

37303



IV CURSO INTERNACIONAL  
DE  
RADIOLOGIA  
VASCULAR E INTERVENCIONISTA  
COMO ALTERNATIVA TERAPEUTICA

# SYLLABUS

directores  
M. MAYNAR  
W.R. CASTAÑEDA-ZUÑIGA  
secretario  
G. LEON

Patrocinado conjuntamente por  
UNIVERSIDAD DE LAS PALMAS DE GRAN CANARIA  
HOSPITAL NUESTRA SEÑORA DEL PINO  
LAS PALMAS. ISLAS CANARIAS. ESPAÑA

  
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UNIVERSITY OF MINNESOTA MEDICAL SCHOOL  
MINNEAPOLIS. USA



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**LAS PALMAS DE G. CANARIA**  
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EL JEFE DE LA CASA DE  
S. M. EL REY

nv.  
49/94

SUS MAJESTADES LOS REYES, accediendo a la peti  
ción que tan amablemente Les ha sido formulada, han  
tenido a bien aceptar la

**PRESIDENCIA DE HONOR**

del "IV CURSO INTERNACIONAL DE RADIOLOGIA VASCULAR  
INTERVENCIONISTA", que se celebrará en Las Palmas de  
Gran Canaria del 22 al 25 de febrero próximo.

Lo que me complace participarle para su cono-  
cimiento y efectos.

PALACIO DE LA ZARZUELA, 28 de Enero de 1994

EL JEFE DE LA CASA DE S.M. EL REY,

SEÑOR RECTOR MAGNIFICO DE LA UNIVERSIDAD DE LAS PALMAS

LAS PALMAS DE GRAN CANARIA

**IV CURSO INTERNACIONAL  
DE  
RADIOLOGIA VASCULAR E INTERVENCIONISTA  
COMO ALTERNATIVA TERAPEUTICA**

**PROGRAMA**

directores  
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UNIVERSITY OF MINNESOTA MEDICAL SCHOOL  
MINNEAPOLIS. USA

**MARTES, 22 de Febrero. 1994**

- 17:00 - 17:30    **INAUGURACION OFICIAL**
- 17:30 - 18:00    **SESION PLENARIA**  
**LA EVOLUCION DE LA RADIOLOGIA CARDIOVASCULAR INTERVENCIONISTA**  
*Dr. W. R. Castañeda - Zúñiga*
- 18:00 - 18:45    **MESA REDONDA**  
**ALTERNATIVAS TERAPEUTICAS EN LA MEDICINA DE HOY**  
*Sr. J. E. Abele*  
*Dr. J. M. Capdevila*  
*Dr. H. Coons*  
*Dr. J. Cueto*  
*Dr. D. D. Liermann*  
*Dr. M. Maynar*  
*Dr. E. Moreno*  
*Sr. R. Ranalli*  
*Dr. P. Rossi*  
*Dr. J. C. Parodi*  
*Moderador : Dr. W. R. Castañeda-Zúñiga*
- SIMPOSIUM SOBRE TROMBOLISIS**
- 18:45 - 18:52    **CONFERENCIA**  
**RESULTADOS A LARGO PLAZO DE LA TERAPEUTICA TROMBOLITICA**  
*Dr. T. O. McNamara*
- 18:52 - 18:59    **CONFERENCIA**  
**LISIS POR SPRAY A IMPULSOS**  
*Dr. K. Kandarpa*
- 18:59 - 19:06    **CONFERENCIA**  
**RESULTADOS A LARGO PLAZO DE LA TERAPEUTICA TROMBOLITICA**  
**EN OCLUSIONES CRONICAS**  
*Dr. J. M. Pulido-Duque*
- 19:06 - 19:13    **CONFERENCIA**  
**RESULTADOS DE LA TERAPEUTICA TROMBOLITICA EN EL MANEJO**  
**DE LA OCLUSION DE LA ARTERIA RENAL**  
*Dr. T. A. Sos*
- 19:13 - 19:20    **CONFERENCIA**  
**TROMBECTOMIA MECANICA**  
*Dr. S. M. Tadavarthy*
- 19:20 - 20:00    **MESA REDONDA**  
**TROMBOLISIS**  
*Dr. K. Kandarpa*  
*Dr. T. O. McNamara*  
*Dr. J. M. Pulido-Duque*  
*Dr. T. A. Sos*  
*Dr. S. M. Tadavarthy*

**MIERCOLES, 23 de Febrero. 1994**

- 07:30 - 07:35 **OBSERVACIONES**  
*Dr. E. Górriz*
- 07:35 - 07:40 **PRESENTACION DEL CASO**  
*Dr. E. Ortiz*
- 07:40 - 08:40 **CASO EN VIVO**  
**TIPS TECNICA DE ROSCH. US IV.**  
*Dr. J. Goicolea (IVUS)*  
*Dr. E. Martin*  
*Dr. M. Maynar*  
*Moderador : Dr. J. Cabrera*
- 08:40 - 08:55 **SEGUIMIENTO DE LOS CASOS DE 1992**  
*Dr. P. Rossi*
- 08:55 - 09:05 **CONFERENCIA**  
**CIRUGIA EN EL HEPATOMA**  
*Dr. E. Moreno*
- 09:05 - 09:15 **CONFERENCIA**  
**TRATAMIENTO PERCUTANEO DEL SINDROME DE BUDD-CHIARI**  
*Dr. M. Fava*
- 09:15 - 09:25 **CONFERENCIA**  
**ESCLEROTERAPIA DE VARICES ESOFAGICAS**  
*Dr. J. Bosch*
- 09:25 - 09:35 **CONFERENCIA**  
**EVOLUCION DE LA TECNICA DEL TIPS**  
*Dr. J. Rosch*
- 09:35 - 09:40 **PRESENTACION DEL CASO**  
*Dr. J. L. Rodríguez San Román*
- 09:40 - 10:40 **CASO EN VIVO**  
**TECNICA DEL TIPS GUIADO POR ULTRASONIDO**  
*Dr. I. Bilbao*  
*Dr. J. Goicolea (IVUS)*  
*Dr. J. M. Longo*  
*Dr. G. M. Richter*  
*Moderador : Dr. J. Rosch*
- 10:40 - 10:50 **CONFERENCIA**  
**U.S. DUPLEX EN EL SEGUIMIENTO DEL TIPS**  
*Dra. J. G. Letourneau*
- 10:50 - 11:00 **CONFERENCIA**  
**RESULTADOS DEL TIPS**  
*Dr. P. Rossi*
- 11:00 - 11:30 **DESCANSO**  
Piscolabis en la zona de la Exposición Técnica

**MIERCOLES, 23 de Febrero. 1994**

- 11:30 - 12:15 **MESA REDONDA**  
**TIPS**  
*Dr. I. Bilbao*  
*Dr. J. Bosch*  
*Dr. J. Cabrera*  
*Dr. M. Maynar*  
*Dr. E. Moreno*  
*Dr. G. M. Richter*  
*Dr. J. Rosch*  
*Dr. P. Rossi*  
*Dr. H. Rousseau*
- 12:15 - 12:20 **PRESENTACION DEL CASO**  
*Dr. J. Hernández - Navarro*
- 12:20 - 13:00 **CASO EN VIVO**  
**PROTESIS ESOFAGICA**  
*Dr. M. Maynar*  
*Dr. J. M. Pulido-Duque*  
*Moderadora : Dra. T. Sala*
- 13:00 - 14:20 **DESCANSO**
- 14:30 - 14:35 **PRESENTACION DEL CASO**  
*Dr. J. Hernández - Navarro*
- 14:35 - 15:30 **CASO EN VIVO**  
**LITOTRIZIA LAPAROSCOPICA POR LASER. TECNICA ENDOSCOPICA**  
*Dr. M. Maynar*  
*Dr. J. Ramírez*  
*Moderador : Dr. A. Soriano*
- 15:30 - 16:20 **MESA REDONDA**  
**INTERVENCION EN PATOLOGIA BILIAR**  
*Dr. J. M. Bordas*  
*Dr. H. G. Coons*  
*Dr. R. F. Dondelinger*  
*Dr. J. Hernández Romero*  
*Dr. E. Moreno*  
*Dr. J. Ramírez*  
*Dr. P. Rossi*  
*Dr. Ch.L. Zollikofer*
- 16:20 - 16:30 **CONFERENCIA**  
**RESULTADOS DEL STENT STRECKER EN LA OBSTRUCCION ESOFAGICA**  
*Dr. R. Reyes*
- 16:30 - 16:40 **CONFERENCIA**  
**PROTESIS ESOFAGICA RECUBIERTA WALLSTENT**  
*Dr. P. Winzeler*

**MIERCOLES, 23 de Febrero. 1994**

- 16:40 - 17:20 **MESA REDONDA**  
**ESOFAGO**  
*Dr. M. Maynar*  
*Dr. J. Rosch*  
*Dra. T. Sala*  
*Dr. P. Strecker*  
*Dr. P. Winzeler*
- 17:20 - 17:50 **DESCANSO**  
Piscolabis en la zona de la Exposición Técnica
- 17:50 - 18:00 **CONFERENCIA**  
**ENDOPROTESIS BILIAR ERCP**  
*Dr. J. M. Bordas*
- 18:00 - 18:10 **CONFERENCIA**  
**LAPAROSCOPIA EN PATOLOGIA ABDOMINAL**  
*Dr. J. Cueto*
- 18:10 - 18:20 **CONFERENCIA**  
**INTERVENCION PANCREATICA PERCUTANEA**  
*Dr. R. F. Dondelinger*
- 18:20 - 18:30 **CONFERENCIA**  
**DRENAJE PERCUTANEO DE ABCESOS**  
*Dr. H. G. Coons*
- 18:30 - 18:40 **CONFERENCIA**  
**ABORDAJE PERCUTANEO VIA BILIAR**  
*Dr. M. Fava*
- 18:40 - 19:30 **MESA REDONDA**  
**TECNICA ENDOSCOPICA VERSUS TECNICA PERCUTANEA**  
*Dr. J. M. Bordas*  
*Dr. J. Bosch*  
*Dr. H. G. Coons*  
*Dr. J. Cueto*  
*Dr. R. F. Dondelinger*  
*Dr. M. Fava*  
*Dra. T. Sala*
- 19:30 **CIERRE**
- 21:30 **COCTEL EN EL HOTEL SANTA CATALINA**



**JUEVES, 24 de Febrero. 1994**

- 07:30 - 07:35    **OBSERVACIONES**  
*Dr. M. Maynar*
- 07:35 - 07:40    **PRESENTACION DEL CASO**  
*Dr. P. Rubio*
- 07:40 - 08:10    **CASO EN VIVO**  
**ANGIOGRAFIA CON CO<sub>2</sub>**  
*Dr. I. F. Hawkins*  
*Dr. L. K. A. Tan*  
*Moderador : Dr. J. M. Rius*
- 08:10 - 08:20    **CONFERENCIA**  
**US DUPLEX Y US INTRAVASCULAR EN LA EVALUACION DIAGNOSTICA**  
**DE LA ENFERMEDAD VASCULAR PERIFERICA**  
*Dra. J. G. Letourneau*
- 08:20 - 08:35    **CONFERENCIA**  
**ANGIOGRAFIA TAC/RNM**  
*Dr. W. P. Th. M. Mali*
- 08:35 - 08:45    **CONFERENCIA**  
**ANGIOGRAFIA DIGITAL CON CO<sub>2</sub>**  
*Dr. I. F. Hawkins*
- 08:45 - 08:55    **CONFERENCIA**  
**NUEVA TECNICA DE ULTRASONIDOS PARA EL FLUJO VOLUMEN ARTERIAL**  
*Sr. R. Ranalli*
- 08:55 - 09:05    **CONFERENCIA**  
**ENDOSCOPIA INTRAVASCULAR**  
*Dr. I. F. Hawkins*
- 09:05 - 09:15    **CONFERENCIA**  
**EXPLORACION FISICA VASCULAR**  
*Dr. P. Lanzer*
- 09:15 - 09:45    **MESA REDONDA**  
**METODOS DE DIAGNOSTICO VASCULAR**  
*Dr. I. F. Hawkins*  
*Dr. P. Lanzer*  
*Dra. J. G. Letourneau*  
*Dr. W. P. Th. M. Mali*  
*Dr. E. Martin*  
*Sr. R. Ranalli*  
*Dr. J. M. Rius*  
*Dr. L. K. A. Tan*
- 09:45 - 10:10    **DESCANSO**  
**Piscolabis en la zona de la Exposición Técnica**

**JUEVES, 24 de Febrero.1994**

- 10:10 - 10:20 **CONFERENCIA**  
PATOFISIOLOGIA DE LA ANGIOPLASTIA  
*Dr. P. M. Consigny*
- 10:20 - 10:25 **PRESENTACION DEL CASO**  
*Dr. E. Sotgiu*
- 10:25 - 11:10 **CASO EN VIVO**  
ANGIOPLASTIA TIBIOPERONEAL  
*Dr. T. Garnica*  
*Dr. T. A. Sos*  
*Moderador : Dr. V. Cabrera*
- 11:10 - 11:20 **CONFERENCIA**  
ATP. EL PUNTO DE VISTA DEL CIRUJANO  
*Dr. J. M. Estevan - Solano*
- 11:20 - 11:30 **CONFERENCIA**  
RESULTADOS A LARGO PLAZO DE LA ATP EN LA ENFERMEDAD VASCULAR PERIFERICA  
*Dr. C. J. Tegtmeier*
- 11:30 - 12:15 **MESA REDONDA**  
ANGIOPLASTIA  
*Dr. V. Cabrera*  
*Dr. J. M. Estevan - Solano*  
*Dr. P. Lanzer*  
*Dr. P. M. Consigny*  
*Dr. E. C. Martin*  
*Dr. D. E. Schwarten*  
*Dr. T. A. Sos*  
*Dr. P. Strecker*  
*Dr. C. J. Tegtmeier*
- 12:15 - 12:20 **PRESENTACION DEL CASO**  
*Dra. A. Cruz*
- 12:20 - 13:00 **CASO EN VIVO**  
PROTESIS FEMOROPOPLITEA US IV.  
*Dr. H. G. Coons*  
*Dr. A. H. Cragg*  
*Dr. J. Goicolea (IVUS)*  
*Moderador : Dr. J. A. Jiménez - Cossío*
- 13:00 - 14:20 **DESCANSO**
- 14:30 - 14:35 **PRESENTACION DEL CASO**  
*Dra. M. Hermida*

**JUEVES, 24 de Febrero. 1994**

- 14:35 - 15:45 CASO EN VIVO**  
**PROTESIS INTRA-AORTICA PERCUTANEA**  
*Dr. V. Cabrera*  
*Dr. J. Goicolea (IVUS)*  
*Dr. J. C. Parodi*  
*Dr. C. J. Schonholz*  
*Dr. J. Palmaz*  
*Moderador : Dr. J. M. Capdevila*
- 15:45 - 16:15 MESA REDONDA**  
**PROTESIS PERCUTANEAS**  
*Dr. J. M. Callejas*  
*Dr. J. M. Capdevila*  
*Dr. A. H. Cragg*  
*Dra. C. Cuesta*  
*Dr. E. C. Martin*  
*Dr. J. Palmaz*  
*Dr. J. C. Parodi*  
*Dr. Ph. Piquet*  
*Dr. P. Strecker*
- 16:15 - 16:25 CONFERENCIA**  
**CIRUGIA ILIACO-FEMORAL**  
*Dr. V. Cabrera*
- 16:25 - 16:35 CONFERENCIA**  
**CIRUGIA DEL ANEURISMA DE AORTA**  
*Dra. C. Cuesta*
- 16:35 - 16:45 CONFERENCIA**  
**PROTESIS AORTICAS PERCUTANEAS**  
*Dr. J. C. Parodi*
- 16:45 - 16:55 CONFERENCIA**  
**RESULTADOS DE LAS PROTESIS AORTICAS PERCUTANEAS**  
*Dr. Ph. Piquet*
- 16:55 - 17:05 CONFERENCIA**  
**ANGIOPLASTIA TRANSLUMINAL Y TROMBOLISIS CON PENTASON-POLISULFATO DE SODIO POR VIA ORAL**  
*Dr. L. Horvath*
- 17:05 - 17:15 CONFERENCIA**  
**RESULTADOS DE LAS PROTESIS FEMOROPOPLITEAS PERCUTANEAS**  
*Dr. A. H. Cragg*
- 17:15 - 17:25 CONFERENCIA**  
**STENTS INTRAVASCULARES EN LA ENFERMEDAD VASCULAR PERIFERICA**  
*Dr. E. C. Martin*
- 17:25 - 17:45 DESCANSO**  
**Piscobabis en la zona de la Exposición Técnica**

**JUEVES, 24 de Febrero. 1994**

**17:45 - 18:30 MESA REDONDA**  
**ALTERNATIVAS TERAPEUTICAS EN LA PATOLOGIA OCLUSIVA**  
*Dr. V. Cabrera*  
*Dr. A. H. Cragg*  
*Dra. C. Cuesta*  
*Dr. J. M. Estevan - Solano*  
*Dr. T. O. McNamara*  
*Dr. C. J. Schonholz*  
*Dr. S. M. Tadavarthy*

**SIMPOSIUM SOBRE STENTS**

**18:30 - 18:37 CONFERENCIA**  
**RESULTADOS A LARGO PLAZO DEL STENT STRECKER EN ARTERIA ILIACA**  
*Dr. P. Strecker*

**18:37 - 18:44 CONFERENCIA**  
**RESULTADOS DE LOS STENTS EN ARTERIA FEMORAL**  
*Dr. D. D. Liermann*

**18:44 - 18:51 CONFERENCIA**  
**USO DEL STENT BALON EXPANDIBLE EN COMBINACION CON PTFE**  
*Dr. J. C. Palmaz*

**18:51 - 18:58 CONFERENCIA**  
**RESULTADOS A LARGO PLAZO DEL WALLSTENT**  
*Dr. Ch. L. Zollikofer*

**18:58 - 19:05 CONFERENCIA**  
**RESULTADOS A LARGO PLAZO DEL STENT GIANTURCO EN VENAS**  
*Dr. J. Rosch*

**19:05 - 19:45 MESA REDONDA**  
**PROTESIS VERSUS STENT**  
*Dr. J. M. Callejas*  
*Dr. J. A. Jiménez - Cossío*  
*Dr. D. D. Lierman*  
*Dr. E. C. Martin*  
*Dr. J. C. Palmaz*  
*Dr. J. Rosch*  
*Dr. P. Strecker*  
*Dr. Ch. L. Zollikofer*

**19:45 CIERRE**

**VIERNES, 25 de Febrero. 1994**

- 07:30 - 07:35 **OBSERVACIONES**  
*Dr. M. Maynar*
- 07:35 - 07:40 **PRESENTACION DEL CASO**  
*Dr. J. Carreira*
- 07:40 - 08:25 **CASO EN VIVO**  
**EMBOLIZACION DE MALFORMACION A-V**  
*Dr. E. Górriz*  
*Dr. L. Guimaraens*  
*Dr. F. Yakes*  
*Moderador: Dr. J. M. Estevan-Solano*
- 08:25 - 08:35 **CONFERENCIA**  
**ACTUALIZACION DE LA EMBOLIZACION**  
*Dr. M. A. Herrera*
- 08:35 - 09:05 **MESA REDONDA**  
**EMBOLIZACION**  
*Dr. J. M. Estevan-Solano*  
*Dr. L. Guimaraens*  
*Dr. M. A. Herrera*  
*Dr. J. Theron*  
*Dr. J. Rosch*  
*Dr. P. Strecker*  
*Dr. F. Yakes*
- 09:05 - 09:35 **DESCANSO**  
Piscolabis en la zona de la Exposición Técnica.
- 09:35 - 09:40 **PRESENTACION DEL CASO**  
*Dr. C. Campo*
- 09:40 - 10:10 **CASO EN VIVO**  
**RESERVORIO PARA ACCESO VASCULAR**  
*Dr. R. Reyes*  
*Dr. J. M. Pulido-Duque*  
*Moderador: Dr. J. Aguiar*
- 10:10 - 10:20 **CONFERENCIA**  
**VASCULITIS DE TAKAYASU: RESULTADOS DE LA ATP**  
*Dr. L. K. A. Tan*
- 10:20 - 11:00 **MESA REDONDA**  
**CATETERES PERMANENTES**  
*Dr. C. Campo*  
*Dr. J. A. Jiménez-Cossío*  
*Dr. R. Reyes*  
*Dr. T. A. Sos*  
*Dr. L. K. A. Tan*  
*Dr. C. J. Tegtmeyer*

**VIERNES, 25 de Febrero. 1994**

- 11:00 - 11:05 **PRESENTACION DEL CASO**  
*Dra. V. Alvarez-Santullano*
- 11:05 - 12:05 **CASO EN VIVO**  
**ANGIOPLASTIA CAROTIDEA**  
*Dr. J. Goicolea (IVUS)*  
*Dr. D. E. Swarten*  
*Dr. C. J. Tegmeyer*  
*Moderador: Dra. C. Cuesta*
- 12:05 - 12:15 **CONFERENCIA**  
**ANGIOPLASTIA BRAQUIOCEFALICA**  
*Dr. J. Theron*
- 12:15 - 12:25 **CONFERENCIA**  
**ANGIOPLASTIA CAROTIDEA. RESULTADOS DEL PROTOCOLO U.S.A.**  
*Dr. D. E. Swarten*
- 12:25 - 13:00 **MESA REDONDA**  
**ANGIOPLASTIA SUPRAORTICA**  
*Dra. V. Alvarez-Santullano*  
*Dr. V. Cabrera*  
*Dr. A. Cubero*  
*Dra. C. Cuesta*  
*Dr. L. Guimaraens*  
*Dr. A. Mayol*  
*Dr. D. E. Swarten*  
*Dr. C. J. Tegmeyer*  
*Dr. J. Theron*
- SIMPOSIUM SOBRE HIPERTENSION RENO-VASCULAR**
- 13:00 - 13:07 **CONFERENCIA**  
**ATP EN LA HIPERTENSION RENO-VASCULAR**  
*Dr. T. A. Sos*
- 13:07 - 13:14 **CONFERENCIA**  
**ATERECTOMIA EN LA ESTENOSIS DE ARTERIA RENAL**  
*Dr. C. J. Tegmeyer*
- 13:14 - 13:21 **CONFERENCIA**  
**STENTS PALMAZ EN LA ESTENOSIS DE ARTERIA RENAL**  
*Dr. J. C. Palmaz*
- 13:21 - 13:28 **CONFERENCIA**  
**WALLSTENT EN LA ESTENOSIS DE ARTERIA RENAL**  
*Dr. H. Rousseau*
- 13:28 - 13:35 **CONFERENCIA**  
**CIRUGIA EN LA HIPERTENSION RENO-VASCULAR**  
*Dr. J. M. Capdevila*



**VIERNES, 25 de Febrero. 1994**

**13:35 - 13:42    CONFERENCIA**  
**TRATAMIENTO MEDICO DE LA HIPERTENSION RENO-VASCULAR**  
*Dr. J. C. Rodríguez*

**13:42 - 14:15    MESA REDONDA**  
**HIPERTENSION RENO-VASCULAR**  
*Dr. J. M. Callejas*  
*Dr. J. M. Capdevila*  
*Dr. T. O. McNamara*  
*Dr. J. C. Palmaz*  
*Dr. J. C. Rodríguez*  
*Dr. H. Rousseau*  
*Dr. D. E. Swarten*  
*Dr. T. A. Sos*  
*Dr. C. J. Tegtmeyer*

**14:15            CLAUSURA**  
*Dr. M. Maynar*

**Cuidado de pacientes**  
**Dr. C. AÑEZ**  
**Dr.A. HERNANDEZ**  
**Dr.J. MAYNAR**  
**Dr.A. RODRIGUEZ**  
**Dr.J. RUBIO**

## IV CURSO INTERNACIONAL DE RADIOLOGIA VASCULAR INTERVENCIONISTA COMO ALTERNATIVA TERAPEUTICA

### CONFERENCIAS

#### CIRUGIA EN HEPATOMA

Dr. E. Moreno, (10')

#### TRATAMIENTO PERCUTANEO DEL SINDROME DE BUDD-CHIARI

Dr. Mario Fava (10')

#### ESCLEROTERAPIA DE VARICES ESOFAGICAS

Dr. J. Bosch, (10')

#### EVOLUCION DE LA TECNICA DEL TIPS

Dr. J. Rosch, (10')

#### U.S. DUPLEX EN EL SEGUIMIENTO DEL TIPS

Dra. J.G. Letourneau, (10')

#### RESULTADOS DEL TIPS

Dr. P. Rossi, (10')

#### RESULTADOS DEL STENT STRECKER EN LA OBSTRUCCION ESOFAGICA

Dr. R. Reyes, (10')

#### PROTESIS ESOFAGICA RECUBIERTA WALLSTENT

Dr. P. Winzeler, (10')

#### ENDOPROTESIS BILIAR. ERCP

Dr. J.M. Bordas, (10')

#### LAPAROSCOPIA EN PATOLOGIA ABDOMINAL

Dr. J. Cueto-García, (10')

#### INTERVENCION PANCREATICA PERCUTANEA

Dr. R.F. Dondelinger, (10')

#### DRENAJE PERCUTANEO DE ABCESOS

Dr. H.G. Coons, (10')

#### U.S. DUPLEX E INTRAVASCULAR EN LA EVALUACION DIAGNOSTICA DE LA ENFERMEDAD VASCULAR PERIFERICA.

Dra. J.G. Letourneau, (10')

#### ANGIOGRAFIA TAC/RNM

Dr. W.P.Th.M. Mali, (15')

#### ANGIOGRAFIA DIGITAL CON CO<sub>2</sub>

Dr. I.F. Hawkins, (10')

#### NUEVA TECNICA DE ULTRASONIDOS PARA EL FLUJO VOLUMEN ARTERIAL

Sr. R. Ranalli (10')

#### ENDOSCOPIA INTRAVASCULAR

Dr. I.F. Hawkins, (10')

#### EXPLORACION FISICA VASCULAR

Dr. P. Lanzer, (10')

#### PATOFISIOLOGIA DE LA ANGIOPLASTIA

Dr. P. M. Consigny (10')

#### ATP. EL PUNTO DE VISTA DEL CIRUJANO

Dr. J.M. Estevan, (10')

#### RESULTADOS A LARGO PLAZO DE LA ATP EN LA ENFERMEDAD VASCULAR PERIFERICA

Dr. C.J. Tegtmeier, (10')

#### CIRUGIA ILIACO-FEMORAL

Dr. V. Cabrera, (10')

#### CIRUGIA DEL ANEURISMA DE AORTA

Dra. C. Cuesta, (10')

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#### STENTS INTRAVASCULARES EN LA ENFERMEDAD VASCULAR PERIFERICA

Dr. E.C. Martin, (10')

#### ACTUALIZACION DE LA EMBOLIZACION

Dr. M.A. Herrera, (10')

#### VASCULITIS DE TAKAYASU: RESULTADOS DE LA ATP

Dr. L.K.A. Tan, (10')

#### ANGIOPLASTIA BRAQUIOCEFALICA

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#### ANGIOPLASTIA CAROTIDEA. RESULTADOS DEL PROTOCOLO U.S.A.

Dr. D.E. Swarten, (10')

### SIMPOSIUM SOBRE TROMBOLISIS

#### RESULTADOS A LARGO PLAZO DE LA TERAPEUTICA TROMBOLITICA

Dr. T.O. McNamara, (7')

#### LISIS POR SPRAY A IMPULSOS

Dr. K. Kandarpa, (7')

#### RESULTADOS A LARGO PLAZO DE LA TERAPEUTICA TROMBOLITICA EN OCLUSIONES CRONICAS

Dr. J.M. Pulido-Duque, (7')

#### RESULTADOS DE LA TERAPEUTICA TROMBOLITICA EN EL MANEJO DE LA OCLUSION DE LA ARTERIA RENAL

Dr. T.A. Sos, (7')

## **IV CURSO INTERNACIONAL DE RADIOLOGIA VASCULAR INTERVENCIONISTA COMO ALTERNATIVA TERAPEUTICA**

### **TROMBECTOMIA MECANICA**

Dr. S.M. Tadavarthi, (7')

### **TERAPIA TROMBOLITICA EN OCLUSION DE ARTERIA RENAL**

Dr. M. Fava, (7')

### **SIMPOSIUM SOBRE STENTS**

#### **RESULTADOS A LARGO PLAZO DEL STENT STRECKER EN ARTERIA ILIACA**

Dr. P. Strecker, (7')

#### **RESULTADOS DE LOS STENTS EN ARTERIA FEMORAL**

Dr. D.D. Liermann, (7')

#### **USO DEL STENT BALON-EXPANDIBLE EN COMBINACION CON PTFE**

Dr. J.C. Palmaz, (7')

#### **RESULTADOS A LARGO PLAZO DEL WALLSTENT**

Dr. Ch.L. Zollikofer, (7')

#### **RESULTADOS A LARGO PLAZO DEL STENT GIANTURCO EN VENAS**

Dr. J. Rosch, (7')

### **SIMPOSIUM SOBRE HIPERTENSION RENO-VASCULAR**

#### **ATP EN LA HIPERTENSION RENO-VASCULAR**

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#### **ATERECTOMIA EN ESTENOSIS DE ARTERIA RENAL**

Dr. C.J. Tegtmeier, (7')

#### **STENTS PALMAZ EN ESTENOSIS DE ARTERIA RENAL**

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#### IV CURSO INTERNACIONAL DE RADIOLOGIA VASCULAR INTERVENCIONISTA COMO ALTERNATIVA TERAPEUTICA

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**FOURTH INTERNATIONAL COURSE  
ON  
VASCULAR AND INTERVENTIONAL RADIOLOGY  
AS A THERAPEUTIC ALTERNATIVE**

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**TUESDAY, February 22, 1994**

- 17:00 - 17:30    **OFFICIAL OPENING**
- 17:30 - 18:00    **PLENARY LECTURE**  
**THE EVOLUTION OF CARDIOVASCULAR AND INTERVENTIONAL RADIOLOGY**  
*W. R. Castañeda - Zúñiga, M.D.*
- 18:00 - 18:45    **PANEL DISCUSSION**  
**THERAPEUTIC ALTERNATIVES IN MEDICINE TODAY**  
*Mr. J. E. Abele*  
*J. M. Capdevila, M.D.*  
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*P. Rossi, M.D.*  
*J. C. Parodi, M.D.*  
*Moderator : W. R. Castañeda-Zúñiga, M.D.*
- SYMPOSIUM ON THROMBOLYSIS**
- 18:45 - 18:52    **LECTURE**  
**LONG TERM RESULTS ON THROMBOLYTIC THERAPY**  
*T. O. McNamara, M.D.*
- 18:52 - 18:59    **LECTURE**  
**PULSE SPRAY LYSIS**  
*K. Kandarpa, M.D.*
- 18:59 - 19:06    **LECTURE**  
**LONG TERM RESULTS OF THROMBOLYTIC THERAPY**  
**IN CHRONIC OCCLUSIONS**  
*J. M. Pulido-Duque, M.D.*
- 19:06 - 19:13    **LECTURE**  
**RESULTS OF THROMBOLYTIC THERAPY IN THE MANAGEMENT**  
**OF RENAL ARTERY OCCLUSION**  
*T. A. Sos, M.D.*
- 19:13 - 19:20    **LECTURE**  
**MECHANICAL THROMBECTOMY**  
*S. M. Tadavarthy, M.D.*
- 19:20 - 20:00    **PANEL DISCUSSION**  
**THROMBOLYSIS**  
*K. Kandarpa, M.D.*  
*T. O. McNamara, M.D.*  
*J. M. Pulido-Duque, M.D.*  
*T. A. Sos, M.D.*  
*S. M. Tadavarthy, M.D.*

**WEDNESDAY, February 23, 1994**

- 07:30 - 07:35    **REMARKS**  
*E. Górriz, M.D.*
- 07:35 - 07:40    **CASE PRESENTATION**  
*E. Ortiz, M.D.*
- 07:40 - 08:40    **LIVE CASE**  
**TIPS ROSCH TECHNIQUE. IVUS**  
*J. Goicolea, M.D. (IVUS)*  
*E. Martín, M.D.*  
*M. Maynar, M.D.*  
*Moderator : J. Cabrera, M.D.*
- 08:40 - 08:55    **FOLLOW-UP 1992 CASES**  
*P. Rossi, M.D.*
- 08:55 - 09:05    **LECTURE**  
**SURGERY IN HEPATOMA**  
*E. Moreno, M.D.*
- 09:05 - 09:15    **LECTURE**  
**PERCUTANEOUS TREATMENT OF BUDD-CHIARI'S SYNDROME**  
*M. Fava, M.D.*
- 09:15 - 09:25    **LECTURE**  
**SCLEROTHERAPY OF ESOPHAGEAL VARICES**  
*J. Bosch, M.D.*
- 09:25 - 09:35    **LECTURE**  
**TECHNICAL EVOLUTION OF TIPS**  
*J. Rosch, M.D.*
- 09:35 - 09:40    **CASE PRESENTATION**  
*J. L. Rodríguez San Román, M.D.*
- 09:40 - 10:40    **LIVE CASE**  
**TIPS ULTRASOUND GUIDED TECHNIQUE**  
*I. Bilbao, M.D.*  
*J. Goicolea, M.D. (IVUS)*  
*J. M. Longo, M.D.*  
*G. M. Richter, M.D.*  
*Moderator : J. Rosch, M.D.*
- 10:40 - 10:50    **LECTURE**  
**DUPLEX U.S. IN TIPS FOLLOW-UP**  
*J. G. Letourneau, M.D.*
- 10:50 - 11:00    **LECTURE**  
**TIPS RESULTS**  
*P. Rossi, M.D.*
- 11:00 - 11:30    **BREAK**  
**Coffee and refreshments at the Technical Exhibits area**

**WEDNESDAY, February 23, 1994**

- 11:30 - 12:15**    **PANEL DISCUSSION**  
**TIPS**  
*I. Bilbao, M.D.*  
*J. Bosch, M.D.*  
*J. Cabrera, M.D.*  
*M. Maynar, M.D.*  
*E. Moreno, M.D.*  
*G. M. Richter, M.D.*  
*J. Rosch, M.D.*  
*P. Rossi, M.D.*  
*H. Rousseau, M.D.*
- 12:15 - 12:20**    **CASE PRESENTATION**  
*J. Hernández - Navarro, M.D.*
- 12:20 - 13:00**    **LIVE CASE**  
**ESOPHAGEAL PROSTHESIS**  
*M. Maynar, M.D.*  
*J. M. Pulido-Duque, M.D.*  
*Moderator : T. Sala, M.D.*
- 13:00 - 14:20**    **BREAK**
- 14:30 - 14:35**    **CASE PRESENTATION**  
*J. Hernández - Navarro, M.D.*
- 14:35 - 15:30**    **LIVE CASE**  
**LAPAROSCOPY LASER LITHOTRIPSY. ENDOSCOPIC GUIDING**  
*M. Maynar, M.D.*  
*J. Ramírez, M.D.*  
*Moderator : A. Soriano, M.D.*
- 15:30 - 16:20**    **PANEL DISCUSSION**  
**INTERVENTION IN BILIARY PATHOLOGY**  
*J. M. Bordas, M.D.*  
*H. G. Coons, M.D.*  
*R. F. Dondelinger, M.D.*  
*J. Hernández Romero, M.D.*  
*E. Moreno, M.D.*  
*J. Ramírez, M.D.*  
*P. Rossi, M.D.*  
*Ch.L. Zollikofer, M.D.*
- 16:20 - 16:30**    **LECTURE**  
**RESULTS OF STRECKER STENT IN ESOPHAGEAL OBSTRUCTION**  
*R. Reyes, M.D.*
- 16:30 - 16:40**    **LECTURE**  
**THE COVERED OESOPHAGEAL WALLSTENT**  
*P. Winzeler, M.D.*

**WEDNESDAY, February 23, 1994**

- 16:40 - 17:20**    **PANEL DISCUSSION**  
**ESOPHAGUS**  
*M. Maynar, M.D.*  
*J. Rosch, M.D.*  
*T. Sala, M.D.*  
*P. Strecker, M.D.*  
*P. Winzeler, M.D.*
- 17:20 - 17:50**    **BREAK**  
Coffee and refreshments at the Technical Exhibits area
- 17:50 - 18:00**    **LECTURE**  
**ERCP. BILIARY ENDOPROSTHESIS**  
*J. M. Bordas, M.D.*
- 18:00 - 18:10**    **LECTURE**  
**LAPAROSCOPY IN ABDOMINAL PATHOLOGY**  
*J. Cueto, M.D.*
- 18:10 - 18:20**    **LECTURE**  
**PERCUTANEOUS PANCREATIC INTERVENTION**  
*R. F. Dondelinger, M.D.*
- 18:20 - 18:30**    **LECTURE**  
**PERCUTANEOUS ABSCESS DRAINAGE**  
*H. G. Coons, M.D.*
- 18:30 - 18:40**    **LECTURE**  
**PERCUTANEOUS APPROACH OF THE BILIARY TRACT**  
*M. Fava, M.D.*
- 18:40 - 19:30**    **PANEL DISCUSSION**  
**ENDOSCOPIC VERSUS PERCUTANEOUS TECHNIQUE**  
*J. M. Bordas, M.D.*  
*J. Bosch, M.D.*  
*H. G. Coons, M.D.*  
*J. Cueto, M.D.*  
*R. F. Dondelinger, M.D.*  
*M. Fava, M.D.*  
*T. Sala, M.D.*
- 19:30**            **ADJOURN**
- 21:30**            **COCKTAIL AT THE SANTA CATALINA HOTEL**

**THURSDAY, February 24, 1994**

- 07:30 - 07:35    **REMARKS**  
*M. Maynar, M.D.*
- 07:35 - 07:40    **CASE PRESENTATION**  
*P. Rubio, M.D.*
- 07:40 - 08:10    **LIVE CASE**  
**CO<sub>2</sub> ANGIOGRAPHY**  
*I. F. Hawkins, M.D.*  
*L. K. A. Tan, M.D.*  
*Moderator : J. M. Rius, M.D.*
- 08:10 - 08:20    **LECTURE**  
**DUPLEX US AND IV US IN THE DIAGNOSTIC EVALUATION**  
**OF PERIPHERAL VASCULAR DISEASE**  
*J. G. Letourneau, M.D.*
- 08:20 - 08:35    **LECTURE**  
**CT/RM ANGIOGRAPHY**  
*W. P. Th. M. Mali, M.D.*
- 08:35 - 08:45    **LECTURE**  
**CO<sub>2</sub> DIGITAL ANGIOGRAPHY**  
*I. F. Hawkins, M.D.*
- 08:45 - 08:55    **LECTURE**  
**ARTERIAL VOLUME FLOW BY A NEW ULTRASONIC TECHNIQUE**  
*Mr. R. Ranalli*
- 08:55 - 09:05    **LECTURE**  
**INTRAVASCULAR ENDOSCOPY**  
*I. F. Hawkins, M.D.*
- 09:05 - 09:15    **LECTURE**  
**VASCULAR PHYSICAL EXAMINATION**  
*P. Lanzer, M.D.*
- 09:15 - 09:45    **PANEL DISCUSSION**  
**VASCULAR DIAGNOSIS METHODS**  
*I. F. Hawkins, M.D.*  
*P. Lanzer, M.D.*  
*J. G. Letourneau, M.D.*  
*W. P. Th. M. Mali, M.D.*  
*E. Martin, M.D.*  
*Mr. R. Ranalli*  
*J. M. Rius, M.D.*  
*L. K. A. Tan, M.D.*
- 09:45 - 10:10    **BREAK**  
Coffee and refreshments at the Technical Exhibits area

**THURSDAY, February 24. 1994**

- 10:10 - 10:20 **LECTURE**  
PATHOPHYSIOLOGY OF ANGIOPLASTY  
*P. M. Consigny, M.D.*
- 10:20 - 10:25 **CASE PRESENTATION**  
*E. Sotgiu, M.D.*
- 10:25 - 11:10 **LIVE CASE**  
TIBIOPERONEAL ANGIOPLASTY  
*T. Garnica, M.D.*  
*T. A. Sos, M.D.*  
*Moderator : V. Cabrera, M.D.*
- 11:10 - 11:20 **LECTURE**  
PTA. SURGEONS POINT OF VIEW  
*J. M. Estevan - Solano, M.D.*
- 11:20 - 11:30 **LECTURE**  
LONG TERM RESULTS OF PTA IN PERIPHERAL VASCULAR DISEASE  
*C. J. Tegtmeier, M.D.*
- 11:30 - 12:15 **PANEL DISCUSSION**  
ANGIOPLASTY  
*V. Cabrera, M.D.*  
*J. M. Estevan - Solano, M.D.*  
*P. Lanzer, M.D.*  
*P. M. Consigny, M.D.*  
*E. C. Martin, M.D.*  
*D. E. Swarten, M.D.*  
*T. A. Sos, M.D.*  
*P. Strecker, M.D.*  
*C. J. Tegtmeier, M.D.*
- 12:15 - 12:20 **CASE PRESENTATION**  
*A. Cruz, M.D.*
- 12:20 - 13:00 **LIVE CASE**  
FEMOROPOPLITEAL GRAFT IVUS  
*H. G. Coons, M.D.*  
*A. H. Cragg, M.D.*  
*J. Goicolea, M.D. (IVUS)*  
*Moderator : J. A. Jiménez - Cossío, M.D.*
- 13:00 - 14:20 **BREAK**
- 14:30 - 14:35 **CASE PRESENTATION**  
*M. Hermida, M.D.*

**THURSDAY, February 24, 1994**

- 14:35 - 15:45 LIVE CASE**  
**PERCUTANEOUS INTRA-AORTIC GRAFT**  
*V. Cabrera, M.D.*  
*J. Goicolea, M.D. (IVUS)*  
*J. C. Parodi, M.D.*  
*C. J. Schonholz, M.D.*  
*J. Palmaz, M.D.*  
*Moderator : J. M. Capdevila, M.D.*
- 15:45 - 16:15 PANEL DISCUSSION**  
**PERCUTANEOUS GRAFT**  
*J. M. Callejas, M.D.*  
*J. M. Capdevila, M.D.*  
*A. H. Cragg, M.D.*  
*C. Cuesta, M.D.*  
*E. C. Martin, M.D.*  
*J. Palmaz, M.D.*  
*J. C. Parodi, M.D.*  
*Ph. Piquet, M.D.*  
*P. Strecker, M.D.*
- 16:15 - 16:25 LECTURE**  
**ILIAC-FEMORAL SURGERY**  
*V. Cabrera, M.D.*
- 16:25 - 16:35 LECTURE**  
**AORTIC ANEURISM SURGERY**  
*C. Cuesta, M.D.*
- 16:35 - 16:45 LECTURE**  
**PERCUTANEOUS AORTIC GRAFT**  
*J. C. Parodi, M.D.*
- 16:45 - 16:55 LECTURE**  
**RESULTS OF PERCUTANEOUS AORTIC GRAFTING**  
*Ph. Piquet, M.D.*
- 16:55 - 17:05 LECTURE**  
**TRANSLUMINAL ANGIOPLASTY AND EFFECTIVE THROMBOLYSIS  
WITH SODIUM PENTOSANE GIVEN ORALLY**  
*L. Horvath, M.D.*
- 17:05 - 17:15 LECTURE**  
**RESULTS OF PERCUTANEOUS FEMOROPOPLITEAL GRAFTING**  
*A. H. Cragg, M.D.*
- 17:15 - 17:25 LECTURE**  
**INTRAVASCULAR STENTS IN PERIPHERAL VASCULAR DISEASE**  
*E. C. Martin, M.D.*
- 17:25 - 17:45 BREAK**  
Coffee and refreshments at the Technical Exhibits area

**THURSDAY, February 24, 1994**

- 17:45 - 18:30 PANEL DISCUSSION**  
**THERAPEUTIC ALTERNATIVES ON OCCLUSION PATHOLOGY**  
*V. Cabrera, M.D.*  
*A. H. Cragg, M.D.*  
*C. Cuesta, M.D.*  
*J. M. Estevan - Solano, M.D.*  
*T. O. McNamara, M.D.*  
*C. J. Schonholz, M.D.*  
*S. M. Tadavarthy, M.D.*

**SYMPOSIUM ON STENTS**

- 18:30 - 18:37 LECTURE**  
**LONG TERM RESULTS OF STRECKER STENT IN ILLIAC ARTERY**  
*P. Strecker, M.D.*
- 18:37 - 18:44 LECTURE**  
**RESULTS OF FEMORAL ARTERY STENTING**  
*D. D. Liermann, M.D.*
- 18:44 - 18:51 LECTURE**  
**USES OF BALLOON EXPANDABLE STENTS IN COMBINATION WITH PTFE**  
*J. C. Palmaz, M.D.*
- 18:51 - 18:58 LECTURE**  
**LONG TERM RESULTS OF WALLSTENT**  
*Ch. L. Zollikofer, M.D.*
- 18:58 - 19:05 LECTURE**  
**LONG TERM RESULTS OF GIANTURCO STENTS IN VEINS**  
*J. Rosch, M.D.*
- 19:05 - 19:45 PANEL DISCUSSION**  
**GRAFT VERSUS STENT**  
*J. M. Callejas, M.D.*  
*J. A. Jiménez - Cossío, M.D.*  
*D. D. Liermann, M.D.*  
*E. C. Martin, M.D.*  
*J. C. Palmaz, M.D.*  
*J. Rosch, M.D.*  
*P. Strecker, M.D.*  
*Ch. L. Zollikofer, M.D.*

**19:45 ADJOURN**



**FRIDAY, February 25, 1994**

- 07:30 - 07:35    **REMARKS**  
*M. Maynar, M.D.*
- 07:35 - 07:40    **CASE PRESENTATION**  
*J. Carreira, M.D.*
- 07:40 - 08:25    **LIVE CASE**  
**A-V MALFORMATION EMBOLIZATION**  
*E. Górriz, M.D.*  
*L. Guimaraens, M.D.*  
*F. Yakes, M.D.*  
*Moderator: J. M. Estevan-Solano, M.D.*
- 08:25 - 08:35    **LECTURE**  
**EMBOUZATION UPDATE**  
*M. A. Herrera, M.D.*
- 08:35 - 09:05    **PANEL DISCUSSION**  
**EMBOUZATION**  
*J. M. Estevan-Solano, M.D.*  
*L. Guimaraens, M.D.*  
*M. A. Herrera, M.D.*  
*J. Theron, M.D.*  
*J. Rosch, M.D.*  
*P. Strecker, M.D.*  
*F. Yakes, M.D.*
- 09:05 - 09:35    **BREAK**  
Coffee and refreshments at the Technical Exhibits area
- 09:35 - 09:40    **CASE PRESENTATION**  
*C. Campo, M.D.*
- 09:40 - 10:10    **LIVE CASE**  
**VASCULAR ACCESS PORT**  
*R. Reyes, M.D.*  
*J. M. Pulido-Duque, M.D.*  
*Moderator: J. Aguiar, M.D.*
- 10:10 - 10:20    **LECTURE**  
**TAKAYASU'S VASCULITIS: PTA RESULTS**  
*L. K. A. Tan, M.D.*
- 10:20 - 11:00    **PANEL DISCUSSION**  
**INDWELLING CATHETERS**  
*C. Campo, M.D.*  
*J. A. Jiménez-Cossío, M.D.*  
*R. Reyes, M.D.*  
*T. A. Sos, M.D.*  
*L. K. A. Tan, M.D.*  
*C. J. Tegtmeyer, M.D.*

**FRIDAY, February 25, 1994**

- 11:00 - 11:05 **CASE PRESENTATION**  
*V. Alvarez-Santullano, M.D.*
- 11:05 - 12:05 **LIVE CASE**  
**CAROTID ANGIOPLASTY**  
*J. Goicolea, M.D. (IVUS)*  
*D. E. Swarten, M.D.*  
*C. J. Tegtmeyer, M.D.*  
*Moderator: C. Cuesta, M.D.*
- 12:05 - 12:15 **LECTURE**  
**BRACHIO-CEPHALIC ANGIOPLASTY**  
*J. Theron, M.D.*
- 12:15 - 12:25 **LECTURE**  
**CAROTID ANGIOPLASTY RESULTS OF U.S.A. CLINICAL TRIAL**  
*D. E. Swarten, M.D.*
- 12:25 - 13:00 **PANEL DISCUSSION**  
**SUPRA-AORTIC ANGIOPLASTY**  
*V. Alvarez-Santullano, M.D.*  
*V. Cabrera, M.D.*  
*A. Cubero, M.D.*  
*C. Cuesta, M.D.*  
*L. Guimaraens, M.D.*  
*A. Mayol, M.D.*  
*D. E. Swarten, M.D.*  
*C. J. Tegtmeyer, M.D.*  
*J. Theron, M.D.*
- SYMPOSIUM ON RENO-VASCULAR HYPERTENSION**
- 13:00 - 13:07 **LECTURE**  
**PTA IN RENO-VASCULAR HYPERTENSION**  
*T. A. Sos, M.D.*
- 13:07 - 13:14 **LECTURE**  
**ATHERECTOMY IN RENAL ARTERY STENOSIS**  
*C. J. Tegtmeyer, M.D.*
- 13:14 - 13:21 **LECTURE**  
**PALMAZ STENTS IN RENAL ARTERY STENOSIS**  
*J. C. Palmaz, M.D.*
- 13:21 - 13:28 **LECTURE**  
**WALLSTENT IN RENAL ARTERY STENOSIS**  
*H. Rousseau, M.D.*
- 13:28 - 13:35 **LECTURE**  
**SURGERY IN RENO-VASCULAR HYPERTENSION**  
*J. M. Capdevila, M.D.*

**FRIDAY, February 25, 1994**

- 13:35 - 13:42    **LECTURE**  
                  **MEDICAL TREATMENT IN RENO-VASCULAR HYPERTENSION**  
                  *J. C. Rodríguez, M.D.*
- 13:42 - 14:15    **PANEL DISCUSSION**  
                  **RENO-VASCULAR HYPERTENSION**  
                  *J. M. Callejas, M.D.*  
                  *J. M. Capdevila, M.D.*  
                  *T. O. McNamara, M.D.*  
                  *J. C. Palmaz, M.D.*  
                  *J. C. Rodríguez, M.D.*  
                  *H. Rousseau, M.D.*  
                  *D. E. Swarten, M.D.*  
                  *T. A. Sos, M.D.*  
                  *C. J. Tegtmeyer, M.D.*
- 14:15            **ADJOURN**  
                  *M. Maynar, M.D.*

**Care of Patients**  
**C. AÑEZ, M.D.**  
**A. HERNANDEZ, M.D.**  
**J. MAYNAR, M.D.**  
**A. RODRIGUEZ, M.D.**  
**J. RUBIO, M.D.**

## IV INTERNATIONAL COURSE ON VASCULAR AND INTERVENTIONAL RADIOLOGY AS A THERAPEUTIC ALTERNATIVE

### LECTURES

#### **SURGERY IN HEPATOMA**

- E. Moreno, M.D. (10')

#### **PERCUTANEOUS TREATMENT OF BUDD-CHIARI'S SYNDROME**

- M. Fava, M.D. (10')

#### **SCLEROTHERAPY OF ESOPHAGEAL VARICES**

- J. Bosch, M.D. (10')

#### **TECHNICAL EVOLUTION OF TIPS**

- J. Rosch, M.D. (10')

#### **DUPLEX US IN TIPS FOLLOW-UP**

- J.G. Letourneau, M.D. (10')

#### **TIPS RESULTS**

- P. Rossi, M.D. (10')

#### **RESULTS OF STRECKER STENT IN ESOPHAGEAL OBSTRUCTION**

- R. Reyes, M.D. (10')

#### **THE COVERED OESOPHAGEAL WALLSTENT**

- P. Winzler, M.D. (10')

#### **ERCP. BILIARY ENDOPROSTHESIS**

- J.M. Bordas, M.D. (10')

#### **LAPAROSCOPY IN ABDOMINAL PATHOLOGY**

- J. Cueto-Garcia, M.D. (10')

#### **PERCUTANEOUS PANCREATIC INTERVENTION**

- R.F. Dondelinger, M.D. (10')

#### **PERCUTANEOUS ABSCESS DRAINAGE**

- H.G. Coons, M.D. (10')

#### **DUPLEX US AND IV US IN THE DIAGNOSTIC EVALUATION OF PERIPHERAL VASCULAR DISEASE**

- J.G. Letourneau, M.D. (10')

#### **CT/MR ANGIOGRAPHY**

- W.P.Th.M. Mali, M.D. (15')

#### **CO<sub>2</sub> DIGITAL ANGIOGRAPHY**

- I.F. Hawkins, M.D. (10')

#### **ARTERIAL VOLUME FLOW BY A NEW ULTRASONIC TECHNIQUE**

- Mr. R. Ranalli (10')

#### **INTRAVASCULAR ENDOSCOPY**

- I.F. Hawkins, M.D. (10')

#### **VASCULAR PHYSICAL EXAMINATION**

- P. Lanzer, M.D. (10')

#### **PATHOPHYSIOLOGY OF ANGIOPLASTY**

- P. M. Consigny, Ph.D. (10')

#### **PTA. SURGEON'S POINT OF VIEW**

- J.M. Estevan, M.D. (10')

#### **LONG TERM RESULTS OF PTA IN PERIPHERAL VASCULAR DISEASE**

- C.J. Tegtmeier, M.D. (10')

#### **PERCUTANEOUS AORTIC GRAFTS**

- J.C. Parodi, M.D. (10')

#### **INTRAVASCULAR STENTS IN PERIPHERAL VASCULAR DISEASE**

- E.C. Martin, M.D. (10')

#### **ILIAC-FEMORAL SURGERY**

- V. Cabrera, M.D. (10')

#### **AORTIC ANEURYSM SURGERY**

- C. Cuesta, M.D. (10')

#### **TRANSLUMINAL ANGIOPLASTY AND EFFECTIVE THROMBOLYSIS WITH SODIUM PENTOSANE POLYSULPHATE GIVEN ORALLY**

- L. Horvath, M.D. (10')

#### **RESULTS OF PERCUTANEOUS AORTIC GRAFTING**

- Ph. Piquet, M.D. (10')

#### **RESULTS OF PERCUTANEOUS FEMOROPOPLITEAL GRAFTING**

- A.H. Cragg, M.D. (10')

#### **EMBOLIZATION UPDATE**

- M.A. Herrera, M.D. (10')

#### **TAKAYASU'S VASCULITIS: PTA RESULTS**

- L.K.A. Tan, M.D. (10')

#### **BRACHIO-CEPHALIC ANGIOPLASTY**

- J. Theron, M.D. (10')

#### **CAROTID ANGIOPLASTY RESULTS OF USA CLINICAL TRIAL**

- D.E. Schwarten, M.D. (10')

#### **SYMPOSIUM ON THROMBOLYSIS**

#### **LONG TERM RESULTS OF THROMBOLYTIC THERAPY**

- T.O. McNamara, M.D. (7')

#### **PULSE SPRAY LYSIS**

- K. Kandarpa, M.D. (7')

#### **LONG TERM RESULTS OF THROMBOLYTIC THERAPY IN CHRONIC OCCLUSIONS**

- J.M. Pulido-Duque, M.D. (7')

#### **RESULTS OF THROMBOLYTIC THERAPY IN THE MANAGEMENT OF RENAL ARTERY OCCLUSION**

- T.A. Sos, M.D. (7')

#### **MECHANICAL THROMBECTOMY**

- S.M. Tadavarthy, M.D. (7')

#### **THROMBOLYTIC THERAPY ON RENAL ARTERY OCCLUSION**

- M. Fava, M.D. (7')

**SYMPOSIUM ON STENTS**

**LONG TERM RESULTS OF STRECKER STENT IN ILIAC ARTERY**

- P. Strecker, M.D. (7')

**RESULTS OF FEMORAL ARTERY STENTING**

- D.D. Liermann, M.D. (7')

**LONG TERM RESULTS OF PALMAZ STENT**

- J.C. Palmaz, M.D. (7')

**LONG TERM RESULTS OF WALLSTENT**

- Ch.L. Zollikofer, M.D. (7')

**LONG TERM RESULTS OF GIANTURCO STENT IN VEINS**

- J. Rosch, M.D. (7')

**SYMPOSIUM ON RENO-VASCULAR HYPERTENSION**

**PTA IN RENO-VASCULAR HYPERTENSION**

- T.A. Sos, M.D. (7')

**ATHERECTOMY IN RENAL ARTERY STENOSIS**

- C.J. Tegtmeier, M.D. (7')

**PALMAZ STENTS IN RENAL ARTERY STENOSIS**

- J.C. Palmaz, M.D. (7')

**WALLSTENT IN RENAL ARTERY STENOSIS**

- H. Rousseau, M.D. (7')

**SURGERY IN RENO-VASCULAR HYPERTENSION**

- J.M. Capdevila (7')

**MEDICAL TREATMENT OF RENO-VASCULAR HYPERTENSION**

- J.C. Rodriguez, M.D. (7')

**IV INTERNATIONAL COURSE ON VASCULAR AND INTERVENTIONAL RADIOLOGY AS THERAPEUTIC ALTERNATIVE**

**GUEST FACULTY**

Mr. John E. ABELE	Co-Chairman and Director. Boston Scientific Corporation. Watertown, Massachusetts, U.S.A.	Jorge CUETO-GARCIA, M.D.	Department of Surgery. Hospital Ingles. Ciudad de Mexico, MEXICO.
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**IV INTERNATIONAL COURSE ON VASCULAR AND INTERVENTIONAL RADIOLOGY AS THERAPEUTIC ALTERNATIVE**

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<b>Jacques THERON, M.D.</b>	Professor and Head of the Department of Radiology. Faculté de Médecine. Centre Regional Hospitalier et Universitaire de Caen. Caen, FRANCE.	<b>Alberto CUBERO, M.D.</b>	Director of Neurology. Hospital Nuestra Senora del Pino. Las Palmas. Canary Islands, SPAIN.
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**IV INTERNATIONAL COURSE ON VASCULAR AND INTERVENTIONAL RADIOLOGY AS THERAPEUTIC ALTERNATIVE**

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Enrico SOTGIU, M.D.	Division of Vascular Surgery. Hospital Nuestra Senora del Pino. Las Palmas. Canary Islands, SPAIN.

The in-vivo demonstrations will be performed in the Interventional Vascular Radiological Unit at the Nuestra Senora del Pino Hospital and will be shown interactively on giant screens installed at the Course Congress Hall

**General Moderator**

**W.R. CASTANEDA-ZUNIGA, M.D.**

**Course Coordinators at Auditorium, M.D.**

**E. GORRIZ, M.D.**

**J.M. PULIDO-DUQUE, M.D.**

**Course Coordinators at Angiographics Suites**

**T. GARNICA, M.D.**

**M. HERRERA, M.D.**

**K. E. NAJARIAN, M.D.**

**R. REYES, M.D.**

**Course Coordinators at Video Center**

**M. de BLAS, M.D.**

**J.M. FELICES, M.D.**

**C. GERVAS, M.D.**

**Care of Patients**

**C. AÑEZ, M.D.**

**A. HERNANDEZ, M.D.**

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**APPRECIATION**

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**THE OFFICE OF CONTINUING MEDICAL EDUCATION,  
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**THE COMMITTEE OF CONTINUING MEDICAL EDUCATION (CME)  
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AWARDS THE EDUCATIONAL CONTENT OF THIS COURSE  
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FELLOWS IN CLINICAL RADIOLOGY OR CLINICAL ONCOLOGY  
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**THE UNIVERSITY OF LAS PALMAS DE GRAN CANARIA  
VALIDATES THIS 30 HOURS COURSE FOR  
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### **Unit of Vascular and Interventional Radiology**

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Flora Moreno  
Carmen Dolores Machín  
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Bienvenida - Welcome

## Bienvenida

**Fue en Octubre de 1988 cuando se organizó el Primer Curso. No han pasado aún seis años, y nueva tecnología y nuevas posibilidades han hecho su aparición en el esperanzador campo de la Terapéutica Percutánea, de la nueva Cirugía sin Bisturí.**

**La posibilidad de ofrecer un Shunt Porto Cava con anestesia local, el intento de cambiar los tradicionales by-pass por puentes metálicos introducidos de forma percutánea, la prometedora investigación de crear stents cubiertos para aneurismas aórticos, son algunas de las ideas y realidades que este campo de la medicina ofrece al mundo sanitario.**

**Pero pensemos que no sólo es esta parte de la medicina la que se encuentra en continua evolución; los métodos endoscópicos, la broncoscopia, la cirugía laparoscópica... intentan también ayudar por métodos menos cruentos a la evolución de las enfermedades, mientras por otra parte la cirugía tradicional se convierte en la mejor medicina: la eliminación y reposición del órgano dañado: el trasplante.**

**Es éste conjunto de acontecimientos los que nos tiene que obligar a organizar los nuevos conceptos médicos; es decir, tenemos que formar nuevas generaciones de profesionales en las nuevas técnicas. Para ello no existe más que un método: crear especialistas.**

**No es justo ampararse en conocimientos aprendidos en el pasado para aplicar nuevas técnicas que basadas en su escasa morbilidad parecen fáciles, convirtiéndose en poco útiles en manos sin entrenamiento. No es morbilidad directa lo que con esto se produce, es algo de significado muchas veces peor: la no atención correcta de un paciente simplemente por falta de experiencia.**

**Hoy día la medicina exige trabajar en equipo. Aprendamos cada uno una parte y juntemonos para aplicar de forma conjunta nuestra experiencia.**

**Este Cuarto Curso Internacional, donde diferentes especialistas nos hemos reunido con el mismo objetivo: ayudar al enfermo, tiene que servir para comenzar un camino que asegure que todo profesional que trate o diagnostique a un paciente con los nuevos métodos diagnóstico-quirúrgicos deba tener un entrenamiento oficialmente reconocido.**

**Sedís todos Bienvenidos a Las Palmas.**

**MANUEL MAYNAR, M.D.  
Jefe de la Unidad de Radiología  
Vascular e Intervencionista  
Hospital Ntra. Sra. del Pino  
Las Palmas de Gran Canaria  
España**



## Welcome

*It was October of 1988 when the First Course was organized. Six years have not yet passed and new technology and new possibilities have appeared in the encouraging field of Percutaneous Therapy, the new Surgery without Scalpel.*

*The possibility of offering a Porto-cava Shunt under local anesthesia, the attempt to change the traditional by-passes for metallic bridges introduced percutaneously, the promising research to create covered stents for aortic aneurisms are some of the ideas and realities that this medical field offers to the medical world.*

*But let us think that this is not the only part of the Medicine which finds itself in continuous evolution; endoscopic methods, bronchoscopy, laparoscopic surgery ... are also trying to help the evolution of illness through less invasive methods. While, on the other hand, the traditional surgery turns into the best medicine: the elimination and reposition of the diseased organ: the transplant.*

*It is this array of events that has to obligate us to organize the new medical concepts; that is, we have to form new generations of professionals in the new techniques. In order to do that there is only one way: create specialists.*

*It is not correct to shelter ourselves under the knowledge learned in the past to apply new techniques, which appear to be easy because of the low morbidity, but become useless in non-experienced hands. It is not direct morbidity what is produced with that, it is something which most of the times means something worse: the incorrect attention of a patient just because of the lack of experience.*

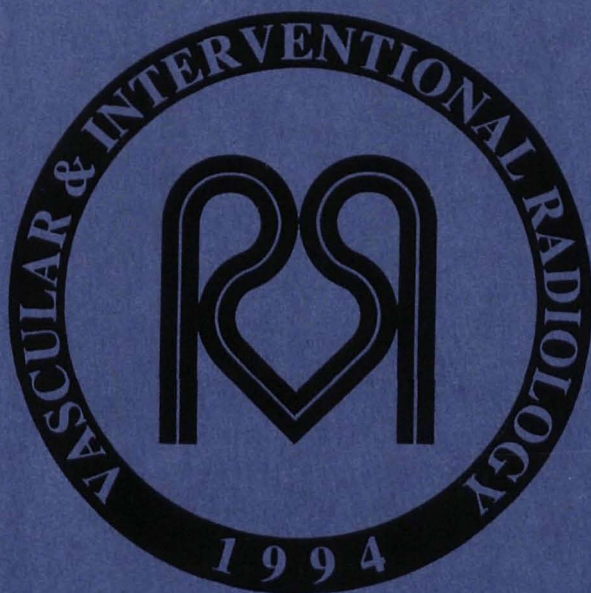
*Today, medicine demands teamwork. Let each one of us learn a part and later apply our experiences together.*

*This Fourth International Course, where different specialists have gathered with the same objective to help the ill has to be used to initiate a path that reassures that every professional that treats or diagnoses a patient with the new diagnostic-surgical methods ought to have an officially recognized training.*

*Welcome to Las Palmas.*

*MANUEL MAYNAR, M.D.  
Director of Vascular and  
Interventional Radiology  
Hospital Ntra. Sra. del Pino  
Las Palmas de Gran Canaria  
Spain*





Seguimiento de los casos de 1992  
Follow-up 1992 cases

## **Seguimiento de los Casos Curso 1992**

### **STENT ILIACO**

Realizado por el Dr. A.H. Cragg.

Varón de 56 años, diabético, fumador, con enfermedad vascular periférica, grado IIB.

La angiografía mostraba lesiones estenóticas severas en ambas arterias ilíacas, oclusión en la arteria femoral común izquierda y pobre salida distal.

En febrero de 1992, fueron insertados dos stents de Cragg en la arteria ilíaca derecha.

La angiografía demuestra permeabilidad del stent dos años más tarde.

El paciente presenta ahora claudicación grado IIA.

### **REVASCULARIZACION PERIFERICA COMPLEJA**

Realizado por los Dres. H. Coons y M. Maynar.

Varón de 42 años, obeso, fumador, hipertenso, con enfermedad vascular periférica, grado IIB.

La angiografía demostraba oclusión del tercio distal de la arteria femoral superior (AFS) con reconstitución de la arteria poplítea.

Debido a la obesidad del paciente, fue realizada recanalización y ATP del segmento ocluido mediante aproximación poplítea.

Dos años después, la angiografía demostró reoclusión de la AFS.

### **EMBOLIZACION HEPATICA**

Realizado por los Dres. H. Carrasco y H. Uchida.

Mujer de 45 años que presentaba cáncer de mama con metástasis única en el lóbulo hepático derecho.

La biopsia hepática fue positiva para adenocarcinoma.

## **Follow-up Cases 1992 Course**

### **ILIAC STENT**

Performed by A.H. Cragg, M.D.

56 year-old man diabetic and smoker with peripheral vascular disease stage IIB.

The angiography showed severe stenotic lesions in both iliac arteries, occlusion of the left SFA and poor run-off.

In February 1992 two Cragg stents were inserted in the right iliac artery.

Angiography showed patency of the stent two years later.

The patient now present claudication Stage IIA.

### **COMPLEX PERIPHERAL REVASCULARIZATION**

Performed by H. Coons, M.D. and M. Maynar, M.D.

42 year-old patient obese, smoker, hypertensive with peripheral vascular disease stage IIB.

Angiography showed occlusion of the distal third of the SFA with reconstitution in the popliteal artery.

Due to the obesity of the patient, recanalization and PTA of the occluded segment was performed through the popliteal approach.

Two years later the angiography showed reocclusion of the SFA.

### **HEPATIC EMBOLIZATION**

Performed by H. Carrasco, M.D. and H. Uchida, M.D.

45 year-old woman presenting breast cancer with a unique metastasis in the right hepatic lobe.

Hepatic biopsy was positive for adenocarcinoma.

La lesión hepática fue embolizada mediante Gelfoam y 4-epi-adriamicina.

Dos meses más tarde la TC demostraba metástasis hepática en ambos lóbulos y la paciente falleció un año después de la embolización.

The hepatic lesion was embolized with Gelfoam and 4-epi-adriamycine.

Two months later CT showed multiple hepatic metastasis in both lobes and the patient died one year after embolization.

### **STENT RENAL**

Realizado por los Dres. G.J. Becker y T.Garnica.

Varón de 64 años, diabético, fumador, con una historia clínica de hipertensión severa.

La angiografía demostraba estenosis severa de la arteria renal izquierda.

En febrero de 1992 fue insertado un stent de Palmaz en la arteria renal izquierda.

Su estado era normal hasta su fallecimiento, un año más tarde, debido a una hemoptisis severa.

### **RENAL ARTERY STENT**

Performed by G.J. Becker, M .D. and T. Garnica, M.D.

64 year-old male diabetic, smoker with a clinical history of severe hypertension.

The angiography showed severe stenosis of the left renal artery.

In February 1992 a Palmaz stent was inserted in the left renal artery.

He was doing well for a year then he died due to a severe hemoptysis.

### **SHUNT PORTO-CAVA PERCUTANEO**

Realizado por los Dres G. Richter, M. Robinson y T. Roehren.

Mujer de 60 años con historia clínica de hemorragia en varices esofágicas.

La paciente fue admitida en el hospital debido a un nuevo episodio hemorrágico.

Dos años más tarde el gradiente porto-cava era de 24 mm Hg, descendiendo a 13 mm Hg tras ATP.

La paciente permanece asintomática.

### **PERCUTANEOUS SHUNT PORTO-CAVA**

Performed by G. Richter, M.D.; M. Robinson, M.D. and T. Roehren, M.D.

60 year-old woman with clinical history of esophageal variceal bleeding.

The patient was admitted to hospital due to a new bleeding episode.

Two years later the portocaval gradient was 24 mm Hg. descending to 13 mm Hg. after PTA.

The patient remains asymptomatic.

### **STENT ESOFAGICO**

Realizado por los Dres. M.Maynar y R. Rostagno.

Varón de 25 años que presentaba disfagia debido a adenocarcinoma del tercio distal del esófago.

La endoscopia mostraba estenosis severa del esófago distal.

### **ESOPHAGEAL STENT**

Performed by M. Maynar, M.D. and R. Rostagno, M.D.

25 year-old patient presenting with dysphagia due to adenocarcinoma of the distal third of the esophagus.

#### IV CURSO INTERNACIONAL DE RADIOLOGIA VASCULAR INTERVENCIONISTA COMO ALTERNATIVA TERAPEUTICA

Se introdujo un stent esofágico de Nitinol de 15 cm. El paciente falleció dos meses más tarde debido a hemorragia gastrointestinal superior.

El stent permaneció permeable.

Endoscopy showed severe stenosis of the distal esophagus.

A 15 cm. Nitinol esophageal stent was inserted. The patient died two months later due to upper gastrointestinal hemorrhage.

The stent remained patent.

#### **IMPLANTACION DE CATETER DE HICKMAN**

Realizado por los Dres. J.M. Pulido y R. Reyes.

Varón de 29 años que presentaba leucemia mieloblástica aguda.

Fue implantado un catéter de infusión de quimioterapia en febrero de 1992.

Un años más tarde el paciente fallece debido a proceso subyacente. El catéter permaneció permeable.

#### **HICKMAN CATHETER IMPLANTATION**

Performed by J.M. Pulido, M.D. and R. Reyes, M.D.

Twenty nine year old male presenting acute myeloblastic leukemia.

A Hickman catheter for chemotherapy infusion was placed in February 1992.

One year later the patient died due to his underlying process. The catheter was still patent.

#### **DISCECTOMIA PERCUTANEA**

Realizado por los Dres. G. Onik y F. Robaina.

Mujer de 47 años que presentaba hernia discal derecha en L4-L5 con severo dolor de espalda.

En febrero de 1992 se realizó una discectomía percutánea con buenos resultados.

Dos años más tarde se encuentra asintomático

#### **PERCUTANEOUS NUCLEOTOMY**

Performed by G. Onik, M.D. and F. Robaina, M.D.

47 year old female presenting right L4-L5 discal hernia with severe back pain.

In February 1992 a percutaneous nucleotomy was performed with good results.

Two years after the procedure the patient is asymptomatic.

#### **STENT PROSTATICO**

Realizado por los Dres. S. Isorna y L.M. Pérez.

Varón de 73 años con historia clínica de prostatismo. Debido a sus pobres condiciones clínicas con un alto riesgo en cirugía abierta, se insertó un stent prostático.

Tras dos años sin problemas de micción, el paciente falleció de ataque cardíaco.

#### **PROSTATIC STENT**

Performed by S. Isorna, M.D. and L.M. Pérez, M.D.

73 year-old patient with clinical history of prostatism. Due to his poor clinical conditions with high risk for open surgery, a prostatic stent was inserted.

After two years without mictional problems, the patient died of a heart attack.



### FRAGMENTACION DE CALCULO BILIAR

Realizado por los Dres. F.J. Miller y R. Tobio.

Mujer de 60 años que presentaba cólico biliar.

Se realiza litotricia mecánica con buenos resultados.

Dos años más tarde la ultrasonografía abdominal demostró aparición de un nuevo cálculo y se realizó colecistectomía laparoscópica.

### LITOTRIZIA CON LASER

Realizado por los Dres. F. Castañeda y S. Isorna.

Mujer de 42 años con cólico renal debido a ureterolithiasis.

El cálculo permaneció tres meses tras la primera urografía. Se procedió mediante litotricia con láser en febrero de 1992.

Dos años más tarde, la paciente permanece asintomática y la urografía IV no muestra anomalía alguna.

### BILIARY CALCULUS FRAGMENTATION

Performed by F.J. Miller, M.D. and R. Tobio, M.D.

60 year-old female presenting biliary colic.

Mechanical lithotripsy was performed with good results.

Two years later abdominal ultrasonography showed new gall-bladder stones and laparoscopic cholecystectomy was performed.

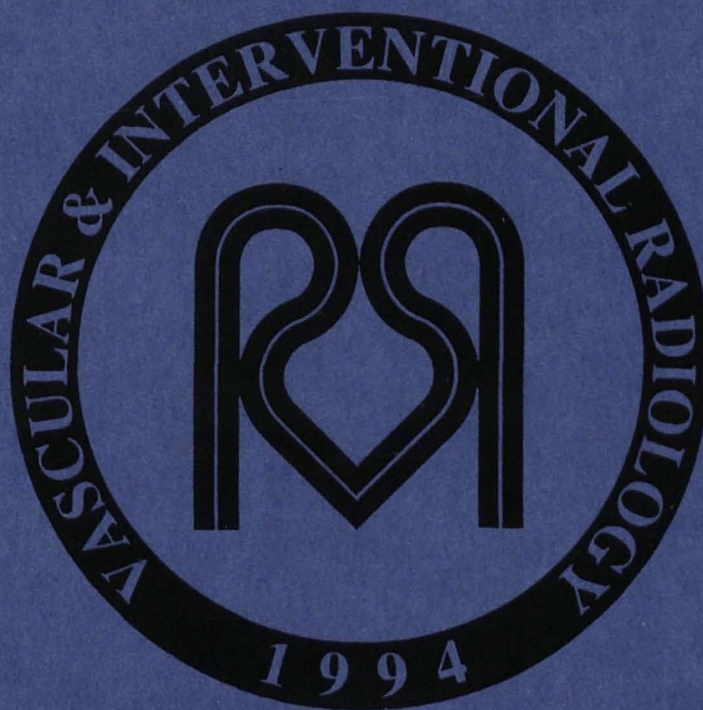
### LASER LITHOTRITY

Performed by P. Castañeda, M.D. and S. Isorna, M.D.

42 year-old patient with renal colic due to ureterolithiasis.

The calculus remained three months after the first urography and thus laser lithotripsy of the calculus was decided and it was performed in February 1992.

Two years later the patient remains asymptomatic and IV urography showed no abnormalities.



# SYLLABUS



## Reservorios Implantables para Acceso Vascular

DR. J. AGUIAR MORALES<sup>1</sup>, DR. U. BOHN SARMIENTO<sup>1</sup>,  
DR. J.M. PULIDO DUQUE<sup>2</sup>, DR. M. MAYNAR MOLINER<sup>2</sup>.

Los reservorios de implantación subcutánea, conectados al árbol vascular mediante un catéter, constituyen un sistema eficaz y seguro de acceso a la red arterial o venosa sin necesidad de utilizar de forma directa las vías periféricas, pudiéndose también acceder a cavidades naturales, como la peritoneal o el espacio subaracnoideo.

El diseño de estos sistemas, cada vez más perfeccionados, permiten su implantación subcutánea en la región pectoral, antebraquial o abdominal. Consta de una parte llamada reservorio fabricada con acero, titanio o polisulfona, el cual se aloja en una cavidad subcutánea realizada "exprofeso", al cual se conecta un catéter de silicona, que se introduce tras tunelización en la vía vascular. El reservorio, dispone de un septum o membrana de silicona autosellable, que permite su punción directa desde la piel, resistiendo hasta 2000 punciones sin roturas o fisuras que provoquen fugas del material administrado, a condición de que se empleen agujas de diseño y bisel adecuadas.

Los diferentes modelos de reservorios implantables que existen en el mercado, difieren fundamentalmente por su peso, tamaño, material empleado en su fabricación y sistemas de conexión catéter-reservorio, oscilando su peso entre 25 gr. para los de acero inoxidable; 16 gr para los de titanio y 3.3 gr los de polisulfonas.

Estos sistemas de reservorios, están dirigidos fundamentalmente a la administración de drogas citotóxicas en los pacientes oncológicos, evitándoles de esta forma el continuo traumatismo de las vías periférica, así como las flebitis química y riesgos de extravasación de drogas, capaces de provocar severos fenómenos de necrosis tisular y dermatitis vesicante.

Además, la búsqueda y punción de las vías

periféricas, resulta con frecuencia difícil y dolorosa para los pacientes, provocando en ellos disconfort y frustración con pérdida de tiempo en el personal de enfermería que les asiste. Los sistemas de referencia, vienen a resolver todas estas dificultades, constituyendo un método de trabajo eficaz, de gran comodidad para los pacientes y con escasa morbilidad, aunque tienen ciertas limitaciones de uso.

La colocación de un sistema portal venoso en la región pectoral, precisa de dos fases. En la primera, se fabrica una bolsa subcutánea para alojar el reservorio, el cual se fija a la fascia pectoral mediante puntos de anclaje. Posteriormente, se introduce el catéter por vía percutánea en el subclavia, llevándolo hasta la aurícula derecha y tunelizando el extremo proximal hasta su conexión con el reservorio. Asegurada la conexión, se comprueba la estanqueidad del sistema, la correcta ubicación distal del catéter en el 3º-4º espacio intercostal por visión radiológica y angiográfica, así como la ausencia de neumotórax, procediéndose a la sutura de las incisiones quirúrgicas.

El sistema queda listo para su utilización al 4º día, precisando hasta ese momento de lavado y sellado diario con 5 ml. de suero fisiológico heparinizado (100 u/ml).

La utilización en la práctica de los reservorios, requiere de una asepsia rigurosa, así como el uso de agujas especiales con un bisel adecuado, para evitar fisuras en el septum. Para administrar drogas, o extraer muestras de sangre de destinadas a controles analíticos, se localiza el portal mediante palpación, insertándose la aguja a través de la piel hasta el fondo del reservorio. Se procede al lavado previo y posterior administración de la medicación indicada en bolus, perfusión o bomba de infusión automática, lavando por último el sistema y

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sellándolo con suero heparinizado. En todo momento, se evitará dejar la vía abierta o extraer con presión negativa para evitar el reflujo.

Finalizado el programa de quimioterapia, se procede a retirar el reservorio. Si por alguna razón se decide conservarlo por algún tiempo, es necesario su lavado y sellado periódico (cada 20 días en nuestra experiencia), para evitar la trombosis del catéter.

Manteniendo de forma rigurosa las condiciones de uso, el número de complicaciones es pequeño y su duración prolongada. Un paciente de nuestra serie, mantuvo su reservorio durante casi un año después de terminar su tratamiento, sin que durante ese tiempo, por fallo administrativo, se realizasen los oportunos cuidados de lavado y sellado. Sorpresivamente, al ir a retirarlo, se comprobó el perfecto funcionamiento del mismo, lo que da una idea de la durabilidad de estos sistemas.

La duración prolongada de los reservorios, poca morbilidad, excelentes condiciones de comodidad y estética para el paciente, así como seguridad y ahorro de tiempo para enfermería, cualifican al procedimiento de forma plena para su utilización clínica. Por otro lado, al ser un sistema cerrado al exterior, presenta menos posibilidades de infección respecto a otros sistemas abiertos tipo Hickman, en los que se precisa una mayor cobertura de enfermería, con ciertas limitaciones para la actividad física del paciente.

Sin embargo, también tienen algunas limitaciones, sobre todo referidas al volumen o viscosidad de las soluciones a perfundir, relacionadas con el calibre del catéter, siendo más adecuados para perfusiones cortas o mediante bombas de perfusión automáticas con cantidades limitadas de diluyente. La situación hematológica de determinados enfermos, con serios problemas de coagulación, impiden la utilización de sistemas portales por el riesgo de sangrado local con la punción y la presión dentro de la bolsa subcutánea, siendo en ellos preferibles los sistemas exteriores.

Durante los años 1992-3, hemos colocado a nuestros pacientes oncológicos 132 sistemas de reservorios subcutáneos, de los cuales 43 han sido antebraquiales y 89 pectorales.

La selección de pacientes para colocación de reservorios, ha estado en función del mal estado de las vías periféricas o por tratarse de pacientes incluidos en programas de quimioterapia

prolongados, con los consiguientes riesgos de extravasación o tromboflebitis química.

Hemos tenido 16 complicaciones, lo que supone un 12.12% del total de la serie. En la comunicación previa, habíamos colocado 40 reservorios, en los que tuvimos un 22.5% de complicaciones (9 casos). En los siguientes 92 implantes, el porcentaje ha sido de un 8.68% (7 casos), lo cual ilustra sobre la importancia de la experiencia en la minimización de la morbilidad.

Las complicaciones globales incluyen:

	nº de casos
1. Trombosis del catéter	5
2. Fiebre relacionada con el sistema	5
3. Precipitación química	2
4. Neumotórax	2
5. Flebitis	1
6. Fallo de anclaje	1
total.....	16 (12.12%)

1. Documentadas angiográficamente y posteriormente por examen del catéter después de su retirada.
2. En todos los casos se procedió a retirada y recolocación de implante. En dos de ellos se documentó el germen responsable (e. dorado y p. aureuginosa). La punta o extremo del catéter, se envían sistemáticamente a estudios microbiológicos.
3. En un mismo paciente y por dos ocasiones consecutivas, se produjo la precipitación química de las drogas administradas, con obstrucción de toda la luz del catéter por un material de aspecto pseudotrombótico.
4. Se produjeron dos neumotórax: uno tardío a las 48 horas, en una paciente con abordaje por subclavia izquierda y otro inmediato rápidamente detectado y de fácil resolución con medidas conservadoras, mientras que el anterior precisó de drenaje-aspiración pleural.

En ningún momento se produjeron situaciones críticas a pesar de tratarse de pacientes inmunocomprometidos de alto riesgo.

5.6. El episodio de flebitis se manejó de forma sintomática y el fallo de anclaje, con movilidad anormal del reservorio, necesitó de reimplante, por el dolor y las dificultades que generaba a la hora de administrar el tratamiento al no poderse delimitar

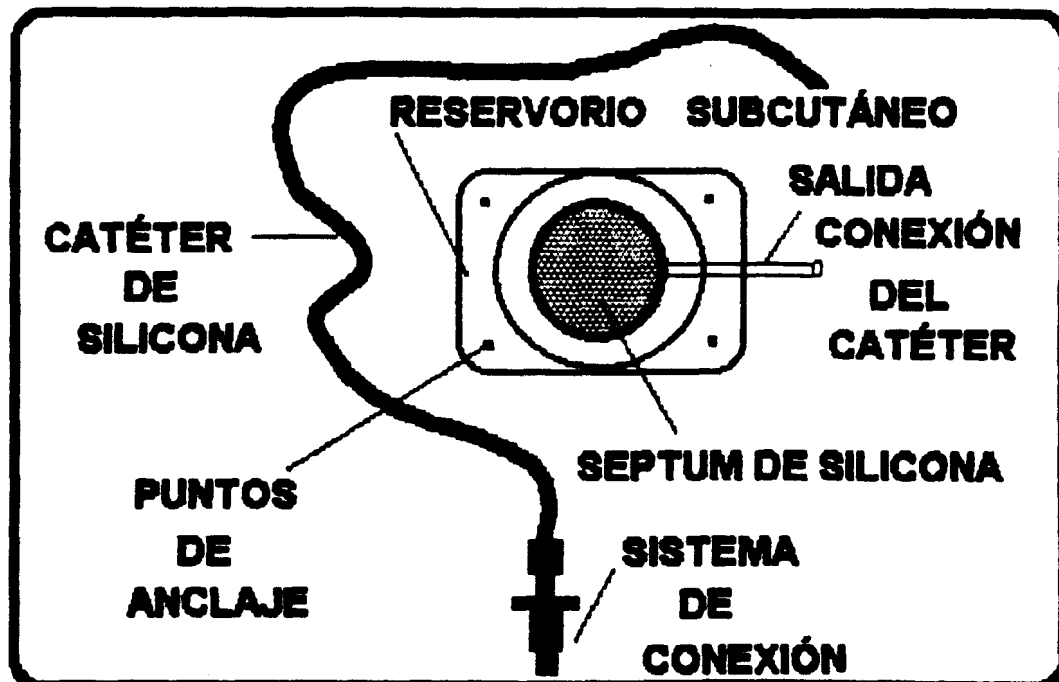


perfectamente el lugar de punción.

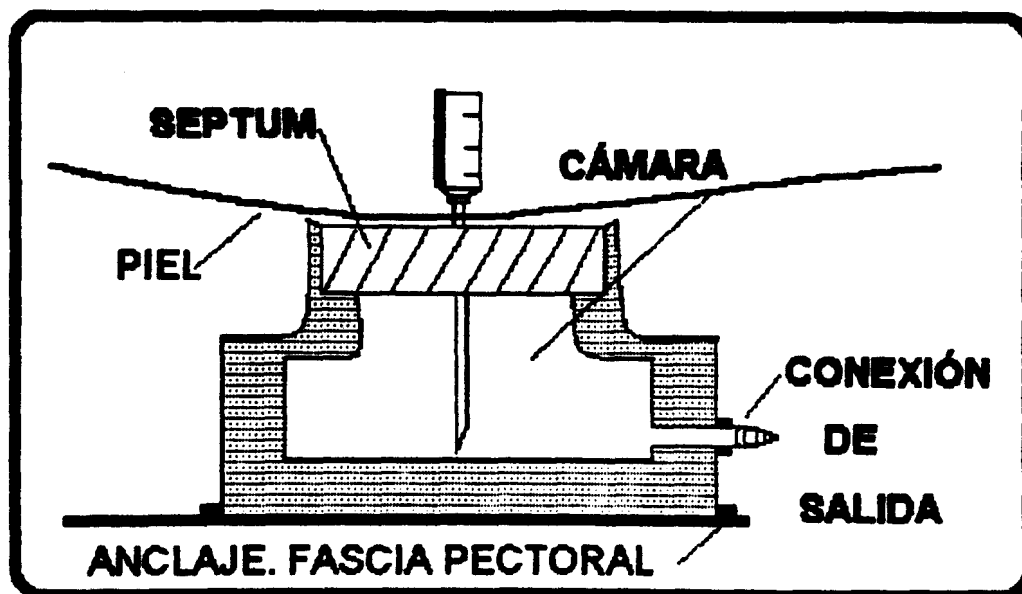
Vamos a considerar aparte un tema, que no incluimos en la categoría de complicaciones. Se trata de episodios aislados de falta de flujo en el sistema, resueltos con medidas simples sin necesidad de retirarlo, tales como acodamientos; fenómenos de vacío, al quedar el extremo distal del catéter adherido a la pared vascular o episodios sin causa aparente que ceden tras forzar la limpieza del sistema con control angiográfico.

Podemos concluir, en resumen que los sistemas de reservorios subcutáneos utilizados para administración de quimioterapia citotóxica, constituyen un método con baja tasa de morbilidad, alto grado de comodidad para el paciente y mínimas a necesidades de mantenimiento.

Como inconvenientes, señalar su desventaja respecto al Hickman, para la administración de grandes cantidades de líquidos o soluciones viscosas; su dificultad para colocarlo en pacientes con problemas de coagulación y la interferencia que producen en técnicas de imagen como la TAC o RNM. Hoy día, se han introducido nuevos materiales que obvian este tipo de interferencias, así como problemas relacionados con la radioterapia de áreas en las que esté incluido el reservorio.



ESQUEMA DE UN SISTEMA IMPLANTABLE SUBCUTÁNEO



VISIÓN LATERAL DEL RESERVORIO SUBCUTÁNEO.

## Complicaciones en Radiología Vascular Intervencionista que Amenazan la Vida del Paciente

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La realización de procedimientos diagnósticos y terapéuticos en Radiología constituye la principal demanda para la práctica de técnicas anestésicas fuera del quirófano. El papel de anestesiólogo en la sala de RxVi es garantizar la seguridad y el confort del paciente y facilitar el procedimiento en aquellos casos en los que no colabora.

La prevención y detección de complicaciones así como la rapidez en el tratamiento son fundamentales, sobretodo en aquellas que amenazan potencialmente la vida del paciente. Dentro de las complicaciones podemos distinguir las secundarias a la patología del paciente, a la técnica en sí y a la utilización de medios de contraste (MC).

Este trabajo tiene como objetivo revisar aspectos de interés clínico de las reacciones adversas a los MC. Las complicaciones derivadas de la situación clínica del paciente son tan amplias que escapan del objetivo de esta revisión y las relacionadas con las técnicas de RxVi son brevemente resumidas en la TABLA I.

### REACCIONES ADVERSAS A LOS MEDIOS DE CONTRASTE

La incidencia de complicaciones por el uso de MC iodados varía entre un 2 y un 10%. La mayor parte de ellas son leves y no requieren tratamiento. Contribuyen a la aparición de reacciones adversas al MC el método de inyección (inyección lenta o en bolo), la técnica o localización anatómica a estudiar (son más frecuentes en arteriografías coronarias y del SNC y colangiografías) e historia previa de atopía o alergia.

Reacciones tales como edema facial o laríngeo, arritmias cardíacas, broncospasmo severo o colapso circulatorio se producen 1/3000 ó 1/4000

procedimientos en los que se utilizan MC. De éstos, el 96% sufren reacciones reversibles con un tratamiento rápido y agresivo. La mortalidad debida al uso de MC es de 1/14.000 a 1/117.000.

Las reacciones a los MC pueden ser idiosincráticas y no idiosincráticas.

#### *Reacciones no idiosincráticas a los medios de contraste*

Son debidas a la toxicidad o a las características físico-químicas del MC (hiperosmolaridad). Incluyen náuseas, vómitos, arritmias, edema pulmonar, colapso circulatorio, nefropatía y agravación de patologías preexistentes.

Los aditivos de los MC son causantes de trastornos hemodinámicos y electrolíticos (por ejemplo el citrato sódico o el edetato sódico actúan como quelantes del calcio por lo que tienen efecto inotrópico negativo).

El fallo renal agudo por MC es la tercera causa de insuficiencia renal intrahospitalaria, precedida por los estados de bajo gasto y los procedimientos quirúrgicos. La prevalencia de nefropatía por MC varía entre 0 a 31%. Son factores de riesgo la existencia de nefropatía previa, la diabetes mellitus, el mieloma múltiple, la insuficiencia cardíaca severa (clase IV) y la hiperuricemia. Otros factores de riesgo en el caso de existencia de nefropatía previa son: deshidratación, HTA, enfermedad vascular periférica, proteinuria, edad mayor de 65 años y la inyección de MC proximal a los vasos renales.

De forma profiláctica debe restringirse la cantidad de MC, hidratación previa al estudio y uso de diuréticos como manitol o furosemida. Los contrastes de baja osmolaridad siendo menos hipertónicos son, en principio, menos citotóxicos pero esto no está demostrado para la nefropatía.

### *Reacciones idiosincráticas a los medios de contraste*

Tienen un mecanismo de producción alérgico o "alérgico-like". Existen cuadros que simulan una reacción anafilactoide, como la estimulación vagal, que cursan con hipotensión y bradicardia, y responden a la administración de atropina.

Los antecedentes de alergia duplican la frecuencia de reacciones al MC, y cuatuplican la aparición de reacciones severas. En 15-60% de los pacientes con antecedentes de reacción al MC se reproducen en un segundo contacto, y en el 20% de ellos cursa con clínica similar.

Otros factores que se asocian con una mayor frecuencia son: la inyección iv, una dosis de yodo superior a 20g, la edad, la presencia de cardiopatía y trastornos electrolíticos.

Las reacciones adversas a los MC pueden clasificarse en: leves (urticaria, broncospasmo, hipotensión arterial y convulsiones) y severas (hipotensión arterial, cianosis, anoxia, edema de pulmón, angor y arritmias).

### **TRATAMIENTO DE LAS REACCIONES ANAFILACTICAS GRAVES**

**L**as primeras medidas son: parar la administración del alérgeno, mantener la permeabilidad de la vía aérea, administrar O<sub>2</sub> y soporte circulatorio.

El angioedema y el laringospasmo se tratan con adrenalina en aerosol (3 inhalaciones de 0,16-0,20 mg), en nebulización (8-15 gotas de adrenalina 0,25% en 2 ml de SF), sc ó iv a dosis de 0,3-0,5 ml en adultos. Si no responde al tratamiento se realiza cricotiroidotomía.

En caso de hipotensión grave o shock, se administran 25-50 ml/kg de cristaloides o coloides. Puede ser necesario el uso de adrenalina 3-5 µg/kg iv. La persistencia de hipotensión requerirá la infusión continúa de adrenalina 1-4 µg/kg/min.

Si el paciente presenta broncospasmo persistente, hipertensión pulmonar o fallo ventricular derecho es útil el isoproterenol a dosis de 0,01-0,02 µg/kg/min. Si el broncospasmo persiste a pesar de los β<sub>2</sub>-adrenérgicos, se le administra aminofilina 5-6 mg/kg en 20 min. En caso de mejoría, antes de extubar al paciente, debe valorarse la persistencia de angioedema que nos impida la reintubación si

procede.

Los glucocorticoides son útiles para prevenir las reacciones tardías, Metilprednisolona 1 mg/kg/6h durante las primeras 24 horas o dexametasona 4-20 mg iv. También son útiles los antihistamínicos H<sub>1</sub> como la difenhidramina a 1 mg/kg.

En caso de parada cardiorespiratoria se iniciarán prontamente las maniobras de reanimación cardiopulmonar y cerebral. Las arritmias cardíacas se tratarán con antiarrítmicos, cardioversión o ambos.

### **PROFILAXIS**

*En pacientes sin historia previa de reacciones a medios de contraste.*

Cualquier paciente puede tener una reacción adversa al MC durante su administración por primera vez. Las dosis de prueba se han abandonado debido a la aparición de reacciones severas. La premedicación con antihistamínicos y corticoides reduce la aparición de reacciones adversas. Una historia previa de alergia (fiebre del heno, asma, etc) pueden acompañarse de un riesgo elevado de reacciones, pero el riesgo de reacciones grave no está aumentado.

*Pacientes con historia previa de reacciones a los medios de contraste.*

Estos pacientes no siempre vuelven a sufrir una reacción adversa. La incidencia de nueva reacción varía entre 17-60%. Dada la imposibilidad de predecir en que pacientes se producirá de nuevo una reacción al MC, se han usado distintas pautas de premedicación. La administración de prednisona, difenhidramina y efedrina reduce la incidencia de reacciones adversas a 4,1% en pacientes de alto riesgo. La administración de antihistamínicos H<sub>2</sub> no reducen el riesgo.

La premedicación junto con un MC de baja osmolaridad atenúa el riesgo de reacción por debajo del 1%, frente al 9% cuando se premedica y se utilizan MC convencionales.

*Manejo del paciente con historia previa de reacciones a medios de contraste en procedimientos electivos.*

1. Valorar el tipo de reacción previa y documentar la necesidad de realización de la técnica en cuestión.

**TABLA I. COMPLICACIONES EN RADIOLOGIA VASCULAR INTERVENCIONISTA.**

<p><b>COMPLICACIONES PROPIAS DE LA TECNICA:</b></p> <p>A) Lesión vascular arterial y/o venosa:</p> <p>    a) - Hematoma.</p> <p>    b) - Disección.</p> <p>    c) - Pseudoaneurisma.</p> <p>    d) - Fístula arterio-venosa.</p> <p>B) Neumotórax.</p> <p>C) Hidrotórax.</p> <p>D) Quilotórax.</p> <p>E) Derrame y taponamiento pericárdico.</p> <p>F) Embolismo aéreo.</p> <p>G) Embolia de partículas sólidas.</p> <p>H) Arritmias cardíacas.</p> <p>I) Lesión nerviosa.</p>
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**TABLA II. PREMEDICACION EN PACIENTES CON HISTORIA DE REACCIONES A LOS MEDIOS DE CONTRASTE EN PROCEDIMIENTOS ELECTIVOS.**

FARMACO	DOSIS	VIA	MOMENTO ADMINISTRACION
Prednisona	50 mg.	v.o.	13,7 y 1 h. antes del proc.
Difenhidramina	50 mg.	v.o./i.m.	1 h. antes del proc.
Efedrina *	25 mg.	v.o.	1 h. antes del proc.

\* Contraindicada en pacientes con cardiopatía isquémica y arritmias.

**TABLA III. PREMEDICACION EN PACIENTES CON HISTORIA DE REACCIONES A LOS MEDIOS DE CONTRASTE EN PROCEDIMIENTOS DE URGENCIA.**

FARMACO	DOSIS	VIA	MOMENTO DE ADMINISTRACION
Hidrocortisona	200 mg.	i.v.	*
Difenhidramina	50 mg.	i.m.	**

\* Inmediatamente antes del procedimientos, repitiendo cada 4 horas durante la realización del mismo.

\*\* Inmediatamente antes del procedimiento.

#### IV CURSO INTERNACIONAL DE RADIOLOGIA VASCULAR INTERVENCIONISTA COMO ALTERNATIVA TERAPEUTICA

2. Informar al paciente de la necesidad del procedimiento y del riesgo que supone.
3. Explicar el objetivo de la premedicación y la disminución del riesgo que supone su administración.
4. Premedicación (TABLA II).
5. Uso de medios de contraste de baja osmolaridad.
6. Disponer de un equipo de reanimación y contar con la colaboración de un anestesiólogo.
9. Dswarte R.D. Special considerations of allergic drug reactions. En Patterson R., Grammer L.C., Greenberger P.A., Zeiss C.R. eds: Allergic diseases. Diagnostic and management. Filadelfia, J.B. Lippincot Co., 1993; 395-552.

Antes de realizar el procedimiento es obligatorio una valoración por un alergólogo.

*Manejo del paciente con historia previa de reacciones a medios de contraste en procedimientos de urgencia.*

1. Premedicación (TABLA III).
2. Utilizar medios de contraste de baja osmolaridad.

"Estas pautas deben realizarse incluso cuando la administración de contraste no sea intravascular".

#### Referencias.

1. Gillies B.S., Anesthesia outside the operating room. En Barash P.G., Cullen B.F., Stoelting R.D., eds. Clinical Anesthesia. Filadelfia. J.B. Lippincot Co, 1992; 1465-1477.
2. A.J.R. 1987; 149: 665-670
3. Mayo Clin Proc. 1989; 64: 976-985.
4. Wolfson B., Hetrick W.D., Dastur K.J., Neuroradiologic procedures. En: Newfield P., Cottrell J. eds. Neuroanesthesia: handbook of clinical and physiologic essentials. Little, Brown and Co, 1991; 416-429.
5. Weiss M.E., Levy J.H. Immunologic complications. En: Benumof J.L., Saidman L.J., eds. Anesthesia and perioperative complications. St. Louis, Mosby Year Book, 1992; 378-395.
6. Reich D.L., Kaplan J.A. Complications of cardiovascular access: En: Benumof J.L., Saidman L.J., eds. Anesthesia and perioperative complications. St. Louis, Mosby Year Book, 1992; 378-395.
7. JAMA 1992; 25: 2855.
8. Sullivan T.J. Drug allergy. En Middleton E, Reede Ch E, Ellis E.F., Adkinson N.F., Yunginger J.W., Busse W.W. eds: Allergy. Principles and practice. Chicago, Mosby, 1993; 1.726-1746.

## Ecografía y Tips

J.I. BILBAO Y J.M. LONGO<sup>1</sup>

La derivación portosistémica percutánea intrahepática (DPPI-TIPS) es una técnica eficaz, con indicaciones ya establecidas en el tratamiento de las complicaciones derivadas de la hipertensión portal en pacientes cirróticos. Actualmente, es una alternativa terapéutica a la cirugía y la escleroterapia en el tratamiento de las hemorragias digestivas por varices gastro-esofágicas.

La técnica inicial, presentada por Richter, incluía la punción portal transhepática con el fin de identificarla para poder realizar su punción transyugular y facilitar la ulteriores manipulaciones de catéteres, guías, etc... Sin embargo, esta punción transhepática de la porta elevaba considerablemente la morbilidad derivada del procedimiento. Por ello, diferentes técnicas menos cruentas de identificación portal han sido desarrolladas y publicadas. Entre ellas, la portografía indirecta, la venografía enclavada, la cateterización de vena umbilical o técnicas quirúrgicas de cateterizaciones de vena mesentérica y ulterior opacificación portal.

Otra posibilidad es la identificación y localización ecográfica (US) de la vena porta. El uso de esta técnica permite, a su vez, monitorizar el procedimiento guiando las diferentes maniobras y pasos del TIPS (selección de vena hepática, punción portal, dilatación del tracto y liberación de la prótesis).

La distribución vascular hepática está sujeta a múltiples variaciones anatómicas que, en ocasiones, dificultan considerablemente la realización del TIPS cuando éste se practica sólo con control fluoroscópico. Del mismo modo, los hígados cirróticos son habitualmente pequeños y con una morfología distorsionada por la fibrosis.

En determinados casos, los recorridos vasculares intraparenquimatosos se encuentran totalmente alterados por la propia enfermedad hepática (p. ej.: Síndrome de Budd-Chiari), siendo necesario un

control más directo del avance transhepático de las agujas que el obtenido "sólo" con fluoroscopia.

El hígado cirrótico es, como se ha dicho, habitualmente pequeño, siendo la "posibilidad de maniobra" de las agujas intrahepáticas también pequeña. Es por ello que, en ocasiones, ocurre la perforación de la cápsula hepática y la punción de la vesícula biliar como grave complicación del procedimiento. Estas eventualidades son mucho menos frecuentes, por no decir nulas, cuando se emplea monitorización ecográfica.

Por último, los US intraprocedimiento posibilitan la menor utilización de contraste yodado (en estos pacientes con función renal habitualmente deteriorada) siendo el tiempo medio del procedimiento comparativamente menor, como ha sido recientemente presentado.

El empleo añadido de US-Doppler-color permite conocer los cambios que sucesivamente se van produciendo en la velocidad y dirección del flujo portal durante el procedimiento. La información con ella obtenida, junto con los datos hemodinámicos y la angiografía, será la base para los sucesivos controles que se realizarán en estos pacientes.

Diferentes estudios publicados han coincidido en señalar al US-Doppler-color como un excelente método, incruento y barato, para el control evolutivo de los TIPSS. Los cambios de velocidad y dirección del flujo portal, la presencia de irregularidades en los contornos del shunt y la vena hepática, así como los cambios en la morfología y dirección de flujo de los vasos colaterales son signos especialmente útiles para detectar estenosis y/o obstrucciones del TIPS. La sensibilidad y especificidad de la técnica puede alcanzar el 90 %. Por ello, la detección incruenta de alteraciones de flujo, etc... indicará la necesidad de realizar técnicas más agresivas para tratar estas complicaciones estenóticas, desgraciadamente frecuentes, en la evolución de los TIPSS.

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## Transjugular Intrahepatic Portocaval Shunt in Partially Thrombosed Portal Veins

J.I. Bilbao et al: Tips in Partially Thrombosed Portal Veins

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### ABSTRACT

During transjugular intrahepatic portocaval shunt (TIPS), needle insertion under ultrasound control facilitates portal puncture minimizing the need of blood aspiration to confirm entry. This fact is important in cases where TIPS is indicated and an intraportal thrombus is present, because needle entry within the vessel is accurately controlled. Once in the portal vein, thrombi can be fragmented by balloon angioplasty dilatation without the need of fibrinolytic therapy. Finally, the rise in portal blood flow velocity probably contributes to the progressive disappearance of the remnant portal thrombi. This is a report of 3 cases of partial porta vein thrombosis in which TIPS were successfully carried out with recovery of portal vein patency and overall clinical improvement.

**KEY WORDS:** Portal vein, US - Portal vein, thrombosis - Hypertension, portal - Shunt, portocaval - Liver, interventional procedures.

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Transjugular intrahepatic portocaval shunt (TIPS) is an affective palliative procedure for variceal bleeding in cirrhotic patients with portal hypertension. Since the initial description in humans by Richter et al (1), different technical advances have been reported providing a clear reduction in the duration of the procedure as well as in the rate of complications. One of these technical modifications is ultrasound control of the needle during portal vein puncture (2). In our

experience, this technique has also proven to be especially useful in shunting patients with partially thrombosed portal veins in which portal catheterization can be difficult.

In this report, we present 3 patients with liver cirrhosis and intraportal thrombi in whom a percutaneous portocaval shunt was safely performed by using ultrasonographic control as an adjunct to fluoroscopy during the procedure. These patients represent 5% of our total number of TIPS and were the only cases presenting with portal thrombosis. In all 3 cases, intraportal thrombi disappeared during follow-up without the need for any additional thrombolytic or anticoagulant therapy.

### CASE REPORTS

#### Case 1.

A 54 year-old male with a 15 year-old history of hepatitis was admitted to our hospital for hematemesis and melena. Emergency endoscopy revealed active bleeding from huge gastroesophageal varices which was successfully controlled by sclerotherapy. The diagnosis of liver cirrhosis with severe portal hypertension and positive serology for hepatitis virus C was established and the patient was started on beta-blockers and diuretics. An abdominal ultrasound study revealed a patent portal vein. He was discharged after 20 days with improved clinical status.

Ten months later he was hospitalized due to severe

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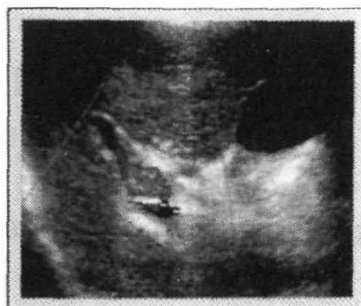
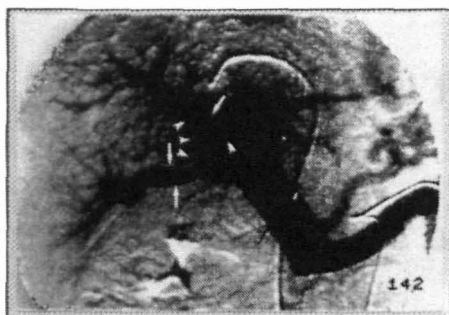
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**Fig. 1.A)** Indirect portography from a splenic angiography. A filling defect is observed within the right portal branches extending into the main portal vein. **1.B)** Color-Doppler ultrasound during transjugular portal puncture. The tip of the needle (arrow) is clearly seen across the clot reaching the nonthrombosed part of the portal lumen. **1.C)** Direct trans-shunt portography just after TIPS placement. Multiple filling defects are clearly observed in both right and left portal branches. Small thrombi are seen within the main portal trunk (arrows) **1.D)** Direct portography (one month after TIPS) through the intrahepatic shunt with the catheter tip placed in the splenic vein. No thrombi are observed neither in the portal vein nor in the intrahepatic branches.

deterioration of liver function and an increase in ascites which was refractory to medical treatment. The patient was included in the liver transplant program. Endoscopy revealed marked increase in gastroesophageal varices. A color-Doppler ultrasound revealed partial occlusion of the portal vein due to the presence of a thrombus extending into both right and left portal branches. Portal blood flow velocity was 5 cm/sec. The presence of partial portal thrombosis, a past history of variceal bleeding, and severe ascites refractory to medical treatment justified TIPS as a temporizing measure while awaiting liver transplantation.

An indirect portography performed just before the TIPS procedure revealed the presence of a thrombus that almost entirely occluded the right portal branches extending into the main portal vein (Fig. 1A). A 10F sheath (William Cook, Europe, Bjaeverskov, Denmark) was inserted into the right internal jugular vein and, after the selection of the most adequate path between the hepatic vein and the intrahepatic portal branches, a 16G transjugular biopsy needle (William Cook, Europe) was placed in the right hepatic vein. Needle passage through the liver parenchyma was controlled with both ultrasound (Acuson 128 x P/10, Acuson, Mountain View, CA, USA) and fluoroscopic guidance. The tip of the needle was advanced across the portal thrombus, only a few drops of blood could be aspirated (Fig. 1B). Contrast was then injected and the intravascular positioning of the needle was confirmed. A hydrophilic guidewire (Terumo Corporation, Tokyo, Japan) was carefully advanced

through the thrombus as far as the nonthrombosed portion of the portal vein. The needle was retrieved and 5F multiperforated straight catheter (Cordis, Europe, Oosteend, The Netherlands) was then advanced down to the mesenteric vein. Central venous and portal pressures were measured and portosystemic gradient was then calculated .

An Amplatz super-stiff guidewire (Medi-Tech/Boston Scientific, Waterlown, MA, USA) was then advanced and a 5F, 8 mm-diameter balloon angioplasty catheter (Schneider Europe AG, Zurich, Switzerland) was utilized for the dilatation of the intrahepatic tract and for the disruption of the portal thrombus. Finally, a Wallstent (Medinvent SA, Lausanne, Switzerland) vascular prosthesis 77 mm long and 9 mm wide was placed within the shunted area with its distal 2 cm within the portal vein lumen (Fig. 1C). The portosystemic gradient was again measured .

Follow-up portography, pressure measurements and ultrasonography performed 1 month later revealed complete shunt and portal vein patency (Fig. 1D) with good hemodynamic function, and portal blood flow velocity of 28 cm/sec. The patient has maintained excellent clinical status since then with no further episodes of ascites or variceal hemorrhage, and has been withdrawn from the liver transplant waiting list.

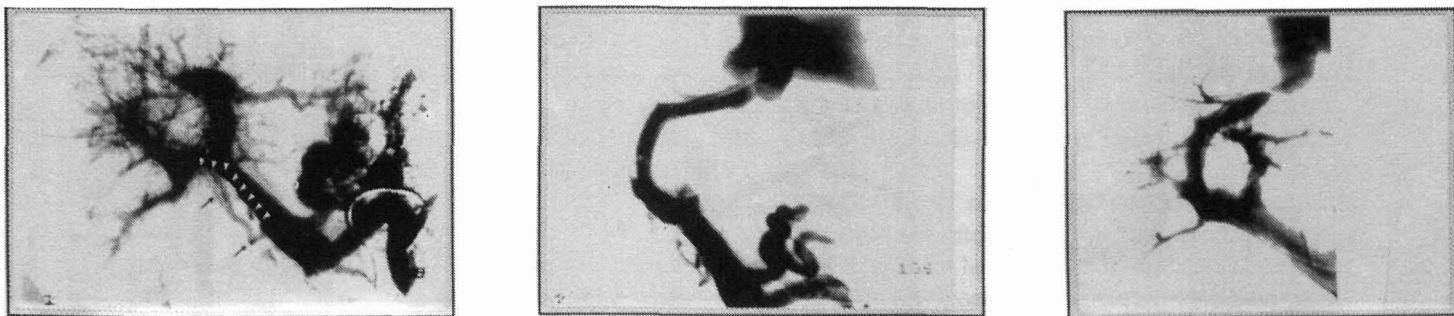


Fig. 2.A) Indirect portography from a splenic angiography. A thrombus is observed in the portal trunk extending into the right portal vein. Collateral periportal vessels (arrows) and gastric varices (arrowheads) are also observed. 2.B) Direct portography 3 days after TIPS shows adequate shunt patency and nonopacification of the intrahepatic portal branches. 2.C) Selective portogram, performed just after figure 2B, with the tip of the catheter in the bifurcation of the portal vein. Several thrombi are observed obstructing the left portal vein.

### Case 2

A 56 year-old female with chronic active hepatitis, diagnosed 6 years previously, presented with massive bleeding secondary to grade IV esophageal varices which was controlled with endoscopic sclerotherapy. Liver cirrhosis with positive serology for virus C was detected. Repeat endoscopy 2 months later showed huge gastric varices in addition to the esophageal ones.

The patient was referred to our institution for inclusion in the liver transplant program. Ultrasound work-up showed a thrombus partially occluding the main portal vein extending into the right branch with a flow velocity of 10 cm/sec. Angiography confirmed the diagnosis and revealed the presence of a massive gastric variceal network as well as collateral recirculation of periportal branches (Fig. 2A).

Because of the difficulty in endoscopic sclerosis of gastric varices in the face of possible recurrent bleeding and the presence of a portal thrombus that could render a liver transplant difficult, TIPS was indicated.

Using the same technique as in the previous case, the right portal vein was reached by US-guided transjugular puncture and, after advancing a 5F straight catheter, venous pressures and portosystemic gradient were measured. Portal wall dilatation with the angioplasty catheter was especially difficult, with the balloon waist lasting for 12 min. A Wallstent vascular prosthesis (62 mm long and 9 mm wide) was deployed in the dilated liver tract and right portal vein down to the main trunk, displacing the previously disrupted

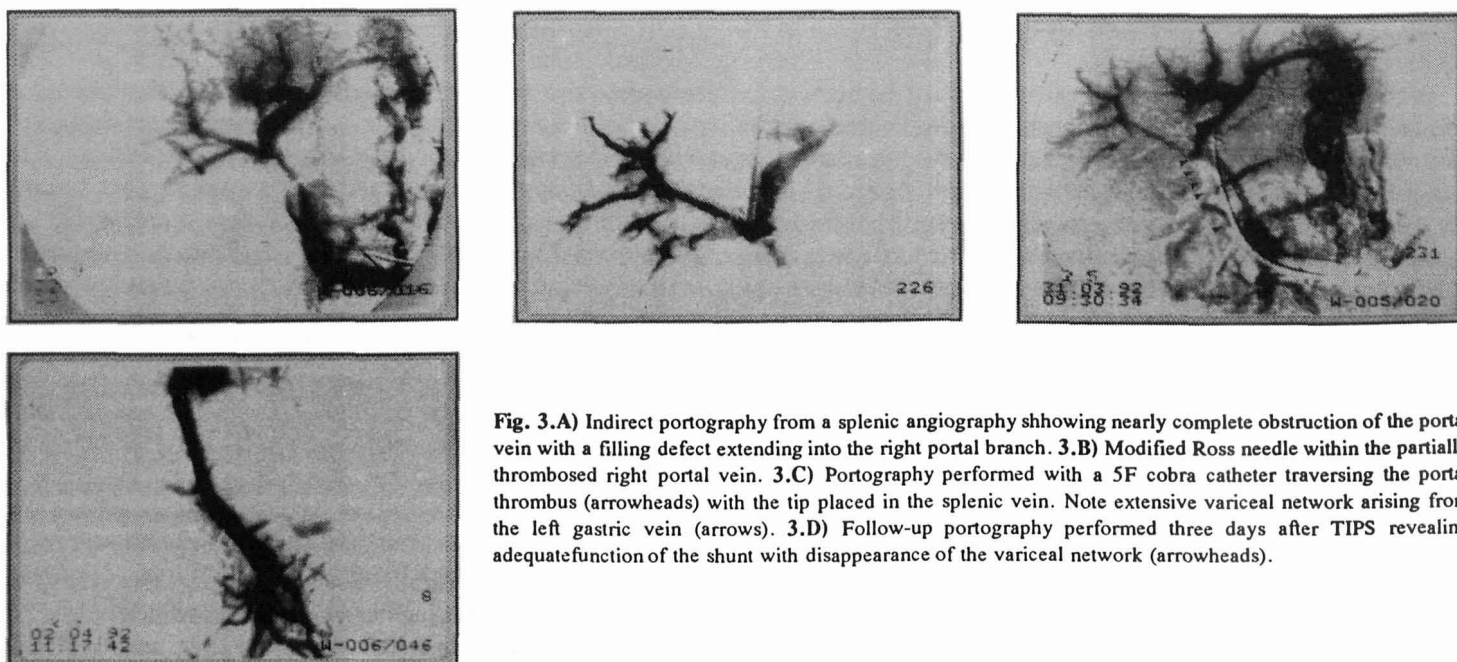
thrombus. A 9F sheath (USCI, Billerica, MA, USA) was left in the jugular vein through which the prosthesis was recatheterized 3 days later, and a repeat portography revealed good contrast passage through the stent and, unexpectedly, complete thrombosis of the left portal vein (Fig. 2B-2C).

At this point, the portosystemic gradient was 12 mm Hg. An expectant attitude was taken and neither fibrinolytics nor anticoagulants were given. Follow-up ultrasound studies revealed progressive disappearance of the thrombus, with a portal blood flow velocity of 37 cm/sec.

Direct portography performed 1 month later revealed complete shunt and portal vein patency without any evidence of thrombi and with clear diminution in gastric varices. Portosystemic gradient was 8 mm Hg at this time. The patient had no further variceal bleeding and is currently awaiting liver replacement.

### Case 3

A 56-year-old male with a diagnosis of cirrhosis since 1988 presented with a 2-month history of a progressive deterioration in its clinical status with lethargy, anorexia, and abdominal distension due to ascites. The patient was admitted to our hospital and at gastroscopy, huge gastroesophageal varices were observed. Color-Doppler ultrasound revealed a patent portal vein with hepatopetal flow at a velocity of 10 cm/sec. The right portal vein was not clearly visualized but the presence of a thrombus could not be ruled out.



**Fig. 3.** A) Indirect portography from a splenic angiography showing nearly complete obstruction of the porta vein with a filling defect extending into the right portal branch. B) Modified Ross needle within the partially thrombosed right portal vein. C) Portography performed with a 5F cobra catheter traversing the porta thrombus (arrowheads) with the tip placed in the splenic vein. Note extensive variceal network arising from the left gastric vein (arrows). D) Follow-up portography performed three days after TIPS revealing adequate function of the shunt with disappearance of the variceal network (arrowheads).

On the third hospital day he presented with massive bleeding from the varices, which was temporarily controlled with an intravenous infusion of Somatostatin. Six hours later, the decision to perform a percutaneous portocaval shunt was made.

Emergency ultrasound immediately prior to the procedure revealed a thrombus in the main portal vein extending into its right branch. This finding was confirmed by indirect portography from both splenic and superior mesenteric angiography (Fig. 3A).

Percutaneous portocaval shunt was performed immediately using the same technique described in the previous cases. The right portal vein was punctured under ultrasound control, but neither hydrophilic nor steerable guidewires could pass through the well-organized thrombus in the right portal vein (Fig. 3B). Different branches of this right portal vein were punctured but a guidewire could not be safely placed in the main portal vein. The left portal vein was then catheterized and a hydrophilic guidewire was inserted through the portal thrombus as far as the splenic vein (Fig. 3C). Portosystemic gradient was recorded. After tract dilatation and thrombus disruption with the balloon, two Wallstents vascular prostheses (9 mm wide and 62 and 77 mm long) were deployed, the first displacing the portal thrombus and the second within the liver tract. Both portography and pressure measurements revealed good shunt function.

Ultrasound and portographic follow-up studies performed 3 days later revealed patency of both the portal vein (flow velocity of 19 cm/sec) and the intrahepatic shunt (Fig. 3D). One month after the procedure, the patient had remained stable with reduced ascites and no recurrence of variceal bleeding. He refused repeat portography, and only ultrasound studies could be performed in order to confirm shunt patency. Unfortunately, the patient died 3 months later due to severe deterioration of liver function.

## DISCUSSION

Until recently, the presence of portal thrombosis was a relative contraindication to liver transplantation. The development of new surgical techniques now allows successful liver replacement in such cases (3). Nevertheless, portal thrombosis increases the risk of variceal bleeding in cirrhotic patients with portal hypertension who are awaiting liver transplantation.

Percutaneous transhepatic access for balloon dilatation of portal stenoses (4,5) or for mechanical or pharmacological disruption of portal thrombi has been previously reported (6-9); it obtains a clear decrease in portal pressure and hence, lowers the risk of variceal bleeding. Recently, Harville et al (10) reported a case of a noncirrhotic patient with pre-hepatic portal hypertension secondary to portal thrombosis. In this patient, portal patency was regained through balloon angioplasty followed by the deployment of two metallic stents within the

portal vein.

In liver transplant candidates with repeated episodes of variceal hemorrhage, TIPS is a useful technique as a temporizing measure to control portal hypertension and to stabilize clinical status before liver replacement (11).

Due to the risk of hemorrhage, transhepatic placement of catheters in the portal vein when performing TIPS has been avoided (12,13). Thus, other methods, such as ultrasound, have been resorted to for needle guidance during the procedure (2). The continuous control of the needle tip during portal puncture provides accuracy and minimizes the need of blood aspiration to confirm entry. Only in our third patient was this step difficult and, in spite of the intraluminal placement of the needle tip, the guidewire could not be advanced, probably because of the presence of a well-organized old thrombus. For this reason, left portal puncture through the same hepatic vein was necessary for the catheterization of the thrombosed portal vein.

When the tip of needle is placed within the portal lumen, hydrophilic guidewires are especially useful because they can be safely advanced through the thrombosed vessel until the splenic or the superior mesenteric vein. A possible complication of this part of the procedure is stripping of the guidewire during its advancement through the beveled needle, thus, special care should be taken and excessive manipulation avoided. However, neither in the presented cases nor in the rest of our series, in which this combination (hydrophilic guidewire and beveled needle) has been utilized (55 patients), has such a complication occurred.

Portal thrombi can be mechanically fragmented, dislodged, and pushed into collaterals (4). Most of the disrupted thrombi flow through the shunt to the general circulation and, as observed by Ring (14), are surprisingly well tolerated, without any pulmonary sequela. Residual clots are safely compressed against the portal wall with the metallic prostheses.

In all patients, a marked decrease in portosystemic gradients was demonstrated. Both direct portography and color-Doppler ultrasound follow-up studies revealed rapid blood flow across a completely patent shunt. Three days after the procedure, our second patient unexpectedly presented complete thrombosis of the left portal vein. Due to the high risk of hemorrhage, an

expectant attitude was taken and no further manipulations were made nor medical treatment given. Careful follow-up studies showed the progressive disappearance of the thrombus probably due to the rise in portal blood flow velocity.

All 3 cases showed marked initial clinical improvement after TIPS, though our third patient died 3 months later. The other 2 patients remain stable with good shunt flow, without portal thrombosis and no ascites nor recurrent variceal bleeding episodes.

This brief report demonstrates the usefulness of ultrasonographic guidance in performing TIPS and the value of this procedure in regaining patency of partially thrombosed portal veins.

## References

1. Richter G.M., Noe G., Palmaz J.C., Roessel M., Slegertetter V., Franke M., Gerok W., Wenz W. (1990) Transjugular intrahepatic portocava stent shunt: Preliminary clinical results. *Radiology*; 174: 1027-1030.
2. Longo J.M., Bilbao J.I., Rousseau H.P., Joffre F.G., García-Villarreal I., Vinel J.P., Sangro B. (1992) Color-Doppler US guidance in transjugular placement of intrahepatic portosystemic shunts. *Radiology*; 184: 281-284.
3. Stieber A.C., Zetti G., Todo S., Tzakis A.G., Fung J.J., Marino I., Casavilla A., Selby R.R., Starzl T.E. (1991) The spectrum of portal thrombosis in liver transplantation. *Ann Surg.*; 213: 199-206.
4. Olcott E.W., Ring E.J., Roberts J.P., Ascher N.L., Lake J.R., Gordon R.L. (1990) Percutaneous transhepatic portal vein angioplasty and stent placement after liver transplantation: Early experience *JVIR*; 1: 17-22.
5. Scantlebury V.P., Zajko A.B., Esquivel C.O., Marimo I.R., Starzl T.E. (1989) Successful reconstruction of late portal vein stenosis after hepatic transplantation. *Arch Surg*; 124: 503-505.
6. Helling T.S. (1985) Thrombosis and recanalization of the portal vein in liver transplantation: A case report. *Transplantation*; 40: 336-448.
7. Uflacker R., Lima S. (1980) Percutaneous transhepatic portography for obliteration of gastroesophageal varices in partial and total portal vein occlusion. *Radiology*; 137: 325-330.
8. Bilbao J.I., Rodríguez-Cabello J., Longo J., Zornoza G., Páramo J., Lecumberri F.J. (1989) Portal thrombosis: percutaneous transhepatic treatment with Urokinase. *Case Report. Gastrointest Radiol.*, 14: 326-328.
9. Uflacker R., Alves M.A., Cantisani G.G., Souza H.P., Wagner J., Moraes L.F. (1985) Treatment of portal vein obstruction by percutaneous transhepatic angioplasty. *Gastroenterology*; 88: 176-180.

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10. Harville L.E., Rivera F.J., Palmaz J.C., Levine B.A. (1992) Variceal hemorrhage associated with portal vein thrombosis: Treatment with a unique portal venous stent. *Surgery*; 111: 585-590.
11. Roberts J.P., Ring E.J., Cake J.R., Sterneck M., Ascher N.L. (1991) Intrahepatic portocaval shunt for variceal bleeding hemorrhage prior to liver transplant. *Transplantation*; 52: 160-162.
12. Richter G.M., Noeldge G., Palmaz J.C., Roessle M. (1990) The transjugular intrahepatic portosystemic stent shunt (TIPSS): Results of a pilot study. *Cardiovasc Intervent Radiol*; 13: 200-207.
13. Richter G.M., Noeldge G., Roessle M., Palmaz J.C. (1991) Evolution and clinical introduction of TIPSS, the transjugular intrahepatic portosystemic stent-shunt. *Semin Intervent Radiol*; 8: 331-340.
14. Ring E.J. (1991) Interventions in portal hypertension. In: Mueller P.R., van Sonnenberg E., Becker G.J. (eds): *Syllabus: a diagnostic categorical course in interventional radiology*. Radiological Society of North America, Oak Brook, Ill pp 219-227.



## Endoprótesis en las Ictericias Obstructivas Extrahepáticas de Etiología Maligna

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Los tumores que obstruyen las vías biliares suelen tener mal pronóstico a pesar de que las técnicas de imagen permiten establecer el diagnóstico de forma muy precoz a partir de la primera manifestación clínica, generalmente dolor abdominal, ictericia, detección de una masa en una exploración ecográfica del hígado y con menor frecuencia después de detectar colestasis biológica. El pronóstico y las posibilidades de tratamiento son peores si el tumor asienta a nivel de hepático común o en la zona de la placa hiliar. En estos casos las posibilidades terapéuticas se limitan a la colocación de drenajes biliares internos mediante cirugía, endoscopia digestiva o punción percutánea.

La ultrasonografía (US) es uno de los métodos más eficaces en la localización de lesiones tumorales que afectan a las vías biliares, puesto que permite evidenciar la dilatación de la vía biliar intrahepática hasta la zona donde se produce la obstrucción. En algunos casos permite apreciar la masa tumoral en la zona próxima al hilio, en otros únicamente detecta un colédoco distal de tamaño normal, y en la mayoría de ocasiones no visualiza el conducto biliar extrahepático. Cuando esto sucede, la ausencia de imágenes de cálculos en la vesícula, junto a la dilatación de la vía biliar intrahepática y a la falta de visualización del colédoco, sugiere la existencia de una masa tumoral a nivel de la placa hiliar o del hepático común, como causa de la ictericia. La coexistencia de litiasis vesicular y tumores de la región hiliar, en ausencia de metástasis hepáticas, puede ser motivo de errores en la interpretación de los datos de la ultrasonografía. En estos casos la dilatación de la vía biliar hasta determinado nivel sin observar litiasis coledocal, asociada a la presencia de cálculos en la vesícula biliar, justifica que se indique la aplicación de otros métodos diagnósticos, y lo mismo sucede cuando no hay un completo acuerdo entre los datos clínico-biológicos y los ofrecidos por la ultrasonografía (US). Es también

imprescindible asegurar un diagnóstico completo (lugar y naturaleza de la obstrucción), mediante la utilización de varias técnicas diagnósticas en los pacientes con elevado riesgo quirúrgico.

Las técnicas alternativas a la US para el estudio de la colestasis, son aquellas que permiten la obtención de un "mapa" completo de la vía biliar mediante la inyección directa de contraste radiológico en los conductos biliares. La colangiografía percutánea con aguja fina o colangiografía transparietohepática (CTPH), según la U. de Chiba (K. Okuda)<sup>(1)</sup> es una de las más eficaces, cuando hay una marcada dilatación de las vías biliares intrahepáticas, puesto que en estas circunstancias, la posibilidad de conseguir la colangiografía es muy elevada (entre 90 y 100 % de los casos). Esta alta rentabilidad diagnóstica de la CTPH se ve gravada por una relativamente elevada incidencia de complicaciones. En un estudio prospectivo efectuado por nosotros, la frecuencia con que la CTPH generó complicaciones fue del 6,5 %<sup>(2)</sup>, de las que un porcentaje elevado necesitaron cirugía urgente. Se cita también un porcentaje de alrededor del 5 % de infecciones biliares secundarias a la punción percutánea<sup>(3)</sup>. Otra desventaja atribuible a este método, es la dificultad de obtener buenas imágenes radiológicas en los casos de obstrucción completa en los que la bilis y la visión global del árbol biliar exige tiempo o maniobras complejas. Una de ellas consiste en el vaciado de la bilis y posterior relleno con contraste. Para ello es preciso sustituir la aguja fina por una cánula de material plástico y de mayor diámetro, lo que aumenta el riesgo de complicaciones. Otra opción es esperar algunas horas y efectuar un nuevo estudio radiológico. Por otra parte, cuando la obstrucción biliar se produce a nivel del hilio hepático, la punción percutánea puede tener dificultades para delimitar las ramas del lóbulo izquierdo<sup>(4)</sup>. La técnica de la colangiografía percutánea permite el drenaje de la vía biliar tanto al exterior como al duodeno. El drenaje externo se consigue al sustituir la aguja fina

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utilizada para el diagnóstico por un catéter de mayor diámetro que facilita el drenaje desde los colectores biliares próximos al hilio hepático al exterior. Esta técnica puede utilizarse como un método temporal de reducir la colestasis entre el diagnóstico y la cirugía. El drenaje interno percutáneo se consigue al introducir a través de la aguja de colangiografía percutánea un fiador que debe superar la obstrucción de la vía biliar y llegar al duodeno. Este fiador sirve de guía para la colocación de una cánula cuyo extremo distal se sitúa en la zona de la obstrucción y permite el paso de la bilis desde los conductos intrahepáticos al duodeno. Estas "prótesis" biliares colocadas por vía percutánea presentan una relativamente alta tasa de complicaciones entre las que se deber citar el cole y hemoperitoneo, las infecciones abdominales y biliares y la dificultad en obtener un drenaje eficaz de ambos lóbulos en casos de que la obstrucción afecte al confluente<sup>(4)</sup>.

La Colangiopancreatografía Retrógrada Endoscópica (CPRE) es otra técnica diagnóstica que puede poner de manifiesto la morfología de la vía biliar al rellenarla con contraste radiológico, en este caso inyectado por vía retrógrada a través de la papila de Vater. Esta exploración no es cruenta y sus complicaciones, generalmente pancreatitis, suelen ser leves y controlarse mediante tratamiento médico. La frecuencia con que la CPRE origina pancreatitis, que en las series retrospectivas se consideraba relativamente baja, parece más alta cuando se han efectuado estudios prospectivos y llega hasta cifras de 16 % en un estudio en que incluye la exploración del páncreas y la zona peripancreática mediante tomografía computarizada<sup>(5)</sup>. Otra complicación, menos banal y que se produce con cierta frecuencia en los pacientes con obstrucción de la vía biliar es la colangitis que suele precisar el drenaje urgente de la vía biliar<sup>(6, 7)</sup>. El mayor inconveniente de la CPRE reside en que el porcentaje de canulación con que se consigue observar la vía biliar viene limitado por el porcentaje de canulación de la papila de Vater y en el caso de las obstrucciones por tumores, por la posibilidad de que el contraste supere la estenosis. Se considera que el éxito de la canulación electiva y profunda del colédoco oscila entre 80 y 95 % de casos<sup>(8, 9)</sup> mientras que no está establecida la frecuencia con que el contraste consigue superar la estenosis y por lo tanto, delimitar la lesión obstructiva en su porción proximal. Por otra parte, cuando se consigue hacer llegar el contraste a la vía biliar supraestenótica, si no se puede drenar seguidamente la vía biliar por métodos endoscópicos, es necesario indicar el

drenaje precoz (dentro de las 48 horas siguientes) por otros métodos, puesto que el riesgo de colangitis es elevado, a pesar de que administren antibióticos de amplio espectro a dosis adecuadas<sup>(10)</sup>.

Cada vez con mayor frecuencia se diagnostican masas tumorales que afectan a la vía biliar produciendo ictericia y malabsorción, en pacientes con estado general conservado y una buena calidad de vida. Hasta el advenimiento de la radiología y de la endoscopia terapéuticas, la cirugía era el único procedimiento viable y se indicaba para obtener un drenaje paliativo de la bilis. El procedimiento quirúrgico suele ser temporalmente eficaz cuando la masa tumoral afecta al colédoco distal y deja libre una buena porción de la vía biliar principal extrahepática. Esto permite, que en los casos más favorables pueda realizarse una coledoco-duodenostomía o una coledocogastrostomía. En otras situaciones en las que la anastomosis de la vía biliar debe realizarse con asas desfuncionalizadas del delgado, los resultados también son aceptables aunque la complejidad de la cirugía y su morbi-mortalidad aumenta. En un tercer grupo de pacientes el drenaje únicamente puede conseguirse mediante calibración y colocación de drenajes en la porción estenosada de la vía biliar. Los casos más difíciles son los que cursan con invasión tumoral de la placa hiliar y de la zona del confluente. En ellos no es infrecuente que por cirugía no se pueda acceder a la vía biliar intrahepática (Estenosis tipo II y III de la clasificación elaborada por Cremer)<sup>(11)</sup>. En esta situación la calibración seguida de la colocación de conductos de drenaje suele ser muy difícil y a veces imposible, por lo que la laparotomía puede saldarse sin maniobras terapéuticas.

La cirugía en los pacientes con ictericia obstructiva por tumores malignos del páncreas tiene una elevada morbi-mortalidad operatoria (30 días) que hace pocos años se situaba en cifras del orden del 20 %<sup>(12)</sup> independientemente del tipo de cirugía realizada. Actualmente parece que estos porcentajes se han reducido, pero la tasa de complicaciones severas después de pancreático-duodenectomía se eleva al 18 % en una serie reciente de 96 pacientes sometidos a esta intervención quirúrgica, cuando se consideró la posibilidad de realizarla como técnica curativa (con extirpación completa de la masa tumoral en la cabeza del páncreas)<sup>(13)</sup>. Sin embargo, la extirpación radical de los tumores de la cabeza del páncreas sólo se considera posible, según algunos estudios prospectivos, en un porcentaje muy escaso de pacientes (2 %)<sup>(14)</sup>. El drenaje biliar temporal antes de la cirugía parece que sólo está

justificado en las obstrucciones distales<sup>(15)</sup>. En los tumores más altos, situados al nivel de la placa hiliar y hepático común la mortalidad es más elevada y en muchos casos se ve agravada por la imposibilidad de realizar un drenaje de ambos lóbulos y no es infrecuente que sea imposible calibrar y colocar un drenaje. En estos casos si se considera indicada la cirugía no debe utilizarse ninguna técnica derivativa temporal<sup>(16)</sup>.

La endoscopia terapéutica, en el caso de la colocación de drenajes biliares internos, presenta similares problemas que la cirugía. La máxima dificultad en la colocación de drenajes se produce cuando los tumores afectan al confluente, en la estenosis de curso tortuoso y cuando existe invasión de las paredes de los conductos biliares. Esta última circunstancia dificulta la progresión de las guías que posteriormente deben permitir el avance del drenaje biliar elegido. Como ventaja de los drenajes introducidos por vía retrógrada mediante CPRE, debe citarse el hecho de que puede ser utilizada como una técnica intermedia para mejorar el estado general y los resultados de la cirugía, en pacientes que posteriormente serán intervenidos quirúrgicamente con pretensión curativa, aunque también pueden utilizarse como terapéutica definitiva. En cualquier caso, si la técnica endoscópica fracasa (cateterización de la papila o calibración de la estenosis tumoral) los intentos endoscópicos no suelen impedir ni dificultar la técnica quirúrgica. En este aspecto los drenajes colocados por vía endoscópica aventajan a los que se introducen por vía percutánea, que con frecuencia lesionan la vía biliar a nivel del cabo proximal y dificultan su utilización para anastomosis quirúrgicas<sup>(16)</sup>.

La colocación de drenajes biliares internos por vía endoscópica es relativamente sencillo<sup>(17)</sup> y en la actualidad se pueden colocar tubos de drenaje de material plástico, también denominadas "prótesis biliares" de 12 F<sup>(18, 19, 20)</sup> o incluso de 14 F, utilizando para las primeras endoscopios con canal operativo de 3,6 mm (OLYMPUS TJF) y para las segundas un canal de 5 mm (OLYMPUS TJF Type M20). En condiciones experimentales, el débito de cualquiera de estos tubos de drenaje supera ampliamente la producción de bilis por parte del hígado<sup>(21)</sup>. También hay estudios clínicos retrospectivos que ponen de manifiesto que los tubos en forma de cola de cerdo o con múltiples orificios laterales condicionan una mayor precocidad en la obstrucción de estas "prótesis biliares"<sup>(22)</sup> y se ha demostrado en clínica que los drenajes rectos y con el menor número de orificios

laterales, ofrecen menor resistencia al paso de la bilis y por lo tanto tienen menor tendencia a la obstrucción<sup>(23)</sup>. La necesidad de prevenir la obstrucción de los drenajes biliares internos (DBI) (colonización bacteriana, formación de una película biológica y posterior precipitación de los solutos de la bilis), ha motivado investigaciones sobre el tipo de material con que están contruidos estos drenajes de plástico. Así Leung publica en *Endoscopy* en 1992 investigaciones in vitro sobre la menor adherencia bacteriana en tubos de drenaje recubiertos por una lámina de plata, sin que haya evidencias de que estos resultados tengan traducción clínica<sup>(24)</sup>.

La mayor parte de las primeras publicaciones sobre "prótesis biliares" colocadas a través de endoscopia, incidieron en el número de drenajes colocados con éxito, así como en las complicaciones<sup>(25, 26, 27, 28)</sup>, aunque sólo algunas permitían establecer comparaciones con técnicas alternativas<sup>(29)</sup>. Por otra parte, en las series más numerosas se mezclaban pacientes muy distintos y no se atendía a la situación de las estenosis tumorales. Así Walta<sup>(30)</sup> en una serie de 84 pacientes en los que se colocaron 135 drenajes biliares incluía 4 lesiones quirúrgicas, 3 pacientes con estenosis inflamatorias y 77 obstrucciones malignas. De ellas, sólo siete correspondían a estenosis situadas en hepático común o en hilio hepático. En estos pacientes con estenosis proximales, la supervivencia fue mucho menor que en los pacientes con estenosis distales, de forma que las que afectaban al hilio sólo tuvieron una supervivencia media de 34 días, los que afectaban al hepático común sobrevivieron una media de 96 días, mientras que los tumores de la zona de la ampolla de Vater y a tumores pancreáticos tuvieron una supervivencia media de más de 200 días.

Hace unos años se dispone de drenajes de malla metálica expandible que consiguen un a mayor dilatación de la vía biliar y por lo tanto un drenaje eficaz<sup>(31)</sup>. El amplio diámetro obtenido que llega a ser de 8 a 10 mm facilita la rápida remisión de los síntomas<sup>(32)</sup>. Un amplio estudio multicéntrico que incluye 103 pacientes en el que se utilizaron prótesis metálicas autoexpandibles tipo Wallstent consiguieron una eficacia en la colocación del 97 %, aunque sólo incluyeron a aquellos pacientes en quienes la guía para la introducción de la prótesis conseguía superar la zona del obstáculo tumoral. El seguimiento de estos pacientes fue de 145 días, durante este período la tasa de colangitis fue de 18%, la supervivencia media de 125 días y la de obstrucción por barro biliar de 5 %. Aunque se



trata de un estudio no controlado, esta tasa de obstrucción por barro biliar es muy inferior al 21 % aceptado en otras series de la literatura en que se habían utilizado drenajes de plástico. Sin embargo a este 5 % hay que añadir un 7 % de obstrucción por crecimiento intraluminal del tumor<sup>(33)</sup>.

Los drenajes expansibles, que se pueden colocar tanto por vía retrógrada a través de duodenoscopios convencionales o por punción transhepática comportan algunos problemas, tanto para su colocación como para mantener su permeabilidad. Los estudios sobre la eficacia de las prótesis metálicas expandibles han puesto en evidencia que con los actuales diseños hay un elevado porcentaje de casos en el que la técnica de colocación se ve dificultada o impedida por el mal funcionamiento de la prótesis en el momento de colocarla (16 % en el Wallstent y 17 % en el drenaje tipo Strecker)<sup>(34)</sup> y también se describen complicaciones relacionadas con la compresión inducida por el cuerpo metálico del drenaje<sup>(35, 36)</sup>. El segundo problema de los drenajes biliares autoexpansibles es la obstrucción que sólo en un bajo porcentaje se debe a la formación de barro biliar, la mayor parte de los casos se debe al crecimiento del tumor a través de las mallas que es significativamente menor y se produce más tarde que en los drenajes de plástico<sup>(33, 37)</sup>, aunque para algunos autores no parece ser distinta a la de los drenajes de plástico de mayor diámetro<sup>(38)</sup>. Para solucionar la obstrucción se han descrito maniobras periódicas de reducción de la masa tumoral intraluminal<sup>(39)</sup>. Otro inconveniente estriba en la imposibilidad de retirarlo o movilizarlo una vez colocado, aunque este problema está resuelto con una nueva prótesis investigada por Eran Goldin de la Universidad de Hadasa que utiliza un drenaje de níquel-titanio constituido por una lámina espiral de la que puede tirarse de uno de sus extremos, desplegarla y retirarla a través del canal operativo de un endoscopio. No hay trabajos que estudien de forma prospectiva el crecimiento intraluminal del tumor en este nuevo tipo de DBI, pero parece que no impide el crecimiento del tumor a través de las espiras<sup>(40)</sup>.

Todas esas circunstancias justifican la utilización de la técnica endoscópica para colocar drenajes biliares internos de la que existen amplias series en la literatura (Tabla 1.). Con la mayor frecuencia estos drenajes biliares se considera que ofrecen ventajas respecto a la cirugía que también se utiliza como una técnica paliativa en ictericias obstructivas tumorales del hepático común y de la zona del hilio

hepático lo que autoriza a utilizar indistintamente ambos tratamientos.

Por ello hemos comparado la eficacia de la colocación de drenajes biliares internos por endoscopia con la técnica de CPRE, utilizando drenajes 12 F y la eficacia de la cirugía convencional en el tratamiento paliativo de los tumores que afectan la vía biliar principal a nivel de hepático común e hilio hepático valorando comparativamente: 1-la posibilidad de colocar los drenajes, tanto por endoscopia como por cirugía, 2-la eficacia en obtener el drenaje de la vía biliar, aspecto que es más complejo cuando la tumorización afecta la región confluyente, 3-la morbilidad y mortalidad generada por cada técnica y establecer 5-la supervivencia, 6-calidad de vida de los pacientes de cada grupo y finalmente en el grupo bajo tratamiento endoscópico evaluar la necesidad de cambiar los drenajes que tienden a ocluirse ya que el plástico queda pronto recubierto por una película biológica sobre la que se deposita progresivamente el barro biliar<sup>(41)</sup>.

En este estudio comparativo la realización de uno u otro tratamiento fue aceptado tanto por el paciente como por su médico.

Los pacientes en los que se indicó la colocación de endoprótesis (drenaje biliar interno por vía endoscópica) fueron sometidos a una mínima papilotomía para facilitar la introducción del drenaje a través del orificio papilar. En todos los casos se utilizó un drenaje "recto" según Olympus, de calibre 12 F (4 mm de diámetro externo y 2,8 de diámetro interno) utilizando un endoscopio OLYMPUS TJF.

La exploración endoscópica fue realizada bajo efecto de sedantes y profilaxis antibiótica con clindamicina (600 mg) y gentamicina (80 mg) administrados por vía intramuscular.

Se consideraron fracasos de la endoscopia todos los casos de imposibilidad de cateterización de la papila de Vater así como la imposibilidad de colocar el drenaje a través de la estenosis. Si el drenaje era completo o parcial, se valoró tanto desde el punto de vista radiológico como biológico y clínico en el seguimiento. En el grupo de pacientes con indicación quirúrgica también se siguieron idénticos criterios de valoración : calibración conseguida; y drenaje completo o parcial de la vía biliar intrahepática.

Los resultados se analizaron independientemente de

que el fracaso de una técnica obligara a utilizar otra alternativa terapéutica en cuyo caso no se consideró la evolución, pero sí las complicaciones inmediatas y dependientes del intento terapéutico.

Para conocer las complicaciones y los primeros datos del seguimiento, los pacientes se mantuvieron ingresados durante al menos una semana después, tanto de la colocación endoscópica del drenaje, como de la descompresión quirúrgica de la vía biliar.

El seguimiento incluyó la valoración de la colestasis a la semana, a los 15 y 30 días, para efectuar más tarde controles mensuales. La valoración de la "calidad de vida" se efectuó según el "performans status", según la escala ECOG, en cada control clínico, después del alta del paciente y de forma ambulatoria. Semanalmente se estableció contacto telefónico para conocer la evolución clínica.

En el control clínico se valoraron distintos parámetros entre los que cabe destacar la disminución clínica de la ictericia y del prurito, el aspecto y consistencia de las heces.

También se controló la aparición de fiebre y se trató de determinar su etiología mediante práctica de hemocultivos.

Para el cálculo de la supervivencia se utilizaron las curvas de Kaplan Meier.

En el curso de 18 meses se incluyeron en el estudio 32 pacientes (11 ♂ y 21 ♀) con edades entre 40 y 94 años con una media de 68 años. En 23 ocasiones los pacientes fueron propuestos para la colocación de un drenaje biliar interno por vía endoscópica y en 9 se indicó el drenaje paliativo por cirugía. El grupo sometido a drenaje endoscópico tuvo una edad media superior en nueve años respecto al grupo en el que se indicó cirugía y la naturaleza de la obstrucción, en cada uno de los dos grupos, se indica en la tabla 2, donde se puede apreciar que la mayor frecuencia corresponde a tumores de la vesícula biliar invadiendo la región del hepático común próxima al hilio, bien por contigüidad, bien por la afectación de las adenopatías hiliares. El segundo tumor, por su frecuencia fue el que invadía la placa hiliar.

El drenaje endoscópico se consiguió en 15 de los 23 intentos (65 %), mientras que de los nueve intentos de solucionar de forma paliativa la obstrucción mediante técnicas quirúrgicas, esto sólo

se consiguió en tres casos (33 %). La mortalidad operatoria de cada técnica (Tabla 3) fue similar, puesto que fallecieron dentro de los 30 días siguientes al drenaje endoscópico 7 de los 23 pacientes en los que se intentó (30 %) y tres de los nueve pacientes operados (33 %).

De los 9 pacientes tratados por cirugía, en dos les fue colocado un drenaje Kher, entre uno de los lóbulos hepáticos y el colédoco por debajo de la tumoración y a uno le fue practicada una calibración de los conductos biliares del lóbulo derecho e izquierdo, según la técnica descrita por Terblanche y los seis restantes (66 %) fueron sometidos a una laparotomía, sin que se pudiera efectuar ninguna medida terapéutica para drenar la vía biliar.

Tanto los fracasos de la endoscopia, como de la cirugía fueron motivados por la imposibilidad de superar la estenosis, generalmente por estar invadida por el tumor.

Las complicaciones infecciosas (Tabla 4) fueron más frecuentes en el grupo sometido a drenaje biliar endoscópico donde catorce de los 23 pacientes (60 %) presentaron fiebre y se detectó un hemocultivo positivo para uno o varios gérmenes, mientras que esta complicación sólo se detectó en uno de los nueve pacientes tratados por cirugía (11%).

La supervivencia de los pacientes tratados por endoscopia osciló entre una semana y seis meses. La principal complicación de este tratamiento fue la colangitis que se presentó en 14 de los 23 pacientes (60 %) siendo la causa de muerte en siete (30 %). Dos pacientes presentaron pancreatitis en relación a la colangio pancreatografía retrógrada endoscópica (CPRE) que se resolvió con tratamiento médico entre uno y cuatro días después de la exploración. En el grupo quirúrgico varió entre los tres días y los ocho meses y las complicaciones propias de la cirugía condicionaron una mortalidad a los 30 días del 33 %.

La supervivencia valorada en semanas se expresa de forma gráfica en las curvas de Kaplan Meier (Figura 1) donde puede apreciarse que no existen diferencias significativas entre las dos curvas, si bien destaca que ambas técnicas motivan una mortalidad precoz importante, evidenciable por la inmediata caída de la curva de supervivencia, que es menos marcada para la técnica endoscópica y más rápida para la cirugía posiblemente en relación con las secuelas de la propia intervención

quirúrgica en los pacientes operados. En el grupo quirúrgico la mortalidad del cincuenta por ciento se alcanza a las cuatro semanas, mientras que este mismo porcentaje se alcanza a las ocho semanas para el grupo de drenajes endoscópicos.

La media de la calidad de vida de los pacientes de cada uno de los grupos (Figura 2) fue similar en un seguimiento que alcanza cinco meses en los cinco supervivientes portadores de drenajes endoscópicos y ocho meses en dos supervivientes del grupo quirúrgico.

Posteriormente en una serie más amplia de 68 pacientes se analizaron los resultados en relación al nivel de la obstrucción según esta afectara al hilio hepático, región del hepático común y la zona de cabeza del páncreas y ampolla de Vater. La eficacia en la colocación alcanzó el 75 % siendo menor en el hilio que en el hepático común y en la zona distal de la vía biliar. Los porcentajes de eficacia fueron de 65, 73 y 80 % respectivamente (Figura 3). La mortalidad global de la serie fue de 19,6 % siendo mayor en el grupo de pacientes con obstrucciones a nivel del hilio hepático (6/21) que en los que la obstrucción afectaba el hepático común o colédoco distal, donde la mortalidad a los 15 días fue del 13 % (Figura 4). La supervivencia media de esta serie calculada según Kaplan Meier no fue significativamente distinta al separar DBI proximales y distales a nivel de colédoco (Figura 5). Diez pacientes fallecieron por colangitis relacionados con la colocación del drenaje. La infección biliar fue uno de los motivos que indicaron la colocación del drenaje biliar interno en cinco pacientes, mientras que en otros cinco la colangitis se presentó después de la colocación del drenaje.

Los resultados del estudio comparativo entre endoprótesis y cirugía y los del análisis de la serie de 68 pacientes permiten establecer que la colocación de drenajes biliares internos por vía endoscópica en los tumores del hepático común e hilio hepático es una técnica relativamente sencilla, excepto cuando hay invasión tumoral de las paredes de la vía biliar a nivel de la estenosis, puesto que esta circunstancia dificulta la progresión de las guías que luego permitirán el avance de la "prótesis". La mayor complicación de la técnica es la colangitis, especialmente por gérmenes Gram negativos, cuya frecuencia se consiguió reducir utilizando una adecuada cobertura antibiótica en los días que siguieron a la colocación del drenaje endoscópico. Deben resolverse problemas derivados de la sobreinfección de las vías biliares, debe considerarse que la colocación de drenajes endoscópicos constituye una alternativa terapéutica en las obstrucciones biliares altas de naturaleza tumoral. Sin embargo esa afirmación es tanto más válida cuando los drenajes biliares se colocan en obstrucciones tumorales más bajas donde las dificultades técnicas son menores.

Por lo tanto los drenajes biliares internos, colocados por vía endoscópica constituyen una alternativa a la cirugía en caso de tumores inextirpables que precisan tratamiento paliativo y en pacientes con un muy elevado riesgo quirúrgico aún en patología benigna (Tabla 5). También deben ser considerados como tratamiento temporal en las fístulas biliares que no responden a la esfinterotomía simple o en las colangitis por litiasis biliar no pueden ser resueltas inmediatamente mediante esfinterotomía y en tumores ampulares o de cabeza de páncreas que luego podrán ser valoradas para cirugía curativa.

Tabla 1

**DRENAJE BILIAR ENDOSCOPICO**  
 Datos obtenidos de distintas series.

OBSTRUCCION			NUM	EFICACIA COLOCA-CION	SUPERVIVEN-CIA	MORTALIDAD	COMPLICACIONES		HOSPITALIZA-CION
Autor (año)	Tipo	Situación				30 b	30 d.	TARDIAS	
SIEGEL (86)	TUM	PANCREA	277	89%	X 4 meses	18%	17%	-	3,5 días
HUIBREG-TSE (86)	LQVB	HEP. COM	29	93%	-	-	-	-	-
KIIL (87)	TUM/LQV B/LIT	VARIA	399	92%	-	22%	-	-	-
NEOPTOLOMEOS (87)	TUM AMPULAR	DISTAL	21	81%	13%(5 años)	-	23%	-	-
BICKER-STAFF (90)	TUM AMPULAR	DISTAL	17	82%	X 12 meses	6%	17%	-	-
SOOMER (90)	LITIASIS	DISTAL	34	100%	seguim. 26 m.	3%	-	23%	-

**Leyenda**

TUM.: Tumor maligno.  
 HEP. COM.: Hepático Común.  
 LQVB.: Lesión quirúrgica de la vía biliar.

Tabla 2

**ICTERICIA OBSTRUCTIVA POR NEOPLASIAS BILIARES ALTAS**  
**ENDOSCOPIA vs CIRUGIA**  
**NATURALEZA DE LA OBSTRUCCION**

OBSTRUCCION POR	DREN. END.	DREN. QUIR.	TOTAL
NEOPLASIA VESICULA BILIAR	9	5	14
COMPRESION HILIO H.	5	-	5
COLANGIOCARCINOMA	2	1	3
NEOPLASIA HILIO H.	7	3	10
<b>TOTAL</b>	<b>23</b>	<b>9</b>	<b>32</b>
<b>EDAD</b>	<b>71 = 12</b> (entre 40 y 94 a)	<b>62 = 19</b>	<b>68 = 12</b>

Dren. End.: Drenaje endoscópico  
 Dren. Quir.: Drenaje quirúrgico  
 H.: Hepático

Tabla 3

**ICTERICIA OBSTRUCTIVA POR NEOPLASIAS BILIARES ALTAS  
ENDOSCOPIA vs CIRUGIA  
EFICACIA TECNICA Y MORTALIDAD**

DRENAJE BILIAR	EFICACIA	MORTALIDAD 30 d.
ENDOSCOPICO	15/23 (65%)	7/23 (30%)
QUIRURGICO	3/9 (33%)	3/9 (33%)

Tabla 4

**ICTERICIA OBSTRUCTIVA POR NEOPLASIAS BILIARES ALTAS  
ENDOSCOPIA vs CIRUGIA  
COMPLICACIONES INFECCIOSAS**

DRENAJE BILIAR	NUMERO	(%)	GERMENES	
ENDOSCOPICO	14/23	(60%)	Pseudomonas	4
			Gram. neg.	7
			Klebsiella	1
QUIRURGICO	1/9	(11%)	Pseudomonas	1

Tabla 5

**INDICACION DE COLOCACION DE DRENAJE BILIAR INTERNO MEDIANTE COLANGIOGRAFIA RETROGRADA**

**TEMPORALES**

**TUMORES**

- NEOPLASIAS QUE AFECTAN COLEDOCO DISTAL (Lygidakis 87)

**PROCESOS BENIGNOS**

- FISTULAS BILIARES QUE NO RESPONDAN A ESFINTEROTOMIA (Goldin 90)
- LITIASIS BILIAR Y COLANGITIS CON CALCULOS QUE POR SU TAMAÑO NO PUEDEN RETIRARSE INICIALMENTE (Akiyama 90)
- ESTENOSIS TRAUMATICA DE LA VIA BILIAR PRINCIPAL (Deviere 87)

**DEFINITIVOS**

**NEOPLASIAS**

- TRATAMIENTO PALIATIVO EN TUMORES SIN POSIBILIDADES QUIRURGICAS (Sonnenfeld 86)

**PROCESOS BENIGNOS**

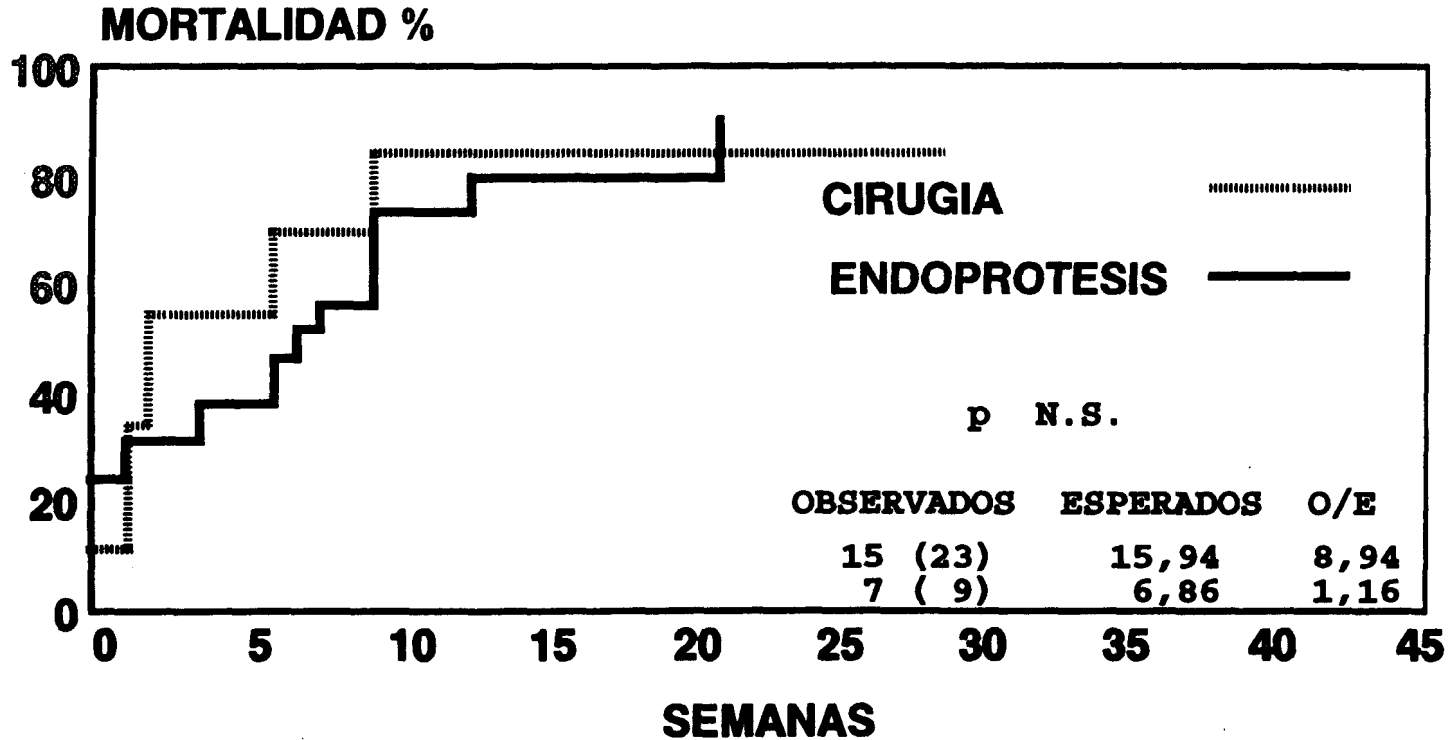
- LITIASIS BILIAR CON CALCULOS GRANDES SIN POSIBILIDADES DE SOLUCION ENDOSCOPICA Y EN PACIENTES CON EXCESIVO RIESGO QUIRURGICO (Sommers 90)

# ICTERICIA OBSTRUCTIVA POR NEOPLASIAS

## SUPERVIVENCIA

### ENDOPROTESIS (23) vs CIRUGIA (9)

30

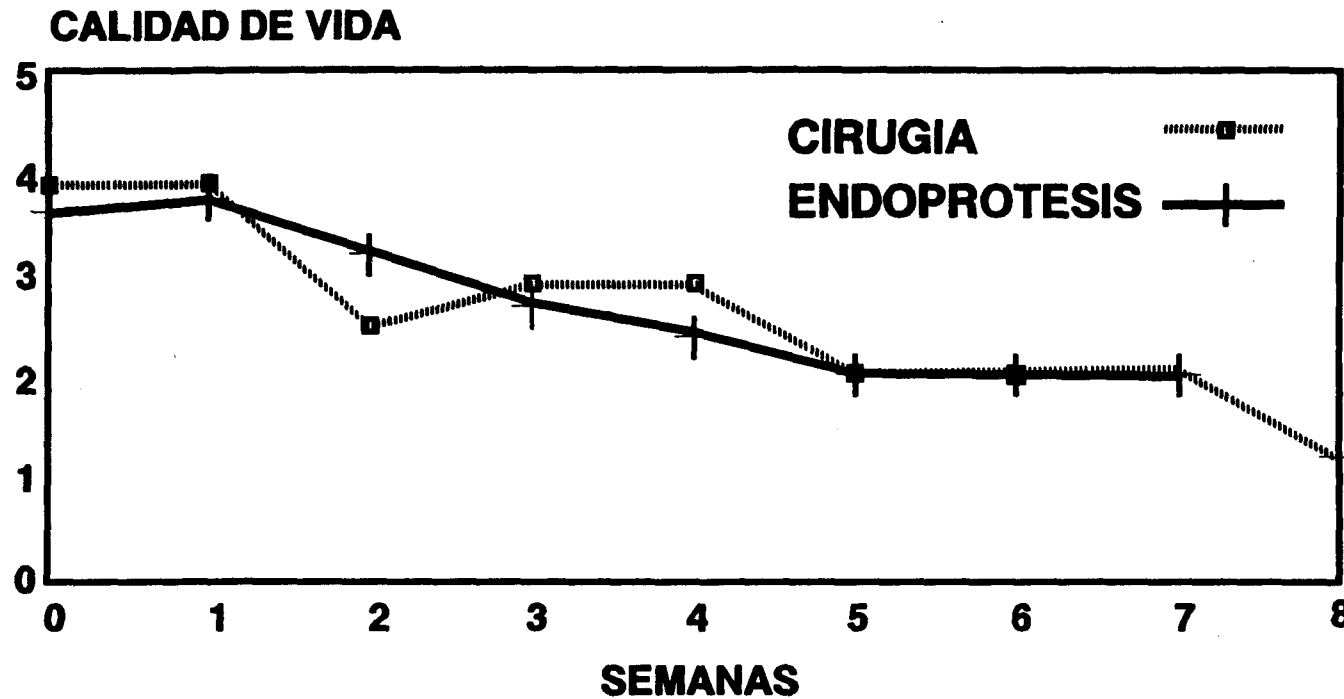


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Figura 1

# ICTERICIA OBSTRUCTIVA POR NEOPLASIAS

## ENDOPROTESIS vs CIRUGIA CALIDAD DE VIDA ("PERFORMANS STATUS")



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Figura 2

# DRENAJE BILIAR INTERNO POR ENDOSCOPIA

## EFICACIA

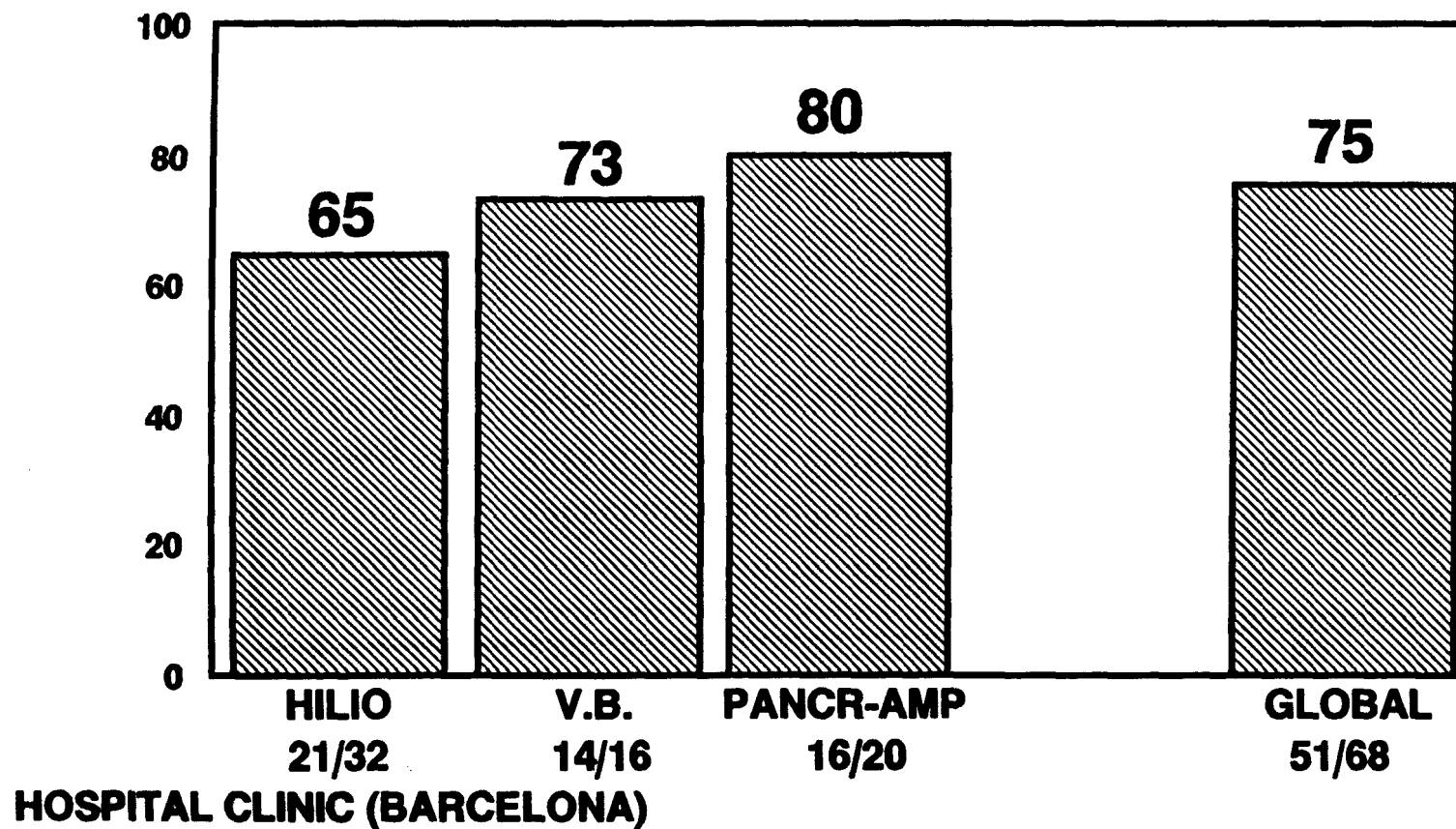
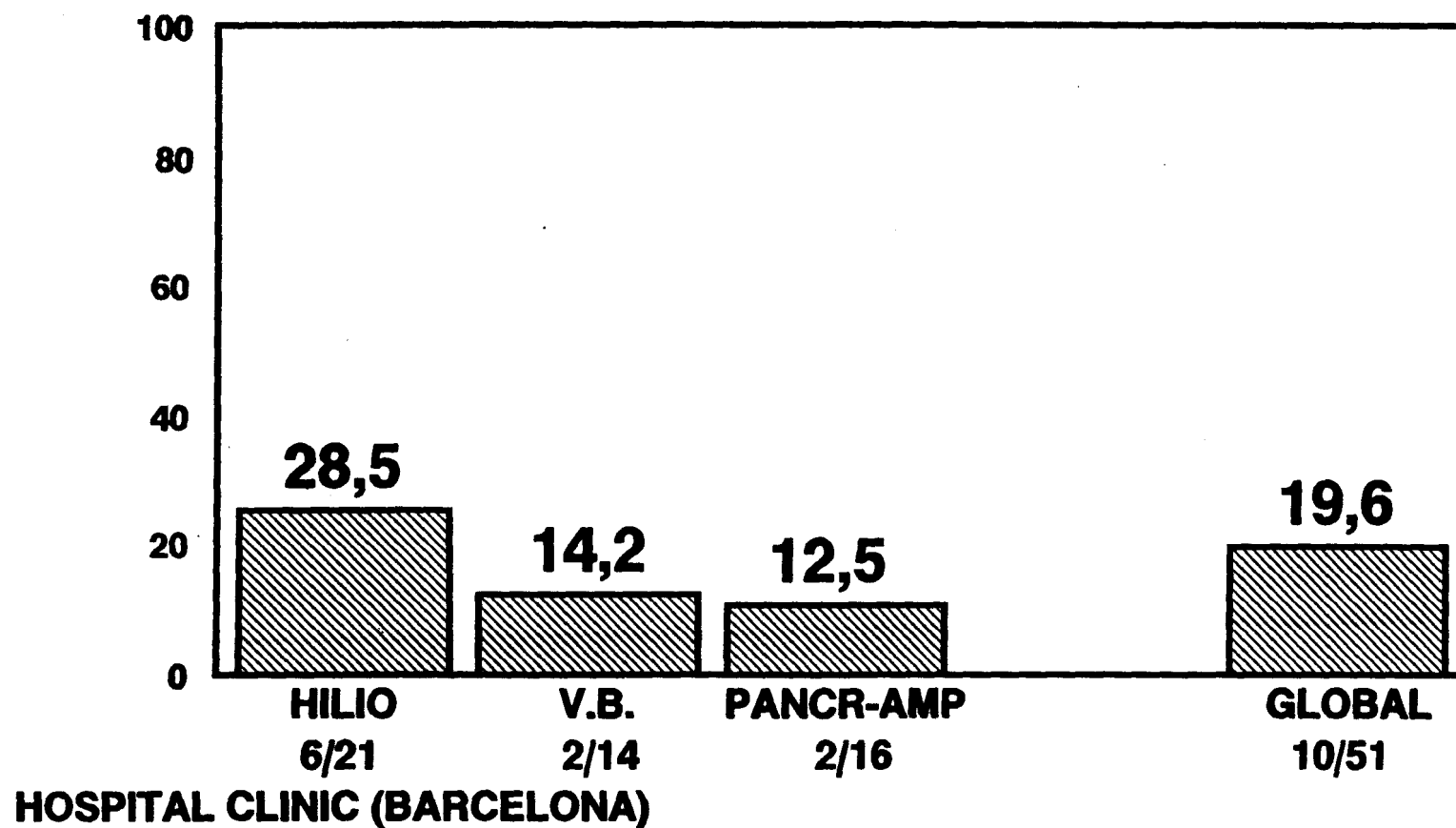


Figura 3

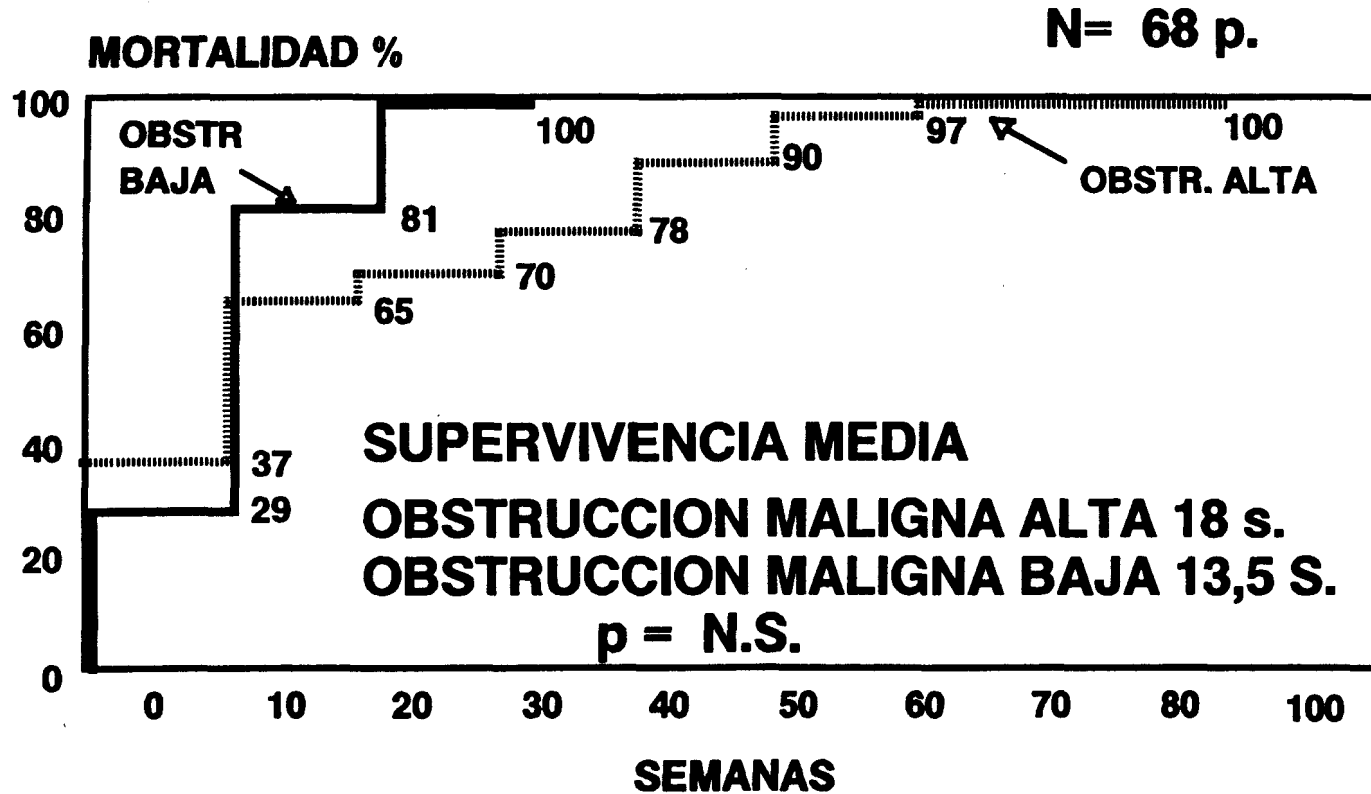


# DRENAJE BILIAR INTERNO POR ENDOSCOPIA MORTALIDAD A LOS 15 DIAS



# DRENAJE BILIAR INTERNO POR ENDOSCOPIA

## SUPERVIVENCIA ACTUARIAL (KAPLAN MEIER)



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Figura 5

**Pies de Figura**

**Figura 1**

**Curvas de actuariales de mortalidad, expresada en semanas, según Kaplan Meier, de los pacientes con drenajes biliares quirúrgicos y endoscópicos. No hay diferencias significativas entre ambas curvas.**

**Figura 2**

**Calidad de vida según la clasificación de "Performance Status". Las cifras se obtuvieron calculando la media de la calidad de vida, según la citada clasificación en relación al número de supervivientes en cada momento de la evolución.**

**Figura 3**

**La eficacia en la colocación de los drenajes biliares internos por endoscopia es inversamente proporcional a la dificultad técnica y esta es mayor cuanto más alta sea la obstrucción biliar. De todas formas también hay dificultades en las neoplasias del colédoco distal, que son más patentes cuando hay invasión tumoral del duodeno o gran deformación de la región.**

**Figura 4**

**La mortalidad de los drenajes biliares puede evaluarse a los 15 días ya que las complicaciones ligadas a la técnica son precoces. La mortalidad es tanto más elevada cuanto más difícil resulta la colocación de la endoprótesis.**

**Figura 5**

**Curvas actuariales de mortalidad en función de la situación de la obstrucción maligna de la vía biliar. Las tumoraciones pancreáticas tienen una evolución más rápida que otros tumores, lo que justifica que la supervivencia media de las tumoraciones que estenosan el colédoco distal tengan una mortalidad más precoz que las del hepático común y región hiliar.**

**Referencias.**

1. Okuda K., Tanikawa k., Emura T. et al. Nonsurgical, percutaneous transhepatic cholangiography. Diagnostic significance in medical problems of the Liver. *Am J Dig Dis* 1974; 19: 21-36.
2. Bordas J.M. La colangiografía percutánea versus la colangio-pancreatografía retrógrada endoscópica en el diagnóstico de la colestasis. En "Actualidades en Gastroenterología y Hepatología" Ed J. Rodes. C. Chantar. J.R. Prous Ed. Barcelona 1990.
3. Dick R., Platts A., Gilford J., Reddy K., Irving J.D. The Carey-Coons percutaneous biliary endoprosthesis: a three-centre experience in 87 patients. *Clin. Radiol.* 1987; 38: 175-178.
4. Madsen B. Transhepatic versus endoscopic interventional procedures in the biliary tract. *Ann Radiol. (Paris)* 1986; 29: 153-157.
5. Thoeni R.F., Fell S.C., Goldberg H.I. CT detection of asymptomatic pancreatitis following ERCP. *Gastrointest. Radiol.* 1990; 15: 291-295.
6. Geenen J.E. ERCP and the problems of sepsis. *Gastrointest Endosc.* 1982; 28: 197-199.
7. Deviere J., Motte S., Dumonceau J.M., Serruys E., Thys J.P., Cremer M. Septicemia after endoscopic retrograde cholangiopancreatography. *Endoscopy* 1990; 22: 72-75.
8. Bordas-Alsina J.M. Endoscopia digestiva (Colangiopancreatografía retrógrada). p 97-98 Eh: Mitjans de diagnostic en la medicina primaria. Ll. Canosa, J. Costa y J. Rodés Eds Salvat 1984.
9. Familiari L. La técnica della colangiopancreatografía retrograda endoscopica e tecniche diagnostiche speciali. p 25-45. En il cateterismo endoscopico della papilla di Vater. Piccin. Padova Italia 1985.
10. Kiil J., Kruse A., Rokkjaer M. Endoscopic biliary drainage. *Br J. Surg* 1987; 74: 1087-1090.
11. Deviere J., Baize J., De Toeu J., Cremer M. Long-Term follow-up with hilar malignant stricture treated by endoscopic internal biliary drainage. *Gastrointest Endosc* 1988; 34: 95-101.
12. Sar G.M., Cameron L.J. Surgical management of unresectable carcinoma of the pancreas. *Clinical Review. Surgery* 1982; 91: 123-133.
13. Grace P.A., Pitt H.A., Tompkins H.A., Derbesten L., Longmyre W.P. Jr. Decreased morbidity and mortality after pancreatoduodenectomy. *Am J. Surg* 1986; 151: 141-149.
14. Tuchiye R., Node T., Harada N., et al. Collective review of small carcinomas of the pancreas. *Ann Surg* 1986; 303: 77-81.
15. Lygidakis N.J., Brummelkamp W.H., Huijbregtse K., Tytgat G.N. Different response to preliminary biliary drainage in proximal versus distal malignant biliary obstruction. *Surg Gynecol Obstet* 1987; 164: 159-162.
16. Lewis W.D., Cady B., Rohrer R.J., Jenkins R.L., Benotti P.N., McDermott W.V. Jr. Avoidance of transhepatic drainage prior to hepaticojejunostomy for obstruction of the biliary tract. *Surg Gynecol Obstet* 1987; 165: 381-386.
17. Cotton P.B. Endoscopic methods for relief of malignant obstructive jaundice. *World J. Surg.* 1984; 8: 854-861.
18. Cotton P.B. Improved biliary decompression with large caliber endoscopic prosthesis. *Gastrointestinal Endoscopy* 1984; 30: 377-378.
19. Siegel J.H. Combined endoscopic dilation and insertion of large diameter endoprosthesis for bile duct obstruction. *Gastrointest Endosc* 1984; 30: 91-92.
20. Siegel J.H. Improved biliary decompression with large caliber endoscopic prosthesis. *Gastrointest Endosc* 1984; 30: 21-23.
21. Rey J.F., et al Experimental study of biliary endoprosthesis efficiency. *Endoscopy* 1985 Jul 1985; 17: 145-148.
22. Soehndre N., Binmoeller K., Seitz F., Grimm H. Biliary endoprostheses without side holes have longer patency. *Endoscopy* 1992; 24: 635A.
23. Coene P.P., Groen A.K., Cheng J., Out M.M., Tytgat G.N., Huijbregtse K. Clogging of biliary endoprostheses: a new perspective. *Gut* 1990; 31: 913-917.
24. Leung J.W.C., Lau G.T.C., Sung J.J.Y., Costerton J.W. Decreased bacterial adherence to silver coated stent material: an in vitro study. *Gastrointest Endosc* 1992; 38: 338-340.
25. Nishimura O. Efficacy of endoprosthesis for internal biliary drainage in patients with nonresectable carcinoma of the proximal bile duct. Nishimura O. et al. *Japan J. Surg.* 1984; 14: 339-344.
26. Gabrielsson N., Granqvist S., Sonnenfeld T. Endoscopically inserted endoprosthesis in inoperable biliary obstruction. *Acta Radiol* 1985; 26: 57-61.
27. Siegel J.H., Snady H. The significance of endoscopically placed prosthesis in the management of biliary obstruction due to carcinoma of the pancreas: results of nonoperative decompression in 277 patients *Am J. Gastroenterol.* 1986; 81: 634-641.
28. Andreani T., Buffet C., Etienne J.P. Jaundice caused by neoplastic obstruction. Role of non surgical palliative treatment. *Gastroenterol Clin Biol* 1986; 10: 308-318.
29. Sonnenfeld T., Gabrielsson N., Grandquist S., Perbeck L. Nonresectable malignant bile duct obstruction. Surgical bypass or endoprosthesis? *Acta Chir Scand* 1986; 152: 297-300.
30. Walz D.C., Fausel C.S., Brant B. Endoscopic biliary stents and obstructive jaundice. *Am J Surg* 1987; 153: 444-447.
31. Alvarado R., Palmaz J.C., Garcia O.J., Tio F.O., Rees C.R. Evaluation of polymer-coated balloon-expandable stents in bile ducts. *Radiology* 1989; 170: 975-978.

**IV CURSO INTERNACIONAL DE RADIOLOGIA VASCULAR INTERVENCIONISTA COMO ALTERNATIVA TERAPEUTICA**

32. Huibregtse K., Cheng J., Coene P.P.L.O., Fockens P., Tytgat G.N.J. Endoscopic placement of expandable metal stents for biliary strictures- A preliminary report on experience with 33 patients. *Endoscopy* 1989; 21: 280-282.
33. Huibregtse K., Carr-Locke D.L., Cremer M., Domachke W., Fjockens P., Poerster E., Hagenmuller E., Hatfield A.R., Blefibre J.F., Liguory C.L., Matzen P., Neuhaus H., Sugai B.M., Williams S. Biliary stent occlusion- A problem solved with self-expanding metal stents?. *Endoscopy* 1992; 24: 391-394.
34. Bethge N., Wagner H.J., Knyrm K., Zimmermann H.B., Stark E., Pausch J., Vakil N. Technical failure of biliary metal stent deployment in a series of 116 applications. *Endoscopy* 1992; 24: 395-400.
35. Ee H., Laurence B.H. Hemorrhage due to erosion of a metal biliary stent through the duodenal wall. *Endoscopy* 1992; 24: 431-432.
36. Steenbergen W.V., Van Aken I., Ponette E. Acute pancreatitis complicating the insertion of selfexpandable biliary metal stent. *Endoscopy* 1992; 24: 440-442.
37. Lammer J., Klein G.E., Kleinert R., Hausegger K., Einspieler R. Obstructive jaundice: use of expandable metal endoprosthesis for biliary drainage: work in progress. *Radiology* 1990; 177: 789-792.
38. Adam A., Chetty N., Roddie M., Yeung E., Benjamin I.S. Selfexpandable stainless steel endoprosthesis for treatment of malignant bile duct obstruction. *AmJ Roentgenol* 1991; 156: 321-326.
39. Cremer M., Deviere J., Sugai B., Baize M. Expandable biliary metal stents for malignancies: endoscopic insertion and diathermic cleaning for tumor ingrowth. *Gastrointest Endosc* 1990; 36: 451-457.
40. Goldin E., Beyer M., Safre T., Wengrower D., Globerman O., Fich A. Results of a new self-expandable and removable metal stent for biliary obstruction. *Endoscopy* 1993; 25: 330A.
41. Leung J.W.C., Ling T.K.W., Kung J.L.S., Wallace-Owen J. The role of bacteria in the blockage of biliary stents. *Gastrointest Endosc* 1988; 34: 19-22.

## New Approaches in the Pharmacological Treatment of Portal Hypertension

JAIME BOSCH<sup>1</sup>

### CLINICAL/HAEMODYNAMIC CORRELATIONS IN THE PHARMACOLOGICAL TREATMENT OF PORTAL HYPERTENSION

The pharmacological treatment of portal hypertension is based on the assumption that a sustained reduction in portal pressure shall reduce the incidence of the complications of portal hypertension. Actually, it is well known that for varices to develop and rupture, the portal pressure gradient (usually evaluated as the hepatic venous pressure gradient or HVPG) shall increase above 12 mm Hg. Recent studies have confirmed that when HVPG is reduced below this threshold gradient the patient is totally protected from the risk of variceal haemorrhage, the varices progressively decrease in size and survival is significantly increased. In addition, it is likely that when HVPG is very high, a substantial reduction (i.e., of more than 20% of baseline value) may also translate into a significant decrease in the risk of variceal bleeding, even if the final value is above the 12 mm Hg threshold gradient. This is because variceal rupture is thought to occur when the tension exerted on the walls of the varices, which is directly related to variceal pressure, exceeds a critical value. Therefore, a marked decrease in portal pressure (which leads to at least a similar fall in variceal pressure) shall markedly decrease variceal tension and the likelihood of variceal haemorrhage.

### LIMITATIONS OF PROPRANOLOL THERAPY

Propranolol and nadolol are now well accepted for the prevention of variceal bleeding or rebleeding. However, we have shown that only 24% of patients decrease HVPG by more than 20% after propranolol administration, and that only in 12% of

the cases the final HVPG is  $\leq 12$  mmHg. On the other hand, nearly 40% of patients fail to exhibit a reduction in HVPG following propranolol. It appears therefore that in order to improve the results of pharmacological therapy with propranolol, we have either to select patients exhibiting better hemodynamic response (which would limit the applicability of therapy), or to develop alternative therapies that can offer effective reductions in HVPG in a greater proportion of patients.

Other limitations of therapy with non-selective beta-blockers are due to their contraindications and side effects. Among the former, the more frequently encountered are chronic obstructive lung disease, A-V heart blocks, arrhythmias, psychosis and insulin-dependent diabetes with past history of hypoglycemia. Side effects are relatively frequent (about 15% patients), but severe events (bronchospasm, etc) are rare. The more frequent complaints are fatigue (that often is associated with marked bradycardia, with heart rate below 60 bpm) and sleep disorders. Although complications from propranolol therapy in cirrhosis have never been lethal, side effects are important in as much as they detract from compliance. Nadolol is easier to administer because of more prolonged half-life (allowing once-a-day administration) and is eliminated by the kidneys, which makes its dosage easier than that of propranolol. Also, it has been suggested that since it does not cross the blood-brain barrier it is less likely to cause central effects and side effects. However, this has not been adequately investigated in cirrhosis.

### ALTERNATIVE PHARMACOLOGICAL TREATMENT FOR PORTAL HYPERTENSION

There has been a lot of attention paid to the possible use of vasodilators in recent years. These

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drugs may reduce portal pressure by decreasing the vascular resistance to portal and collateral blood flow, and also, by promoting reflex splanchnic vasoconstriction as a response to reduced mean arterial and cardiac filling pressures. A theoretical advantage of vasodilators over beta-blockers is that the former may allow to reduce portal pressure without impairing liver perfusion. Its most important limitation is that all vasodilators reduce arterial blood pressure, which may be poorly tolerated by patients with advanced cirrhosis (especially in those with ascites and edema), who are frequently hypotensive.

Nitrovasodilators such as isosorbide dinitrate or isosorbide 5-mononitrate does cause marked reductions in HVPG in acute administration, but much less in chronic therapy. Tolerance to nitrovasodilators may be due to true pharmacological tolerance, to the activation of endogenous neurohumoral vasoactive systems, and to plasma volume expansion. The latter suggests the possibility that the association of a diuretic may enhance the effects of these compounds in HVPG (in analogy with findings in the treatment of arterial hypertension). Otherwise, it is unlikely that nitrovasodilators *alone* may become useful agents in the treatment of portal hypertension, because of their small effect on portal pressure. Molsidomine has effects that are superimposable to those of isosorbide dinitrate and is thought not to cause tolerance, but this has not been assessed by long-term comparative studies in patients with portal hypertension.

Serotonin S<sub>2</sub>-receptor blockers were introduced following the observation that mesenteric veins from portal hypersensitive to the vasoconstrictor effects of serotonin. Ritanserin and other serotonin S<sub>2</sub>-receptor blockers such as ketanserin have been shown to be able to reduce portal pressure in portal hypertensive models and/or in patients with cirrhosis. This is in part due to lowering in portal-collateral resistance. It is important to remark that selective S<sub>2</sub> antagonists do not cause any decrease in arterial pressure. The potential for the use of these drugs, either alone or in combination with other agents requires further investigation.

Clonidine is a centrally acting alpha-2 adrenergic agonist, which results in reduced adrenergic output. Clonidine reduces portal pressure by decreasing portal resistance and splanchnic inflow. Arterial pressure is also markedly reduced, but in this case it does not appear to be associated to adverse effects on renal function or sodium handling. The

magnitude of the fall in portal pressure is slightly higher than that achieved with propranolol.

Spirolactone has been shown to reduce HVPG in patients with cirrhosis. This is based on the known relationship between plasma volume and portal pressure. In addition, the reduction in plasma volume caused by spironolactone attenuates the increase in cardiac output observed in cirrhosis and triggers vasoactive mechanisms that decrease the splanchnic blood flow. There is a potential for spironolactone to maintain and enhance the decrease in portal pressure caused by nitrovasodilators or propranolol. Clearly, the degree of plasma volume expansion is an important variable influencing portal pressure, which has not been taken into account in the past. Concomitant use of diuretics may explain in part some conflicting results observed in previous studies and clinical trials.

### COMBINATION THERAPY

It is unlikely that any single agent may offer a fall in portal pressure marked enough to provide effective protection from the risk of variceal bleeding or rebleeding to most patients with cirrhosis. This is probably easier to achieve using combinations of drugs acting through different mechanisms. Again, this is a situation in which the strategy for the treatment of portal hypertension may profit from the experience gained in the treatment of systemic hypertension.

The combined administration of propranolol and isosorbide 5-mononitrate has been shown to cause a significantly greater reduction in HVPG than either drug alone. Acute and long-term hemodynamic studies have shown that this drug association markedly reduces the number of patients who do not reduce portal pressure, while increasing the proportion of cases in which HVPG is markedly decreased. This is thought to be due to the fact that isosorbide 5-mononitrate prevents the increase in portal resistance caused by propranolol. This association is also superior to propranolol alone in maintaining liver perfusion and hepatic function, while the beneficial effect reducing azygos blood flow is unchanged. Whether these greater hemodynamic effects translate into better clinical results should be verified by randomized controlled studies.

As already mentioned, other drug combinations have potential for a synergistic effect reducing

portal pressure. These include the association of propranolol or nadolol with an S2-blocker, the association of propranolol or nadolol with spironolactone, the association of a nitrovasodilator (or molsidomine) and diuretics, or the simultaneous use of three drugs (beta-blocker + nitrovasodilator + spironolactone, or beta-blocker + S2-antagonist + spironolactone). Obviously, further research is needed to clarify these important questions.

## References

1. García Tsao G., Groszmann R.J., Fisher R.L., Conn H.O., Atterbury C.E., Glickman M. Portal pressure, presence of gastroesophageal varices and variceal bleeding. *Hepatology* 1985; 5: 419-424.
2. Groszmann R.J., Bosch J., Grace N.D., et al. Hemodynamic events in a prospective randomized trial of propranolol vs. placebo in the prevention of the first variceal hemorrhage. *Gastroenterology* 1990; 99: 1401-1407.
3. Feu F., Bordas J.M., García-Pagán J.C., Bosch J., Rodés J. Double blind investigation of the effects of propranolol and placebo on the pressure of esophageal varices in patients with portal hypertension. *Hepatology* 1991; 13: 917-922.
4. Pereira O., García-Pagán J.C., Feu F., et al. Factors influencing the portal pressure response to propranolol administration in patients with cirrhosis. *Hepatology* 1991; 14: 133A.
5. Bosch J., Mastai R., Kravetz D., et al. Effects of propranolol on azygos venous blood flow and hepatic and systemic hemodynamics in cirrhosis. *Hepatology* 1984; 4: 1200-1205.
6. García-Pagán J.C., Feu F., Navasa M., et al. Long-term haemodynamic effects on isosorbide-5-mononitrate in patients with cirrhosis and portal hypertension. *J. Hepatol* 1990; 11: 189-195.
7. García-Pagán J.C., Navasa M., Bosch J., Dru C., Fizueta P., Rodés J. Enhancement of portal pressure reduction by the association of isosorbide-5-mononitrate to propranolol administration in patients with cirrhosis. *Hepatology* 1990; 2: 230-238.
8. García-Pagán J.C., Feu F., Bosch J., Rodés J. Propranolol compared with propranolol plus isosorbide-5-mononitrate for portal hypertension in cirrhosis. A randomized controlled trial. *Ann. Intern. Med.* 1991; 114: 869-873.



# Tratamiento intervencionista de la HTA vasculorrenal.

ANGIOPLASTIA TRANSLUMINAL PERCUTÁNEA RENAL

J. Montañá Figuls y J.M.<sup>a</sup> Callejas Pérez

## Introducción

La angioplastia transluminal percutánea de arteria renal (ATP de AR) es el procedimiento de revascularización renal con menos riesgo para el paciente y con más probabilidades de éxito.

La ATP se empezó a practicar en zonas periférica y cardíaca en los años 1978 y 1979<sup>1,2</sup>. Después de hacerse pública su utilización a nivel renal, esta técnica se ha popularizado con rapidez en los últimos años<sup>3</sup>.

## Técnica

La ATP cateteriza con precisión por procedimientos angiográficos, la arteria renal estenosada. Se hace pasar a través de la estenosis un catéter especial, que tiene un globo casi en su extremo, que se puede hinchar a voluntad con una presión controlada (fig. 23.1). Al hincharlo con una presión de entre cinco a diez atmósferas, en el centro de la estenosis, se provoca su dilatación, que en general persiste después de retirado el catéter<sup>4,5,6</sup>.

Aunque es una técnica bastante simple, debe ser practicada sólo por personal especializado y en centros donde pueda ser

cederse sin demora a la solución de las posibles complicaciones. A pesar de ser una técnica de naturaleza angiográfica o radiológica, no debe practicarse sin un entorno quirúrgico adecuado.

## Indicaciones

Los beneficios de la ATP en las estenosis de arteria renal pueden considerarse desde dos aspectos: a) mejoría o curación de la hipertensión, y b) mejoría de la función renal.

Los dos aspectos van muchas veces relacionados y, sobre todo, son esenciales en los pacientes monorrenos, ya sea por trasplante renal o porque tengan un solo riñón por otras causas.

Podemos dividir a los enfermos tributarios de tratamiento mediante ATP, en los tres grupos siguientes:

1. Estenosis posquirúrgicas (figs. 23.2 y 23.3). Tanto en los injertos renales por insuficiencia renal, como en las intervenciones por hipertensión (autotrasplante, *bypass*), pueden producirse estenosis. La reintervención quirúrgica de estos pacientes presenta muchos problemas técnicos y pocas probabilidades de éxito. El tratamien-

TRATAMIENTO DE LA HIPERTENSIÓN ARTERIAL

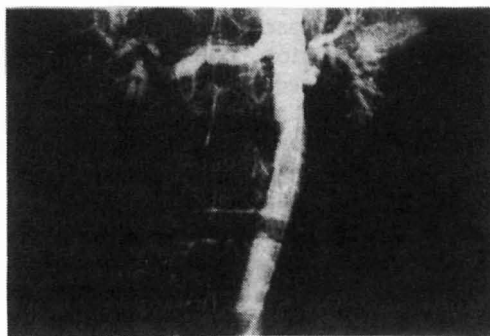
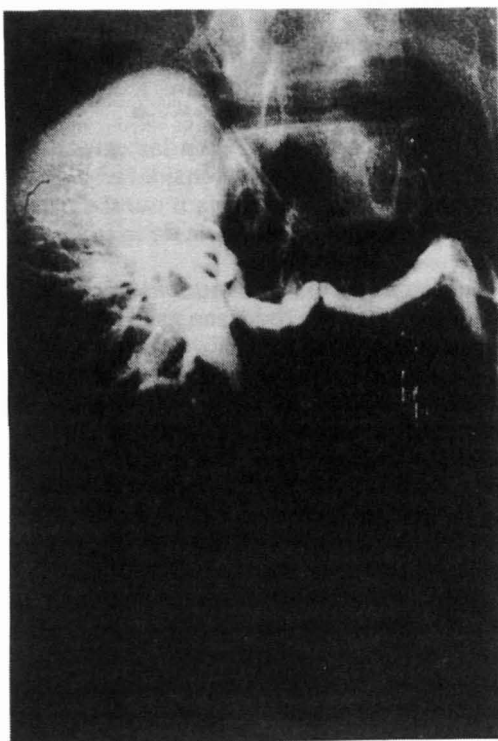


**Fig. 23.1.** Catéter de Grüntzig, con el globo hinchado, en el centro de una estenosis de arteria renal. Se observa la hendidura que provoca la estenosis en el globo.

to de elección es en estos casos la ATP, al conseguir excelentes resultados en la mayoría de las ocasiones. Merece resaltarse, en el caso de los pacientes trasplantados, que además de solucionar su hipertensión, se les restablece también la función renal.

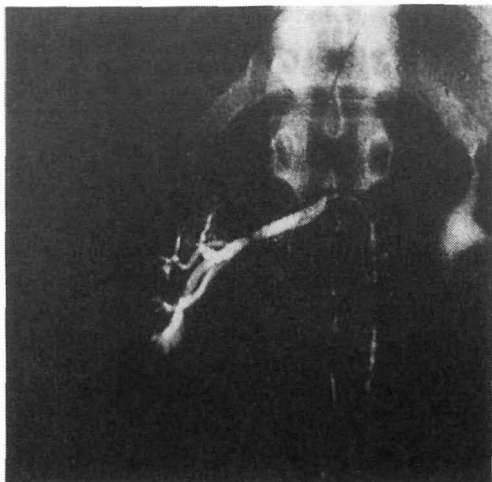
2. Estenosis displásicas (figs. 23.4, 23.5, 23.6 y 23.7). En las displasias de arteria renal, el primer tratamiento de elección es también la ATP. Los resultados en cuanto a la curación de la HTA son muy buenos y superponibles a los resultados quirúrgicos, con una menor morbilidad. Existen sin embargo, desde el punto de vista técnico, algunas dificultades, sobre todo cuando las estenosis son muy cerradas, o en algunos casos en que las múltiples irregularidades impiden el correcto desplazamiento de la guía angiográfica o del catéter.

3. Estenosis ateromatosas (figs. 23.8 y 23.9). En las estenosis ateromatosas debe tenerse siempre en cuenta al conjunto de la enfermedad ateromatosa, puesto que la afectación renal no es, por lo general,

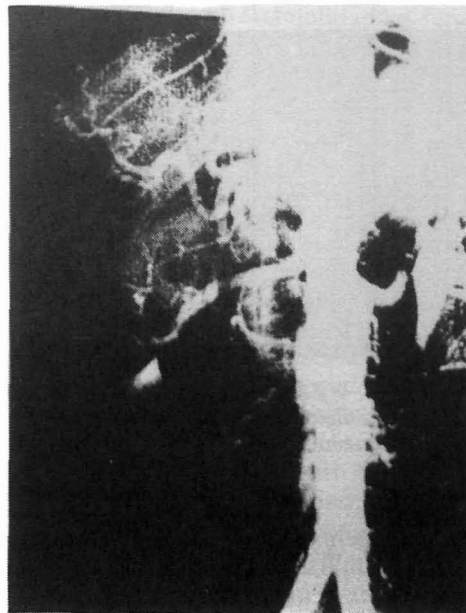


**Figs. 23.2. y 23.3.** Estenosis de una anastomosis terminoterminal quirúrgica. Desaparición de dicha estenosis en la comprobación.

TRATAMIENTO INTERVENCIONISTA DE LA HTA VASCULORRENAL



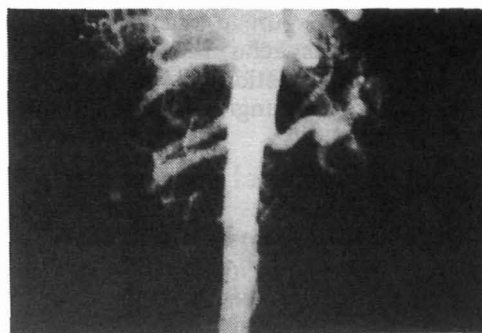
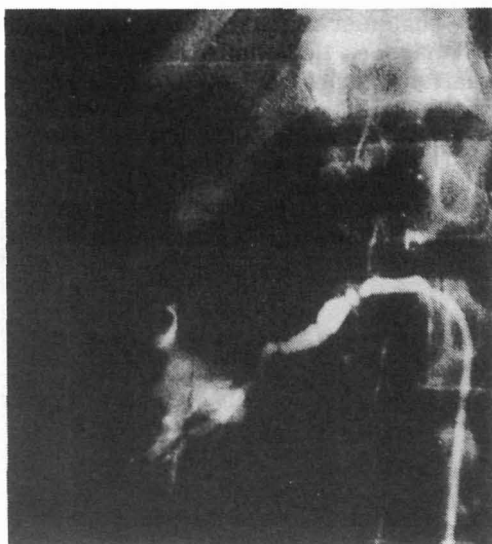
**Figs. 23.4 y 23.5.** Estenosis displásica de arteria renal, con una buena dilatación de la misma después de la angioplastia.



la única en estos pacientes. Éstos presentan un alto riesgo quirúrgico y, excepto si presentan lesiones asociadas aortoiliacas quirúrgicas, debe plantearse como primera opción la angioplastia.

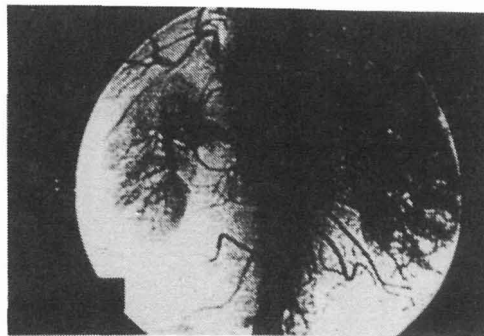
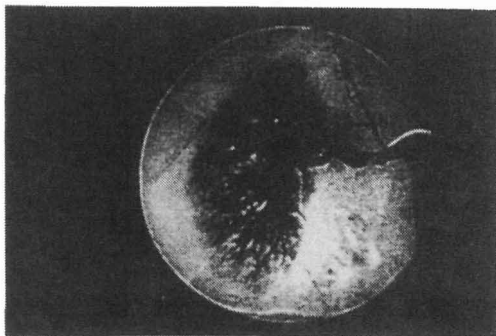
Sin embargo, es importante señalar que la dilatación no siempre será efectiva, so-

bre todo cuando se trata de placas de ateroma aórticas que afectan al *ostium* renal, que, en ocasiones, son imposibles de dilatar (figs. 23.10 y 23.11). Además, por la propia evolución de la enfermedad, no son infrecuentes las reestenosis. Cuando se trata de pacientes con muchos años de evolu-



**Figs. 23.6 y 23.7.** Doble arteria renal derecha, con estenosis por displasia de la arteria inferior. Aortografía postangioplastia con muy buena permeabilidad de dicha arteria.

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**Figs. 23.8. y 23.9.** *Importante estenosis ateromatosa de la primera porción de arteria renal derecha. Aortografía digital de comprobación, con desaparición de la estenosis, observándose en cambio una imagen de pequeña disección subintimal.*

ción, no siempre será efectiva la dilatación para normalizar la tensión. No obstante, será útil en muchos casos para mejorar la función renal, sobre todo cuando se trata de pacientes monorrenos, que están abocados con rapidez a una insuficiencia renal.

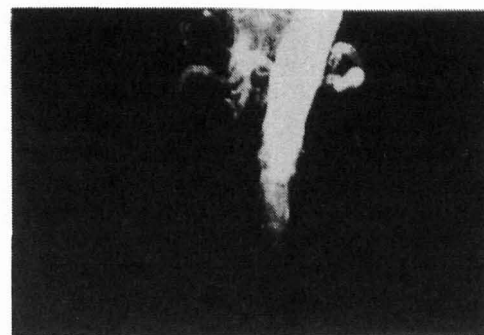
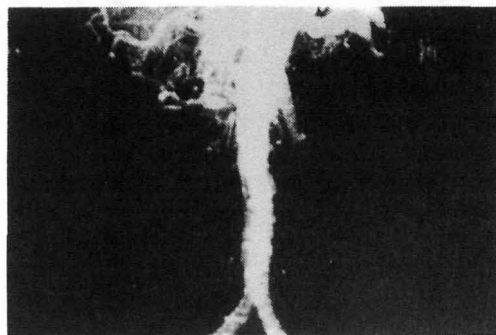
### Metodología

#### Diagnóstico de la HTA vasculorrenal

En un período de pocos años, la aparición de la angiografía digital ha revolucionado los procedimientos para diagnosticar la hipertensión secundaria a la estenosis de arteria renal. Según lo acostumbrado, el paciente con sospecha clínica de HTA vasculorrenal es sometido en primer lugar a la práctica de una angiografía digital intra-

venosa (ASD/i.v.), que proporciona una visión directa de las arterias y permite afirmar o descartar la estenosis de arterias renales. Aprovechando el contraste de la ASD/i.v., conviene practicar una urografía descendente, que proporciona datos importantes morfofuncionales del aparato urinario. Si en vez de practicar la ASD/i.v. por punción de una vena del codo, como es lo habitual, se realiza mediante cateterismo femoral, se puede aprovechar este último y hacer una extracción de sangre de las venas renales para la determinación de reninas centrales.

Debe tenerse en cuenta que la ASD/i.v. es una exploración de *screening*, por lo que ante cualquier duda en la interpretación de las imágenes, debe hacerse un estudio intraarterial, para comprobar las lesiones existentes.



**Figs. 23.10. y 23.11.** *Paciente monorreno, con estenosis severa del ostium de la arteria renal. Buena permeabilidad de dicha arteria renal tras la dilatación.*

### Técnica de la angioplastia transluminal

Con el diagnóstico probable de estenosis de arteria renal, conseguido por angiografía digital, se programan a la vez la arteriografía de comprobación de las lesiones y la angioplastia. El paciente debe tomar, dos días antes, medicación antiagregante. Por ejemplo: persantín 300 mg/día y ácido acetilsalicílico 500 mg/día.

Se hace la arteriografía, normalmente por cateterismo femoral, y si se comprueba la estenosis, se decide tratamiento, inyectando antes, en la arteria renal que corresponda, 4.000 U de heparina y 40 mg de lidocaína (para evitar trombosis y espasmos arteriales). Se pasa la guía angiográfica a través de la estenosis y se desliza a continuación sobre ella el catéter de dilatación. Cuando el globo está situado en el centro de la estenosis, se procede a hincharlo. Para controlar el proceso se rellena el globo con contraste y se administra una presión de unas 5 a 7 atmósferas durante 30-60 segundos. Según la resistencia de la estenosis a dilatarse, se repite varias veces dicho proceso. Por último, se retira el catéter de dilatación y se realiza una aortografía de comprobación para evaluar el resultado obtenido.

Después de la dilatación, el paciente debe permanecer ingresado unas 48 horas, para controlar el punto de punción y adaptar la medicación hipotensora a la nueva situación creada.

### Control posterior

Después de practicar una angioplastia, el paciente debe seguir recibiendo medicación antiagregante durante un tiempo no inferior a tres meses. Es conveniente, en algunos casos, prolongar mucho más dicho tratamiento.

En general, si los resultados clínicos son buenos, no es necesario practicar angiografías digitales de comprobación; pero cuando un paciente presenta un empeoramiento clínico, no debe dudarse en practicar dicha angiografía y, en el caso de que se compruebe una nueva estenosis, iniciar de nuevo todo el proceso.

### Fisiopatología

No son conocidos en su totalidad los fenómenos que tienen lugar en una arteria estenosada que ha sido dilatada, y la razón de que ésta permanezca permeable. Las primeras teorías formuladas a este respecto establecían cinco fenómenos principales: compresión de la placa de ateroma; redistribución de la placa; embolización de pequeñas partículas; regresión de la placa de ateroma por mecanismo de fagocitosis, y por último, estrechamiento de la pared arterial<sup>7</sup>.

Algunos puntos de las teorías no tardaron en ser rebatidos, en especial los que hacían referencia a la compresión y redistribución de la placa de ateroma; su dureza hace que sea compresible con dificultad<sup>8</sup>.

Por el contrario, el estrechamiento de la pared parece ser uno de los mecanismos fundamentales para devolver una buena luz a las arterias con las paredes engrosadas. Por regla general se produce una rotura de la íntima, y en los ateromas, una impactación de la placa, en las capas muscular y adventicia, aumentando entonces el calibre externo de la arteria.

Las imágenes angiográficas inmediatas a la angioplastia ofrecen en ocasiones imágenes de disección intimal secundaria a la rotura, pero nada tienen que ver con el resultado clínico obtenido<sup>9</sup>. La evolución espontánea de dicha rotura intimal es cicatrización con formación de luz más amplia (curación). Pero también puede evolucionar a una formación de reestenosis. De aquí la importancia de realizar un correcto tratamiento antiagregante posterior.

### Resultados de la ATP de arteria renal

Es difícil dar un único resultado final, en una técnica como la ATP, ya que, en general, se trata de grupos de pacientes heterogéneos, en los que la causa de hipertensión no sólo puede ser la estenosis de arteria renal existente. Por tanto, un buen resultado angiográfico, con una buena restitución de la luz de la arteria, puede no ser un buen resultado clínico.

Se obtiene una buena dilatación morfológica de la estenosis en cerca del 90 % de los pacientes a los que se practica una dilatación de arteria renal.

En relación al resultado en cuanto a la HTA, deben clasificarse los pacientes en los tres grupos siguientes: a) pacientes curados, es decir, que se convierten en normotensos tras la dilatación, sin necesitar ningún tratamiento médico; b) pacientes con una mejoría clínica, en los que se consigue un mejor control de la hipertensión, con menor medicación, y c) un grupo de enfermos sin ningún cambio tras la dilatación<sup>10</sup>.

El resumen de los resultados de la abundante literatura sobre el tema, coincide con la experiencia personal de un grupo de 112 pacientes, y es el siguiente: pacientes curados, un 25 %; pacientes con mejoría clínica, un 55 %, y pacientes a los que la angioplastia no ha aportado ningún beneficio, un 20 %. Al hablar de los resultados, es importante constatar una mejoría de la función renal en un 15 % de los pacientes.

Según qué autores, la recurrencia de las estenosis ocurre entre un 12 y un 22 %. Esta incidencia es claramente más alta en los pacientes ateromatosos. También debe tenerse presente que en las reestenosis se puede practicar nuevamente una angioplastia sin mayores problemas. De todas maneras, cuando la recidiva se presenta de forma precoz en los primeros meses, el pronóstico desaconseja una nueva dilatación, y es más aconsejable el tratamiento quirúrgico.

### **Complicaciones de la ATP de arteria renal**

Las complicaciones de la angioplastia de arteria renal oscilan entre el 5 y el 10 %, y son en general menores. Las principales son los hematomas en el punto de punción (arterias femoral o axilar).

Además son posibles espasmos de las arterias intrarrenales y embolización a dis-

tancia de pequeños émbolos. Entre las complicaciones más graves se encuentran la trombosis de arteria renal o su disección, llegando a originar un hematoma renal o incluso retroperitoneal. Por ello es importante realizar las angioplastias con un entorno quirúrgico adecuado.

En nuestra serie personal de 112 pacientes no ha sido necesario, a pesar de ello, proceder a ningún tratamiento quirúrgico de urgencia, y sólo en un caso fue necesario tratar quirúrgicamente un hematoma de la zona de punción femoral.

### **BIBLIOGRAFÍA**

1. Gruntzig A, Kumpe DA. Technique of percutaneous transfemoral angioplasty with the Gruntzig balloon catheter. *AJR* 1979; 132: 547-552.
2. Gruntzig A, Senning A, Siegenthaler WE. Non-operative dilatation of coronary artery stenosis: Percutaneous transluminal coronary angioplasty. *N Eng J Med* 1979; 301: 61-68.
3. Gruntzig A, Vetter W, Meier B et al. Treatment of renovascular hypertension with percutaneous transluminal dilatation of a renal artery stenosis. *Lancet* 1978; 1: 801-802.
4. Schwarten DE. Transluminal Angioplasty of Renal Artery Stenosis: 70 experiences. *AJR* 1980; 135: 969-974.
5. Puijlaert CBAJ, Boomsma JHB, Ruijs JHJ. Transluminal renal artery dilatation in hypertension: technique, results and complication in 60 cases. *Urol Radiol* 1981; 2: 201.
6. Tegtmeier J, Kellum CD, Ayers C. Percutaneous transluminal angioplasty of the renal arteries. Results and long term follow up. *Radiology* 1984; 153: 77-84.
7. Wolf GL, Le Veen RF, Ring EJ. Potential mechanism of angioplasty. *Cardiovasc Intervent Radiol* 1984; 7: 11-17.
8. Zollkofer ChL. Experimental Basis of Percutaneous Transluminal Angioplasty. *Frontiers in European Radiology* 1987; 5: 51-135.
9. Castañeda-Zúñiga WR, Tadavarthy SM, Laerum F, et al. Pseudo intramural injection following percutaneous transluminal angioplasty. *Cardiovasc Intervent Radiol* 1984; 7: 104-108.
10. Tegtmeier J, Kellum CD. Percutaneous Transluminal Angioplasty of the Renal Arteries. En: Castañeda-Zúñiga W, Murthy Tadavarthy S, eds. *Interventional Radiology*. Williams and Wilkins, 1988.

# Tratamiento intervencionista de la HTA vasculorrenal.

TRATAMIENTO QUIRÚRGICO

J.M.<sup>a</sup> Callejas Pérez

## Introducción

En 1836, Bright relacionó el riñón con la HTA. Pasados 100 años, Goldblatt demostró que la compresión o estenosis de la arteria renal provocaba atrofia del riñón e hipertensión<sup>1</sup>. Tras este descubrimiento, los pacientes con riñones de pequeño tamaño, mostrados en la pielografía, y afectados de hipertensión, se trataban, por lo general, con nefrectomía, sobre todo tras la comunicación del estudio de Butler en 1937, que descubría la desaparición de la hipertensión en los pacientes nefrectomizados por pielonefritis<sup>2</sup>. Dean y Foster<sup>3</sup> señalan su sorpresa ante la ausencia de comprobación anatomopatológica de lesiones estenosantes u obliterantes de la arteria renal, en los pacientes nefrectomizados durante aquellos años. En 1956, Smith revisó 576 casos de pacientes nefrectomizados por hipertensión y riñón pequeño; descubrió que sólo habían curado su hipertensión menos de un 30 %, por lo que llegó a la conclusión de que las nefrectomías se deben limitar a indicaciones urológicas, sin pretender curar la hipertensión, en todos los casos<sup>4</sup>.

En el campo de las relaciones etiopatogénicas entre las estenosis de la arteria renal y la hipertensión se producen varios hechos trascendentes: la descripción por

Leadbetter y Burkland, en 1938, del primer caso de displasia fibromuscular de arteria renal en un niño de 5 años, afecto de hipertensión severa y riñón pélvico malfuncionante<sup>5</sup>; la observación de Freeman, en 1954, de la normalización de la HTA tras una endarterectomía de la aorta y de las dos arterias renales estenosadas<sup>6</sup>, y la primera anastomosis esplenorrenal, para tratar una estenosis de la arteria renal izquierda, que Thompson y Swithwick habían practicado dos años antes<sup>7</sup>.

Después del descubrimiento de Dos Santos (aortografía) y Seldinger (arteriografía selectiva), el desarrollo de la angiografía favoreció, en las décadas de los cincuenta y sesenta, los diagnósticos de estenosis de arteria renal, practicándose casi siempre un tratamiento quirúrgico, como la endarterectomía o la técnica de *by-pass* o derivación, propuestas por Poutasse, DeBackey y Morris<sup>8-9</sup>, entre otros. Los resultados obtenidos con dichas técnicas en la normalización de la TA sólo fueron satisfactorios en la mitad de los casos. Se comprobó, así, que la relación entre la estenosis renal y la hipertensión no siempre era causal, y se produjo cierto desencanto en el concepto de hipertensión vasculorrenal. El desarrollo de técnicas diagnósticas complementarias, como el test de Howard<sup>10</sup>, o la deter-

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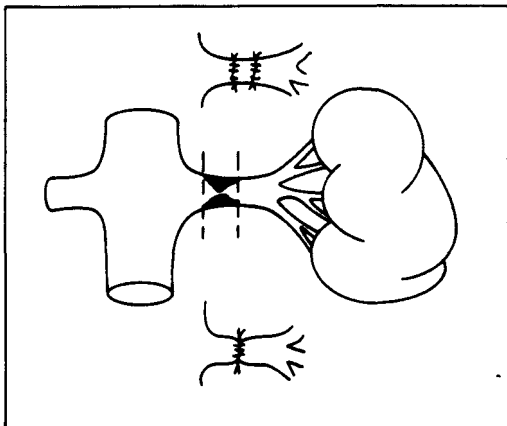


Fig. 24.1. Anastomosis terminoterminal

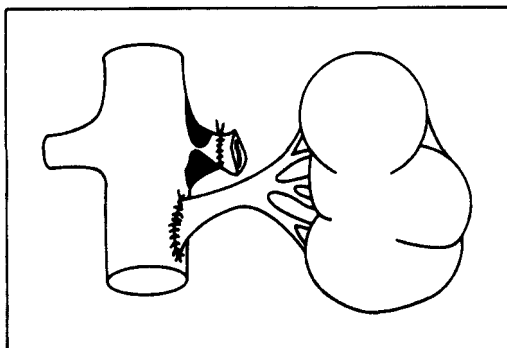


Fig. 24.2. Reimplantación de arteria renal en la aorta.

minación de reninas, ha llevado a la actitud actual: se admite de forma empírica que si una de dichas pruebas es positiva, la respuesta al tratamiento quirúrgico es satisfactoria en más del 90 % de los casos<sup>11</sup>.

Por último, muchos nefrólogos y cirujanos vasculares, en la actualidad, han asumido la gran importancia del concepto: las lesiones estenosantes de las arterias renales no deben tratarse sólo con la pretensión de normalizar la hipertensión, sino con la intención de evitar la progresión de la enfermedad ateromatosa, que lleva indefectiblemente a su obliteración, con la consiguiente anulación funcional del riñón.

Revisaremos a continuación el tratamiento de las lesiones estenosantes de la arteria renal, según las distintas opciones

desde el punto de vista de la técnica quirúrgica.

**Resección de la lesión estenosante y anastomosis terminoterminal**

Se podrá realizar en casos excepcionales de lesión circunscrita en una arteria renal larga. Las características anatomopatológicas de las lesiones ateromatosas displásicas imposibilitan casi siempre esta técnica. Por otra parte, las anastomosis terminotermiales de arterias de pequeño calibre, con una mala localización quirúrgica, producen una elevada incidencia de estenosis residuales (fig. 24.1).

**Reimplantación de la arteria renal en la aorta**

Está indicada en la lesión circunscrita del ostium, con arteria renal larga y movilizable. Tiene el inconveniente de que las lesiones ateromatosas suelen extenderse a la pared aórtica, imposibilitando y dificultando la técnica, si la arteria renal es de un calibre importante (fig. 24.2).

**Endarterectomía o extirpación de la placa ateromatosa estenosante**

Se utiliza también en las lesiones limitadas al ostium. En la práctica, se limita muchas veces la técnica porque las lesiones ateromatosas suelen ser difusas. No debe utilizarse en los casos de fibrodisplasia y lesiones calcificadas. Dado el tamaño de la arteria renal, obliga a practicar una angioplastia en parche, con vena safena o material protésico; esto dificulta y alarga la intervención (fig. 24.3).

Otros inconvenientes son el riesgo de embolización distal de material ateromatoso, y las complicaciones derivadas de la fragilidad de la pared arterial endarterectomizada (hemorragia, aneurismas).

Se utiliza, si es posible, en los casos de estenosis del ostium bilateral, pues permite practicar la revascularización, con una única incisión en la aorta extendida en las dos arterias renales (fig. 24.4). En los casos de lesiones obliterantes de la aorta, asociadas a estenosis del ostium de la arteria



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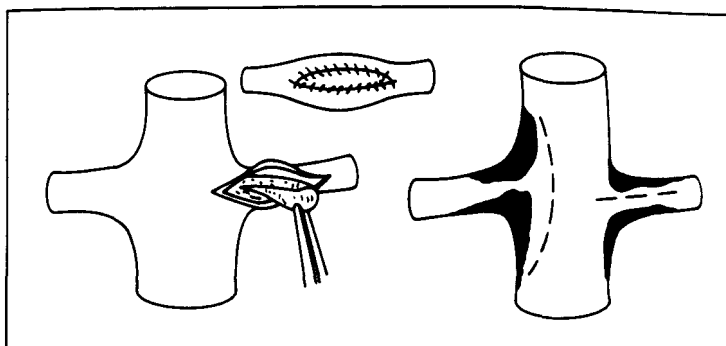


Fig. 24.3. Endarterectomía.

renal, es posible practicar endarterectomías renales transaórticas, con buenos resultados.

**By-pass aorticorrenal**

Es la técnica más utilizada por su simplicidad, ventajas hemodinámicas evidentes y versatilidad. Se han demostrado los mejores resultados, a corto y largo plazo. Según el estado de la pared aórtica y la topografía lesional, puede elegirse en cada caso el lugar más idóneo para la anastomosis. Si el calibre de la arteria renal es terminoterminal o terminolateral, las condiciones hemodinámicas son las mejores. No es preciso una disección extensa, y se puede utilizar material autógeno (vena safena o arteria hipogástrica) o prótesis de Dacron o PTFE. Pueden obtenerse los mejores resultados y un mínimo de complicaciones empleando la vena safena interna, aunque se han descrito casos de dilatación aneurismática tardía, así como posibles plicaturas. Si la vena safena interna no reúne las condiciones de calibre adecuado y estado normal de sus paredes, debe recurrirse a la implantación de una prótesis (fig. 24.5).

**By-pass iliacorrenal**

Es una técnica de recurso, utilizada en el caso de que la aorta no pueda emplearse como origen del *by-pass*, por la patología extrema de sus paredes. Implanta una prótesis de Dacron o de PTFE, con anastomosis terminolateral en la iliaca común, o terminoterminal o terminolateral en la arteria renal (fig. 24.6).

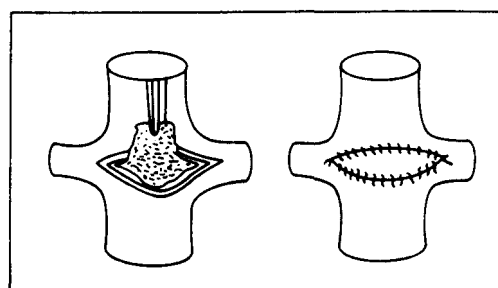


Fig. 24.4. Extirpación de una placa ateromatosa.

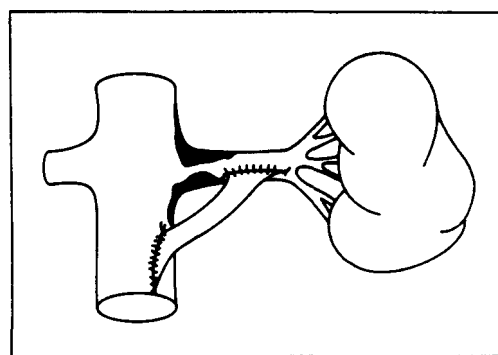


Fig. 24.5. By-pass aorticorrenal.

**By-pass prótesis aorticorrenal**

La frecuencia de asociación de lesiones aórticas y renales es muy elevada; surge la necesidad de abordar las dos localizaciones en un mismo acto quirúrgico. El tratamiento más utilizado en la actualidad, para tratar las lesiones estenosantes u ocluyentes aortoiliacas, es la prótesis bifurcada aorto-bifemoral. Si se aprecian estenosis renales

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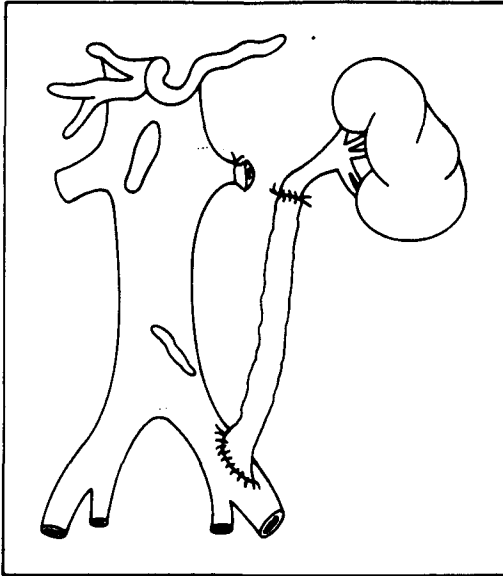


Fig. 24.6. By-pass iliacorrenal.

significativas, deben ser tratadas durante la misma intervención, aunque el paciente no sea hipertenso, con objeto de prevenir la progresión de la enfermedad, que provoca aparición de hipertensión e insuficiencia renal. Desde un punto de vista técnico, la solución más simple es la colocación de un *by-pass* desde la prótesis aórtica hasta la arteria renal estenosada, utilizando el mismo material protésico o la vena safena (fig. 24.7).

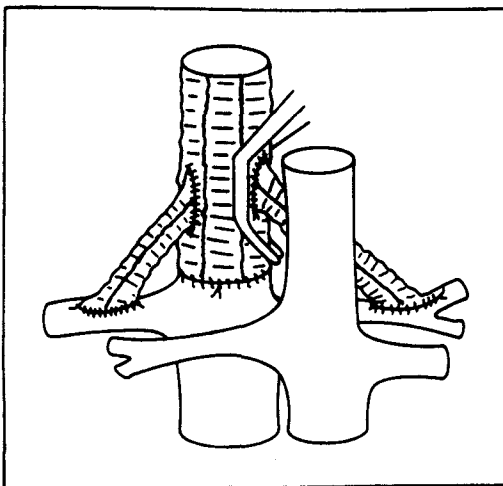


Fig. 24.7. By-pass prótesis aorticorrenal.

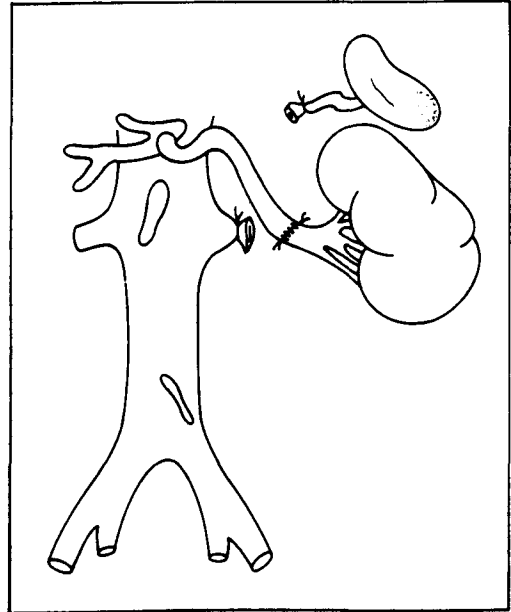


Fig. 24.8. Anastomosis esplenorrenal.

**Anastomosis esplenorrenal**

Revasculariza el riñón mediante sección de la arteria esplénica terminal y anastomosis con la arteria renal distal a la lesión estenosante, ya sea en disposición terminoterminal o terminolateral. No es preciso practicar esplenectomía; el bazo se nutre por los vasos cortos. Se utiliza, en especial, en la arteria renal izquierda. Su gran inconveniente son las dificultades técnicas de la movilización y preparación de la arteria esplénica, que en muchas ocasiones es de escaso calibre, corta o afecta de lesiones ateromatosas. Sus ventajas son el no precisar actuación sobre la aorta, utilización de prótesis, y poder utilizarse incluso, abordaje por lumbotomía (fig. 24.8).

**Revascularización a partir de arterias viscerales**

Debe distinguirse entre anastomosis directas, como la hepatorrenal o la gastroduodenorrenal, y derivaciones que precisan de la interposición de un injerto, como la anastomosis mesentericorrenal.

La anastomosis hepatorrenal o gastroduodenorrenal se utiliza en el lado derecho

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y tiene los mismos inconvenientes y limitaciones que las citadas para la anastomosis esplenorrenal, incrementada en la hepatorenal por el riesgo isquémico del hígado (fig. 24.9).

En la revascularización renal a partir de la arteria mesentérica debe interponerse un injerto o *by-pass* de vena safena o material protésico, por lo que la técnica pierde una de sus principales justificaciones.

**Autotrasplante renal y cirugía *ex situ***

Se trata de aplicar a la cirugía de la arteria renal los recursos técnicos adquiridos con el autotrasplante renal. Está indicado, sobre todo, en lesiones extensas ateromatosas o fibrodisplasias de la arteria renal y sus ramas terminales. Consiste en seccionar la arteria y vena renal y exteriorizar el riñón conservando intacto el uréter. Tras la perfusión renal con suero frío, se procede a la reparación que requieran las lesiones de la arteria renal y sus ramas, ya sea por sustitución de vena safena o de arteria hipogástrica. Una vez completada la revascularización renal, se comunican la arteria y vena renal en la hipogástrica o iliaca externa, según las técnicas habituales de autotrasplante renal.

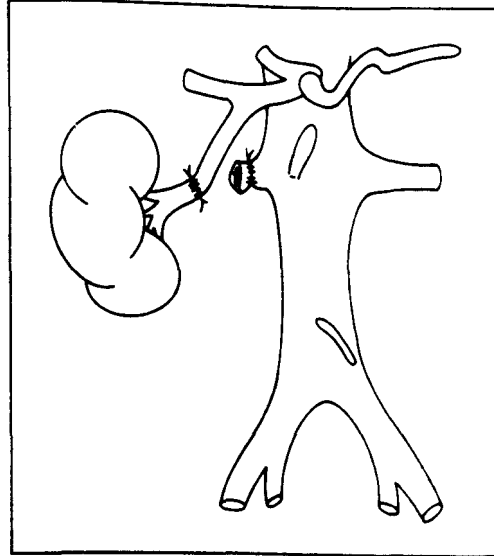


Fig. 24.9. Anastomosis hepatorenal.

Es una técnica compleja, que obliga a una extensa disección, anastomosis arterial y venosa y asiento ectópico del riñón; sólo se justifica como tratamiento de la hipertensión vasculorrenal en las lesiones de la arteria renal y sus ramas, tan extensas y complejas, que obliguen a una reparación *ex situ* de las mismas (fig. 24.10).

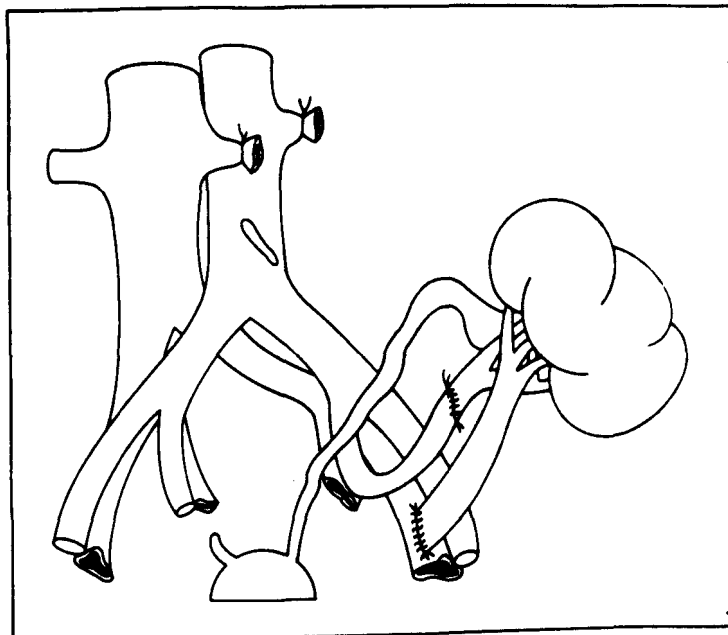


Fig. 24.10. Autotrasplante renal.

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Otro factor de indicación del autotrasplante es la imposibilidad de utilización quirúrgica de la aorta abdominal, por patología severa de sus paredes.

**Tratamiento de las estenosis secundarias al trasplante renal**

Una de las complicaciones que se desarrolla en el riñón trasplantado es la estenosis arterial, que suele aparecer entre los 6 meses y 2 años del trasplante. Afecta con frecuencia al segmento de la arteria renal del riñón trasplantado. La primera actitud terapéutica debe ser la angioplastia percutánea, y si ésta no es efectiva, la angioplastia en parche con vena safena o prótesis, la resección del segmento ateromatoso, anastomosis terminoterminal o técnicas más complejas como la reimplantación en la arteria iliaca, directa o mediante interposición de un segmento de vena safena.

**Estado actual del tratamiento quirúrgico**

En la actualidad, la mayoría de autores con experiencia admiten que la técnica que ha demostrado mejores resultados a corto y largo plazo, con menor número de complicaciones, es el *by-pass* aorticorrenal con vena safena autógena, tanto en estenosis ateromatosas como en fibrodisplasias. Se utiliza una prótesis de Dacron o PTFE sólo si la vena safena no reúne las condiciones mínimas exigidas.

La endarterectomía queda limitada a lesiones ateromatosas estrictamente ostiales, en especial bilaterales, y a los casos que deba practicarse junto a una resección aórtica por patología estenosante u ocluyente.

En pocas ocasiones son posibles técnicas simples, como la resección y anastomosis terminoterminal o la reimplantación de la arteria renal en la aorta.

El autotrasplante renal debe quedar limitado a casos con topografía lesional extensa de la arteria renal y sus ramas termina-

les, que exija su sustitución o reparación mediante cirugía *ex situ*.

La revascularización renal a partir de las ramas viscerales de la aorta (anastomosis esplenorrenal en el lado izquierdo y hepatorrenal, gastroduodenorrenal o mesentérico renal en el lado derecho) es considerada como técnica de recurso, y se emplea cuando fracasan las técnicas de elección o no es posible acceder a la aorta por su patología severa, o por intervenciones previas.

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**BIBLIOGRAFÍA**

1. Goldblatt H. Studies on experimental hypertension. *J Exp Med* 1934; 59: 347.
2. Butler AM. Chronic pyelonephritis and arterial hypertension. *J Clin Invest* 1973; 16: 889.
3. Dean RH et al. Surgery of the renal artery. Haimovici H, 1984.
4. Smith HW. Unilateral nephrectomy in hypertensive disease. *J Urol* 1956; 76: 685.
5. Leadbetter WF, Burkland CE. Hypertension in unilateral renal disease. *J Urol* 1938; 39: 611.
6. Freeman NE, Leeds FH et al. Thromboendarterectomy for hypertension due to renal artery occlusion. *JAMA* 1954; 156: 1.077.
7. Thompson JE, Swithwick RH. Human hypertension due to unilateral renal disease, with special recurrence to renal artery lesions. *Angiology* 1952; 3: 493.
8. Poutasse EG, Dustan HP. Arteriosclerosis and renal hypertension: Indications for aortography in hypertensive patients and results of surgical treatment obstructive lesions of renal artery. *JAMA* 1957; 165: 1.521.
9. DeBackey ME, Morris GC Jr et al. Lesions of the renal artery. Surgical technic and results. *Am J Surg* 1964; 107: 84.
10. Howard JE, Connor TB. Use of differential renal function studies in the diagnosis of renovascular hypertension. *Ann Surg* 1973; 177: 755.
11. Stamey TA, Nudelman IJ et al. Functional characteristics of renovascular hypertension. *Medicine* 1961; 40: 347.

## La Utilización de Catéteres Venosos Centrales en Oncohematología

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### INTRODUCCION

La utilización de catéteres que permitan un acceso a una vía venosa central es un requisito imprescindible en el tratamiento actual de los pacientes con enfermedades onco-hematológicas.

El tipo de intensidad del tratamiento quimioterápico, la administración de soporte hemoterápico y nutricional durante los prolongados períodos de pancitopenia y toxicidad gastrointestinal inducidos por el tratamiento, así como la necesidad de efectuar frecuentes determinaciones analíticas hacen inapropiado el uso de vías venosas periféricas en nuestros pacientes.

Los catéteres venosos centrales permanentes (CVCP), siendo una necesidad para el equipo médico y una comodidad para el paciente, no están exentos, sin embargo, de alteraciones en su colocación y funcionamiento que pueden convertirlos en una fuente de morbilidad y mortalidad importantes.

Una indicación correcta del tipo de CVCP necesario en cada caso, la implantación de dicho sistema por un equipo experto y un manejo adecuado del catéter contribuyen al funcionamiento adecuado del mismo y a la disminución de sus complicaciones.

### TIPOS DE CATETERES VENOSOS CENTRALES PERMANENTES

Básicamente, los catéteres actuales son modificaciones de los sistemas de alimentación parenteral a través de la vena subclavia desarrollados a partir de 1969.

Consisten en un tubo de Silastic, material escasamente trombogénico, radio-opaco y atóxico, cuya punta queda alojada en la entrada de la aurícula derecha; la porción extravenosa del catéter

recorre a continuación un trayecto subcutáneo en la región torácica anterior, finalizando en una porción distal, aquella que utilizamos para la infusión de medicación, que puede estar abocada al exterior (CVCP parcialmente implantable tipo Hickman) o bien permanecer subcutánea en forma de reservorio (CVCP totalmente implantable tipo Port-a-Cath).

Las ventajas de los catéteres totalmente implantables radican en un menor riesgo de infección, menos necesidades de mantenimiento y criterios estéticos. Estas ventajas potenciales se dan sobre todo en aquellos casos en que la frecuencia de utilización no es muy alta. Para los pacientes con alto requerimiento de uso del catéter y sobre todo en el contexto de tratamientos actuales con altas dosis de quimioterapia y trasplantes de Médula Osea, los catéteres parcialmente implantables ofrecen mayor utilidad.

### CUIDADOS DEL CATETER VENOSO CENTRAL PERMANENTE

Debido a la situación de inmunodepresión del paciente y a la vía abierta directamente al torrente circulatorio, los CVCP presentan un alto riesgo de infección.

La fuente de infección es tanto la piel del propio paciente como las manos del personal que maneja el sistema de infusión.

La limpieza de la zona de salida del catéter parcialmente implantable (tipo Hickman) se realizará diariamente; es muy importante el mantenimiento de una asepsia rigurosa principalmente en los 2-3 primeras semanas de la implantación del catéter, cuando la reacción fibrosa subcutánea alrededor del anillo de Dacron aún no se ha producido. A partir de entonces y en pacientes no neutropénicos es suficiente con cubrir el catéter con gasas estériles. Los CVCP totalmente implantables, al permanecer en la región subcutánea, no requieren cuidados especiales una

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vez curada la herida quirúrgica de su implantación.

Los CVCP deben ser heparinizados siempre después de cada uso. Se utiliza una solución de Heparina diluida a 100 U/ml. en la cantidad suficiente para rellenar todo su trayecto. En los catéteres parcialmente implantables es suficiente con 3-4 ml.; para los totalmente implantables hay que añadir además la cantidad necesaria para llenar el reservorio subcutáneo.

Cuando el catéter no se está utilizando, debe igualmente heparinizarse: diariamente en los casos de CVCP parcialmente implantables y cada 3-4 semanas en los totalmente implantables.

### **COMPLICACIONES DE LOS CVCP**

#### **1.- NO INFECCIOSAS**

**A.-MECANICAS**

**B.- TROMBOTICAS**

#### **2.- INFECCIOSAS**

**A.- PUNTO DE ENTRADA**

**B.- TUNEL SUBCUTANEO**

**C.- BACTERIEMIA**

#### *Complicaciones no infecciosas*

El dato de presentación más frecuente de las complicaciones mecánicas es la imposibilidad de extraer sangre o infundir líquidos a través de un catéter que había funcionado correctamente con anterioridad. Entre las complicaciones no infecciosas se distinguen dos grupos principales:

**Mecánicas:** Las fracturas de la porción externa del catéter son generalmente visibles y pueden ser reparadas sin necesidad de retirar el mismo. Las fracturas internas pueden ser detectadas en una Rx. de tórax como una discontinuidad en la línea radiopaca del catéter. A menudo, sin embargo, es necesaria la administración de medio de contraste. Las fracturas en el trayecto subcutáneo o intravascular requieren la retirada del catéter. El mal funcionamiento por emigración del catéter, bien subcutáneo bien intravascular, es más frecuente. La salida del catéter al espacio subcutáneo requiere la retirada del mismo. La emigración espontánea del catéter, dentro del espacio intravascular, hacia la vena yugular, subclavia contralateral o venas intercostales puede ocurrir tanto en el momento de su colocación como a lo largo de su funcionamiento. Necesitan de su reposición en el sitio correcto mediante control radiológico y generalmente no es necesaria su retirada total.

**Trombóticas:** Las complicaciones trombóticas de los CVCP ocasionan dos problemas clínicos diferentes:

1.- **Oclusión trombótica de la punta del catéter:** es la causa más frecuente de alteración en la función del mismo. La prevención, con heparinización rigurosa después de cada utilización del catéter, es la mejor solución del problema. El método de diagnóstico en la inyección de contraste dentro del catéter. Una vez se ha producido la obstrucción de la punta del catéter, puede intentarse su recanalización mediante la administración de Urokinasa ( de 1-3 ml. de una solución que contenga 5000 U./ml.) dejándola actuar en el interior del catéter durante 30 minutos. Esta maniobra puede ser repetida de nuevo si no se logra la recanalización inicial del catéter. En aquellos casos en que las maniobras anteriores no han tenido éxito se debe intentar la infusión de Urokinasa (1 vial de 250.000 U. administrado a razón de 40.000 U./hora durante 6 horas). Con la infusión de Urokinasa se recupera la función del 90% de los catéteres trombosados; para el 10% restante de los casos, es necesaria la retirada del catéter y su recambio por otro.

2.- **Trombosis de la vena subclavia:** la trombosis sintomática y el desarrollo de un síndrome de vena cava superior se produce en menos del 1% de los pacientes sometidos a trasplante de Médula Osea. El diagnóstico de sospecha se basa en la presencia de síntomas tales como dolor en brazo, hombro o cuello, prominencia de las venas del tórax y hombro, así como edemas en brazo y cuello. La confirmación de la trombosis se hará mediante una venografía de la extremidad afectada. La ecografía de la zona es muy específica para detectar trombosis establecida pero poco sensible. El tratamiento de la trombosis venosa establecida es la anticoagulación (Heparina 1000 U./hora) intentando prevenir la producción de TEP. También se ha utilizado el tratamiento trombolítico. Cuando la anticoagulación está contraindicada por la presencia de pancitopenia y mucositis intensas, se mantendrá el catéter en su lugar demorando la administración de heparina y fibrinolíticos hasta que la contraindicación se resuelva.

3.- **Trombosis no sintomática de la subclavia:** es una complicación descrita recientemente; el 25% de los pacientes en el trasplante autólogo de Médula Osea presentan una oclusión total de la subclavia y otro 34% desarrollan trombos parcialmente oclusivos. El significado clínico de estos trombos se desconoce, pero ante la posibilidad de

producción de TEP, esta circunstancia debe ser tenida en cuenta ante pacientes que presenten síntomas pulmonares.

### *Complicaciones infecciosas*

Constituyen el problema clínico más frecuente en el contexto de pacientes inmunodeprimidos portadores de catéteres venosos centrales permanentes. En relación con el catéter, las complicaciones infecciosas tienen dos formas de presentación:

- 1.- Infección del punto de salida y subcutánea.
- 2.- Bacteriemia.

Generalmente la primera, aunque no sea clínicamente visible, es causa de la segunda. Otra fuente importante de bacteriemias son las infecciones transmitidas por el personal que maneja el catéter. Por lo tanto, la prevención de la colonización e infección del sitio de salida del catéter y el lavado riguroso de las manos del personal que lo maneja son extremadamente importantes. La infección del punto de salida del catéter se define como la presencia de eritema, induración y/o dolor a la presión en un trayecto de 2 cm. Los mismos síntomas en un trayecto mayor de 2 cm. desde el punto de salida del catéter al exterior se clasifica como infección del túnel subcutáneo.

La determinación de que la bacteriemia en el paciente inmunodeprimido que no presenta alteraciones en el punto de salida o en el trayecto subcutáneo está producida por el CVCP requiere el aislamiento del mismo germen en hemocultivos tomados a través del catéter y de una vía periférica.

La infección del punto de salida del catéter responde bien al tratamiento antibiótico; no ocurre lo mismo en las infecciones del trayecto subcutáneo del catéter que, generalmente, obligan a la retirada del mismo.

En más del 50% de los casos, las bacteriemias relacionadas con el catéter están producidas por staphilococcus epidermis y pueden ser solucionadas con tratamiento antibiótico adecuado. Las bacteriemias por otros gérmenes como Cándida o Pseudomona no suelen responder a tratamiento antibiótico y no necesita, para su solución, la retirada del catéter.

### CONCLUSIONES

**A** pesar de la lista de agravios que supone el repaso anterior a las complicaciones producidas por los CVCP, el balance riesgos/beneficios es extremadamente favorable al uso de los mismos.

Sin ellos es imposible el tratamiento con dosis altas de quimioterapia y trasplante de Médula Osea. Con ellos aumenta la aceptación de dichos tratamientos por parte de los pacientes y, por lo tanto, su calidad de vida.

## Elección del Método

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Tres décadas de dedicación personal al estudio de la patología vascular, bien merecen una cierta reflexión sobre lo acontecido y algunas consideraciones sobre los espectaculares avances surgidos en el campo del diagnóstico, pronóstico y tratamiento de las enfermedades arteriales.

Remontándonos a 1964, por su influencia en los años siguientes, es útil recordar la introducción del concepto de que la arteriopatía obstructiva es siempre "segmentaria" y, por tanto, tratable localmente (De Bakey). Aquel planteamiento suponía hacer abstracción de que la enfermedad obstructiva arterial, especialmente la degenerativa, acostumbra a ser sólo una eclosión etio-patogénica y sindrómica de una topografía lesional concreta, cuando en realidad lo que sucede es que estamos ante un episodio esporádico, aunque no infrecuente, de una enfermedad sistémica: la arteriosclerosis.

Bajo esta doctrina impulsada por la Escuela de Houston, adquiere una gran relevancia el diagnóstico angiográfico de estas lesiones y las imágenes conseguidas, cada vez más precisas, constituyen desde aquel momento el único objetivo terapéutico, casi siempre de tipo quirúrgico revascularizador.

Aparecen y se prodigan a partir de entonces, las nuevas y espectaculares técnicas de cirugía arterial que han dado décadas de esplendor a nuestra especialidad y han permitido continuos avances en el campo de la implantación protésica, dando verdadero contenido y acreditación definitiva a una especialidad: la cirugía vascular.

Sin embargo, si profundizamos en esta realidad evidente y analizamos la multiplicidad de artículos aparecidos en la literatura de las décadas 60 y 70, repletas de series numerosas explicitadas o indicadas casi siempre sobre la base de una imagen angiográfica, olvidando no sólo el contexto genérico del paciente, las consecuencias hemodinámicas de dicha lesión y, sobretodo, que un gesto quirúrgico esmerado es sólo un remedio paliativo puntual de una manifestación local de la enfermedad sistémica. Puesto a exagerar la situación, me viene a la memoria la frase de Martorell "operan Vds. arteriografías y no enfermos" (!) y aquella otra "los injertos, cuando están indicados no van bien y cuando van bien, no están indicados"...

Pese a todo, hay que reconocer los repetidos esfuerzos para mejorar la calidad de la técnica quirúrgica y la búsqueda de un progresivo refinamiento en los métodos de anestesia y reanimación, que globalmente han permitido obtener resultados cada vez mejores e incrementar el número de pacientes que se benefician de estos avances.

En otro orden de ideas, es evidente que el seguimiento y evaluación de los resultados de la cirugía de medio y largo plazo, ha condicionado, en cierta forma, la acreditación y progresiva sofisticación de los llamados métodos no-invasivos de diagnóstico que, asociados a la arteriografía, nos han permitido aprender que no sólo es importante localizar una lesión, sino también conocer y medir las consecuencias hemodinámicas que de ellas se derivan, su importancia pronóstica y su influencia en el momento de sentar la indicación terapéutica.

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Y es en este abigarrado contexto, donde los avances en el conocimiento de la enfermedad arterial adquieren su mayoría de edad actual, se estandarizan las indicaciones y los procedimientos frente a una de las causas de mayor morbi-mortalidad conocida en el mundo: la arteriopatía oclusiva o ectasiante. De aquí se deriva un incremento de las líneas de investigación en el campo del diagnóstico y del tratamiento, aunque con los objetivos múltiples y no siempre bien coordinados. La tecnología diagnóstica con el Duplex color, la angiografía digital y la reciente angiografía por resonancia, permiten nuevos avances terapéuticos en el campo de la farmacología, la cirugía y hasta la "mal" llamada "cirugía sin bisturí"...

Y ¿cómo podríamos acotar la situación actual?

Los estudios epidemiológicos, un mejor conocimiento de la prevalencia de la enfermedad arterial y, muy especialmente, la mayor esperanza de vida que incrementa el número de pacientes de edad avanzada, ha producido un importante aumento de la demanda de asistencia por una población caracterizada por una patología compleja, portadora de un mayor número de factores de riesgo y una multifocalidad lesional característica de las arteriopatías degenerativas.

Al mismo tiempo, los constantes e imparable avances tecnológicos aportan nuevos métodos terapéuticos, con el consiguiente incremento del armamentario terapéutico, con posibilidades de indicaciones diversas, que muchas veces también dependen de factores no siempre son coincidentes con una correcta y estandarizada aplicación, en base a los resultados a medio y largo plazo. Es decir, la aplicación de un nuevo método terapéutico no siempre se indica con bases pragmáticamente establecidas y un conocimiento profundo de su eficacia, sino que algunas veces está condicionada por el afán irrefrenable de aplicar "lo más nuevo" o "lo más espectacular".

En la actualidad y con las posibilidades que tenemos a nuestro alcance, consideramos que la elección del método debería ir previamente condicionada por la esperanza de vida del

paciente, el entorno socio-económico en el que está inmerso, teniendo también en cuenta la alta morbi-mortalidad cardiológica y cerebrovascular de estos pacientes, cuya curva de supervivencia es imprescindible analizar.

Concretándonos a las técnicas de reconstrucción arterial, éstas tienen el objetivo de revascularizar tejidos u órganos isquémicos, conservar la función del miembro y evitar, dentro de lo posible, la progresión de la enfermedad.

En este contexto, la indicación terapéutica debe basarse en la edad biológica del paciente y las peculiaridades de su entorno socio-laboral, los factores de riesgo que presenta, la repercusión hemodinámica de la patología lesional, directamente relacionada con el grado evolutivo del síndrome isquémico que requiere la actitud terapéutica, la multifocalidad lesional características de las arteriopatías degenerativas, la valoración del riesgo quirúrgico y la habituación del equipo, fruto de un entrenamiento previo y una experiencia continuada, a una técnica determinada.

En la actualidad, las técnicas de revascularización que tenemos a nuestra disposición son las convencionales directas (endarterectomía, sustitución protésica y derivación tipo by-pass), las de recurso (by-pass extranatómicos) y las técnicas de revascularización "a distancia" más conocidas como *procedimientos percutáneos endoluminales* de revascularización (angioplastia con balón, con o sin stent, la aterectomía, la angioplastia láser, la trombectomía por aspiración y la aplicación locoregional de fibrinolíticos).

La aplicación de cada uno de estos procedimientos y la constatación de su eficacia, está permanentemente sujeta a controversia, destacando una supuesta competitividad entre la revascularización quirúrgica y la percutánea endoluminal, así como la aún vigente, entre endarterectomía y prótesis artificial.

Lo irracional de estas controversias, en parte influidas por un afán de protagonismo o por

los intereses comerciales de marketing, las firmas comerciales se olvidan frecuentemente de aspectos que me parecen importantes: por un lado, no se ha conseguido aún la prótesis vascular "ideal", pese al largo camino recorrido y el considerable dispendio económico de las muchas líneas de investigación dedicadas a este tema y a que no son nada desdeñables las complicaciones de las prótesis actuales (trombosis, infección y pseudoaneurismas).

Por otro lado, tampoco están plenamente acreditados, pese a la amplia experiencia existente, los beneficios a medio y largo plazo de la angioplastia con balón y, mucho menos, las otras variantes técnicas endoluminales. Y si a esto añadimos el alto costo de la sanidad y la crisis económica generalizada, parece difícil justificar la aplicación prematura y no siempre justificada de cada una de estas posibilidades técnicas.

Estas definiciones, seguramente precipitadas y con bases seguramente discutibles, no significan que los logros y avances conseguidos hasta la fecha en el manejo de la enfermedad arterial sean menospreciables, pues la realidad es que en la década de los 90, el cirujano, el radiólogo intervencionista, el hemodinamista y el cardiólogo disponen de métodos y tecnología lo suficientemente eficaces si son aplicados convenientemente en cada caso concreto y con la garantía suficiente de una baja morbi-mortalidad.

## Cirugía de la Hipertensión Reno-Vascular

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Es ampliamente conocido que el tratamiento de la hipertensión vásculo-renal ha cambiado profundamente en los últimos 15 años, debido a un mejor conocimiento de la historia natural de la patología lesional, a la mejora de los métodos diagnósticos (la mayoría no invasivos), a la eficaz asociación de drogas sinérgicas y también por los buenos resultados y baja morbi-mortalidad de la angioplastia endoluminal percutánea (ATP).

Además, el clínico ya diferencia la nefropatía parenquimatosa isquémica de la hipertensión arterial vásculo-renal, pues no siempre aquella cursa con cifras tensionales altas y, en cambio, el estado final de aquella es la insuficiencia renal irreversible (1, 2, 3). En la actualidad, los objetivos terapéuticos se dirigen más a salvar el complejo nefrónico-tubular que a conseguir una remisión de las cifras tensionales, es decir, mejorar o prevenir la insuficiencia renal, más que "curar" la hipertensión.

Aunque las drogas hipotensoras, casi siempre administradas en forma múltiple o asociada, son el tratamiento habitual en la mayoría de estos enfermos, la controversia actual radica en la disyuntiva entre las técnicas convencionales de revascularización quirúrgica o, por el contrario, utilizar la angioplastia percutánea con balón.

Diversos factores y no sólo la morbi-mortalidad quirúrgica, ni la menor agresividad y bajo costo de la ATP, condicionan la elección del método terapéutico. Entre ellos, hay que mencionar que aún es difícil determinar los mecanismos "causa-efecto", entre patología lesional causal e hipertensión arterial o nefropatía isquémica.

Los métodos diagnósticos tradicionales (pielograma minutado, hipertensión de corta evolución, determinación de reninas en venas renales, etc...), ha perdido actualidad, ya que el nefrograma post-captopril, la determinación de actividad renina periférica post-captopril (4) y, muy especialmente el Duplex ultrasónico de las arterias renales y del parénquima tienen una mayor fiabilidad (5). La velocidad sistólica media, obtenida en la a. renal con el Duplex, permite, en manos expertas, detectar una reducción del diámetro del vaso superior al 60%, lo cual generalmente se considera como una causa de hipertensión vásculo-renal. Los parámetros Dópler sobre el parénquima renal (una longitud inferior a 8 cm. y la ausencia de señal de flujo), son también sugestivos de una oclusión arterial. En las estenosis unilaterales, el Duplex permite cuantificar una disminución de la perfusión y en ausencia de patología intrínseca parenquimatosa, el cociente entre las velocidades sistólicas máximas registradas en ambos riñones, tiene una buena correlación con el porcentaje de función renal individual determinada mediante la renografía isotópica (6).

En cuanto al test del captopril (7), continúa la controversia sobre su fiabilidad real, pero hay evidencia de que la determinación de la actividad plasmática de reninas periféricas, después de una dosis única de Captopril, permite identificar pacientes con una alta probabilidad de que su hipertensión sea debida a una estenosis arterial superior al 60%. En definitiva, la utilización combinada del Duplex y la determinación de reninas periféricas post-captopril proporcionan resultados muy fiables en la identificación de una hipertensión vásculo-renal.

Desde el punto de vista terapéutico, la

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este tipo de pacientes, especialmente dirigido a conseguir cifras tensionales normales, sabemos que una postura más agresiva de revascularización renal, está no sólo justificada por la hipertensión, sino también y sobretodo, por el "salvamiento nefrónico" y preservación de la función renal (8).

Las indicaciones anatómicas de revascularización, ATP o cirugía convencional, están directamente ligadas al tipo de patología lesional de la arteria renal (9), hiperplasia fibromuscular, placa estenosante arteriosclerótica o aneurismas. La indicación de angioplastia en las lesiones fibrodifásicas es absoluta (10) cuando la hipertensión no se corrige mediante drogas hipotensoras. En cambio, cuando la lesión es arteriosclerótica, la indicación varía según la localización y extensión de la patología oclusiva. Están demostrados los buenos resultados de la ATP en las lesiones tronculares que respetan el ostium y las ramas distales, siendo en este caso ésta la postura terapéutica más correcta (10, 11). En cambio, cuando la estenosis es ostial yuxta-aórtica, la cirugía convencional ofrece, como mínimo, una mejor y más segura revascularización (12, 13, 14) y cuando las lesiones se sitúan en las ramas distales, la cirugía de banco "ex-situ" es la técnica de elección (15, 12).

Uno de los aspectos que perpetúa la actual controversia entre ambos procedimientos, es la disparidad en la forma de valorar los resultados, ya que en la mayoría de series publicadas, los parámetros no siempre son coincidentes y, desde luego, el simple criterio morfológico de una imagen post-angioplastia mostrando una patología lesional "curada", no significa que hayamos solucionado el problema clínico. En este sentido, es comúnmente aceptado (16) que un paciente está "curado" cuando la tensión arterial post-tratamiento no supera las cifras de 140/90 mmHg, se considera que ha "mejorado" si hay una reducción de la presión diastólica en 10-15 mmHg o se consiguen cifras tensionales normales con medicación asociada. Finalmente, el "fallo terapéutico" es evidente cuando ninguna de las posibilidades anteriormente mencionadas ha sido observada.

Por otro lado, no se debería ignorar que la ATP es una técnica relativamente invasiva, en la que no son infrecuentes las complicaciones (diseción intramural, oclusión trombótica, perforación, embolización distal renal y agravamiento de la insuficiencia renal, secundaria al contraste). En la serie de Ramsay (17) con 691 pacientes tratados con ATP, se reporta un 9,1% de complicaciones, siendo éstas más frecuentes en la corrección de las lesiones arterioscleróticas. Canzanello (11), sobre 100 pacientes, reporta 2 exitus después de la ATP, agravamiento de la insuficiencia renal en el 26% y complicaciones mecánicas de un 14%. En cualquier caso, hoy por hoy, la angioplastia con balón, es indudablemente la técnica de primera elección en la displasia fibromuscular (9) y en las lesiones segmentarias tronculares arterioscleróticas (8).

Otro aspecto a considerar, cuando se compara ATP y cirugía es la problemática del costo económico, pues no está demostrada una diferencia significativa a favor de la ATP. En el estudio de Weibull (18) se compara el coste terapéutico en 21 pacientes con ATP y 16 con cirugía, concluyéndose que este último fue un 12% más caro, pero en 32 de los 37 casos de la serie que tenían lesiones arterioscleróticas, la permeabilidad fue mejor en el grupo quirúrgico (100%) que en el de la ATP (62%), así como la regresión de las cifras tensionales (81% vs. 52%). En cualquier caso, serán necesarios estudios más numerosos para comprobar la realidad del supuesto bajo costo de la angioplastia con balón.

En relación a las técnicas convencionales de revascularización quirúrgica, los avances han sido considerables en la última década debido al perfeccionamiento de las técnicas anestésicas y de reanimación y a la proliferación imaginativa de nuevas variantes técnicas. Los clásicos by-pass aorto-renales con homoinjertos de hipogástrica o vena safena, siguen siendo el método habitual cuando la aorta abdominal no está afectada por la arteriosclerosis (19). Cuando la vena safena no es utilizable, el PTFE es la prótesis que ofrece mejores resultados (20) y la endarterectomía está ampliamente preconizada por autores del prestigio de Stoney (21) y Thevenet (22).

Cuando las lesiones están situadas en la ramas distales de la arteria renal, el tratamiento extracorpóreo con reconstrucción microquirúrgica vascular y auto-transplante, es mandatorio y hay grupos (23, 15) con una amplia experiencia en este campo y con unos resultados a medio y largo plazo excelentes.

En pacientes ancianos con arteriosclerosis severa de la aorta abdominal, el by-pass aorto-renal o la endarterectomía pueden no ser practicables y, entonces cobra actualidad la técnica de by-pass espleno-renal (24) para la arteria renal izquierda o el by-pass hepato-renal (25) para la derecha. Recientemente, en este tipo de pacientes con arteriosclerosis generalizada y con el común denominador de un nivel crítico de insuficiencia renal, el uso de la vía supraceláca desde la porción inferior de la aorta torácica para la revascularización simultánea de las arterias renales y de la aorta abdominal, está siendo utilizado con resultados excelentes a corto plazo (26,27).

La literatura reciente que hemos revisado nos permite afirmar que las técnicas quirúrgicas de revascularización convencional en la actualidad pueden ser llevadas a cabo con un alto índice de buenos resultados (19, 13, 14). En la displasia fibromuscular, la morbi-mortalidad es muy baja (oscilando entre 2,1% y 6,1% en los casos de lesiones arterioscleróticas). En cambio, los grupos que preconizan la revascularización renal bilateral simultánea (821), tienen una morbi-mortalidad más elevada, especialmente cuando se le asocia además una revascularización de la aorta abdominal. (28, 29). Siguiendo los estándares de los resultados previamente mencionados, la mayoría de series con experiencia quirúrgica, mencionan que el 60% de los pacientes se consideran curados, que el 40% han mejorado y el índice de fallo postoperatorio es inferior al 10% cuando la patología lesional es una fibrodisplasia muscular (13). En pacientes portadores de lesiones arterioscleróticas, pocos pacientes están considerados como "curados", pero la mayoría han "mejorado" en el postoperatorio, precisando dosis menor de medicación hipotensora. Una explicación de estas diferencias en los resultados en los pacientes con lesiones arterioscleróticas, sería

que la hipertensión vásculo-renal en pacientes ancianos a menudo coexiste con una hipertensión esencial. Un estudio reciente de Van Bockel (13) observa en una serie con un seguimiento medio de 9 años, que la hipertensión fue curada o mejorada en el 79% de los casos y una estabilización de la función renal en el postoperatorio, que oscila entre el 75 y el 89% de los casos.

Como conclusiones forzosamente esquemáticas de lo que antecede, podríamos enfatizar los siguientes:

1. La multiplicidad de las vías de circulación colateral compensatoria y el hecho de la duplicidad renal explican el que, durante mucho tiempo, lesiones de las arterias renales sigan asintomáticas.
2. El diagnóstico anatómico de dicha patología lesional no plantea, en la actualidad, problemas insolubles, ya que los métodos no invasivos, los funcionales y la angiografía digital ofrecen un abanico de posibilidades diagnósticas muy importante.
3. Pese a los avances de la angioplastia endoluminal y los resultados de la cirugía convencional, en los casos de lesiones complejas y enfermos poli-arteriales, hoy por hoy, son técnicas no siempre justificables para normalizar las cifras tensionales.
4. Otra cosa bien distinta, es su utilidad en la prevención de la insuficiencia renal, ya que en la actualidad, constituye la indicación fundamental de los métodos de revascularización renal. De esta forma, una terapéutica potencialmente curativa, ha pasado a ser una técnica preventiva, dependiendo de un mejor conocimiento de la historia natural de las enfermedades de la arteria renal y habiendo desplazado progresivamente la filosofía de las indicaciones de revascularización. Podríamos pues afirmar, sin excesivo rubor, que la terapia "preventiva" de la insuficiencia renal, hoy por hoy, está tan justificada como la de los aneurismas y las

estenosis de carótida.

5. En la actualidad, el tratamiento más recomendable de esta patología lesional, seguramente es la angioplastia endoluminal percutánea que parece tan eficaz como la cirugía y con menor morbi-mortalidad. Sin embargo, su eficacia en las lesiones muy distales u ostiales, es muy discutible y, seguramente, inferior a la cirugía convencional.

En definitiva, son imprescindibles estudios prospectivos randomizados para comparar ambas técnicas, cosa nada fácil, dada la diferencia del tipo, situación y extensión de la patología lesional del pedículo aorto-renal y una no siempre uniforme manifestación clínica de los pacientes.

#### Referencias.

1. Schreiber M.J. et al. The natural history of atherosclerotic and fibrous renal artery disease. *Urol Clin N. Am*, 1984; 11:383-392.
2. Harding M.B. et al. Renal artery stenosis: prevalence and associated risk factors in patients undergoing routine cardiac catheterization. *J. Am. Soc. Nephrol.*, 1992; 2: 1608-1616.
3. Tollefson D.E. et al. Natural history of atherosclerotic renal artery stenosis associated with aortic disease. *J. Vasc. Surg.* 1991; 14: 327-331.
4. Nally J.V. et al. State-of-the art review: Captopril renography-pathophysiological considerations and clinical observations. *Sem. Nucl. Med.* 1992; 22: 85-97.
5. Hansen K.J. et al. Renal duplex sonography:evaluation of clinical utility. *J. Vasc. Surg.*,1990; 12: 227-236.
6. Sos T.A. et al. Percutaneous transluminal renal angiography in renovascular hypertension due to atheroma or fibrous dysplasia. *N. Engl. J. Med.*, 1983; 309: 274-279.
7. Svetky L.P. et al. Prospective analysis of strategies for diagnosing renovascular hypertension. *Hypertension*, 1989; 14: 247-257.
8. Libertino J.A. et al. Renal revascularization to preserve and restore renal function. *J. Urol.*, 1992; 147: 1485-1487.
9. Cicuto K.P. et al. Renal artery stenosis: anatomic classification for percutaneous angioplasty. *A.J.R.*, 1981; 137: 599-601.
10. Sos T.A. Angioplasty for the treatment of azotemia and renovascular hypertension in atherosclerotic renal artery disease. *Circulation*, 1991; 83 (Suppl 1); 1: 162-166.
11. Canzanello V.J. et al. Percutaneous transluminal renal angioplasty in management of atherosclerotic renovascular hypertension: results in 100 patients. *Hypertension*, 1989; 13: 163-172.
12. Van Bockel J.H. et al. Extracorporeal renal artery reconstruction for renovascular hypertension. *J. Vasc. Surg.*, 1991; 13: 101-111.
13. Van Bockel J.H. et al. Surgical treatment of renovascular hypertension caused by arteriosclerosis: influence of preoperative factors on blood pressure control early and late after reconstructive surgery. *Surgery*, 1987; 101: 698-705.
14. Dean R.H. et al. Failed percutaneous transluminal renal angioplasty: experience with lesions requiring operative interventions. *J. Vasc. Surg.*, 1987; 6: 301-307.
15. Barral X. et al. Dysplasies des branches de l'artère rénale: résultats tardifs de la chirurgie ex vivo. *Ann. Chir. Vasc.*, 1992; 6: 225-231.
16. Novick A.C. et al. Trends in surgical revascularization for renal artery disease: ten years' experience. *JAMA*, 1987; 257: 498-501.
17. Ramsay L.E. et al. Blood pressure response to percutaneous transluminal angioplasty for renovascular hypertension. An overview for published series. *B.M.J.*, 1990; 300: 569-572.
18. Weibull H. et al. Clinical outcome and health care costs in renal revascularization: percutaneous transluminal renal angioplasty versus reconstructive surgery. *Br. J. Surg.*, 1991; 78: 620-624.
19. Mercier C. et al. Occlusive disease of the renal arteries and chronic renal failure: the limits of reconstructive surgery. *Ann. Vasc. Surg.*, 1990; 4: 166-170.
20. Cormier J.M. et al. Renal artery revascularization with polytetrafluoroethylene bypass graft. *Ann. Vasc. Surg.*, 1990; 4: 471-478.
21. Stoney R.J. et al. Renal endarterectomy through the transected aorta: a new technique for combined aortorenal atherosclerosis. *J. Vasc. Surg.*, 1989; 9: 224-233.

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- with polytetrafluoroethylene bypass graft. *Ann. Vasc. Surg.*, 1990; 4: 471-478.
21. Stoney R.J. et al. Renal endarterectomy through the transected aorta: a new technique for combined aortorenal atherosclerosis. *J. Vasc. Surg.*, 1989; 9: 224-233.
22. Thevenet A. L'endartériectomie. In Dubost C., Carpentier A. (eds.) *Actualités de Chirurgie Cardio-vasculaire (3<sup>a</sup> série)*. Paris, Masson, 1984, pp. 77-100.
23. Sicard G. et al. Renal autotransplantation: an alternative to standard renal revascularization procedures. *Surgery*, 1988; 104: 624-630.
24. Khauli R. et al. Splenorenal bypass in the treatment of renal artery stenosis: experience with 69 cases. *J. Vasc. Surg.*, 1985; 2: 547-551.
25. Chibaro E.A. et al. Use of hepatic circulation for renal revascularization. *Ann. Surg.*, 1984; 199: 406-411.
26. Novick A.C. Use of the thoracic aorta for renal revascularization. *J. Urol.*, 1990; 143: 77-79.
27. Barral X. et al. Revascularisation des membres inférieurs à partir de l'aorte supra-coeliaque. *Ann. chir. Vasc.*, 1986; 1: 30-35.
28. Piquet P. et al. Lésions athéroscléreuses aorto-rénales: résultats du traitement chirurgical simultané. *Ann. Chir. Vasc. Surg.*, 1988; 2: 319-325.
29. Branchereau A. et al. Chirurgie simultanée de l'aorte sous-rénale et des artères rénales. *Ann. Chir. Vasc.*, 1992; 6: 232-238.



## Current Status of Revascularization Techniques

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The past ten years have witnessed the development of innumerable new devices designed for the management of atherosclerotic/thrombotic vascular disease. Among these are laser angioplasty devices, atherectomy catheters, intravascular stents, intravascular ultrasound and angiography. In this presentation, we will discuss some of the most prominent devices, with particular emphasis on those that are currently commercially available.

### LASER ANGIOPLASTY

Laser angioplasty had a tremendous growth in early 1980s, with a subsequent fall to almost absent applications. Apparently, very few companies remain and most of them have shifted their research and development efforts toward the development of better laser systems (excimers), as well as a better recognition of plaque versus media through computerized manipulations. While indeed there will be a place for a laser device which removes plaque, undoubtedly a better safety margin with lack of damage to adjacent vascular wall and better long-term results will be required before lasers gain widespread application again.

### ATHERECTOMY DEVICES

Many atherectomy catheters have been developed, again with the aim of debunking the atherosclerotic vascular obstruction. Of the devices available, there are basically two main designs:

1.- Rotational Devices: Rotating at high speed, these devices help cross lesions while at the same time "micropulverize" the atherosclerotic plaque. The fragments produced by these devices typically embolize distally, but since their size has been said to be smaller than red cells, they do not cause any clinical complications.

2.- Cutting Devices: In these group are included those devices in which the plaque is cut and removed from the vessel. The hypothetical advantages of the cutting atherectomy devices over the rotational pulverizing devices is that the material is removed from the body preventing distal embolization of fragments which, in patients with poor vascular bed to begin with, could compromise the outcome. The widest experience with any of these devices has been reported with the Simpson Atherocath, with long term patencies at three years around 70%.

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Most of the other atherectomy devices available on the market require an associated transluminal angioplasty to complete the restoration of the vascular lumen to the original diameter. Longer-term results are required with most of the devices to assess whether the results obtained with these procedures are better than those obtained with plain old balloon angioplasty.

### **INTRAVASCULAR STENTS**

Intravascular stents were developed for the management of complications of transluminal angioplasty, such as abrupt reclosure as a consequence of extensive dissections, thrombosis, and/or elastic recoil. Recently, it has been said that primary stenting should be performed in order to improve the results of angioplasty. This has not been proven and long-term follow-ups are required before this can be advocated. Intravascular stents can be divided into two large groups: balloon-expandable stents and self-expandable stents. All available stents are made of different metals. The stent that has been most widely used in the United States is the Palmaz balloon expandable stent, with a correlative clinical success rate of 93% at ten months and 86% at twenty months.

Similar results have been reported with the Wallstent (Schneider, Inc.) in the iliac vessels and by European investigators. Other stents that will be discussed during the presentation include the Strecker, the Rabkin and the Cragg stents.

### **INTRAVASCULAR ULTRASOUND**

Intravascular ultrasound has been developed in response to the need to assess the atherosclerotic lesions from the inside, rather than from just evaluating the angiographic pictures. Intravascular ultrasound has the advantage over angiography in that it allows one to see through the wall, rather than to just see what is in the lumen of the vessel. In addition, intravascular ultrasound allows guidance of interventional procedures by assessing the amount and location of remaining tissue in the vascular wall. Its usefulness will be evaluated by the outcome of long-term results in patients in whom intravascular ultrasound was used in a randomized trial compared against the traditional angiographic methods.

## Pathophysiology of Angioplasty

P. MACKE CONSIGNY, Ph. D.<sup>1</sup>

### MECHANISMS OF BALLOON ANGIOPLASTY

The purpose of balloon angioplasty is to irreversibly enlarge the lumen of stenotic arteries. Several mechanisms have been proposed to explain angioplasty-induced arterial enlargement including (1) the compression of plaque (the "snowball" effect); (2) the extrusion of water from the plaque; (3) the embolization of plaque into the distal vasculature; and (4) the "irreversible" stretching of the arterial wall. Of these proposed mechanisms, "irreversible" stretching appears to be the dominant mechanism.

### ACUTE PATHOPHYSIOLOGIC EFFECTS OF ANGIOPLASTY

Angioplasty increases lumen diameter by stretching and cracking the atherosclerotic plaque while stretching the underlying arterial wall. This process induces a number of acute pathophysiologic changes in the arterial wall which are summarized below.

#### *Endothelial denudation*

The luminal surfaces of both normal and atherosclerotic arteries are covered with a monolayer of endothelial cells. These cells release a number of factors which participate in normal arterial function. The process of angioplasty destroys these cells and, in doing so, dramatically modifies arterial wall physiology. The physiologic changes that result from endothelial denudation include:

*Increased vasoconstriction and vasospasm* due to decreased capacity to produce the vasodilators prostacyclin (PGI<sub>2</sub>) and endothelium-derived

relaxing factor (EDRF).

*Increased platelet adhesion and aggregation* due to decreased capacity to produce the inhibitors of platelet adhesion/aggregation PGI<sub>2</sub> and EDRF.

*Increased luminal thrombosis* due to decreased capacity to produce the fibrinolytic agent tissue plasminogen activator (tPA).

*Increased arterial wall permeability* due to the loss of the semipermeable barrier provided by the endothelium and the tight junctions that join these cells.

#### *Cracking of Plaque*

The process of angioplasty stretches the arterial wall and in doing so often fractures the atherosclerotic plaque. The process of plaque fracture can result in:

*Stimulation of platelet adhesion/aggregation* by exposure of thrombogenic surfaces such as collagen.

*Thrombus formation* by release tissue factor (thromboplastin, Factor III) from within the plaque. Tissue factor activates the extrinsic pathway of thrombin generation resulting in further platelet activation and fibrin formation.

*Vasoconstriction/vasospasm* either at or down stream of the site of angioplasty subsequent to the release of thromboxane (TXA<sub>2</sub>) and serotonin (5-hydroxytryptamine, 5HT) from activated platelets.

#### *Lysis of medial smooth muscle cells*

Histologic and biochemical studies have demonstrated that the arterial stretching and compression that occurs during angioplasty results

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in the lysis of smooth muscle cells in the media. Destruction of these cells can have varying effects on the ability of the artery to undergo vasoconstriction.

*Diminished vasoconstriction / arterial paralysis.* If smooth muscle cell destruction is concentric, then the ability of the artery to constrict will most likely be diminished and may be abolished if the magnitude of injury is great enough.

*Enhanced vasoconstriction / vasospasm.* If smooth muscle cell destruction is eccentric, then the uninjured segment of artery can still constrict and may go into vasospasm. The likelihood that of vasospasm is enhanced by the absence of endothelium-derived vasodilators (PGI<sub>2</sub>, EDRF) and the presence of platelet-derived vasoconstrictors (TXA<sub>2</sub> and 5HT).

#### *Stretching of connective tissue*

In order for angioplasty to irreversibly increase lumen diameter, the mechanical properties of the artery must be altered. Histologic and physiologic studies of balloon dilated arteries have revealed the following changes:

*Stretching and tearing of internal elastic lamina.* Fracture of the internal elastic lamina may permit further expansion of the artery, since an intact internal elastic lamina may limit expansion of the artery.

*Increased arterial wall stiffness.* Physiologic studies have demonstrated that the balloon dilated artery is less compliant / more stiff than the undilated artery. This loss of compliance or loss of elastic recoil is the result of an overstretching of the connective tissue and smooth muscle cells within the arterial wall.

### **LONG-TERM PATHOPHYSIOLOGIC EFFECTS OF ANGIOPLASTY**

**A**lthough angioplasty produces an immediate increase in lumen diameter, it may also initiate an arterial repair process that can ultimately result in a reduction in lumen diameter, a phenomenon referred to as restenosis. The acute angioplasty-induced changes in the artery that may ultimately contribute the process of restenosis are summarized below.

#### *Platelet deposition and release of platelet-*

#### *derived growth factor (PDGF)*

Upon injury to the arterial wall, platelets adhere their alpha granules which contain platelet-derived growth factor (PDGF), a potent stimulator of the smooth muscle cell migration and proliferation that contributes to restenosis.

#### *Cell lysis and the release of basic fibroblast growth factor (bFGF)*

Angioplasty destroys endothelial cells, smooth muscle cells, and extracellular matrix. This destruction results in the release of a growth factor, basic fibroblast growth factor (bFGF), which is also a mitogen for smooth muscle cells. Experimental observations that support a role for bFGF in the early stages of restenosis include: (1) bFGF is released from injured and dying endothelial and smooth muscle cells and damaged extracellular matrix; (2) smooth muscle cells have high affinity bFGF receptors; (3) bFGF stimulates smooth muscle cell proliferation; (4) the infusion of bFGF into animals that have undergone balloon injury promotes intimal hyperplasia; and (5) the infusion of anti-bFGF antibodies partially inhibits medial smooth muscle proliferation after balloon injury.

#### *Release of PDGF by cells other than platelets*

Recent studies have demonstrated that PDGF is produced by many cells other than platelets including endothelial cells, smooth muscle cells and macrophages. There is considerable evidence that this non-platelet PDGF participates in the smooth muscle cell proliferation that is responsible restenosis. This evidence includes: (1) platelet deposition is limited to the first few days after angioplasty but smooth muscle cell migration and proliferation continues for several weeks after angioplasty; (2) macrophages, capable of producing PDGF are recruited to the artery by angioplasty-induced cell death; and (3) angioplasty stimulates the expression of the mRNAs for the A-chain and the B-chain of PDGF by cells within the dilated artery.

#### *Angioplasty-induced production of other growth factors*

Several growth factors are produced either on the luminal surface or within the balloon dilated artery. These factors include:

**Insulin growth factor-1 (IGF-1).** Several observations implicate IGF-1 in post-injury smooth muscle cell proliferation including: (a) IGF-1 is a progression factor which acts synergistically with PDGF to stimulate smooth muscle proliferation in vitro; (b) IGF-1 gene expression is increased by arterial injury and remains elevated during the period of intimal proliferation and endothelial regeneration.

**Thrombin.** Observations indicating thrombin in the restenosis process include: (a) thrombin is produced in high concentration on the luminal surface of injured arteries, (b) restenotic lesions often contain thrombus undergoing cellular reorganization, (c) thrombin receptors are present on smooth muscle cells, (d) thrombin stimulates the proliferation of serum-treated smooth muscle cells, (e) thrombin stimulates endothelial cells to produce PDGF which could promote further proliferation, and (f) hirudin, a thrombin inhibitor, reduces intimal proliferation after angioplasty of rabbit femoral arteries.

**Angiotensin.** Observations implicating angiotensin in restenosis include (a) angiotensin converting enzyme activity is present within the arterial wall, (b) the infusion of angiotensin promotes intimal hyperplasia, and (c) angiotensin converting enzyme inhibitors and angiotensin receptor antagonists retard intimal smooth muscle hyperplasia after balloon injury.

### **Matrix deposition**

Histologic examination of lesions obtained from both animals and man have revealed that a large component of the lesion is extracellular matrix. Furthermore, in vitro studies have documented that transforming growth factor-beta, which is produced within the artery in the later stages of restenosis, stimulates smooth muscle cells to secrete extracellular matrix proteins including collagen and fibronectin.

### **Re-endothelialization**

The luminal surfaces of normal and atherosclerotic arteries are covered by a monolayer of endothelial cells. These cells produce several factors (heparin, EDRF, prostacyclin) that inhibit smooth muscle cell migration and proliferation. Recent studies have demonstrated that re-endothelialization inhibits intimal hyperplasia. For example, intimal hyperplasia is not observed in areas of rat carotid arteries that re-endothelialized within 5 days after injury. In additional studies, an inverse linear

relationship has been observed between the magnitude of intimal hyperplasia and the magnitude of re-endothelialization.

### **Selected References**

1. Consigny P.M., Cragg A.H., Waller B.F. Pathophysiology of Vascular Intervention. In *Vascular Diseases: Surgical and Interventional Therapy*. Strandness E.D. Jr. and Van Breda A. (eds). New York: Churchill Livingstone Inc., pp. 31-53, 1993.
2. Consigny P.M. Platelet-derived Growth Factor Release after Angioplasty. In *Growth Factors and the Cardiovascular System*. Cummins P. (ed). Boston: Kluwer Academic Publishers, pp. 261-274, 1993.
3. Consigny P.M., Tulenko T.N., Nicosia R.F. Immediate and long term effects of angioplasty on the rabbit iliac artery. *Arteriosclerosis* 6: 265-276, 1986.
4. Consigny P.M. and Bilder G.E. Expression and release of smooth muscle cell mitogens in the arterial wall after balloon angioplasty. *J. Vascular Medicine Biology*. 4: 1-8, 1993.
5. Forrester J.S., Fishbein M., Helfant K. et al. Paradigm for restenosis based on cell biology. Clues for development of new preventive therapies.. *J. Am. Coll. Cardiol.* 1991; 79: 1374-1387.
6. Liu M.W., Roubin G.S., King S.B. Restenosis after coronary angioplasty: potential biological determinants and the role of intimal hyperplasia. *Circulation* 1989; 79: 1374-1387.
7. Libby P., Schwartz D., Brogi E., Tanaka H., Clinton S.K. A cascade model for restenosis. A special case of atherosclerosis progression. *Circulation* 1992; 86 (Suppl III): III-47 - III-52.
8. Haudenschild C.C. Pathobiology of restenosis after angioplasty. *Am. J. Med.* 1993; 94 (Suppl 4A): 40S-44S.

## Percutaneous Femoropopliteal Graft Placement

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Approximately two years ago, we began investigation of a technique for percutaneous placement of a femoropopliteal bypass graft (1). The purpose of this technique was to treat patients who had long segment femoropopliteal occlusive disease, which was not considered amenable to angioplasty because of the high restenosis rate of the technique in this situation. The technique involves placement of a conventional bypass graft into the superficial femoral artery. In this report, I described the initial application of this technique as well as a modification of our initial technique using a new dacron-covered, self-expandable stent graft.

### PROCEDURE

To date, this procedure has been reserved for patients with long segment femoropopliteal stenosis or occlusion who would otherwise be candidates for femoropopliteal bypass. Our protocol requires patients to have good iliac inflow, either native or corrected, as well as two vessel tibial runoff. In the United States, our experience has been limited to the use of PTFE graft material. We use conventional thin-walled PTFE (Gore and Associates, Flagstaff, Arizona).

To prepare the graft, we removed the spiral outer wrap on the graft material itself. This thins out the graft slightly and also allows us to dilate the graft by 1-2 mm if necessary once the graft has been placed. At present, our preferred graft stent combination involves suturing of two Palmaz stents, one on each end of the graft using interrupted 6-0 polypropylene sutures. The compressed stents are placed inside the graft with only a small amount of stent protruding outside of the graft. The graft is then mounted on a 6 mm x 10 cm angioplasty balloon and the stents are crimped in place.

After performance of conventional angiography and angioplasty via an antegrade femoral puncture, a long 12F sheath is placed across the segment of the femoral artery to be treated. A custom-made sheath has been created for this purpose (Daig Inc., Minneapolis, MN). The balloon graft assembly is

then loaded into a short segment of 12F sheath material. This is then back-loaded over a wire onto the hemostasis end of the 12F sheath. The balloon and stent graft combination are then advanced into the sheath and down to the site of intended deployment. The 12F sheath is then withdrawn and the balloon is inflated to deploy the stent and graft.

The major pitfall of this technique is that friction inside the sheath tends to force the crimped stents off of the balloon. We have been able to deploy these stent grafts through 11F sheaths but find that the 12F sheath minimizes friction and allows more reliable deployment on this fashion.

We have experimented with an alternative implantation technique for the PTFE material using Wallstents (Schneider Inc., Plymouth, MN). This technique requires a popliteal puncture and a through-and-through wire. While this technique has the advantage of allowing placement of a flexible stent which we find desirable in the femoral artery, it is cumbersome to place grafts using this technique and we no longer perform this technique.

We have recently begun investigation of a new stent graft which is presently undergoing clinical trials in Europe. This is a nitinol self-expandable stent covered by heparin impregnated dacron graft material. The graft material is ultrathin and allows deployment of the stent graft through a 9F sheath, (Mintec, Inc., La Ciotat, France) (2). With this system, the stent graft comes loaded in a 9F cartridge. This is back-loaded on a 9F sheath which is placed across the intended delivery site. The stent is advanced up to the point of delivery using a 9F pusher catheter. The sheath is then withdrawn to deploy the stent which is self-expanding. The stent graft can then be dilated using conventional angioplasty balloons after placement.

### RESULTS

Our initial technique using PTFE graft material has been applied to a small cohort of twelve patients. Technical success was achieved in eleven of twelve patients. All of these patients had grafts

placed in the femoropopliteal segment. Ten of twelve patients had favorable clinical responses with normalization of ankle/brachial indices and relief of symptoms. At mean follow up of 12 months, the primary patency is 50% (6/12). Secondary patency is 75% (9/12). Four patients had thrombosis of their graft during the follow up interval and underwent successful thrombolysis. One patient thrombosed twice and subsequently went on to have a femoropopliteal bypass.

In two patients, the cause of thrombosis was thought to be stenosis due to compression of a Palmaz stent placed in the adductor canal region. In one patient, intimal hyperplasia occurred at both ends of a Wallstent placed in the graft. One patient had thrombolysis of the graft without apparent stenosis.

In the month prior to this writing (December, 1993), nine implants of the dacron/nitinol graft have been performed in Europe, all technically successful. Follow up is not yet available. The use of the graft for treatment of aneurysmal disease, TIPS, and biliary obstruction is also underway. A randomized trial of the dacron/nitinol graft comparing it to conventional femoropopliteal bypass is also being organized in Europe. Application for a U.S. clinical trial is also pending.

## **DISCUSSION**

**D**uring the past ten years, multiple techniques have been investigated whose aim is to improve on the long-term success of balloon angioplasty. Balloon angioplasty is known to have suboptimal results in the treatment of long segment disease particularly in the infrainguinal vessels. In this situation, dilatation of lesions greater than 7 cm in length carries a high rate of restenosis.

Alternative therapy such as laser and atherectomy have been applied vigorously to treatment of this subset of patients with suboptimal results. Any technique which could improve on the high restenosis rate of long segment femoropopliteal revascularization would find application as an alternative to femoropopliteal bypass.

Late restenosis in femoral angioplasty is usually due to intimal hyperplasia and plaque progression. When long segments are treated, the chance of this occurring at some point along the segment increases thus making the restenosis rate of these lesions higher than is seen with short segment

disease. The technique of intraluminal graft placement is intuitively appealing since it may allow isolation of the diseased segment and prevent the ingrowth of intimal hyperplasia or plaque responsible for restenosis. In this situation, the graft may act as a barrier along its length to the development of intimal hyperplasia. The risk of restenosis may be reduced to the conventional surgical problem of anastomotic failure which is known to carry a lower restenosis rate than long segment balloon angioplasty.

In its present development, the technique of percutaneous vascular graft placement has many pitfalls. With rigid stents, there is a risk of compression when the stents are placed in the infrainguinal vessels. This occurred in two patients in our preliminary series. We also observed intimal hyperplasia at the end of one of the stent/grfts.

It is likely that these grafts will suffer from the same problems associated with surgical anastomoses, that is, turbulence, pannus formation and restenosis. The graft size must also be matched to the size of the inflow and outflow vessels. Development of tapered grafts and grafts of varying diameter may expand their use in this regard. As with surgical bypass grafting, the status of the inflow and outflow vessels will likely play a significant role in determining long-term patency of individual grafts.

Future applications of this technique which are now being investigated include the use of grafts in TIPS. Graft material on a stent may limit tissue ingrowth which is a common cause of restenosis after this procedure. The use of stents grafts for aneurysmal disease is now well underway. We anticipate percutaneous coronary graft placement in Europe by the time of this meeting.

This is an exciting area of interventional therapy at the present time. It is still in its infancy and it is not clear yet how great a role it will play. The author encourages the use of the judicious clinical trials to prevent a repetition of the "laser fiasco" of the 1980's.

## **References.**

1. Cragg A.H., Dake M.D. Percutaneous femoropopliteal graft placement. *JVIR* 1993; 4: 455-463.
2. Cragg A.H., DeJong S.C., Barnhart W.H., Landas S.K., Smith T.P. Nitinol intravascular stent: Results of preclinical evaluation. *Radiology* 1993; 189: 775-778.

## Cirugía Laparoscópica Abdominal

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Desde los reportes iniciales en 1987 de P. Mouret y F. Dubois, Francia, del éxito obtenido en la Colectomía Laparoscópica se ha producido un cambio dramático en la práctica de la cirugía abdominal muy en particular de la vesícula y vías biliares extra hepáticas. En Septiembre de 1992 en la reunión mundial de Consenso en Bethesda, Md., E.U.A., el acuerdo unánime fue de establecer que la Colectomía Laparoscópica es el procedimiento de elección, debido a sus grandes ventajas como son: reducción importante del dolor y sufrimiento post-operatorio, ausencia de íleo y complicaciones respiratorias, rápida reintegración a las actividades rutinarias, mejor resultado cosmético, etc.

Como consecuencia lógica y al mejorar la tecnología médica se han ampliado las indicaciones y a la fecha se practican en numerosos centros hospitalarios de varios países en el mundo, otros procedimientos tales como la apendicectomía, procedimientos de diagnóstico diverso, vagotomías, procedimientos antireflujo, y otros en ginecología, en urología con resultados que inicialmente parecen ser satisfactorios y comparables a los obtenidos en cirugía convencional aunque obviamente se requiere de un seguimiento cuidadoso y prolongado para confirmar estos resultados iniciales.

En otros casos como los de plastías inguinales, cirugía del colon, histerectomías, se practica este tipo de cirugía solamente en centros especializados con protocolos de trabajo estrictos para que en estudios prospectivos y bien controlados se pueda establecer qué grado de utilidad representa el abordaje por estos métodos mini-invasivos teniendo en cuenta siempre que lo más importante para los cirujanos es el bienestar, seguridad y confort del paciente.

De manera innegable en las evaluaciones objetivas que se llevan y se llevarán a cabo deberán incluirse relaciones costo-beneficio toda vez que en la actualidad este es un aspecto esencial en la práctica moderna de la medicina asistencial.

Durante esta breve presentación se tratará de ilustrar lo antes mencionado.

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## Percutaneous transgastric internal drainage, endoscopy and stenting of the pancreatic duct; a new technique

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**Key words:** Pancreaticoscopy, percutaneous; Pancreatic endoprosthesis, percutaneous; Pancreatic duct, catheter drainage; Interventional radiology, pancreatic duct

### Introduction

Percutaneous opacification of the pancreatic duct with ultrasound and computed tomography guidance [1,2] and percutaneous external ductal drainage [3] were described as alternatives to endoscopic cannulation when transpapillary access failed. We report the feasibility of percutaneous transgastric transpapillary drainage, percutaneous endoscopy and percutaneous stenting of the pancreatic duct.

### Case report

A 73-year-old male without medical history, except extrasystoles, presented with upper abdominal pain, with recent exacerbation. The patient lost 7 kg of weight. Physical examination was normal. Serum creatinine was 1.60 mg%, serum amylase 137 U/l, CEA 4.84 U/ml, and CA 19.9 800 U/ml. Abdominal ultrasound examination showed a well defined hypoechoic 2-cm mass in the head of the pancreas, the distal pancreatic duct was dilated at 8 mm. Computed tomography of the upper abdomen confirmed enlargement of the anteroposterior diameter of the pancreatic head at 4.5 cm, body and tail of the pancreas were atrophic. The pancreatic duct was dilated at 8 mm. The pancreatic mass was hypodense and its margins unclear, the superior mesenteric vein was compressed by the mass. Three percutaneous pancreatic biopsies with a 1.2 mm cutting needle were negative. Coeliac, su-

perior mesenteric and left gastric arteriography was performed: the superior mesenteric vein was encased at the level of the pancreatic mass. The common hepatic artery was slightly displaced cephalad. Two 5 mm pseudoaneurysms of the gastroduodenal artery were present. Percutaneous biopsy of the pancreatic mass with angiography guidance was negative. ERC showed normal bile ducts, cannulation of the pancreatic duct failed. ERP was attempted twice, and failed despite of a sphincterotomy.

A percutaneous diagnostic opacification of the pancreatic duct was planned. The dilated pancreatic duct was punctured after computed-tomography planning under fluoroscopic guidance with a 22G needle, by a transgastric approach. Opacification confirmed dilation of the pancreatic duct at the level of body and tail, and tumoral narrowing in the head. Anatomic distribution of the pancreatic ducts was normal. Percutaneous transgastric catheterization of the pancreatic duct was performed under local anesthesia and sedation, allowing further biopsies and pancreaticoscopy. The opacified pancreatic duct was punctured under fluoroscopic control with a F5 Teflon-sheathed needle at the level of the pancreatic body; a 0.035 torque-controlled guide wire was negotiated through the narrowed cephalic pancreatic duct and the papilla. A F8 biliary drainage catheter was placed percutaneously in the pancreatic duct, with the distal end in the duodenum and contrast medium was injected through the catheter (Fig. 1). The drainage catheter was exchanged for a 2.8 mm flexible endoscope (Surgitek, Racine, WI 53404) over a 0.035 guide wire after dilatation of the percutaneous and transgastric track with F9 semirigid dilators. Video-pancreaticoscopy was recorded. At the cephalic level,

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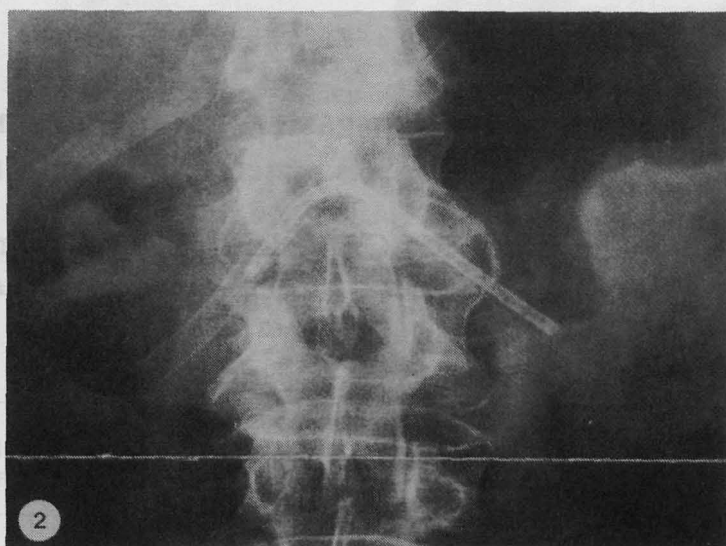
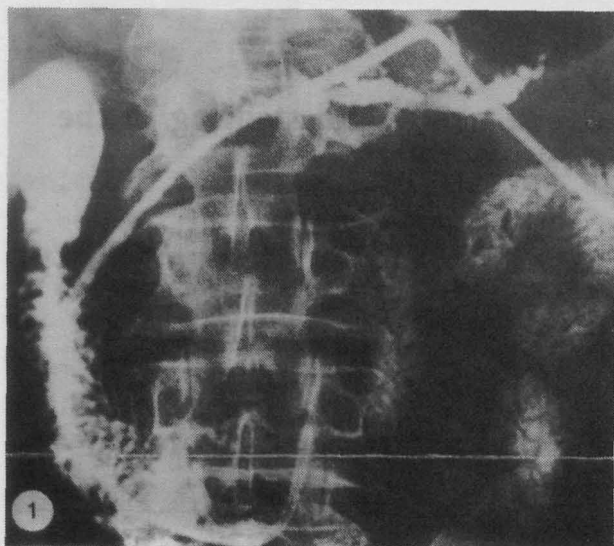


Fig. 1. A F8 drainage catheter with sideholes was inserted percutaneously through the stomach in the pancreatic duct at the level of the body of the pancreas; the distal extremity of the catheter was located in the duodenum. Opacification confirmed stricture of the pancreatic duct in the head and post-obstructive dilatation in the body and tail.

Fig. 2. Abdominal radiograph taken 3 days after percutaneous stenting of the pancreatic duct showed complete expansion of the expandable metal prosthesis (faintly visible) in the pancreatic head and good position of the F8 plastic prosthesis between the pancreatic duct and the stomach.

the mucosa of the pancreatic duct was hemorrhagic, without endoluminal tumor growth. Mural biopsy of the pancreatic duct was repeated, and was negative. A 4-cm-long expandable metal Wallstent prosthesis (Medinvent, Lausanne, Switzerland) with a diameter of 6 mm at full expansion was placed percutaneously in the pancreatic duct over a 0.035 guide wire. Duodenal protrusion of the prosthesis through the papilla was less than 5 mm, as confirmed by simultaneous transoral duodenoscopy. The pancreatic duct was inspected again after stent placement with the percutaneous endoscope. Perfect opening of the metal stent was confirmed. A pancreatic fistula at the puncture site was obviated by temporary percutaneous placement of a stent between the pancreatic duct and the stomach. A F8, 4-cm-long plastic prosthesis with sideholes was selected and inserted over the guide wire with a pusher. Neither gastric nor papillary bleeding was observed endoscopically after pulling out the endoscope from the stomach. The day after the procedure, clinical course of the patient was uneventful, leucocyte count was 10 300, serum amylase 181 UI/l, hemoglobine 11.8 g% and hematocrite 37.5%. Abdominal radiographs performed 3 days after the procedure showed complete opening of the expandable pancreatic metallic stent and good position of the gastropancreatic plastic stent (Fig. 2).

The general condition of the patient deteriorated progressively over the following weeks although the patient could leave the hospital. Death from pancreatic cancer occurred 2 months after percutaneous pan-

creatic duct stenting without exacerbation of abdominal pain during survival.

### Discussion

Among progress in therapeutic endoscopy, sphincterotomy of the pancreatic duct, nasopancreatic drainage or drug infusion, stone fragmentation and extraction, endoscopic inspection and stenting have evolved as alternatives to surgical procedures in the management of chronic pancreatitis [4-6] and malignant tumor of the papilla [7]. Percutaneous access to the pancreatic duct represents an alternative to endoscopy when technical difficulties or inadequate anatomical conditions prevent retrograde catheterization of the papilla. Percutaneous access to the pancreatic duct remained limited to diagnostic opacification in the literature [1-2], although percutaneous external drainage of the pancreatic duct was described in acute pancreatitis [3]. A prerequisite for percutaneous puncture is sufficient dilatation of the pancreatic duct allowing its identification with ultrasonography or computed tomography. When the pancreatic duct is dilated more than 3 to 5 mm, percutaneous puncture is successful in 90% [1]. A percutaneous transgastric approach is advisable, since a temporary prosthesis can be placed between the pancreatic duct and the stomach, obviating for a pancreatic leak after completion of a procedure and later endoscopic withdrawal of material. Computed tomography is particularly useful for planning the procedure:

the slice showing optimal contact between the pancreatic body and the posterior wall of the stomach should be selected for the percutaneous approach, as for internal gastric drainage of pancreatic pseudocysts [8]. Coiling of the guide wire in the stomach during percutaneous catheterization of the pancreatic duct is avoided by the use of a stiff guide wire. Placement of a working sheath through the walls of the stomach facilitates insertion of a drainage catheter, a prosthesis or an endoscope. No particular difficulty was noticed in the reported patient during antegrade catheterization with the guide wire of the strictured pancreatic duct. Intraductal guide wire manipulation must be carried out carefully as the pancreatic duct shows multiple side branches and its wall is less resistant than the bile ducts.

In the clinical case reported, transpapillary drainage, pancreaticoscopy and stenting of the pancreatic duct were performed by an exclusively percutaneous transgastric approach. The combined peroral-percutaneous technique commonly used in the biliary system [9], when endoscopic cannulation failed, can also be applied to the pancreatic duct, reducing the risk for pancreatic trauma. Potential complications of percutaneous transgastric puncture of the pancreatic duct are pneumoperitoneum, peritonitis, sepsis, pancreatitis, hemorrhage and vagal shock. Pancreatic bleeding and pancreatitis are minimized by a proper technique. Optimal cross-section localization of the dilated pancreatic duct reduces the number of attempts of percutaneous puncture. When the pancreas is atrophic, bleeding and pancreatitis are unlikely to occur during percutaneous puncture. Metallic expandable stents introduced percutaneously [10] or endoscopically [11] in the biliary system can also be inserted in the strictured pancreatic duct, provided no intraluminal tumor growth is present, which could eventually proliferate through the meshwork of the prosthetic wall and lead to occlusion. The flexible metallic stents expand to 6 mm and more, but need a F7 introducer catheter. Standard plastic prostheses can be used as an alternative choice. In the same way, mini-endoscopes [12] in regular use for cholangioscopy, ureteroscopy and angioscopy pass through a F9 introducer sheath. Clinical indication for the procedure in the reported patient was confirmation of pancreatic malignancy and relief from pain by treating ductal obstruction. Intraductal biopsy remained negative despite of endoscopic aid, but abdominal pain was not exacerbated until the patient de-

ceased. Although diffusion of this technique will be limited, we advocate in the future the percutaneous transgastric approach to the pancreatic duct in chronic pancreatitis, when endoscopic treatment has failed. Percutaneous balloon dilatation of the pancreatic sphincter, antegrade sphincterotomy, intraductal mechanical, ultrasonic or laser lithotripsy are other techniques which can be performed by the percutaneous route, in the same setting. Failure of the percutaneous procedure can be related to a non-dilated pancreatic duct, inappropriate anatomical conditions which make a transgastric approach impossible, and failure of antegrade catheterization of the papilla. A drawback of the percutaneous approach is the difficulty of inspection or catheterization of the whole pancreatic duct.

## References

- 1 Ohto M, Karasawa E, Kimura K, Saisho H, Ono T, Okuda K, Tsuchiya Y. Ultrasonically guided percutaneous contrast medium injection and aspiration biopsy using a real-time puncture transducer. *Radiology* 1980; 136: 171-176.
- 2 Haaga JR, Highman LM, Cooperman AV, Owens FJ. Percutaneous CT-guided pancreatography and pseudocystography. *AJR* 1979; 132: 829-830.
- 3 Gobien RP, Stanley JH, Anderson MC, Vujic I. Percutaneous drainage of pancreatic duct for treating acute pancreatitis *AJR* 1983; 141: 795-796.
- 4 Takekoshi T, Maruyama M, Sugiyama N et al. Retrograde cholangiopancreatography (in Japanese). *Gastroenterol. Endosc.* 1975; 17: 678.
- 5 Cremer M, Toussaint J, Dunham F. Endoscopic management of chronic pancreatitis. *Gastrointest. Endosc.* 1980; 26: 65-69.
- 6 Fuji T, Amano H, Harima K, Aibe T, Asagami K, Kinusaw K, Aryama S, Takemoto T. Pancreatic sphincterotomy and pancreatic endoprosthesis. *Endoscopy* 1985; 17: 69-72.
- 7 Huijbregtse K, Tytgat GNJ. Carcinoma of the ampulla of Vater: the endoscopic approach. *Endoscopy* 1988; 20: 223-226.
- 8 Hancke S, Henriksen FW. Percutaneous pancreatic cystogastrostomy guided by ultrasound scanning and gastroscopy. *Br. J. Surg.* 1985; 72: 916-917.
- 9 Kerlan RK Jr, Ring EJ, Pogany AC, Jeffrey RB Jr. Biliary endoprotheses: insertion using a combined peroral-transhepatic method. *Radiology* 1984; 150: 828-830.
- 10 Dick R, Gillams J, Dooley JS. Self-expandable stainless steel braided endoprosthesis for biliary strictures. *Radiology* 1988; 169 (P): 25.
- 11 Neuhaus H, Hagenmuller F, Classen M. Self-expanding biliary stents: preliminary clinical experience *Endoscopy* 1989; 21: 225-228.
- 12 Foerster ECh, Schneider MU, Stommer P, Runge U, Domschke W. Miniscopes in gastroenterological endoscopy: inspection of the gallbladder and the biliary and pancreatic duct systems in autopsy specimens. *Endoscopy* 1988; 20: 316-320.

## A.T.P. 15 Años de Experiencia Opinión del Angiólogo y Cirujano Vascular

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Cuando hace 15 años (1979) se introdujo en nuestro hospital la Angioplastia Transluminal Percutánea (ATP) como una nueva posibilidad terapéutica en el tratamiento de las lesiones segmentarias, estenosantes u obstructivas, de las arterias periféricas (1), nos hacíamos muchas preguntas sobre la bondad y el futuro de la nueva técnica. Sabíamos que, como cualquier nueva forma de tratamiento, debería demostrar, no sólo su eficacia inicial, sino también la duración de sus poco conocidos efectos endoarteriales (2), con el paso de los años. Por otra parte, los riesgos de su realización y los costes generados por tan novedosos métodos, deberían ser "competitivos", clínica y económicamente, en comparación con la terapia quirúrgica convencional (endarterectomía, prótesis), habitualmente utilizada para la corrección de dichas lesiones (3,4). Introducía a su vez, un cambio de mentalidad que afectaba tanto a radiólogos como a cirujanos, que bruscamente veían alterados sus roles en el manejo de estos pacientes. El Cirujano Vascular, tradicionalmente ejecutor de la terapia, pasaba a sentar la indicación clínica del procedimiento que sería realizado por el Angiorradiólogo, hasta ese momento encargado exclusivamente del aspecto diagnóstico.

Muchas de estas preguntas y consideraciones, muy poco contrastadas en la literatura mundial en aquel momento (5,7), explicaban los recelos, dudas, desconfianzas y suspicacias, que muchos radiólogos y cirujanos tenían en aquel momento, no solo en nuestro país, sino en todo el mundo. Se abrió un interesante y variado debate que en muchos aspectos, aún hoy permanece sin resolverse. Con pocas excepciones, en nuestro país, inicialmente, la técnica tuvo poca aceptación, e incluso un cierto grado de rechazo, más por desenfocados protagonismos de unos y otros que por su eficacia. En nuestro hospital, el nuevo método fue poco a poco asentándose y "cogiendo sitio" entre el no muy variado arsenal terapéutico que para el tratamiento de las arteriopatías periféricas

disponíamos en aquellos años. El entusiasmo y la amplitud de miras tanto del Cirujano Vascular (Dr. J.G. Pumarino) como del Angiorradiólogo (Dr. M. Maynar), que por entonces dirigían las respectivas unidades, fueron la principal razón para poder decir hoy, 15 años después, y con más de 1.000 procedimientos de ATP realizados en todos los sectores arteriales que: "La ATP es una variante terapéutica de *total validez* en el manejo de los pacientes portadores de arteriopatías periféricas".

Si bien esta aseveración previa es la conclusión más importante de nuestra experiencia en estos años (8,9), hay muchas circunstancias y variados aspectos consecuentes a este enfoque terapéutico que merecen ser comentados:

- Influencia de la ATP, en términos generales, en la existencia y manejo de los pacientes arteriopáticos.
- Modificaciones terapéuticas en función del sector arterial afectado.
- Influencia de la ATP en el seguimiento de estos enfermos. Resultados. Complicaciones.
- Cómo se han ido modificando las indicaciones con el paso del tiempo. Contraindicaciones, etc.

Estas y otras preguntas precisarían de varios tratados para su contestación minuciosa y pormenorizada, que se escapa tanto de la intención del autor, como de las posibilidades y finalidad del trabajo. Si podemos exponer de forma esquemática, algunos hechos significativos que reflejan y resumen nuestra actual forma de pensar al respecto. Ante todo, tres consideraciones, que no por obvias deja de ser importante recalcarlas y sobre todo, hacerlas reales en nuestro trabajo cotidiano:

- Imprescindible colaboración entre Angiorradiólogos y Cirujanos Vasculares.
- Ausencia de criterios competitivos entre las distintas alternativas terapéuticas.
- Indicaciones selectivas e *individualizadas*.

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Desde su introducción como método terapéutico en nuestro hospital, los parámetros asistenciales no han disminuido en términos cualitativos ni cuantitativos, aumentando tanto el número de intervenciones quirúrgicas de cirugía arterial, como el de amputaciones de extremidades por arteriopatías (Figura I).

Esta aparente paradoja, por otra parte constatada en auditorías de otros grupos europeos y americanos (10-16), tiene en nuestra opinión una doble explicación; por una parte, el progresivo aumento de la esperanza de vida con un crecimiento paulatino de población añosa, en la cual la prevalencia arteriopática es elevada (17); por otra parte, el mejor conocimiento de esta patología tanto por la clase médica como por la población que, día a día demanda mayores cotas de salud. Es decir, hay realmente más pacientes y se diagnostican con más precisión. Las previsiones demográficas, según las cuales una de cada cinco personas tendrá más de 65 años en el año 2.000, prevén en consecuencia, un continuado aumento de la demanda por arteriopatías periféricas en los próximos decenios (18, 19).

Si analizamos el tipo de tratamiento en relación con los diversos sectores afectados (Figuras II y III), observamos algunos hechos significativos en relación con la ATP. Así, en el sector aórtico, la influencia ha sido nula ya que su indicación es anecdótica; sí, en cambio, se ha modificado sustancialmente la técnica quirúrgica en este sector, con una casi total desaparición de la endarterectomía aórtica a favor de las prótesis arteriales. Cuando se asocian lesiones en las arterias renales a la patología aórtica y se considera indicada su corrección, en casos seleccionados hemos realizado ATP renal previa a la cirugía aórtica, disminuyendo los nada despreciables riesgos de la cirugía combinada aorto-renal; si bien el número de pacientes es pequeño, seis, los resultados han sido muy satisfactorios. El sector iliaco es el que más ha notado en su manejo terapéutico la aparición de la ATP, disminuyendo de forma notoria la cirugía aislada sobre el eje iliaco y aumentando significativamente las técnicas de dilatación percutánea.

Por otra parte, la dilatación ilíaca juega un papel de gran interés en las lesiones combinadas ilio/femoro/poplíteas, con mucho, las más frecuentemente halladas en nuestros pacientes (más del 70% de los casos), ya que nos permite tratar ambos sectores (ATP iliaca más cirugía femoropoplíteas o distal), con una notable

disminución del riesgo para el paciente; los resultados de este planteamiento asociativo son muy satisfactorios (20,21).

En el sector femoro-poplíteo las variaciones han sido mínimas desde la introducción de la ATP, tanto por la complejidad del sector como por los criterios restrictivos de indicación en función de la morfología lesional angiográfica. Si bien en ocasiones se combinan ATP y cirugía en grados muy avanzados de isquemia, los pésimos resultados obtenidos durante el comienzo de nuestra experiencia, han hecho que sus indicaciones sean actualmente muy seleccionadas.

En términos generales podemos afirmar que de una actividad muy posibilista inicial (8), hemos pasado a unas indicaciones mucho más seleccionadas y restringidas en función de las características clínicas y angiográficas de cada paciente (9). En su combinación con los fibrinolíticos, está desempeñando un interesante papel, en el tratamiento de las lesiones secundarias que se desarrollan después de la cirugía arterial revascularizadora (estenosis de bocas anastomóticas, trombosis protésicas, etc), con frecuencia reiterativas y complejas en su resolución duradera (22,23).

Mantenemos como única contraindicación absoluta sectorial, el eje carotídeo de destino encefálico (carótida común, carótida interna), por el riesgo incontrolable y potencialmente desastroso de embolismo cerebral que conlleva la manipulación de estas arterias cuando son portadoras de lesiones aterotrombóticas (24, 25).

Los resultados obtenidos con la ATP en el tratamiento de las arteriopatías periféricas, están en función de las características clínicas de los pacientes (edad, grado clínico, enfermedades asociadas, etc), y de la morfología y extensión de la lesión angiográfica. Es importante recordar el carácter progresivo y polifocal de la enfermedad causal, arterioesclerosis, que condiciona la historia natural de la enfermedad y hace muy complejo el control y seguimiento de estos pacientes, siendo muy difícil encontrar grupos homogéneos para su posible evaluación comparativa, dadas las numerosas variables existentes que caracterizan esta patología (26-29). Podemos esperar, sin embargo, buenos resultados a largo plazo (30, 31) cuando se trata de indicaciones *electivas* (estenosis ilíacas, estenosis u obstrucciones cortas femoropoplíteas, grados de isquemia poco evolucionados, ...), muy variables en las indicaciones *opcionales*, cada vez



más restrictivas e individualizadas (9, 32-36) y decepcionantes cuando se trata de indicaciones de *recurso*, para intentar salvar una extremidad como alternativa a la amputación (37, 38). Sí quiero señalar, que hemos comprobado en ciertos casos, permeabilidad de sectores arteriales tratados mediante ATP en seguimientos superiores a diez años, lo que en mi opinión, tiene un gran valor positivo respecto a la bondad de esta modalidad terapéutica.

Las complicaciones graves, empeoramiento del estado clínico previo (trombosis, embolismo, etc) o hemorragia, que precisan urgente resolución quirúrgica (39, 40), han disminuido muy significativamente con la progresiva experiencia de los Angiorradiólogos y las mejoras en el utillaje disponible, como se refleja en la Figura IV.

Si bien se prescribe de forma rutinaria y empírica, no tenemos constancia del posible efecto beneficioso de la medicación antiagregante/anticoagulante como terapia coadyuvante en el seguimiento a medio y largo plazo, siendo confusos y poco concluyentes los estudios existentes (41-44).

En resumen, la ATP ha supuesto, en nuestra experiencia asistencial de estos últimos quince años, una terapéutica de gran utilidad para nuestros pacientes que, con escasos riesgos, se han beneficiado de sus conocidas características de menor agresividad, mayor confort, menor tiempo de ingreso, etc, y en casos seleccionados, de excelentes resultados en el seguimiento evolutivo de su enfermedad.

## APÉNDICE

**E**s indudable que, desde su casual introducción hace 30 años, la idea o el planteamiento de tratar lesiones arteriales distantes a través de un catéter percutáneo, ha revolucionado muy positivamente la orientación terapéutica de estos pacientes. Por otra parte, paralelamente y gracias a ella, han surgido un gran número de técnicas que abren nuevos caminos y posibilitan resolver situaciones hasta ese momento impensables o de muy alto riesgo para la terapia quirúrgica convencional (45-52).

Estamos, y no quiero pecar de grandilocuente pero sí de sincero, ante una nueva era terapéutica en la que se va a modificar de forma espectacular el diagnóstico y tratamiento de un número de enfermedades. Ante esta situación hay que

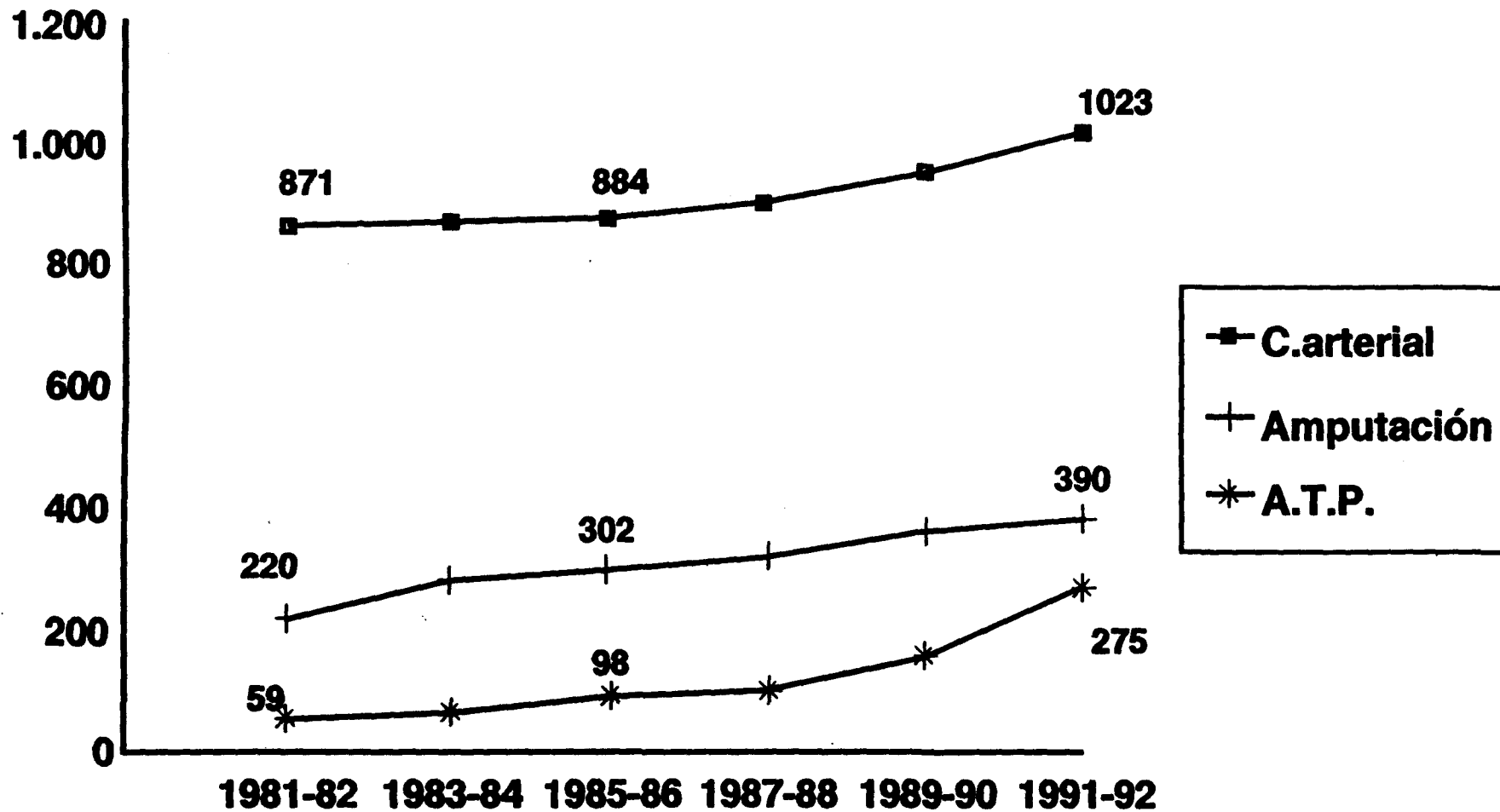
reaccionar con valentía y espíritu abierto, y por supuesto, con seriedad y prudencia.

Lo que, en mi opinión, no puede ni debe suceder, es que determinados sectores médicos por trasnochados personalismos u otros oscuros intereses, intenten frenar este apasionante futuro que hemos tenido la fortuna de conocer en sus comienzos. Obviamente hay que actuar con rigor y evitar los sensacionalismos que tan indeseables consecuencias tienen para pacientes y médicos. Sociedades científicas, comités de expertos, normativas o standards, comites de credenciales, etc, tienen la palabra y esperamos que la acción, para poder arbitrar y controlar la buena práctica de estos procedimientos (53-60).

En el concreto terreno que me compete, como Angiólogo y Cirujano Vascular, quiero manifestar mi opinión, por otra parte nunca ocultada, acerca de la nominada por algunos Terapéutica Percutánea. Hemos constatado, en estos 15 años, como los Angiorradiólogos de nuestro hospital han evolucionado en su quehacer diario desde la introducción de la ATP y otras variadas técnicas de terapia percutánea que abarcan un gran número de patologías. Desde hace varios años dedican su actividad profesional *exclusiva y permanentemente* (guardias), a la Angiorradiología (diagnóstica y terapéutica) y, en menor cuantía, a otras variantes de Radiología Intervencionista. Su volumen de trabajo es creciente y la demanda con frecuencia les desborda. Su habilidad en la realización de las técnicas se ve favorecida por la *especificidad* de su trabajo. El principal beneficiado de esta gran experiencia es el paciente y de forma indirecta sus acciones altamente especializadas, repercuten muy positivamente en los parámetros hospitalarios (ingresos, estancias, costes, etc). Demanda progresiva, volumen de pacientes creciente, *especificidad* de los métodos, exclusividad funcional, mejora de resultados,... ¿Qué más se necesita para poder afirmar que estamos ante una *nueva especialidad?* Es cierto que las connotaciones administrativas, académicas, asistenciales y docentes que esta afirmación conlleva, ha sido, es y será objeto de interminables polémicas y discusiones, pero no es menos cierto que cada vez se hace más patente la necesidad de dar respuesta a las nuevas preguntas que nos marca el proceso tecnológico, favoreciendo la investigación y creando nuevas unidades con capacidad docente de Terapéutica Percutánea, adecuadamente planificadas y dotadas, integradas en grupos hospitalarios de trabajo multidisciplinario, que sean capaces de forma

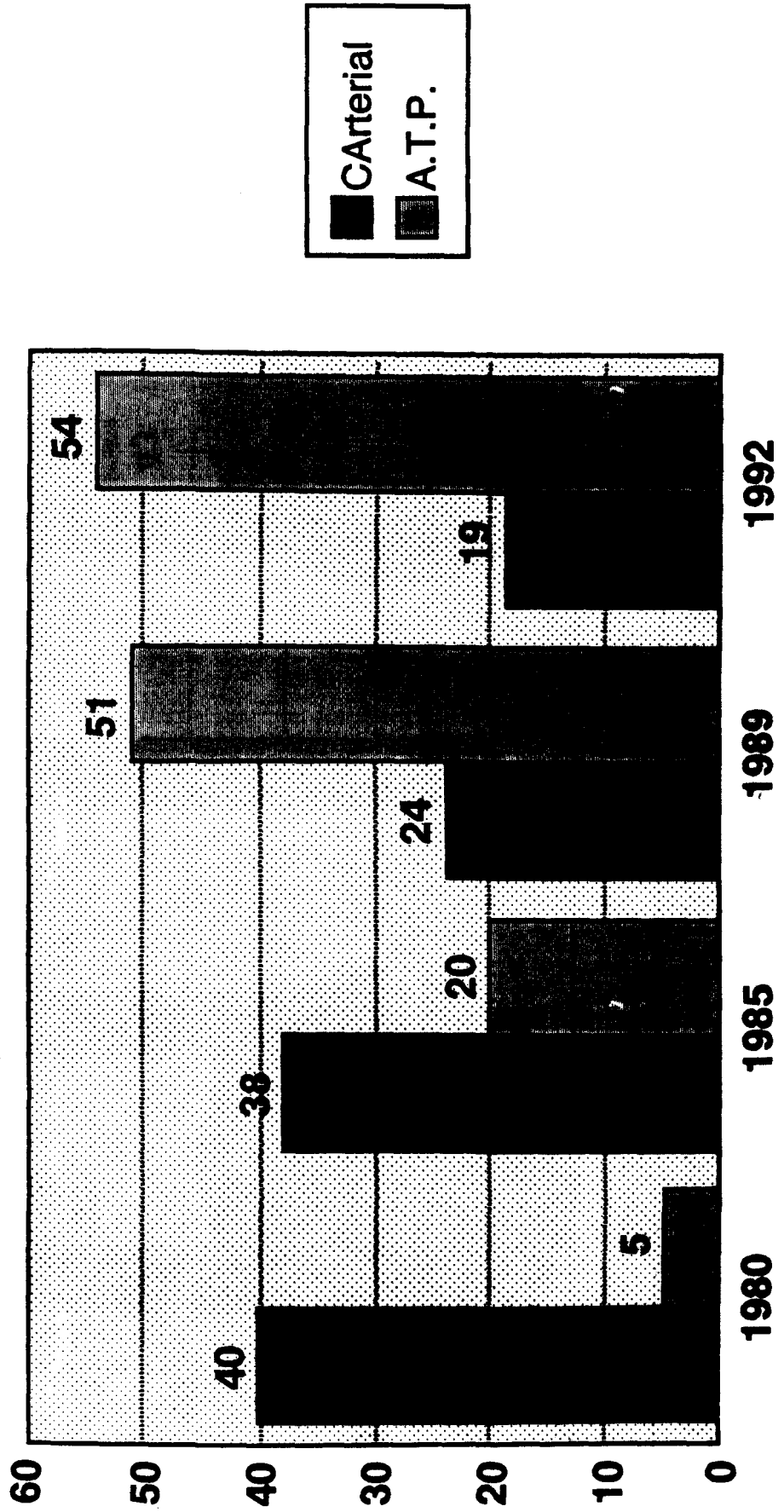
# FIGURA 1

## Relación Cirugía/A.T.P./Amputación

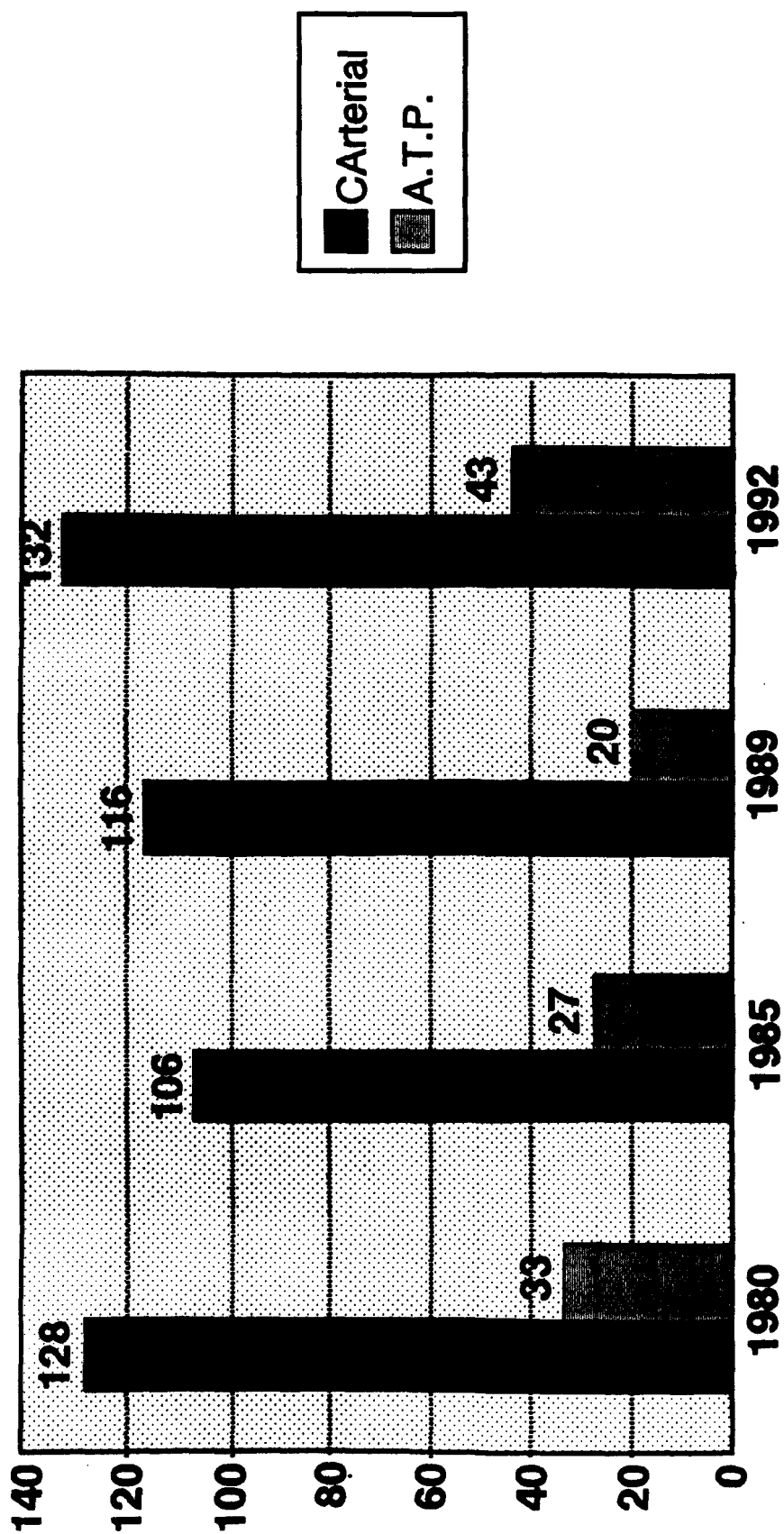


# FIGURA II

## SECTOR ILIACO (1980-93).RELACION CIRUGIA/A.T.P.



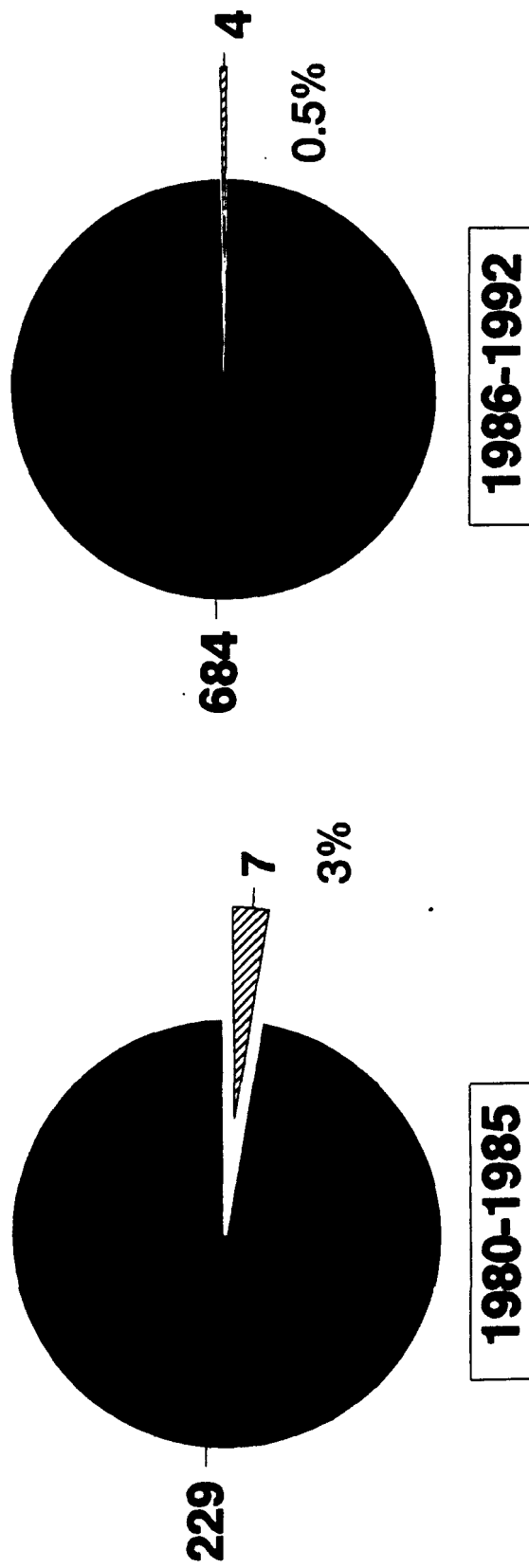
**FIGURA III**  
**SECTOR FEMORO-POPLITEO (1980-93).RELACION CIRUGIA/A.T.P.**





# FIGURA IV

## ASTURIAS 1980-93. COMPLICACIONES GRAVES DE A.T.P.



paulatina de comprometerse con las exigencias del progreso y no detenerse ante el permanente desafío que supone la mejora de la salud.

## Referencias

1. Maynar M., García-Pumarino J.L., Estevan J. ATP en MMII. Comunicación preliminar. Radiología, 1981; 23:73-82.
2. Castañeda W. et al. The mechanisms of balloon angioplasty. Radiology; 135: 565-570.
3. Douvilet P. The cost of underutilization of ATP for PAD. *New Engl J Med*, 1984; 95-102.
4. Kinnion et al. Cost incentives for peripheral angioplasty. *Am J Roentgenol*, 1985; 145:1241-1244.
5. Wierny et al. Long-term rests in 100 consecutive patients treated by ATP. Radiology, 1974; 543-548.
6. Dotter C. ATP. A long view. Radiology, 1980; 561-565.
7. Council report. PTA. Council of scientific area. JAMA, 1984; 764-768.
8. Estevan J. y otros. ATP. 5 años de experiencia. *Angiología*, 1986; 38: 59-72.
9. Estevan J. y otros. ATP. 10 años de experiencia. *Angiología*, 1991; 43: 55-65.
10. Tunis S. et al. The use of PTA, bypass surgery and amputation in the management of PAOD. *New Engl J Med*, 1991; 325: 556-562.
11. Earnshaw J. et al. The importance of vascular surgical audit to surgeons, patients and purchasers. *Eur J Vasc Surg*, 1992; 6: 540-544.
12. Jamadar D. et al. The impact of PTA on the workload of the vascular radiological and vascular surgical units of a district general hospital. *J Internat Radiol*, 1993; 7: 139-144.
13. Campbell W. et al. Auditing for vascular surgical audit. *Br J Surg*, 1987; 74: 98-100.
14. Holsworth J. Five year vascular audit from a district hospital. *Br J Surg*, 1991; 78: 601-606.
15. Michaels J. et al. Prospective audit of vascular surgical emergencies in a district hospital. *Br J Surg*, 1991; 78: 1271-1272.
16. Sayers D. et al. Changing trends in the management of lower limb ischaemia. *Br J Surg*, 1993; 80: 1269-1273.
17. Estevan J. y otros. Aspectos epidemiológicos de las arteriopatías periféricas. *Clin Cardiovasc*, 1993; 11: 39-47.
18. Fowkes F. et al. Edimburg artery study: prevalence of asymptomatic and symptomatic PAD in the general population. *Internat J Epidemiol*, 1991; 20: 384-392.
19. Estevan J. y otros. Aplicación de los datos epidemiológicos a la organización de las arteriopatías. *Angiología*, 1989; 41: 52-61.
20. Shah R. et al. Donor iliac angioplasty and crossover femoro-femoral by-pass. *Am J Surg*, 1992; 164: 295-298.
21. Brewster D. et al. Long-term results of combined iliac angioplasty and distal surgical revascularization. *Ann Surg*, 1989; 210: 324-331.
22. London N. et al. Interventional Radiology in the maintenance of infrainguinal vein graft patency. *Br J Surg*, 1993; 80: 187-193.
23. Beck A. et al. Angioplastie endoluminale, thrombolysis in situ et endoprotheses arterielle. Resultats immediates et a long term: a propos de 5.216 interventions. *Sem Hôp Paris*, 1991; 67: 1146-1158.
24. Mathias K. et al. PTA of proximal subclavian artery occlusions. *Cardiovasc Intervent Radiol*, 1993; 16: 214-218.
25. Kachel R. et al. PTA of supra-aortic arteries specially the internal carotid artery. *Neuroradiology*, 1991; 33: 191-194.
26. Kumpe D., Rutherford R. PTA for lower extremity ischaemia. In *Vascular Surgery*, Rutherford Ed. Saunders, 1989, pag 754-764.
27. Cristol R. Evaluation du risque de restenoses apres angioplaste peripherique. *J Malat Vasc* 1993; 18: 96-98.
28. Holm J. et al. Chronic lower limb ischaemia. A prospective randomized controlled study comparing the 1-year results of vascular surgery and PTA. *Eur J Vasc Surg*, 1991; 5: 517-522.
29. Creasy T. et al. Is PTA better than exercise for claudication? *Eur J Vasc Surg*, 1990; 4: 135-140.
30. Joffre F. et al. Resultates a long term des angioplasties femoro-poplitees. In Branchereau A ed. *CVN*, 1992; pag 39-51.
31. Insall R. Long-term results of double balloon PTA of the aorta and iliacs arteries. *Eur J Vasc Surg*, 1993; 7: 31-36.
32. Humiak M. et al. Risk and benefit of femoropopliteal PTA. *J Vasc Surg*, 1993; 17: 183-194.
33. Rooke T. et al. PTA in the lower extremities: a 5-year experience. *Mayo Clin Procc*, 1987; 62: 85-91.
34. Johnston K. Iliac arteries: reanalysis of results of balloon angioplasty. *Radiology*, 1993; 186: 207-212.
35. Thorninger B. Patency after iliac and femoropopliteal PTA. *Acta Radiol*, 1992; 33: 29-36.
36. Dalsing M. et al. Outcome predictor in selection of balloon angioplasty and surgery for PAOD. *Surgery*, 1991; 110: 636-644.
37. Matsi P. et al. Chronic critical lower limb ischaemia. Prospective study of angioplasty. *Radiology*, 1993; 188: 381-387.
38. Blair J. et al. PTA versus surgery for limb-threatening

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- ischaemia. *J Vasc Surg*, 1989; 9: 698-703.
39. Gardiner J. et al. Complications of PTA. *Radiology*, 1986; 159: 201-208.
40. Hasson J. et al. Complications of PTA. *Surgery*, 1990; 108: 748-753.
41. Antiplatelets trialists collaboration. Secondary prevention of vascular disease by prolonged antiplatelet treatment. *Br Med J*, 1988; 296: 320-332.
42. Ameli F. et al. ATP sin anticoagulación. *Ann Cir Vasc (ed. español)*, 1989; 3: 240-243.
43. Sarin S. et al. When do vascular surgeons prescribe antiplatelet therapy. *Eur J Vasc Surg*, 1993; 7: 6-13.
44. Fihn S. et al. Risk factors for complications of chronic anticoagulation. *Ann Intern Med*, 1993; 118: 511-520.
45. Ahn S. et al. Endovascular surgery for PAOD. A critical review. *Ann Surg*, 1992; 216: 3-17.
46. Strecker E. et al. Iliac and femoro-popliteal vascular occlusion disease treated with flexible tantalum stents. *Cardiovasc Intervent Radiol*, 1993; 16: 158-164.
47. Lammer J. et al. Pulsed excimerlaser versus continuous wave Nd: YAG laser versus conventional angioplasty of PAOD. *Lancet*, 1992; 340: 1183-1188.
48. Castañeda W. Percutaneous Thrombectomy. In "Syllabus" of International Course of Interventional Radiology, Las Palmas (Spain), 1992. 26-28 de Febrero, pag 131-139.
49. Kim et al. Peripheral directional atherectomy: 4 year experience. *Radiology*, 1992; 183: 773-777.
50. Laborde J. et al. Intraluminal by-pass of AAA: feasibility study. *Radiology*, 1992; 184: 185-190.
51. Muller C. et al. Effectiveness and safety of ultrasonic atherosclerotic plaque ablation. *Cardiovasc Intervent Radiol*, 1993; 16: 303-307.
52. Lierman D. et al. Endovascular radiotherapy of intimal hyperplasia in peripheral vessels. *Cardiovasc Intervent Radiol*, 1993 (supp) pag S 33.
53. Fernández Cruz L. Nuevos enfoques en la solución de problemas quirúrgicos: los nuevos cirujanos. En "Syllabus" del III Curso de Radiología Intervencionista. Dr M. Maynar, Las Palmas, 1992. pag 251-253.
54. Hollier L. Presidential address: influence of nonsurgical intervention on vascular surgical practice. *J Vasc Surg*, 1989; 9: 627-629.
55. String S. et al. Interventional procedures for the treatment of vascular disease: recommendations regarding quality assurance development, credentialing criteria and education. *J Vasc Surg*, 1989; 9: 736-739.
56. Rutherford R. Endovascular surgery: the new challenge. *J Vasc Surg*, 1989; 10: 208-210.
57. Porter J. Terapia intervencionista: un criterio alternativo. *Ann Cir Vasc (ed. español)*, 1991; 5: 101-102.
58. Frisch R. Técnicas endovasculares y Cirugía vascular. *Ann Cir Vasc (ed. español)*, 1991; 5: 1-3.
59. Levin D. et al. Training standards for physicians performing peripheral angioplasty and other percutaneous peripheral vascular interventions. *Circulation*, 1992; 86: 1348-1350.
60. White R. et al. Endovascular surgery credentialing and training for vascular surgeons. *J Vasc Surg*, 1993; 17: 1095-1102.

## Fístulas Arteriovenosas Durales Raquídeas con Drenaje Venoso Medular

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Individualizadas en 1.977 por Kendall y Logue (6), después precisadas por Merland en 1.980 (8). Actualmente, están bien caracterizadas. Corresponden a la mayor parte de los angiomas llamados retrorredulares de la clasificación de las malformaciones arteriovenosas medulares, de Djindjian (2). La fisiopatología de la afectación medular permite incriminar una hiperpresión venosa medular, cuyo mecanismo, no está aún claramente elucidado. El tratamiento, ya sea la obliteración de la fístula por vía endovascular o por vía quirúrgica, obtiene unos resultados funcionales que van a depender de la precocidad del tratamiento.

### DEFINICION

Se trata de una fístula arteriovenosa directa, microscópica, (150 micras de diámetro), sin interposición de nido angiomaso, entre una o varias arteriolas meníngeas y una vena, que se drena a contracorriente, de manera centrípeta hacia las venas perimedulares. La fístula, es extramedular, de muy bajo flujo, situada en el espesor de la duramadre. Se asocia constantemente a una perturbación del drenaje venoso medular normal, cuyas referencias normales hacia el espacio epidural-lumbosacro, no son visibles.

### ASPECTOS CLINICOS

Se observa un neto predominio masculino (7 hombres por cada mujer), en la quinta década de la vida (edad media 58 años). El cuadro clínico que presentan, es el de una mielopatía de nivel dorso-lumbar, de constitución progresiva, de 6 meses a 2 años, agravándose regularmente hasta la paraplejia, en de 2 a 4 años. El comienzo de la enfermedad es

raramente brutal, frecuentemente insidioso, y se manifiesta por una afectación, sensitiva, motora o esfinteriana, frecuentemente aislada al principio. Los problemas sensitivos, son los más frecuentes, afectando a los miembros inferiores de forma asimétrica: radiculalgia de tipo L<sub>4</sub>, L<sub>5</sub> o S<sub>1</sub>; o parestesia de tipo de quemazón o de hormigueos. La afectación puede ser únicamente motora, de tipo de claudicación medular. Excepcionalmente, el comienzo puede estar marcado únicamente por problemas esfinterianos y/o genitales. Independientemente del modo del comienzo, la evolución, se hace en varios meses hacia una afectación asociada sensitivo-motora y esfinteriana, de diversos grados, provocando un cuadro de radiculomielopatía dorso-lumbar de gravedad variable, que está esencialmente en función de la duración de la evolución antes del tratamiento. La evolución, es generalmente progresiva; sin embargo, una agravación brutal más o menos regresiva, aparece a veces. Esta agravación, puede ser espontánea o provocada, durante circunstancias particulares principalmente posturales (posición sentada durante mucho tiempo) o excepcionalmente, después de una mielopatía se manifiesta, según los casos, por diferentes cuadros neurológicos, que son, por orden de frecuencia decreciente: síndrome del cono medular; síndrome de cola de caballo; paraparesia espasmódica aislada. El nivel superior de la afectación sensitiva, aparece excepcionalmente por encima de D<sub>10</sub>; el sufrimiento medular es esencialmente dorso-lumbar, cualquiera que sea el nivel de la fístula arteriovenosa.

### EXAMENES COMPLEMENTARIOS

El estudio del L.C.R., muestra en general un aumento moderado de la proteinorraquia entre 0,60 y 1,50 gr/l sin reacción celular asociada.

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La Resonancia Magnética, debe ser la prueba diagnóstica a realizar en primer lugar, utilizando la secuencia ponderada en T<sub>2</sub>, en los planos, sagital y transversal, donde suele apreciarse una hipers señal a nivel de la médula dorsal baja y del cono medular. Esta hipers señal, puede ser visible en incidencia sagital, pero es frecuentemente confirmada por los cortes axiliares transversos. Está en relación con el sufrimiento isquémico crónico de origen venoso del cono medular (4). Es menos frecuente, observar una importante dilatación vascular.

Si la Resonancia Magnética es negativa, es conveniente realizar, como antiguamente, una mielografía dorso-lumbar con medios de contraste hidrosolubles (10), que muestra casi constantemente, la presencia de improntas vasculares anormales, muy evocadoras de una malformación arteriovenosa medular. Es muy importante realizar una técnica perfecta con tomografía, en cortes finos (A-P y laterales). La imagen típica es la de una dilatación de las venas retromedulares, tortuosas, extendidas sobre toda la médula dorso-lumbar hasta el nivel cervical. A veces, las imágenes son menos evidentes: es decir, presencia de una dilatación de grado variable, visible únicamente en tomografía (particularmente de las venas perimedulares); dilatación visible únicamente sobre 2 ó 3 segmentos raquídeos, siempre patológica en la región dorsal media y alta; dilatación de las venas radicales lumbares. Otras anomalías que pueden estar asociadas: presencia de una canal lumbar estrecho, protrusiones discales.

La Angiografía Medular, está formalmente indicada ante un cuadro de mielopatía progresiva, con Resonancia Magnética o mielografía revelando, imágenes vasculares francamente anormales o simplemente dudosas o ante la existencia de una hipers señal del cono medular. Dicha angiografía, identifica y localiza la fístula arteriovenosa, el lugar exacto de la comunicación arteriovenosa en el canal y precisa las particularidades del drenaje venoso. Permite localizar al arteria espinal anterior y sus relaciones eventuales con la arteria aferente de la fístula.

Desde el punto de vista técnico, es importante comenzar por una arteriografía bifemoral retrógrada simultánea (9) que nos permite generalmente apreciar la arteria de Adamkiewicz y todas las arterias dorso-lumbo-sacras, simplificando considerablemente la exploración, que es delicada en pacientes de edad, en los que aparece este tipo de patología.

## RESULTADOS

La fístula arteriovenosa, es frecuentemente única aunque a veces, es, múltiple, puede estar localizada a cualquier nivel del canal raquídeo dorsal, lumbar o sacro, con una predilección particular por las regiones dorsal media (D<sub>3</sub> a D<sub>7</sub>) y dorso-lumbar (D<sub>12</sub> a L<sub>3</sub>). La localización menos frecuente y quizá excepcional es a nivel cervical. (Nosotros, hemos encontrado una fístula dural cervical a nivel C<sub>6</sub>, en un hombre con un paraparesia progresiva que se convirtió bruscamente en paraplejia después de una mielografía).

La arteria aferente, puede ser una intercostal, una arteria lumbar, una arteria sacra (5) y en los casos excepcionales, una arteria cervical. La comunicación arteriovenosa, situada en la duramadre, es directa, microscópica, de bajo flujo, entre una o varias arteriolas meníngeas de fino calibre y una única vena dilatada de drenaje.

La vena eferente, presenta una trayecto ascendente intradural hacia la médula, donde se reúne con el sistema venoso perimedular, que está dilatado, anterior o posterior. La circulación venosa, presenta una dirección, prácticamente siempre ascendente hacia las venas de la base del cráneo, que parecen la principal vía de salida, es frecuentemente muy lenta (la totalidad en la red venosa medular no es opacificada hasta los 40 o 60 segundos), las eferencias epidurales normales dorso-lumbo-sacras, no son opacificadas, (salvo raras excepciones, cuando el drenaje es descendente).

El enlentecimiento del sistema venoso medular, está igualmente demostrado por el estudio del retorno venoso de la arteria espinal anterior, que normalmente es visible entre 15 a 20 segundos (7) siendo invisible en caso de las fístulas incluso después de los 50 a 60 segundos.

Al final del estudio angiográfico, la decisión terapéutica, puede ser tomada, teniendo en cuenta el nivel del origen de la arteria de Adamkiewicz, en relación al de la fístula, así como las posibilidades de un cateterismo hiperselectivo estable, para poder realizar una obliteración endovascular de la comunicación arteriovenosa.

## FISIOPATOLOGIA

El sufrimiento medular, responsable de un cuadro de mielopatía progresiva de estos enfermos, es

secundario a una isquemia medular crónica por hiperpresión venosa medular. La hiperpresión venosa reduce el gradiente de presión arteriovenosa intramedular, perturbando la irrigación normal de la médula con constitución de un edema intersticial medular y de dilataciones venosas intramedulares que llevan muy progresivamente a una necrosis isquémica definitiva. El mecanismo de hiperpresión venosa, no ha sido completamente elucidado: existe una fístula arteriovenosa drenándose a contracorriente en las venas perimedulares, pero esta fístula es microscópica y de bajo flujo, no permitiendo explicar por ella misma, la dilatación venosa observada. La perturbación de la circulación venosa medular objetivada por la ausencia de eferencias epidurales dorso-lumbo-sacras persiste después del tratamiento de la fístula, independientemente de la importante mejora funcional, que es frecuentemente observada. Por último, el origen adquirido o congénito de la fístula arteriovenosa como de un problema venoso medular no ha sido determinado actualmente.

#### DIAGNOSTICO DIFERENCIAL

Desde el punto de vista clínico, las fístulas dures son características. Sin embargo, es importante no confundir la hiperseñal del cono medular y región dorsal baja, observadas en la Resonancia Magnética, con un tumor intramedular o con placas aisladas de esclerosis múltiple.

#### TRATAMIENTO

La exclusión de la comunicación arteriovenosa por vía endovascular o por cirugía, está siempre indicada ya que permite frecuentemente una recuperación funcional notable e incluso inesperada o simplemente una estabilización del estado neurológico, mientras que la evolución natural de la enfermedad se realiza siempre hacia una paraplejía completa.

#### TRATAMIENTO QUIRURGICO

Las técnicas actuales son simples, ya sea el clipaje simple intradural de la vena de drenaje, antes de su conexión al sistema venoso medular, ya sea el clipaje asociado a la exéresis del fragmento de duramadre conteniendo la zona fistulosa.

#### TRATAMIENTO ENDOVASCULAR POR EMBOLIZACION

La técnica necesita un cateterismo superselectivo de la arteria nutricia; con verificación preembolización de la ausencia de anastomosis de esta arteria con la arteria espinal anterior. El material de embolización utilizado, tiene que ser fluido; actualmente, se utiliza el Hystoacril (n-butilcianoacrilato) cuyo tiempo de polimerización es modificado por la mezcla con Lipiodol. La eficacia de la embolización, exige la oclusión de la zona fistulosa y del pie de la vena de drenaje, con el fin de evitar una revascularización rápida por anastomosis proveniente de arterias adyacentes. El control arteriográfico, debe ser efectuado algunos días más tarde para verificar la ausencia de repermeabilización de la fístula.

#### INDICACIONES

En equipos expertos, la embolización no necesita anestesia general y es realizada, durante la arteriografía; es el tratamiento de elección y de realización simple. Dos condiciones fundamentales deben tenerse en cuenta: primero, ausencia de arteria espinal anterior al mismo nivel de la fístula, que contraindica el abordaje endovascular y segundo, posibilidad de cateterismo superselectivo estable.

El clipaje quirúrgico está únicamente reservado de primera intención, si existe una de las dos causas citadas anteriormente; o secundariamente, en caso de fallo por repermeabilización de la fístula después de la embolización.

#### RESULTADOS

La desaparición de la fístula (por técnicas quirúrgicas o endovasculares) va seguida de una mejoría funcional, que va a ser mucho más espectacular dependiendo de la precocidad del tratamiento. Esta mejoría, es frecuentemente sensible desde los primeros días que siguen al tratamiento o se dibuja más lentamente en algunas semanas. Sin embargo, cuando no se observa ninguna mejoría a los 3 meses del tratamiento, es necesario realizar un control arteriográfico, a la búsqueda de una revascularización de una fístula o eventualmente, de la presencia de otra fístula arteriovenosa del mismo tipo. Es muy importante, en un número de casos no despreciable observar la recuperación de una paraplejía flácida. La mejoría

funcional, es frecuentemente motora, mientras que los problemas sensitivos sobre todo las parestesias, pueden tardar más tiempo en regresar, siendo esta regresión, raramente total. Los problemas esfinterianos o genitales son igualmente, de una mejoría muy lenta.

### CONCLUSION

La existencia de una mielopatía dorso-lumbar de forma progresiva sobre todo en el hombre, es una indicación formal para realizar una Resonancia Magnética y en caso de ser negativa de una mielografía cuidadosa, necesitando obligatoriamente la realización de tomografías en cortes muy finos. En este contexto, la presencia de imágenes vasculares conduce siempre a una exploración con una arteriografía medular. Esta, debe opacificar todos los pedículos arteriales vértebro-raquídeos, cervico-dorso-lumbo-sacros, empezando por la región lumbo-sacra con el fin de buscar una fístula arteriovenosa dural, en la que le retorno venoso de la arteria de Adamkiewicz, está ausente o muy retrasado. Cuando la angiografía es negativa y las imágenes por Resonancia Magnética o por mielografía son características, es necesario explorar las vertebrales y las carótidas, pues existen fístulas A-V dures intracraneales, que se drenan preferentemente por las venas medulares y que se revelan por una sintomatología medular (12).

Un tratamiento es siempre propuesto, empezando por la embolización selectiva, que es el método de elección. El tratamiento quirúrgico (11), nos parece reservado actualmente a las contraindicaciones de orden anatómico o técnico de la embolización (1). Dicho tratamiento permite recuperaciones funcionales inesperadas.

### Referencias

1. Barth M.O., Chiras J., Rose M., Vega Molina J., et Bories J. Résultats de l'embolisation des fistules artério-veineuses dures rachidiennes à drainage veineux périmédullaire. Neurochirurgie, 1984; 30 n° 6: 381-386.
2. Djindjian M. Les malformations artério-veineuses de la moelle épinière. Paris IV. Pitié-Salpêtrière, 1976.
3. Djindjian R. Angiographie de la moelle épinière. Masson et Cie, édit., Paris 1970.
4. D. Dormont, E. Assouline, F. Gelbert, A. Helias, P. Halimi, J. Chiras, J. Bories, D. Doyon, J.J. Merland. Malformations artério-veineuses médullo rachidiennes. Étude en. I.R.M. J. Neuroradiol, 1987; 14: 351-364.
5. Heindel C., Duggen G.S. et Guinio F.C. Spinal arteriovenous malformation with hypogastric blood supply. Neuroradiology, 1977; 13: 181-189.
6. Kendal B.E., Logue V. Spinal epidural angiomatous malformations draining into intrathecal veins. Neuroradiology, 1977; 13: 181-189.
7. Launay M., Chiras J., et Bories J. Angiographie médullaire: temps veineux. Aspects normaux. Perspectives d'application en pathologie. J. Neuroradiol, 1979; 6 n° 4: 287-315.
8. Merland J.J., Riche M.C., Chiras J. Les fistules artério-veineuses intracanales extramédullaires à drainage veineux médullaire. J. of Neuroradiol, 1980; 7: 271-320.
9. Melki J.P., Riche M.C., Reizine D., Assouline E., Aymard A., Merland J.J. L'artériographie bi-fémorale simultanée rétrograde sous-pression. J. Neuroradiology, 1986; 13: 62-70.
10. N'Diaye, Chiras J., Meder J.F., Barth M.O., Koussa A., Bories J. Apport de la myélographie aux hydrosolubles dans les fistules artério-veineuses dures rachidiennes à drainage veineux périmédullaire. J. Neuroradiol, 1984; 11: 327-339.
11. Symon L., Kuyama H., Kendall B. Dural arteriovenous malformations of the spine. Clinical features and surgical results in 55 cases. J. of Neurosurg., 1984; 60: 238-247.
12. Woimant F., Merland J.J., Riche M.C., de Liege P., Bertrand N.J., Bacri D., Haguenu M. et Pepin B. Syndrome bulbo-médullaire en rapport avec une fistule artério-veineuse (FAV) méningée du sinus latéral à drainage veineux médullaire. Rev. Neurol, 1972; 138 n° 6-7: 559-566.

# Angioscopy: current techniques

6

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Examination of the internal surface of arteries has been attempted using a variety of devices. Because these systems have been cumbersome to use, angioscopy has not been widely used. Advances in miniaturization of optical fibers and endovascular intervention, however, have revived interest in angioscopy. This study illustrates a variety of angioscopes (0.85 to 3.2 mm in diameter) and experiences in the peripheral circulation. Both percutaneous and intraoperative approaches are described, using either saline solution or CO<sub>2</sub> gas flush as the visualization medium. A total of 32 procedures were performed (23 intraoperative; 9 percutaneous). Visualization of the arterial lumen provided clear detail of the lesion in all percutaneous cases. In 3 of these procedures, CO<sub>2</sub> gas was used. In the intraoperative approach, clear detail of the obstruction was noted in 16 of 23 arteries (70%). In 5 (22%), visualization was intermittent and in 2 (9%), visualization was not feasible. Via the percutaneous approach, the duration of the procedure was  $30 \pm 7$  min (mean  $\pm$  standard deviation) and the visualization time was  $20 \pm 4$  sec. In the intraoperative setting, the duration of the procedure was  $20 \pm 5$  min with a longer visualization time at  $37 \pm 5$  sec, which was significantly greater compared to percutaneous procedures ( $p < 0.001$ ). The volume of saline solution used in the percutaneous approach ranged from 500 to 800 cc and intraoperatively from 170 to 500 cc. Morphologic characterization of the interarterial lesions showed whitish yellowish atherosclerotic plaque and glistening red brown globulated fresh thrombus. Organizing thrombus was grey in color. Observations following laser recanalization using a "hybrid" probe showed thermal charring along the arterial lumen surface. Visualization following balloon angioplasty showed intimal flaps, large dissections and occasionally a false lumen. Angioscopy did not predict or alter perforations with laser recanalization; however, it did help in selection of lesions to avoid perforation. Angioscopy provided a method for (1) evaluation of the nature of the arterial blockage, (2) guidance of interventional procedures and (3) assessment of the results following interventional procedures. These data, if correlated to short and long term patency, may provide further insight into the mechanism of arterial interventions and help predict the outcome of the procedure.

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## INTRODUCTION

The concept of angioscopy dates back to 1913 when Rhea and Walker used a rigid illuminated tube in an attempt to visualize the endocardium.<sup>1</sup> Sixty years later, Shore et al and Gable et al revived interest in angioscopy by using a flexible device to perform endoscopy of great vessels.<sup>2</sup> This step, however, was limited by the large size of the angioscope with respect to the arterial lumen. Technical problems such as frequent fiber fractures and a lack of flexibility discouraged the early users. Recently, the miniaturization of fiber optic bundles and the advent of interventional procedures in arteries renewed interest in angioscopy. Furthermore, improved techniques to obtain a clear visual field such as balloon-occluding catheters have helped to overcome some of the difficulties previously encountered.



At present, angiography is a reliable method to assess the intravascular pathomorphology by direct three dimensional color imaging, thus providing more detail of the vascular structure than routine angiography. Angioscopes have been used both in peripheral and coronary arteries during intraoperative and percutaneous procedures. The purpose of this study is to demonstrate the various angioscopic techniques in the diagnosis of vascular lesions and its potential use for the control of interventional procedures such as laser recanalization, balloon angioplasty and bypass surgery.

## MATERIAL AND METHODS

### Equipment used for angiography

This study was accomplished with seven types of angioscopes (Fig. 1). These included the following:

(1) A 0.85 mm outer diameter angioscope with an illuminating optical bundle (IOB) surrounding a viewing optical bundle (VOB) (Miniflex™, American Edwards Lab, Irvine, CA).

(2) A 1.0 mm angioscope with an IOB surrounding a VOB (Miniflex™, American Edwards Lab).

(3) A 1.4 mm angioscope with an IOB surrounding a VOB (Olympus mode IPF 12, Olympus Corp. of America, New Hyde Park, NY).

(4) A 1.7 mm angioscope with a VOB and a surrounding array of illuminating fibers (Optiscope™ model 310, Trimedyne, Inc., Santa Ana, CA).

(5) A 1.7 mm angioscope with an IOB, a VOB and a 0.8 mm working channel (Olympus prototype).

(6) A 2.0 mm angioscope with a VOB and two working channels (0.9 and 0.6 mm). Illumination was provided by an optical fiber passed through one of the two channels (Trimedyne, Inc.).

(7) A 3.2 mm angioscope with an IOB, a VOB and a 1.2 mm working channel. This scope also had tip deflection capability (Olympus model BF3C4).

A flexible plastic sheath surrounds and protects the fiber optic bundles and the working

channels. These angioscopes have a spatial resolution ranging from 50  $\mu\text{m}$  to 200  $\mu\text{m}$ , a minimum focal length from 3 mm to 5 mm and a full angle of view, from 30° to 50°.

Illumination was provided by a xenon cold lamp (Karl Storz 487, Culver City, CA or Trimedyne Model 319). The images were relayed to a low light sensitive RGB camera (Pulnix TMC-54) and viewed online using a high resolution color monitor (Sony DVM-1910). Permanent video recordings (Sony 3/4 inch video tape recorder, Model VO 5800) were obtained (Fig. 2).

### Interventional procedures







Interventional techniques evaluated with angiography included peripheral laser recanalization, balloon angioplasty, and bypass surgery. The laser and balloon procedures were detailed in other reports.<sup>3-6</sup> In brief, laser recanalization was done using a 2 mm Spectraprobe-PLR™ (Trimedyne) to recanalize totally occluded arterial segments. A continuous-wave argon laser (Endocoagulator, HGM Model 20S, Salt Lake City, UT) was used to energize the probe. Percutaneous transluminal angioplasty (PTA) was done using 4 to 7 mm diameter and 5 cm long balloon catheters (Microvasive, Inc.). Bypass surgery was done using either prosthetic graft (PTFE or woven dacron) or in situ vein graft. Standard bypass surgical techniques were used.




### Patient selection

Thirty patients aged 52 to 80-years, with either severe intermittent claudication or rest pain had angioscopic evaluation. Each patient had previous angiography showing total obstruction of the superficial femoral or popliteal arteries. Informed consent was obtained from all patients for both the interventional procedure and the angiography.

### Percutaneous angiography

Angioscopes measuring 1 to 1.7 mm in diameter were utilized via a percutaneous approach. Prior to utilization, the angioscopes were sterilized using ethylene oxide. The camera and adapter were then put in a sterile plastic bag and

Angioscopes Characteristics	American Edwards Miniflex	Olympus PF 12	Trimedynne Optiscope model 310	AIS & Olympus prototype	Microvasive Visicath	Olympus BF 3C4
Cross section						
Outer diameter	1.0 (3F)	1.4 (4F)	1.7 (5F)	1.7 (5F)	2.0 (6F)	3.2 (9F)
Flexibility	++	++++	+++	+++	+	steerable

○  
illuminating bundle

○  
viewing bundle

●  
working channel

Fig. 1 Description of seven commercially available angioscopes, six of which were utilized in this study. The angioscope manufacturer is listed in the top row and the characteristics are listed in the vertical column. The second row shows the cross section of the angioscope with the configuration of the illumination bundle, viewing bundle, and working channel. The third row shows the outer dimensions of the angioscopes, from the smallest, 3 Fr, to the largest, 9 Fr. The bottom row describes the angioscope flexibility, ranging from + (least flexible) to + + + + (most flexible). The steerable angioscope is the only one with tip maneuverability.

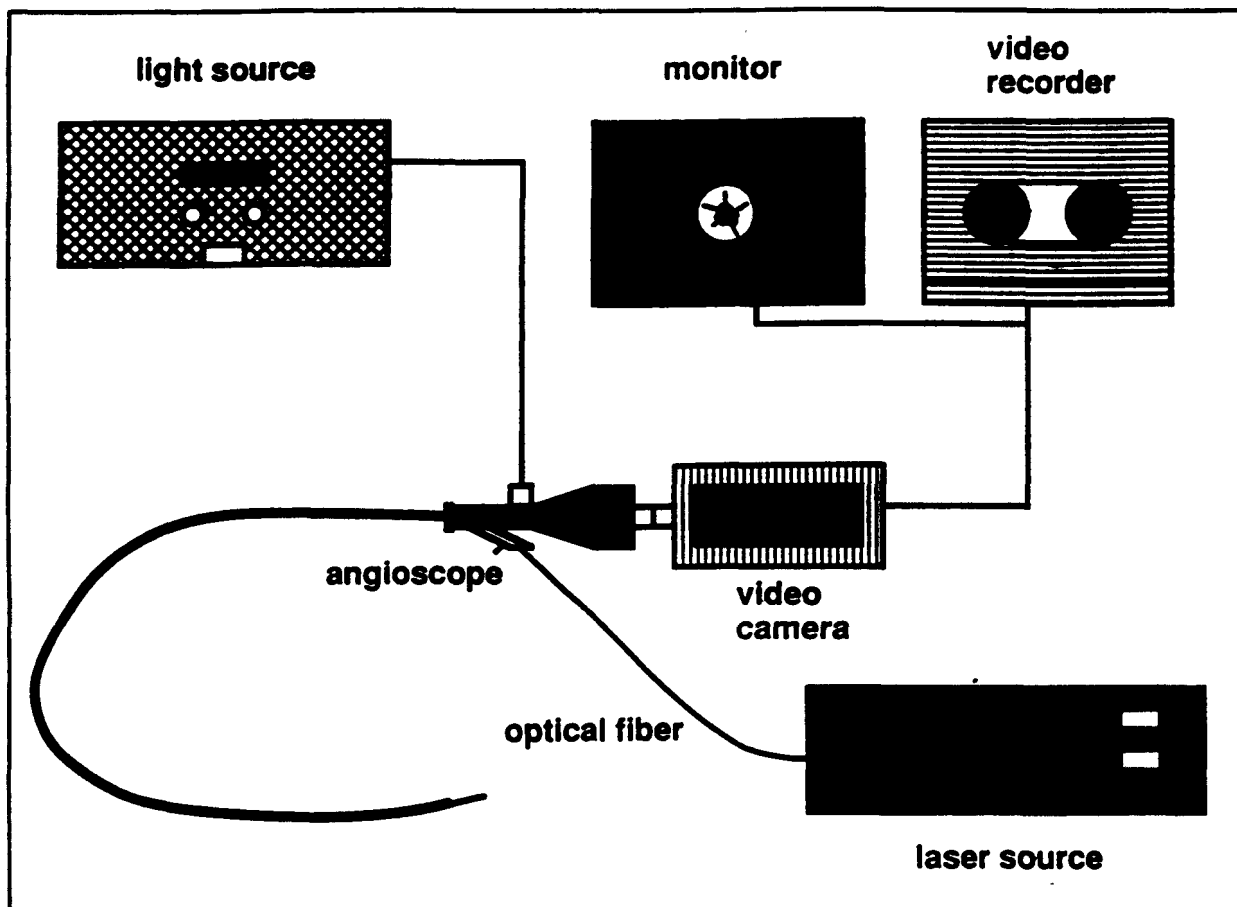


Fig. 2 Standard angioscope setup is shown. This includes a light source, TV monitor, video recorder, video camera and angioscope. An interventional device, such as a laser, can be used during angioscopy. An optical fiber, from the laser source, is shown extruding from the tip of the angioscope.

placed on the fluoroscopy table. The angioscope was connected to a video camera using a C-mount adapter. An 8-French introducer sheath was placed into the superficial femoral artery in an antegrade fashion using the standard Seldinger technique. The angioscope was then loaded inside a 7-French thin-wall catheter with a Tuohy-Borst connector at the hub. The tip of the angioscope was advanced to the catheter tip and this position secured by tightening of the Tuohy-Borst connector. The light intensity and focus of the angioscope were then adjusted to provide a clear image on the video monitor prior to insertion into the artery.

The guiding catheter with the angioscope were inserted into the sheath and gently advanced in an antegrade fashion into the femoral artery. The position of the angioscope tip was monitored with fluoroscopy. In order to prevent clot formation on the lens at the angioscope tip, heparinized saline solution was infused continuously around the angioscope (1 U/ml) from a pressurized bag at 300 mm Hg. Contrast material was injected intermittently to define the location of the vascular obstruction while simultaneously viewing the lumen with the angioscope. In order to obtain a clear field of view, the blood column in front of the scope was displaced using one or more of the following techniques:

(1) 60 cc of saline solution flushed by hand injection at a rate of 2 ml/sec for 30 seconds either through or around the angioscope via the guiding catheter or the working channel of the angioscope.

(2) 60 cc of CO<sub>2</sub> gas flush using hand injection or by constant pressure from a special CO<sub>2</sub> injector.

(3) Balloon inflation at the catheter tip temporarily occluding antegrade blood flow while flushing saline through the catheter to clear the forward blood column.

In each patient, saline or CO<sub>2</sub> flushes were given separately or in an alternating fashion depending on the ability to visualize the images while attempting to limit the volume of saline solution used. The angioscope was held in a fixed position at the tip of the guiding catheter and coaxial positioning obtained by gentle rotation and withdraw-advance movement of the catheter in order to obtain images of the arterial lumen,

rather than the arterial wall.

### Intraoperative angioscopy

Both small and large size angioscopes were used in the intraoperative setting. A cut-down over the common femoral artery was done as part of the standard surgical procedure for either bypass, endarterectomy, embolectomy, or laser recanalization. After exposure of the femoral vessels, an arteriotomy was performed and the angioscope advanced into the distal circulation. In this setting, antegrade blood flow was interrupted by clamping of the common femoral artery above the arteriotomy. For the smaller diameter angioscopes (1-1.7 mm), the flushing techniques to clear the forward blood column were the same as those described for the percutaneous approach. When the larger angioscopes (2-3 mm) were employed, an occluder balloon at the angioscope tip was used, if it was available, and saline flush via a working channel was sufficient to clear the field for visualization. When no occluder balloon was available, simultaneous proximal flushing through the introducer sheath and distal flushing from the tip of the angioscope were needed to obtain a clear field.

### Angioscopy during arterial interventions

Arterial intervention included laser recanalization, balloon angioplasty and peripheral bypass surgery. Angioscopy was performed before, during and/or after the arterial intervention in order to diagnose the nature of the lesion, guide the laser and/or evaluate the immediate results of the procedure. Laser recanalization was done using a 2.0 or 2.5 mm Spectraprobe-PLR™. Argon laser power was delivered using a dosimetry matrix by starting at 5 watts for 1 sec and 0.2 sec pauses. Power was increased by 1 watt increments until successful recanalization was achieved. The probe tip was positioned against the arterial obstruction under angioscopic visualization. Visualization was maintained throughout the recanalization process. Fluoroscopy was also used simultaneously during recanalization. After completion of laser recanalization, the new channel was inspected using one of the thinner angioscopes (1-1.7 mm). If balloon angioplasty was needed to enlarge the residual arterial steno-

sis, then angiography was used to evaluate the arterial lumen following the procedure. When bypass surgery or thrombectomy were done, angiography was used to assess the final result.

During the interventional procedures, all patients received 10,000 U of heparin. After completing the angiography, the angiogram, guiding catheter and sheath were removed. Hemostasis of the entry site was obtained by standard suture closure of the artery in the intraoperative setting or by hand pressure in case of percutaneous procedures. Anticoagulation was subsequently reinstated using heparin.

### Data analysis

Data were expressed as mean values  $\pm$  1 standard deviation. Visualization times were compared using unpaired two-tailed Student t test.

## RESULTS

Thirty-two angiographic examinations were performed in 30 patients during laser recanalization, 23 were done via arteriotomy during intraoperative procedures and 9 via percutaneous approach. No complication related to angiography was noted either immediately or at short-term followup.

### Duration of procedure

*Percutaneous approach* — The average duration of the procedure was  $30 \pm 7$  min (mean  $\pm$  SD). The visualization time was  $20 \pm 4$  sec using saline solution and/or CO<sub>2</sub> gas. An average of 5 (range 3-8) visualizations was attempted per procedure.

*Intraoperative approach* — The average duration of the procedure was  $20 \pm 5$  min (mean  $\pm$  SD). The visualization time was  $37 \pm 5$  sec. Visualization time was significantly longer in the intraoperative procedures compared to the percutaneous procedures ( $p < 0.001$ ).

### Volume of fluid

*Percutaneous approach* — The volume of saline solution which was used in each case ranged from 500 cc to 800 cc.

*Intraoperative approach* — The volume of saline solution was used in each case ranged from 170 cc to 500 cc.

### Images

*Percutaneous approach* — In all nine cases done, visualization of the vascular lumen provided clear detail of the obstruction site. Saline solution was used as the only visualization medium in 6 of the 9 procedures. Saline solution was used in an alternating fashion with CO<sub>2</sub> gas in the 3 other procedures.

*Intraoperative approach* — Visualization of the vascular lumen provided clear detail of the obstruction in 16 of the 23 arteries (70%). In 5 (22%), visualization was intermittent either because of a lack of coaxial position, low light intensity and presence of a clot at the angiogram tip. In 2 (9%) procedures, images were not sufficiently clear to identify vascular structures; this was due to a technical problem with the monitor and camera system. Saline solution was used as the sole viewing medium in 21 of the 23 procedures and alternated with CO<sub>2</sub> gas in the 2 other procedures.

### Morphology of arterial obstruction

Occluding atherosclerotic plaques were identified in 24 procedures as whitish-yellowish lesions blocking the lumen. Fresh occlusive thrombus was visualized in 6 arteries and appeared as glistening red-brown, lobulated or smooth surfaced (Video Sequences 3, 6). Occasionally, older organized thrombus appeared to merge with the plaque and was greyish with areas of brown streaks. Above the occlusion site, mural thrombus partially occluding the lumen and laying flat against the arterial wall was often noted. Intimal flaps were frequently noted along the vascular surface in atherosclerotic areas not previously instrumented. Ulcerated plaques were also occasionally identified in areas of dense plaque formation.

### Guidance for interventional procedure

Angiography done during laser recanalization was used to place the laser probe in a coaxial position to avoid inadvertent irradiation of the arte-

rial walls. This, however, did not seem to prevent perforation during laser recanalization. The tip of the fiber could be observed in one plane only while advancing beyond the tip of the angioscope. The repetitive blue-green light flashes during argon laser activation interrupted visualization by saturating the camera sensors. Bubbles of boiling saline solution generated by the laser vaporization also intermittently obstructed the field of view (Video Sequence 1). Once blood flow was reestablished across the arterial obstruction, it became increasingly difficult to clear the field of view for satisfactory visualization without the use of an occluder balloon.

### Evaluation of arterial interventions by angioscopy

*Following laser recanalization* — The walls of the new channel created by the laser could be identified as regular, smooth arterial lumen surfaces. The diameter of the channel was not much wider than the diameter of the probe tip used for recanalization. The wall of the channel was charred and white flecks embedded in the arterial wall suggested calcific deposits. Intimal flaps could also be readily visualized (Video Sequence 1). When recanalization resulted in communication with the distal arterial segment, backflow of blood was observed as the angioscope was passed beyond the recanalized segment for further inspection of the arterial lumen.

None of the 5 arterial perforations that occurred during lasing were detected by angioscopy. Nevertheless, the following example typifies the utility of angioscopy to prevent perforation. In an intraoperative case (Video Sequence 2), lasing was initiated under fluoroscopic guidance only. The laser probe could be seen making an acute turn toward the arterial wall, which suggested imminent perforation. The probe was removed and a 2 mm angioscope advanced up to the lesion. This revealed a dense intraluminal rock-like calcification. Since continuous-wave argon thermal lasing is not known to penetrate this type of obstruction, no further lasing attempts were made and a bypass was performed.

Another example typifies the utility of angioscopy to elucidate the angiographic observations with respect to the cause of an obstruction.

During a percutaneous case, laser recanalization appeared to be successful in crossing the obstruction in a distal superficial femoral artery; however, the angiogram revealed an interruption of contrast flow to the popliteal artery. It was assumed that a subintimal channel was created and the procedure was not successful. During angioscopy, branching vessels in the recanalized segment were clearly visualized, indicating that the channel was not subintimal; however, an organized thrombus was seen obstructing the lumen at the site of contrast interruption by angiography. During this case, simultaneous angioscopic and angiographic images were obtained. This demonstrated the feasibility of clear angioscopic visualization through the contrast medium.

*Following balloon angioplasty (BA)* — Four arterial segments were inspected after laser-assisted balloon angioplasty, using angioscopy. In 3 of 4 balloon-dilated arterial segments, intimal flaps could be clearly identified. Two had a large dissection with a false lumen channel (Video Sequence 7). Also, fresh thrombus embedded in the arterial wall was noted in one case following BA (Video Sequence 4).

*Following surgical procedures* — Arterial-vein graft or prosthetic graft anastomosis were inspected in 3 patients. This revealed a clean suture line without flaps. In an additional case, following embolectomy, angioscopy demonstrated layering of the vascular lumen with old thrombus still adherent to the vascular walls. Numerous intimal flaps were also observed in this case.

## DISCUSSION

Success in obtaining sufficient visualization to recognize intravascular structures during angioscopy depends greatly on the approach and the technique used. For example, intraoperative procedures were easier to perform than percutaneous procedures. In our series, the longest duration of visualization and the shortest duration of procedure were obtained intraoperatively. This is primarily related to the ability to interrupt antegrade blood flow. Also, the straight configuration of the peripheral arteries helped to steer the angioscope. Percutaneous angioscopy, however, is technically more difficult, usually requiring tem-

porary balloon occlusion. Nevertheless, percutaneous angiography is feasible and with miniaturization of devices, may provide a tool for precise description of arterial morphology. Since angiographic studies were initiated in the operating room, certain technical difficulties were encountered in the early part of the learning curve. This explains the reduced visibility in 5 cases and technical failure in 2 other cases which were reported in the intraoperative setting.

Preliminary studies reported that angiography could be a useful tool to guide laser angioplasty and prevent vessel perforation.<sup>7</sup> Our study suggests that angiography does not greatly alter arterial perforation rates. However, a better definition of the nature of the obstruction may change the approach to the intervention, thus avoiding perforation, as demonstrated by the example of the calcified obstruction (Video Sequence 2).

Angiography provides more detail than is usually obtained by standard angiography.<sup>8,9</sup> In our study, the procedure was safe and the images provided detailed information about the obstruction. The duration of the angiographic examination can be short enough not to interfere with the interventional procedure. This study shows that angiography can provide additional imaging data which can be used to complement standard angiography. This imaging data was found to be most useful in the three following areas:

*Assessment of arterial occlusion* — Angiography can differentiate thrombus from atherosclerotic lesion and also provided data about the age of the thrombus.<sup>10,11</sup> This distinction is important since fresh thrombi may be treated using thrombolytic therapy as a preferred approach.<sup>12</sup> Angiographic recognition of thrombus is often underestimated.<sup>13</sup> This may be related to passage of contrast all around the thrombus so that only changes in density of the lumen are seen but not the degree of lumen obstruction. Thus, angiography may provide a more accurate morphopathologic analysis to help in the selection of the most appropriate interventional technique (i.e., laser, thrombectomy, atherectomy, or thrombolysis).<sup>8,14,15</sup>

*Guidance for interventional procedures* — Angiography allowed coaxial positioning of the Spectraprobe™ tip in the occluded vessel and confirmed contact of the probe tip with the ob-

struction. In spite of the ability to visualize the relative position of the Spectraprobe™ to the arterial wall, angiography did not alter the perforation rate. This is due to the inability to visualize the probe's direction once it enters the obstruction. Nevertheless, in our study, it was possible to identify bifurcations and heavily calcified plaques which are a frequent site of perforation,<sup>4</sup> and thus prevent imminent perforations.

*Assessment of interventional procedure* — The laser-formed channel or balloon dilated lumen can be visualized as a cross-sectional image.<sup>16</sup> Angiography provides a qualitative assessment of the immediate results, detailing the degree of charring, residual lumen size and intimal tears. To obtain a more accurate evaluation of the lumen size, a computer-based image processing method was recently reported by Friedl et al.<sup>17</sup> Using this approach, precise determination of arterial lumen diameter before and after recanalization may be obtained.

Many of the initial technical limitations of angioscopes, such as lack of steerability and stiffness, have been greatly resolved. In our study, the major difficulty was a sustained displacement of blood from the field of view. Caution must be exercised with respect to excess fluid delivery especially in patients with heart failure or renal disease.<sup>18</sup> Hawkins et al proposed infusion of CO<sub>2</sub> gas to increase the duration of clear visualization.<sup>19</sup> In our series, however, no major difference in viewing time could be noted in the 3 procedures done with CO<sub>2</sub> gas. The advantage of CO<sub>2</sub> gas compared to saline solution is its low viscosity and high compressibility. In addition to its application as a visualization medium, CO<sub>2</sub> gas may be used as an efficient lasing medium.<sup>20</sup> Finally, CO<sub>2</sub> gas may reduce the volume of saline solution required during an angiographic procedure.

Another technique to clear the field of view is the use of a guiding-balloon catheter which has been reported by Uchida et al.<sup>21,22</sup> This method utilizes an occluder balloon at the tip of a 9 Fr. guiding catheter. The angioscope is passed through the guiding catheter and flush solution is infused around the angioscope to clear the field. This technique can reduce the volume of flushing medium needed to displace back flow of blood. Also, the use of a foot-paddle control high-

pressure irrigation pump seems to facilitate the clearing of blood from the field of view.

In spite of these advantages and its low risk, angiography has potential limitations. For example, the examination of arterial segments facing against the direction of blood flow may be very difficult, due to the inability to displace the blood column. Therefore, percutaneous angiography of the iliac arteries and aorta via a retrograde approach is greatly limited. Another situation may be in small arteries such as the coronaries with several tandem lesions. The angioscope may not be able to cross beyond the proximal lesion to evaluate the distal arterial segments.<sup>23</sup>

Another application of angiography has been in the detection of congenital cardiac lesions. Sargin has reported on this technique during cardiac surgery for congenital heart disease.<sup>24</sup> This was especially useful in detecting the presence of anomalous pulmonary venous return in association with atrial septal defects.

At present, another problem is the cost of angioscopes. In the era of communicable diseases, such as hepatitis and Acquired Immune Deficiency Syndrome, the issue of cheap and disposable angioscopes needs to be addressed. Already, high quality plastic optical fibers are being developed for such purposes.

Other intravascular imaging techniques, such as endovascular ultrasound, are also being developed. The advantages of ultrasound include the ability to obtain images in a blood-filled artery, the ability to obtain measurements of arterial wall and plaque thickness, and the ability to assess plaque composition. Calcific plaque is easily detected; however, soft plaque and fresh clot may not be as clearly defined. The major limitation of current ultrasound systems lies in their inability to view in the forward direction; the images produced are of the vascular wall immediately surrounding the ultrasound catheter, not of the lumen in front of the catheter tip. At present, angiography provides more in-depth forward viewing of the vascular lumen.

## SUMMARY

Although angiography has been available for many years, practical application was not possible until the advent of flexible fiber optic scopes.

Recent developments in interventional arterial techniques have revived interest in angiography; however, large and bulky angioscopes still limited practical use. Additionally, the lack of an effective blood displacement technique in order to see the interior of the vascular lumen dampened early enthusiasm. Many of these problems have been mostly resolved. Smaller diameter angioscopes with optic bundles less than 0.5 mm in diameter are now available. Guiding catheters with occluder balloons at their tips have greatly improved the percutaneous approach for angiography. Consequently, angiography may finally overcome many of its earlier limitations.

Although bypass surgery is still considered as the gold standard for treatment of obstructive vascular disease, other interventional techniques, including balloon angioplasty, laser, and atherectomy, have gained acceptance for treatment of arterial lesions. Certain devices are better suited for particular types of lesions. Endovascular imaging techniques and especially angiography appear to be useful to determine the procedure of choice by assessing the intra-arterial pathomorphology (i.e., fresh thrombus, plaque, calcification). This ability, as well as the guidance of recanalization and the assessment of interventional results are valuable applications for angiography.

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## REFERENCES

1. Rhea L, Walker IC. Cited by Cutler et al. Arch Surg 1913;9:689.
2. Gable WJ, Iris RE. Experimental intracardiac visualization. New Eng J Med 1967;276:1397.
3. Abela GS, Seeger JM, Barbieri E, et al. Laser angioplasty with angioscopic guidance in humans. J Am Coll Cardiol 1986;8:184-92.
4. Abela GS. Laser recanalization: a basic and clinical perspective. Thorac Cardiovasc Surgeon 1988;36:137-41.



5. Abela GS, Seeger JM, Fry RS, et al. Percutaneous laser recanalization of totally occluded human peripheral arteries: a technical approach. *Dynamic Cardiovasc Imaging* 1988;4:302-8.
6. Seeger JM, Abela GS. Angioscopy as an adjunct to arterial reconstructive surgery: a preliminary report. *J Vasc Surg* 1986;4:315-20.
7. Lee G, Ikeda RM, Stobbe D, et al. Intraoperative use of dual fiberoptic catheter for simultaneous in vivo visualization and laser vaporization of peripheral atherosclerotic obstructive disease. *Cathet Cardiovasc Diagn* 1984;10:11-6.
8. White GH, White RA, Kopchok GE, Klein SR, Wilson SE. Intraoperative video angioscopy compared with arteriography during peripheral vascular operations. *J Vasc Surg* 1987;6:488-95.
9. Grundfest WS, Litvack F, Sherman T, et al. Delineation of peripheral and coronary detail by intraoperative angioscopy. *Ann Surg* 1985;202:394-400.
10. Ferris EJ, Ledor K, Ben-Avi DD, et al. Percutaneous angioscopy. *Radiology* 1985;157:319-22.
11. Hickey A, Glick D, Litvack F. Peripheral vascular and coronary bypass graft angioscopy. *Dynamic Cardiovasc Imaging* 1987;1:80-3.
12. Tomaru T, Uchida Y, Nakamura F, Sonoki H, Tsukamoto M, Sugimoto T. Enhancement of arterial thrombolysis with native tissue type plasminogen activator by pretreatment with heparin or batroxobin: an angioscopic study. *Am Heart J* 1989;2:275-82.
13. Sherman CT, Litvack F, Grundfest W. Coronary angioscopy in patients with unstable angina pectoris. *N Engl J Med* 1986;315:913-9.
14. Uchida Y, Masuo M, Tomaru T, Kato A, Sugimoto T. Fiberoptic observation of thrombosis and thrombolysis in isolated human coronary arteries. *Am Heart J* 1986;4:691-6.
15. Rees MR, Gehani AA. Percutaneous high-resolution angioscopy: its role in evaluating new angioplasty techniques (abstr). *Radiology* 1988;169(P).
16. Lee G, Garcia JM, Corso PJ, et al. Correlation of coronary angioscopic to angiographic findings in coronary artery disease. *Am J Cardiol* 1986;58:238-41.
17. Friedl SE, Abela GS, Tomaru T, Barbeau GR, Haley RA. Quantitative endovascular angioscopy. In: *Optical Fibers in Medicine IV*, A. Katzir, ed., Proc. SPIE 1989; 1067:197-202.
18. Towne JB. Complications of vascular endoscopy. In: Moore WS, Ahn SS, eds. *Endovascular Surgery*. Philadelphia: Saunders, 1989:114-7.
19. Hawkins IF, Mladinich CRJ, Akins EW, Silverman SH. Use of carbon dioxide gas to improve visualization during angioscopy (abstr). *Abstracts RSNA* 1988.
20. Silverman SH, Khoury AI, Seeger JM, Tomaru T, Hawkins IF, Abela GS. Effect of CO<sub>2</sub> and blood media on laser probe temperature. *Las Surg Med* 1989;9:17-21.
21. Uchida Y, Hasegawa K, Kawamura K, Shibuya I. Angioscopic observation of the coronary luminal changes induced by percutaneous transluminal coronary angioplasty. *Am Heart J* 1989;4:769-76.
22. Uchida Y, Tomaru T, Nakamura F, Sonoki H, Sugimoto T. Fiberoptic angioscopy of cardiac chambers, valves, and great vessels using a guiding balloon catheter in dogs. *Am Heart J* 1988;6:1297-1302.
23. Sherman T. Clinical problems in coronary angioscopy. In: *Optical Fibers in Medicine IV*, A. Katzir, ed., Proc. SPIE 1989;1067:158-71.
24. Sargin M. Angioscopy of congenital cardiac lesions. In: Abela G., ed. *Lasers in Cardiovascular Medicine and Surgery: Fundamentals and Techniques*. Boston: Kluwer Academic Publishers, 1989;411-22.

## CO<sub>2</sub> Angiography: A Safer Contrast Agent for Renal Vascular Imaging?

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### ABSTRACT

Although the new nonionic contrast agents are safer than ionic agents, renal insufficiency and even death still occur occasionally. Therefore, we have explored the use of carbon dioxide (CO<sub>2</sub>) as an alternative angiography contrast agent used in combination with digital subtraction angiography. Clinical observations have been made in over 800 patients. The images obtained are of equivalent diagnostic quality compared with those using conventional iodinated contrast agents. Recent advances in imaging, including "stacking", provide images comparable with iodinated contrast. Very small vessels, equivalent to third-order branches of the renal artery, can be imaged satisfactorily with CO<sub>2</sub>. Occasional studies with CO<sub>2</sub> yield information not apparent with iodinated contrast agents, including excellent visualization of arteriovenous shunts, collateral circulations or filling defects in malignant tumors, as well as minute amounts of arterial bleeding. Many of the advantages and disadvantages of CO<sub>2</sub> derive from its special physical and chemical properties. The advantages include no allergic potentiation and no renal metabolism of CO<sub>2</sub>, because CO<sub>2</sub> is cleared by the lungs and does not recirculate. Other advantages include delivery by very small catheters because of the low viscosity of CO<sub>2</sub>, minimal discomfort on injection, and very low cost. However, the low-density and compressibility of CO<sub>2</sub> poses some special problems. Imaging requires digital subtraction angiography with electronic enhancement and injections require an experienced investigator and, ideally, a dedicated CO<sub>2</sub> injector. The dedicated CO<sub>2</sub> injector provides calculated, controlled dosing and rates for injection, while excluding the possibility of air contamination. The

buoyancy of CO<sub>2</sub> inhibits good filling of dependent vessels. Accordingly, CO<sub>2</sub> does not normally produce good nephrographic images, although proximal renal arteries are normally shown clearly. Experimental studies in dogs, whose renal arteries have been injected repeatedly with supramaximal doses of CO<sub>2</sub>, demonstrate only transient changes in renal blood flow and no endothelial cell damage. However, these studies also showed clearly that renal ischemia can occur due to a "vapor lock" phenomenon if the kidney is positioned vertically above the injection site, and recurrent injections are given without time for absorption of the arterially-delivered CO<sub>2</sub> boluses. Uncontrolled studies in over 800 patients have confirmed that CO<sub>2</sub> likely has a very low renal toxicity. At the University of Florida, CO<sub>2</sub> is the radiologic contrast agent of choice in patients with renal insufficiency, especially those with diabetes mellitus, and in those with pre-existing allergy to contrast agents. Further controlled clinical studies are required to define the true clinical utility and safety of CO<sub>2</sub> compared to conventional radiologic contrast agents.

**Index Words:** angiography, nephrotoxicity, renal insufficiency, carbon dioxide, iodinated contrast agent.

### INTRODUCTION

Although considerable progress has been made in vascular imaging with ultrasound, computerized tomography and magnetic resonance angiography, iodinated contrast angiography remains the gold standard for all vascular imaging. Since the introduction of iodine as an angiographic contrast agent in the early 1920s, the safety of contrast agents in angiographic procedures has improved markedly. Unfortunately, procedural complications

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still occur. Their overall incidence is approximately 1.7% for the femoral approach (1); renal insufficiency and even death are reported on rare occasions (2). The newer nonionic contrast agents cause less adverse reactions (3); however, they are very expensive and several studies have failed to show that the incidence of nephrotoxicity is consistently reduced (4-8). However, some recent papers demonstrate a lower incidence of renal insufficiency with nonionic, compared to ionic, contrast agents in patients with pre-existing renal disease (9-11). The risk of renal failure with any contrast agent is increased in patients with renal insufficiency and diabetes. Therefore, many physicians are reluctant to recommend angiography in these high risk patients, even though they recognize that the consequences of undiagnosed renovascular hypertension and incapacitating peripheral vascular disease may result in a poor outcome for the patient. Therefore, there is much interest in the development and evaluation of safer forms of renal vascular imaging.

Since CO<sub>2</sub> is totally eliminated by the lungs in a single pass, if it is injected distal to the renal arteries, there is no impact whatsoever on renal function. Moreover, recent evidence suggests that selective renal injections also have a low toxicity for the kidneys (12).

Many of the special advantages and disadvantages of CO<sub>2</sub> as a radiographic contrast agent are predictable from its special physical and chemical properties. Therefore, these will be reviewed. A physician who is considering requesting a renal CO<sub>2</sub> arteriogram should be aware of the principles of the technique that his/her patient is to undergo. This should enable the physician to plan the procedure rationally with the radiologist, thus selecting appropriate patients, and guard against potential adverse effects. Therefore, the methods used in CO<sub>2</sub> angiography will be reviewed.

CO<sub>2</sub> was first injected intraperitoneally in 1914, to outline the abdominal structures radiographically (13). The first intravascular use of CO<sub>2</sub> was in the 1950s, for demonstration of pericardial effusion. CO<sub>2</sub> (100-200 ccs) was injected into a antecubital vein with the patient in the left lateral decubitus position, thereby trapping the CO<sub>2</sub> in the right atrium (14-16). The pericardial effusion was imaged between the gas density in the right atrium and the gas density of the lung. Before CO<sub>2</sub> was used in patients, it underwent extensive animal testing, using both intravenous and arterial routes of administration (17,18). Even with massive

intravascular injections, only minimal and transient changes in arterial blood gas composition were observed. Very large volumes could be injected repeatedly and indefinitely in dogs, if time were allowed between injections for the CO<sub>2</sub> to be eliminated by the lungs.

We initially used CO<sub>2</sub> as an arterial contrast agent in 1971, using a cut-film subtraction technique. The CO<sub>2</sub> was delivered with a hand syringe. It produced diagnostic images both in the kidney and in the extremities. However, the photographic subtraction techniques were time-consuming and, in general, produced suboptimal images.

With the advent of digital subtraction angiography (DSA) in 1980, this "low-density contrast agent" became more readily imaged(19).

In general, the final images that can be obtained with CO<sub>2</sub> DSA are almost identical to those obtained with liquid iodinated contrast. However, the physical properties of the gas are quite different from those of liquid contrast.

Iodinated contrast injected into the vascular system mixes with blood, and is excreted quantitatively over time in the glomerular filtrate. The high atomic number of the iodine atoms inhibit passage of the X-ray beam and produce a "shadow" on the X-ray film. The density of the image is improved by either injecting more iodine (i.e., injecting at a higher rate and volume) or increasing the concentration of the iodine in the contrast fluid. Both of these maneuvers will increase the total dose of the contrast agent administered and, therefore, will increase the incidence of toxic effects.

The principles of vascular imaging with CO<sub>2</sub> are quite different from those of conventional liquid contrast. Vessels are imaged with CO<sub>2</sub> by totally displacing the blood from the lumen of the vessel. This allows more X-rays to penetrate the vessel, since the density of the gas is lower than that of the surrounding soft tissues. If the blood is totally replaced with CO<sub>2</sub>, the addition of more CO<sub>2</sub> will not improve the image. Since there is only a minimal difference in attenuation of the X-ray beam between the gas-filled vessel and the surrounding soft tissue, this density differential must be enhanced using DSA. This effectively subtracts out the images caused by the bone and soft tissue, leaving only the CO<sub>2</sub> image which can be further enhanced electronically. Sophisticated software is

now available which permits not only electronic enhancement of the contrast agent, but also allows "stacking" of multiple images when only one segment of the vessel is filled (20). This produces a single composite image in which the entire artery is visualized. The stacking technique permits injection of a smaller volume of CO<sub>2</sub>, which traverses the artery as a single large bubble. Since its passage conforms to the interior of the vessel, if multiple images are obtained at a rapid rate, images of this single "bubble" which have displaced the blood, can be assembled to produce a single diagnostic image (Figure 1). This is not possible with iodinated contrast which simply mixes with blood and is diluted by it.

It is clear from this analysis that the techniques needed to image CO<sub>2</sub> are considerably more stringent than those for iodinated contrast. DSA requires the patient to be absolutely motionless. Peristaltic bowel gas motion also will degrade the image.

CO<sub>2</sub> images that are comparable to iodinated contrast can be obtained only if: (1) high resolution DSA X-ray equipment is available, (2) no motion occurs, and (3) the blood is completely displaced from the vessel.

#### *Physical and Chemical Properties of CO<sub>2</sub> that are Revelant to its Use in Angiography*

The gaseous properties of CO<sub>2</sub> provides a unique contrast agent whose most prominent advantages and disadvantages are summarized in Table 1. These must be thoroughly understood to permit consistent, safe delivery and for correct interpretation of the images.

#### **Low Viscosity**

There are several consequences of the extremely low viscosity of CO<sub>2</sub> that are relevant to its use as an imaging agent.

1. This permits its delivery via very small catheter (as small as 1.5 French) which greatly reduces the risk of procedural complications.
2. The low viscosity allows CO<sub>2</sub> to flow from the artery to the vein without apparent blockage of the capillary bed. We have demonstrated that, when large volumes of CO<sub>2</sub> are injected into the aorta of a dog, the CO<sub>2</sub> returns to the inferior vena cava and right heart, and disappears from the pulmonary arteries within a few seconds.

Since the lungs eliminate the CO<sub>2</sub> in a single pass, if small volumes are injected (less than 200 cc/sec) and if time is allowed between the injections, CO<sub>2</sub> can be injected in unlimited volumes. However, it is very important to stress that if a single very large bolus of CO<sub>2</sub> is injected inadvertently, the CO<sub>2</sub> can displace the blood from the right heart, resulting in a "vapor lock" phenomenon and death.

3. The low viscosity apparently permits arterial venous (AV) shunting of the agent through tumors (20,21), better collateral filling (20), an detection of minute amounts of arterial bleeding which may not be imaged with the more viscous liquid contrast. We have injected CO<sub>2</sub> selectively in over 20 malignant hypernephromas. The majority demonstrated immediate AV shunting with good opacification of the inferior vena cava which was not seen with iodinated contrast.

#### **Buoyancy**

The extreme buoyancy of CO<sub>2</sub> results in selective filling of nondependent portions of the vascular bed. For accurate imaging, the blood should be either totally displaced from the vessel or the region of interest placed above the injection site. With the patient lying supine, nondependent branches of the abdominal aorta, such as the celiac axis, superior mesenteric artery and inferior mesenteric artery, always fill, even with injections of very small amounts of CO<sub>2</sub>. In contrast, the lumbar arteries are very difficult to fill since they are more posterior. The origins of the renal arteries usually fill well; however, the more distally renal arteries are more difficult to fill since they are posteriorly located and are below the injection site.

The buoyancy properties of CO<sub>2</sub> can be illustrated by the analogy of injecting helium into a hollow tree. If the tree is in the normal upright position, the helium will float into all of its branches. If it is inverted, it will be very difficult to fill the distal branches and if the tree is lying on its side, only the nondependent half of the tree will fill.

The buoyancy properties may explain why occasionally the left renal artery is not seen. This is secondary to counter-clockwise rotation of the aorta that can occur in older patients, which results in more posterior location of the origin of the left renal artery. This problem can be overcome by rotating the patient in the opposite direction (lateral

decubitus position or a semi-prone position). The renal artery is then nondependent and will fill readily. It can be imaged by cross-table orientation of the X-ray beam even with a very small amount of CO<sub>2</sub>.

Buoyancy can also be a disadvantage if the vector of forces of the buoyancy exceeds the kinetic energy of the venous flow in any end-arterial type organ such as the kidney, bowel, or elevated lower extremity. This can lead to entrapment of CO<sub>2</sub> in the vascular space of the organ. If repeated injections are made at frequent intervals, there is potential for a "vapor lock" leading to organ ischemia. This phenomenon is unusual because of the high solubility of CO<sub>2</sub> in blood (20 X that of oxygen in blood) which implies that, if the CO<sub>2</sub> did block capillaries, the ischemia should be very short-lived since the CO<sub>2</sub> should dissolve very rapidly. In practice, the injection of CO<sub>2</sub> in areas where it can potentially become trapped should cause only transient ischemia, providing due time is allowed between successive CO<sub>2</sub> injections.

#### **Invisibility and Compressibility**

The invisible and compressible nature of CO<sub>2</sub> poses unique problems for its reliable and safe delivery into the vascular system. Medical grade CO<sub>2</sub> is available in a highly pure form in high pressure CO<sub>2</sub> cylinders. Most portable cylinders contain over three-million ccs of the gas at atmospheric pressure. Gas is transferred from the high pressure cylinder via a gas regulator.

If the regulator malfunctions, an excessive volume of the CO<sub>2</sub> may be loaded into a hand-held syringe or an angiographic injector's cylinder. There is also a danger that CO<sub>2</sub> could be contaminated with room air via loose connecting Luer lock fittings.

A more common problem derives from the ready compressibility of CO<sub>2</sub>. When the injection is made with a hand-held syringe or angiographic injector, as the cylinder of the syringe is advanced, the gas will initially be compressed and will not emanate from the tip of the catheter into the vascular system. When the pressure in the syringe exceeds the resistance of the catheter and the arterial pressure, the majority of the gas will be delivered in the last fraction of a second resulting in an "explosive" type of delivery. Such a high velocity injection will fill the vessels for only a very short time. This problem has led to the requirement for a very high frame rate to image the gas. With standard DSA equipment, this may exceed its

capacity to image the vessel. This problem has been reduced by the development of "stacking programs" which summates successive fragments of the CO<sub>2</sub> image to form a composite image (see above).

Clearly, there are problems with reliable and safe delivery of CO<sub>2</sub>. Therefore, we have recently developed a device that delivers CO<sub>2</sub> in a controlled, nonexplosive manner. The CO<sub>2</sub> passes through multiple valves, and its passage is monitored by successive pressure sensors. The system is EKG- and pressure-gated to allow the injection of a higher volume of CO<sub>2</sub> during systole, and a lower volume during diastole. This provides a uniform displacement of blood from the vessel. The entire injector system is hermetically sealed to prevent air contamination. It also incorporates a closed system with a micro-filter which provides sterility of the gas and automatic closed flushing to prevent catheter clotting. This device is not yet commercially available since it is under evaluation by the Federal Drug Agency. However, with practice, CO<sub>2</sub> can be quite reliably delivered by a hand-held syringe or by mechanical injectors, proving care is taken to use a disposable CO<sub>2</sub> cylinder and to guard against severe, explosive-type delivery by moving the syringe cylinder forward at a reduced velocity as the injection terminates. This requires a little practice. This technique has been described (20,22).

#### *Diagnostic Accuracy and Clinical Experience of CO<sub>2</sub> Angiography*

The low-density image of CO<sub>2</sub> is more difficult to visualize and interpret than iodinated contrast. However, with meticulous technique elevating the area of interest, restraining the patient to prevent motion, and decreasing bowel gas motion with glucagon, diagnostic images can be obtained in greater than 90% of the studies. In practice, CO<sub>2</sub> is often selected in high risk patients, especially those with renal insufficiency. Sometimes even multiple injections of CO<sub>2</sub> do not provide truly diagnostic information. In these circumstances, a single injection of iodinated contrast with DSA or cut film will produce a diagnostic study that has limited the risk of nephrotoxicity by severely curtailing the total dose of iodinated contrast agent used.

#### *Clinical Experience with CO<sub>2</sub> Angiography*

CO<sub>2</sub> is most reliable for examination of peripheral vascular disease of the lower extremity, since the images are not degraded by bowel gas motion.

Recently, we performed a retrospective study of 128 CO<sub>2</sub> arteriograms obtained in 115 patients (23). Comparison studies with iodinated contrast were available in 98 of these patients. Many patients had concomitant medical illnesses that increases the risk of arteriography. These included renal insufficiency in 70 patients (serum creatinine concentration greater than 1.5 mg/dl), hypertension requiring multiple drugs in 85, diabetes mellitus in 44, severe coronary artery disease in 62, and cerebral vascular disease in 27. In this series, 47 abdominal arteriograms were performed for abdominal aneurysmal disease, renal artery stenosis (Figure 2), or mesenteric ischemia. Most patients also had an extremity study. The CO<sub>2</sub> arteriograms were rated to be of good or excellent quality in 117 cases (91%). There was diagnostic agreement between the CO<sub>2</sub> and standard contrast studies in 95% of the cases. Subsequent operative findings confirmed the correct diagnosis in 92% of the cases with CO<sub>2</sub>. The diagnostic insufficiencies were due to inadequate visualization of the infrapopliteal arteries in seven cases. Although 80 patients presented with renal insufficiency and five with recent renal transplantation, no increase in serum creatinine concentration was detected in any patient in whom only CO<sub>2</sub> as used. One case of contrast-induced nephrotoxicity was noted where both CO<sub>2</sub> and iodinated contrast were used together.

Equally good results were found in a second study of high risk patients. This group had renal artery stenosis and required reimplantation of their renal arteries to address poorly controlled hypertension or deteriorating renal function (24). All the CO<sub>2</sub> arteriograms were considered diagnostic (Figure 3). There were no complications detected in this study, and the serum creatinine concentration remained stable after the CO<sub>2</sub> arteriograms in these patients.

Preliminary data is also available on 15 patients with renal transplant that were studied with CO<sub>2</sub> angiograms. Again, there was good visualization of the renal arteries, including second and third order branches (Figure 4). No elevation in serum creatinine concentration or other complications were detected during follow-up. It is important to stress that these observations were not blinded, controlled studies. Therefore, some minor adverse effects of CO<sub>2</sub> may have escaped detection.

Because of the low-density of CO<sub>2</sub> and the nondependent position of the normally oriented kidneys, a nephrogram is not usually done. However, the proximal renal artery and up to the third-order branches are usually well seen.

Unfortunately, accurate and interlobular arteries are not well imaged because they are usually obscured by overlying bowel gas and minor degrees of motion in the majority of patients. In patients with suspected small vessel disease, such as arteritis, CO<sub>2</sub> can be used to rule out aortic abnormalities and main renal artery stenosis. However, standard cut films and iodinated contrast is recommended to image small vessels. By combining techniques, a diagnostic study can be obtained with as little as 10 cc of nonionic contrast.

The ability to use CO<sub>2</sub> in unlimited quantities makes it ideal for complicated interventional procedures, especially in patients with renal insufficiency. We have performed several angioplasties, including two cases of renal artery stenosis solely with CO<sub>2</sub>.

#### *Renal Toxicity of CO<sub>2</sub> Angiography*

When CO<sub>2</sub> is used for run-off and extremity studies, the injection is made distal to the renal arteries. Therefore, there is no impact whatsoever on renal function, since the CO<sub>2</sub> is eliminated by the lungs in a single pass. Even CO<sub>2</sub> aortography provides only a small CO<sub>2</sub> load to the kidney, since the buoyancy of CO<sub>2</sub> inhibits good filling of the distally and posteriorly placed renal arteries. Indeed, a nephrogram is not usually seen during supine aortography. The experience of the follow-up of patients after CO<sub>2</sub> angiography at our institution encourages us to anticipate a low prevalence of post-CO<sub>2</sub> renal insufficiency. Moreover, we now have clinical experience in over 800 patients that have had CO<sub>2</sub> angiography (19, 20, 22-24). We have not seen evidence of clinically significant renal insufficiency that could be attributed to CO<sub>2</sub> injection. Moreover, other centers have not reported patients with renal toxicity secondary to CO<sub>2</sub> angiography (25-27).

However, these studies of the effects of CO<sub>2</sub> of renal function in patients were not rigorous. Therefore, to assess the renal toxicity of selective intra-arterial CO<sub>2</sub> injections, we undertook a renal toxicity study in which large volumes of CO<sub>2</sub> were injected directly into the renal arteries of 12 dogs (12). The contralateral kidney was used as a control. Nuclear medicine studies with [<sup>131</sup>I]-orthoiodohippurate (Hippuran) and [<sup>99m</sup>Tc]-DMSA were obtained before CO<sub>2</sub> was injected, immediately after, and 24 hours later. Light transmission and scanning electron microscopy of the kidneys was obtained at autopsy 72 hours after the injection of CO<sub>2</sub>. Overall, we detected a mean

decrease in renal blood flow of 6% immediately after the CO<sub>2</sub> was injected but no change from baseline by 24 hours. Histologic examination of the renal arterial and capillary endothelium with scanning electron microscopy, revealed no changes in any dog. However, changes of acute tubular necrosis were seen in one of the kidneys, and minimal ischemic changes were seen in the parenchyma of two kidneys. All of these kidneys were vertically oriented (Figure 5). Indeed, the animal that developed acute tubular necrosis had an especially good quality nephrogram, because the CO<sub>2</sub> may have been trapped in the kidney. This study was designed to subject the kidneys to a supramaximal CO<sub>2</sub> dose. The CO<sub>2</sub> was injected repeatedly every 2 minutes, which could lead to a prolonged "vapor lock" phenomenon preventing blood flow. However, if time is allowed between injections (as is routine clinical practice), the high solubility of the CO<sub>2</sub> should result in only transient renal ischemia. This theory is supported by the preliminary result of an ultrasound study in patients in which the kidney was imaged after injection of CO<sub>2</sub>. If the kidney was horizontal, the CO<sub>2</sub> bubbles cleared in approximately 30 seconds. However, if the kidney was vertical, approximately 2 minutes was required to clear CO<sub>2</sub> from the renal cortex (unpublished data; Hawkins). We conclude from this experience that intrarenal arterial injection of CO<sub>2</sub> appears to be quite safe, providing that time is allowed to clear CO<sub>2</sub> from the circulation between injections. In practice, the patient's position should be changed, or 2-5 minutes should elapse between injection to ensure that CO<sub>2</sub> does not get trapped in the kidneys and lead to ischemia.

#### *Neurotoxicity*

At the University of Florida, we do not use CO<sub>2</sub> in arterial studies above the diaphragm, since a preliminary study in rats suggested that CO<sub>2</sub> may be neurotoxic. We have, however, used CO<sub>2</sub> in the cerebral circulation in over 15 canines without any untoward reactions (22). Shifrin, et al. (29) have reported similar results in canines and cite absence of untoward reaction in patients. However, until CO<sub>2</sub> studies are available in primates, we hold that CO<sub>2</sub> is clearly contraindicated in any situation in which the cerebral circulation could be exposed. The report of neurotoxicity in the rat also raises concern about spinal cord toxicity. To explore this possibility, we injected large volumes of CO<sub>2</sub> into the aorta of 10 dogs in the prone position. No neurological deficit was noted. However, we continue to be reluctant to use CO<sub>2</sub> in the prone position, whereas it may percolate into spinal

arteries until it is tested systematically in primates.

#### *Respiratory Complications of CO<sub>2</sub> Angiography*

Intuitively, it seems that repeated injections of CO<sub>2</sub> should not be used in patients with severe pulmonary compromise. However, we have undertaken CO<sub>2</sub> angiography in 10 patients with severe respiratory failure, and have not detected any obvious increase in arterial blood pCO<sub>2</sub> levels after the studies. However, patients who are acidotic with elevated pCO<sub>2</sub> values require monitoring of arterial blood gases. In practice, the volume of CO<sub>2</sub> injected is quite small compared to the rates of endogenous CO<sub>2</sub> production.

#### *Discomfort*

The vast majority of patients experience no, or minimal, sensation during CO<sub>2</sub> injection with a controlled, dedicated device. With the more explosive delivery from a hand-held syringe, the injection of large amounts of CO<sub>2</sub> can give discomfort similar to that provided by injection of nonionic contrast agents. With the new stacking programs, only very small amounts of CO<sub>2</sub> need to be injected; this usually results in no sensation whatsoever.

#### *Other Complications of CO<sub>2</sub> Angiography*

We have experienced only one significant complication that was attributed to CO<sub>2</sub>. This was a patient who developed severe diarrhea after receiving over 200 ccs of CO<sub>2</sub> injected into a large abdominal aneurysm over a short time. The inferior mesenteric artery arose from this aneurysm, resulting in continual exposure to CO<sub>2</sub> from the trapped gas in the aneurysm. Apparently, the CO<sub>2</sub> which was trapped in the aneurysm, producing a "vapor lock" phenomenon, resulted in mesenteric ischemia. Endoscopy demonstrated the appearance of ischemic colitis. Fortunately, the diarrhea abated after 6 hours and endoscopy three days later was normal. We believe that this is the same mechanism that produced ischemia, in kidneys positioned vertically, in the canine study (12).

### CONCLUSIONS AND CURRENT RECOMMENDATIONS

In the past, the use of CO<sub>2</sub> has been extremely helpful in providing diagnostic studies in patients at high risk for nephrotoxicity because of renal insufficiency, and in those who have had a prior

severe contrast reaction. CO<sub>2</sub> occasionally provides additional information that cannot be obtained with iodinated contrast by visualizing collateral circulation, demonstrating AV shunting in malignant tumors, and detecting minute amounts of arterial bleeding. Occasionally, vascular malignant tumors can be detected with CO<sub>2</sub> that appear totally avascular with iodinated contrast. Also in the past, the delivery and imaging of CO<sub>2</sub> has been very demanding, time-consuming, and clearly not as reliable as iodinated contrast agents. With the development of more sophisticated programs for DSA and a user-friendly delivery system, as well as the use of tilt tables, the studies have become much more reliable and safer for the patient. The diagnostic accuracy is now similar to studies with conventional iodinated contrast. The apparent lack of renal toxicity warrants this additional effort in patients with renal insufficiency. Unfortunately, the automated delivery systems will not be commercially available for at least one year. However, CO<sub>2</sub> can be delivered by hand or with an angiographic injector. One must be familiar with the properties of CO<sub>2</sub>, and be prepared to obtain multiple views in several positions for accurate imaging. Very frequently, CO<sub>2</sub> alone may not provide all the necessary diagnostic information. However, the addition of a small amount of iodinated contrast medium will result in a diagnostic study with a reduced risk of renal toxicity.

It is important to emphasize that much of the information on CO<sub>2</sub> angiography has been obtained either from nonrigorous clinical studies or more detailed examinations in relatively small numbers of dogs. Therefore, a larger trial involving a more systematic and quantitative analysis of the accuracy, applicability, and toxicity of CO<sub>2</sub> compared to conventional contrast angiography, is required before the true place of this presently investigational agent can be securely stated.

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#### References:

1. Hessel S.J., Adams D.F., Abrams H.L. Complications of angiography. *Radiology* 1981; 138: 273-281

2. Cavo J.J., Trindade E., McGregor M. The risks of death and of severe nonfatal reactions with high- versus low-osmolality contrast media: a meta-analysis. *A.J.R.* 1991; 156:825-832.

3. Katayama H., Yamaguchi K., Kozuka T., et al. Averse reactions to ionic and nonionic contrast media. *Radiology* 1990; 175: 621-628.

4. Kinnison M.L., Powe N.R., Steinberg E.P. Results of randomized controlled trials of low-versus high- osmolality contrast media. *Radiology* 1989; 170: 381-389.

5. Smith H.J., Levorstad K., Berg K.J., et al. High dose urography in patients with renal failure: a double blind investigation of iohexol and metrizoate. *Acta Radiol* 1985; 26: 213-220.

6. Winfield A.C., Dray R.J., Kirchner F.K. et al. Iohexol for excretory urography: a comparative study. *A.J.R.* 1983; 141:571-573.

7. Cacayorin E.D., Bernstein A.D., Fruehan C.T., et al. Intravenous digital subtraction angiography with iohexol. *AJNR* 1983; 4: 329-332.

8. Berns A.S. Nephrotoxicity of contrast media. *Kidney Int.* 1989; 36: 730-740.

9. Lautin E.M., Freeman N.J., Schoenfeld A.H., et al. Radiocontrast-associated renal dysfunction: a comparison of lower-osmolality and conventional high-osmolality contrast media. *A.J.R.* July 1991; 157: 59-65.

10. Harris K.G., Smith T.P., Cragg A.H., et al Nephrotoxicity from contrast material in renal insufficiency: ionic versus nonionic agents. *Radiology* 1991; 179: 849-852.

11. Moore R.D., Steinberg E.P., Powe N.R. et al. Nephrotoxicity of high-osmolality versus low-osmolality contrast media: randomized clinical trial. *Radiology* 1992; 182: 649-655.

12. Hawkins I.F., Mladinich C.R.J., Storm B., et al. Short term effects of selective renal arterial CO<sub>2</sub> administration on the dog kidney. *J..V.I.R.* (in press).

13. Harvard B.M., White R.R., Walsh J.F. Experimental studies in acute retroperitoneal carbon dioxide insufflation. *J. Urol* 1959; 8: 481-485.

14. Paul R.E., Durant T.M., Oppenheimer M.J., Stauffer H.M. Intravenous carbon dioxide for intracardiac gas contrast in the roentgen diagnosis of pericardial effusion and thickening. *A.J.R.* 1957; 78: 224-225.

15. Turner A., Meyers H.I., Jacobson G., Lo W. Carbon dioxide cineangiography in the diagnosis of pericardial disease. *A.J.R.* 1966; 97: 342-9.

16. Bendib M.B.T., Toumi M., Boudjellab A. Carboxyangiographie et carboxyangiographie elargi en cardiologie. *Ann Radiol* 1977; 20: 673-686.

17. Moore R.M., Braselton C.W. Injections of air and carbon dioxide into a pulmonary vein. *Ann Surg* 1940; 112: 212-8.

18. Oppenheimer M.J., Durant D.M., Stauffer H., Stewart



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- G.H., Lynch P.R., Barrera F. Cardiovascular-respiratory effects and associated changes in blood chemistry. *Am J. Physiol* 1956; 186: 325-34.
19. Hawkins I.F. Carbon dioxide digital subtraction arteriography. *A.J.R.* 1982; 139: 19-24.
  20. Hawkins I.F., Akins E.W., Seeger J.M.: Carbon dioxide (CO<sub>2</sub>) as a contrast for angiography and angioscopy, in Ahn S.S., Moore W.S. (2nd ed): *Endovascular Surgery*, Philadelphia, W.B. Saunders, 1991, pp 473-480.
  21. Takeda T., Ido K., Nishimura G., Hashimoto S., Kyo E., Okawa S., Nakatsuka S., Miura H., Kobayashi S., Tanaka T., Hiramatsu K. Intraarterial digital subtraction angiography with carbon dioxide: superior detectability of arteriovenous shunting. *Cardiovasc Intervent Radiol* 1988; 11: 101-107.
  22. Hawkins I.F., Herrera M.A. Carbon dioxide has promise as an arterial contrast agent. *Diagn. Imag.* 1985;7: 82-84.
  23. Seeger J.M., Self S., Harward T.R., Flynn T.C., Hawkins I.F. Carbon dioxide gas as an arterial contrast agent. *Annals of Surgery*, Vol. 217, 1993; 6: 688-698.
  24. Harward T.R., Smith S., R.V.T., Hawkins I.F., Seeger J.M. Follow-up evaluation after renal artery bypass surgery with use of carbon dioxide arteriography and color-flow duplex scanning. *J. Vasc. Surg* 1993; 18: 23-30.
  25. Miller F.J., Mineau D.E., Koehler P.R., Nelson J.A., Luers P.D., Sherry R.A., Lawrence F.P., Anderson R.E., Kruger R.A. Clinical intraarterial digital subtraction imaging: use of small volumes of iodinated contrast material or carbon dioxide. *Radiology* 1983; 148: 273-278.
  26. Krasny V.R., Hollmann J.P., Gunther R.W. Erste erfahrungen mit CO<sub>2</sub> als gasformiges kontrastmittel in der DSA. *Fortachr Rontgenstr* 1988; 146,4: 450-454.
  27. Weaver F.A., Pentecost M.J., Yellin A.E., Davis S., Finck E., Teitelbaum G. *J. Vasc. Surg* 1991; 13: 266-273.
  28. Coffey R. Quisting R.G., Mickle J.P., Hawkins I.F., Ballinger W.B.: The cerebrovascular effects of intra-arterial CO<sub>2</sub> on quantities required for diagnostic imaging. *Radiology* 1984; 151: 405-410.
  29. Shifrin E.G., Plich M.B., Verstandig A.G., Gomori M. J. *Cardiovaac Surg* 1990; 31: 603-606.

**Table 1: Special Properties of Carbon Dioxide Relevant To Its Use in Angiography**

Special Property	Consequence
Low viscosity	<ul style="list-style-type: none"> <li>● Only Small catheter required for delivery</li> <li>● AV shunting seen in malignant tumors with CO<sub>2</sub></li> <li>● Shunting of agent through AV fistulae</li> <li>● Improved visualization of vascular tumors</li> <li>● Detection of minute degrees of arterial bleeding</li> <li>● Better collateral filling.</li> </ul>
Buoyancy	<ul style="list-style-type: none"> <li>● Selective filling of non-dependent vessels</li> <li>● Potential to miss filling of dependent vessels</li> <li>● Entrapment of CO<sub>2</sub> in elevated organs.</li> </ul>
High solubility	<ul style="list-style-type: none"> <li>● Rapid disappearance from arterial tree.</li> </ul>
Compressibility	<ul style="list-style-type: none"> <li>● Difficulty in providing controlled injection.</li> </ul>
Pulmonary Clearance	<ul style="list-style-type: none"> <li>● No recirculation</li> <li>● Limited exposure of kidney to agent even with direct intra-arterial injection.</li> </ul>

Summary of the major special physical and chemical properties of CO<sub>2</sub> and their consequences for its use as an angiographic agent. For further discussion, see text.

**Table 2: Summary of Relative Advantages and Disadvantages of CO<sub>2</sub> Compared to Conventional Iodinated Contrast Renal Angiography.**

Advantages of CO <sub>2</sub>	<ul style="list-style-type: none"> <li>● Low cost.</li> <li>● Low viscosity reduces the catheter size requirements.</li> <li>● Apparent absence of nephrotoxicity if "vapor lock" phenomenon is prevented .</li> <li>● No contraindication in patients with iodine allergy.</li> <li>● No recirculation allowing unlimited quantities to be injected.</li> <li>● Enhanced visualization of AV anastomoses and malformations, certain vascular tumors, or bleeding sites.</li> <li>● Minimal discomfort.</li> </ul>
Disadvantages of CO <sub>2</sub>	<ul style="list-style-type: none"> <li>● Inadequate diagnostic visualization in a small percentage of studies.</li> <li>● Potential for "vapor lock" ischemia, if repeated rapid injections are given and the patient is not properly positioned.</li> <li>● Can fail to image dependent renal arteries if the patient is not repositioned.</li> <li>● Distal renal arteries are difficult to image.</li> <li>● It produces a poor nephrogram.</li> <li>● Requires sophisticated DSA programming and preferably a dedicated injector.</li> <li>● May prolongs procedure time.</li> <li>● Potential, unexplored danger of neurotoxicity.</li> <li>● Potential danger of air embolization if CO<sub>2</sub> system is not hermetically sealed.</li> <li>● True benefits and toxicity yet to be rigorously evaluated in blinded, controlled trials.</li> <li>● Presently investigational.</li> </ul>

## The Amplatz Clot Macerator: Initial Clinical Experience

MARCOS A. HERRERA, M.D.<sup>1</sup>

The Amplatz clot macerator was used in twenty vessels in twelve patients for the mechanical removal of thrombus. Age range from 24 to 71 years; eight male and four female. Venous thrombosis was treated in eleven patients and arterial thrombosis in one. Of the eleven patients with venous thrombosis, seven were located to the subclavian vein; three to the axillary; two in the superior vena cava; two in the inferior vena cava; two in the femoral vein; and two in the pulmonary arteries. The patient with arterial involvement had thrombosis of a limb of an aortobifemoral bypass graft.

An 8-Fr device was used in all cases in combination with a 10-Fr Schneider guiding catheter.

By using an Iris adapter, contrast medium could be injected during the clot maceration while preventing blood loss. Manual suction was applied during the mechanical maceration. Total running time was, on average, three minutes.

Significant removal of thrombus was achieved in all patients (70-80%), with a residual narrowing of 20-30% in 80% of the patients, and of 40-50% in 20% of the patients. No major pulmonary emboli was detected by lung scans after the venous procedures. Angioplasty and/or surgical correction of the underlying anatomic problem was undertaken in all cases. The Amplatz macerator is an effective device for the removal of thrombus.

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## Transluminal angioplasty and effective thrombolysis with sodium pentosane polysulphate given orally

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**Abstract.** A case report of a 52-year-old female is presented whose recent arterial thrombosis had been traversed by a transluminal angioplasty Teflon catheter leaving only a narrow channel for blood flow. Transluminal dilatation was not performed because the recently developed thrombus showed only weak adherence to the vascular wall. During 10 days of sodium pentosan polysulphate treatment orally the clot resolved resulting in a well established distal flow. The case indicates a new opportunity to treat recently developed thrombi in the vascular system especially in those patients cases having some pathway for blood flow beside the clot.

**Key words:** Transluminal angioplasty – Thrombolysis – Fibrinolysis – Arterial thrombosis – Atherosclerosis – Orally induced thrombolysis – Sodium pentosane polysulphate

Transluminal angioplasty is an effective method of treating chronic arterial occlusions, but to recanalize recently occluded arteries has proved risky in terms of a high thromboembolic complication rate. In these cases selective thrombolysis has been the recommended treatment of choice. In some cases, however, selective lysis cannot be accomplished. In such cases vascular surgery has been the only alternative treatment.

Recommendations for oral fibrinolytic stimulants appeared in the literature in 1980 [6], used in combination with percutaneous transluminal angioplasty and some preliminary experiences were published even earlier [5]. The removal of remaining clots or atheromata after transluminal angioplasty with orally induced thrombolysis was the goal of our early investigations [7, 8, 9, 10]. Even with a two year old occlusion, the lower segment of the abdominal aorta and both common iliac arteries have been successfully cleared of deposits [10].

### Case report

A fiftytwo-year-old female presented with claudication and a walking distance of just over 200 meters. There was a slight murmur over the right femoral artery, with good pulsation on both sides. There were no palpable pulses below the femoral region on the left side. The Doppler-index was as low as 0.6 at rest on this side. At angiography a 1 cm long subtotal stenosis was observed in the proximal popliteal segment of the limb artery (Fig. 1). The patient was discharged and instructed to stop smoking, to follow a special exercise program in order to stimulate collaterals to develop and to take Trental 400<sup>®</sup> 2 x 1 dragees and Xavin<sup>®</sup> 3 x 1 tablets a day. Three months later the patient returned with a worsening of symptoms. Her walking distance was as short as 30 meters although the patient had stopped smoking 4 weeks prior to her return. A new angiogram revealed a 9.5 cm long occlusion of the left popliteal artery (Fig. 2). Transluminal angioplasty was attempted but abandoned because of the softness of the occluding clot. To recanalize with a catheter technique was considered too risky because of the lack of adherence of the clot to the vascular wall (Fig. 3). The patient refused surgical intervention. She was discharged from hospital on SP54<sup>®</sup> 150 mg orally twice a day at least one hour before meal, Trental 400<sup>®</sup> 1 dragee twice a day, Xavin<sup>®</sup> 3 times 1 tablet (150 mg xanthinon nicotinate per tablet) a day. She was instructed to continue her exercise regime. On the 10th day after the attempted angioplasty her leg became swollen and hyperemic with pain in the calf. After hospitalization on the 15th "postangioplasty" day angiography was performed with the surprising finding result of a clot-free, wide lumen of the previously occluded popliteal segment on the left side. This lumen was even wider than the neighbouring arterial segments showing that the intact, mechanically untouched deposits on the adjacent vessel wall has not been lysed (Fig. 4.-5.).

### Discussion

The first observations on sulphated polyanions (poly-saccharides) were published as early as 1949 [15]. These negatively charged substances may initiate separation between plasminogen activators and their inhibitors [3, 13] allowing availability for fibrinolytic activation. In some patients, however, no activation of the fibrinolytic system can be obtained by adding polyanions to the collected plasma in vitro [3]. This phenomenon can be encountered with human medication as well (our

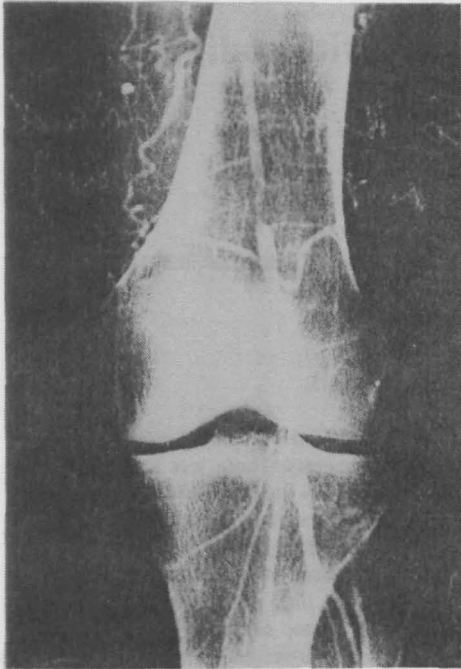


Fig. 1. Fiftytwo-year-old female, subtotal stenosis (1 cm) of the left popliteal artery

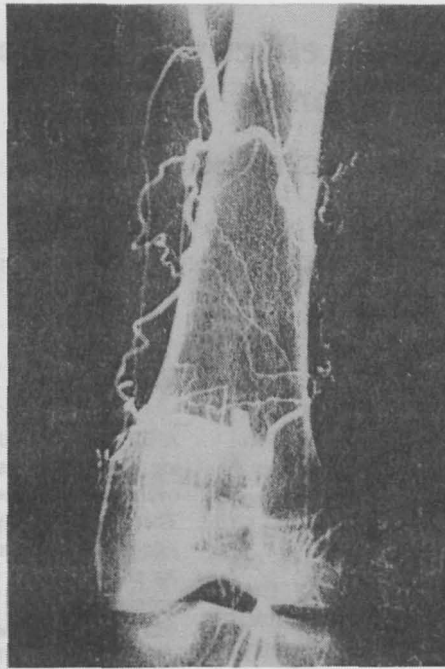


Fig. 2. Three months, the stenosis is a complete occlusion (9.5 cm)

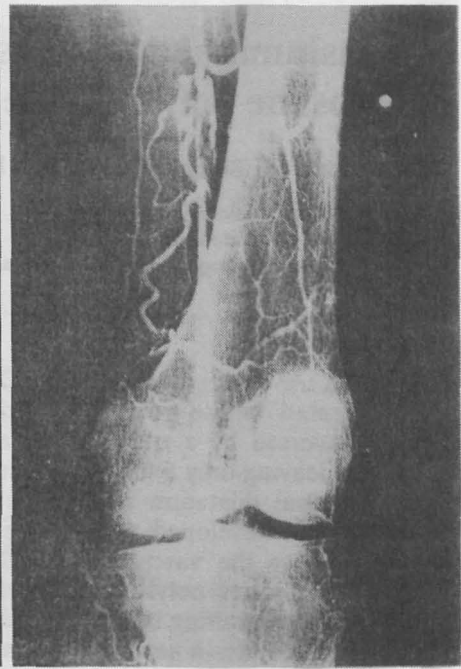
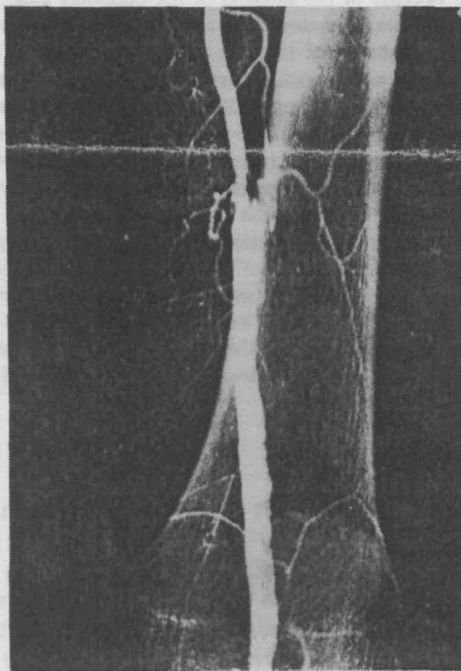
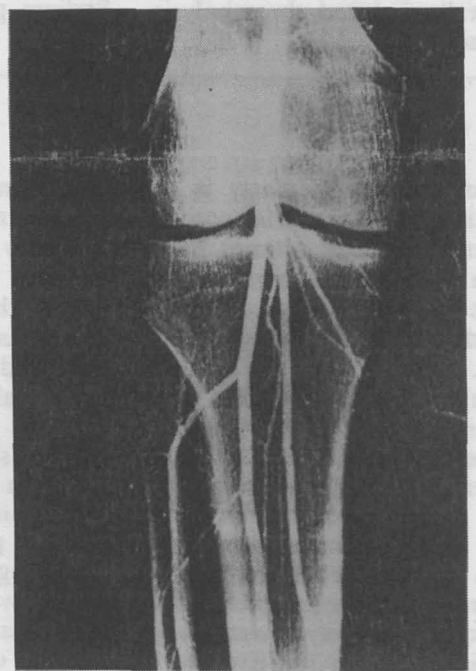


Fig. 3. During attempted transluminal angioplasty, occluding material easily traversed with guide wire and F5 Teflon catheter



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Fig. 4. and 5. Angiography, 15 days later, shows recanalisation

unpublished experience). The thrombolytic effect of sodium pentosan polysulphate (SP54) was experimentally established by Halse [4]. Administration of SP54<sup>R</sup> in man resulted in activation of fibrinolysis in whole plasma as well as in the euglobulin fraction, associated with lowering of antiplasmin activity [1]. SP54<sup>R</sup> stimulated lipolytic activity, and had moderate antiinflammatory effects [11]. The drug prevented lipid deposition in cholesterol-fed rabbits [2]. Pentosan polysulphate potentiates and prolongs nicotinic acid-induced fibrinolysis [14].

It might be expected that with orally induced thrombolytic stimulation an entirely new era will arrive with regard to arterial and perhaps venous thrombosis and their prevention. The effectiveness of such a medication is quite difficult to evaluate for lack of randomized studies so far. But earlier data [5–10, 12, 13, 15, 16] suggest that this phenomenon deserves the attention of interventional radiologists, angiologists or even vascular surgeons. The result of such a treatment may be striking especially if there is a pathway in the occluding clot made by catheter technique to give the chance to the enzymes to meet their counterparts, the fibrin molecules.

## References

1. Coccheri S, Genarri P, Loreti A (1963) L'attivazione della fibrinolisi mediante polisaccaridi acidi. Parte I. Aspetti biologici. *Clin Ter* 25:3–9
2. Coccheri S, Loreti A, Genarri P (1963) L'attivazione della fibrinolisi mediante polisaccaridi acidi. Parte II. Prospettive terapeutiche di un nuovo polisolfostere del pentoano. *Clin Ter* 25:103–112
3. Coccheri S, De Rosa V, Cavallaroni K, Poggi M (1978) Activation of fibrinolysis by means of sulphated polysaccharides. Present status and perspectives. In: Davidson JF, Rowan RM, Samama MM, Desnoyers PC (eds) *Progress in Chemical Fibrinolysis and Thrombolysis*. Raven Press, New York, pp 461–473
4. Halse Th (1954) Polyelectrolytes as activators of fibrinolysis and thrombolysis. *Sangre, Barcelona* 9:149–164
5. Horváth L, Illés I, Varró J (1978) Complications of transluminal angioplasty excluding the puncture site complications. In: Zeitler E, Grüntzig A, Schoop W (eds) *Percutaneous Vascular Recanalization*. Springer, Berlin Heidelberg New York, pp 126–139
6. Horváth L (1980) Percutaneous transluminal angioplasty: importance of anticoagulant and fibrinolytic drugs. *Am J Roentgenol* 135:951–954
7. Horváth L, Illés I, Fendler K, Herzfeld I (1982) Adjuvant long-term treatment during and following percutaneous transluminal angioplasty. In: Veiga-Pires JA (ed) *Intervention Radiology 2*. Excerpta Medica, Amsterdam, pp 347–352
8. Horváth L, Illés I, Fendler K, Herzfeld I (1982) Pharmaceutical therapy as a major part of percutaneous transluminal angioplasty. *Ann Radiol* 25:489–490
9. Horváth L (1983) Results with additional use of SP54. In: Dotter CT, Zeitler E, Grüntzig A (eds) *Percutaneous Transluminal Angioplasty*. Springer, Berlin Heidelberg New York Tokyo, pp 95–97
10. Horváth L, Rauth J, Fendler K, Molnár Z, Romhányi M (1985) Behandlung des Leriche-Syndroms mit Dotter-Angioplastie und oraler Fibrinolyse. In *Ungarisch. Orv Hetil* 126:641–656
11. Kalbhen DA (1973) Pharmacological studies on the anti-inflammatory effect of a semisynthetic polysaccharide (pentosan polysulphate). *Pharmacology* 9:74–81
12. Kollár L, Rozsos I, István M, Kiss T (1989) Pentosan-polysulfat bei peripherer arterieller Verschlusskrankheit im Stadium II. nach Fontaine. *Perfusion* 2:52–56
13. Marsh N, Gaffney P (1984) Stimulation of fibrinolysis by the heparinoid pentosan polysulphate (SP54). A proposal for the mechanism. VIII. Thrombosis Congress Istanbul, June 4–7
14. Menghini P, Poggio (1974) Thrombolytic therapy with non-enzymatic indirect agents. *Minerva Cardioangiol* 22:101 (Abstract)
15. Ungar G, Mist SH (1949) Observations on the release of serum fibrinolysis by specific antigen peptone and certain polysaccharides. *J Exp Med* 90:39–46
16. Vinazzer H, Haas S, Stemberger A (1980) Influence on the clotting mechanism of sodium pentosan polysulphate (SP54) in comparison to commercial beef lung sodium heparin. *Thromb Res* 20:57–68

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## Consideraciones sobre la utilización de Stent y By-pass en los Sectores Aortoiliaco y Femoropoplíteo

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La evolución y a veces la "involución" de la Cirugía Vascul ar en la últimas décadas ha traído como consecuencia modificar técnicas de revascularización en determinados sectores arteriales. Concretamente, en los sectores aortoiliaco y femoropoplíteo hemos vivido considerables innovaciones en relación con las técnicas utilizadas.

La década de los 70 se vio influenciada por las extensas endarterectomías, con excelentes resultados en el sector aortoiliaco y muy deficientes en el femoropoplíteo. La complejidad de la técnica y la elevada morbimortalidad especialmente en el sector aortoiliaco hizo abandonar prácticamente esta técnica.

Desde comienzo de los 80, los by-pass femoropoplíteos con vena safena interna invertida y los aortobifemorales, con material sintético vinieron a constituir las técnicas más empleadas. La incorporación de los by-pass femoro-femorales abrió otra alternativa para la resolución de las lesiones aortoiliacas, así como los by-pass "in situ", para las lesiones distales.

Las técnicas de radiología intervencionista surgieron como terapéutica alternativa y como generalmente ocurre en todo tratamiento revolucionario, se ampliaron excesivamente sus indicaciones. La experiencia adquirida por los angi radiólogos en los últimos años, esta consiguiendo situar estas posibilidades terapéuticas en su justo lugar.

Las angioplastias transluminales y los "stents" han contribuido a ampliar, modificar y mejorar la revascularización en estos sectores. Concretamente en el sector aortoiliaco el "stent" encuentra a nuestro entender, su máxima indicación, fundamentalmente en las lesiones estenosantes u obliterantes segmentarias de iliaca común y en ocasiones como complemento de técnicas

derivativas: by pass femoro-femoral o femoropoplíteo, ante la presencia de lesiones combinadas.

En el sector femoropoplíteo, somos más escépticos en la indicación de "stent", prefiriendo el by-pass con vena safena interna invertida.

En la siguiente Tabla, establecemos las indicaciones de revascularización en los sectores aortoiliaco y femoropoplíteo:

SECTOR AORTOILIACO	SECTOR FEMOROPOPLITEO
• By-Pass material sintético	By-Pass vena safena interna invertida
• Stent	By-Pass "in situ"
• Stent iliaco + By-Pass femoro-femoral	
• Stent iliaco + By-Pass femoropoplíteo	

La estrecha colaboración de angi radiólogos con cirujanos vasculares contribuirá a mejorar la valoración en conjunto de las lesiones arteriales y establecer aquella terapéutica que implique los mejores resultados inmediatos y tardíos.

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## Critical Analysis of Peripheral Arterial Thrombolysis

KRISHNA KANDARPA, M.D.

The goal of intraarterial thrombolytic therapy (ITT) is to rapidly restore blood flow to the ischemic limb and to identify underlying obstructive lesions for treatment by surgical and/or percutaneous techniques. ITT is gaining wider acceptance by referring physicians as its role in the treatment of peripheral arterial occlusions (PAO) is clarified in terms of: 1) patient selection by clinical and arteriographic criteria (1-3), and 2) sites and types of conduits amenable to treatment -The present discussion will be limited to the use of ITT in lower extremity arterial ischemia where there is most experience. However, experience with upper extremities (8), dialysis fistulas (9,10), and coronary & cerebral vessels is growing-, eg., arteries of the extremities, bypass grafts, etc. (4-10). Thrombotic or embolic arterial occlusions causing worsening claudication or limb-threatening ischemia are amenable to ITT. Both acute and chronic thrombi have been successfully lysed (2,3,6). Absolute contraindications to ITT include patients with irreversibly -SVS Category III ischemia- ischemic limbs (2), active internal bleeding, intracranial neoplasms, and recent stroke or craniotomy.

Although ITT has a high success -clinical success is defined as complete thrombus lysis and improvement in clinical status- rate (about 85 % 90% initial clinical success rate for urokinase) and acceptably low rate for significant complications (eg., major bleeding <3%, and death <2%) (2,5,6), the treatment is resource-intensive and expensive. Major contributors to the cost of therapy are expensive lytic agents, the requisite serial angiographic follow-up, and the near universal practice of intensive care (ICU) monitoring during prolonged enzyme infusion. In addition, morbidity increases with duration of therapy (11). None of the clinically used lytic agents can differentiate between an occlusive thrombus causing ischemia and one that is formed in response to vascular injury (e.g., at a puncture site); therefore, bleeding complications are an inherent problem with thrombolytic therapy, albeit

to varying degrees. Attempts to make this treatment easier on the patient and physician center around reducing the duration of therapy by modifying dosing technique (11), using faster acting enzymes (12,13), infusing enzymes more efficiently (14,15), and minimizing or even eliminating ICU monitoring (16).

At the present time, urokinase (UK) is preferred over streptokinase (SK) because of it has a lower incidence of complications (UK vs. SK: amputations, 8% vs. 16%, major bleeding 3% vs. 11%, and death, 1.6% vs. 1.9%) and greater efficacy (initial clinical success rate of 90% for UK vs. 60% for SK). In addition, McNamara (1) has reported a significant shortening of the mean duration of treatment with UK (18 hours, versus 42 hours for SK). This is confirmed by the experience of others as well (4,5,11). Some of these differences may be attributable to the pharmacokinetics of these drugs (17). Streptokinase has to form a complex with a molecule of plasminogen before it can convert a second molecule of plasminogen to plasmin, the enzyme which lyses fibrin. Thus, streptokinase depletes plasminogen (the substrate needed for fibrinolysis) twice as rapidly as urokinase or tissue-plasminogen activator (rt-PA), both of which directly activate plasminogen. This may account for the longer duration of treatment with SK. Streptokinase also causes a more rapid and profound depletion in fibrinogen than does UK or rt-PA. Fibrinogen is needed for the production of fibrin, the main component of the hemostatic plug which prevents hemorrhage. Finally, there are two important consequences to the fact that SK is a bacterial protein. First, patients exposed to prior streptococcal infections may have antibodies which may cause hypersensitivity reactions during SK infusion or, if present in high titres, may reduce the efficacy of thrombolysis with this drug. Second, patients exposed to SK, which is antigenic, develop antibodies to it early during treatment and may require up to six months before they can be reexposed to it (18). Several recent studies which have considered the risks and benefits of ITT with



UK versus SK have concluded that treatment of peripheral arterial occlusions with urokinase is safer and more cost-effective (7,19-21), although UK is the more expensive drug.

Presently, urokinase (UK) is also preferred over rt-PA for ITT in the lower extremities, although initial clinical success rates are similar, because UK has lower reported incidence of complications (UK vs. rt-PA: amputations, 8% vs. 17%, major bleeding 3% vs. 10-15%, and death, 1.6% vs. 4-10%). Tissue-plasminogen activator has an increased affinity to fibrin-bound plasminogen (17,18), but it can also convert circulating plasminogen to plasmin at much slower rates. This fibrin-specificity was thought to endow rt-PA with a more rapid lytic effect. In an early study using rt-PA for thrombolysis in peripheral artery and bypass graft occlusions, Graor et al. (12) reported a 92% success rate for lysis of peripheral thrombotic occlusions (of mean duration < 6.5 days) with a mean lysis time of 3.9 hours, and no major complications! However, a recent randomized comparison (13) of rtPA to urokinase for treatment of peripheral arterial and graft occlusions (of mean duration < 12 days) showed that although lysis progressed more rapidly with rtPA, there was no significant difference either in the number of patients achieving complete lysis at 24 hours or in the clinical outcome at thirty-days between the two matched groups. Furthermore, there was a significantly greater suppression of fibrinogen levels in the rtPA group with more major bleeding complications. The rtPA dosing was similar in the two studies, but preliminary transthorbus bolusing and concomitant IV heparin anticoagulation were employed in all patients in the randomized trial (13). A recent study which specifically compared the relative risk of stroke with different lytic agents in patients being treated for acute myocardial infarction found that, although the incidence of stroke was not increased with thrombolysis per se, rt-PA had an excess of strokes in comparison with streptokinase (22). This latter finding is in agreement with the findings of the ISIS-3 study (23). The higher complication rates generally reported with rt-PA may ironically be related to its fibrin-specificity and its inability to differentiate between pathologic and physiologic thrombus.

More recently, Graor et al. (24) reported the findings of a retrospective study which compared the efficacy and safety of local intraarterial infusion of SK (n=200), UK (n=200), or rt-PA (n=65) for peripheral arterial occlusions. Results were as follows: 1) complete thrombolysis with clinical

improvement: SK = 60%, rt-PA = 91%, and UK = 95%; 2) major bleeding: SK = 28%, rt-PA = 12%, and UK = 6%; 3) intracranial hemorrhage: SK = rt-PA = 2%, UK = none; and 4) death: SK = 4%, rt-PA = 2%, UK = none. Their obvious conclusion from this study was that *UK was the safest and most efficacious of the three drugs compared*. The question of whether rt-PA or any of the newer and more expensive lytic agents (eg., pro-UK or Scu-PA, APSAC) will, with further accumulated experience, replace UK, as UK has replaced SK, is as yet unsettled. Cost-effectiveness studies comparing UK to rt-PA are awaited.

Methods for more efficient use of enzyme include preliminary bolusing of the entire thrombus prior to the initiation of infusion (11), optimization of treatment doses (12,25,26), use of several new catheter systems which bathe the thrombus more effectively during infusion (27-29), and forced infusion of the enzyme into the thrombus (pulsed-spray) in order to mechanically disrupt the clot and to create a greater surface area for enzyme action (30,31). In a retrospective comparison of two groups of patients receiving two different transthorbus bolus doses (52K IU vs. 230K IU) but similar infusion regimens, Sullivan et al. (11) reported that with high-dose bolusing the time to completion of thrombolysis was significantly reduced (from 33.6 hrs to 10.4 hrs.). In contradistinction, in a randomized prospective trial comparing two dose regimens of urokinase (50K IU bolus plus 50K IU/hr for 24 hours versus 250K IU bolus plus 250K IU/hr four hrs. and then 125K IU/hr for 20 hours), Cragg et al. (25) found no significant differences between the two groups of patients, except for higher costs associated with the higher dose of UK used. Similarly, a study by Berridge et al. (26) used rtPA for thrombolysis of peripheral arteries at a much lower dose (0.5 mg/hr) than Graor et al. (3.5-7.0 mg/hr) (12), on occlusions of longer duration (mean = 17 days) with a reported mean duration of treatment of 29 hours, but with comparable clinical outcome. They concluded that low-dose rtPA was as clinically effective (albeit slower acting) as the high-dose regimens reported, with the additional benefit of reduced systemic lytic effects. Although the optimal dose for neither UK nor rtPA has been firmly established, these studies suggest that larger doses are not necessarily more clinically efficacious.

In earlier animal experiments, we (15) and others (14) had shown that forceful infusion of enzyme into the thrombus accelerates thrombolysis. We have recently completed a randomized prospective

clinical trial comparing conventional slow infusion to forced intrathrombotic infusion for treatment of PAO (31). Initially both groups of patients received intra-thrombus spray-bolusing of UK through a tip-occluded multi-sideslit (pressure responsive orifice) catheter (AngioDynamics, Glens Falls, NY), but then one group (n=13) received slow continuous-infusion (IMED infusion pump, San Diego, CA) while the other (n=12) continued to receive spray-infusion (prototype pulse-spray injector, AngioDynamics, Glens Falls, NY) for the four hour duration of the protocol. We were able to achieve antegrade flow and significant lysis ( $\geq 95\%$ ) of the thrombus in the majority of patients on both groups in under four hours. However, the difference in total treatment time (100% lysis of thrombus with clinical improvement) between the two groups was not only not statistically significant, but it was also longer by a factor of ten compared with the total treatment time (under two hours) reported by others (30). When the two groups of our study were combined and the results of total treatment times were compared to our own recently documented historical record (13), we found that a significantly greater number of the study patients (78%) were completing their treatment within twenty-four hours than our patients previously did. We attribute this shortening to the aggressive initial spray bolusing which both groups in this study received. Restoration of brisk antegrade flow may reduce the effectiveness of continued pulse-spray infusion, perhaps accounting for the lack of a significant difference in total treatment time. In addition, pulse-spray infusion was as safe as conventional infusion in our study. Based on these findings, we would recommend early and aggressive pulse-spray bolusing of the thrombus by the enzyme. It should be noted that pulse-spray thrombolysis appears to be quite effective in reducing treatment time of thrombosed dialysis fistulae where thrombi are usually acute and detected early (9,10,30).

Controversies in thrombolytic therapy as elsewhere in interventional radiology stem from lack of information or, as is often the case, the inability to directly compare studies. Factors such as differences in initial thrombus burden might account for some of the difference in reported treatment times. Some of the differences are due to initial study design (eg., patient selection criteria, equivalence of lytic activity of drug doses chosen, treatment endpoints, etc.), but others are in standards of practice (eg., completion of treatment does not necessarily correspond to total lysis of thrombus). In other words, whereas one

practitioner may accept residual thrombus at the end of treatment (32), another may continue infusion overnight in order to eliminate all thrombus (29). We favor the latter practice because of the preponderance of clinical evidence, especially in the literature on thrombolysis for acute myocardial infarction, that residual thrombus is itself highly thrombogenic (33,34), and that thrombolysis itself may be associated with in vivo activation of the coagulation system (35-37).

The use of concomitant intravenous heparin during urokinase thrombolysis has reduced the incidence of pericatheter thrombus formation to about 5%. In fact one of the most distressing set-backs during therapy is caused by rethrombosis due to inattention to maintaining therapeutic anticoagulation levels. An IV bolus of heparin of 70mg/kg followed by continuous infusion at 700-1000 units/hr to maintain a PTT between 80-100 seconds is recommended. However, the anticoagulation achieved in any individual with a given dose of heparin is highly variable (38). Therefore, we have recently shifted to following activated clotting times and maintaining them at 250-300 seconds during therapy.

Although we anticoagulate all of our patients intravenously with heparin, we have not explored the benefits of mixing heparin directly to the UK solution as has been recently advocated (32). Although, admixing heparin may be of benefit in preventing rethrombosis in acute platelet-rich arterial thrombi as would be found in dialysis fistulae, there may be no such benefit in older arterial thrombi which are generally devoid of platelets. In addition, once flow is reestablished systemic heparinization should prevent rethrombosis. LeBlang et al. (39), have recently reported that they found no significant difference in outcome whether the heparin was given intravenously or whether it was given directly into the intraarterial sheath. We have also not studied the benefits of suction-thrombectomy for "debulking" thrombus (40). In our forced-infusion study, we have limited our pulsing rate to two per minute, and there may be a benefit to increasing this rate at least during the initial bolusing (41). Recently, there have been experimental reports on the acceleration of thrombolysis by the addition of local ultrasonic energy (42), but these ideas need to be tested clinically.

The ultimate goal of accelerated thrombolysis should be to shorten the entire treatment time, including adjunctive angioplasty if necessary, to

under two hours. Long-term efficacy of this treatment depends on detecting and correcting underlying obstructive lesions (5,43). One group has reported that patients undergoing thrombolysis may be monitored safely outside of an intensive care setting (16). We do not recommend this practice presently, but if it is found to be universally safe, a considerable cost reduction will be realized even if the goal of single-session therapy is not achieved in the near future.

### References

1. McNamara T.O., Fisher J.R. Thrombolysis of Peripheral Arterial and Graft Occlusions: Improved results using high-dose urokinase. *AJR* 1985; 144: 769-775.
2. McNamara T.O., Bomberger R.A., Merchant R.F. Intra-Arterial Urokinase as the Initial Therapy for Acutely Ischemic Limbs. *Circulation* 1991; 83 (s1): I-106 - I-109.
3. Lupatelli L., Barzi F., Corneli P., et al. Selective Thrombolysis with Low-dose Urokinase in Chronic Arteriosclerotic Occlusions. *Cardiovasc Intervent Radiol* 1988; 11: 123-126.
4. Gardiner G.A., Koltun W., Kandarpa K., et al. Thrombolysis of Occluded Femoropopliteal Grafts. *AJR* 1986; 146: 831-833.
5. Sullivan K.L., Gardiner G.A., Kandarpa K., et al. Efficacy of Thrombolysis in Infringuinal Bypass Grafts. *Circulation* 1991; 83 (s1): I-99 - I-105.
6. Motarjeme A. Thrombolytic Therapy in Arterial Occlusion and Graft Thrombosis. *Seminars in Vascular Surgery* 1989; 2: 155-178.
7. Traugher P.D., Cook P.S., Micklos T.J., Miller F.J. Intraarterial Fibrinolytic Therapy for Popliteal and Tibial Artery Obstruction: Comparison of Streptokinase to Urokinase. *AJR* 1987; 149: 543-456.
8. Lambiasi R.E., Paoletta L.P., Haas R.A., Dorfman G.S. Extensive Thromboembolic Disease of the Hand and Forearm: Treatment with Thrombolytic Therapy. *JVIR* 1991; 2: 210-208.
9. Brunner M.C., Matalon T.A., Patel S.K., et al. Ultrarapid Urokinase in Hemodialysis Access Occlusion. *JVIR* 1991; 2: 503-506.
10. Kumpe D.A., Cohen M.A.H., Durham J.D. Treatment of Failing and Failed Hemodialysis Access Sites: Comparison of Surgical Treatment with Thrombolysis/Angioplasty. *Semin -Vasc Surgery* 1992; 5: 118-127.
11. Sullivan K.L., Gardiner G.A., Shapiro M.J., et al. Acceleration of Thrombolysis with a High-dose Transthrombus Bolus Technique. *Radiology* 1989; 173: 805-808.
12. Graor R.A., Risius B., Lucas F.V., et al. Thrombolysis with Recombinant Human Tissue-type Plasminogen Activator in Patients with Peripheral Artery and Bypass Graft Occlusions. *Circulation* 1986; 74 (s1): I-15 - I-20.
13. Meyerovitz M.F., Goldhaber S.Z., Reagan K., et al. Recombinant Tissue-type Plasminogen Activator versus Urokinase in Peripheral Arterial and Graft Occlusions: A Randomized Trial. *Radiology* 1990; 175: 75-78.
14. Valji K., Bookstein J.J. Fibrinolysis with Intrathrombic Injection of Urokinase and Tissue-type Plasminogen Activator: results in a new model of subacute venous thrombosis. *Invest Radiol* 1987; 22: 23-27.
15. Kandarpa K., Drinker P.A., Singer S.J., Caramore D. Forceful Pulsatile Local Infusion of Enzyme Accelerates Thrombolysis: *in vivo* evaluation of a new delivery system. *Radiology* 1988; 168: 739-744.
16. Routh W.D., Tatum C.M., Barton R.E., et al. Urokinase Infusion: Feasibility of Monitoring for Complications in a Non-Intensive Care Setting. *JVIR* 1991; 2: 69-72.
17. Holden R.W. Plasminogen Activators: Pharmacology and Therapy. *Radiology* 1990; 174: 993-1001.
18. Marder V., Sherry S. Thrombolytic Therapy: Current Status. *N Engl J Med* 1988; 318: 1512-1520.
19. van Breda A., Graor R.A., Katzen B.T., et al. Relative Cost-Effectiveness of Urokinase versus Streptokinase in the Treatment of Peripheral Vascular Disease. *JVIR* 1991; 2: 77-87.
20. Janosik J.E., Bettmann M.A., Kaul A.F., Souney P.F. Therapeutic Alternatives for Subacute Peripheral Arterial Occlusion: Comparison by outcome, length of stay, and hospital charges. *Invest Radiol* 1991; 26:921-925.
21. Belkin M., Belkin B., Buckman C.A., et al. Intra-arterial Fibrinolytic Therapy: Efficacy of Streptokinase vs. Urokinase. *Arch Surg* 1986; 121: 769-773.
22. Vaitkus P.T., Berlin J.A., Schwartz S., Barnathan E.S. Stroke Complicating Acute Myocardial Infraction. *Arch Intern Med* 1992; 152: 2020-2024.
23. ISIS-3: A Randomized Comparison of Streptokinase vs. Tissue Plasminogen Activator vs. Anistreplase and of Aspirin plus Heparin vs. Aspirin alone among 41,299 cases of Suspected Acute Myocardial Infraction. *The Lancet* 1992; 339: 753-770.
24. Graor R.A., Olin J, Bartholomew J.R., et al. Efficacy and Safety of Intraarterial Local Infusion of Streptokinase, Urokinase, or Tissue Plasminogen Activator for Peripheral Arterial Occlusion: A Retrospective Review. *J Vasc Med Biol* 1990; 2: 310-315.
25. Cragg A.H., Smith T.P., Corson J.D., et al. Two Urokinase Dose Regimens in Native Arterial and Graft Occlusions: Initial Results of a Prospective, Randomized Clinical Trial. *Radiology* 1991; 178: 681-686.
26. Berridge D.C., Gregson R.H.S., Makin G.S., et al. Tissue Plasminogen Activator in Peripheral Arterial Thrombolysis. *Br J Surg* 1990; 77: 179-182.
27. Hicks M.E., Picus D., Darcy M.D., Kleinhoffer M.A. Multilevel Infusion Catheter for use with Thrombolytic

#### IV INTERNATIONAL COURSE ON VASCULAR AND INTERVENTIONAL RADIOLOGY AS THERAPEUTIC ALTERNATIVE

- Agents. *JVIR* 1991; 2: 73-75.
28. Mewissen M.W., Minor P.L., Beyer G.A., Lipchik E.O. Symptomatic Native Arterial Occlusions: Early Experience with "Over-the-wire" Thrombolysis. *JVIR* 1990; 1: 43-47.
  29. McNamara T.O. The Use of Lytic Therapy with Endovascular "Repair" for the Failed Infrainguinal Graft. *Seminars in Vascular Surgery* 1990; 3: 56-65.
  30. Valji K., Roberts A.C., Davis G.B., Bookstein J.J. Pulsed-spray Thrombolysis of Arterial and Bypass Grafts Occlusions. *AJR* 1991; 156: 617-621.
  31. Kandarpa K., Chopra P.S., Aruny J.E., et al. Intraarterial Thrombolysis of Lower Extremity Occlusions: Prospective, Randomized Comparison of Forced Periodic Infusion and Conventional Slow Continuous Infusion. *Radiology* 1993; 188: 861-867.
  32. Valji K., Bookstein J.J., Roberts A.C., Sanchez R.B. Occluded Peripheral Arteries and Bypass Grafts: Lytic Stagnation as an End Point for Pulse-Spray Pharmacomechanical Thrombolysis. *Radiology* 1993; 188: 389-394.
  33. Fuster V., Stein B., Ambrose J.A., et al. Atherosclerotic Plaque Rupture and Thrombolysis: Evolving Concepts. *Circulation* 1990; 82(suppl II): 47-59.
  34. Arnout J., Simoons M., de Bono D., et al. Correlation Between Level of Heparinization and Patency of the Infarct-Related Coronary Artery After Treatment of Acute Myocardial Infarction with Alteplase (rt-PA). *J Am Coll Cardiol* 1992; 20: 513-519.
  35. Owen J., Friedman K.D., Grossman B.A., et al. Thrombolytic Therapy with Tissue Plasminogen Activator and Streptokinase Induces Transient Thrombin Activity. *Blood* 1988; 72: 616-620.
  36. Fitzgerald D.J., Catella F., Roy L., Fitzgerald G.A. Marked Platelet Activation in vivo after Intravenous Streptokinase in Patients with AMI. *Circulation* 1988; 77: 142-150.
  37. Baglin T.P., Luddington R., Jennings I., Richards E.M. Thrombin Generation and Myocardial Infarction during Infusion of tissue-Plasminogen Activator. *The Lancet* 1993; 341: 504-505.
  38. Rath B., Bennett D.H. Monitoring the Effect of Heparin by Measurement of Activated Clotting Time during and after PTCA. *Br Heart J* 1990; 63: 18-21.
  39. LeBlang S.D., Becker G.J., Benenati J.F., et al. Low-dose Urokinase Regimen for the Treatment of Lower Extremity Arterial and Graft Occlusions: Experience in 132 cases. *JVIR* 1992; 3: 475-483.
  40. Starck E.E., McDermott J.C., Crummy A.B., et al. Percutaneous Aspiration Thromboembolectomy. *Radiology* 1985; 156: 61-66.
  41. Kandarpa K., Drinker P.A., Caramore D., Singer S.J. Accelerated thrombolysis with pulsatile infusion of enzyme: effect of frequency of pulsation and age of thrombus. *Joint Meeting of CIRSE and SCVIR, Berlin, West Germany, June 15, 1988 (abstr.)*.
  42. Tachibana K. Enhancement of Fibrinolysis with Ultrasound Energy. *JVIR* 1992; 3: 299-303.
  43. McNamara T.O., Bomberger R.A. Factors Affecting Initial and 6 Month Patency Rates after Intraarterial Thrombolysis with High Dose Urokinase. *Am J Surg* 1986; 152: 709-712.

## Interventional Radiology

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## Intraarterial Thrombolysis of Lower Extremity Occlusions: Prospective, Randomized Comparison of Forced Periodic Infusion and Conventional Slow Continuous Infusion<sup>1</sup>

A prospective randomized controlled trial compared forced infusion (FI) of urokinase (UK) with conventional slow continuous infusion (CI) in 25 patients with 25 acutely ischemic lower limbs. Demographics, ischemia categories, and infusion rates and doses were similar for both groups. A preliminary single-pass bolus of UK was injected into the thrombus in all patients with a pulsed-spray technique, and heparin was administered. UK was then infused with a CI pump ( $n = 13$ ) or a prototype pulsed-spray pump ( $n = 12$ ). The primary end point was patency, defined as at least 95% thrombolysis by volume, with brisk antegrade flow occurring within 4 hours. Eleven of the 12 patients (92%) who underwent FI and nine of the 13 (70%) who underwent CI had patency within 4 hours. However, 10 patients who underwent FI and nine who underwent CI had residual thrombi prolonging infusion. No significant differences between the two groups were apparent in speed of lysis, initial success rates, complication rates, or 30-day clinical outcome. Lytic therapy, however, was completed within 24 hours in 18 of 23 (78%) successfully treated patients ( $P = .01$ ).

**Index terms:** Arteries, peripheral, 92.7214 • Arteries, stenosis or obstruction, 92.7214 • Arteries, transluminal angioplasty, 92.1274 • Grafts, stenosis, 92.452 • Thrombolysis, 92.1274 • Urokinase, 92.1274

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LOCAL intraarterial thrombolytic therapy is useful for restoring blood flow relatively rapidly to an ischemic limb and for identifying underlying obstructive lesions for further treatment. Although regional thrombolysis with urokinase (UK) has a high initial success rate and an acceptably low rate of major complications (1-3), the treatment has remained resource-intensive and expensive. Major contributors to cost are the lytic agents, the requisite serial angiographic follow-up, and the near universal practice of monitoring in the intensive care unit during prolonged enzyme infusion. In addition, morbidity increases with duration of therapy (4). Attempts to make this procedure easier on all concerned center on reducing the duration of therapy by modifying dosing technique (4), infusing enzymes more efficiently (5,6), using faster acting enzymes (7,8), and minimizing or even eliminating intensive care monitoring (9).

Because conventional (slow) local infusion techniques do not deliver optimal concentrations of lytic enzymes into the thrombus where clot-bound plasminogen can be converted into plasmin, methods for more efficient use of enzymes have evolved and include (a) preliminary "lacing" of the entire thrombus (intrathrombus bolus technique) before initiation of slow infusion (4), (b) use of one of several new catheter systems that slowly infuse the entire length of thrombus more effectively (10-12), and (c) frequent periodic forced infusion (FI) (with a pulsed-spray technique) of the lytic enzymes into the thrombus,

which mechanically disrupts the clot and creates a greater surface area for enzyme action (13,14).

In 1989, Bookstein et al (15) used UK for pulsed-spray thrombolysis and reported mean times for completion of lysis in 10 peripheral arterial bypass grafts and seven native arteries of 91 and 75 minutes, respectively. Recently, the same group (13) confirmed these results, reporting mean times for completion of lysis in 18 bypass grafts and 15 native arteries of 93 and 65 minutes, respectively. The data were analyzed retrospectively in these uncontrolled studies. Those lysis times are dramatically shorter than the 24-72 hours generally reported in the literature (16-20). Because such reduction in total treatment time could have a profound influence on the further acceptance and utilization of thrombolysis for the initial recanalization of ischemic limbs, we designed the following prospective randomized, controlled study. The purpose of our study was to determine whether, after administration of a preliminary single-pass pulsed-spray lacing (bolus) of UK into the thrombus, continued FI results in a safe and substantial acceleration of thrombolysis when compared with conventional slow continuous infusion (CI).

### MATERIALS AND METHODS

This study was conducted with the approval of our institution's Human Research Committee. From January 1, 1990, through July 31, 1992, we performed thrombolysis in 75 lower extremities. Some of these patients and/or their referring physicians elected not to participate in the research protocol, while others were excluded on the basis of the criteria listed below. We were able to obtain informed consent from 27 patients who met the in-

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**Abbreviations:** CI = continuous infusion, FI = forced infusion, UK = urokinase.

clusion criteria. One patient withdrew consent before undergoing angiography, and one patient was eliminated from the study because the thrombus could not be crossed with a guide wire. Thus, 25 patients were randomly placed into one of the two treatment groups.

Patients were eligible for inclusion, before arteriographic evaluation, if they were older than 18 years of age and their medical history and results of physical examination and noninvasive evaluation were consistent with lower extremity native arterial or bypass graft thrombotic or embolic occlusion of less than or equal to 30-day duration. Patients with any of the following criteria were excluded: (a) symptoms and signs of irreversible limb ischemia (Society for Vascular Surgery/International Society of Cardiovascular Surgery category III ischemia), (b) active internal bleeding within the past 3 weeks, (c) cerebrovascular accident within 1 year, (d) intracranial neoplasm or intracranial or intraspinal surgery within the past 2 months, (e) major thoracoabdominal surgery, including biopsy, within the past 10 days, (f) open heart surgery within the past 3 weeks, (g) severe impairment of hepatic function, (h) severe uncontrolled hypertension, (i) recent major trauma or cardiopulmonary resuscitation, (j) history of emboli from cardiac source, (k) subacute bacterial endocarditis, (l) severe coagulopathy, (m) diabetic hemorrhagic retinopathy, (n) lactating female, pregnancy, or unexplained amenorrhea for three or more days beyond expected menses, (o) inability to give informed consent, and (p) inability to cross the entire thrombus with a guide wire.

The flow chart of the study protocol is shown in Figure 1. Patients were randomized by selection of consecutively numbered sealed envelopes to undergo treatment by means of either CI ( $n = 13$ ) or FI ( $n = 12$ ). The mean age of the patients in both treatment groups was  $63.9 \text{ years} \pm 12$ . There were seven men and six women in the CI group and four men and eight women in the FI group. UK (Abbokinase; Abbott Laboratories, North Chicago, Ill) was intraarterially administered to all patients with a 5-F pulsed-spray catheter (AngioDynamics, Glens Falls, NY) with pressure-responsive side holes distributed over its distal 10 cm. This catheter can serve as an end-hole catheter when the tip-occluding wire is removed.

A preliminary bolus of UK was intra-thrombotically administered to all patients with the pulsed-spray technique. Heparin was administered intravenously to all patients. For the CI group, the bolus was hand injected by withdrawing concentrated UK from a 10-mL syringe connected to the reservoir port of the pulsed-spray connector and injecting the UK forcefully (simulating the action of the pulsed-spray pump) with a 1-mL tuberculin syringe connected to the injection port. This was followed by slow CI (continuous infusion pump; IMED, San Diego, Calif) through the end hole (the tip-occluding wire was removed), which was embedded in the

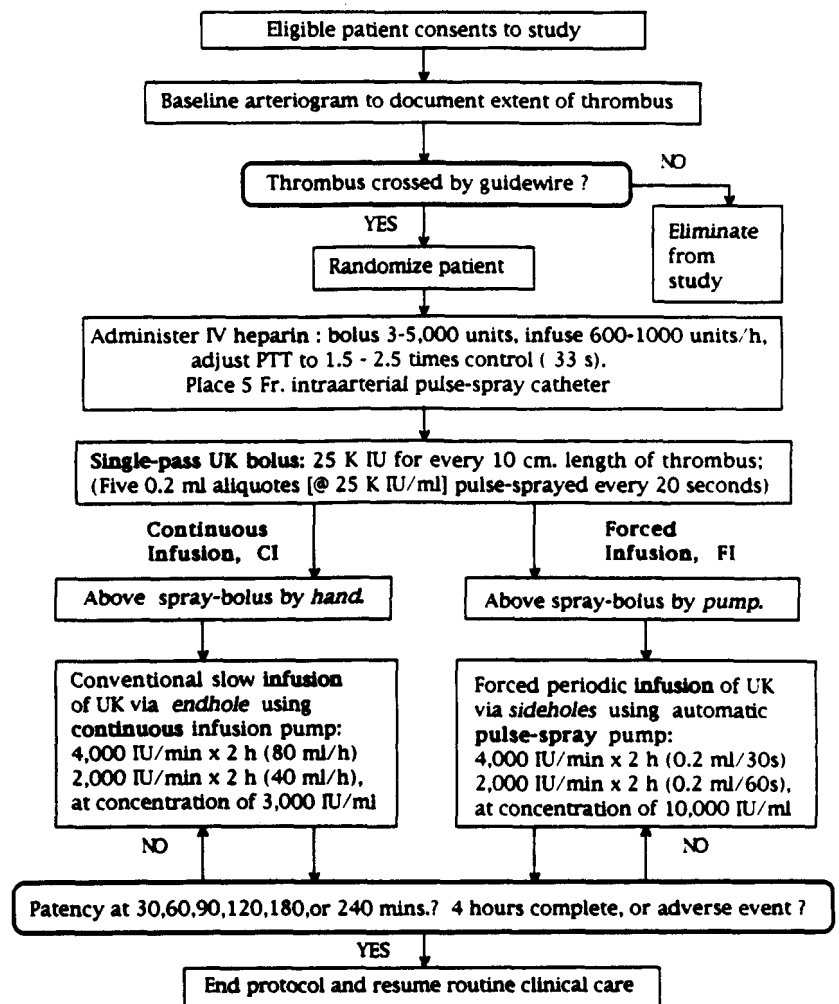


Figure 1. Flow chart shows procedural protocol for lower extremity thrombolysis.

proximal thrombus. For the FI group, an investigational prototype automatic pulsed-spray pump (AngioDynamics) was used for both the initial bolus and the subsequent FI technique (pulsed spray via side holes) for up to the 4-hour duration of the protocol. Administration of the preliminary bolus was completed in all patients within 5-10 minutes.

UK concentrations and infusion rates were calculated to provide equivalent doses within a given period regardless of the treatment technique employed and were comparable with the high doses used in previous studies (13,15). During bolus administration, the catheter was initially placed at the distal end of the thrombus and retracted proximally in 10-cm sections, lacing the entire thrombus (ie, depositing enzyme locally) as the catheter was turned about its axis after each spray. After the bolus was administered, the catheter was placed proximally in the thrombus for both groups. The CI group received slow infusion of UK via the end hole. For the FI group, the distal 10 cm of the catheter was embedded in the thrombus and UK was administered forcefully

with the pulsed-spray technique. The research protocol lasted for a maximum of 4 hours; during this time, the patients were monitored closely in the angiography suite.

Angiography was performed frequently to monitor for progress of thrombolysis with use of low-osmolarity contrast agents. Intraarterial digital subtraction angiograms were obtained unless substantial changes were observed fluoroscopically, necessitating a cut-film single leg run-off. When marked lysis was noted and the thrombus was no longer present around the catheter, the catheter was advanced into the thrombus (proximally for the CI group and 10 cm for the FI group). A cut-film arteriogram was obtained in all patients at completion of thrombolysis.

The milestones for progression of therapy were defined as follows: *Recanalization* refers to the establishment of a continuous channel of at least the diameter of a wire throughout the length of the thrombus with or without substantial flow, *patency* refers to our end-point criterion of 95% or greater thrombolysis by volume with brisk antegrade flow, and *completion of treatment*

**Table 1**  
**Associated Risk Factors**

Risk Factors	Treatment Group	
	CI (n = 13)	FI (n = 12)
Diabetes mellitus	3	3
Hypertension	10	8
Smoking history	10	5
Coronary artery disease	6	6
Hypercoagulable state	2	1
Prior vascular surgery	9	12

Note.—None of the differences between the groups is statistically significant.

required total lysis (100%) without any trace of residual thrombus.

The primary end point of this study was 95% or greater lysis of the initial thrombus volume with brisk antegrade flow (patency) at any time within the first 4 hours of treatment, as estimated with angiography. The research protocol was discontinued if patency was achieved (as determined by consensus of the interventional radiology team) or if the 4-hour limit was reached. Acute complications, adverse effects of treatment, or an unacceptable worsening in the condition of the patient were also reasons for terminating UK administration. After completion of the thrombolytic therapy, routine clinical care was resumed. If further thrombolysis was required, UK was administered at 1,000 U/min (20 mL/h via the end hole with the IMED pump).

Thrombus volume was estimated retrospectively from measurements of post-thrombolysis vessel diameter and length in regions where thrombus was previously present. If diameter varied, the volumes of segments of different diameters were added. In addition, an arbitrary runoff score (ranging from 0 to 3) was established as follows: 1 point was given for each continuous vessel, 0.5 was given if the vessel was present but discontinuous, and 0 was given if the vessel was absent.

The duration of UK administration and the size of the population sample were based on two previous studies. In a prospective randomized comparison of recombinant tissue-type plasminogen activator and UK for peripheral arterial thrombolysis, Meyerovitz et al (8) employed a conventional UK infusion technique similar to the slow CI regimen used here (but without using the pulsed-spray technique to administer the initial bolus) and found that none of their patients achieved 95% lysis within 4 hours. Conversely, Bookstein et al (15) reported that 94% of their patients treated with pulsed-spray techniques and UK doses similar to those used in this study achieved "complete" thrombolysis within 2 hours. For the sake of sample size estimation (21), we assumed that the success rate for patency within 4 hours with conventional CI was actually 20% and that with pulsed-spray FI was 80%. This would bias the calcula-

tion toward a higher sample size requirement. On the basis of these assumptions, a sample size of six patients in each group would provide a one-sided  $\alpha$  error of 0.05 and an 80% power ( $\beta = 0.2$ ). Our sample size was twice that required, and the probability of an 80% success rate with FI versus a 20% rate with conventional CI resulting from chance alone would be less than 5%.

The collected data were analyzed for significance of differences between the groups with regard to (a) the number of cases achieving patency within 4 hours, (b) the mean times to achieve various milestones of treatment, and (c) the rates of successful initial clot lysis. In addition, we also analyzed the proportion of limbs salvaged at 30 days with lytic therapy alone or with adjunctive percutaneous and/or surgical intervention as well as the rate and severity of complications. Determination of the statistical significance of differences between population mean values was performed with the Student *t* test, and comparison of proportions (rates) was made with the Fisher exact test. A *P* value of  $\leq .05$  was considered significant.

## RESULTS

Tables 1 and 2 list the demographic characteristics of the two patient groups and the ischemia status of their treated limbs, respectively. None of the differences was statistically significant, although the patients who underwent FI had a greater proportion of more severely ischemic limbs and all had grafts treated ( $P = .06$ ).

Patency was achieved within 4 hours in nine of the 13 (70%) patients who underwent CI and 11 of the 12 (92%) patients who underwent FI ( $P = .16$ ). One patient in each group chose to discontinue participation in the study during the first 4 hours. Thus, 100% of patients in the FI group and 75% of patients in the CI group ( $P = .12$ ) achieved patency in less than 4 hours. Three other patients who underwent CI failed to achieve 95% lysis; treatment was discontinued in two patients, one because of extravasation of contrast material at a graft anastomosis and the other because of acute reocclusion of a native artery after a vasovagal episode. There was no significant difference in the mean bolus dose ( $P = .54$ ), the mean dose to achieve patency within 4 hours ( $P = .43$ ), or the mean total dose infused for completion of thrombolysis ( $P = .44$ ) between the two groups (Table 3).

Table 4 shows the mean times required to achieve the various milestones of treatment. Although patency was achieved within 4 hours in most patients, based on our stringent

standard of practice that requires complete lysis with no residual thrombus (and perhaps because some judgment was involved in determining the "exact" amount of thrombus to undergo lysis), the team in conjunction with the referring vascular surgeon almost always decided to continue infusion overnight. In fact, complete lysis was achieved in only two patients in each group in less than 4 hours. The mean hospital stay was 10 days  $\pm$  10 for the CI group and 9 days  $\pm$  6 for the FI group. There was no statistically significant difference between the two groups in any of these treatment milestones, including the hospital stay.

Table 5 shows the number of patients in each group who achieved complete lysis within 4 hours, within 24 hours, and in more than 24 hours. The difference in the number of patients in each group achieving lysis in less than 24 hours (eight in the CI group and 10 in the FI group) and those requiring more than 24 hours (three in the CI group and two in the FI group) was not significant ( $P = .32$ ).

There was no significant difference in the limb-related complications between the two groups. Emboli occurred in five patients in each group. All emboli were small (2–3 mm) except for a large distal embolus from the iliac artery in a patient undergoing CI. In addition, most of these emboli (three of the five in the CI group and four of the five in the FI group) resolved within the observation period with continued UK infusion. Groin hematoma ( $> 2$  cm in diameter) developed in two patients in each group; none required surgical evacuation. Three of the 12 patients undergoing FI and one of the 13 patients undergoing CI needed red blood cell transfusion within 72 hours of therapy (not necessarily related to blood loss from lytic therapy), but the difference between the groups was not statistically significant ( $P = .27$ ). Similarly, there was no significant difference in the number of patients who experienced concurrent rethrombosis (two in the CI group, one in the FI group), compartment syndrome (two in the CI group, none in the FI group), or amputation (two in the CI group, none in the FI group) in less than 30 days ( $P = .2$ ). The one patient in the FI group with a hypercoagulable state experienced recurrence of severe concurrent thrombosis during therapy and underwent prolonged treatment of 44 hours. There were two patients in the CI group who had a hyperco-

agulable state; thrombolysis was unsuccessful in one within the protocol period (4 hours), and the other had a successful outcome with an uneventful clinical course.

Other complications (occurring within 30 days) not related directly to the treated limb were comparable between the two groups. Acute renal failure occurred in none of the patients in the CI group and one patient in the FI group, rigors occurred in one patient in the CI group and none in the FI group, and trace hematuria occurred in none of the patients in the CI group and one patient in the FI group. One patient who underwent CI had minor changes at electrocardiography without subsequent myocardial infarction. No significant differences were noted between the two groups, including two deaths (occurring within 30 days) in the FI group ( $P = .2$ ) that were not directly related to lytic therapy. One patient died of myocardial infarction within the first week after therapy, and the other died of pneumonia in the third week after therapy. Two patients in each group were hospitalized for more than 2 weeks.

Therapeutic success was defined as a positive clinical outcome at 30 days with resolution or improvement of ischemic symptoms. The treatment was considered a failure if occlusion of a treated graft or artery recurred, the limb was amputated, or the patient died. Five patients in the CI group and seven patients in the FI group underwent thrombolytic therapy alone (two in each group) or with angioplasty (three in the CI group, five in the FI group). There was no significant difference in successful revascularization rates between these two subgroups (five of five patients in the CI group vs six of seven in the FI group). Four of eight patients in the CI group and only one of five patients in the FI group subsequently underwent successful surgical revascularization procedures. Although the number of patients who subsequently underwent surgical revascularization is small, the success rate for the patients who underwent FI was significantly lower ( $P < .05$ ) than that in the patients who underwent thrombolytic therapy alone or with angioplasty. The significance is not altered if the deceased patients are excluded. In comparison, this difference between the similar CI subgroups (five of five patients vs four of eight) was not significant ( $P = .1$ ).

Table 6 shows various 30-day clinical outcomes. There is no difference at

**Table 2**  
Ischemia Status of the Treated Limbs

Parameter	Treatment Group	
	CI (n = 13)	FI (n = 12)
SVS category	4 viable limbs, 9 threatened limbs	1 viable limb, 11 threatened limbs
Mean symptom duration (d)	13 (9.4)	8 (8.3)
Mean estimated clot volume (mL)	18 (17)	15 (12)
Mean run-off score	1.31 (1.03)	1.16 (0.98)
No. of native arteries	4	0
No. of grafts	9	12

Note.—There were 25 acute thrombotic occlusions. SVS = Society for Vascular Surgery. Numbers in parentheses are standard deviations. None of the differences between the groups is statistically significant.

**Table 3**  
Mean UK Dose (U/h) by Treatment Milestone

Treatment Milestone	Treatment Group	
	CI	FI
Administration of initial bolus	202,000 ± 105,000	179,000 ± 74,000
Patency within 4 h	585,000 ± 179,000	532,000 ± 115,000
Completion of lysis	2,103,000 ± 1,792,000	1,620,000 ± 1,115,000

Note.—Data are given as mean ± standard deviation. None of the differences between the groups is statistically significant.

the end of the procedure in the rates of thrombolysis success or failure between the two groups ( $P = .09$ ). Similarly, there are no statistically significant differences in the rates of continued claudication, limb salvage or loss, death, or positive clinical outcome (nine of the 13 patients [69%] in the CI group, seven of the 12 [59%] in the FI group).

### DISCUSSION

In theory, forceful intrathrombic infusion of plasminogen activators accelerates thrombolysis by disrupting the thrombus and increasing the surface area available for enzymatic action. Early laboratory experiences had suggested that the rate-limiting step in thrombolysis is the slowness in diffusion of enzyme from the clot's surface into its interstices (22) and that thrombolysis could be accelerated by macerating the clot and mixing it directly with the enzyme (23). In later animal studies (5), a marked increase was reported in the rate of lysis with use of supplemental intrathrombic spray injection of enzymes when compared with parathrombic slow infusion. We have shown, in an in vivo study (6), that periodic FI of streptokinase into rabbit inferior vena cava thrombi accelerated thromboly-

**Table 4**  
Times to Achieve Treatment Milestones

Treatment Group	Recanalization (min)	Patency within 4 h (min)	Completion of Lysis (h)
CI	40 ± 19	92 ± 36	28 ± 26
FI	42 ± 20	95 ± 50	20 ± 14

Note.—Data are given as mean ± standard deviation. Differences between the groups are not statistically significant.

**Table 5**  
Time for Completion of Thrombolysis

Treatment Group	Time to Achieve Complete Lysis		
	≤ 4 h	≤ 24 h	> 24 h
CI (n = 11)	2	8	3
FI (n = 12)	2	10	2

Note.—Data do not include two patients in the CI group who failed to achieve lysis in less than 4 hours.

sis when compared with conventional slow infusion. However, the advantages observed in in vivo laboratory models should not necessarily be expected to translate proportionately to clinical practice.



**Table 6**  
**Thirty-day Clinical Outcome**

Outcome	Treatment Group	
	CI (n = 13)	FI (n = 12)
Initial thrombolysis success	8	11
Initial thrombolysis failure	5	1*
Adjunctive surgical procedure necessary	8	5
Claudication at 30 d	2	3
Limb salvage	11	10
Limb loss	2	0
Death	0	2
Positive clinical outcome	9	7

Note.—None of the differences between the groups is statistically significant.

\* This patient experienced a partial reocclusion.

Of the patients who remained in our study, patency was achieved in less than 4 hours in 75% of patients who underwent CI and all of the patients who underwent FI, although this difference is not statistically significant. Similarly, times for completion of lytic therapy after the 4-hour formal research protocol were not different, suggesting that, on the whole, the two infusion techniques used in this study were equally efficacious with respect to speed of lysis. Indeed, except for a mild trend toward faster completion of lytic therapy and a smaller dose of UK with FI, there were no significant differences in the immediate results, complications, or 30-day clinical outcomes between the two treatment groups.

It is notable that the time required in our trial for completion of thrombolysis was longer by a factor of 20 relative to the time of only 1–1.5 hours reported by Valji et al (13) and Bookstein et al (15), who used pulsed-spray methods and UK infusion rates and doses nearly identical to those used herein. Mewissen et al (11) used a different side-hole catheter to administer UK into eight occluded native arteries with a pulsed-spray technique and reported completion of lysis in a mean of 0.8 hour with an infusion rate of 600,000 U/h—which is much greater than the rate used by us. However, in a later study using the same pulsed-spray pump with similar UK doses and treatment end points, Hallisey et al (24) were able to demonstrate a shortening of therapy time in only three of their nine patients (mean infusion time, 2 hours). The remaining six patients required a mean infusion time of 23 hours to

achieve complete thrombolysis—which is similar to that reported herein.

Discrepancies in reported duration of thrombolytic therapy are often difficult to explain because of the inability to directly compare studies. Factors such as patient selection criteria, limb ischemia status, and thrombus age and burden might account for some of the differences. Other differences might be due to study design or choice and definition of treatment end points. Whereas one practitioner may accept a residual thrombus or small distal emboli at the end of treatment (13,25), another may choose to continue infusion overnight to eliminate all evidence of thrombus (12). We favor the latter practice for the treatment of peripheral arteries because of the preponderance of clinical evidence that a residual thrombus is highly thrombogenic (26).

In our study, some of the difference in the mean time to completion of treatment between the groups (28 hours for the CI group vs 20 hours for the FI group) may be related to the fact that, as a group, the patients who underwent CI completed their research protocols slightly earlier in the day (by approximately 1.5 hours [ $P = .15$ ]) than did those who underwent FI. This may have resulted in a slight increase in the mean interval to subsequent (out-of-protocol) angiographic follow-up for progression of lysis (16.7 hours for the CI group vs 13.9 hours for the FI group,  $P = .06$ ), which was almost always performed early the next morning. Such practical considerations in treating and monitoring patients may also contribute substantially to the differences in the time for completion of thrombolysis reported in the literature.

In a prospective randomized trial completed at our institution just before initiation of this study, Meyerovitz et al (8) compared the efficacies of recombinant tissue-type plasminogen activator and UK for thrombolysis of acute peripheral arterial and graft occlusions. The demographic and clinical characteristics of the patients in their study were comparable with those of the present study. Specifically, in the patients receiving UK, Meyerovitz et al slowly injected a bolus (lacing) of 60,000 U of UK by hand via an end-hole catheter as it was withdrawn from the distal to the proximal part of the thrombus. Then UK was administered with the CI technique by using an IMED pump with a dose and intravenous anticoagulation regimen identical to those

of our CI group. The bolus technique used by Meyerovitz et al differs from our technique in two ways: We injected a preliminary bolus of UK into the thrombus with a pulsed-spray technique via a side-hole catheter, and we used a larger dose.

None of the patients in the study by Meyerovitz et al achieved complete lysis within the first 4 hours, while six patients achieved it within 24 hours and 10 in more than 24 hours. In comparison with our CI group, the difference between the number of patients completing lysis within 24 hours or more almost achieves statistical significance ( $P = .07$ ). A similar comparison with the FI group shows a significant difference ( $P = .02$ ). If the results from our CI and FI groups are combined and compared with those from the study by Meyerovitz et al, the increase in the number of patients completing lysis within 24 hours becomes significant ( $P = .01$ ). Although conclusions must be drawn cautiously from historical comparisons, there is nonetheless a suggestion that this reduction in treatment time when compared with our own prior experience (8) can only be credited to one change in technique—namely, the initial intrathrombotic administration of a bolus with a pulsed-spray technique, which was the common denominator in the treatment protocol of both groups in this study. This was an unexpected finding.

In a randomized prospective trial comparing two dose regimens of UK (a 50,000-IU local bolus administered with an end-hole catheter plus 50,000 IU/h for 24 hours versus a 250,000-IU local bolus plus 250,000 IU/h for 4 hours and then 125,000 IU/h for 20 hours), Cragg et al (27) found no statistically significant differences between the two groups of patients with respect to duration of infusion or mean time for completion of lysis. Thus, the increase in the proportion of cases completed in less than 24 hours in the present study may be attributed to the action of initial forceful pulse-spray administration of a bolus through a side-hole catheter, rather than to the larger mean bolus dose in comparison with Meyerovitz et al (8).

FI does not appear to carry an added risk over conventional slow infusion methods. Because development of distal emboli is a feared complication of FI, a diligent effort was made to record all embolic events, regardless of clinical importance. Most emboli, however, resolved dur-

ing the treatment period. The remaining emboli either needed no further intervention or were removed at surgery performed for other reasons.

The higher rate of observed emboli in our study may be due to many factors aside from diligent observation. First, it is possible that administration of the initial bolus in both groups caused thrombus fragmentation resulting in a greater number of emboli. Interestingly, continued FI beyond the initial bolus did not appear to increase the rate of emboli formation in the FI group. Second, Bookstein et al (23), who reported an emboli rate of 4%, attributed this to leaving a distal 2-cm plug of thrombus untreated for the first 15 minutes. However, during this early period, which corresponds to our preliminary bolus administration, we saw no emboli. All of our embolic events occurred much later in the treatment. Moreover, we prefer to reestablish flow as soon as possible. Indeed, we found that for both groups, the time to achieve patency increased linearly with the number of run-off vessels ( $r = .77, P < .05$  for the CI group;  $r = .74, P < .05$  for the FI group). This suggests that the presence of large collateral vessels reconstituting the distal run-off vessels may provide a lower-resistance pathway for blood flow and delay recanalization of the occluded conduit. Because these collateral vessels alone are unable to prevent ischemia, it would seem prudent to rapidly recanalize and reestablish flow through the occluded conduit. Finally, we note that, in general, the rates of other important acute and late complications are not different from those reported previously (28,29).

Although the number of patients in our study is small, those who were not successfully treated with thrombolytic therapy alone or with angioplasty, and thus required surgery, did worse—a finding that was more significant in the FI group. We report the association but propose no causative relationship. All of the patients who underwent FI had previously undergone surgical vascular procedures ( $P = .06$ ) and tended to have more severe ischemia. Moreover, most patients in the FI group did not require surgery and had a high success rate, arguing against FI being a cause of poor surgical outcome. The 30-day clinical outcome in both groups was similar.

We expected that the effect of the continued FI with the automatic pump would outweigh that of the preliminary bolus and that it would accelerate thrombolysis significantly

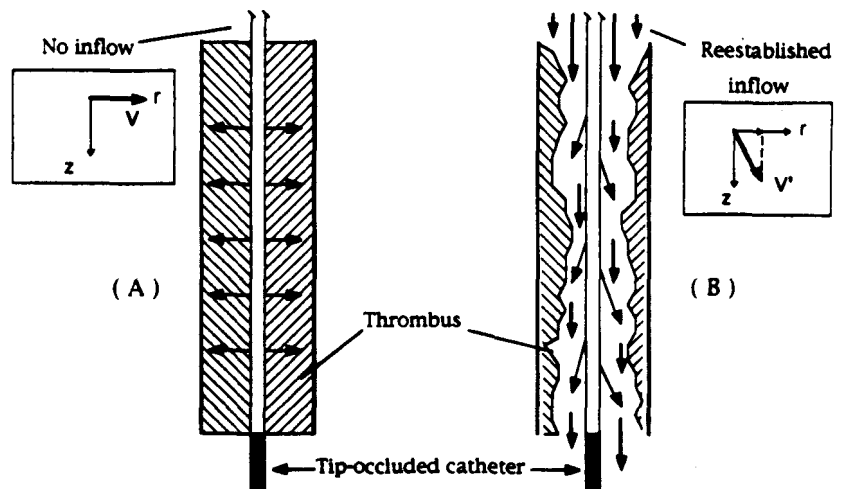


Figure 2. (A) Schematic of a thrombosed vessel with a concentrically placed side-hole catheter. The full force of the exiting jet of fluid is directed radially into the thrombus. (B) As blood flow is reestablished, the fluid jets are redirected toward the flow; the radial component ( $V'[r]$ ) of the jet velocity is insufficient to penetrate the thrombus.

when compared with conventional slow local infusion. However, we did not find such an added effect. It is possible that FI quickly loses its effectiveness. As shown in Figure 2, when the thrombus surrounds the catheter, the full force of the radially exiting jet is available to penetrate the thrombus. However, as brisk antegrade blood flow is reestablished, it redirects the jets so that the radial component is decreased and the impact on the receding clot surface is reduced. Thus, the pulsed-spray technique is most effective during the initial bolus administration and may be less effective once antegrade flow has been reestablished.

We have not studied the benefits of suction thrombectomy as a "debulking" procedure during thrombolysis (30). Results from recent experiments demonstrating acceleration of thrombolysis by the addition of local ultrasonic energy (31) are encouraging, but these techniques must be tested clinically. The ultimate goal of research in accelerating thrombolysis should be to shorten the entire treatment time, including adjunctive angioplasty, to about 2 hours. There is no convincing evidence in the literature suggesting that termination of lytic therapy and early angioplasty in the presence of residual thrombus is safe or clinically efficacious.

Of note, LeBlang et al (16), in a retrospective analysis, reported a mean infusion duration time of 21 hours with use of a continuous, low-dose (mean, 87,000 U/h) overnight drip infusion of UK without transthoracic administration of a bolus and (in general) monitoring progression of

lysis with angiography. If properly applied, this may be one presently available approach to minimizing resource utilization. Another group has reported that patients undergoing thrombolysis may be monitored safely in a non-intensive care setting (9). If this practice is found to be universally safe, a considerable cost reduction will be realized even if the goal of single-session therapy is not achieved in the near future.

In conclusion, continued FI of UK does not significantly accelerate thrombolysis or improve 30-day clinical outcome when compared with CI when both groups receive a preliminary bolus by means of the pulsed-spray technique. We were unable to reproduce the short treatment times reported by Valji et al (13) and Bookstein et al (15), although we used comparable doses and techniques. However, we are now able to achieve complete lysis in most patients within 24 hours, probably because of the administration of the initial pulsed-spray bolus. This should result in a decrease in the need for intensive care monitoring. The role of automation (the pulsed-spray pump) is one of adding convenience and consistency. We have limited our pulsing rate to two per minute. It is possible that increasing the pulse rate may increase the rate of lysis. We have not explored the flexibility, nor optimized the use, of the programmable automated pump. ■

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**References**

1. McNamara TO, Bomberger RA, Merchant RF. Intra-arterial urokinase as the initial therapy for acutely ischemic lower limbs. *Circulation* 1991; 83(suppl I):106-109.
2. Sullivan KL, Gardiner GA, Kandarpa K, et al. Efficacy of thrombolysis in infrainguinal bypass grafts. *Circulation* 1991; 83(suppl I):99-105.
3. Motarjeme A. Thrombolytic therapy in arterial occlusion and graft thrombosis. *Semin Vasc Surg* 1989; 2:155-178.
4. Sullivan KL, Gardiner GA, Shapiro MJ, Bonn J, Levin DC. Acceleration of thrombolysis with a high-dose transthorbus bolus technique. *Radiology* 1989; 173:805-808.
5. Valji K, Bookstein JJ. Fibrinolysis with intrathrombotic injection of urokinase and tissue-type plasminogen activator: results in a new model of subacute venous thrombosis. *Invest Radiol* 1987; 22:23-27.
6. Kandarpa K, Drinker PA, Singer SJ, Caramore D. Forceful pulsatile local infusion of enzyme accelerates thrombolysis: in vivo evaluation of a new delivery system. *Radiology* 1988; 168:739-744.
7. Graor RA, Risius B, Lucas FV, et al. Thrombolysis with recombinant human tissue-type plasminogen activator in patients with peripheral artery and bypass graft occlusions. *Circulation* 1986; 74(suppl I):15-20.
8. Meyerovitz MF, Goldhaber SZ, Reagan K, et al. Recombinant tissue-type plasminogen activator versus urokinase in peripheral arterial and graft occlusions: a randomized trial. *Radiology* 1990; 175:75-78.
9. Routh WD, Tatum CM, Barton RE, et al. Urokinase infusion: feasibility of monitoring for complications in a non-intensive care setting. *JVIR* 1991; 2:69-72.
10. Hicks ME, Picus D, Darcy MD, Kleinhofer MA. Multilevel infusion catheter for use with thrombolytic agents. *JVIR* 1991; 2:73-75.
11. Mewissen MW, Minor PL, Beyer GA, Lipchick EO. Symptomatic native arterial occlusions: early experience with "over-the-wire" thrombolysis. *JVIR* 1990; 1:43-47.
12. McNamara TO. The use of lytic therapy with endovascular "repair" for the failed infrainguinal graft. *Semin Vasc Surg* 1990; 3:56-65.
13. Valji K, Roberts AC, Davis GB, Bookstein JJ. Pulsed-spray thrombolysis of arterial and bypass graft occlusions. *AJR* 1991; 156:617-621.
14. Kandarpa K, Meyerovitz MF, Aruny JE, et al. Thrombolysis by forced infusion of urokinase: a prospective randomized controlled clinical trial (abstr). *JVIR* 1991; 2:7.
15. Bookstein JJ, Fellmeth B, Roberts A, Valji K, Davis GB, Machado T. Pulsed-spray pharmacomechanical thrombolysis: preliminary clinical results. *AJR* 1989; 152:1097-1100.
16. LeBlang SD, Becker GJ, Benenati JF, Zemel G, Katzen G, Sallee SS. Low-dose urokinase regimen for the treatment of lower extremity arterial and graft occlusions: experience in 132 cases. *JVIR* 1992; 3:475-483.
17. Gardiner GA, Koltun W, Kandarpa K, et al. Thrombolysis of occluded femoropopliteal grafts. *AJR* 1986; 146:831-833.
18. Van Breda A, Graor RA, Katzen BT, et al. Relative cost-effectiveness of urokinase versus streptokinase in the treatment of peripheral vascular disease. *JVIR* 1991; 2:77-87.
19. Traughber PD, Cook PS, Micklos TJ, Miller FJ. Intraarterial fibrinolytic therapy for popliteal and tibial artery obstruction: comparison of streptokinase to urokinase. *AJR* 1987; 149:453-456.
20. Belkin M, Belkin B, Bucknam CA, Straub JJ, Lowe R. Intra-arterial fibrinolytic therapy: efficacy of streptokinase vs urokinase. *Arch Surg* 1986; 121:769-773.
21. Colton T. Inference on means. In: *Statistics in medicine*. 19th ed. Boston, Mass: Little, Brown, 1974; 142-146.
22. Bookstein JJ. Clinical and laboratory experience with selective thrombolysis. *Ann Radiol* 1984; 27:322-323.
23. Bookstein JJ, Saldinger E. Accelerated thrombolysis: in vitro evaluation of agents and methods of administration. *Invest Radiol* 1985; 20:721-735.
24. Hallisey MJ, Parker BC, Meranze SG, Rholl KS, van Breda A. Preliminary experience with a mechanical pulse-spray device in the treatment of arterial occlusions (abstr). *JVIR* 1992; 3:34.
25. Valji K, Bookstein JJ, Roberts AC, Sanchez RB. Occluded peripheral arteries and bypass grafts: lytic stagnation as an end point for pulse-spray pharmacomechanical thrombolysis. *Radiology* 1993; 188:389-394.
26. Fuster V, Stein B, Ambrose JA, et al. Atherosclerotic plaque rupture and thrombosis: evolving concepts. *Circulation* 1990; 82(suppl II):47-59.
27. Cragg AH, Smith TP, Corson JD, et al. Two urokinase dose regimens in native arterial and graft occlusions: initial results of a prospective, randomized clinical trial. *Radiology* 1991; 178:681-686.
28. Kaufman JA, Bettmann MA. Thrombolysis of peripheral vascular occlusions with urokinase. *Semin Intervent Radiol* 1992; 9:159-165.
29. Durham JD, Rutherford RB. Assessment of long-term efficacy of fibrinolytic therapy in the ischemic extremity. *Semin Intervent Radiol* 1992; 9:166-173.
30. Starck EE, McDermott JC, Crummy AB, et al. Percutaneous aspiration thromboembolectomy. *Radiology* 1985; 156:61-66.
31. Tachibana K. Enhancement of fibrinolysis with ultrasound energy. *JVIR* 1992; 3:299-303.

## Intravascular Ultrasound: Implications for Intervention

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During the past several decades angiography has been well established as the "gold standard" for detailed assessment of arterial structures. Depending on the type of imaging system employed, detailed evaluation of diverse portions of the circulation can be obtained over large areas of the vascular system. It provides a two-dimensional detailed evaluation of vessels, including branches and collateral circulation in patients with occlusive disease. By definition, angiography provides images of the luminal surface, showing irregularities, stenosis, and occlusions but providing no information about the vessel wall deep to the lumen. Angiographers in clinical practice have long heard comments from our surgical colleagues regarding underestimation of extent of disease based on their correlative observations at the time of surgery. In addition, intra- and inter-observer variability limits the quantitative assessment of disease and therapeutic interventions. 1 y 2

The recent development of clinically useful intravascular ultrasound has allowed for the first time, the interventionalist to obtain unique and additional information about the vessel wall compared to angiography. The continued miniaturization of ultrasound technology and concomitant efforts of catheter technology has facilitated this growth. At the same time, significant advances in percutaneous intervention for vascular disease as made the information obtained by intravascular ultrasound more directly relevant to intervention. The purpose of this discussion is to review the recent development in intravascular ultrasound (IVUS) technology and to define its clinical benefit in daily practice of interventional vascular radiology.

### TECHNICAL CONSIDERATIONS IN IVUS

Successful acquisition of IVUS images involves a combination of both ultrasound and catheter

technologies. Because of the invasive nature of the examination is limited to adjunctive use during diagnostic and therapeutic procedures. Characteristics such as flexibility, tractability over a guidewire, radiopacity, and durability are as important as the technological aspects of the ultrasound transducers and imaging consoles. If one cannot place the IVUS catheter in the area of interest, a failure of acquisition occurs as surely as if the ultrasound imaging device failed. This represents a unique problem to manufacturers of ultrasound equipment and has lead to cooperative efforts with catheter manufactures.

Investigators have evaluated both phased array (multiple small transducers at the catheter tip) and mechanical types of transducer for use in intravascular ultrasound with each approach having advantages and limitations. Phased array systems (e.g. Endosonics, Intertherapy) employ multiple crystals (32-64) to simulate and reconstruct a cross-sectional 2D image. Advantages of this approach include the use of a catheter with no moving parts, facilitating "over the wire" designs, and having theoretical advantages for integration into interventional devices such as angioplasty balloons and atherectomy devices. Limitations include the need for more computer power to handle data and secondary increase in cost. Additionally, imaging of larger vessels such as those in the peripheral circulation has been more difficult, however high quality images have been obtained in the coronary circulation.

Mechanical transducers employ a rotating transducer driven by an externally supplied motor drive (Diasonics/Boston Scientific Corporation. Cardiovascular Imaging Systems CVIS). Frequently, internal mirrors are used reduce the imaging window of the transducer and further enhance image quality. Catheters used with these types of systems are somewhat larger (4.8-8F) and use "monorail" or "over the wire" introduction

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utilizing small (0.016-0.025 in) guides. Traversing the aortic bifurcation or delivering the IVUS catheter selectively to visceral vessels is still somewhat difficult without using a guiding catheter.

IVUS catheters have been evaluated in a variety of frequencies from 20-40 MHz. These frequencies are considerably higher than those used in conventional external vascular ultrasound (5 MHz), taking advantage of improved resolution resulting from shorter wave lengths. In the peripheral circulation, the most widely used types are 20 MHz, allowing penetration of 1.5-2.0 cm. These frequencies have produced images of consistently high quality, but recently IVUS catheters of 30 MHz have been under evaluation.

Use of intravascular ultrasound requires a sheath for introduction, and guide wires of smaller size than usual for exchange (0.014-0.025 in). Since the catheters are dedicated use, it requires an additional step during the procedure, adding procedure time. With experience this can be limited to less than five minutes. Additional cost is added, however, not only by the ultrasound machine, but by the disposable catheters needed for imaging. To justify this increase in procedure time and cost, this imaging modality must provide unique information, that in some way enhances understanding of the procedure and effects therapeutic decision-making.

#### **UNIQUE INFORMATION PROVIDED BY INTRAVASCULAR ULTRASOUND**

**A**ngiography provides detailed longitudinal information about vessels, but is limited in ability to assess the vessel wall. Intravascular ultrasound allows 2D axial imaging of the vessel, providing information about the luminal surface as well as the other histologic layers. Post processing can allow three dimensional reconstruction of imaged segments further enhancing the quality of this information. It requires no ionizing radiation, no injections of fluids, and there is no inherent time limit on imaging. Additionally the information is digitized and may be suitable for quantitative analysis such as diameter, area, and percent stenosis calculations, as well as useful in attempting tissue characterization.

Investigation of ultrasound applied in vessels in vivo and in vitro have demonstrated similar findings. The three histologic layers of the arterial wall can be clearly identified and have distinctive ultrasonic characteristics. 3,4,5

The intima is hyperechoic with a varying coarse and smooth echogenic pattern. In normal arteries it is quite thin, but can increase significantly if intimal hyperplasia develops post intervention. A distinct thin hypoechoic layer is now felt to represent media. The adventitia is hyperechoic with an indistinct outer margin where the outer wall of the artery is covered by surround soft tissue. Calcium within the wall of a vessel produces acoustic shadowing, a typical sonic characteristic. Generally this calcium is deep to the intima and does not result in degradation of the image. Plaque is hyperechoic but may have a mixed pattern with hypoechoic areas. This is compatible with the known histologic complexity of plaque, including predominantly fibrous material mixed with lipid components. Thrombus is hyperechoic but has a frequently distinctive fine pattern recognizable by experienced observers. Flowing blood is easily identified during real-time examination with fine moving with the cardiac cycle. Vessel wall elasticity may be evaluated subjectively during imaging. Variables in ultrasonic appearance of vessels occurs due to differences in manufacturer, gain settings during examination, frequency of transducer, and vessel size. None the less, the characteristics described above are reproducible and predictable, making them clinically useful.

More recently authors have begun to assess the potential of ultrasound to distinguish plaque types in smaller (coronary) muscular arteries. Potkin and colleagues 6 compared ultrasound images with corresponding histologic sections of excised coronary arteries. The authors were able to correctly predict the presence of fibrous and calcific plaque with an accuracy of 77 % and 83 % respectively. Both of these types of plaque are highly echogenic, with calcification producing typical acousting shadowing 7. It was considerably harder to determine the presence of lipid containing or mixed plaque with 23 % and 43 % success respectively. Since lipid filled areas are hyperechoic, they may be obscured by areas of calcification, or surround tissue. None the less, there is hope that improved technology and further investigation will allow improvement in the potential that ultrasound offers for tissue specificity.

#### **CLINICAL APPLICATIONS**

**T**he unique information offered by intravascular ultrasound has its greatest value during the treatment of vascular disease by percutaneous

methods. Additionally, our experience at the Miami Vascular Institute has shown it to be of value in some cases of trauma where two dimensions longitudinal imaging has inherent limitations.

### VESSEL MEASUREMENT

While digital subtraction angiography has provided significant clinical benefit in performing diagnostic and therapeutic vascular procedures, one of the significant limitations has been in the difficulty in determining accurate cross-sectional diameter. While most DSA systems have measurement programs, their accuracy has been suspect particularly when trying to acquirement tolerances of less than 1.0 mm. One of the benefits of intravascular ultrasound is the ability to accurately measure 8,9 not only vessel diameter, but cross-sectional area and percent stenosis 10 as well. Other potential benefits include the potential for evaluating vessel compliance. These measurements can be done "on line" without significant procedural delay. These measurements have proven valuable in determining balloon size for PTA, atherectomy device size, and measuring accurate residual stenoses post procedure. They have also been of value in obtaining accurate sizing prior to stent deployment.

### USE DURING TRANSLUMINAL ANGIOPLASTY

Ultrasound observations during the performance of percutaneous transluminal angioplasty have documented the increased sensitivity for calcium, as well as the significant residual luminal narrowing that is present post PTA. Our clinical experience confirms the observations in coronary arteries of Tobis et al 11: residual atheroma of significance is still present (average residual stenosis=73% by area), suggesting a possible cause of inherent restenosis or early failure. Early prototypes of balloon catheters integrated with central transducers are currently being evaluated, and may at the least contribute to a better understanding of the angioplasty process.

IVUS provides definitive information about lesion location, percent stenosis, lesion length and extent of calcifications and is therefore useful as an adjunct during angioplasty of peripheral lesions 12. Intravascular ultrasound offers particular advantages in patients with decreased renal function and/or diabetes by reducing contrast injections used for localization and monitoring of the procedure.

Because of increased ability to evaluate the vessel wall, IVUS offers great promise in increasing in vivo understanding of the angioplasty process, hopefully leading to better outcomes or therapy.

### DETECTION OF INTIMAL DISSECTIONS

Intimal dissections, both spontaneous and iatrogenic, are well visualized by 2D cross-sectional ultrasound imaging. While spontaneous dissections are generally detected by other means IVUS has been shown 13,14,15 to be useful in detection of aortic dissections. While more conventional methods, such as CT scan, or angiography, may more simply establish this diagnosis, IVUS may be of assistance during angiography in detecting the precise point of re-entry or termination of the dissection.

More frequent use during intervention has shown ultrasound to be more sensitive in detecting the presence and extent of post-PTA dissections, than angiography. One would expect this superiority since dissections are longitudinal and IVUS images perpendicular to the plane of dissection. In many cases fully appreciating the extent of dissection will influence therapeutic decision-making: placing an intravascular stent, prolonged balloon inflation, atherectomy.

Dissections have been noted to be of two predominant types: localized longitudinal and spiral. Either type may extend to compromise the lumen, but spiral dissections are generally a cause of immediate luminal compromise and will generally lead to endovascular stent placement.

### USE DURING ATHERECTOMY

Percutaneous atherectomy is a method of treatment of vascular disease relying on removal of plaque to restore luminal patency rather than "controlled injury" associated with balloon angioplasty. Some investigators believe this method may be superior to PTA 16,17 but this has not been well established in the scientific literature.

It seems intuitive that a procedure to remove plaque should depend on an adequate method of determining a therapeutic end-point which has traditionally been angiography. A recent study at our institution 18 has confirmed previously reported observations in the coronary arteries 19, angiography provides a gross underestimation of residual plaque compared to intravascular

ultrasound. Yock has previously considered the use of IVUS 20. In our own series in patients who determined to have an adequate result (% stenosis(10%) 68% of lesions were found to have residual stenosis)30%, the traditional end point of intervention. One would assume that assessment of the efficacy of atherectomy has been limited by these significant observations and repeat studies should be performed to re-evaluate outcomes. For this reason IVUS has been utilized routinely in our own institution to determine vessel size as well as percent stenosis pre and post intervention.

Three dimensional reconstructions of vessel walls can be accomplished in practical real-time utilizing adjunctive workstation technology. This type of display allows a more intuitive look at the vessel wall, extent of plaque or dissection, and provides a more complete assessment of the effect of therapy. Clinical value has led IVUS manufactures to consider including this type of software in base units.

Recently, investigators have considered the integration of IVUS into atherectomy devices 21,22. This would allow on-line assessment of the efficacy of plaque removal and reduce procedure time. While only in its early stages, it appears that eventually technological hurdles will be overcome in achieving this goal.

#### **USE OF IVUS DURING STENT PLACEMENT**

Intravascular stents have been proven to be of value in the treatment of iliac artery disease where angioplasty has produced a suboptimal result. Indications for use include significant intimal dissections compromising flow, complex lesions, total occlusions and complex lesions. As previously reported by Palmaz 23, ideal stent placement results in integration of the endovascular device into the arterial lumen by endothelialization of the stent surface. Recently, it has become apparent that incomplete deployment of the stent will result in delayed or incomplete endothelial covering. Therefore it has become relevant to ascertain whether complete stent inflation has occurred.

In clinical trials conducted at the Miami Vascular Institute 15, intravascular ultrasound was used to assess degree of stent inflation (abutment) against the arterial wall after deployment with satisfactory result based on angiography. In 20% lesions, stents were noted to be incompletely deployed, requiring

additional intervention with a larger balloon to achieve complete deployment. Stent struts are clearly identified by their regularly spaced highly echogenic appearance.

IVUS may also be useful in evaluating the extent of intimal dissection and in particular, the effectiveness of stenting in eliminating the adverse effects of dissection. Following recanalization of total occlusions, when questions arise as to whether subintimal passage has occurred, IVUS may provide the answers. Ultrasound is also a sensitive detector of intimal hyperplasia which may develop within the stent, although no specific echogenic pattern has been noted for this entity, having the same appearance as normal intima.

#### **FUTURE DIRECTIONS**

Intravascular ultrasound will continue to improve as more investigations consider its importance. Clearly technologies are being explored to integrate IVUS capabilities with interventional devices including PTA balloons and atherectomy devices, with several concepts being explored 24. Additional emphasis will be made on image quality and processing, including three dimensional reconstructions in real time. The practical result of these efforts will be more accurate assessments and control on interventions, hopefully resulting in improved initial and long term outcome.

#### **CONCLUSIONS**

Intravascular ultrasound technology has made significant advances since its first clinical application. Smaller and more flexible catheters, in conjunction with improved image quality have greatly reduced catheter time, making IVUS less cumbersome and more practical during intervention. Unique information obtained includes tissue specific data about the vessel wall deep to the lumen, more sensitive and accurate demonstration of extent of plaque as well as extent of calcification, and accurate measurement of luminal diameter area, and percent stenosis. Ultrasound also has the potential for specific tissue characterization: detecting fibrous, calcific, or lipid-laden plaque as well as the presence of thrombus. This information may have value in determining which intervention might be best suited for a particular lesion. Additionally, early prototypes of devices (PTA balloons, atherectomy devices, and lasers) in which IVUS is integrated offers hope for more precise localization and on

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line assessment of efficacy of therapy. Three dimensional reconstructions may be performed in or near real time, providing additional perspectives about extent of disease and effects of therapy. The established increased sensitivity over angiography makes ultrasound a suitable method of comparison of various types of interventional devices. In the current environment of critical cost effective analysis, IVUS contributes significantly to the efficacy of intervention which will hopefully lead to better long term results. Studies are underway to evaluate the effect of this increased accuracy on long term outcome. Based on our clinical experience, intravascular ultrasound would be a useful imaging tool to the active interventionalist.

#### References

1. Fisher L.D., Judkins M.P., Lesperance J., et al. Reproducibility of coronary arteriographic reading in the coronary artery surgery study (CASS). *Cathet Cardiovasc Diag* 1982; 8: 565-75.
2. Siegel R.J., Swan K., Edwards G., et al. Limitations of post-mortem assessment of human coronary artery size and luminal narrowing: differential effects of tissue fixation and processing on vessels with different degrees of atherosclerosis. *J Am Coll Cardiol* 1985; 5: 342-6.
3. Gussenhoven W.J., Essed C.E., Frietman P., Mastik F., Lancee C., Slager C., Serruys P., Gerritsen P., Pieterman H., and Bom N. Intravascular echographic assessment of vessel wall characteristics: a correlation with histology. *Int J Card Imaging* 1989; 4: 105-116.
4. Mallery J.A., Tobis J.M., Griffith J., Gessert J., McRae M., Moussabek O., Bessen M., Moriuchi M., Henry W.L. Assessment of normal and atherosclerotic arterial wall thickness with an intravascular ultrasound imaging catheter. *Am Heart J* 1990; 119(6): 1392-1400.
5. Segel R.J., Ariani M., Fshbein M.C., Chae J.S., Park J.C., Maurer G., Forrester J.S. Histopathologic validation of angiography and intravascular ultrasound. *Circulation* 1991; 84(1): 109-117.
6. Potkin B.N., Bartorelli A.L., Gessert J.M., Neville R.F., Almagor Y., Roberts W.C., Leon M.B. Coronary artery imaging with intravascular high-frequency ultrasound. *Circulation* 1990; 81: 1575-1585.
7. Gussenhoven E.J., Essed C.E., Lancee C.T., Mastik F., Frietman P., Van Egmond F.C., Rieber J., Bosch H., Urk H.V., Roelandt J., Bom N. Arterial wall characteristics determined by intravascular ultrasound imaging: an in vitro study. *J Am Coll Cardiol* 1989; 14: 957-962.
8. St. Goar F.G., Pinto F.J., Alderman E.L., Fitzgerald P.J., Stadius M.L., Popp R.L. Intravascular ultrasound imaging of angiographically normal coronary arteries: an in vivo comparison with quantitative angiography. *J Am Coll Cardiol* 1991; 18(4): 958-8.
9. Sheikh K.H., Davidson C.J., Kisalo K.B., Harrison J.K., Himmelstein S.I., Kisso J., Bashore T.M. Comparison of intravascular ultrasound external ultrasound and digital angiography for evaluation of peripheral artery dimensions and morphology. *Am J Cardiol* 1991; 67(9): 817-22.
10. Pandian N.G., Kreis A., O'Donnell. Intravascular Ultrasound Estimation of Arterial Stenosis. *J Am Soc Echo* 1989; 2: 390-7.
11. Tobis J.M., Mahon D., Lehman K., Mariuchic M., Honye J., Henry W.L. Intracoronary Ultrasound imaging After Balloon Angioplasty. *Circulation* 1990; 82(4): 175: III-676.
12. Isner J.M., Rosenfield K., Losordo D.W., et al. Percutaneous intravascular US as adjunct to catheter-based interventions: preliminary experience in patients with peripheral vascular disease. *Radiology* 1990; 175: 61-70.
13. Cavaye D.M., French W.J., White R.A., Lerman R.D., Mehringer C.M. Intravascular ultrasound imaging of an acute dissecting aortic aneurysm: a case report. *J Vasc Surg* 1991; 13(4): 510-12.
14. Pande A., Meier B., Fleisch M., Kammerlander R., Simonet F., Lerch R. Intravascular ultrasound for diagnosis of aortic dissection. *Am J Cardiol* 1991; 67(7): 662-3.
15. Williams D.M., Simon H.J., Marx M.V., Starkey T.D. Acute Traumatic Aortic Rupture: Intravascular Ultrasound Findings. *Radiology* 1992; 182: 247-249.
16. Simpson J.B., Selmon M.R., Robertson G.C., et al. Transluminal thectomy for occlusive peripheral vascular disease. *Am J Cardiol* 1988; 61: 96G-101G.
17. Selmon M., Robertson G., Hinohara T., et al. Factors associated with restenosis following successful peripheral atherectomy. *J Am Coll Cardiol* 1989; 13: 108A (abstract).
18. Katzen B.T., Benenati J.F., Becker G.J., Zemel G. Role of Intravascular Ultrasound in Peripheral Atherectomy and Stent Placement. *Circulation* 1991; 84: II-542 (abstract).
19. Ehrlich S., Honye J., Mahon D., Bernstein R., Tobis J. Unrecognized stenosis by angiography documented by intravascular ultrasound. *Cathet-Cardiovasc-Diagn* 1991; 3: 198-201.
20. Yock P.G., Fitzgerald P.J., Linker D.T., Angelsen B.A. Intravascular ultrasound guidance for catheter-based coronary interventions. *J Am Coll Cardiol* 1991; 17(6 Suppl B): 39B-45B.
21. Yock P.G. Catheter apparatus. European patent No. 0234951. 1987.
22. Yock P.G. Personal communication. February 1991.
23. Palmaz J. Intravascular Stents. Syllabus: A Categorical Course in Interventional Radiology. RSNA 1991 December. pp 185-192.
24. Crowley R.J., Hamm M.A., Joshi C.D., Roberts G.T. *Int J Card Imaging* 1991; 6: 145-6.



## Vascular Examination

PETER LANZER<sup>1</sup>

Even in the high technology era the physical exam remains the principle means to assess patients with vascular disease. Traditionally, a single vascular territory was examined. More recently, accounting for the systemic nature of vascular disease a more comprehensive approach is preferred.

The exam consists of eliciting the medical history and performing a thorough bodily inspection, palpation and auscultation. Finally, the standard blood pressure measurements are taken.

### MEDICAL HISTORY

Medical history provides a summary of patient's health problems and where relevant of his socio-economical background. In form of a structured interview the patient's permanent medical record is obtained.

Ordinarily the physician begins the interview with inquiring about the current primary and secondary cardio-vascular symptoms. Patient's free narration is completed by recording the degree of interference of symptoms with patient's life style. The onset of symptoms, provoking circumstances, duration, frequency, severity and response to relieving attempts should be noted.

The physician proceeds to inquire about the history of the present symptoms, or if applicable of the already known specific cardio-vascular disease.

Next, a brief overview of patient's other (non cardio-vascular) medical problems (major illnesses operations or accidents) is provided and the list of all current medications including dosages and timetables are drawn up.

Eliciting the cardiovascular risk profile and detailed history of the previous cardiovascular diseases

represent the final focus of the medical history taking. In the former, specifically the absence or presence of arterial hypertension, tobacco use, genetic and familiar predisposition, lipoprotein, carbohydrate, coagulation, homocystein and iron metabolism disorders, obesity, level of physical activity and negative stressors should be noted. Each risk factor should be individually documented (etiology, duration, level of control and complications, number of cigarette packs per day in years in smokers, the severity of familiar predisposition, the type of obesity, etc.) In high risk patients additional information might be useful (e.g. dietary habits). The laboratory results of the latest metabolic evaluations should be noted. A complete list of previous cardiovascular diseases is drawn up, a detailed description of each cardiovascular illness including previous findings, medical records and other relevant documents' is furnished.

Inquiry about patient's social background completes the oral part of the physical examination. Other components of the standard medical history taking (e.g. review of systems) are not mandatory and remain at the discretion of the physician.

Following the interview the physician proceeds with a thorough physical examination of the patient. The focus of attention is the status of the heart and of the circulatory (arterial, venous, lymphatic) system. The secondarily involved organ systems (e.g. nervous system in stroke victims) are also examined in detail. The exam is completed by an orienting general medical examination. Conducted by a master the exam provides all necessary and essential clues to design the most streamlined economical and straight forward diagnostic and occasionally also a therapeutic regimen for a given individual patient. The essential components of a comprehensive physical vascular examination will be reviewed and discussed.

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##### **References**

1. **Lanzer P., Roesch J. (eds.) : Vascular diagnostics. Springer Verlag, Heidelberg, 1994.**
2. **Kappert A. (ed.) : Lehrbuch und Atlas der Angiologie. Verlag Hans Huber, Bern, 1987.**
3. **Perloff D., Grim C., Flack J. et al.: Human blood pressure determination by sphygmomanometry. Circulation 88: 2460-2470, 1993..**

## Doppler Ultrasound

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**R**ecent technologic advances in conventional and color-Doppler duplex sonography have afforded dramatic changes in the utility of non-invasive vascular imaging. These advances permit high-resolution imaging of the vessels and perivascular tissues and simultaneous Doppler assessment of flow within these vessels. The advantages to the patient of such non-invasive technologies over conventional invasive studies are obvious; however, successful use and interpretation of duplex sonography requires an understanding of not only the strengths, but also the limitations, of the modality.

### PERIPHERAL VENOUS SYSTEM

**T**he accuracy of compression ultrasound in the diagnosis of deep venous thrombosis in the femoropopliteal system is widely recognized. The addition of Doppler interrogation to the basic compression examination permits assessment of flow by the study of spontaneous waveform characteristics, as well as flow responses to distal augmentation or proximal compression or Valsalva maneuvers. The latter maneuvers aid in the diagnosis of valvular incompetence, as reversal of flow can be seen in the vein below the level of incompetent veins. Additionally, comparison Doppler studies between the veins of the symptomatic and asymptomatic sides often provide useful information regarding venous stenosis caused by either intrinsic or extrinsic factors. In the veins, stenosis is suggested by the presence of high velocity flow, loss of respiratory phasicity, turbulence, and abundant flow through geographically contiguous collateral veins.

The basic compression ultrasound study of the femoropopliteal system can be extended when additional data is deemed necessary for the referring physician or the interventional radiologist. For example, if anticoagulation is to be undertaken for deep venous thrombosis of the calf, these vessels can be examined with color Doppler and augmentation techniques. If the proximal extent of a femoral vein thrombus cannot be defined, the

examination should be continued proximally into the iliac veins or even the inferior vena cava to, in part, help assess the need for diagnostic venography or therapeutic filter placement. When filter placement is anticipated, patency of the potential jugular or femoral venous access sites can be also verified by duplex sonography. After filter placement, the location of the filter can be checked by sonography and caval flow assessed by Doppler above, at, and below the level of the filter; similarly complications at the venous insertion site can be studied.

Conventional and color duplex sonography are becoming increasingly important in the diagnosis of upper extremity venous pathology. These clinical situations usually relate to central catheter complications; however, specific and less common entities, such as effort thrombosis and thoracic outlet syndrome, can also be diagnosed with these modalities.

Because of the location of subclavian and innominate veins, compression techniques cannot be applied to these vessels. Consequently, studies of the upper extremity veins rely, to a much greater extent than do those of the lower extremity, on analysis of waveforms and comparison with the other side. Upper extremity veins are characterized by strikingly dynamic flow, reflecting transmission of the cardiac hemodynamics. Respiratory phasicity is also superimposed on these transmitted pulsation. Collateral vessels can demonstrate normal or near-normal waveforms; misidentification of these collateral vessels can result in diagnostic problems.

With central catheter placement complete or non occlusive thrombosis of the vein can occur, as can focal or long segment stenosis. When there is complete thrombosis, the vessel may be expanded with clot of varying echogenicity, incompressible, if accessible to compression, and without flow; collateral vessels may also be seen. Mural or free-floating thrombus can be seen without complete occlusion of the vessel and dampen the waveforms, and may increase the velocity to flow. In thoracic outlet syndrome flow alterations and even cessation flow occur with abduction of the shoulder.

**PERIPHERAL ARTERIAL SYSTEM**

**I**nterest in duplex assessment of lower extremity arterial disease is due in part to the accepted utility of the modality in carotid pathology and deep vein thrombosis and in part to the expanded therapeutic options available for revascularization. However, diagnostic criteria for the lower extremity arteries are much less well-defined than are those for the carotid arteries.

Hemodynamically significant stenosis are usually characterized by at least a doubling of peak systolic velocity, increased turbulence and, on occasion, as with complete occlusion, alteration of the distal waveform to a monophasic signal.

Clinical settings in which duplex sonography is useful include suspected stenosis or occlusion of the native peripheral arteries. Knowledge of the level of disease may be useful even in a patient destined for arteriography to help plan the diagnostic, and possibly therapeutic, angiographic approach. In patients who have undergone surgical arterial reconstruction, duplex sonography can be used routinely to diagnose complications that may predispose to graft failure or used in symptomatic patients, to diagnose clinically significant graft stenoses or occlusions. Doppler features thought to predict graft failure include anastomotic or non-anastomotic stenosis, low graft velocities from inflow disease, cardiac failure or diffuse graft narrowing, and poor distal run-off. The presence and location of hemodynamically significant arteriovenous fistulas can also be define by duplex sonography, permitting accurate radiologic or surgical correction.

If arteriography or radiologic intervention has been undertaken, duplex sonography is useful in defining puncture site complications, such as hematoma, pseudoaneurysm, or arteriovenous fistula. Pseudoaneurysms are characterized by the presence of a pulsatile perivascular mass, with a communicating neck that demonstrates either turbulent to-and-for flow or high velocity flow. Compression over the neck of a pseudoaneurysm with color Doppler monitoring may obviate the need for surgical intervention. Arteriovenous fistulas alter the proximal venous and arterial waveforms, with the vein being more pulsatile than usual and the artery more low resistance in nature; these waveform alterations can often be used to precisely define the level of the arteriovenous fistula, even when the fistula itself cannot be seen.

**CONCLUSION**

**T**he application of conventional and color Doppler sonography to a variety of clinical problems is increasingly sophisticated. These non-invasive modalities are useful in diagnosis and in the follow-up of treatment for pathology in both the peripheral venous and arterial systems.

**References:**

1. Basile R.M., Walters B.D., and Curletti E.L. Image-directed Doppler, ultrasonography in the management of failing infrainguinal bypass grafts. *J. Clinic Ultrasound* 11990; 18: 132-137.
2. Beidle T.R., Brom-Ferral, R., Letourneau J.G., A. Review of Duplex Sonography of Infringuinal Bypass Grafts. in press *A.J.R.*
3. Burbidge S.J., Finlay D.E., Letourneau J.G., Longley D.G. Effects of Central Venous Catheter Placement on Upper Extremity Duplex Ultrasound, *J.V.I.R.*, 1993; 4: 399-404
4. Campbell W.B. Doppler waveform analysis in the management of the lower limb arterial disease. *Ann R. Coll Surg Engl* vol. 68, 1986.
5. Coughlin B.F. and Paushter D.M. Peripheral pseudoaneurysms: evaluation with duplex U.S. *Radiology* 1988; 168 339-342.
6. Fellmenth B.D., Roberts A.C., Bookstein J.J., Freischlag J.A., Forsythe J.R., Buckner N.K. and Hye R.J., Postangiographic femoral artery injuries: nonsurgical repair with US-guided compression. *Radiology* 1991; 178:671-675.
7. Foley D.W., Middleton W.D., Lawson T.L., Erickson S., Quiroz F.A., Macrander S. Color Doppler ultrasound imaging of lower-extremity venous disease. *A.J.R.* 1989; 152:371-376.
8. Helvie M.A. and Rubin J. Evaluation of traumatic groin arteriovenous fistulas with duplex Doppler sonography. *A.J.R.* 1998; 1500: 1177-1180.
9. Igidbashian V.N., Mitchell D.G., Middleton W.D., Schwartz R.A. and Goldberg B.B. Iatrogenic femoral arteriovenous fistula: diagnosis with color Doppler imaging. *Radiology* 1989; 170: 749-752.
10. Kohler T.R., Nance D.R., Cramer M.M., Vandenburghe N., Standness D.E. Duplex scanning for diagnosis of aortoiliac and femoropopliteal disease: a prospective study. *Circulation* 1987; 76(5): 1074-1080.
11. Longley D.G., Finlay D.E., Letourneau J.G. Sonography of the Upper Extremity and Jugular Veins *A.J.R.*, 1993; 160: 957-962.
12. Mantoni M. Deep venous thrombosis: longitudinal study with duplex U.S. *Radiology* 1991; 179: 271-273.

#### **IV INTERNATIONAL COURSE ON VASCULAR AND INTERVENTIONAL RADIOLOGY AS THERAPEUTIC ALTERNATIVE**

13. Middleton W.D., Erickson S., Melson G.L. Perivascular color artifact: pathologic significance and appearance on color Doppler U.S. images. *Radiology* 1989; 171: 647-652.
14. Moneta G.L., Strandness D.E. Jr. Peripheal arterial duplex scanning. *J. Clin Ultrasound* 1987; 15: 645-651.
15. Mulligan S.A., Matsuda T., Lanzer P., Gross G.M., Routh W.D., Keller F.S., Koslin D.B., Berland L.L., Fields M.D., Doyle M., Cranney G.B., Lee J.Y. and Pohost G.M. Peripheal arterial occlusive disease: Prospective comparison of M.R. angiography and color duplex U.S. with conventional angiography. *Radiology* 1991; 178: 695-700.
16. Murphy T.P., Cronan J.J. Evolution of deep venous thrombosis; a prospective evaluation with U.S. *Radiology* 1990; 177: 543-548.
17. Nazarian G.K., Finlay D.E., Letourneau J.G., Schwabacher S.M. Air Along a New Dialysis Graft: Inability to Demonstrate Flow by Doppler Sonography, in press *J.V.I.R.*
18. Polak J.F., Culter S.S. and O'Leary D.H. Deep veins of the calf: assessment with color Doppler flow imaging. *Radiology* 1989; 171: 481-485.
19. Polak J.F., Donaldson M.C., Whitmore A.D., Mannick J.A. and O'Leary D.H. Pulsatile masses surrounding vascular prostheses: real-time U.S. color flow imaging. *Radiology* 1989; 170: 363-366.
20. Rose S.C., Zwiebel W.J., Nelson B.D., Priest D.L., Knighton R.A., Brown J.W., Lawrence P.F., Stults B.M., Reading J.C., Miller F.J. Symptomatic lower extremity deep venous thrombosis: accuracy, limitations, and role of color duplex flow imaging in diagnosis. *Radiology* 1990; 175: 639-644.
21. Yucel E.K., Fisher J.S., Egglin T.K., Geller S.C. and Waltman A.C. Isolated Calf venous thrombosis: diagnosis with compression U.S. *Radiology* 1991; 179: 443-446.
22. Zierler R.E. Duplex and color-flow imaging of the lower extremity arterial circulation. *Semin Ultrasound, C.T., M.R.* 1990; 11(2):168-179.

## Early Sonographic Evaluation of the Transjugular Intrahepatic Portosystemic Shunt (TIPS)

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**Abstract.** The purpose of this study was to evaluate duplex and color Doppler findings in patients before and within 24 h after transjugular intrahepatic portosystemic shunts (TIPS). Conventional duplex and color Doppler were used in the assessment of 19 patients who underwent TIPS as part of a prospective protocol. Patients were examined within 24 h before and after the procedure. Before TIPS, patency, flow direction, and peak flow velocity in the main portal vein and hepatic artery were studied, as well as patency and flow direction in hepatic veins, splenic vein, and inferior vena cava (IVC). Immediately after the procedure, sonographic identification of stent position, shunt patency, and flow dynamics were evaluated and patency and flow direction of hepatic veins, splenic vein, and IVC were determined. The portogram performed at the end of the procedure was compared with the 24-h sonographic studies after TIPS to determine sonographic/angiographic correlation. No intraparenchymal abnormalities or perihepatic fluid collections were detected after the procedure. The metallic stent was clearly seen in all patients. Mean peak shunt flow velocities were  $139 \pm 50$  cm/sec within 24 h after TIPS. Absence of flow through the shunt was correctly identified in one case and confirmed angiographically. Mean peak flow velocity in the portal vein before TIPS was  $22 \pm 13.6$  cm/sec and increased to  $43.6 \pm 9.1$  cm/sec after TIPS ( $p < 0.05$ ). The hepatic artery peak systolic velocity increased from  $77 \pm 51$  cm/sec before TIPS to  $119 \pm 53$  cm/sec after the

procedure ( $p = 0.029$ ). Conventional duplex and color Doppler ultrasound proved to be a useful non-invasive diagnostic method to assess patients who have undergone TIPS. We propose its use as the primary diagnostic modality in these patients.

**Key words:** TIPS—Ultrasound, liver—Hepatic artery—Portal hypertension

The transjugular intrahepatic portosystemic shunt (TIPS) is a relatively new procedure that provides an alternative to surgically created portosystemic shunts in the treatment of the complications of portal venous hypertension, particularly bleeding varices [1–3]. The shunt is created by forming a tract from the hepatic venous circulation to the portal venous system through the hepatic parenchyma; patency of the shunt is maintained with metallic stents [1, 2]. The long-term clinical impact of TIPS procedures has not yet been established and the expected hepatic hemodynamic changes involving the shunts themselves, the portal and the hepatic venous systems, and the hepatic artery are not yet understood. Color Doppler ultrasound has been used successfully in the follow-up of patients with surgically created portosystemic shunts, decreasing the need for angiographic evaluation [4–7]. On this basis, we decided to establish a protocol to evaluate all patients undergoing TIPS at our institution using duplex and color Doppler sonography. The purpose of this paper is to describe our findings in an attempt to define expected hemodynamic changes and to present the correlation of sonographic findings with angiography.

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## Materials and Methods

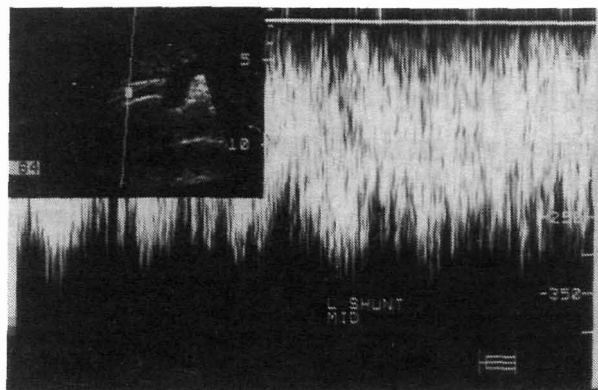
Nineteen patients (11 females and 8 males) underwent TIPS between September 1991 and July 1992. The mean age of the patients was 49.6 years (range 18–81 years). All of our patients had chronic liver disease and portal hypertension. The underlying cause of liver cirrhosis was ethanol abuse in 11 patients, viral hepatitis in 3, chemotherapeutic drugs in 2, congenital hepatic fibrosis in 2, and portal hypertension secondary to diffuse metastatic liver disease in 1 patient. The indication for TIPS was bleeding esophageal varices in 9 patients and control of refractory ascites in 10. The technical aspects of the TIPS procedure have previously been described [1–3]. In our series, the Wallstent (Schneider Inc., Plymouth, MN) was utilized to create the portosystemic shunt in all cases. The technical success was 100%. Portal venograms of the newly created shunt were performed in all patients before the procedure was terminated.

All patients undergoing TIPS in our institution are entered into a prospective study protocol which includes routine sonographic and angiographic shunt evaluation. Sonography is routinely performed within 24 h before TIPS, within 24 h after TIPS, at the time of discharge from the hospital, at 1–2 months, at 3 months, and at 3-month intervals thereafter. Angiography is performed at the time of the TIPS procedure and at 6-month follow-up intervals. Additionally, angiographic evaluation of the shunt is performed at any interval if suspicion of shunt occlusion or malfunction is found on sonographic evaluation.

Sonographic studies are performed using an Ultramark-9 HDI unit (Advanced Technology Laboratories, Bothell, WA, USA) or an Acuson-128 XP-10 unit (Acuson, Mountain View, CA, USA). Conventional duplex and color Doppler imaging are performed with phased and curved linear array and sector transducers (2.25, 3.5, and 5.0 mHz). Studies performed within the 24 h before the TIPS procedure include a thorough vascular assessment using conventional duplex and color Doppler sonography. Angle correction is used in all velocity determinations. The main, right, and left portal veins are carefully studied to determine patency, flow direction, and flow velocity. Hepatic artery patency and peak systolic velocities are determined. The splenic vein, hepatic veins, and inferior vena cava are examined for patency and flow direction.

The sonographic study performed within 24 h after TIPS focuses on shunt function and hemodynamic changes in the hepatic arterial and portal circulations, and careful evaluation of the parenchyma and perihaptic spaces for the detection of fluid collections. Stent position and anatomic relation to the portal and hepatic veins are determined. Peak shunt flow velocities are obtained in the proximal (hepatic vein) end, midportion, and distal (portal vein) segments of the shunt. The purpose of the 24-h post-TIPS study is twofold: 1) to detect any immediate complications, and 2) to establish normal baseline values for shunt flow velocities, hepatic artery peak systolic velocity, and portal hemodynamics in all patients. Sonographic studies performed within 24 h after TIPS are compared with the portogram done at completion of the procedure. The portogram is then considered the diagnostic gold standard. In cases in which sonographic findings suggest shunt occlusion, angiographic evaluation of the shunt is recommended. In those patients who undergo angiographic evaluation as a result of abnormal findings, the portogram performed after ultrasound is used for comparison and considered the gold standard.

This paper addresses the results of sonography 24 h before and 24 h after TIPS and compares these findings to the post-TIPS portogram in an effort to establish expected sonographic hemodynamics in the shunt and hepatic vasculature following TIPS. Descriptive analysis of the data obtained from the pre-TIPS evaluation and 24-h follow-up studies are presented using mean values  $\pm$  1 SD. Mean velocities are compared using the paired *t*-test; *p*-values of less than 0.05 are considered statistically significant.



**Fig. 1.** Doppler sonography through the midportion of a well-functioning shunt demonstrates the expected turbulent high velocity blood flow (300 cm/sec) within the stent. The walls of the stent are well visualized within the liver parenchyma.

## Results

Nineteen patients who underwent TIPS at our institution are included; 17 were evaluated before TIPS. In two patients, the TIPS procedure was performed emergently due to rapid clinical deterioration, and an ultrasound could not be obtained before TIPS. All 19 patients were evaluated within 24 h after TIPS. The stents were clearly seen in all 19 patients. The walls of the stent were seen as highly echogenic parallel lines (Fig. 1). The liver parenchyma provided an excellent window for stent visualization. No intrahepatic fluid collections suggestive of hematoma or biloma were identified in any of the patients after TIPS.

### Sonographic-Angiographic Correlation

Within 24 h of a technically successful TIPS, ultrasound detected the presence of shunt flow in 18 patients. Ultrasound correctly predicted the absence of blood flow in 1 case. An angiogram performed immediately after the ultrasound confirmed an occluded stent due to stent retraction and subsequent dislodgement from the portal vein lumen. Sonographic examination immediately following shunt revision with placement of an additional stent confirmed shunt patency. In one patient we incorrectly raised the question of nonocclusive thrombus at a site of high velocities (350 cm/sec) and incomplete color filling of the shunt. The stent was found to be normal at angiography. We now recognize this as

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**Table 1.** Peak stent velocities (cm/sec) at 24 h

	n	Mean ± SD	Min	Max
Proximal (HV)	14	171 ± 69	60	300
Midportion	17	155 ± 63	60	300
Distal (PV)	17	106 ± 73	20	275

**Table 2.** Hepatic artery peak systolic velocity (cm/sec)

	n	Mean ± SD	Min	Max
Pre-TIPS	15	75 ± 49	20	170
24 h post-TIPS	17	118 ± 55	35	220

a technical pitfall of ultrasound which we did not recognize early in our learning curve.

#### Shunt Flow Dynamics

The shunts of all 19 patients were sonographically evaluated within 24 h after successful TIPS. The color flow pattern within the shunt is characterized by complete filling of the shunt lumen with color, and evidence of turbulent, high velocity flow manifested by color aliasing which appears predominantly in the most proximal segment of the shunt. Due to technical factors, a complete complement of shunt velocity measurements was not obtained in all 19 patients. However, at least one shunt velocity determination was made in each patient. Peak velocities were obtained in the proximal (hepatic vein) portion in 14 patients, in the midportion in 17, and in the distal (portal vein) segment in 17 patients. These velocities were used in our statistical analysis. At 24 h, the mean peak velocity in the proximal portion of the stent was 171 ± 69 cm/sec, in the midportion it was 155 ± 63 cm/sec, and in the distal portion, 106 ± 73 cm/sec (Table 1). The difference in peak velocities within the different segments of the stent was not statistically significant.

The mean shunt flow velocity in the 19 patients evaluated at 24 h was 139 ± 50 cm/sec. In two patients, flow through the shunt could not be demonstrated when using the 3.5 MHz transducer. This technical problem was overcome by using a 2.25 MHz transducer to examine the patients; adequate flow through the shunt was demonstrated in both cases.

#### Portal Vein and Hepatic Artery Velocity Changes

The flow velocity in the main portal vein before TIPS was 22 ± 13.6 cm/sec, with antegrade portal flow

in all patients. After TIPS, portal vein flow velocity was 43.6 ± 9.1 cm/sec ( $p < 0.05$ ). Antegrade portal venous flow was confirmed in the main portal vein in all patients after the procedure.

Hepatic artery peak systolic velocity was obtained in 15 of the 19 patients in the pre-TIPS evaluation and in 17 patients at the 24-h follow-up. Two patients did not have an ultrasound before TIPS. In two patients with massive ascites, the hepatic artery peak systolic velocity could not be adequately assessed due to technical factors before TIPS and at 24 h after TIPS, and were excluded from analysis. Mean hepatic artery peak systolic velocity was 75 ± 49 cm/sec in the pre-TIPS patient group and increased to 118 ± 55 cm/sec at the 24-h follow-up (Table 2). In the 14 patients who had both pre- and 24-h post-TIPS hepatic artery velocities available, the mean peak systolic velocity increased from 77 ± 51 cm/sec to 119 ± 53 cm/sec. The mean difference between the pre- and post-TIPS values was 42 cm/sec ( $p = 0.029$ ).

#### Discussion

In the establishment of a new therapeutic vascular procedure such as TIPS, development of sensitive and specific noninvasive means of postprocedural evaluation is desirable. Early literature concerning TIPS has largely focused on technique, patency rates, clinical outcomes, and angiographic studies [1-3]. Two recent articles have been written on the duplex sonographic evaluation of patients undergoing this procedure, and have shown that sonographic evaluation of the TIPS shunt is an adequate, accessible, noninvasive method that provides useful hemodynamic information in this complex set of patients [8, 9]. Taking into account that duplex sonography has been shown to be an effective means of evaluating surgical portosystemic shunts [4, 5, 7], we think it has considerable potential in the evaluation of patients who have undergone TIPS, in part because of the ease of intrahepatic stent visualization in the majority of patients [8, 9]. The additional use of color Doppler sonography enhances vessel identification and flow documentation, thereby theoretically increasing the sensitivity of this examination.

Even in a prospective sonographic study such as this, acquisition of a perfectly complete database is difficult to accomplish for two reasons. First, all patients have advanced cirrhosis, often with ascites, and can be difficult to evaluate sonographically. Secondly, compliance with follow-up appointments and examinations is often less than desirable. Nonetheless, certain important findings are apparent.

Our data from functioning stents show that flow

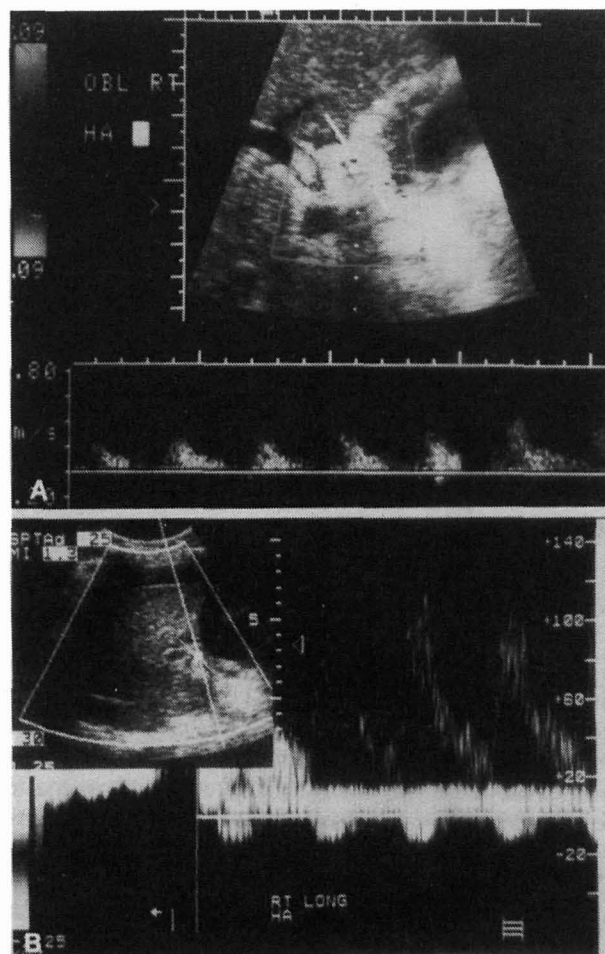


through the shunt is characterized by a broad range of velocities, typically with high peak velocities; this correlates with the findings of Surratt et al. [9]. Careful angle correction is required to obtain accurate flow velocity determinations. Such high flow velocities through the shunt might be expected, as the blood flow from the portal system is shunted directly from the main portal vein (usually 15–17 mm inner diameter) through a shunt that not only offers lower vascular resistance but also has an inner diameter of 8–9 mm [10]. The pressure differential afforded by a patent portosystemic shunt should direct the majority of splanchnic flow through a small caliber shunt; flow velocities would, therefore, be expected to be high. This also explains the increase in main portal vein flow velocities after TIPS, which was statistically significant. This finding correlates with those of Surratt et al. [9] and supports the strength of this finding.

Interestingly, we have found that flow velocities vary in different segments of the shunt, with a trend toward higher velocities at the proximal (hepatic vein) and midportions of the shunt; however, no significant differences in the velocities were found. Greater pressure differentials are expected in the proximal and midportions of the shunt and, therefore, are probably responsible for the slightly higher flow velocities in these segments. We anticipate that analysis of stent flow velocity will provide useful information regarding shunt function as further clinical experience is obtained [11].

The lowest flow velocity obtained in our group of patients was 20 cm/sec. This low value in retrospect was probably a technical error. This was the second TIPS patient we studied and we probably chose a suboptimal angle and cursor position. Nonetheless, flow velocities in this particular patient consistently remained well below the mean for this patient population.

Duplex sonography has been shown to be an excellent method for determination of shunt patency [4, 5, 8, 9], and in our series there was excellent sonographic/angiographic correlation within 24 h of TIPS when compared with the post-TIPS portogram. As surgical shunts are frequently difficult to visualize sonographically [4, 5, 12], one would expect comparable or better diagnostic results for sonography in TIPS patients, as the intrahepatic location of the shunt is more amenable to sonographic assessment. Our preliminary data support this. In addition, we have found that the use of lower frequency Doppler transducers (2.5 MHz) is helpful in examining large patients, patients with massive ascites, or shunts suspected to have low flow velocities if no flow signal is obtained within the shunt with a higher frequency Doppler capacity transducer. These tech-



**Fig. 2.** **A** Doppler sonography of the hepatic artery before TIPS demonstrates a monophasic waveform with peak systolic velocity of approximately 40 cm/sec. **B** Doppler sonography of the hepatic artery 24 h after TIPS (same patient as **2A**) demonstrates significant elevation of hepatic artery velocity to greater than 100 cm/sec. Superimposed portal venous flow is seen.

nical problems have also been addressed by Surratt et al. [9] and correlate with our findings.

The possibility of stenosis within the stent is a major diagnostic consideration in these patients. Metallic stents, in general, are susceptible to intimal hyperplasia and thrombus formation which may threaten adequate shunt function and even patency [13]. As this may be corrected by balloon angioplasty and/or atherectomy before complete shunt occlusion occurs, early detection is critical. We hope that the establishment of expected shunt velocities immediately after TIPS will provide an important baseline in these patients. Any significant deviation from expected normal velocities might indicate shunt mal-

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function. More data will be needed to support this theory.

There is a significant increase in hepatic artery velocity within the first 24 h following TIPS (Fig. 2A and B). Successful placement of the shunt diverts the portal flow from the liver. Given that most of the blood flow to the liver is provided by the portal vein [14], even in patients with portal hypertension, we think that this elevated hepatic arterial flow in shunted patients is a hemodynamic response to preserve liver perfusion [15], previously described as hepatic arterial buffer response [16–18]. Similar findings have been documented in series analyzing nonselective, surgical portosystemic shunts [14]. Further analysis of patients with functional and dysfunctional stents will be required to establish whether this hepatic artery response has any prognostic significance related to stent or hepatic function or to patient survival.

We conclude that sonography is an excellent noninvasive method of assessing the hemodynamic changes in patients who have undergone TIPS. The duplex and color Doppler sonographic criteria used to characterize properly functioning and dysfunctional stents will likely be more clearly defined in the near future with careful and thorough assessment of these complicated, but interesting, patients.

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## References

- Zemel G, Katzen BT, Becker GJ, Benenati JF, Sallee DS (1991) Percutaneous transjugular portosystemic shunt. *JAMA* 266:390–393
- Zemel G, Becker FJ, Bancroft JW, Benenati JF, Katzen BT (1991) Technical advances in transjugular intrahepatic portosystemic shunts. *RadioGraphics* 12:615–622
- Richter GM, Noeldge G, Palmaz J, Roessle M, Siegerstetter V, Frank M, Gerok W, Wenz W, Farthmann E (1990) Transjugular intrahepatic portacaval stent shunt: Preliminary clinical results. *Radiology* 174:1027–1030
- Grant EG, Tessler FN, Gomes AS, Holmes CL, Perrella RR, Dverinckx AJ, Busuttill RW (1990) Color Doppler imaging of portosystemic shunts. *AJR* 154:393–397
- Helton WS, Montana MA, Duyer DC, Johansen K (1988) Duplex sonography accurately assesses portacaval shunt patency. *J Vasc Surg* 8:657–660
- Patriquin H, Lafortune M, Burns PN, Dauzat M (1987) Duplex Doppler examination in portal hypertension: Technique and anatomy. *AJR* 149:71–76
- Ackroyd N, Gill R, Griffiths K, Kossoff G, Reeve T (1986) Duplex scanning of the portal vein and portosystemic shunts. *Surgery* 99:591–597
- Longo JM, Bilbao JI, Rousseau HP, Garcia-Villareal L, Vinel JP, Zozaya JM, Joffre FG, Prieto J (1993) Transjugular intrahepatic portosystemic shunt: Evaluation with Doppler sonography. *Radiology* 186:529–534
- Surratt RS, Middleton WD, Darcy MD, Melson GL, Brink JA (1993) Morphologic and hemodynamic findings at sonography before and after creation of a transjugular intrahepatic portosystemic shunt. *AJR* 160:627–630
- Zieler RE (1990) Hemodynamic considerations in evaluation of arterial disease by Doppler US. *Clin Diagn Ultrasound* 26:13–22
- LaFortune M, Patriquin H, Pomier G, Huet PM, Weber A, Lavoie P, Blanchard H, Breton G (1987) Hemodynamic changes in portal circulation after portosystemic shunts: Use of duplex sonography in 43 patients. *AJR* 149:701–706
- Finn JP, Gibson RN, Dunn GD (1987) Duplex ultrasound in the evaluation of portacaval shunts. *Clin Radiol* 38:87–89
- Vorwerk D, Günther RW (1991) Stenting of peripheral arteries. *Semin Intervent Radiol* 8:264–272
- Sherlock S. (1989) Ascites In: Sherlock S (ed): *Diseases of the liver and biliary system*. 8th ed. Blackwell Scientific Publications, Oxford, 129–151
- Spina GP, Santambrogio R, Opocher E, Gattoni F, Baldini V, Cucchiario G, Uslenghi C, Pezzvoli G (1990) Early hemodynamic changes following distal splenorenal shunt for portal hypertension: Comparison of surgical techniques. *World J Surg* 14:115–122
- Henderson JM, Gilmore GT, Mackay GJ, Galloway JR, Dodson TF, Kutner MH (1992) Hemodynamics during liver transplantation: The interactions between cardiac output and portal venous and hepatic arterial flows. *Hepatology* 16:715–718
- Lautt WW (1985) Mechanism and role of intrinsic regulation of hepatic arterial blood flow: Hepatic arterial buffer response. *Am J Physiol* 249:G549–G556
- Lautt WW, Legare DJ, D'Almeida MS (1985) Adenosine as putative regulator of hepatic arterial flow (the buffer response). *Am J Physiol* 248:H331–H338

## Stent Implantation in Femoropopliteal Arteries Problems and Solutions

DIETER D. LIERMAN<sup>1</sup>

### ABSTRACT

**R**ecurrent stenosis or occlusions by intimal hyperplasia occurs in up to 40 % of any kind stent implantations in femoropopliteal arteries and greatly restricts the indication for stent implantations. Repeat balloonangioplasty (PTA) and subsequent prophylactic endovascular radiotherapy with a surfacedose of 12 Gy using an iridium 192 source was investigated as a means to reduce or eliminate further recurrence. 20 patients underwent confirmatory diagnostic atherectomy. PTA or laser recanalization and radiation in one session. All 20 had developed recurrent stenosis/occlusion 6-8 months after original stent implantations.

One of the 20 has redeveloped recurrent obstruction two years above the irradiated area. 19 patients have not redeveloped recurrent obstruction between 7 to 43 months after this treatment, which up to date showed no short term or long term complications. We conclude that this limited experience is promising enough to warrant further study.

An additional aspect is the treatment of severe dissections. We analyzed, in how far a transient stent implantation may be an alternative to permanent stentimplantation in the treatment of therapyresistant dissections.

### INTRODUCTION

**T**he development of vascular endoprotheses or stents opened new treatment options for arterial occlusive disease unmanageable with balloon PTA alone (1-8). PTA and stent implantation are associated with iatrogenic trauma to the vessel wall considered the trigger mechanism for intimal hyperplasia (10-18). Therefore, recurrent stenosis secondary to intimal hyperplasia remained a

frequent problem for infrainguinal arterial stents usually within 6 months after placement (9,19-21). For the peripheral stents recurrent stenosis and occlusion reaches up to 40 % (9).

The good results reported for the radiotherapy of keloids (22-25) prompted us to develop our therapeutic concept of prophylactic irradiation to reduce this hyperplastic vessel wall reaction. Small caliber radiation sources using the afterloading technique were previously applied to bile ducts (26). We applied this technique of endovascular radiotherapy (ERAD) to 20 patients with recurrent stenosis after stenting.

An additional aspect is the treatment of severe dissections. In most cases a repeated PTA with inflation for time periods of 2 min to 5 min is sufficient to cause an occlusion of the false lumen after dissections and to avoid any surgical treatment. In cases of persistence of dissection with severe flowreduction, the stent implantation is the only interventional method to guarantee a patent lumen. Unfortunately we had a lot of restenosis in case of permanent stent implantation in case of intimal hyperplasia. We analyzed, in how far a transient stent implantation may be an alternative to permanent stentimplantation in the treatment of therapyresistant dissections.

### MATERIAL AND METHODS

**P**atients older than 70 years with recurrent, clinically relevant stenoses or occlusions in the stented vascular segment less than 8 months after placement and without contraindications to anticoagulation were eligible for ERAD.

Before undergoing repeat balloon PTA or laser recanalization with a special matted sapphire tip which allows treatment without subsequent PTA in the stented vascular segment, biopsy by Simpson

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atherectomy was performed for histological confirmation of intimal hyperplasia. So far we have treated 8 women and 12 men, aged 70 - 84 years. All patients had a history of several PTAs for occlusions and reocclusions in the superficial femoral artery before being treated by placement of one or several tantalum stents. The length of the stented vascular segments ranged from one to 14 cm. In all 20 cases, histology revealed intimal hyperplasia as the cause of the restenosis.

ERAD was performed under heparinization. After PTA or laser recanalization of the restenosed, stented vascular segments, a 9 French straight catheter (William Cook, Europe) was introduced through a 9 F femoral introducer sheath over a guidewire until its tip was positioned below the segment to be treated. The 9 F catheter tip was tapered (0.5 cm) so that the coaxially introduced 5 F end-occluded catheter (Nucletron, Veenendal, Netherlands "Theranostic") and the calibrated wire in it could not advance through its tip. The calibrated wire with a diameter of 1,1 mm (Nucletron, Veenendal, Netherlands "Theranostic") first determined the exact length of the stented vascular segment which determined the distance to which the afterloading probe was later inserted. The 9 F catheter and the coaxial advanced end occluded 5 F catheter with the calibrated wire was positioned so, that its distal section marks the lower end of the irradiation field which extended 1 cm beyond the actual stent location. The calibrated wire was then withdrawn while the 5 F end-occluded catheter and the 9 F catheter remains in place. The 5 F catheter accommodates the 1,1 mm iridium probe, which was of the same diameter as the calibrated wire, after being connected to the outlet valve of the iridium 192 source. We used a Nucletron (Micro) Selectron High Dose Radiation (HDR) treatment planning system (Nucletron, Veenendal, Netherlands) version 10.10, for the exact calculation, supervision and guidance of the afterloading procedure. The radiation source was iridium 192 at a strength of 10,000 Ci. The reference dose was 12 GY delivered in about 200 sec, depending on the source activity and the isodose length. After ERAD the catheters were removed and manual pressure applied carefully to the puncture site for 10-20 minutes. Patients were systemically heparinized for 72 hours and continued on Coumarin anticoagulation for 6 months.

Clinical patient follow-up was performed including ankle arm indices (AAI) before, immediately after, 3 and 6 months later, and then every 6 months. Additional examination by Magnetic resonance

angiography (MRA) was performed before and after the procedure and at 6- month intervals using FLASH gradient spin-echo sequences with a flip angle of 30° in a vertical and coronal projection with a 1.0 Tesla unit and a neck coil (26). In addition intravenous DSA and high resolution CT were performed after 6 and 12 months.

Concerning the aspect of severe dissections we used a temporary stentimplantation in peripheral arteries. In 10 cases of a therapyresistant dissection after recanalization of an occluded vesselsegment we implanted tantalum stents in the dissection area for at least 24 h to 48 h. The length of the stented vesselsegment ranged from 4 cm to 12 cm, the diameter from 5 mm to 6 mm. The introducer sheath remained at the punctureside and the patients get 1000 I.U. of heparin/h using a perfusion system for the whole time. After this period, the stents can be easily withdrawn by a special forcepsystem without any damage of the vesselwall. This technique had been used since experimental extractions showed excellent results for the tantalum stent while other stent types can not be extracted interventionally.

## RESULTS

In all 20 patients, repeat PTA was performed without leaving behind any residual stenosis in the stented segment. Subsequent ERAD with 192 iridium delivered 12 Gy to the vessel wall in all cases, with an exposure time of about 200 sec. ERAD added about 45 minutes in each case; most of this extra time was taken up by transporting the patients between the interventional lavatory and the afterloading room, which are located in different buildings. No post-procedure bleeding at the puncture site occurred. Follow-up for 2 patients is now 43 months and the mean follow-up for all is 21 months. The clinical stage improved in 11 patients from Fontaine's stage IIb to stage I and in 7 patients from stage III to stage I. The improvement in 2 cases was only from stage II to stage IIa, the limiting factor in these cases being a contralateral occlusion. No deterioration of the clinical stage and no restenosis was found at follow-up in 19 patients. Occlusion in one case in the origin of the SFA two years after irradiation therapy cannot be probed. This patient was treated by bypass surgery. Neither CT and MRA revealed any radiation-induced changes to the tissues surrounding the vessel. The patients did not report any discomfort during or after ERAD and no delayed complications have emerged to date.

Concerning the indication for temporary stent implantation, all dissections had disappeared after successful treatment. All stents had been easily removed by a special forceps after 24 h to 48 h without any damage of the vessel wall. The patients were followed between 3 months and 14 months without restenosis. All patients were treated with cumarine or ASS as a long term therapy. For the follow-up is relative short, a comparative analyzes to permanent stent implantation is to early.

## DISCUSSION

At this early stage, ERAD as a therapeutic concept to prevent recurrent intimal hyperplasia in stented vascular segments should be performed only after very careful consideration of the potential benefits and risks. The method can be compared with low dose irradiation of non-malignant lesions, e. g. prophylactic irradiation against keloids, irradiation of hemangiomas, Peyronie's disease or any anti-inflammatory radiotherapy (22-25). Use of the contact radiation therapy in afterloading technique has to be strictly separated from any kind of external beam irradiation. It keeps the radiation dose to the tissue surrounding the target organ and consequently at the same time the somatic risk to a minimum. The dose for ERAD is derived from treatment of keloids. Irradiation of keloids is performed as fractionated contact therapy to a fixed total dose. The radiation source is strontium 90 (25). We calculated the equivalent dose for single step iridium 192 afterloading treatment as 12 Gy. Treatment of a vessel wall by fractionated radiation would be impractical. Compared to external beam irradiation, the contact radiation dose decays steeply protecting the surrounding tissue. The dose of intraluminal iridium 192 drops from a surface dose of 12 Gy to 8.77 Gy at 4 mm, 5.51 Gy at 6 mm, 3.96 Gy at 8 mm and 3.03 Gy at 10 mm (Fig. 3). The risk of damage to the adjacent nerve tissue can be regarded as low. Reported tolerance of peripheral nerves to a single radiation dose is 15 Gy (27,28). For ethical reasons and in order to minimize the risk of radiation-induced malignant transformation, our pilot study was restricted to patients over 70 years old. In addition, these patients had a history of multiple restenoses of the particular vascular segment at short intervals even before stent placement and a Fontaine's stage of IIb to IV. We discussed our therapeutic concept and any alternatives in detail with the patients. Intraarterial afterloading therapy was feasible as small caliber sources were available from developments made for malignant lesions of the bile

duct (29).

Theories about the development of intimal hyperplasia in arteries (10, 11, 12), and the role of PTA (13,15) or stents (16, 17, 18, 30) led us to adopt the concept of treatment for keloids (20-25) to the treatment of intimal hyperplasia. Although the small number of patients so far treated does not allow any definitive conclusions, we think there is a good chance that this treatment suppresses hyperplasia inside the stents. The initial results are certainly encouraging. With the exception of the above-described somatic risk of irradiation, we do not see any other relevant short-term or long-term side effects or complications. One potential short-term effect that could have been expected is an increased risk of thrombosis secondary to edema or inflammatory reaction to the radiation, but this was not observed in any case. Long-term effects of radiotherapy such as scar formation with arterial constriction after about 50 Gy and are unlikely to occur after the endovascular application of 12 Gy. The dose used by us, however, causes significant reduction of mitosis only in the most exposed cells and only partial cell necrosis. This effect together with the reduction of the rate of myofibroblast migration might be the reason for the absence of restenosis in our population. Of all the suggested treatment regimes for intimal hyperplasia (31-39), ERAD is the only one so far that can be used successfully under clinical conditions. The discovery of mediators of intimal hyperplasia secondary to any kind of vessel wall injury led to the development of substances which disrupt or reduce this process. Unfortunately, the substances are directed against only one or two of the growth factors, while others remain unaffected (40-45). Another model favors a genetic influence on the vessel wall tissue (46-49). The results of experiments with the implantation of so-called covered stents in animals are not particularly encouraging, as hyperplasia occurs at the transition zone between stent and vessel wall (50,51).

There are also some preliminary experimental models in which the stent is prepared with heparin or chemotherapeutic substances in order to reduce intimal hyperplasia after stent implantation (51). At least some biocompatible new stent materials with different characteristics have been developed in animal models (52,53). However, most of these models have not yet been tested clinically. We conclude that the ultimate value of stents in the therapy of arterial occlusive disease of femoropopliteal arteries depends on successful

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inhibition of intimal hyperplasia. ERAD presents a promising clinical technique.

The first positive results after transient stentimplantation may show an alternative to permanent stentimplantation and surgical therapy in case of severe dissections in peripheral vessels. Tantal stents are the best for easy rescue after a defined time without damage of the vesselwall, while other commercial available stent types are not retrievable by interventional methods. The use of the tantalum stent in treatment of severe dissections can be recommended.

#### Literature

1. Cragg, A., Lund, G., Rysavy, J., Castaneda, F., Castaneda-Zuniga, W., Amplatz, K. (1983) Nonsurgical placement of arterial endoprotheses: A new technique using nitinol wire. *Radiology* 147: 261-263.
2. Mass, D., Zollikofer, C.L., Largadere, F., Senning, A. (1984) Radiological Follow up of transluminal inserted vascular indoprotheses: an experimental study using expanding spirals. *Radiology* 152: 659-663.
3. Palmaz, J.C., Sibbitt, R. R., Reuter S. R., Tio, F. O., Rice, W. J. (1985) Expandable intraluminal graft: A preliminary study. *Radiology* 156: 73-77.
4. Wright, K.C., Wallace, S., Charsangavej, C., Carrasco, CH., Gianturco. (1985) Percutaneous endovascular stents: an experimental evaluation. *Radiology* 156: 69-72.
5. Duprat, G., Wright, K., Charsangavej, C., Wallace, S., Gianturco, C. (1987) Self expanding metallic stents for small vessels: an experimental evaluation. *Radiology* 162: 469-472.
6. Sigwart, U., Puel, J., Mirkovitch, V., Joffre, F., Kappenberger, L. (1987) Intravascular stents to prevent occlusion and restenosis after transluminal angioplasty. *N Engl. J. Med.* 316: 701-706.
7. Strecker, E. P., Ber, G., Schneider, B., Freudenberg, N., Weber, H., Wolf, H.R.D. (1988) A new vascular balloon-expandable prosthesis; experimental studio and first clinical results. *J. Interv. Radiol.* 3: 59-62.
8. Rabkin, J. E., Germanshev, V. G. *The Rabkin Nitinol Coust Stent: A Five Year Experience in Intervent. Radiol.*, W. R. Castaneda-Zuniga, S. M. Tadavarthy Ed. Williams and Wilkins Baltimore, 1992; Vol II: 576-581.
9. Liermann, D., Strecker, E. P., Peters, J. (1992) *The Strecker Stent: Indications and Results in iliacal and femoropopliteal arteries.* *J. Cardio Vasc. Interv. Radiol.*, 15-5: 298-305.
10. Imparato, A. M., Bracco, A., Kim, G. E., Zeff, R. (1972) Intimal and neointimal fibrous proliferation causing failure of arterial reconstructions. *Surgery* 172; 6: 1007-1017.
11. Spaet, T.H., Stemermann, M.B., Veith, F.J., Lejniaks, I. (1975) Intimal injury and regrowth in the rabbit aorta. Medial smooth muscle cells as a source of neointima. *Circ. Resa* 36:58-70.
12. Clowes. A. W., Karnovsky, M.J. (1977) Suppression by heparin of smooth muscle cell proliferation in injured arteries. *Nature* 265:625-626.
13. Laerum, B.F., Vloder, Z., Castaneda-Zuniga, W.R., Edwards, J.E., Amplatz, K. (1982) The mechanism of angioplasty. *Fortschr. Röntgenstrahlen* 136:573-576.
14. Le Veen, R.F., Wolf, G.L., Villanueva, T.G. (1983) New rabbit atherosclerotic model for the investigation of transluminal angioplasty. *Invest. Radiol.* 17:470-475.
15. Faxon, D.P., Sanborn, T.A., Weber, V.J. (1984) Restenosis following transluminal angioplasty in experimental atherosclerosis. *Arteriosclerosis* 4:189-195.
16. Palmaz, J.C., Hunter, G., Carson, S.N., French, W.S. (1983) Postoperative carotid restenosis due to neointimal fibromuscular hyperplasia. *Radiology* 148-3:699-702.
17. Rollins, N., Wright, K.C., Charsangavej, C., Gianturco, C., (1987) Selfexpanding metallic stents: preliminary evaluation in an atherosclerotic model. *Radiology* 163:739-742.
18. Rousseau, H., Joffre, F., Raillat, C., Julian, M., Pierragi, M.T., Chlabi, F., Bouissou, H. (1989) Self expanding endovascular stent in experimental atherosclerosis. *Radiology* 170:773-778.
19. Triller, J., Mahler, F., Thalmann, D.H.R. (1989) Die vaskuläre Endoprothese bei femoropoplitealer Verschlusskrankheit. *Fortschr. Röntgenstr.* 150:328-334.
20. Günther, R.W., Vorwerk, D., Bohndorf, K., Peters, I., El Din, A., Mesmer, D. (1989) Iliac and femoral artery stenosis and occlusion: treatment with intravascular stents. *Radiology* 172:725-730.
21. Rousseau, H., Puel, J., Joffre, F., Sigwart, U., Duboucher, C., Imbert, C., Knight, C., Kropf, L., Wallsten, H. (1987) Selfexpanding endovascular prosthesis: an experimental study. *Radiology* 164:709-714.
22. Baensch, W. (1937) Über die Strahlenbehandlung der Keloide. *Strahlentherapie* 60:204-209.
23. Krüger, A. (1954) Über Keloide und Ihre Behandlung

**IV INTERNATIONAL COURSE ON VASCULAR AND INTERVENTIONAL RADIOLOGY AS THERAPEUTIC ALTERNATIVE**

- unter besonderer Berücksichtigung der Strahlentherapie.  
Strahlentherapie 93:426-433.
24. Levy, D.S., Salter, M.M., Roth, R.E. (1981) Postoperative Irradiation in the Prevention of Keloids. *Am. Roentg.* 127:509-510.
  25. Wagner, W., Schopohl, B., Böttcher, H.D., Schnepfer, E. (1989) Ergebnisse der Narbenkeloidprophylaxe durch Kontaktbestrahlung mit Strontium 90. *Röntgenpraxis* 42:248-252.
  26. Liermann, D., Berkefeld, J. (1993) MRI Examination of implanted endovascular prosthesis in vessels - First results In *Stents*, Liermann, D., ed., Marianne Liebert, New York in press.
  27. Le Couteur, R.A., Gilette, E.L., Powers, B.E., Child, G., Mc Chesney, S.L., Ingram, J.T. (1989) Peripheral neuropathies following experimental intraoperative radiation therapy (IORT) *Int. J. Radiat. Oncol. Biol. Phys.* 17:583-590.
  28. Kinsella, T.J., Sindelar W.F., De Luca, A.M. (1988) Threshold dose for peripheral nerve injury following intraoperative radiotherapy (IORT) in a large animal model. *Int. J. Radiat. Oncol. Biol. Phys.* 1:205-211.
  29. Brambs, H.J., Freund, U., Bruggmoer, G., Wannemacher, M. (1987) Kombinierte intraduktale perkutane Radiotherapie bei malignen Gallengangsobstruktionen mit anschließender prothetischer Versorgung. *Onkologie* 10:84-89.
  30. Barth, K.H., Virmani, R., Strecker, E.P., Savin, M., Lindisch, D., Matsumoto, A.H., Teitelbaum, G.P. (1990) Flexible tantalum stents implanted in aortas and iliac arteries: Effects in normal canines. *Radiology* 175:91-96.
  31. Betz, E., Hämmerle, H., Strohschneider, T. (1984) Vergleich von Wirkungen einzelner Pharmaka auf die Proliferation von Gefäßmuskulzellen in vivo und in vitro in Gefäßwandelemente in vivo und in vitro. H. Fischer, E. Betz Eds.:43-57. *Wiss. Verlagsgesellschaft Stuttgart, Germany.*
  32. Heras, M., Chesebro, J.H., Penny, W.J., Baily, K.R., Badimon, K., Fuster, V. (1989) Effects of thrombin inhibition on the development of acute platelet thrombus deposition during angioplasty in pigs. Heparin versus recombinant hirudin, a specific thrombin inhibitor. *Circulation*, 79:657-665.
  33. Courrier, J.W., Pow, T.K., Haudenschild, C.C., Miniham, A.C., Faxon, D.P. (1991) Low molecular weight heparin (enoxaparin) reduces restenosis after iliac angioplasty in the hypercholesterolemic rabbit. *J. Am. Coll. Cardiol.*, 17:118B-125B.
  34. Hagen, B. (1993) Einflüsse der medikamentösen Nachbehandlung auf die mittelfristigen Ergebnisse von Stent-Implantationen der A. femoropoplitea. In *Stents*, Liermann, D. Ed., Marianne Liebert, New York in press.
  35. Ellis, S. G., Roubin, G. S., Wilentz, J., King, S. B., Douglas, J., Weintraub, W. S., Thomas, R. G., Cox, N. R. (1987) Results of a randomized trial of heparin and aspirin vs. aspirin alone for prevention of acute closure and restenosis after angioplasty (PTCA). *Circulation*; 76: 213-218.
  36. Hoepf, L. M., Elbadawi, M., Cohn, M., Dachelet, R., Peterson, C., De Weese, J. A. (1979). Stenosis and immunosuppression effect on anastomotic intimal hyperplasia in femoral arterial dacron bypass grafts. *Arch Surg*, 114: 273-276.
  37. Pepine, C. J., Hirschfeld, J. W., MacDonald, R. G. (1990). A controlled trial of corticosteroids to prevent restenosis after coronary angioplasty. *Circulation*, 81: 1753-1761.
  38. Reis, G. J., Boucher, T. M., Sipperly, M. E. (1989) Randomized trial of fish oil for prevention of restenosis after coronary angioplasty. *Lacet*; 2: 1753-1761.
  39. Kramach, D. M., Aspen, A. J., Rozler, L. J. (1980) Suppression of experimental atherosclerosis by the Ca + + antagonist lanthanum. *J. Clinical Invest.*; 65: 967-981.
  40. Powell, J. S., Clozel, J. P., Müller, R. K. (1989) Inhibitors angiotensin-converting enzyme prevent myointimal proliferation after vascular injury. *Science*, 245: 186-188.
  41. Liu, M. W., Roubin, G. S., Robinson, K. A. (1990) Trapidil in preventing restenosis after balloon-angioplasty in the atherosclerotic rabbit. *Circulation*, 81: 1089-1093.
  42. Dartsch, P. C., Betz, E., Ischinger, T. (1991) Wirkung von Dihämatoporphyrin-Derivaten auf kultivierte glatte Muskelzellen des Menschen aus normalen und atherosklerotisch veränderten Gefäßsegmenten-Übersicht über bisherige Ergometrische Therapie *Z. Kardiol.*, 80: 6-14.
  43. Betz, E., Hämmerle, H., Strohschneider, T. (1985) Inhibition of smooth muscle cell proliferation and endothelial permeability with flurazazine in vitro and in experimental atheromas. *Res. Exp. Med.*, : 325-340.
  44. Nilsson, Jan, Sjölund, M., Palmberg, L., Thyberg, J., Heldin, C. H. (1985) Arterial smooth muscle cells in primary culture produce a platelet-derived growth factor-like protein. *Cell Biol.*, Vol 82: 4418-4422.
  45. Castellot, J. J., Jr., Addonizio, M. L., Rosenberg, R., Karnovsky, M. J. (1990). Cultured endothelial cells produce a heparin like inhibitor of smooth muscle cell growth. *J. Cell Biol.*, : 373-379.
  46. Dichak, D. A., Neville, R. F., Zwiebel, J. A., Freeman, S. M., Leon, M. B., Anderson, W. F. (1989) Seeding of intravascular stents with genetically engineered



#### **IV INTERNATIONAL COURSE ON VASCULAR AND INTERVENTIONAL RADIOLOGY AS THERAPEUTIC ALTERNATIVE**

- endothelial cells.  
*Circulation*, 80: 1347-1353.
47. Nabel, E. G., Plautz, G., Nabel, G. J. (1990)  
Site specific expression in vivo by direct gene transfer into the arterial wall.  
*Science*, 249: 1285-1288.
48. Wilcox, J. N. (1991)  
Analysis of local gene expression in human atherosclerotic plaques by in situ hybridization.  
*Trends Cardiol. Med.* , 1: 17-24.
49. Leclerc, G., Inan, J. M., Kearny, M., Simons, M., Safian, R. D., Bain, D. S., Weir, L. (1992)  
Evidence implicating nonmuscle myosin in restenosis: Use of in situ hybridization to analyze human vascular lesions obtained by directional atherectomy.  
*Circulation*, 85: 1-11.
50. Roeren, T., Palmaz, J. C., Garcia, O., Rees, C. R., Tio, F. O. (1990)  
Percutaneous vascular grafting with a coated stent.  
*Radiology*, 177: 202.
51. Strecker, E. P., Hagen, B., Liermann, D., Wakhloo, A. Y., Schneider, R., Mayer, S., Freudenberg, N. (1992)  
Experimental and clinical experiences in the prevention and treatment of stent-restenoses.  
*Abst. IAGS., Nürnberg, Vol I: 35.*
52. Slepian, M. J., Schindler, A. (1988)  
Polymeric endoluminal paving/sealing: a biodegradable alternative to intracoronary stenting.  
*Circulation*, 78: 400-414.
53. Murphy, J. G., Schwartz, R. S., Kennedy, K., Edwards, R. E., Vliestra, R. E., Holmes, D. R. (1990)  
A new biocompatible polymeric coronary stent: design and early results in the pig model.  
*J. Am. Coll. Cardiol.* , 1S: 10SA.



## Color Doppler US Guidance in Transjugular Placement of Intrahepatic Portosystemic Shunts<sup>1</sup>

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Percutaneous placement of portosystemic shunts requires access to the portal system from a transjugular approach. Color Doppler sonography was used to direct the transjugular puncture in intrahepatic portosystemic shunt procedures in four patients. In each case, the technique allowed quick, safe transjugular puncture of the portal vein and close real-time monitoring of the procedure.

**Index terms:** Hypertension, portal, 94.711, 95.711 • Liver, interventional procedure, 761.12986 • Portal vein, 957.711 • Shunts, portosystemic, 957.453, 982.453 • Ultrasound (US), Doppler studies

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**T**HE transjugular intrahepatic portosystemic shunt (TIPS) technique is emerging as a useful percutaneous alternative to sclerotherapy and surgery in the treatment of portal hypertension. The initial procedure, as reported by Richter et al (1,2), involved a combined transjugular and percutaneous transhepatic portal approach. After percutaneous transhepatic insertion of a stone retrieval basket into the desired portal branch, a needle was placed into the selected hepatic vein by way of the transjugular access. The basket was used as a target for the needle, which was then carefully advanced (under fluoroscopic guidance) out of the hepatic vein, through the liver parenchyma, and into the portal branch. Once the portal branch was reached, successive balloon dilatation and stent

placement allowed the creation of an intrahepatic portosystemic shunt.

Percutaneous transhepatic access to the portal system in advanced cirrhotic patients is not a risk-free procedure (3). In fact, the failure to embolize the transhepatic access tract was the cause of death in one of the nine patients included in the report by Richter et al (2). In view of this risk, most authors have abandoned the transhepatic puncture. Identification of the portal vein with transabdominal ultrasonography (US) or arterial portography has been used as a substitute in planning of the course of the shunt tract (4-8). We describe our experience with color Doppler US-guided transjugular placement of intrahepatic portosystemic shunts.

### Patients and Methods

During the past year, we performed TIPS procedures in 23 patients, by using a right jugular venous approach and no percutaneous transhepatic puncture. In the past four patients in whom we have performed the procedure, we used color-Doppler flow US guidance during the procedure.

**Representative case report.**—A 56-year-old woman was admitted with a history of liver cirrhosis, portal hypertension, ascites, and gastroesophageal varices. She had had five previous episodes of variceal bleeding, and all had been treated with endoscopic sclerotherapy, to which she had become nonresponsive. Surgical portal decompression was ruled out due to her poor clinical status. Placement of a TIPS was decided upon.

At US, a small, retracted liver without nodular parenchymal lesions was seen. Antegrade, hepatopetal flow in a 14-mm-wide portal vein was observed. Prophylactic antibiotics and sedatives were administered intravenously. The right internal jugular vein was punctured percutaneously, and a 10-F introducer sheath with a hemostatic valve (Cook Europe, Bjaeverskov, Denmark) was advanced into the inferior vena cava. A 7-F cobra catheter (USCI, Billerica, Mass) was successively placed into the right and middle hepatic veins, and hepatic venograms were obtained at various projections. The rest of the procedure was performed with additional US monitoring by a radiologist (J.M.L.) with experience in interventional procedures and US. A color Doppler flow system with a 3.5-MHz sector probe (128 XP/10; Acuson, Mountain View, Calif) was used. The presence of ascitic fluid

contributed to the easy visualization of the right lobe of the liver.

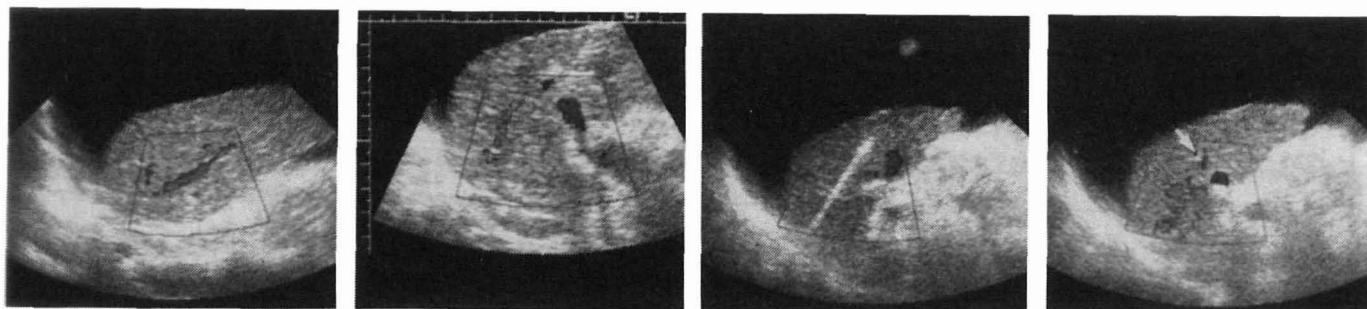
With color Doppler US, the positions of the right and middle hepatic veins in relation to the branches of the right portal vein were easily revealed. The right hepatic vein showed an excessively peripheral course, far from ideal for shunting purposes (Fig 1). The middle hepatic vein was selected, owing to its better orientation and close proximity to the anterior branch of the right portal vein (Fig 2).

With fluoroscopic guidance, a rigid 0.035-inch Amplatz guide wire (Mediatech/Boston Scientific, Watertown, Mass) was inserted into the middle hepatic vein (Fig 3). A transjugular needle (transjugular cholangiography set; Cook Europe) (16-gauge inner transeptal needle; 9-F outer Teflon catheter) was placed over the Amplatz guide wire and advanced into the middle hepatic vein; the tip was left at the middle portion of the vessel. After removal of the guide wire, under US guidance, the needle was advanced gently toward the nearby visualized portal branch. In contrast to the guide wire, which was easily visible at US, the smooth surface of the needle was not so clearly detected. Flash artifacts (9) caused by the displacement of the needle and adjacent liver parenchyma were observed during advancement of the tip (Fig 4). Despite these limitations, sufficient guidance was provided, and blood was aspirated after a single 3-cm advancement through the liver parenchyma in the preselected direction.

Introduction of contrast medium (Omnigraf 300; Juste S.A.Q.F., Madrid) allowed confirmation that a branch of the right portal vein had been reached (Fig 5), and a 0.035-inch guide wire (Radifocus; Terumo, Tokyo) was advanced into the portal system with both intermittent fluoroscopic and US guidance (Fig 6). After removal of the needle, a 5-F straight catheter (Cordis-Europe, Oosteinde, The Netherlands) was advanced distally over the guide wire, which was then replaced with a 0.035-inch Amplatz guide wire.

Dilation of the parenchymal tract connecting the portal system to the punctured hepatic vein was achieved by means of a 5-F 8-mm-diameter angioplasty balloon catheter (Schneider Europe, Buelach, Switzerland) placed over the Amplatz guide wire. Two 120-second, 8-atm inflations were enough to dilate the tract (Fig 7). The "waist"

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 RSNA, 1992



**Figures 1–4.** (1) Longitudinal color Doppler sonogram. The right hepatic vein (blue) is easily identified. Its trajectory, far away from the portal branches, makes it less than convenient for shunting. (2) A sagittal oblique image shows the favorable alignment of the middle hepatic vein (blue) to the right portal vein (red). (3) The echogenic Amplatz guide wire inserted into the middle hepatic vein is easily visible. (4) Color Doppler sonogram obtained during the hepatic puncture. Motion-related color (flash artifacts), although visually disturbing in the frozen image, does not hamper the correct advancement of the needle. The needle tip, reaching the portal vein branch, is identified as a small echogenic dot (arrow).

caused by the portal wall disappeared after 40 seconds of inflation. After the angioplasty balloon catheter was exchanged for a 7-F Wallstent introducer catheter (Medinvent, Lausanne, Switzerland), a 62-mm-long, 8-mm-diameter self-expanding prosthesis (Wallstent; Medinvent) was deployed with combined fluoroscopic and sonographic monitoring (Fig 8).

There is an inherent shortening of the Wallstent prosthesis when it is delivered, and the exact decrease in length and the relative positions of the ends of the prosthesis with respect to the punctured points in the connected vessels are difficult to predict with fluoroscopy alone. In contrast, US allowed for simultaneous direct visualization of both the prosthesis and the vessels during the act of deployment. The exact distance of liver parenchyma to be traversed between the targeted vessels was determined. The positions of the ends of the prosthesis in relation to the exit and entry points on the vessels being connected were readily apparent. This facilitated adequate positioning of the prosthesis, leaving the distal centimeter of each end within the lumen of the hepatic and portal veins, respectively.

**Results**

In the representative case, flow across the shunt from the portal vein to the hepatic vein and the inferior vena cava was observed shortly after the procedure (Fig 9). Injection of contrast material into the portal vein revealed rapid flow across the shunt and into the inferior vena cava (Fig 10). The entire procedure took 40 minutes. The portosystemic pressure gradient dropped from 32 to 15 mm Hg right after the procedure. Five days later, the portosystemic gradient further decreased to 12 mm Hg.

In the three other patients in whom color Doppler US guidance was employed during the procedure, a right

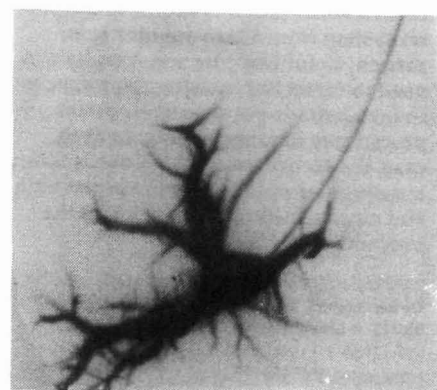
hepatic vein to right portal vein approach was used. The right portal vein was entered at the initial attempt in one case, while in the remaining two cases, a second attempt was necessary, after slight modification of the angle of the needle tip. In all four patients, successful puncture of the portal vein took less than 7 minutes, allowing completion of the entire procedure in 40–105 minutes. In no case did we need to administer more than 100 mL of iodinated contrast material, even with performance of hepatic venography and direct portography.

In all four cases, color Doppler US guidance allowed appropriate selection of the hepatic vein and portal branch and close real-time monitoring of the puncture. Continuous visualization of balloon inflation and prosthesis deployment were easily achieved.

**Discussion**

The TIPS has been a long-desired, attractive alternative to the surgical decompressive approach in the treatment of portal hypertension (10). The initial attempts, at first with some form of tubing (11–13) and later with balloon techniques (14–16), demonstrated feasibility, but long-term patency was not usually achieved. With the advent of either balloon-expandable (1,2,17) or self-expanding (4,18–20) metallic stents, success with TIPS procedures has become more achievable. As stated by Richter et al (1,2), however, considerable procedural problems with stent placement remained to be solved, despite this initial clinical success. The challenge posed by this evolving technique is evidenced by the many technical variations that have been attempted (2,4–8,21).

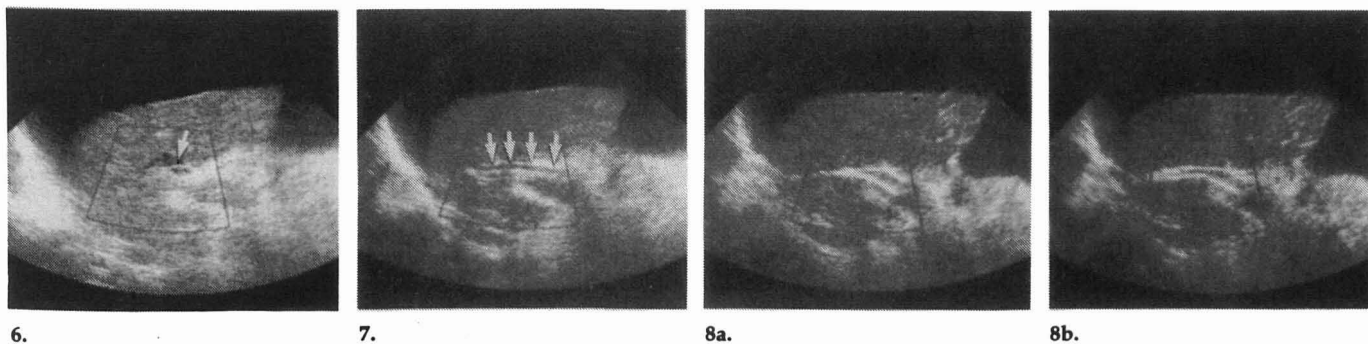
A most critical point to the TIPS procedure has been related to the need for transhepatic portal access to guide the transjugular needle (1). The percutaneous puncture of a small, hard, cirrhotic liver, commonly associated with coagu-



**Figure 5.** Opacification of the peripheral right portal branches at contrast material injection through the transjugular needle confirms successful puncture.

lation disorders and ascites, is at high risk for complications (1,3). In fact, one of the patients reported by Richter et al (2) died from intraperitoneal hemorrhage after embolization of the percutaneous access tract to the portal system failed. As Richter et al initially stated, a transhepatic portal access seemed to be necessary to allow exact planning of the course of the shunt tract (2), but, ideally, this could be obviated by using stereoscopic fluoroscopy, US, or detailed preprocedural sectional imaging (1).

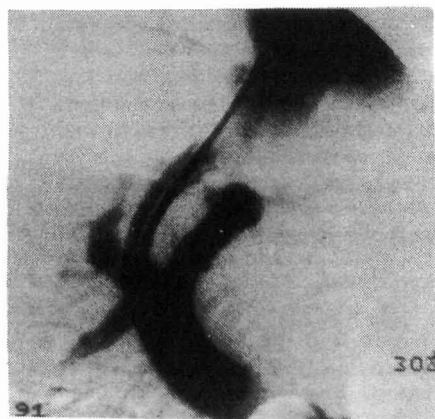
As more experience has been accumulated, transhepatic portal puncture has been demonstrated to be unnecessary (4–8). Previous arterial portography and transabdominal US are employed instead to provide some kind of guidance in identification of the position of the portal vein. In our experience, although previous portographic images are useful to avoid gross miscalculation in advancement of the needle in the coronal plane, exact tridimensional imaging with these is lacking, and the needle tip can be misdirected either ventrally or dorsally away from the targeted portal vein branch. Furthermore, as can be fre-



**Figures 6–8.** (6) A 0.035-inch guide wire, transjugularly placed in the portal vein, is seen as an echogenic line (arrow) across the portal vein lumen. (7) Longitudinal sonogram shows the properly positioned inflated balloon catheter (arrows) in the parenchymal tract. (8) Progressive opening (a, b) of the self-expanding prosthesis is clearly depicted.



**Figure 9.** Color Doppler sonogram shows completely expanded prosthesis with good flow from the portal vein to the hepatic vein.



**Figure 10.** Portal venogram shows portal flow through the shunt toward the inferior vena cava.

roscopic guidance alone, inadvertent puncture of hepatic artery branches or biliary ducts can occur (22), owing to their proximity to portal vein branches. Likewise, “blind” fluoroscopy-guided advancement of the transjugular needle in a small, retracted, and hard liver can result in accidental perforation of the inferior surface of the liver, the underlying gallbladder, or the adjacent colon (23,24). In the presence of ascites, calculation of the true boundaries of the liver with fluoroscopy alone can be even more difficult.

In our preliminary experience, advancement of the transjugular needle with US guidance overcomes most of these shortcomings. We have found color Doppler US to be particularly useful not only in selection of the hepatic vein and portal branch most suitable for shunting, but also in proper placement of the needle and direction of its advancement (25). Adequate balloon dilatation of the parenchymal tract and proper placement of the prosthesis were accurately verified with US. Immediately after the prosthesis was expanded, flow across the shunt was easily demonstrated.

Three other well-known occasional drawbacks with TIPS procedures are the need for a large amount of contrast medium, long duration of the procedure, and, hence, considerable exposure to radiation. Prolonged operation time and excessive exposure to radiation provide inconveniences mainly for the radiologists performing the procedure. On the other hand, the dose of iodinated contrast material is most critical for the patient, since excessive amounts may cause deterioration of renal function (26,27). In our experience, US guidance resulted in a low requirement for iodinated contrast material: Aside from 30–50 mL used in the initial hepatic venography, only 40–50 mL were employed in direct portography performed before and after the TIPS procedure. Color Doppler US monitoring allowed a quick and successful entry into the portal system and thus reasonably short

procedure duration and minimal radiation exposure. ■

#### References

1. Richter GM, Noeldge G, Palmaz JC, et al. Transjugular intrahepatic portacaval stent shunt: preliminary clinical results. *Radiology* 1990; 174:1027–1030.
2. Richter GM, Noeldge G, Palmaz JC, Roessler M. The transjugular intrahepatic portosystemic stent-shunt (TIPSS): results of a pilot study. *Cardiovasc Intervent Radiol* 1990; 13:200–207.
3. Viamonte M, Pereira R, Russell E, LePage J, Meier WL. Pitfalls in transhepatic portography. *Radiology* 1977; 124:325–329.
4. LaBerge JM, Ring EJ, Gordon RL. Intrahepatic portosystemic shunts with the Wallstent expandable metallic endoprosthesis: preliminary results (abstr). *JVIR* 1991; 2:13.
5. Richter GM, Noeldge G, Palmaz JC, Katzen BT, Becker GJ, Roessler M. Initial long-term results after transjugular intrahepatic portosystemic stent placement (abstr). *JVIR* 1991; 2:13.
6. Zemel G, Katzen BT, Becker GJ, Benenati JF. Percutaneous transjugular portosystemic shunt. *Radiology* 1991; 181(P):99–100.
7. Perarnau JM, Nöldge G, Rössle M. Transjugular intrahepatic portosystemic stent-shunt (TIPSS): early results with an improved technique (abstr). *J Hepatol* 1991; 13(suppl 2):S59.
8. Richter GM, Noeldge G, Roessler M, Roerent TH, Kauffmann GW, Palmaz JC. Three-year results of use of transjugular intrahepatic portosystemic stent shunt (abstr). *Radiology* 1991; 181(P):99.
9. Hamper UM, Savader BL, Sheth S. Improved needle-tip visualization by color Doppler sonography. *AJR* 1991; 156:401–402.
10. Zimmon DS. Percutaneous portal-systemic shunts: promise and problems. *Hepatology* 1988; 8:698–699.
11. Rösch J, Hanafee WN, Snow H. Transjugular portal venography and radiologic portacaval shunt: an experimental study. *Radiology* 1969; 92:1112–1114.
12. Rösch J, Hanafee WN, Snow H, Barenfus M, Gray R. Transjugular intrahepatic portacaval shunt: an experimental work. *Am J Surg* 1971; 121:588–592.
13. Gutierrez OH, Burgener FA. Production of nonsurgical portosystemic venous shunts in dogs by transjugular approach. *Radiology* 1979; 130:507–509.
14. Colapinto RF, Stronell RD, Gildiner M, et al. Formation of intrahepatic portosystemic shunts using a balloon dilatation

quently observed at US, pushing of the needle in a hard cirrhotic parenchyma usually produces some extent of downward displacement of the entire liver, rendering inaccurate the landmarks provided by previously obtained portograms. These shortcomings may partly explain the multiple puncture trials (22) occasionally required to gain entrance to the portal vein. Moreover, with fluo-

- catheter: preliminary clinical experience. *AJR* 1983; 140:709-714.
15. Abecassis M, Gordon JD, Colapinto RF, et al. The transjugular intrahepatic portosystemic shunt (TIPS): an alternative for the management of life-threatening hemorrhage (abstr). *Hepatology* 1985; 5:1032A.
  16. Burgener FA, Gutierrez OH. Experimental intrahepatic portocaval shunts created in portal hypertension by balloon angioplasty catheters. *Invest Radiol* 1988; 23:24-29.
  17. Palmaz JC, Sibbitt RR, Reuter SR, Garcia R, Tio FO. Expandable intrahepatic portocaval shunt stents: early experience in the dog. *AJR* 1985; 145:821-825.
  18. Rösch J, Uchida BT, Putnam JS, Buschman RW, Law RD, Hershey AL. Experimental intrahepatic portocaval anastomosis: use of expandable Gianturco stents. *Radiology* 1987; 162:481-485.
  19. Rousseau H, Puel J, Joffre F, et al. Experimental portohepatic shunt with the Wall-stent endoprosthesis (abstr). *JVIR* 1991; 2:12.
  20. Roberts JP, Ring E, Lake JR, Sterneck M, Ascher NL. Intrahepatic portocaval shunt for variceal hemorrhage prior to liver transplantation. *Transplantation* 1991; 52:160-162.
  21. Uchida BT, Putnam JS, Rösch J. "Atraumatic" transjugular needle for portal vein puncture in swine. *Radiology* 1987; 163:580-581.
  22. Bancroft III JW, Becker GJ, Katzen BT, Zemel G, Benenati JF, Sallee DS. Percutaneous transjugular portosystemic shunt (abstr). *Radiology* 1991; 181(P):338.
  23. Kattan KR. The effect of changes in liver volume on the adjacent viscera. *Radiol Clin North Am* 1980; 18:195-207.
  24. Gore RM, Ghahremany GG, Joseph AE, Nemcek AA Jr, Marn CS, Vogelzang RL. Acquired malposition of the colon and gallbladder in patients with cirrhosis: CT findings and clinical implications. *Radiology* 1989; 171:739-742.
  25. McNamara MP Jr. Percutaneous procedures guided by color-flow Doppler sonography. *AJR* 1989; 152:1123-1125.
  26. Abrams HL. The opaque media: physiologic effects and systemic reactions. In: Abrams HL, ed. *Abrams angiography: vascular and interventional radiology*. 3rd ed. Boston: Little, Brown, 1983; 15-39.
  27. Marx M, Bettmann MA. Contrast-induced renal failure. *Postgrad Radiol* 1985; 5:343-349.

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## Transjugular Intrahepatic Portosystemic Shunt: Evaluation with Doppler Sonography<sup>1</sup>

To assess the potential role of ultrasound (US) in evaluating transjugularly inserted intrahepatic portosystemic shunts, the authors performed sonographic, including duplex and color Doppler, studies in 23 patients. Imaging was performed before, during the first week after, and 2-3 months after shunt placement. The prostheses were readily visualized and shunt patency was easily determined with Doppler US in all patients. The mean of the maximum blood flow velocity in the main portal vein increased from 7 (range, 3-16) cm/sec before shunting to 24 (range, 18-47) cm/sec 2-3 months after shunting. Flow reversal was detected in the intrahepatic portal branches in 16 (69%) of the 23 patients. US allowed prompt detection and management of two cases of incomplete stent expansion and one case of shunt stenosis due to pseudo-intimal hyperplasia. Sonography is a valuable tool for evaluation of changes in hepatic hemodynamics after transjugular intrahepatic shunt placement.

**Index terms:** Hypertension, portal, 94.711 • Liver, interventional procedure, 761.12986 • Portal vein, 957.711 • Shunts, portosystemic, 957.453, 982.453 • Ultrasound (US), Doppler studies

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**N**ONSURGICAL creation of an intrahepatic portosystemic shunt has been a long-sought, attractive alternative to both decompressive shunt surgery and endoscopic sclerotherapy in the treatment of variceal bleeding due to portal hypertension. Since the initial work of Rösch et al (1) in 1969, several groups have attempted to create an intrahepatic portosystemic shunt through the transjugular route. Initial attempts with either some form of tubing (1,2) or balloon techniques (3,4) almost always resulted in early shunt closure. Only with the advent of vascular stents, either balloon-expandable or self-expanding, has the transjugular intrahepatic portosystemic shunt (TIPS) technique achieved clinical success (5-8).

Until recently, angiography has been the standard technique for evaluation of transjugularly placed portosystemic stents. Angiography is invasive, however, and has its consequent limitations as a routine follow-up technique. Clearly, less invasive follow-up techniques are highly desirable. Doppler ultrasound (US), either duplex or color-coded, has proved extremely useful in the evaluation of the hepatic vasculature and various surgical portosystemic shunts (9-14). We undertook this study to assess the role of Doppler US in the evaluation of cirrhotic patients with percutaneously inserted intrahepatic portosystemic shunts.

### MATERIALS AND METHODS

From February 1991 to February 1992, 65 cirrhotic patients underwent treatment with the TIPS procedure at our institutions in a cooperative project. Among the 29 TIPS procedures performed at the University Clinic of Navarre in that period, sonographic follow-up as described below was carried out in 23 cases. These cirrhotic patients (alcoholic [ $n = 10$ ], postnecrotic [ $n = 13$ ]) with transjugularly placed intra-

hepatic portosystemic stents were included in this study. The indications for the TIPS were variceal bleeding that had become nonresponsive to sclerotherapy (16 cases) and refractory ascites (seven cases). There were 17 male and 6 female patients, with a mean age of 57 (range, 43-72) years. Four of the patients were classified as having Child A disease; 11 patients, as having Child B disease; and the remaining eight patients, as having Child C disease by means of the Child-Pugh modified scoring system (15).

In all of these patients, self-expanding Wallstent metallic endoprostheses (Medinvent, Lausanne, Switzerland) 43 or 62 mm long, 8-10 mm in diameter, were inserted by using a percutaneous technique. Transjugular access was the only route employed, without any percutaneous transhepatic puncture. The intrahepatic portal system was punctured and catheterized by means of combined fluoroscopic and sonographic control in all patients, color Doppler US guidance (16) being utilized in the last 19 cases. After balloon dilation of the resulting intraparenchymal tract, the metallic self-expanding stents were deployed, allowing the high-pressure portal blood to divert toward the low-pressure hepatic vein. The TIPS insertion techniques have been described in detail elsewhere (5-7,16-18) and will not be discussed here. In 19 cases, right hepatic vein-right portal vein shunting was performed. The right portal vein was connected to the middle hepatic vein in the remaining four patients.

In each of our patients, baseline US evaluation was performed before TIPS insertion. Early sonographic controls were performed during the first week after the procedure. Late sonographic controls were carried out 2-3 months after TIPS insertion.

Patients underwent scanning with an Acuson 128 XP/10 computed sonography system (Acuson, Mountain View, Calif). A 28-mm-aperture phased-array vector transducer, operating at 3.5 MHz, was employed. The Doppler frequency for color imaging was also 3.5 MHz. Patients were

**Abbreviation:** TIPS = transjugular intrahepatic portosystemic shunt.



examined in the supine and left posterior oblique positions after at least a 7-hour fast, and both subcostal and intercostal approaches were used to fully assess the liver and neighboring structures.

All the sonographic studies were performed or directly supervised by one of the authors (J.M.L.) with experience in US and interventional procedures. In the baseline US evaluation, previous to TIPS insertion, the patency and flow characteristics in the main portal vein and its branches were evaluated. Visualization of portosystemic collateral vessels, the size of the spleen, and the presence of ascites were also registered.

In the follow-up sonographic studies, complications of the insertion procedure (eg, hematomas, intrahepatic bile collections, infarcts) were sought. Variations in the visualization of portosystemic collateral vessels, spleen size, and ascites were recorded. Sonographic studies were then focused on the intrahepatic shunt. The caliber, configuration, and relative position of the stent with respect to both the portal branch and the draining hepatic vein were carefully assessed. The presence, direction, and characteristics of flow within the shunt and adjacent vessels were evaluated by using both duplex and color Doppler US.

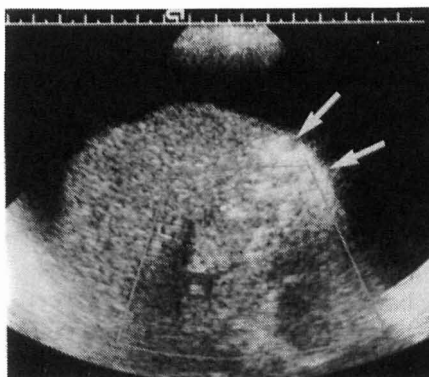
A low pulse repetition frequency was employed to obtain adequate color Doppler sensitivity. Since low frequencies can result in aliasing, which occasionally mimics flow reversal, an adequate sampling rate was employed to avoid misinterpretations (19,20). Furthermore, flow direction assessed with color Doppler US was confirmed by the results of spectral Doppler studies in questionable cases. The angle of insonation was kept to less than 60°, whenever possible, to minimize sampling error in measurement of Doppler shifts. In some instances, obtaining such a satisfactory Doppler angle was difficult. No attempt was made to grade flow volume (21-23).

As part of the follow-up protocol, direct transshunt portography was also performed in all 23 patients by means of transvenous access (usually right femoral

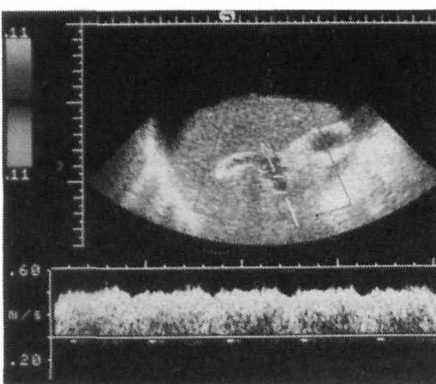
vein) across the shunt during the first week after TIPS insertion and 2-3 months later. Opacification of the shunted portal system, measurement of pressure gradients across the shunt, and variceal evaluation were achieved by means of this technique.

## RESULTS

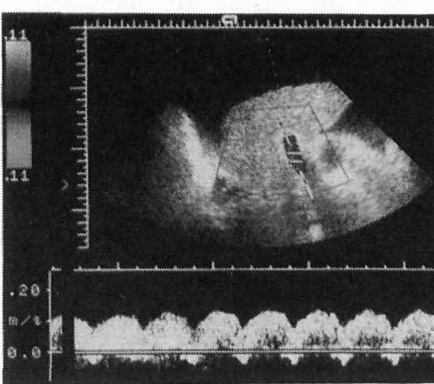
Early sonographic examinations after TIPS insertion showed no parenchymal hematomas, bile collections, segmental liver infarcts, or other intrahepatic findings related to the insertion procedure. Only small and transient echogenic areas in the peripheral liver parenchyma (Fig 1) were observed in seven patients examined during the first hours after TIPS insertion. This finding was possibly related to inadvertent introduction of small microbubbles during the various manipulations (mainly catheter exchanges) required throughout the insertion procedure.



**Figure 1.** Oblique sonogram obtained 2 hours after TIPS placement in a 45-year-old man. Small echogenic areas (arrows), probably due to small microbubbles, are commonly seen during the first few hours after TIPS insertion.



**Figure 3.** (a) On a Doppler study obtained in a 56-year-old man before shunt placement, slow (8-12 cm/sec) flow is seen in the main portal vein. (b) After shunt placement, a fourfold increase in portal flow velocity (up to 40-45 cm/sec) is seen.

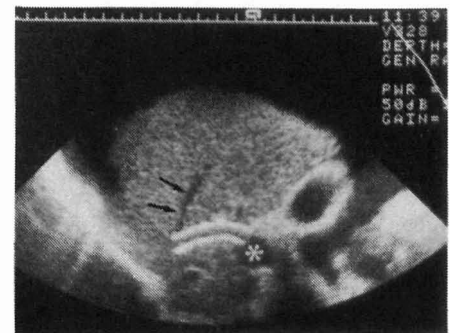


**Figure 4.** Color Doppler sonogram obtained in a 53-year-old man shows brisk flow along the entire length of the stent up to the inferior vena cava (\*). Aliasing (coded red-yellow) (arrow) is prominent in the hepatic vein end of the stent. Antegrade, hepatopetal flow is preserved through the right branch of the portal vein (arrowheads) peripheral to the stent entry point.

The prostheses were adequately visualized in all cases. The morphologic characteristics of the stent were clearly depicted, and the known longitudinal flexibility of the Wallstent prostheses (24) was readily apparent. This characteristic allowed optimal adaptability, the metallic prosthesis conforming to the diverse spatial ori-

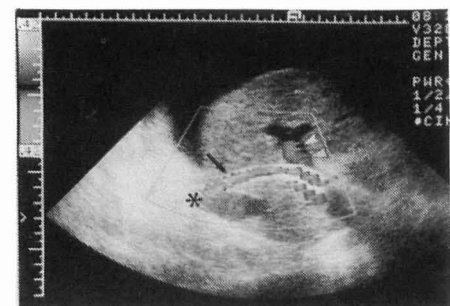


a.



b.

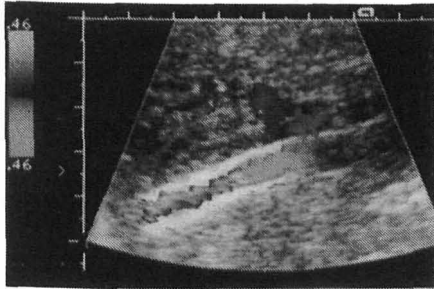
**Figure 2.** TIPS in two different patients. (a) Sonogram obtained in a 52-year-old woman shows the walls of the bent stent as a row of echogenic dots that correspond to the metallic wires. The slightly everted end is seen entering the portal vein. (b) Sonogram obtained in a 45-year-old man shows the prosthesis in its complete length. The smooth curvature of the stent contributes to the optimal hemodynamic orientation of the shunt with respect to the decompressed portal vein (\*) and the draining hepatic vein (arrows).



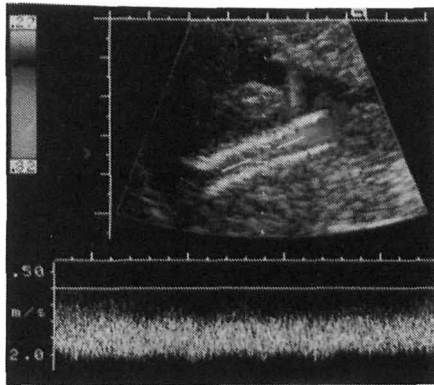
**Figure 4.** Color Doppler sonogram obtained in a 53-year-old man shows brisk flow along the entire length of the stent up to the inferior vena cava (\*). Aliasing (coded red-yellow) (arrow) is prominent in the hepatic vein end of the stent. Antegrade, hepatopetal flow is preserved through the right branch of the portal vein (arrowheads) peripheral to the stent entry point.

entations of the connected vessels (Fig 2). No shadowing effect was caused by the metallic stent (25).

The shunting effect created by the TIPS was reflected in a clear increase



a.



b.

**Figure 5.** Color Doppler sonogram obtained in a 67-year-old man shows fast flow inside a shunt. (a) Aliasing (coded red-yellow) is observed near the hepatic vein end of the shunt. (b) Duplex Doppler measurement of flow velocity inside the shunt. Continuous, high-velocity (up to 200 cm/sec) flow is seen. Spectral broadening is evident. The baseline has been moved to avoid color aliasing.

in portal flow velocity in all cases. The maximum velocities before and after shunting were obtained in each patient (Fig 3). The mean maximum velocity in the main portal vein changed from 7 (range, 3–16) cm/sec before the procedure to 23 (range, 15–45) cm/sec in the early sonographic controls after TIPS insertion and remained with slight oscillations at sonographic controls 2–3 months later (mean, 24 cm/sec; range, 18–47 cm/sec).

In spite of the deep location of the shunts inside the abdomen and the fact that most of them were oriented almost perpendicular to the sound beam, shunt patency was readily demonstrated with color Doppler sonography. Aliasing was frequently observed in color Doppler examinations, reflecting the high flow velocities inside the stent (Fig 4). It was usually more difficult to perform accurate spectral Doppler velocity measurements of the flow across the shunt, mainly because of the difficulty in placing the sample volume at a suitable angle. High-velocity flow across the shunt, ranging from 65 to 220 (mean, 123) cm/sec, was observed in all cases (Fig 5).

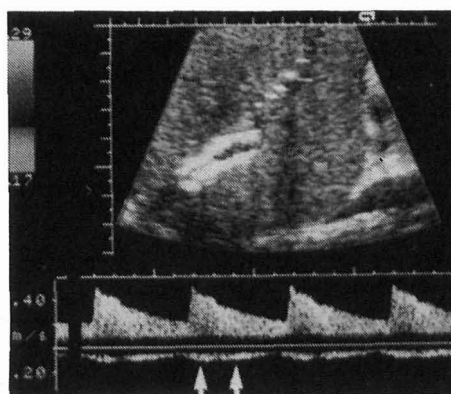
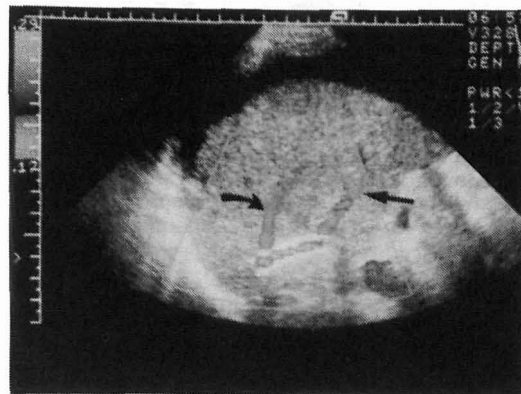
Patency and direction of flow in the intrahepatic portal branches were readily apparent in all cases. All patients showed antegrade, hepatopetal flow toward the liver periphery in these intrahepatic branches before shunting. Early after TIPS insertion, hepatopetal flow was maintained in the intrahepatic portal vein branches in only seven patients (Fig 4). In the remaining 16 patients, a reversal of the flow direction, with the blood

flowing toward the shunt, was observed in the intrahepatic portal vein branches (Fig 6). In the patients in whom antegrade, hepatopetal flow persisted in the portal vein branches after TIPS placement, a small area of reversed color, indicating a local change of flow direction, was frequently observed in the right portal vein branch immediately adjacent to the prosthesis entry point. Careful analysis showed that this finding was due to turbulence in blood flow as it flows around and through the inserted prosthesis (Fig 7). All intrahepatic portal vein branches remained patent, with no occluding clot or partial thrombosis.

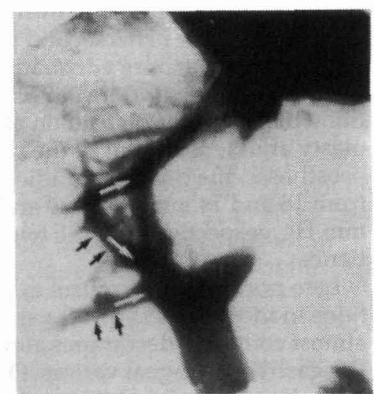
Patency of the stent in the hepatic vein was maintained in all cases (Fig 8). While normal, biphasic flow was present in 18 cases, a continuous, turbulent hepatofugal flow, a frequent finding in the hepatic vein in cirrhotic patients (26), was observed in five cases. The tributaries draining into the hepatic vein with a stent maintained their patency and flow toward the inferior vena cava in all cases.

In a sonographic control examination performed in one patient 2 months after TIPS insertion, a discrete, eccentric narrowing of the lumen, probably due to pseudointimal hyperplasia (27), was observed. The stenosis was dilated with an 8-mm angioplasty balloon. Postdilation images showed complete relief of the stenosis (Fig 9).

The diameter was measured along the entire length of the stent, allowing detection of persistence of incompletely expanded segments in two

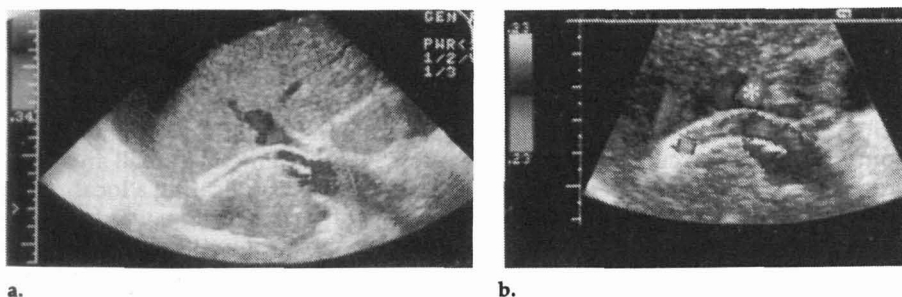


b.

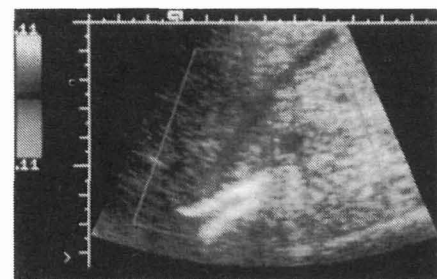


c.

**Figure 6.** (a) Doppler sonogram shows oblique, longitudinal view of the shunt along its main axis in a 45-year-old man. Reversed, hepatofugal flow (coded blue) is seen in a branch of the right portal vein (straight arrow), draining toward the prostheses. The hepatic vein with a stent (curved arrow) is patent and shows normal flow toward the inferior vena cava. (b) Duplex Doppler study confirms flow reversal in the same right portal vein branch. Reversed portal flow (arrows) is seen below the baseline in the opposite direction to that in the accompanying branch of the hepatic artery. (c) Direct transshunt portogram obtained in the same patient. High-pressure injection of contrast material in the main portal vein results in artifactual, transient antegrade filling after the peak of the injection ("mixing artifact") in both the right portal vein branches (black arrows) and hepatic vein. Although later digital subtraction images (not shown) disclose the true flow direction in each vessel (white arrows), care must be taken not to misinterpret this technique-related artifact as true hepatopetal flow in the portal vein branches.



**Figure 7.** (a) Color Doppler sonogram shows antegrade flow (coded red) in the right portal vein branch in a 53-year-old man. A small area of reversed flow (coded blue) is seen where the right portal vein branch is entered by the prosthesis. (Filtering has been set to detect low, venous velocities. Hence, brisk flow inside the stent is not shown in this image). (b) The same finding (\*) is prominent in another patient, a 67-year-old man.

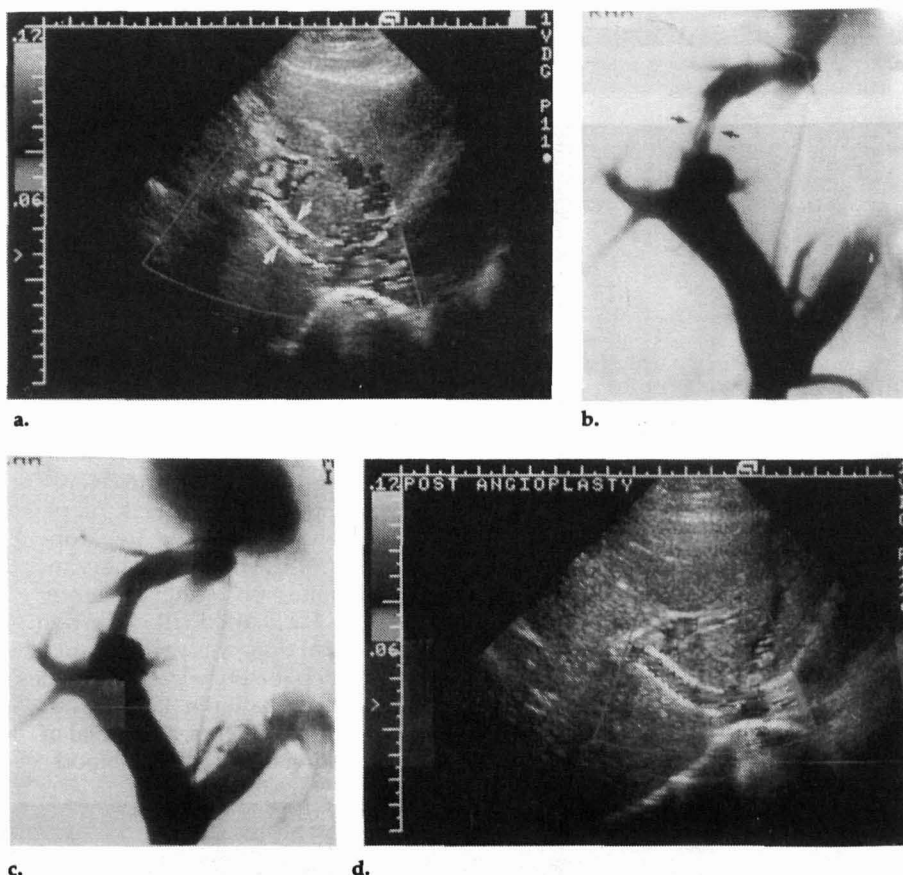


**Figure 8.** Sonogram obtained in a 51-year-old man shows normally directed flow toward the inferior vena cava in the peripheral portion of the hepatic vein with a stent.

patients 3 and 4 days after the procedure. In both of them, initial balloon dilation of the intrahepatic parenchymal tract was performed before stent placement but was not repeated after deployment of the prosthesis. Pressure gradients remained moderately elevated (19 and 14 mm Hg, respectively) at the end of the procedure, and portograms showed persistent incomplete opening of the stents at that moment. Further dilation was not considered necessary in the hope that progressive opening of the self-expanding prosthesis would take place spontaneously, as it frequently occurs in the biliary tract (28).

At subsequent sonographic controls 3 and 4 days after the initial procedures, no further increase in the caliber of the self-expanding prosthesis was observed. Both transshunt portograms and pressure-gradient measurements were similar to previous ones. This prompted additional selective balloon dilation of the shunts. In both cases, after balloon dilation of the prosthesis, a second stent was coaxially deployed, partially overlapping the first one. This second prosthesis allowed a more adequate stent placement in the portal vein (Fig 10). Immediately after the balloon angioplasty and deployment of the second prostheses, the pressure gradients fell from 18 and 14 mm Hg to 10 and 9 mm Hg, respectively, in the two patients.

Late portographic control examinations in most cases showed a striking, almost complete decompression of the gastroesophageal varices. On the other hand, as might be expected, US was not as reliable a method for the evaluation of gastroesophageal collateral vessels, neither before nor after TIPS insertion. Ascites, gastrointestinal gas, and deep location of gastroesophageal collateral vessels accounted for the poor visualization



**Figure 9.** Shunt stenosis. (a) At 2 months after TIPS insertion in a 44-year-old woman, sonogram shows a short stenosis, resulting in a local loss of the color column, in the middle portion of the stent (arrows). (b) Direct (transshunt) portogram confirms the uneven reduction of caliber, probably due to pseudo-intimal proliferation (arrows). (c, d) After balloon angioplasty, restoration of the stent lumen is observed on (c) an angiogram and (d) a color Doppler sonogram.

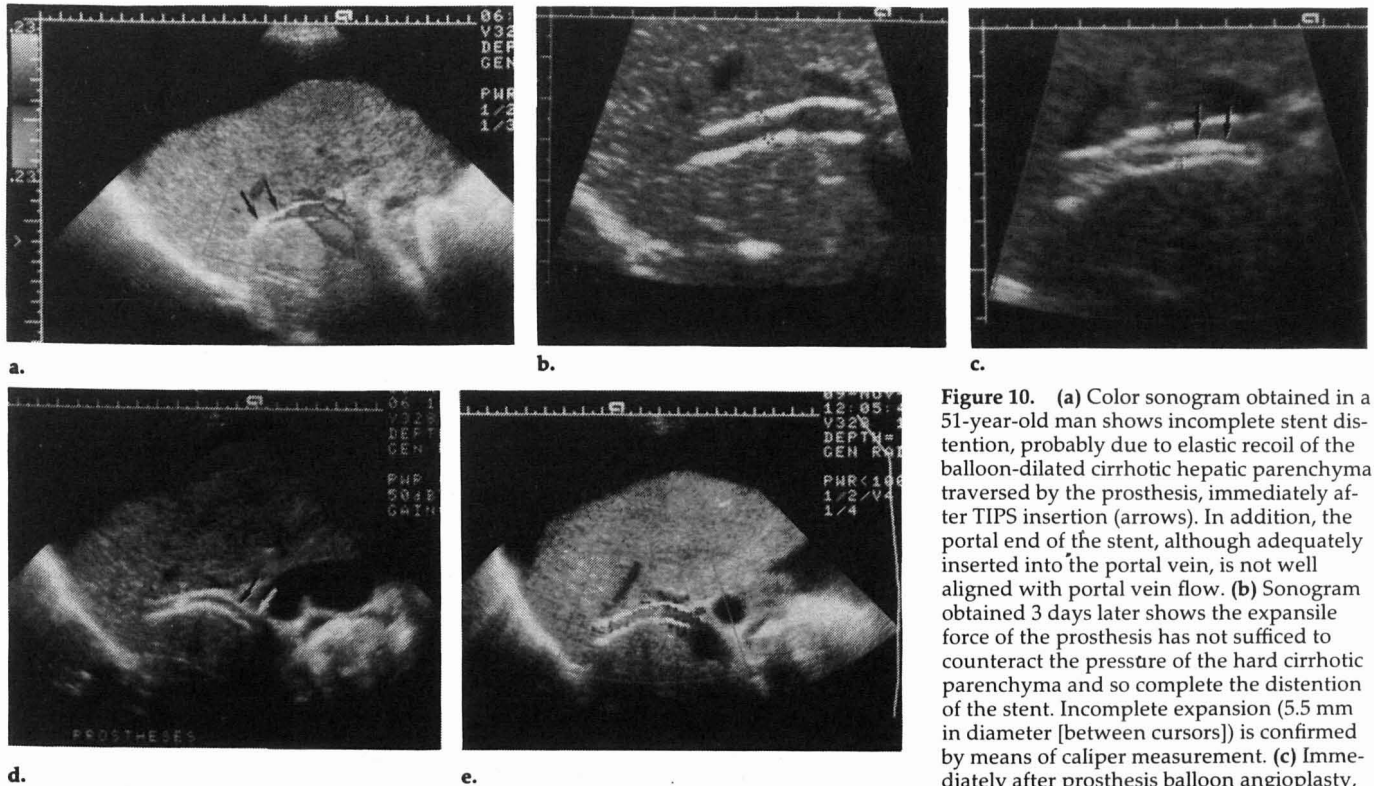
and ensuing difficulty in flow detection in most instances (29).

Apart from the seven patients in which the main indication for TIPS was ascites refractory to diuretic therapy, mild to moderate ascites was detected with US in three other patients before shunt placement. After the procedure, tense ascites was reduced to a minimal amount in five patients and greatly improved in two. The

three cases of mild to moderate ascites resolved completely after the TIPS procedure.

Probably because of the relatively short follow-up study period (30,31), only a moderate decrease in splenic size was detected in most cases after shunting. In the first week after shunting, mean splenic size dropped from a baseline of 15.9 (range, 9.2–24.4) cm to 15.4 (range, 9.6–21.9) cm.





**Figure 10.** (a) Color sonogram obtained in a 51-year-old man shows incomplete stent distention, probably due to elastic recoil of the balloon-dilated cirrhotic hepatic parenchyma traversed by the prosthesis, immediately after TIPS insertion (arrows). In addition, the portal end of the stent, although adequately inserted into the portal vein, is not well aligned with portal vein flow. (b) Sonogram obtained 3 days later shows the expansile force of the prosthesis has not sufficed to counteract the pressure of the hard cirrhotic parenchyma and so complete the distention of the stent. Incomplete expansion (5.5 mm in diameter [between cursors]) is confirmed by means of caliper measurement. (c) Immediately after prosthesis balloon angioplasty, the stent has approached 8 mm in diameter (between cursors). An angiographic catheter (arrows) is seen as a third bright line between the stent walls. (d) Deployment of a second prosthesis, partially overlapping the first and extending into the main portal vein (arrows), was carried out under combined fluoroscopic (not shown) and sonographic monitoring. (e) Color sonogram shows adequate stent caliber and correct alignment with the long axis of the portal vein, resulting in satisfactory flow across the shunt.

Two to 3 months later, however, a significant decrease to 15.1 (range, 9.5–22.1) cm was observed ( $P < .05$ ).

## DISCUSSION

Goldberg and Patel (32) first proposed in 1977 the use of sonography as a noninvasive method of evaluating portosystemic shunts. More recently, duplex Doppler sonography and color Doppler imaging were found to be appropriate evaluation tests to assess the patency of conventional, surgical portosystemic shunts (11–14). We found that US is similarly useful in evaluation of the hepatic hemodynamic situation after percutaneous, nonsurgical portosystemic shunting.

For decades, angiography has been the traditional method of evaluating surgical portosystemic shunts. At present it remains the standard evaluation technique after TIPS insertion. Fortunately, the portal system is readily accessible through the transjugularly placed intrahepatic stent. Selective shunt catheterization allows both opacification of the portal system and direct measurement of the pressure gradient across the shunt. Direct portograms obtained in this way in these patients allow visualization of variceal collaterals. Patency and flow across the shunt are also clearly depicted.

On the other hand, direct portograms offer little information about local, subtle hemodynamic changes occurring in

the intrahepatic portal branches after TIPS placement. As we frequently observed, a low-pressure portal injection of contrast material can result in artifactual nonopacification of some portal branches because of preferential flow through the stent, while a high-pressure injection can overflow into the entire intrahepatic portal system, then momentarily masking any reversed (hepatofugal) flow in these vessels. This report demonstrates that these limitations are easily overcome with Doppler imaging. Due to its noninvasive nature, sonography does not interfere with the complex hemodynamic patterns in the cirrhotic liver with a stent, and misinterpretations of flow direction are thus avoided. As a result, accurate information about the true direction of flow in each individual vessel is provided.

Doppler sonography allowed us to detect true reversal of portal flow in the intrahepatic portal branches in 16 (69%) of 23 patients. This phenomenon, which could be labeled stealing of flow from the neighboring portal branches into the shunt, is obviously difficult to detect with direct transshunt portography. In fact, we cannot find any previous explicit mention of this relevant finding in the literature about TIPS. This reversal of intrahepatic portal flow implies that the TIPS, while adequately lowering the portosystemic pressure gradient, might diminish hepatic portal perfusion. In two of the four patients who showed a slight impairment in hepatic function,

as well as in the two patients who developed mild, transient, new-onset encephalopathy episodes during follow-up, flow reversal was seen in the intrahepatic portal vein branches. Nevertheless, the clinical significance of these local hemodynamic changes is poorly understood at present (33,34) and remains to be evaluated with long-term follow-up.

The crisscross design of the Wallstent allows blood to flow freely through the small holes in its wall. In all of our cases, Doppler imaging, both duplex and color, allowed confirmation of this finding, no thrombosis being observed in the portal or hepatic branches crossed by the prostheses.

As might be expected, flow velocity in the main portal vein increased markedly immediately after shunt placement and remained stable thereafter. While direct portograms provide only a rough, subjective estimation of flow velocity in the portal vein, Doppler sonography allowed measurement of a mean increase of 17 cm/sec in maximum veloc-

ity in that vessel. Similarly, color Doppler US easily depicted high-velocity flow through the shunt in all patients. Shunt patency could be clearly demonstrated in every case. Nevertheless, accurate quantitative flow-velocity measurements inside the stent proved to be more difficult (20-23), because of the almost perpendicular orientation of the stent flow with respect to the transducer in many cases.

The liver, by providing an excellent acoustic window, always allows the metallic prosthesis to be easily visualized. Precise diameter measurements can be performed, and accurate evaluation of the exact position of the intrahepatic stent is consistently provided. We found US particularly useful in detecting cases in which further manipulations were necessary. US allows precise measurement of the stent diameter, greatly facilitating the detection of zones of incomplete stent expansion. Most interesting is that US allows simultaneous visualization of both the shunted hepatic vessels and the prosthesis connecting them. As a result, the relative position and angulation of the end of the prosthesis with respect to the walls of the vessels with stents can readily be displayed. Hence, inappropriate positioning of the ends of the stent can be detected easily. Fortunately, the flexibility of the Wallstent prostheses allowed us to correct the situation by inserting a second prosthesis partially overlapping the first one and protruding farther into the portal vein.

US showed certain limitations in the evaluation of these patients. In contrast with its ability to depict the flow in both the stent and the hepatic vessels, US was not as useful in evaluating the gastroesophageal collateral vessels. Ascites, bowel gas, the body habitus of the patient, and the deep location of these vessels (29) all contributed to poor visualization in most of our patients. As a consequence, the evolutionary changes in the variceal collateral vessels after TIPS placement must be monitored by more invasive means. Although it would be highly desirable to have a noninvasive method of correctly evaluating gastroesophageal collateral vessels and pressure gradients, we are forced to rely on transshunt portography and endoscopy at the moment. Nevertheless, in view of the findings earlier described at Doppler US in the intrahepatic portal branches, we must be cautious in the interpretation of persistent visualization of the gastroesophageal collateral vessels by means of direct portography. Conceivably, artifactual filling and opacification of "empty" variceal collateral vessels, due to a sudden high-pressure injection of contrast ma-

terial in the portal bed, is possible during portography. The theoretical risk of "overdiagnosis" and/or "overtreatment" of variceal collateral vessels must be taken into account.

Finally, accidental puncture of both the intrahepatic arterial branches and biliary ducts (17,18), which could lead to hematoma formation or bile leakage, is not uncommon during misdirected needle passages. Although in the follow-up of the cases reported herein we did not observe either intrahepatic hematomas or bile leaks, we believe that conventional sonography would be the technique of choice in detecting these potential subsequent complications of accidental injury of arterial vessels or biliary ducts.

As stated by Ralls (10), portal blood flow pathophysiologic characteristics are complex and incompletely defined even by means of invasive standard-of-reference techniques such as angiography and percutaneous venography. In this context, Doppler sonography can add valuable information to that provided with angiographic studies. Owing to its noninvasiveness, US allows acquisition of useful hemodynamic data without extrinsic interference by contrast material injections. On the other hand, shunt catheterization remains invaluable in obtaining measurements of portosystemic pressure gradients, visualizing collateral vessels, and performing additional interventional procedures. Once again, the complementary roles of Doppler sonography and angiographic techniques in evaluating the vascular system are appreciated. ■

#### References

- Rösch J, Hanafee WN, Snow H. Transjugular portal venography and radiologic portacaval shunt: an experimental study. *Radiology* 1969; 92:1112-1114.
- Gutierrez OH, Burgener FA. Production of nonsurgical portosystemic venous shunts in dogs by transjugular approach. *Radiology* 1979; 130:507-509.
- Colapinto RF, Stronell RD, Gildiner M, et al. Formation of intrahepatic portosystemic shunts using a balloon dilatation catheter: preliminary clinical experience. *AJR* 1983; 140:709-714.
- Burgener FA, Gutierrez OH. Experimental intrahepatic portacaval shunts created in portal hypertension by balloon angioplasty catheters. *Invest Radiol* 1988; 23:24-29.
- Richter GM, Noeldge G, Palmaz JC, Roessle M. The transjugular intrahepatic portosystemic stent-shunt (TIPSS): results of a pilot study. *Cardiovasc Intervent Radiol* 1990; 13:200-207.
- Richter GM, Noeldge G, Roessle M, Palmaz JC. Evolution and clinical introduction of TIPSS, the transjugular intrahepatic portosystemic stent-shunt. *Semin Intervent Radiol* 1991; 4:331-340.
- Zemel G, Katzen BT, Becker GJ, Benenati JF, Sallee DS. Percutaneous transjugular portosystemic shunt. *JAMA* 1991; 266:390-393.
- Ring EJ, Lake JR, Roberts JP, et al. Using transjugular intrahepatic portosystemic shunts to control variceal bleeding before liver transplantation. *Ann Intern Med* 1992; 116:304-309.
- Becker CD, Cooperberg PL. Sonography of the hepatic vascular system. *AJR* 1988; 150:999-1005.
- Ralls PW. Color Doppler sonography of the hepatic artery and portal venous system. *AJR* 1990; 155:517-525.
- Rice S, Lee KP, Johnson MB, Korula J, Ralls PW. Portal venous system after portosystemic shunts or endoscopic sclerotherapy: evaluation with Doppler sonography. *AJR* 1991; 156:85-89.
- Grant EG, Tessier FN, Gomes AS, et al. Color Doppler imaging of portosystemic shunts. *AJR* 1990; 154:393-397.
- Ralls PW, Lee KP, Mayekawa DS, et al. Color Doppler sonography of portocaval shunts. *JCU* 1990; 18:379-381.
- Hederström E, Forsberg L, Ivancev K, Lundstedt C, Stridbeck H. Ultrasonography and Doppler duplex compared with angiography in follow-up of mesocaval shunt patency. *Acta Radiol* 1990; 31:341-345.
- Pugh RN, Murray-Lyon IM, Dawson JL, Pietroni MC, Williams R. Transsection of the oesophagus for bleeding oesophageal varices. *Br J Surg* 1973; 56:646-649.
- Longo JM, Bilbao JL, Rousseau HP, et al. Color Doppler US guidance in transjugular placement of intrahepatic portosystemic shunts. *Radiology* 1992; 184:281-284.
- Ring EJ. Interventions in portal hypertension. In: Mueller PR, vanSonnenberg E, Becker GJ, eds. *Syllabus: a diagnostic categorical course in interventional radiology*. Oak Brook, Ill: Radiological Society of North America, 1991; 219-227.
- Zemel G, Becker GJ, Bancroft JW, Benenati JF, Katzen BT. Technical advances in transjugular intrahepatic portosystemic shunts. *Radiographics* 1992; 12:615-622.
- Foley WD, Erickson SJ. Color Doppler flow imaging. *AJR* 1991; 156:3-13.
- Mitchell DG. Color Doppler imaging: principles, limitations and artifacts. *Radiology* 1990; 177:1-10.
- Ranke C, Hendrickx P, Roth U, Brassel F, Creutzig A, Alexander K. Color and conventional image-directed Doppler ultrasonography: accuracy and sources of error in quantitative blood flow measurements. *JCU* 1992; 20:187-193.
- Burns PN, Jaffe CC. Quantitative flow measurements with Doppler ultrasound: techniques, accuracy, and limitations. *Radiol Clin North Am* 1985; 23:641-657.
- Sabbà C, Weikim GG, Cicchetti DV, et al. Observer variability in echo-Doppler measurements of portal flow in cirrhotic patients and normal volunteers. *Gastroenterology* 1990; 98:1603-1611.
- Zollhofer CL, Largiadier I, Bruhlmann WF, Uhlischmid GK, Marty AH. Endovascular stenting of veins and grafts: preliminary clinical experience. *Radiology* 1988; 167:707-712.
- Vorwerk D, Guenther KW, Gehl HB, Bohndorf K. Color-coded image-directed Doppler sonography compared with digital subtraction angiography in the follow-up of percutaneous vascular endoprostheses. *JCU* 1990; 18:631-637.
- Longo JM, Eleno A, Bilbao JL, Cabello JR, Aguerreta JD, Fernandez A. Duplex Doppler of the hepatic veins in patients with cirrhosis: pseudoportal pattern (abstr). *Radiology* 1989; 173(P):361.
- LaBerge JM, Ferrell LD, Ring EJ, et al. Histopathologic study of transjugular intrahepatic portosystemic shunts. *JVIR* 1991; 2:549-556.
- Laméris JS, Stoker J, Nijs HGT, et al. Malignant biliary obstruction: percutaneous use of self-expandable stents. *Radiology* 1991; 179:703-707.
- Parvey HR, Eisenberg RL, Givanani V, Krebs CA. Duplex sonography of the portal venous system: pitfalls and limitations. *AJR* 1989; 152:765-770.
- Soper NJ, Rikkers LF. Effect of operations for variceal hemorrhage on hypersplenism. *Am J Surg* 1982; 144:700-703.
- Mutchnick MG, Lerner E, Conn HO. Effect of portacaval anastomosis on hypersplenism. *Dig Dis Sci* 1980; 25:929-938.
- Goldberg BB, Patel J. Ultrasonic evaluation of portacaval shunts. *JCU* 1977; 5:304-306.
- Warren WD. Control of variceal bleeding: reassessment of rationale. *Am J Surg* 1983; 145:8-16.
- Rikkers LF. Portal hemodynamics, intestinal absorption, and postshunt encephalopathy. *Surgery* 1983; 94:126-133.

## Carotid Bifurcation Imaging: Magnetic Resonance Angiography Compared to Conventional Angiography and Doppler Ultrasound\*

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*Purpose: to evaluate the clinical usefulness of the two dimensional "Time of Flight" (2D TOF) Magnetic Resonance Angiography technique (MRA) in imaging the carotid bifurcation as compared to conventional angiography and pulsed and colour Doppler ultrasound. Methods: in 19 patients with possible cerebrovascular disease and eight volunteers, contrast angiography was compared with MRA in 21 carotid bifurcations and with Doppler ultrasound in 23 bifurcations by two independent observers. In 19 bifurcations, all three techniques were available for comparison. Internal carotid arteries were graded normal/minimal disease, mild, moderate or severe stenosis, or occluded. Results: overall agreement between contrast angiography and MRA existed in 62% for one observer and 76% for the other. When MRA and Doppler agreed, agreement between these two investigations and contrast angiography existed in 77-81%. The major problem with MRA was overestimation of moderately stenosed vessels; 50% of the vessels with a moderate stenosis on contrast angiography were judged severely stenosed on MRA. An occlusion was never mistaken for a stenosis by MRA. Evaluating the separate slices, acquired in the 2D TOF MRA investigation, appeared to be essential to avoid this mistake. Conclusion: at present 2D TOF MRA is not clinically useful for diagnosing the degree of carotid artery stenosis. MRA has a clear tendency to overestimate the degree of stenosis especially moderately severe stenoses. To date, there are no objective methods to correct for this mistake. Technical improvements may make MRA a better diagnostic tool in the future.*

*Key Words: Carotid arteries; Carotid arteries angiography; Magnetic resonance angiography (MRA); Doppler ultrasound; Magnetic Resonance (MR) vascular studies.*

### Introduction

Magnetic Resonance Angiography (MRA) is a recently developed technique for imaging blood vessels. Vessels can be imaged in a three dimensional (3D) manner on a regular MR scanner in 30 min, without the use of contrast agents. Its major advantage as compared to conventional contrast angiography is the non-invasive character of MRA which thereby eliminates the risk of vascular injury, stroke and contrast reaction and the costs of post-angiography patient observation.

The purpose of this study was to evaluate the clinical efficacy of the two dimensional "Time of Flight" (2D TOF) MRA technique in imaging the carotid bifurcation on patients and volunteers as compared to contrast angiography and pulsed and colour Doppler ultrasound.

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### Materials and Methods

Between July 1991 and March 1992, MRA of the carotid bifurcation was performed on 19 patients (mean age 58) and eight volunteers (mean age 29); eight were female and 19 were male; ages varied from 6 to 79 years. Indications for the MRA examination of patients were cerebrovascular accidents in 15 and suspected carotid or vertebral artery dissection in one and two patients respectively. One patient was asymptomatic. In one of the volunteers, the bifurcation was situated higher than estimated, so it was not adequately covered by MRA.

Contrast angiography was performed and available for evaluation in 13 patients. In five patients no contrast angiography was performed. In one patient the study was lost. The quality of the intravenous digital subtraction angiogram of one bifurcation was poor and therefore was excluded from evaluation. In some cases the patient had undergone operation on

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one of the carotid arteries in the period between the separate studies; for this reason contrast angiography could finally be compared with MRA in 21 bifurcations and with Doppler in 23 bifurcations. Doppler evaluation was performed on all but one patient and three of the volunteers. Therefore, MRA could be compared with Doppler scanning in 44 bifurcations.

The time intervals between the two or three separate investigations were 2 weeks in two patients, 4 weeks in nine, less than 3 months in four and less than 6 months in three patients. In one patient, 1.5 years elapsed between contrast angiography and MRA. Since this patient had been operated on during this interval, only one bifurcation was left for comparison. This bifurcation was already occluded at the time of the contrast angiography, so comparison with MRA of this bifurcation was legitimate.

Doppler evaluation was performed by an experienced team of technicians with a 7.5 Mhz linear array transducer with pulsed and colour Doppler capabilities (Advanced Technology Laboratories, Inc.). The degree of stenosis was determined by measuring the peak systolic velocity and the end diastolic velocity. Stenoses were graded in one of five categories: 0–39% (normal/minimal stenosis), 40–59% (mild), 60–79% (moderate), 80–99% (severe) or occlusion.

Contrast angiography was performed in other institutions in five patients. Intravenous digital subtraction angiography (DSA) was performed in five patients and intra-arterial contrast injection in eight cases with conventional radiographic imaging (2) or digital subtraction imaging (6) in two planes. The degree of stenosis was calculated by comparing the most stenosed diameter with the diameter distal to the stenosed area where the vessel had a normal appearance. The degree of stenosis was determined by the reports that accompanied the exams and one observer. In those cases in which there was any doubt on the degree of stenosis, a second observer gave his judgement and a final classification was made. The stenoses were classified using the same categories as used in the Doppler evaluations.

2D TOF MRA was performed on a 1.5 Tesla Philips Gyroscan S15 with a 3 mT/m gradient system and an open head/neck receiving coil. Scan parameters were TR 60 ms, TE 14 ms and a 60° flip-angle. Sixty slices were obtained with a thickness of 3 mm and a 1 mm overlap. A cephalad presaturation slab was used so that the only strong signal arises from vessels with a cranially directed flow. On each slice only such vessels (e.g. the carotid and vertebral arteries) appear bright, surrounded by much darker stationary tissue. The examination time was about 20–25 min.

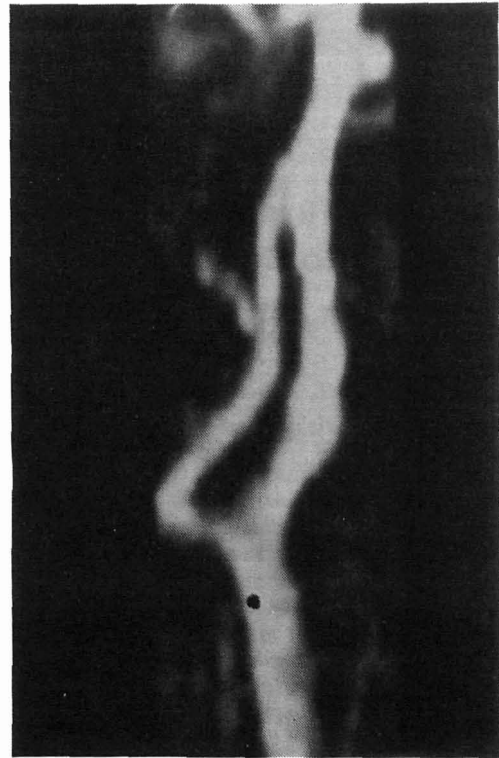


Fig. 1. Normal carotid bifurcation imaged by MRA. Apparent stenosis of external carotid artery caused by artefacts.

After this acquisition, three dimensional computer calculated reconstructions were made by using a Maximum Intensity Projection (MIP) technique, showing 15 different projections of a 12 cm long trajectory of the carotid vessels, rotating around the vertical axis from 0 to 180° (Fig. 1). From each carotid artery two different reconstructions were made: one including the vertebral artery and one of the carotid bifurcation only. These projections as well as all individual slices were recorded on film. These were evaluated by two radiologists independently who were unaware of the results of the other investigations. The degree of stenosis was classified in one of the categories mentioned above.

2D TOF MRA is called 2D because the images are acquired as separate thin (2D) slices. Another method is the 3D TOF MRA, in which the data are acquired from one large (3D) sample volume instead of thin slices. The reconstructions, calculated by the computer using the MIP technique are called 3D, because they can rotate in a movie on a monitor screen, giving it a three dimensional appearance.

## Results

Contrast angiography and Doppler agreed in 18 of 23

Table 1. Degree of internal carotid artery stenosis: Doppler versus contrast angiography

Doppler	Contrast angiography					Total
	0-39%	40-59%	60-79%	80-99%	Occlusion	
0-39%	5	1	—	—	—	6
40-59%	—	—	—	—	—	0
60-79%	—	—	3	1	1	5
80-99%	—	—	2	6	—	8
Occlusion	—	—	—	—	4	4
Total	5	1	5	7	5	23

cases (78%; Table 1). In one patient, Doppler showed a mild stenosis while on contrast angiography (and MRA) an occlusion was seen. Repeated Doppler investigation showed a large branch of the external carotid artery which was mistaken for the internal carotid artery in the first examination. In the other four cases the difference in degree of stenosis was not more than one category.

There was agreement between the findings in contrast angiography and MRA in 13 out of 21 cases for observer 1 and in 16 cases for observer 2 (62 and 76% respectively; Table 2). In no cases was an occlusion mistaken for a stenosis or vice versa. Also the difference in degree of stenosis was never more than

one category. Fifty percent (two out of four) of the moderately stenosed bifurcations on contrast angiography were graded as severely stenosed by MRA by both observers.

There was agreement between Doppler and MRA in 28 out of 44 cases for observer 1 and in 33 cases for observer 2 (64 and 75% respectively). As mentioned above, Doppler showed a stenosis in one case of an occlusion in one bifurcation. When there was disagreement between these two investigations, the difference was more than one category in two cases for observer 1 and in five cases for observer 2 (5 and 11% respectively).

In 19 bifurcations, contrast angiography, MRA

Table 2. Degree of internal carotid artery stenosis: MRA versus contrast angiography

MRA	Contrast angiography					Total
	0-39%	40-59%	60-79%	80-99%	Occlusion	
Observer 1						
0-39%	5	1	—	—	—	6
40-59%	2	—	2	—	—	4
60-79%	—	—	—	1	—	1
80-99%	—	—	2	3	—	5
Occlusion	—	—	—	—	5	5
Total	7	1	4	4	5	21
Observer 2						
0-39%	6	1	—	—	—	7
40-59%	1	—	1	—	—	2
60-79%	—	—	1	—	—	1
80-99%	—	—	2	4	—	6
Occlusion	—	—	—	—	5	5
Total	7	1	4	4	5	21

Table 3. Degree of internal carotid artery stenosis: MRA and Doppler (in agreement) versus contrast angiography

MRA and Doppler	Contrast angiography					Total
	0-39%	40-59%	60-79%	80-99%	Occlusion	
Observer 1						
0-39%	3	1	—	—	—	4
40-59%	—	—	—	—	—	0
60-79%	—	—	—	—	—	0
80-99%	—	—	2	3	—	5
Occlusion	—	—	—	—	4	4
Total	3	1	2	3	4	13
Observer 2						
0-39%	4	1	—	—	—	5
40-59%	—	—	—	—	—	0
60-79%	—	—	1	—	—	1
80-99%	—	—	2	4	—	6
Occlusion	—	—	—	—	4	4
Total	4	1	3	4	4	16

and Doppler were all available for comparison. For observer 1, Doppler and MRA agreed in 13 cases (68%) and for observer 2 in 16 cases (84%). Comparing these cases, in which Doppler and MRA agreed to contrast angiography, there was an overall agreement in 10 out of 13 bifurcations for observer 1 (77%) and in 13 out of 16 cases for observer 2 (81%; Table 3).

### Discussion

In this study contrast angiography and Doppler agreed in 78% of the cases. Results from other centres vary: Jacobs *et al.*<sup>1</sup> reports an overall agreement of 77-87%, which is similar to our results; Riles *et al.*<sup>2</sup> found an agreement in 65%; Withers *et al.*<sup>3</sup> reported an overall agreement of 94%; Steinke *et al.*<sup>4</sup> reported 80%; and Mattos *et al.*<sup>5</sup> 82%. So Doppler is not an infallible technique. Its reliability seems dependent on the skills of the technician who performs the investigation. In one Doppler examination in our study a branch of the external carotid artery was mistaken for a patent internal carotid artery where the internal carotid artery was actually occluded. Occlusion is a contraindication for operation. If the preoperative evaluation had been limited to Doppler, a mistake like this could have resulted in an unnecessary operation.

Contrast angiography is regarded as the "gold standard" for evaluation of vessel stenosis. In Litt's study, two observers agreed 72% of the time on 94 evaluated contrast angiograms of carotid bifurcations, using a similar five category system for the degree of stenosis.<sup>6</sup> This low figure largely reflects the disadvantage of the "forced-choice" ranking systems, which leads to borderline cases being placed in one category by one observer and in an adjacent category by the other observer. Also, in this study contrast angiograms were performed in different centres and by different techniques. The quality of the angiograms that were available for comparison therefore was not uniform. The poor quality resulted in one carotid study to be excluded from this study. For these reasons contrast angiography cannot simply be seen as the gold standard. Important clinical decisions however are often made on basis of contrast angiographic studies.

In this study, agreement between contrast angiography and MRA existed in 62% for one observer and in 76% for the other. This study is rather small in the number of bifurcations examined. In the recent literature, three studies evaluated exclusively the 2D TOF MRA technique for the carotid bifurcation, with classification of the degree of stenosis in five groups. Litt *et al.*<sup>6</sup> compared contrast angiography and MRA in 94 carotid bifurcations, with two separate



observers interpreting the results. Heiserman *et al.*<sup>7</sup> did the same with 73 bifurcations, evaluated by four different observers. Riles *et al.*<sup>2</sup> compared contrast angiography, MRA and Doppler in 74 bifurcations, evaluated by a team of radiologists who came to one judgement on the degree of stenosis. Although the MRA techniques in these studies differ to some extent in the parameters used, an overview of the results of these different studies is very useful in demonstrating the problems that exist at this time with the 2D TOF MRA technique.

In Table 4 the results collected from the studies in the recent literature are grouped together. A total of 553 bifurcations have been examined. Contrast angiography is used as the gold standard, despite the restrictions that exist in doing so. The middle column shows the numbers and percentages in each category in which MRA agreed. Overall agreement was 70%. In the separate studies the agreement varied from 52% (Riles)<sup>2</sup> to 79% (Heiserman).<sup>7</sup>

Closer examination of the separate categories, however, reveals that agreement is very good in the groups of normal and severely stenosed vessels (80 and 95% respectively), but poor in the intermediate groups, the mildly and moderately stenosed vessels (48 and 36% respectively). From a clinical point of view the major problem is overestimation of a moderate stenosis: 61% off the vessels with a moderate stenosis on contrast angiography were judged severely stenosed on MRA. In the three individual studies this percentage varied from 48–76%; in our study it was 50%. Even in the category of mildly stenosed vessels, 10% were seen as severely stenosed on MRA. One of the suggested criteria for operating asymptomatic patients is the existence of a 80–99% stenosis. Many patients would be operated on unnecessarily if MRA were the sole preoperative study.

The tendency to exaggerate the degree and extent of stenosis by MRA is a consequence of excessive signal loss in a stenosed segment, caused by complex flow patterns (Fig. 2). A strong signal is generated by blood flowing in a regular, laminary pattern. The MR apparatus is able to some extent to compensate for changes in flow velocity, but when this change is too large or when flow turbulence beyond a certain degree exists, then the signal diminishes or disappears. In significant stenoses, there is a significant disturbance of flow velocity, which often leads to overestimation of the degree of stenosis by MRA. Distal to a stenosis there is always a region with turbulent flow, which in general leads to overestimation of the length of the stenosis. Flow reversal also causes signal loss due to dephasing and saturation. Flow reversal in post-stenotic areas additionally leads to exaggeration of the length of the stenosis. Reversed flow can occur in the bulbous of normal bifurcations. As a result, in our study an area with decreased signal in the bulbous was seen in some healthy volunteers, which can be misinterpreted as mild stenosis. This in part could explain why in literature 17% of the normal bifurcations were judged as mildly stenosed.

In time, observers become familiar with the artefacts just mentioned and it is possible to adjust for this tendency to overestimate stenoses when interpreting MRAs. There are, however, no objective criteria for doing so; some moderately stenosed vessels are correctly demonstrated by MRA, but others are depicted as severely stenosed. As it is impossible in individual cases to know to what extent artefacts have an influence, interpreting MRAs might become a very subjective matter, in which mistakes can easily be made. To date there are no objective criteria that can be used to correct for these problems and it is questionable whether such criteria can ever

Table 4. Accuracy of MRA versus contrast angiography ("gold standard"): data from 3 recent studies

Stenosis by contrast angiography*	MRA underestimation		MRA correct	MRA overestimation		n
	> 1 category	1 category		1 category	> 1 category	
Normal			142 (80%)	31 (17%)	5 (3%)	178
Mild		28 (25%)	54 (48%)	19 (17%)	11 (10%)	112
Moderate		2 (3%)	28 (36%)	47 (61%)		77
Severe	1 (1%)	3 (2%)	127 (95%)	3 (2%)		134
Occlusion		14 (27%)	38 (73%)			52
Total	1 (< 1%)	47 (8%)	389 (70%)	100 (18%)	16 (3%)	553

\* Degree of stenosis: normal = 0–15% or 0–10% stenosed; mild = 16–49% or 11–50% stenosed; moderate = 50–79% or 50–75% stenosed; severe = 80–99% or 76–99% stenosed; occlusion = 100% stenosis.

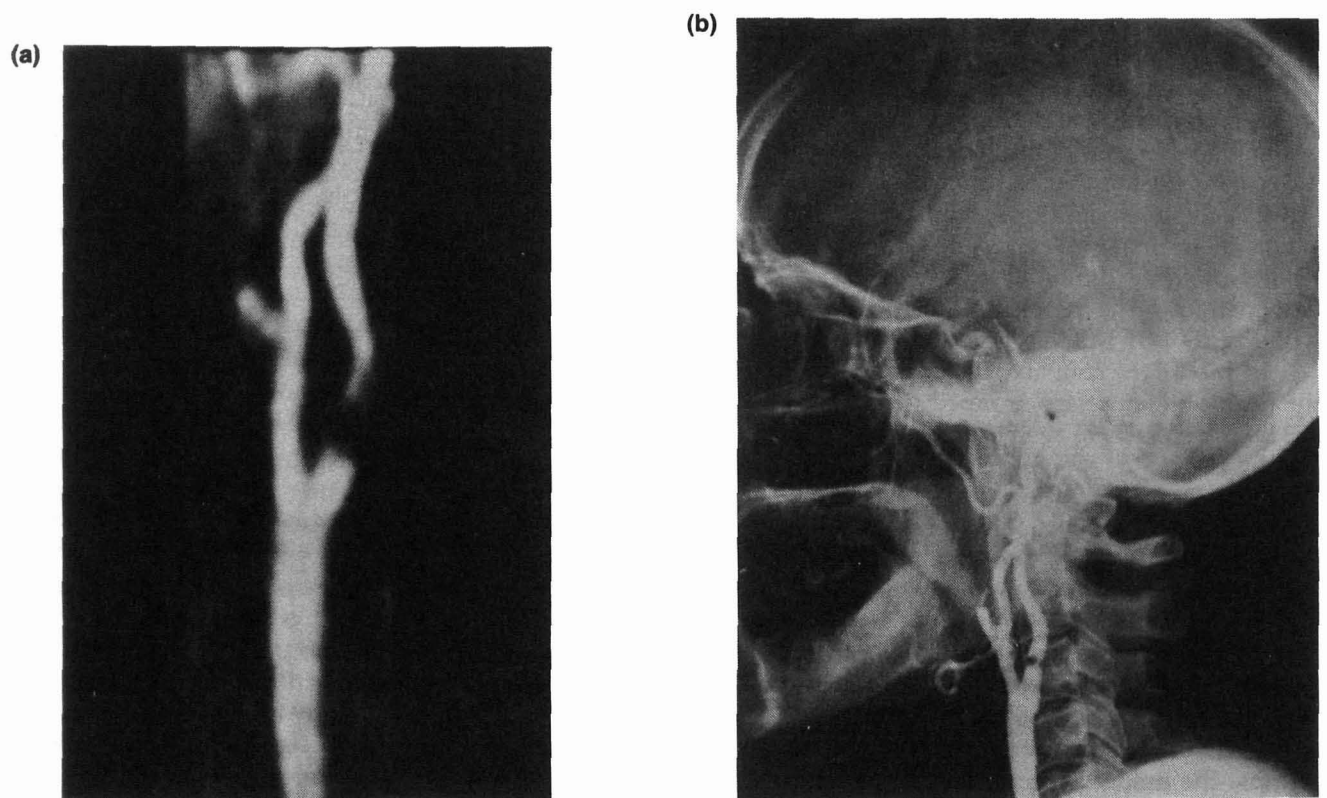


Fig. 2. Exaggeration of severity and length of stenosis by MRA (a) as compared to contrast angiography (b).

be developed. Conceivably, technical improvements may prevent these artefacts in the future: shorter echo times and smaller voxel size theoretically could give significant improvements, as well as specially designed transmitting and receiving coils.

At its current state of development 2D TOF MRA cannot be relied upon when it shows a severe stenosis as there is a significant possibility that the degree of stenosis is, in reality, only moderate. On the other hand, if a severe stenosis exists on contrast angiography, then there is a 95% chance that it will be shown as a severe stenosis on MRA. In this way, MRA might be useful as an initial screening method in order to prevent patients from undergoing an unnecessary contrast angiogram.

Another major problem is the occlusion that is mistaken for a stenosis by MRA. Table 4 shows that, in the other studies, this occurred in 14 out of 52 cases (27%). In these cases, a branch of the external carotid artery was mistaken for the occluded internal carotid artery. This misinterpretation also has a major clinical impact, as occlusion is a contraindication for operation. In our study such a mistake never occurred. However, the observers in our study did note that discriminating these external carotid artery branches

from the internal carotid artery in some cases was very difficult when looking only at the films of the 3 dimensional reconstructions of the carotid bifurcations. It was only after examining the 60 separate slices, also printed on film, that they could make the correct conclusion. On these slices in general, there is symmetry in the course of the vessels on both sides, specially at the level of the beginning of the carotid syphon. If, at this level, on one side no signal arises from the internal carotid artery then the existence of an occlusion is almost certain. In our study these separate slices appeared to be indispensable for a correct interpretation of the results. In the studies from Litt<sup>6</sup> and from Riles<sup>2</sup> in which this mistake occurred (11 and three times) the separate slices were not studied.

A severe stenosis is rarely mistaken for an occlusion on MRA, an occurrence which would otherwise have great clinical importance as well.

It has been suggested that MRA combined with Doppler might replace contrast angiography. Our study indicates that such a combination would be inadequate. Even when MRA and Doppler agreed, there was an agreement of these two studies with contrast angiography in only 77 and 81% (Table 3).



Riles<sup>2</sup> under these conditions found an agreement in 33 out of 49 cases (67%) and found that the combination of Doppler and MRA shows a tendency to overestimate the degree of stenosis. Our study, with its limited numbers of exams also shows this tendency. Therefore, the combination of MRA with Doppler cannot replace contrast angiography at this time.

Other problems with MRA are motion and swallowing artefacts that can impair the quality of the reconstructions and make interpretation very difficult. Additionally, claustrophobia sometimes causes patients to refuse a MRA. Furthermore, patients with pacemakers or some surgical clips cannot undergo MRA.

From these observations we can conclude that 2D TOF MRA alone is not reliable for evaluating the degree of carotid artery stenosis. Especially in the group of moderately stenosed vessels, there is a clear tendency to overestimate the degree of stenosis and there still is no objective way to correct for this overestimation. This is also the case when MRA is combined with Doppler. However, either method can be used as a screening method.

2D TOF is not the only MRA technique. Other techniques are 3D TOF, phase contrast and "black-blood" imaging. Anderson,<sup>8</sup> in a recent study, combined 2D TOF with 3D TOF in one investigation, using complementary information arising from each technique. Despite this strategy, the overall agreement between contrast angiography and MRA was only 64%. In his study a different category system

was used. But again, six out of 11 patients (55%) who had a 60–94% stenosis on contrast angiography appeared to have a 95–99% stenosis on MRA.

There is a possibility that technical improvements may make MRA a better diagnostic tool in the future. But at present, MRA cannot be used to replace contrast angiography.

## References

- 1 JACOBS NM, GRANT EG, SCHELLINGER D, BYRD MC, RICHARDSON JD, COHAN SL. Duplex carotid sonography: criteria for stenosis, accuracy, and pitfalls. *Radiology* 1985; 154(2): 385–391.
- 2 RILES TS, EIDELMAN EM, LITT AW, PINTO RS, OLDFORD F, THOE SCHWARTZENBERG GWS. Comparison of magnetic resonance angiography, conventional angiography and duplex scanning. *Stroke* 1992; 23(3): 341–346.
- 3 WITHERS CY, GOSINK BB, KEIGHTLEY AM, et al. Duplex carotid sonography. Peak systolic velocity in quantifying internal carotid artery stenosis. *J Ultrasound Med* 1990; 9: 345–349.
- 4 STEINKE W, KLOETZSCH C, HENNERICI M, et al. Carotid artery assessed by colour Doppler flow imaging: correlation with standard sonography and angiography. *AJR* 1990; 154: 1061–1068.
- 5 MATTOS MA, HODGSON KJ, RAMSEY DE, BARKMEIER LD, SUMMER DS. Identifying total carotid occlusion with colour flow duplex scanning. *Eur J Vasc Surg* 1992; 6: 204–210.
- 6 LITT AW, EIDELMAN EM, PINTO RS, et al. Diagnosis of carotid artery: comparison of 2DFT Time-of-flight MR angiography with contrast angiography in 50 patients. *AJR* 1991; 156: 611–616.
- 7 HEISERMAN JE, DRAYER BP, FRAM EK, et al. Carotid artery stenosis: clinical efficacy of two-dimensional time-of-flight MR angiography. *Radiology* 1992; 182(3): 761–768.
- 8 ANDERSON CM, SALONER D, LEE RE, et al. Assessment of carotid artery stenosis by MR angiography: comparison with X-ray angiography and colour-coded Doppler ultrasound. *AJNR* 1992; 13: 989–1003.

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## CT Angiography: Application to the Evaluation of Carotid Artery Stenosis<sup>1</sup>

**PURPOSE:** To determine whether computed tomographic (CT) angiography can be used to accurately quantify carotid stenoses and differentiate severe stenoses from occlusions.

**MATERIALS AND METHODS:** CT angiography was used to evaluate 50 carotid arteries in 27 patients who were referred for surgical evaluation after diagnosis of carotid stenosis and who had undergone standard angiography. Four observers read each study separately in a blinded fashion, then all four observers together reviewed those studies in which the individual readings differed to reach a consensus reading.

**RESULTS:** The consensus readings for the two modalities were in agreement on stenosis category in 41 (82%) of the 50 carotid arteries ( $\kappa_w = 0.852 \pm 0.114$ ). The consensus readings in the other nine arteries differed by only one category; seven had less severe stenosis at CT angiography and two had more severe stenosis. The CT angiograms also depicted a variety of additional abnormalities, including loops ( $n = 6$ ), aneurysms ( $n = 2$ ), and ulcers ( $n = 4$ ).

**CONCLUSION:** These results indicate that CT angiography can non-invasively provide most of the information needed before carotid endarterectomy.

**Index terms:** Carotid arteries, stenosis or obstruction, 172.721 • Computed tomography (CT), comparative studies, 172.12116 • Computed tomography (CT), preoperative, 172.12116 • Computed tomography (CT), three-dimensional, 172.12117

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CAROTID endarterectomy is now widely used to treat carotid stenoses and thereby reduce the risk of subsequent stroke (1,2). The North American Symptomatic Carotid Endarterectomy Trial (NASCET) reported an absolute reduction in risk of ipsilateral stroke of 17% at 2 years in patients with greater than 70% carotid stenosis who were treated surgically compared with medically treated patients (1). Therefore, in order to identify patients who will benefit from endarterectomy, Masaryk and Obuchowski have argued that the goals of carotid imaging must be to accurately quantify the degree of stenosis, to differentiate between severe stenosis and occlusion, and to detect any associated abnormalities such as tandem lesions that may affect the surgical procedure (3). In addition, in accomplishing these goals, the imaging modality should subject the patient to a minimum of risk and the health care system to a minimum of cost.

Three imaging modalities, selective carotid arteriography, pulsed and color Doppler ultrasound (US), and magnetic resonance (MR) angiography, have been extensively evaluated in assessing carotid disease. Selective carotid arteriography is the standard against which all other modalities are usually compared. This technique accomplishes all of the anatomic imaging goals as defined by Masaryk and Obuchowski but does so with a small but definite risk of causing a stroke, which is what the evaluation and surgical procedure were meant to prevent, and with the costs of a period of inpatient observation and of treatment of any complications that may

occur (4-6). To reduce these risks and costs, the noninvasive modalities of US and MR angiography have been developed and evaluated. US is widely used as a screening modality. While the accuracy of US in detecting severe stenoses was suboptimal in the NASCET study, other authors have reported accuracies of  $\approx 90\%$  in diagnosing severe stenoses (7,8). The more recently developed technique of MR angiography has improved in accuracy in recent years, and recent reports indicate a sensitivity of over 90% in detecting severe stenoses (9,10). However, the persistent tendency to overestimate stenoses with MR angiography results in positive predictive values for severe stenoses as low as 39% (9). Polak et al have reported that the combination of US and MR angiography may provide sufficient preoperative information in most patients (11). However, both US and MR angiography have been reported to yield false-positive and false-negative results in diagnosing carotid occlusions, a differentiation that is crucial to surgical planning (7,12-14). Heiserman et al recently reported 100% accuracy in differentiating severe stenosis from occlusion using MR angiography (10). Nevertheless, the number of subtotal occlusions studied with MR angiography remains small, and the accuracy of MR angiography in this respect remains unknown.

CT angiography is a new noninvasive vascular imaging technique in which angiographic images are produced by performing three-dimensional (3D) display or reconstruction of vessel anatomy as depicted on the overlapping images obtained with helical CT. In 1984, Heinz et al de-

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**Abbreviations:** DSA = digital subtraction angiography, NASCET = North American Symptomatic Carotid Endarterectomy Trial, 3D = three-dimensional, 2D = two-dimensional.

scribed the use of thin-section dynamic CT to allow direct visualization of carotid atheroma and thrombi and demonstrated the possibility of making 3D reconstructions of the carotid artery (15,16). However, because the CT scanners in use at that time were relatively slow, a bolus of contrast material was followed by a very slow rate of contrast infusion (50–60 drops per minute), and only a small length of carotid artery could be imaged during the injection phase. The recent technical developments of slip-ring CT, which allows very rapid imaging, and independent 3D workstations that allow rapid manipulation of data have reawakened interest in the possibility of using contrast material-enhanced CT for vascular imaging (17–19). This study was therefore performed to ascertain whether these new technical developments could be used to accurately quantify carotid stenoses and differentiate severe stenoses from occlusions.

## MATERIALS AND METHODS

### Patients

All patients with symptomatic carotid disease who were referred for surgical evaluation and who had undergone or were going to undergo angiography were asked to participate in the study. Criteria for referral included angiography performed elsewhere ( $n = 21$ ) or at our institution ( $n = 15$ ) and color and duplex Doppler sonography performed at our institution ( $n = 14$ ). All patients who were asked to participate in the study did so. A variety of angiography techniques were used, including selective carotid angiography, aortic arch injection angiography, and intravenous digital subtraction angiography (DSA). For an artery to be included in the study, at least two angiographic views of the bifurcation had to be available.

A total of 27 patients (54 carotid arteries) were included in the study. They ranged in age from 37 to 79 years (mean, 62.5 years  $\pm$  10.9 [standard deviation]). There were 16 men and 11 women. All were being evaluated for known or suspected atherosclerotic disease except the youngest patient, who had a known dissection involving one carotid artery. Four carotid arteries were excluded from further evaluation for the following reasons: in two instances, there was no corresponding angiographic image of the carotid bifurcation; one artery was affected by a known dissection, a disease not included in the current study; and one CT angiographic study was unsuccessful due to swallowing artifact on the CT scan. Therefore, a total of 50 carotid arteries were available for evaluation. Angiograms were obtained by means of selective carotid injection in 17 arteries, aortic arch injection in 21, and intravenous

injection in 12. In 13 arteries, CT angiography preceded standard angiography by 7.9 days  $\pm$  9.0 (range, 2–28 days). One artery was examined by both methods on the same day. Standard angiography was performed before CT angiography in 36 arteries; 32 were studied within 4 months before CT angiography (mean, 42.7 days  $\pm$  37.9; range, 1–118 days). Two patients (four arteries) were examined with angiography 9 and 12 months before CT angiography.

### Imaging

The CT scans were obtained with a new slip-ring CT scanner capable of performing 50 consecutive rotations in 50 seconds (Tomoscan SR-7000; Philips Medical Systems International, Best, The Netherlands). The patients were positioned supine on the CT gantry table with the head tilted back as far as comfortably possible in order to avoid artifact from fillings in the teeth. A lateral scanogram was then obtained. With use of the scanogram as a guide, the gantry was angled so that it was perpendicular to the presumed course of the carotid arteries. Again with use of the lateral scanogram, the distance from the anterior arch of the first cervical vertebra to the midbody of the seventh cervical vertebra was measured to ascertain the number of gantry rotations required to image this area with use of one rotation per second and a table speed of 3 mm/sec. Typically, 40 rotations were performed, although some patients required as few as 35 or as many as 50.

The section thickness was 3 mm, and a section was reconstructed every 1 mm, resulting in 105–150 3-mm sections each overlapping 2 mm with each adjacent section. The actual number of sections depended on the number of gantry rotations that had been performed. The other CT scan parameters were as follows: 120 kV, 250 mAs, 512  $\times$  512 matrix, and 167-mm field of view. In one patient, a field of view of 156 mm was used, and in another, a field of view of 250 mm was used.

Intravenous contrast material (ioxitalamate [Telebrix; Guerbet Nederland, Gorinchem, The Netherlands] [350 mg iodine per milliliter]) was mechanically injected through a standard catheter placed in an antecubital vein. In most patients, a volume of 120 mL was injected at a rate of 1.8 mL/sec, with a scan delay of 35 seconds. While we were optimizing the contrast material protocol early in the study, variations occurred in the volume and rate of contrast material as well as in scan delay (volume range = 90–140 mL, rate range = 1.4–2.0 mL/sec, and scan delay range = 25–35 seconds). Since the 3D reconstruction technique (described in detail below) allowed exclusion of calcified plaque, a non-contrast-enhanced series was not obtained.

Quiet breathing was permitted during the examination. The patients were strongly encouraged to refrain from swallowing during the period of actual CT data acquisition, usually about 40 seconds.

The scanner then required approximately 15 minutes to reconstruct the sections.

### Three-dimensional Reconstruction

The data from the CT sections were then transferred to an independent workstation for 3D reconstruction (Gyrovie HP, Philips Medical Systems International). A surface-rendering 3D technique was used with a "region-growing" segmentation process as described below (20,21). The reconstructions were performed by one of two operators (E.H.D., M.A.F.) without knowledge of the results of angiographic assessment.

To perform region-growing segmentation, minimum and maximum Hounsfield density levels for the region must be defined. An objective means of determining the minimum Hounsfield density level for the carotid segmentation range was used to minimize operator dependence. First, the two-dimensional (2D) CT images were viewed to determine the site of maximal carotid stenosis. The density of the central portion of the lumen ( $D_L$ ) at that level was either directly measured or calculated. Direct measurement was performed if the lumen was large enough to place a region of interest in the central portion of the lumen away from the periphery of the lumen, where the individual voxels are composed partly of lumen and partly of plaque. When direct measurement was not possible due to small cross-sectional area, the density was calculated by measuring the density of the lumen, again only of the central portion, a few millimeters above and an equal distance below the stenosis and averaging these values. The density of soft tissue ( $D_{ST}$ ) was determined by measuring the density of the ipsilateral sternocleidomastoid muscle. The minimum segmentation level ( $D_{MIN}$ ) was then calculated with the following formula:  $D_{MIN} = D_{ST} + 0.7(D_L - D_{ST})$ . The mean  $D_L$  was 299.6  $\pm$  59.7 (range, 212–447), the mean  $D_{ST}$  was 86.0  $\pm$  13.6 (range, 57–127), and the mean  $D_{MIN}$  was 235.3  $\pm$  43.8 (range, 175–342). The maximum segmentation level was set so that the cortex of the vertebral bodies and thus the central portion of any calcified plaques was excluded from the segmentation range.

This segmentation range was strictly adhered to within several centimeters above and below the stenosis. Toward the more caudal and cranial extents of the carotid arteries, the segmentation range was not applicable due to changes in the concentration of contrast material and beam hardening from the shoulders and skull and mandible. In those locations, a more subjective segmentation range was used to adjust for the change in Hounsfield numbers, since these regions were not crucial to displaying the stenosis.

Having defined the segmentation range, the carotid arteries were then segmented by using a region-growing process in which a "seed" is placed in the vessel lumen and all contiguous pixels that have a density in the segmentation range are included in the defined lumen. Regardless

**Table 1**  
**CT Angiography versus Standard Angiography: Consensus Readings**

Stenosis Category at CT Angiography	Stenosis Category at Standard Angiography			
	Mild	Moderate	Severe	Occluded
Mild	16	3	0	0
Moderate	1	4	3	0
Severe	0	1	14	1
Occluded	0	0	0	7

Note.—Numbers are numbers of consensus readings. Indices of concordance for consensus readings were as follows: percentage agreement = 82.0%, weighted percentage agreement = 94.0%, and  $\kappa_w = 0.852 \pm 0.114$ .

**Table 2**  
**CT Angiography versus Standard Angiography: Individual Readings**

Stenosis Category at CT Angiography	Stenosis Category at Standard Angiography			
	Mild	Moderate	Severe	Occluded
Mild	63	9	0	0
Moderate	6	19	12	0
Severe	0	5	52	6
Occluded	0	0	2	26

Note.—Numbers are numbers of individual readings. Indices of concordance for individual readings were as follows: percentage agreement = 80.0%, weighted percentage agreement = 93.3%,  $\kappa_w = 0.835 \pm 0.057$ .

of the segmentation range used, the edges of calcified plaque will fall within that range owing to partial volume effects. Such calcified plaques were manually removed from the segmented lumen by drawing a boundary line. The cervical portions of the common and internal carotid arteries were segmented, but only the proximal external carotid artery was included, since that artery was not the focus of this study.

A 3D reconstruction of each carotid artery was then performed and displayed by using a shaded-surface display projection. Having viewed the CT images during the segmentation process, the operator had an idea of how the reconstruction should appear. If any unexpected findings were observed in the 3D image, the 2D CT images were reviewed to ensure that no mistake such as inappropriate placement of the seed had been made in the segmentation process. Finally, the view of each carotid bifurcation that showed the most severe luminal narrowing was recorded on film. The entire 3D reconstruction process required between 30 and 45 minutes to produce a 3D image of both carotid arteries.

**Evaluation**

The CT angiograms and standard angiograms were reviewed separately and in a blinded fashion by each of four observers (E.H.D., M.S.v.L., B.C.E., W.P.T.M.M.) in order to quantify the degree of carotid stenosis. The severity of the stenosis was determined by comparing the narrowest diameter of the stenotic segment with the diameter of the internal carotid artery well beyond the bulb (1,22). The stenoses were

graded according to the following classification scheme: mild = 0%–19% diameter reduction, moderate = 20%–69%, severe = 70%–99%, and occlusion = 100%. This classification system maintains the critical criterion of 70% as the level at which a stenosis is graded as severe that was recommended as a result of the NASCET study (1,22).

Each observer first made a subjective assessment of the degree of stenosis. If the carotid artery was normal or occluded, no objective measurement was made. In the other cases, an objective measurement was made by using vernier calipers (accurate to 0.01 mm). The site of maximal stenosis was measured and compared with the more distal, postbulbar internal carotid artery, as in the NASCET study (1). The percentage of diameter reduction was then calculated. When an observer's subjective assessment and objective measurements resulted in different stenosis categories for the same carotid artery, the observer was asked to review the films and make a final decision on the stenosis category. Finally, cases in which there was disagreement between the observers were reviewed by all four observers together in order to reach a consensus.

The stenosis category indicated by the CT angiogram was compared with that indicated by the standard angiogram. A percentage agreement and a weighted percentage agreement between the two studies were calculated for the individual readings as well as for the group consensus reading. The latter statistic weights the results to distinguish between varying degrees of disagreement (eg, one-category disagreement vs two) but does not correct

for chance agreement (23). In addition, a weighted  $\kappa$  index ( $\kappa_w$ ) was calculated for the individual and consensus readings. This index is a measure of concordance with a correction for chance agreement and weights the results to distinguish between varying degrees of disagreement between the two modalities being compared. It is the statistic of choice for measuring the agreement of ordinal data (data in which there are three or more categories that bear a ranked relationship) (23).

**RESULTS**

Fifty carotid arteries were evaluated in this study. The consensus readings of the CT angiograms and those of the standard angiograms were in agreement in 41 (82%) of the 50 arteries (Table 1). The stenosis category at CT angiography was one category less severe than at angiography in seven arteries (14%) and one category more severe in two (4%). No readings were more than one category apart. Seventeen arteries were examined by using a selective carotid injection. There was agreement on stenosis category in 14 (82%) of these 17 arteries. Twenty-one arteries were examined by using arch aortography, and there was agreement in 17 (81%) of these 21 arteries. Twelve arteries were examined by using intravenous DSA, and there was agreement in 10 (83%) of these 12 arteries.

There was a discrepancy between severe stenosis and occlusion in only one case. In that case, patency was observed on the CT angiogram but not on the selective carotid DSA image obtained 28 days later. In retrospect, a "string sign" of pseudo-occlusion may have been present on the standard angiogram, in which case angiography was falsely positive for occlusion.

Each of the 50 arteries was graded separately by four observers, resulting in a total of 200 readings. The individual readings for CT angiography were in agreement with those for angiography in 160 (80%) of the readings (Table 2). The stenosis category at CT angiography was one category less severe in 27 readings (13.5%). The stenosis category at CT angiography was more severe by one category in 13 readings (6.5%). The rates of agreement for the four individual observers were as follows: observer 1, 86%; observer 2, 76%; observer 3, 76%; and observer 4, 82%.

There was a discrepancy between severe stenosis and occlusion in eight of the 200 individual readings. Four of these readings concerned the artery mentioned above that appeared to be

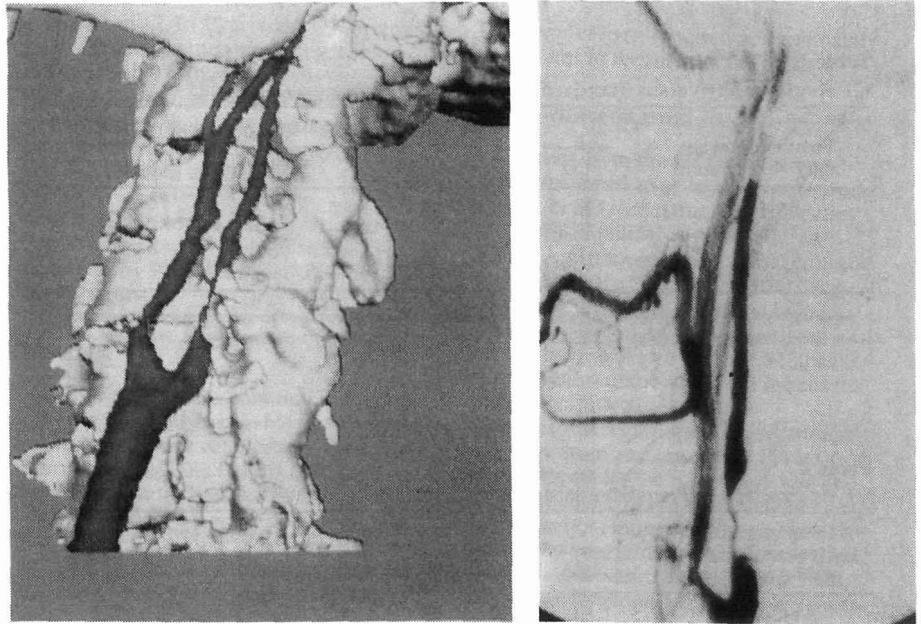
patent at CT angiography but was read as occluded on an angiogram obtained 28 days later. Two other readings were falsely positive for occlusion at angiography owing to failure by two observers to notice a string sign on the angiogram, indicating patency. The other two readings were falsely negative for occlusion at angiography: In one artery, one observer misinterpreted a stenotic external carotid as a patent internal carotid; in another artery, one reader called the cervical carotid patent after observing a patent carotid siphon, which the other readers thought filled through intracranial collateral vessels.

### DISCUSSION

Masaryk and Obuchowski recently argued that a carotid imaging modality should be judged by its ability to allow accurate quantification of the degree of stenosis, differentiation of severe stenosis and occlusion, and detection of associated abnormalities such as tandem lesions that may affect the surgical procedure (3). The purpose of this study was to ascertain if CT angiography could be used to accomplish the first two of these three anatomic imaging goals.

The results of this study suggest that even at this early stage in the development of CT angiography, stenoses can be quantified with this modality. Specifically, the stenosis category indicated by the consensus CT angiographic readings agreed in 82% of cases with that of the consensus angiographic readings. When the results of the two studies differed, they were only one category apart. The correlation of CT angiography with standard angiography varied for the different stenosis categories. Seventeen mildly stenotic arteries were identified with angiography, and CT angiography allowed correct classification of 16 (94%). Eight moderately stenosed arteries were evaluated, and four appeared to be moderately stenosed at CT angiography. There were 17 severely stenotic arteries at angiography, and CT angiographic results were in agreement in 14 (82%) (Fig 1). The other three were classified as moderately stenosed with CT angiography. There were eight arteries interpreted as occluded at angiography. Seven (88%) of these were interpreted as occluded at CT angiography, and one was interpreted as severely stenotic. This case is discussed in detail below.

These results may be compared with those of a recent study by



**Figure 1.** Severe stenosis of the left internal carotid artery is visible on both the CT angiogram (a) and the selective left carotid angiogram (b).

Schwartz et al (19). They compared CT angiography with standard angiography in 40 arteries and found agreement in 92% of cases. There were a few differences between these two studies, which may account for some of the difference in correlation between CT angiography and angiography.

All of the angiograms in the study of Schwartz et al were obtained with selective carotid injection (19). The angiograms in the current study were obtained with aortic arch injection in 21 arteries, selective carotid injection in 17 arteries, and intravenous DSA in 12 arteries. Intravenous DSA is known to occasionally produce erroneous results and is thus not an ideal standard of reference, but we chose to include these 12 arteries since the cases were available (24). To allow for the shortcomings of intravenous DSA, the correlations between the various angiographic techniques and CT angiography were separately analyzed. There was no major difference in correlation with CT angiography between the groups. CT angiography correlated with angiography in 82%, 81%, and 83% of arteries, respectively, for selective carotid injection, aortic arch injection, and intravenous DSA.

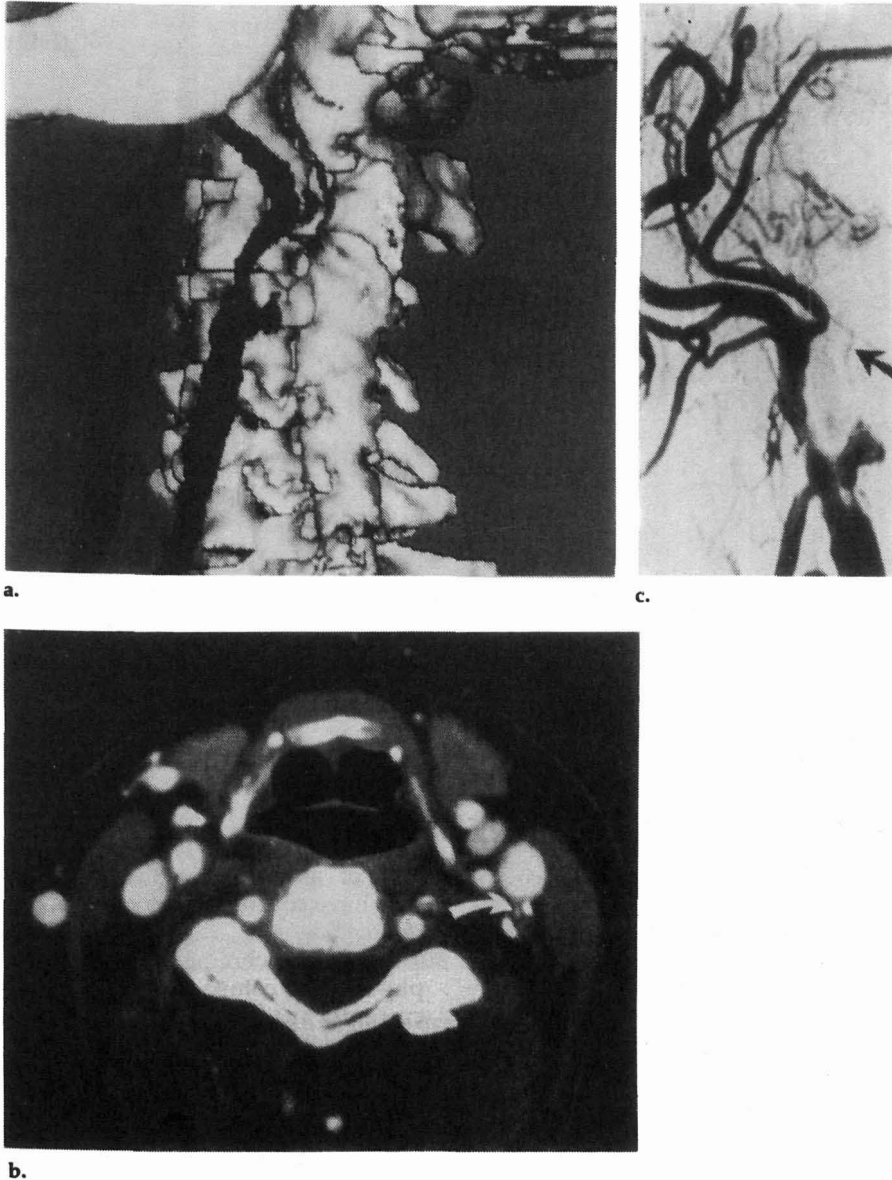
The same number of stenosis categories was used in both studies, and both studies used 70% as the criterion for severe stenosis, as recommended by Fox (22) in response to the NASCET study (1). The only difference in stenosis categories between

the two studies is that Schwartz et al used 30% stenosis as the point of differentiation between mild and moderate stenosis whereas the current study used 20% stenosis for this differentiation.

Perhaps the most important difference between this study and that of Schwartz et al is the method by which the minimum segmentation level was determined. The choice of minimum segmentation level is crucial in use of surface-shaded displays to assess the degree of stenosis. Early in our experience with CT angiography, it became apparent that an objective means of determining this level was needed, since different operators made different subjective assessments and thus produced images of the same carotid artery showing different degrees of stenosis. Schwartz et al addressed this problem by measuring the attenuation value of the intraluminal contrast material at the narrowest point in the artery (19). While this method is appealing due to its ease of application, it adds a degree of variability, since the lumen is very small in cases of moderate to severe stenosis. In such cases, it is difficult to obtain reliable density measurements owing to the small number of pixels representing the lumen and the partial volume averaging of the pixels on the periphery of the lumen with adjacent structures.

The minimum segmentation level used in this study was reached by using the following reasoning: Voxels





**Figure 2.** Severe stenosis versus occlusion. (a) CT angiogram reveals apparent discontinuity in the proximal left internal carotid artery (often seen in severe stenoses with CT angiography), with a patent lumen beyond the discontinuity. Also visible is severe stenosis of the right external carotid artery. (b) CT image demonstrates small contrast material-opacified lumen of left internal carotid artery (arrow). Note the mural calcifications. (c) Selective left carotid angiogram obtained 28 days after CT angiography was interpreted as showing occlusion. There is a vague suggestion of a string sign of pseudo-occlusion (arrow).

ability to allow differentiation of severe stenoses from occlusions, since endarterectomy is possible in the former situation but not in the latter (3). No imaging technique allows 100% accuracy in making this distinction. Even angiography may yield false diagnoses of occlusion by failing to demonstrate slow flow through a highly stenosed lumen (25). Schwartz et al reported 100% accuracy in making this distinction with CT angiography (19). There was only one artery in the current study for which the consensus readings of the two studies disagreed regarding occlusion (Fig 2). In that case, patency demonstrated on the CT angiogram was not demonstrated on the angiogram obtained 28 days later. Review of the contrast-enhanced CT images revealed that an almost punctate opacified lumen was visible centrally within the internal carotid artery on 55 overlapping images covering a distance of 55 mm (all the way up to the most cephalad image), an observation that would appear to indicate that the carotid artery was patent at the time of CT angiography (Fig 2b). It is conceivable that the subtotal occlusion apparently present at the time of CT angiography had progressed to complete occlusion by the time of angiography. However, review of the selective carotid DSA image obtained after CT angiography did suggest a possible string sign, in which case angiography was falsely positive for occlusion (Fig 2c). Perhaps a cut-film arteriogram might have better demonstrated the possible string sign, but such a study was not obtained, since the initial injection image was interpreted as showing occlusion.

One great advantage of CT in general is its ability to allow detection of slight differences in contrast. Since the 2D CT images allow visualization of the course of the internal carotid artery whether patent or not, a very small opacified lumen (which may not be visualized with angiography in

on the edge of the vessel lumen are composed partly of lumen and partly of atherosclerotic plaque. If a voxel is composed of 50% or more vessel lumen, as indicated by its density relative to that of the lumen and plaque, then it should be included in the segmented lumen. Since the plaque was often too small to allow placement of a sufficient region of interest for reliable density measurement, a decision was made to use the density of soft tissue as represented by the ipsilateral sternocleidomastoid muscle at the level of the stenosis as a rough approximation of that of soft plaque. By using this approximation and by including voxels with a density halfway between that of soft tissue and the lumen in a preliminary set of reconstructions, the stenosis on the CT angiograms tended to appear less severe than on the corresponding angiograms.

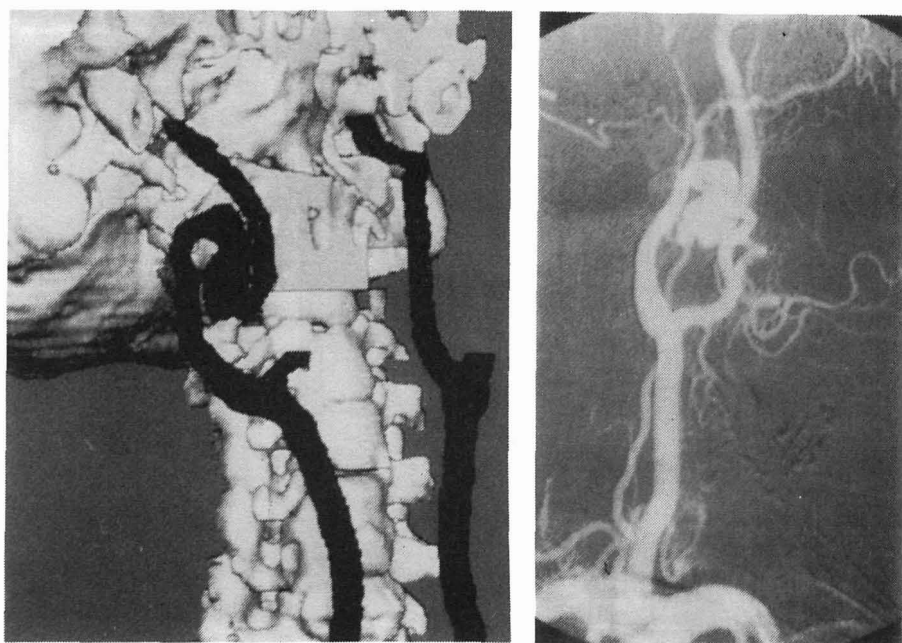
Therefore, an empirical choice was made to include voxels with a density representing 70% lumen and 30% soft tissue. Even with use of this 70% level, there was still a tendency to underestimate stenoses in this study. Seven of the nine arteries for which the stenosis category was not in agreement with that on the angiogram appeared less severe on the CT angiogram. Increasing the minimum segmentation level above that used in this study will decrease this tendency toward underestimation but may concomitantly increase the chance of producing 3D images depicting stenoses that are not real. Since the minimum segmentation level is crucial to accuracy in CT angiography, further research must be done to optimize it.

The second anatomic goal of any carotid imaging modality as defined by Masaryk and Obuchowski is its

cases of pseudo-occlusion) would be expected to be visualized on the 2D CT images, especially since the contrast material is administered over a relatively long time, thus opacifying any patent arterial lumen. A similar use of CT to establish patency of the distal internal carotid artery when cerebral arteriography failed to allow visualization of this structure was reported by Riles et al in 1982 (26). Future research should address the question of whether CT might be more sensitive than typical angiography in detecting pseudo-occlusions. However, until proved otherwise, cut-film arteriography with a prolonged selective injection and prolonged filming remains the standard of reference and should be performed in cases of suspected subtotal occlusion (25).

The third goal of any carotid imaging technique as indicated by Masaryk and Obuchowski should be to detect any associated abnormalities such as tandem lesions that may affect the surgical procedure (3). The technique used in our study was intended to yield a thorough evaluation of the entire cervical carotid artery and did, in fact, result in the display of several abnormalities in addition to stenosis, including carotid loops ( $n = 6$ ), aneurysms ( $n = 2$ ), and ulcers ( $n = 4$ ) (Figs 3, 4). Since the CT scanner used in this study allowed 50 seconds of uninterrupted scanning, we were able to image the entire cervical carotid artery (up to 15 cm of scanned length with a table speed of 3 mm/sec). The capability of this CT scanner to scan for 50 continuous seconds represents a substantial improvement over that used in the study by Schwartz et al, in which only 24 seconds of continuous scanning was performed, resulting in imaging of a smaller length of the carotid artery.

Nevertheless, our technique did not allow imaging of the carotid origin or the carotid siphon, the two locations at which tandem lesions are most frequent (27). The surgical importance of these tandem lesions has been the subject of recent debate. Masaryk and Obuchowski argue that detection of these lesions is crucial before performing carotid endarterectomy, since patients with such lesions are at greater risk for intra- and perioperative strokes and significant cardiac complications (3). Polak argues that such lesions are uncommon and that, in any case, detection of such a lesion would probably not result in postponement of surgery (28). The surgical importance of tandem lesions and



**Figure 3.** Carotid loop with aneurysm and ulcer. (a) CT angiogram, (b) selective carotid angiogram. Both studies demonstrate a normal right carotid bifurcation and a more distal loop, with aneurysmal dilatation and a projecting ulcer on the descending limb. The external carotid arteries were not reconstructed, thus accounting for their amputated appearance on the CT angiogram.

whether CT angiography could be used to detect them are not yet clearly known and may provide the basis for future research.

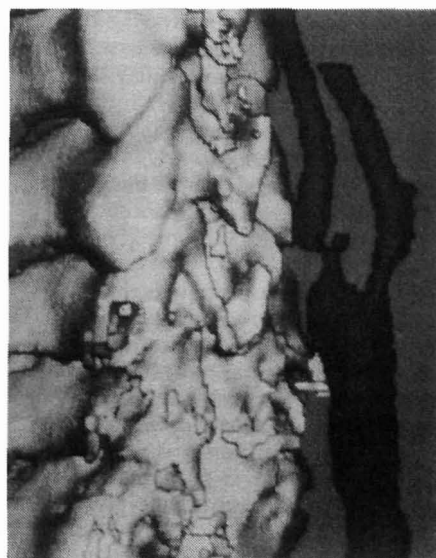
One limitation of the current study is the time interval between CT angiography and standard angiography, raising the possibility of disease progression between performance of the two studies (29). Both studies were performed within 30 days of each other in 31 of the 50 arteries, between 31 and 60 days of each other in five arteries, between 61 and 90 days of each other in six arteries, and more than 90 days apart in eight arteries. The time interval between the two studies in the 41 cases in which agreement was observed was  $58.1 \text{ days} \pm 97.1$ . Disease progression between the two studies might be expected to manifest itself in the nine arteries in which the CT and standard angiograms were in disagreement. In four arteries in which disagreement occurred, the worst disease was seen on the second study, which was obtained  $41.5 \text{ days} \pm 43.7$  after the first study. In the other five cases of disagreement, the worst disease was seen on the first study, which preceded the second study by  $45.4 \text{ days} \pm 34.1$ . Therefore, given the roughly equal distribution of cases in which the first study demonstrated the worst stenosis and those in which the second study demonstrated the worst steno-

sis, disease progression is not a convincing explanation for all of the nine cases of disagreement, although it may have contributed to some of them.

As a new technique, CT angiography must be compared with the other currently available noninvasive modalities. US uses no contrast material and is an excellent screening modality, with a sensitivity as high as 90% in detecting severe stenoses (7,8,12). However, US may result in a false diagnosis of occlusion in cases of severe stenosis (7,9,12,14). In addition, it fails to provide an overview of the carotid circulation, as may be needed to guide surgery (27). Such an overview may allow detection of tandem stenotic lesions or distal loops or coils of the internal carotid artery, such as that seen in Figure 3, that may affect the surgical procedure. These more distal abnormalities are not so well evaluated with US (27). Also, many surgeons prefer confirmation of the US diagnosis before performing endarterectomy, since the surgical procedure itself is associated with a total perioperative complication (death or stroke) rate of 5.8%–7.6% (1,2). CT angiography as performed in this study allowed thorough examination of the entire cervical carotid artery and quantification of any visualized stenosis but did not provide evaluation of the carotid origin or carotid

**Table 3**  
**Statistical Indices and Failure Rates: CT Angiography and MR Angiography**

	Statistical Indices							
	Weighted Kappa Index	Percentage Agreement (%)	Weighted Percentage Agreement (%)	Sensitivity (%)	Specificity (%)	Positive Predictive Value (%)	Negative Predictive Value (%)	Failure Rate (%)
CT angiography								
Current study	0.852	82.0	94.0	82.4	93.9	87.5	91.2	1.9
Schwartz et al (19)	0.929	92.5	97.5	100.0	100.0	100.0	100.0	0.0
MR angiography								
Huston et al (9)	0.731	59.0	89.0	90.0	83.1	39.1	98.6	5.1
Heiserman et al (10)	0.852	79.1	94.6	100.0	94.7	66.7	100.0	7.3



**Figure 4.** Stenosis with ulcer. (a) Aortic arch injection angiogram reveals severe stenosis and ulceration of the proximal right internal carotid artery. (b) CT angiogram depicts the ulcer, but the stenosis appears less severe.

siphon, and thus abnormalities in these regions would have been overlooked had they been present.

MR angiography of the carotid arteries has been the subject of much recent research. MR angiography does provide an overview of the carotid circulation, but its accuracy in diagnosing bifurcation stenoses may be hampered by signal loss in areas of turbulent flow (13,30-33). However, recent studies by Huston et al and Heiserman et al using state-of-the-art 2D time-of-flight MR projection angiography have reported improvements over previous studies in the accuracy of MR angiography (9,10). Since the raw data were reported for both of these MR angiographic studies as well as for the CT angiographic study by Schwartz et al (19), the data can be subjected to statistical analysis for comparison purposes, as shown in Table 3. The values of the statistical indices for each study are affected by

the number of vessels studied, the number of stenosis categories (four or five), the definition of these categories (particularly the definition of severe stenosis, which had a minimum value of 70%, 76%, and 80% in the different studies), and the distribution of disease among these categories. Nevertheless, these statistical indexes do provide some basis for comparison.

All of these studies involve ordinal data (the classification of disease into categories of severity). Therefore, indices of concordance rather than trend are most appropriate for comparison. The most useful of these indices is the weighted  $\kappa$  index ( $\kappa_w$ ), which corrects for chance agreement and weights the results to distinguish between varying categories of disagreement (23). If perfect agreement existed between the two compared modalities (MR angiography or CT angiography vs standard angiography), the index would have a value of

1.0. To calculate sensitivity, specificity, and positive and negative predictive values, the ordinal data must be converted into dichotomous data (data in which there are only two categories, ie, disease is either present or absent). This can be accomplished by defining a positive test result as one that shows the presence of surgical disease (ie, severe stenosis) and a negative test result as one that shows the absence of surgical disease (ie, occlusion or nonsevere stenosis).

The results of this statistical analysis for these two CT angiographic and two recent MR angiographic studies are shown in Table 3. The values for CT angiography for all of these statistical indices compare favorably with those for MR angiography. The largest difference between the two modalities appears to be in the positive predictive values. Owing to the overestimation of stenosis that may occur with MR angiography, the predictive value of a positive MR angiogram (ie, surgical disease is present) can be much less than that of a positive CT angiogram. Thus, there is much less certainty that a patient with a positive MR angiogram actually has surgical disease than when the patient has a positive CT angiogram. The predictive value of a negative result (ie, surgical disease is not present) is very high for both modalities.

Another advantage of CT angiography compared with MR angiography that is evident from Table 3 is the lower failure rate of CT angiography. This failure rate is related to the period of time required for data acquisition during which the patient must remain motionless: a maximum of 50 seconds for CT angiography versus 10-15 minutes for MR angiography. In addition, CT angiography can be performed in patients with cardiac pacemakers and in claustrophobic patients.

CT angiography does, however,



have certain disadvantages that are inherent to the technique. Foremost among these is the use of a bolus of intravenous contrast material, a disadvantage not shared by US and MR angiography. The use of contrast material subjects patients to all of the well-known risks of nephrotoxicity and allergic reaction. In addition, the use of a bolus might be problematic in patients with diminished cardiac output. However, the total volume and rate of contrast material used in this study were essentially the same as those used daily for routine CT applications and thus subject the patient to no more risk than any other contrast-enhanced CT study. Another disadvantage of CT angiography is that there is some degree of operator dependence in performing the segmentation for the 3D reconstruction if the shaded-surface display technique is used. To minimize some of the operator dependence, an objective means of setting the minimum segmentation level was used. However, some operator dependence persists in a few cases when a boundary line must be drawn to exclude contiguous calcified plaques. This form of operator dependence does not exist when maximum intensity projections (MIPs) are used to provide the 3D display, but such projections do result in some difficulty in "seeing through" calcified plaques (18). The most reproducible and accurate technique for the visualization of CT angiographic data remains to be established.

A section by section seed-growing process was used in this study to perform the segmentations and produce the surface-shaded displays. This technique as described herein requires about 30–45 minutes. Both multiplanar reconstruction (MPR) or MIPs would provide a more rapid view, but both have disadvantages and therefore were not used. Sagittal, coronal, or curved plane MPR allows visualization of the course of the carotid artery in many cases but can prove difficult to use in tortuous carotid arteries. In addition, use of this technique can easily result in a misdiagnosis in cases of occlusion of the internal carotid artery if the common carotid artery is aligned with a vertically oriented external carotid branch, which may then be misinterpreted as a patent internal carotid artery. MIP images preserve the CT attenuation information contained in the 2D sections but also require editing to evaluate the lumen in the vicinity of calcified plaques (18).

The stenoses in the current study

were assessed both subjectively with visual inspection and objectively with a pair of vernier calipers accurate to 0.01 mm. Despite the accuracy of the calipers, there was some difficulty in measuring the diameters of the lumen as shown on the films, since the CT angiograms represent a 3D projection of voxels measuring  $0.3 \times 0.3 \times 3.0$  mm that overlap in the longitudinal direction. Thus, close inspection of CT angiograms reveals a contour composed of pixels rather than a smooth contour, and it is difficult to decide exactly where to place the calipers along this bumpy contour for the purpose of measurement. Use of a jeweler's eyepiece as performed for angiography in the NASCET study would result in the same problem (1). Therefore, the stenoses were finally assigned to categories on the basis of the individual reader's combination of visual assessment and objective measurements.

CT angiography is a new technique, and further research will be necessary to assess its role in the evaluation of vascular disease. Certain advances can be expected in the near future as the capabilities of commercially available scanners and workstations are expanded. In particular, development of x-ray tubes that have a higher heat capacity and x-ray detectors with improved sensitivity may allow data acquisition for longer than 50 seconds, which would allow coverage of the same area with thinner sections, thus producing a better approximation of the true anatomy of the vessel with less partial volume effect. Increased use of nonionic contrast agents will reduce the risk of contrast material reaction and may decrease the occurrence of inadvertent swallowing by the patients as the contrast material is administered. In addition, development of subtraction techniques may allow the production of CT angiograms without the need for segmentation on each individual CT image by allowing subtraction of calcified plaques adjacent to the vessel lumen. Nevertheless, even in its current early state of development, CT angiography can provide most of the information needed before carotid endarterectomy and thus, in conjunction with US or MR angiography or both, may eventually reduce the need for preoperative carotid arteriography. ■

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## References

1. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effect of carotid endarterectomy in symptomatic patients with high grade carotid stenosis. *N Engl J Med* 1991; 325:445–453.
2. Ruckley CV, Wildsmith JAW. Carotid endarterectomy: future perspectives. *Eur J Vasc Surg* 1992; 6:229–231.
3. Masaryk TJ, Obuchowski N. Noninvasive carotid imaging: caveat emptor. *Radiology* 1993; 186:325–328.
4. Waugh JR, Sacharias N. Arteriographic complications in the DSA era. *Radiology* 1992; 182:243–246.
5. Grzyska U, Freitag J, Zeumer H. Selective cerebral intraarterial DSA: complication rate and control of risk factors. *Neuroradiology* 1990; 32:296–299.
6. Earnest F, Forbes G, Sandok BA, et al. Complications of cerebral angiography: prospective assessment of risk. *AJR* 1984; 142:247–253.
7. Steinke W, Kloetzsch C, Hennerici M. Carotid artery disease assessed by color Doppler flow imaging: correlation with standard Doppler sonography and angiography. *AJR* 1990; 154:1061–1068.
8. Moneta GL, Edwards JM, Chitwood RW, et al. Correlation of North American Symptomatic Carotid Endarterectomy Trial (NASCET) angiographic definition of 70%–99% internal carotid artery stenosis with duplex scanning. *J Vasc Surg* 1993; 17:152–159.
9. Huston J, Lewis BD, Wiebers DO, Meyer FB, Riederer SJ, Weaver AL. Carotid artery: prospective blinded comparison of two-dimensional time-of-flight MR angiography with conventional angiography and duplex US. *Radiology* 1993; 186:339–344.
10. Heiserman JE, Drayer BP, Fram EK, et al. Carotid artery stenosis: clinical efficacy of two-dimensional time-of-flight MR angiography. *Radiology* 1992; 182:761–768.
11. Polak JF, Kalina P, Donaldson MC, O'Leary DH, Whittemore AD, Mannick JA. Carotid endarterectomy: preoperative evaluation of candidates with combined Doppler sonography and MR angiography—work in progress. *Radiology* 1993; 186:333–338.
12. Hunink MGM, Polak JF, Barlan MM, O'Leary DH. Detection and quantification of carotid artery stenosis: efficacy of various Doppler velocity parameters. *AJR* 1993; 160:619–625.
13. Riles TS, Eidelman EM, Litt AW, Pinto RS, Oldford F, Thoe Schwartzberg GWS. Comparison of magnetic resonance angiography, conventional angiography, and duplex scanning. *Stroke* 1992; 23:341–346.
14. Erickson SJ, Mewissen MW, Foley WD, et al. Stenosis of the internal carotid artery: assessment using color Doppler imaging compared with angiography. *AJR* 1989; 152:1299–1305.
15. Heinz ER, Pizer SM, Fuchs H, et al. Examination of the extracranial carotid bifurcation by thin-section dynamic CT: direct visualization of intimal atheroma in man (part 1). *AJNR* 1984; 5:355–359.
16. Heinz ER, Fuchs J, Osborne D, et al. Examination of the extracranial carotid bifurcation by thin-section dynamic CT: direct visualization of intimal atheroma in man (part 2). *AJNR* 1984; 5:361–366.
17. Dillon EH, van Leeuwen MS, Fernandez MA, Mali WPTM. Spiral CT angiography: a new noninvasive vascular imaging technique. *AJR* 1993; 160:1273–1278.
18. Rubin GD, Dake MD, Napel SA, McDonnell CH, Jeffrey RB. Three-dimensional

- spiral CT angiography of the abdomen: initial clinical experience. *Radiology* 1993; 186:147-152.
19. Schwartz RB, Jones KM, Chernoff DM, et al. Common carotid artery bifurcation: evaluation with spiral CT—work in progress. *Radiology* 1992; 185:513-519.
  20. Vahlensieck M, Lang P, Chan WP, Grampp S, Genant HK. Three-dimensional reconstruction. I. Applications and techniques. *Eur Radiol* 1992; 2:503-507.
  21. Vahlensieck M, Lang P, Chan WP, Grampp S, Genant HK. Three-dimensional reconstruction. II. Optimization of segmentation and rendering of MRI. *Eur Radiol* 1992; 2:508-510.
  22. Fox AJ. How to measure carotid stenosis. *Radiology* 1993; 186:316-318.
  23. Kramer MS, Feinstein AR. Clinical biostatistics. LIV. The biostatistics of concordance. *Clin Pharmacol Ther* 1981; 29:111-123.
  24. Cebul RD, Paulus RA. The failure of intravenous digital subtraction angiography in replacing carotid arteriography. *Ann Intern Med* 1986; 104:572-574.
  25. Ammar AD, Turrentine MW, Farha SJ. The importance of arteriographic interpretation in occlusion or pseudo-occlusion of the carotid artery. *Surg Gynecol Obstet* 1988; 167:119-123.
  26. Riles TS, Posner MP, Cohen WS, Pinto R, Imparato AM, Baumann FG. The totally occluded internal carotid artery: preliminary observations using rapid sequential computerized tomographic scanning. *Arch Surg* 1982; 117:1185-1188.
  27. Geuder JW, Lamparello PJ, Riles TS, Giangola G, Imparato AM. Is duplex scanning sufficient evaluation before carotid endarterectomy? *J Vasc Surg* 1989; 9:193-201.
  28. Polak JF. Noninvasive carotid evaluation: carpe diem. *Radiology* 1993; 186:329-331.
  29. Bock RW, Gray-Weale AC, Mock PA, Robinson DA, Irwig L, Lusby RJ. The natural history of asymptomatic carotid artery disease. *J Vasc Surg* 1993; 17:160-171.
  30. Polak JF, Bajakian RL, O'Leary DH, Anderson MR, Donaldson MC, Jolesz FA. Detection of internal carotid stenosis: comparison of MR angiography, color Doppler sonography, and arteriography. *Radiology* 1992; 182:35-40.
  31. Anderson CM, Saloner D, Lee RE, et al. Assessment of carotid artery stenosis by MR angiography: comparison with x-ray angiography and color-coded Doppler ultrasound. *AJNR* 1992; 13:989-1003.
  32. Masaryk AM, Ross JS, DiCello MC, Modic MT, Paranandi L, Masaryk TJ. 3DFT MR angiography of the carotid bifurcation: potential and limitations as a screening examination. *Radiology* 1991; 179:797-804.
  33. Kido DK, Barsotti JB, Rice LZ, et al. Evaluation of the carotid artery bifurcation: comparison of magnetic resonance angiography and digital subtraction arch aortography. *Neuroradiology* 1991; 33:48-51.

## The U.S. F.D.A. Trial of the Wallstent

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Currently, there is only one stent that is available in the United States. To introduce a second stent requires an FDA trial which began in August 1991 and closed in January 1993. During that time 226 patients were entered. Indications for stent placement were restenosis within 90 days of an angioplasty procedure (17 patients) and iliac occlusions, (13 patients). Otherwise stents were placed for a failure of angioplasty. This was defined as a poor cosmetic result with a 30% or greater residual stenosis, a dissection longer than the dilated lesion, or a pressure gradient more than 5mm Hg in the iliac artery. The exclusion criteria were single or multiple lesions longer than 10cm in length, perforations or aneurysms caused by the angioplasty and patients unsuitable for anticoagulation. One hundred and forty patients had stents placed in the iliac system and 91 patients had femoral stents. Five patients had both femoral and iliac stents.

The 140 patients with iliac stents had 173 lesions stented in 165 limbs at risk. Eighty per cent of these were an SVS Grade I with a mean ABI of .63. In the iliac system, only 13 occlusions were stented, the remaining stents were placed in stenoses (145). The mean length of the occlusions treated were 6.6cm (range 4-13cm) and of the stenoses 3cm (range .1-18cm).

All patients having iliac stents were started on heparin 4 hours after the procedure and it was continued until the following morning. Patients were started on aspirin which continued indefinitely.

The primary angiographic success of stenting in the iliac system was 94.4%. The mean ABI rose from .64 before stenting to .85. Using the Rutherford scale, 78% of the patients were in the +2 and +3 categories and 95% were +1 or greater. At 6 months the mean ABI of the group with iliac stents was .92. By life table analysis, using a change in the Rutherford scale to represent a stent failure, there was an 85% patency at 6 months and a 77% patency at 1 year. Sixty percent of patients had follow up arteriograms at a mean of 6 months

which revealed a 93% patency. The results in the iliac system are little different from the Palmaz trial, both in demographics and results. (1)

Ninety-one patients had femoral Wallstents placed in 118 lesions in 96 limbs at risk.

All patients with femoral stents were heparinized with 5000 units during the implant and started an intravenous heparin 4 hours after the procedure. They were started concomitantly on coumadin and the heparin continued until such time as the prothrombin time was 1 1/2 times normal. This was usually 48 hrs. They continued coumadin for 6 months and took aspirin indefinitely.

In the femoral system 77 stenoses were stented as were 41 occlusions. The average length of the stenoses was 3.7cm, (range 1-30cm) and of the occlusions 9.1cm (range 1-30cm). Whereas the stenoses were predominantly short, occlusions were approximately evenly distributed between 0-5cm, 5-10cm and greater than 10cm in length. The latter were protocol violations. The majority of patients had claudication. Seventy-five per cent were in the Society of Vascular Surgery grade I and the mean ankle brachial index of the femoral group was .66 (SD  $\pm$  0.2).

As a result of stent placement, the stenoses increased in diameter from 1.5mm to 5.4mm and the occlusions increased to 5.45mm. The primary angiographic success was 96.4%. The mean ankle brachial index increased from .66 (SD  $\pm$  .2) to .96 (SD  $\pm$  .21). We also classified the primary clinical success using the Rutherford criteria. If one uses only +3 and +2, which represent very strict criteria, there was an 88% primary success rate. If one includes +1 then the primary success rate is 96%. (+3 implies normalization with an ABI greater than .9 and no symptoms. +2 is an increase in ABI of .1 and an increase in category. +1 represents either one of these criteria but not both). There was no significant difference in the results between stenoses and occlusions.

The initial ABI increase to .96 fell at six months to .92 for both stenoses and occlusions. The latest

follow up is a mean of 8 months. The mean ABI in stenoses is .88 and in occlusions .83. Using the Rutherford classification there is no statistical difference between stenoses and occlusions. At a mean of 8 months 72% of the occlusions are in +3 or +2 category and 79% +1 or greater. There has, therefore, been a clinical deterioration. Angiographic follow up have been achieved in 48 of the 91 patients. At 6 months, 11 patients have occluded or stenosed >50% for a six month angiographic patency of 77%. These results are beginning to mirror those previously described by Sapoval (2) and Zollikofer (3).

The complication rate is typical of multi center trials with a total of 16% but only 2.7% required surgery.

### **References**

1. Palmaz J.C., Laborde J.C., Rivera F.J., et al. Stenting of the iliac arteries with the Palmaz stent: Experience with a multicenter trial. *Cardiovasc Intervent. Radiology* 1992; 15: 291-7.
2. Saporal M.R., Long A.L., Raynaud A.C., et al. Femoropopliteal stent placement: long-term results. *Radiology* 1992; 184: 833-39.
3. Zollikofer C.L., Antonucci F., Markus P., et al. Arterial stent placement with use of the Wallstent: midterm results of clinical experience. *Radiology* 1991; 179: 449-56.

# Transjugular Intrahepatic Portosystemic Shunt: Early Experience with a Flexible Trocar/Catheter System

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**OBJECTIVE.** The purpose of this study was to determine the feasibility of using a flexible trocar/catheter system to create a transjugular intrahepatic portosystemic shunt (TIPS).

**SUBJECTS AND METHODS.** A flexible needle/trocar was used to connect the hepatic vein to the portal vein in 23 patients with portal hypertension and a history of bleeding gastroesophageal varices. Five patients had signs and symptoms of encephalopathy before shunting; in four, the disease was classified as Child's class C and in one, as Child's class B. Nine-millimeter self-expandable metallic stents were used in all patients.

**RESULTS.** The procedure was technically successful in all patients. The mean portosystemic pressure gradient decreased from 26 to 16 mm Hg (range, 7–23 mm Hg). Eight patients have died. One patient in a hepatic coma with hepatorenal failure survived percutaneous portosystemic shunting, but died 2 days after the TIPS procedure was done. After creation of the shunt, three other patients died of unrelated disease without recurrence of bleeding from gastroesophageal varices. Two patients died of massive bleeding from esophageal varices, one 24 hr after shunting and one of disseminated intravascular coagulation after a second attempt to establish a shunt. One patient died of sepsis associated with infected ascitic fluid 2 months after the TIPS procedure was done, and one died of unknown causes 4 months after the procedure was done. The mean follow-up time in the surviving 15 patients was 12 months. Seven patients had recurrence of gastrointestinal bleeding; the recurrence was due to a thrombosed shunt in two, to hepatic vein stenosis in four, and to a high portosystemic pressure gradient (22 mm Hg) after creation of the shunt in one. Hepatic vein stenosis was noted on portal venograms in another three otherwise asymptomatic patients. In four of five patients, encephalopathy was adequately controlled after shunting. The fifth patient with encephalopathy (Child's class C) and an additional patient in whom encephalopathy (Child's class C) developed after the TIPS procedure have had two additional hospital admissions because of difficulty in controlling the encephalopathy. The amount of ascitic fluid decreased or totally disappeared after treatment in all cases. No severe complications associated with creation of a TIPS were observed.

**CONCLUSION.** Use of a flexible trocar/catheter system to create a TIPS is a simple, safe, and moderately reliable means of decompressing the portal vein in patients with portal hypertension.

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Bleeding from esophageal varices in patients with portal hypertension continues to be a difficult therapeutic problem despite improvements in endoscopic sclerosing techniques. Creation of a surgical portosystemic shunt continues to be the most effective method of treatment. However, because of the high morbidity and mortality, efforts to develop other therapeutic approaches have continued [1, 2].

In 1969, Rösch et al. [3] showed the feasibility of creating percutaneous portocaval shunts in animals by connecting the inferior vena cava with the portal vein. During the following decade, a large number of experimental studies [4, 5] were per-

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formed to determine the feasibility of percutaneous creation of portosystemic shunts. In 1982, Colapinto and colleagues [6, 7] reported the first clinical experience in which percutaneous portosystemic shunts were created by dilating the track with angioplasty balloons. Long-term patency was, however, a problem, as the track tended to close in a relatively short time (Dotter CT et al., presented at the annual meeting of the Radiological Society of North America, November 1991).

With the development of vascular stents has come a sudden revival of interest in this technique [8–10]. Recently, Ring et al. [11], Richter et al. [12, 13], and Zemel et al. [14] reported their initial clinical results. In all previous studies, a rigid needle was used to create the transhepatic track. We report our preliminary results with percutaneous establishment of a portosystemic shunt in which a flexible needle was used to create the track.

### Subjects and Methods

From May to October 1991, 17 men and six women 39–70 years old (mean age, 54 years) had a transjugular intrahepatic portosystemic shunt (TIPS) created. All patients were admitted to the hospital because of life-threatening bleeding in the upper gastrointestinal tract due to gastroesophageal varices, and all had a history of liver cirrhosis (alcoholic in 16, idiopathic in three, postinfectious in four), portal hypertension, and gastroesophageal varices. The disease in these patients was classified according to clinical and laboratory findings as Child's C (10), B (six), and A (seven). The number of bleeding episodes varied from one to five (mean, 2.5). Twelve of the patients had had endoscopic sclerotherapy one to 12 times, and 60% of the patients had required one or more blood transfusions. Five of the patients had signs of encephalopathy before the TIPS procedure was done, as shown by the results of standard quantitative tests (Reitan test) and blood ammonia levels. All patients have been taking lactulose and following dietary restrictions to prevent encephalopathy. Creation of the shunt was an elective procedure in 21 patients and an emergency procedure in two, who were admitted with massive bleeding in the upper gastrointestinal tract and did not respond to conservative medical management, including fluid replacement, IV vasopressin, balloon tamponade, and sclerotherapy.

Before creating the shunt, we assessed the anatomy of the portal vein by using indirect portal venography with injection of the superior mesenteric and/or the splenic artery. The diagnostic study was usually performed 1–7 days before creation of the shunt to avoid potential contrast nephropathy from the large volume of contrast medium required for the diagnostic and therapeutic studies. If the patient was referred for an emergency TIPS procedure, the preoperative assessment and the shunting procedure were done at the same time. Hepatic venography was used, either simultaneously or subsequent to indirect portal venography, to assess the anatomy of individual veins and the relationship of the hepatic vein to the branches of the portal vein (Figs. 1A–1C). The wedge hepatic and inferior vena caval pressures were recorded in order to calculate the portosystemic pressure gradient before shunting. In some patients, a wedge hepatic venogram was also obtained to evaluate the anatomy of the portal vein further (Fig. 2). In addition, Doppler sonographic examination of the hepatic and portal veins was used to assess patency of the portal vein and the direction of flow in the portal branches.

The TIPS procedure was performed in the angiographic suite, and local anesthesia was used. No antibiotic prophylaxis was used, and no anticoagulants were given either during or after the procedure. In one patient with severe psychomotor activity, general anesthesia was used. IV midazolam (Versed, Roche Laboratories, Nutley, NJ) was used for sedation. Because dilatation of the transhepatic track caused severe pain, the track was infiltrated with a local anesthetic (lidocaine, 1.5 mg per kilogram of body weight) in the last 15 patients. Infiltration of the track was accomplished by using a catheter with multiple side holes (Cook Multipurpose, Cook, Inc., Bloomington, IN). The catheter was positioned over a guidewire at the level of the parenchymal track. A Y-connector was placed over the wire, and the anesthetic was injected through the side arm of the Y-connector. No adverse reactions were induced by the intraparenchymal injection of lidocaine.

Percutaneous access was obtained through the right internal jugular vein by using the Seldinger technique to place a 10-French sheath. A 7-French multipurpose catheter was used to selectively catheterize the right hepatic vein in 21 cases, the middle hepatic vein in one case, and the left hepatic vein in one case. With fluoroscopic guidance, a flexible needle/trocar was used to establish a transhepatic track from the hepatic vein into the portal vein. For the needle puncture, the needle/trocar system was taken to a medial

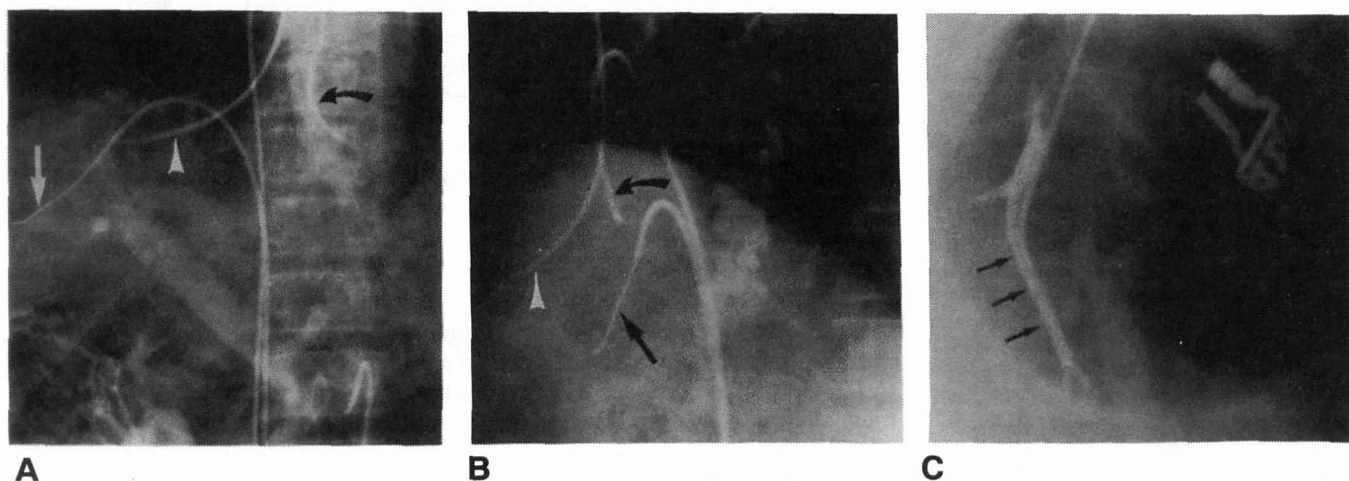


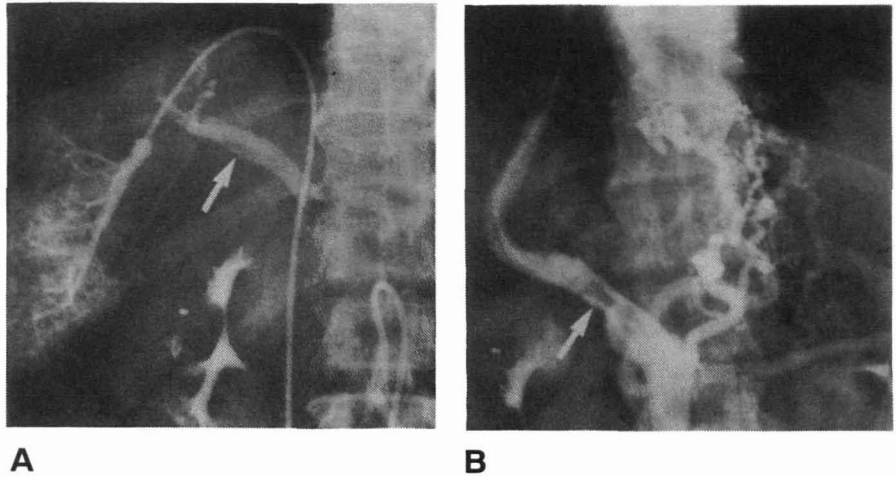
Fig. 1.—A and B, Anteroposterior (A) and lateral (B) indirect portal venograms (injection into superior mesenteric artery) show catheters placed in right (straight arrow), middle (arrowhead), and left (curved arrow) hepatic veins to assess veins' relationship to portal vein. Clearly, right hepatic vein provides shortest and easiest access.

C, Lateral venogram obtained after simultaneous injection in right hepatic vein (arrows) during indirect portal venography. Note relationship of right hepatic vein to portal vein.

TRANSJUGULAR INTRAHEPATIC PORTOSYSTEMIC SHUNT

Fig. 2.—A, Wedge hepatic venogram shows good opacification of portal vein, which has hepatofugal flow. Note filling defect within portal vein (arrow), suggesting partial thrombosis of portal vein.

B, Portal venogram obtained during transjugular intrahepatic portosystemic shunt procedure confirms presence of thrombus in portal vein (arrow) causing significant obstruction of flow, as shown by persistent opacification of gastric varices. Thrombus was fragmented and lysed in order to maintain patency of shunt.



position in the hepatic vein that had been entered in order to form a straighter track. The needle/trocar set consists of a flexible 0.038-in. (1-mm) trocar with a sharp tip sheathed in a 5-French Teflon catheter. The 5-French catheter fits through the lumen of a 10-French Teflon catheter and metal cannula assembly (Fig. 3). Aspiration of blood and subsequent injection of contrast medium confirmed that the portal vein had been entered. A stiff-type angled glidewire (Terumo, Medi-tech, Boston Scientific Corporation, Boston) was used to enter the portal vein in all cases. After passage of the glidewire into the portal system, in order to predilate the track, the locked assembly of the 10-French Teflon guiding catheter/metal cannula and the 5-French Teflon catheter was advanced into the portal vein until the tip of the 10-French guiding catheter was seen within the portal vein; the pressure in the portal vein was then measured. The 5-French catheter and the 10-French guiding catheter/cannula assembly was then removed, and a 9-mm (diameter) angioplasty balloon was introduced to fully dilate the track in the hepatic parenchyma. Pressures in the portal vein and in the hepatic vein were then recorded in order to reassess the portosystemic pressure gradient. Subsequently, the length of the track from the portal vein to the hepatic vein was measured, and a self-expandable Wallstent (Schneider, Zurich) of adequate length was placed in the track and dilated to 9 mm. More than one stent was required when the parenchymal track was long or when placement of the first stent was inaccurate. Pressures in the portal vein, hepatic vein, and inferior vena cava were

then recorded. Next, portal venograms were obtained by injecting contrast material into the superior mesenteric vein to assess the flow through the shunt and evaluate the persistent opacification of the gastroesophageal varices (Fig. 4).

In some cases, a pressure gradient greater than 15 mm Hg was present after placement of the stent. In none of the cases was further dilatation of the stent attempted (Fig. 5). The sheath was subsequently removed, and local pressure was applied at the level of the percutaneous puncture in the neck.

Follow-up of the patient included clinical and Doppler examinations, including baseline evaluation before discharge from the hospital and evaluation 1, 3, 6, and 12 months after discharge. An angiographic follow-up, including pressure measurements, at 6 months or sooner was planned as an outpatient procedure if the patient had any duplex sonographic or clinical evidence of malfunction of the shunt. Endoscopy to rule out the presence of potential bleeding lesions other than esophageal varices was performed only if the patient had evidence of gastrointestinal bleeding.

Results

Mean hospital stay after the TIPS procedure was 6.5 days (range, 2–30 days). The time required for the procedure was from 1.0 to 6.5 hr (mean, 3 hr). The time required and the number of needle passes needed to obtain access to the

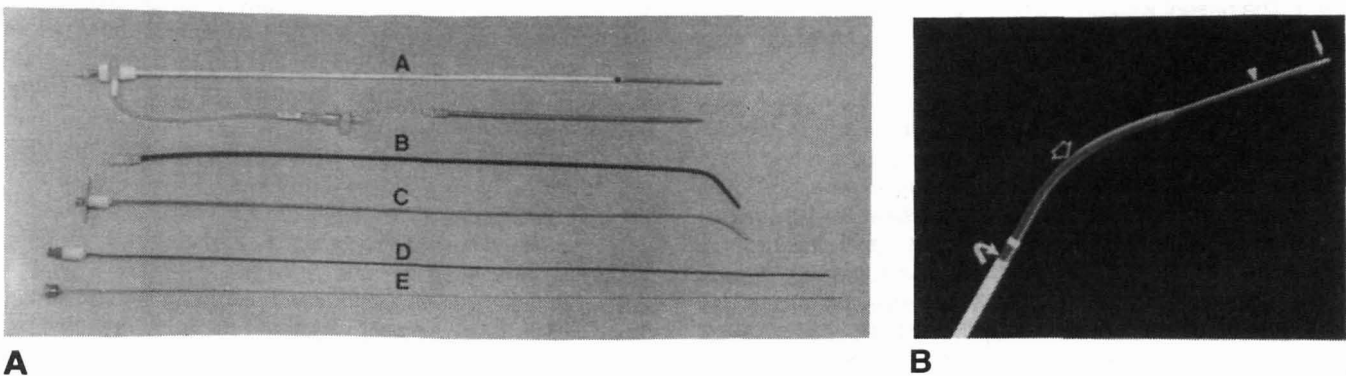


Fig. 3.—A, Photograph shows Rösch portacaval shunt set, RUPS-100 (Cook, Inc., Bloomington, IN). A = 10-French introducing sheath and 10-French vessel dilator, B = 10-French Teflon catheter, C = 14-gauge transjugular needle, D = 5-French Teflon catheter, E = 62.5-cm flexible-tip needle.

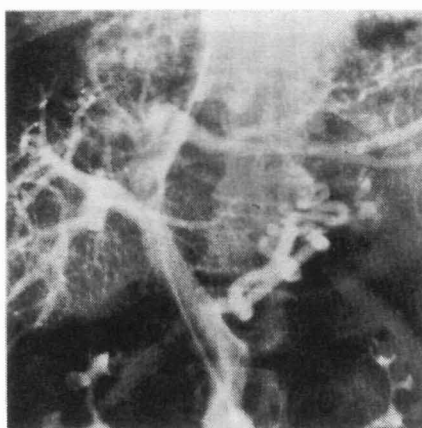
B, Magnified illustration shows assembled set: flexible-tip needle (straight arrow), 5-French Teflon catheter (arrowhead), 10-French Teflon catheter (open arrow), and 10-French introducer sheath (curved arrow).

(Figs. 3A and 3B courtesy of Cook, Inc., Bloomington, IN.)

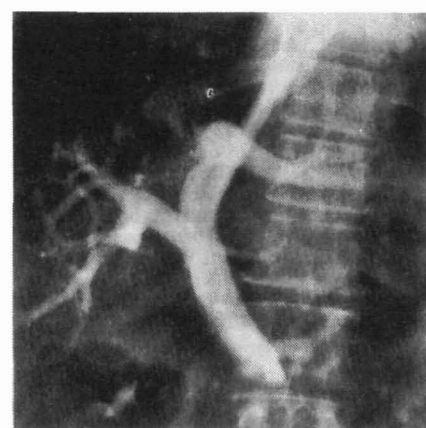




Fig. 4.—Portal venogram obtained after placement of Wallstent shows smooth angle of stent in track. Ends of stent are clearly seen within hepatic and portal veins. Note forward flow into branches of portal vein.



A



B

Fig. 5.—A, Portal venogram obtained before stent dilatation shows extensive gastric varices. B, Portal venogram obtained after stent dilatation shows lack of opacification of gastric varices and preservation of forward flow into branches of portal vein.

portal vein decreased as more experience was gained (learning curve). In our first cases, the mean number of needle passes was 16 (range, 3–25); this decreased to a mean of three (range, 1–7) in our last 10 cases.

Technical success was achieved in all patients. The portosystemic pressure gradient decreased from a mean of  $26 \pm 6$  mm Hg before establishment of the shunt to a mean of  $16 \pm 3$  mm Hg afterward. Angiograms showed antegrade flow through the stent in all patients; antegrade flow through branches of the portal vein was evident in 16 patients, and persistent opacification of the gastroesophageal varices was seen in 14 patients.

Of the 21 patients in whom the right hepatic vein was used, connection with the right portal vein branch was obtained in 12, with the left portal vein in six (at the level of bifurcation), and with the main portal vein at the level of the bifurcation in three. In the patients in whom the middle and left hepatic veins were used, the connection was made with the left portal vein.

The bile duct was punctured in four patients, the gallbladder in one, and a branch of the hepatic artery in one, but no clinical manifestations associated with these events were seen. The patient who had general anesthesia for the procedure was comatose for 2 days but recuperated fully and was discharged 15 days after the TIPS procedure.

The first eight patients in whom the intrahepatic track was not infiltrated with local anesthetic had moderate to severe pain compared with those patients in whom the intraparenchymal track was infiltrated with anesthetic, who had only minimal pain. The follow-up period has been 6–16 months (mean, 12 months). Eight patients died. One patient who was in a hepatic coma and had severe hepatorenal failure before the TIPS procedure died 48 hr after the procedure was done without having come out of the coma. Four patients died 2 months after the TIPS procedure: one of a massive cerebral vascular accident, one of an acute myocardial infarction, one of pneumonia, and one of sepsis due to infection of ascitic fluid. One patient died of massive

bleeding from esophageal varices 24 hr after the TIPS procedure. Another died of disseminated intravascular coagulation after successful establishment of a second TIPS, which was created because of thrombosis of the first one caused by bleeding varices. The eighth died of unknown causes 4 months after the TIPS procedure. Therefore, the 30-day mortality was 4%, and the 90-day mortality was 30%.

The remaining 15 patients have undergone angiographic evaluation, seven because of recurrent bleeding in the upper gastrointestinal tract and eight as a routine 6-month follow-up study. Seven patients had recurrent variceal bleeding. Two of these had a thrombosed shunt 2 and 3 months after the TIPS procedure was done, and creation of a second TIPS was required. Four others had stenosis of the hepatic vein 3–4 months after shunting, and balloon dilatation and stenting were necessary to relieve the obstruction. The seventh patient hemorrhaged because of a persistently elevated (22 mm Hg) portosystemic pressure gradient. These patients have remained asymptomatic since reintervention. Of the eight asymptomatic patients who had a routine angiographic-hemodynamic follow-up, three had stenosis of the hepatic vein but have remained asymptomatic, with patent shunts shown by Doppler sonography. After creation of the shunt, one of the patients who had encephalopathy before the procedure and an additional patient have had two subsequent hospital admissions because of encephalopathy. Ascites was present in 11 patients; it disappeared in four, and the amount of fluid decreased in seven after the TIPS procedure.

## Discussion

The prevalence of gastric and esophageal varices in patients with liver cirrhosis varies widely, from 14% to 77% [15–18], and the prevalence of bleeding from gastroesophageal varices in these patients varies from 13% to 70% [19, 20]. The risk of bleeding is said to be about 10% in patients with liver cirrhosis who have not had any previous episodes



of bleeding from gastroesophageal varices. In patients who have had a previous episode, the risk of rebleeding is 46% [21]. A higher prevalence of bleeding, from 65% to 81% [18, 22], has been reported in a selected group of patients with liver cirrhosis who were followed up prospectively.

Endoscopic sclerotherapy provides good palliation, but as the cause of portal hypertension is not eliminated, the prevalence of rebleeding is high [23–27].

Surgical creation of a portacaval shunt remains the best method for decompression of the portal system and therefore for preventing rebleeding from gastroesophageal varices [1, 20, 28]. Morbidity and mortality are less in patients in whom the surgery is elective, in patients with good residual liver function, and when small H-grafts are used to prevent liver failure and encephalopathy [29].

The concept of a percutaneous transhepatic portacaval shunt evolved from the late 1960s. Rösch et al. [3] described use of a needle for percutaneous transhepatic liver biopsies, and subsequently a percutaneous portacaval shunt in animals was reported. The needles initially described by Rösch et al. and subsequently modified by Colapinto et al. [6] have a rigid shaft and a sharp cutting tip in common. Although the rigidity of the needle allows better torque and, therefore, improved control during the transhepatic puncture, the risk of using such a sharp, rigid instrument, particularly if the operator is inexperienced, might outweigh the advantages of this design. A flexible, Teflon-sheathed stylet has recently been developed by Rösch. We have not had any difficulty in advancing the needle, even through fibrotic livers. Another advantage of this needle is that after passage of the guidewire, the assembly of a 5-French Teflon sheath–metal cannula and 10-French guiding catheter can be easily advanced over the guidewire into the portal vein to predilate the parenchymal track. After the metal cannula/5-French Teflon sheath is removed, an 8- or 9-mm angioplasty balloon can be easily advanced over the guidewire to dilate the parenchymal track.

In our series of 23 patients, our technical success was 100%, as compared with the 75% reported by others [12, 30]. Initial clinical success was achieved in all patients. Recurrent bleeding was seen in 35%, good control of ascites in 63%, and encephalopathy in 9%.

Richter et al. [12] recommended use of a transhepatic approach to the portal vein for guidance during the establishment of the connection between the hepatic vein and the portal vein. Unfortunately, in their series, one death was directly related to the percutaneous transhepatic track; massive bleeding occurred after loss of percutaneous transhepatic access despite the successful creation of a percutaneous portosystemic shunt. Sonographic guidance of the needle during the TIPS procedure has been used [12–14, 31].

We consider it essential to obtain a good quality angiographic evaluation of the patient's anatomy; this includes use of indirect portal venography, hepatic vein venography, wedge hepatic venography, and measurements of wedge hepatic vein pressure to assess the anatomy and the portosystemic pressure gradient. Anteroposterior and lateral angiograms should be obtained in order to evaluate the relationship of the hepatic veins to the portal vein. Additional information obtained from the initial angiographic evaluation

includes the presence of gastroesophageal varices, other sources of bleeding, the status of the hepatic artery circulation, and the direction of flow in the portal vein (hepatofugal, hepatopetal).

Probably the most important information obtained from the angiograms is data on the patency of the portal vein, because thrombosis of the portal vein (Fig. 2) can be present in some patients, particularly those who have had sclerotherapy several times [29]. Doppler sonography can also be used to assess the status of the portal vein and to determine if the flow in this vessel is hepatopetal or hepatofugal. If large collaterals are present, Doppler evaluation of the portal vein can be difficult, and then portal venography is the only accurate method of obtaining this information.

No deaths were directly associated with the TIPS procedure, even though one patient died 48 hr afterwards, because of terminal and irreversible hepatic and renal failure. Pain during dilatation of the transhepatic track was a common problem in the first eight cases. Subsequently, direct injection of local anesthetic into the transhepatic track eliminated this problem [32]. Interestingly, none of these patients had adverse reactions to the intraparenchymal injection of lidocaine. We do not recommend this approach in patients with a history of adverse reactions to this group of drugs. The long duration of the procedure is a relative problem, which has improved as we have become more proficient with the procedure.

The self-expandable Wallstent was used to maintain patency of the shunt. It was chosen because of its flexibility and self-expanding capabilities, which allow the stent to adapt itself to all types of tracks, preventing kinking in areas of angulation and migration of the stent (Fig. 4). Stent migration has been described with other types of stents [14].

The diameter of 9 mm for the stent was chosen on the basis of surgical experimental and clinical data. It has been reported that total decompression of the varices is not necessary to prevent rebleeding from gastroesophageal varices [33, 34]. The ultimate factors that cause variceal bleeding are not fully understood. The hemodynamics of the portal circulation are extremely complex and can be altered by a number of factors. Even though a larger shunt diameter is theoretically desirable to adequately decrease the portal pressure, it is well known that use of a larger shunt may increase the risk of encephalopathy [35]. The idea of creating a small-diameter shunt that will partially divert the portal flow was re-established by Rypins and Sarfeh [29]. The main purpose of this approach was to decrease the risk of encephalopathy by maintaining flow via the portal vein. However, it was found that, in some cases, the shunt would still cause complete diversion of portal blood flow.

Other researchers [34] recommend embolization of gastroesophageal varices after partial portal decompression via surgery. Fourteen of our patients had persistent opacification of their esophageal varices after creation of a TIPS. We decided that embolization was not necessary in one patient because the portosystemic pressure gradient decreased markedly after creation of the shunt, and we thought this was adequate to prevent rebleeding from the esophageal varices. A patient with rebleeding from gastroesophageal varices after a successful TIPS procedure could undergo dilatation of

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the shunt to decrease the portosystemic pressure gradient further and therefore stop the bleeding, or the varices could be easily embolized through the shunt [11, 13]. An increase in the diameter of the TIPS is possible only when the Palmaz stent is used, because the diameter of the Wallstent is fixed. Dilatation of the Wallstents with 10- or 12-mm angioplasty balloons may be attempted. In our experience, this has caused stent shortening and minimal increase in shunt diameter. As further clinical and hemodynamic data are gathered, guidelines for shunt dilation, variceal embolization, and creation of a second shunt will most likely be established. Long-term follow-up data will be critical in this regard.

Creation of a TIPS is a simple, safe, and reliable means of decompressing the portal vein in patients who have portal hypertension associated with liver cirrhosis. Clearly, more data are needed to prove the long-term efficacy of this procedure. Randomized trials comparing its efficacy with the efficacies of surgical shunts and sclerotherapy are warranted.

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## REFERENCES

- McDermott WV. Bleeding esophageal varices. *World J Surg* 1984;8:660-666
- Reynolds TB, Donovan AJ, Mikkelsen WP, Redeker AG, Turril FL, Weiner JM. Results of a 12-year randomized trial of portacaval shunt in patients with alcoholic liver disease and bleeding varices. *Gastroenterology* 1981;30:1005-1011
- Rösch J, Hanafee WN, Snow H. Transjugular portal venography and radiologic portacaval shunt: an experimental study. *Radiology* 1989;92:1112-1114
- Rösch J, Hanafee W, Snow H, Barenfus M, Gray R. Transjugular intrahepatic portacaval shunt: experimental work. *Am J Surg* 1971;121:488-492
- Gutiérrez OH, Burgener FA. Production of nonsurgical portosystemic venous shunts in dogs by transjugular approach. *Radiology* 1979;130:507-509
- Colapinto RF, Stronell RD, Birch SJ, et al. Creation of an intrahepatic portosystemic shunt with a Grüntzig balloon catheter. *Can Med Assoc J* 1982;126:267-268
- Colapinto RF, Stronell RD, Gildiner M, et al. Formation of intrahepatic portosystemic shunts using a balloon dilatation catheter: preliminary clinical experience. *AJR* 1983;140:709-714
- Palmaz JC, Sibbitt RR, Reuter SR, Garcia F, Tio FO. Expandable intrahepatic portacaval shunt stents: early experience in the dog. *AJR* 1985;145:821-825
- Palmaz JC, Garcia F, Sibbitt RR, et al. Expandable intrahepatic portacaval shunt stents in dogs with chronic portal hypertension. *AJR* 1986;147:1251-1254
- Rösch J, Uchida BT, Putnam JS, Buschman RW, Law RD, Hershey AL. Experimental intrahepatic portacaval anastomosis: use of expandable Gianturco stents. *Radiology* 1987;162:481-485
- Ring EJ, Lake JR, Roberts JP, et al. Percutaneous intrahepatic portosystemic shunts to control bleeding pre-transplantation. *Ann Intern Med* 1992;116:304-309
- Richter GM, Noeldge G, Palmaz JC, et al. Transjugular intrahepatic portacaval stent shunt: preliminary clinical results. *Radiology* 1990;174:1027-1030
- Richter GM, Noeldge G, Palmaz JC, Roessie M. The transjugular intrahepatic portosystemic stent-shunt (TIPSS): results of a pilot study. *Cardiovasc Intervent Radiol* 1990;13:200-207
- Zemel G, Katzen BT, Becker GJ, Benenati JF, Sallee DS. Percutaneous transjugular portosystemic shunt. *JAMA* 1991;266:390-393
- Garceau AJ, Boston Inter-Hospital Liver Group. The natural history of cirrhosis: II. The influence of alcohol and prior hepatitis on pathology and prognosis. *N Engl J Med* 1984;271:1173-1179
- Czaja AJ, Wolf AM, Summerskill WHJ. Development and early prognosis of esophageal varices in severe chronic active liver disease (CALD) treated with prednisone. *Gastroenterology* 1979;77:629-633
- Stone WD, Islam NRK, Paton A. The natural history of cirrhosis: experience with an unselected group of patients. *Q J Med* 1968;37:119-132
- Christensen E, Fauerholdt L, Schlichting P, et al. Aspects of the natural history of gastrointestinal bleeding in cirrhosis and the effect of prednisone. *Gastroenterology* 1981;81:944-952
- Baker LA, Smith C, Lieberman G. The natural history of esophageal varices: a study of 115 cirrhotic patients in whom varices were diagnosed prior to bleeding. *Am J Med* 1959;26:228-237
- Olsson R. The natural history of esophageal varices: a retrospective study of 224 cases with liver cirrhosis. *Digestion* 1972;6:65-74
- Resnick RH, Iber FL, Ishihara AM, et al. A controlled study of the therapeutic portacaval shunt. *Gastroenterology* 1974;67:843-857
- Jackson FC, Perrin EB, Felix WR, Smith AG. A clinical investigation of the portacaval shunt: V. Survival analysis of the therapeutic operation. *Ann Surg* 1971;174:672-701
- Warren WD. Control of variceal bleeding. *Am J Surg* 1983;145:8-16
- Cello JP, Grendell JH, Grass RA, Trunkey DD, Cobb EE, Heilbron DC. Endoscopic sclerotherapy versus portacaval shunt in patients with severe cirrhosis and variceal hemorrhage. *N Engl J Med* 1984;311:1589-1594
- Levine BA, Sirinek KR. The portacaval shunt: is it still indicated? *Surg Clin North Am* 1990;70:361-378
- Galampos JT. Portal hypertension. *Semin Liver Dis* 1985;5:277-290
- Rueff B, Prandi D, Degos F, et al. A controlled study of therapeutic portacaval shunt in alcoholic cirrhosis. *Lancet* 1976;1:655-659
- Planas R, Boix J, Broggi M, et al. Portacaval shunt versus endoscopic sclerotherapy in the elective treatment of variceal hemorrhage. *Gastroenterology* 1991;100:1078-1086
- Rypins EB, Sarfeh IJ. Small-diameter portacaval H-graft for variceal hemorrhage. *Surg Clin North Am* 1990;70:395-404
- Abecassis M, Gordon JD, Colapinto RF, et al. The transjugular intrahepatic portosystemic shunt (TIPS): an alternative for the management of life-threatening variceal hemorrhage. *Hepatology* 1985;5:1032A
- Richter GM, Noeldge G, Palmaz JC, et al. Transjugular intrahepatic portacaval stent shunt: preliminary clinical results. *Radiology* 1990;174:1027-1030
- Pulido-Duque JM, Reyes R, Górniz E, et al. Intraparenchymal anesthesia infiltration during transjugular intrahepatic portosystemic shunting. *Radiology* 1992;185:903-904
- Rypins EB, Sarfeh IJ. Partial portal decompression: two approaches to the management of portal hypertension. *Hepatology* 1990;12:370-372
- Coldwell DM, Moore ADA, Ben-Menachem Y, Johansen KH. Bleeding gastroesophageal varices: gastric vein embolization after partial portal decompression. *Radiology* 1991;178:249-251
- Johansen K. Partial portal decompression for variceal hemorrhage. *Am J Surg* 1989;157:479-482

## Long Term Results of Thrombolytic Therapy

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Long term results of thrombolytic therapy depend upon the type of conduit, the location, the need for PTA, the result of PTA, the status of the runoff vessels, and whether thrombolysis was successful. Thrombolysis yields the best long term results in native arteries (iliac > SFA), if lysis was complete, if the runoff is good, and in non-smokers. (Table 7).

The results of "late failure" are different than those following bypass surgery. Late failure after successful thrombolysis is due to stenosis in the previously long occlusion. As a result the patient has the same or less severe symptoms. In contrast, failure of a bypass graft is usually due to advancement of disease at the distal anastomosis and/or the distal vessels.

The graft usually thromboses and the thrombus extends into the next arterial segment(s). Thus, the patients has multilevel disease. This often gives limb threatening signs and symptoms. The incidence of amputation following late failure of a thrombosed bypass graft is higher than following reocclusion or stenosis developing within a previous occlusion. (Table 8,9).

### *Conclusion:*

Thrombolysis makes an enormous difference in the acutely ischemic limb by virtue of saving both lives and limbs. It makes a big difference in chronic occlusions improving the patency versus direct guide wire traversal and PTA as the initial step.

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TABLE 1. INITIAL LYSIS

ACUTE OCCLUSION	COMPLETE	PARTIAL	NONE
ILIAC	95	4	1
SFA	78	21	1
ABF	90	10	0
FERM-POP	75	25	0

TABLE 2. 1° PATENCY % (YEAR)

ACUTE OCCLUSION	0	1	2	3	4	5
ILIAC	98	98	95	90	85	80
SFA	88	85	80	76	69	65
ABF	95	90	75	70	60	45
FERM-POP	85	70	50	32	18	5

TABLE 3. PATENCY % (YEAR)

LYSIS	0	1	2	3	4	5
COMPLETE	100	100	92	80	72	65
PARTIAL	60	48	40	30	22	12
NONE	30	20	15	10	0	0

TABLE 4. INITIAL LYSIS

CHRONIC OCCLUSION	COMPLETE	PARTIAL	NONE
ILIAC	50	30	20
SFA	40	40	20
ABF	85	15	0
FERM-POP	85	5	10

**TABLE 5. 1° PATENCY % (YEAR)**

CHRONIC OCCLUSION	0	1	2	3	4	5
ILIAC	90	90	86	86	80	80
SFA	70	66	60	56	50	45
ABF	95	90	80	72	65	50
FERM-POP	85	80	70	35	0	0

**TABLE 6. PATENCY % (YEAR)**

RUNOFF VESSEL	0	1	2	3	4	5
4	80	78	74	70	63	57
3	78	74	70	69	60	59
2	75	70	66	60	50	43
1	78	70	56	45	20	0

**TABLE 7. BEST LONG TERM PATENCY (FAVOURABLE FACTORS)**

- Complete lysis
- Iliac Artery
- Non-smoker
- Two runoff arteries
- No residual stenosis

**TABLE 8.**

LATE FAILURE	AMPUTATIONS
LYSIS /PTA	RARE
BYPASS GRAFT	2-10%

**TABLE 9. LATE FAILURE**

CONDUIT	LIMB THREAT (%)
NATIVE ARTERY	RARE
BYPASS GRAFT	10-25%

**TABLE 10. BEST LONG TERM PATENCY**

- Native artery → Iliac > SFA
- Lysis recanalizes
- Large vessels
- Singles, short stenosis
- No residual stenosis

**TABLE 11. GRAFT PATENCY**

- Vein > PTFE
- Monitor with U.S.
- Preemptive PTA
- Atherectomy ?
- Stent ?

**TABLE 12. POST-LYSIS (ANTICOAGULANT?)**

- Ectasia
- Slow flow
- ↓ Cardiac output
- 1 vessel runoff

**TABLE 13. POST-LYSIS PATENCY (ANTICOAGULANT?)**

- Emboli
- PTFE graft
- No stenosis to PTA
- Unsuccessful PTA

**TABLE 14. BENEFITS OF LYSIS**

- Convert occlusion to stenosis
- Therefore, shorter lesion
- Minimizes risk of false channel

**TABLE 15. LONG TERM PATENCY (IMPACT OF LYSIS)**

- Stenosis > Occlusion
- Short > Long

**TABLE 16. PATENCY**

- Proxima > Distal
- Big > Small vessel
- Focal > Diffuse
- No residual stenosis
- Good flow/runoff
- Normal risk factors

**TABLE 17.**

- Thrombolysis converts occlusion to stenosis.
- Then patency follows stenosis patterns.

## Uses of balloon expandable stents in combination with PTFE

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### INTRODUCTION

**T**he use of stents in combination with PTFE offers the possibility to treat focal vascular lesions other than stenosis or occlusion in patients whom cannot otherwise undergo surgical repair for any reason. This is feasible in aneurysms, pseudoaneurysms and arteriovenous fistulas by excluding the abnormal vascular lumen with PTFE coaxially placed over stent as illustrated below.

radially expandible if a coaxial balloon is inflated within its lumen. This material may radially elongate with little elastic recoil several times its original diameter before rupture. This typically occurs after stretching 600-700 percent (7). The experience provided by bench and animal testing and the limited clinical endovascular use of this material suggests that radial expansion within the 350-450 percent of the original diameter may be adequate for practical use because the physical and mechanical characteristic of the material are not profoundly altered in this range. This ideal expansion ratio must be kept in mind during the selection of the diameter of the bypass material for a given target lesion.

We mount Impragraft (TM) on balloon expandable stents (Johnson and Johnson Interventional Systems) by placing the graft material coaxially over the stent and cutting it with a scalpel blade to match the length of the stent. We select the stent type trying to match its expansion range to that of the graft as suggested on the table above. The graft is affixed to the stent with 6-0 polypropylene suture with non-cutting needle. The sutures are placed on each quadrant of the stent circumference, 5 mm from the ends as depicted below.

Figure 1: Balloon inflation expands stent and PTFE material simultaneously occluding the lumen of the arteriovenous fistula (F).

It is understood that many of the materials needed to perform these procedures have not been designed for endovascular application and are not yet approved for clinical use as depicted here. However, because of their potential life-saving nature, it may be useful to review these techniques in some detail in case the need arises to use them in a critical situation.

### MATERIALS AND METHOD

**B**ecause of its plastic characteristics and the lack of a reinforcing layer Impragraft (TM) PTFE is

Figure 2: Suture placement to affix PTFE to a coaxial stent. The insert depicts a suture tied around one of the bridges between adjacent struts.

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GRAFT DIAMETER (MM)*	EXPANSION RANGE (MM)	STENT DIAMETER (MM)	SHEATH SIZE (ID. FRENCH)
3 TW	7-8	MEDIUM 2.5 **	10
3.5 TW	8-10	LARGE 3.3***	12
4 TW	10-15		14

Table 1.

\* Impragraft TW (thin wall)

\*\* (Johnson and Johnson Interventional System, P294)

\*\*\* (Johnson and Johnson Interventional System, P308)

The choice of the balloon catheter is critical because of two reasons: a) the need of maintaining the lowest possible profile to allow the smallest introducer sheath diameter. Table 1 indicates suggested sheath sizes for the different stent graft combinations. b) The other important factor is the length of the balloon in relationship with the length of the stent-graft assembly. If the balloon is longer than the stent-graft the balloon will adopt a dumbbell configuration at the beginning of the inflation causing retraction of the graft material and possible tear of the PTFE material at the site of the sutures. These tears may result in longitudinal splitting of the material at higher inflation pressures. If the balloon length is matched with the stent-graft the expansion begins at the center of the assembly preventing suture disruption. In selecting the appropriate balloon length it is important to remember that the nominal balloon length reflects the length of the cylindrical portion of the balloon and disregards the tapered portions at the ends. This indicates that for a 3 cm stent-graft the ideal balloon length should be 2 cm and have short tapered ends. For a stent-graft assembly composed of two 3 cm stents in tandem with a single piece of coaxially mounted graft the balloon should be 4 cm long. After complete expansion, the stent-graft may have tapered ends, requiring repositioning of the balloon and reexpansion of both ends to attain a cylindrical configuration as illustrated below.

Figure 3: A) Excessively long balloon with dumbbell expansion causing retraction of the graft over the stent with damage at the suture sites.  
 B) Shorter balloon expands the stent-graft from the center out. This produces tapered ends.  
 C) Balloon repositioning and inflation expands the tapered end.

To stabilize the stent-graft on the balloon during withdrawal of the sheath a "pusher" catheter can be coaxially mounted over the shaft of the balloon angioplasty catheter. This provides an edge on the back side of the balloon that prevents backward displacement. For balloon catheters with 5 French shafts a pusher can be fashioned out of an 8 French Van Andel catheter with the tapered tip cut off. Balloons catheter with a 6 French chaff accept 8 French Van Andel catheters.

Figure 4: Assembly of balloon angioplasty catheter, pusher catheter and stent-graft.

Stent-graft combinations larger than 15 mm in diameter require pre-expansion of the PTFE because larger balloons generate lower pressures. This determines a smaller margin of safety between the pressure needed to expand the stent-graft and the burst limit of the balloon. The pre-expanded graft must be folded around the stent and balloon catheter like an umbrella and loaded inside a delivery sheath with a hemostatic valve, as illustrated below. The assembly may be sterilized with ethylene oxide prior to use.

GRAFT DIAMETER (MM)*	EXPANSION RANGE (MM)	STENT DIAMETER (MM)	SHEATH SIZE (FRENCH, ID)
5 ST	15-20 (PRE-EXPANDED)	4.3	16
6 ST	20-28 (PRE-EXPANDED)		18

Table 2. (\* Impra TM) PTFE graft conduits of standard wall thickness and their expansion range, matching stent and introducer sheaths.

Figure 5: A large balloon angioplasty catheter is used to pre-expand PTFE (top). The resulting material is sutured to a bridge approximately in the middle of a stent. Therefore, half of the stent is not covered by PTFE (middle). The redundant PTFE is folded with two longitudinal folds running along the catheter shaft, inside of a sheath with a hemostatic valve (bottom).

Unlike stent-graft combinations of 15 mm in diameter or smaller, the large counterparts are delivered to the target site pre-loaded. This means that the sheath is introduced into the vessel with the balloon and the stent-graft inside its lumen. After reaching the desired position, the sheath is withdrawn and the balloon is inflated to deploy the stent. Because using graft material of larger diameter usually involves using greater lengths only part of the graft is supported by stent. The graft ends are applied against the arterial wall by the expanded stent which function as a friction seal. Only one stent is affixed to the PTFE material prior to placement. A distal stent may be placed immediately after the stent graft is deployed.

Figure 6: Large stent-graft is expanded with partial withdrawal of the delivery sheath (left). Complete withdrawal of the sheath allows the PTFE conduit to expand (center). Introduction of a second stent through the sheath is positioned and expanded at the distal end (right).

Table 2 shows suggested PTFE diameter and their expanded diameter range, corresponding stent and introducer sheaths. Balloon angioplasty catheters 15-30 mm in diameter are not available for arterial use. In exceptional situations, valvuloplasty and prostatic dilatation balloons could be used for these purposes. These balloons should have shafts no larger than 7 French to fit the suggested sheaths and overall lengths of 100 cm.

Intraarterial placement of the large vascular access sheaths, size 12 French and larger, requires direct exposure of the vessel. Occasionally, 12 French sheaths may be introduced percutaneously in patients with large, non-tortuous vessels because these procedures may be done, in general, with small doses of heparin. Introduction of 12 and 14 French sheaths into surgically exposed common femoral arteries should begin with a needle puncture followed by over-the-wire exchange. This technique preserves blood flow around the sheath in contrast with the use of arteriotomy and vessel loops. For sheath sizes 16 French and larger, arteriotomy and vessel loops may be used without disadvantage since these diameter sheaths usually interrupts blood flow.

## Endovascular Grafts

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In 1988 a project that had been abandoned by us in 1979 was restarted. The project was related to the transluminal treatment of aneurysms. Since then experiments were performed in 43 dogs. Most of them were used trying to develop a good model for testing several devices for aneurysmal exclusion. The last 8 experiments were performed in Buenos Aires and in San Antonio, Texas with the final design (two balloon expandable stents and a dacron graft overlapping the stent). The device was deployed in the infrarenal aorta in a retrograde fashion from the femoral artery under fluoroscopic guidance. The animal work proved the feasibility of the method.

On September 7, 1990 the first human application of the method was performed in Buenos Aires, the procedure was successful. The same day, the second procedure ended up as the first and only case considered as a primary failure. The stent was improperly deployed. The patient had to be operated upon, but had favorable outcome. As of October 1993. 45 procedures using the graft-stent combination have been performed. 38 aneurysms were treated, one of them was caused by an infrarenal aortic dissection. In addition seven patients with traumatic arteriovenous fistulas and false aneurysms were treated successfully.

Initial primary success was 80 %, mortality rate for the procedure was 5.2 % One patient developed a distal aortic dilatation 18 months after the initial treatment. The distal stents was placed too proximally to the aortic bifurcation leaving part of the aneurysm uncovered by the graft. Thus, this complication was considered to be caused by the mispositioning of the distal stents. Two additional patients in whom only a proximal stent was applied developed distal reflux after 8 and 24 months.

All of our patients were followed by physical examination, color duplex scan, CT scan and arteriography.

The longest follow-up is 36 months and the shortest in one month. With the results we have had until now. We can conclude that the procedure is applicable even in the very high-risk subset of patients we treated. Long-term outcome is still unknown.

## Co-knit Stent/Graft for Endovascular Treatment of Aortoiliac Aneurysms

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### OVERVIEW

**A** new concept for the treatment of abdominal aortic aneurysm has been employed in animal studies to exclude blood flow from artificial aortic aneurysms in mini-pigs. Two patients have been treated with the device.

### TECHNOLOGY AND APPLICATIONS

**T**he co-knit stent/graft, developed by Boston Scientific A/S, Jyllinge, Denmark, is a tubular, balloon-expandable stent/graft co-knit from intertwined strands of tantalum wire and texturized Dacron fiber. In theory, the Dacron fiber would encourage a controlled deposition of fibrin to cover the stent/graft complex, forming a new aortic flowpath and excluding blood flow from an aneurysm. Additionally, it was postulated that the fibrin seal, occurring from in vivo clotting processes, would encourage endothelialization of the stent/graft complex, forming a natural barrier to blood leakage.

### EXPERIMENTAL RESULTS

**E**ight Pitman-Moore mini-pigs underwent aortic resection and surgical insertion of an artificial Dacron aneurysm graft. The aneurysm (3cm diameter × 4cm length) was joined to the native aorta by a 10mm × 1cm tubular graft at the proximal and distal ends. After 15 day recovery period, a 10mm × 6cm co-knit stent was introduced into the false aneurysm graft of 7 mini-pigs. The 8th pig was retained as control. Post-insertion angiograms revealed immediate exclusion of the aneurysm from the new aortic bloodflow path. Autopsies performed at 24 hours (1 pig), 1 week (1 pig), 3 weeks (2 pigs) and 6, 9 and 12 weeks (1 pig each) confirmed fibrin deposition covering the entire co-knit stent/graft and excluding

the aneurysm. From 24 hours to 3 weeks, the fibrin deposition became progressively more organized, as shown in SEMs. At 6, 9 and 12 weeks, pathology studies confirmed the complete endothelialization of the 6cm length co-knit complex. Additionally, a neointima and a neomedial-like layer containing fibrous and collagenous structures was evidenced, suggesting the formation of a neo-vascular wall.

### INITIAL CONCLUSIONS

**T**he initial animal study of the tantalum/dacron co-knit stent suggest that the concept of utilizing the body's own fibrin to form a natural, permanent barrier to blood flow could be used for clinical treatment of abdominal aortic aneurysms. The first clinical placement of the device in an iliac aneurysm resulted in aneurysm exclusion and continued patency at 5 month follow-up. A patient presenting an infra-renal aortic aneurysm was successfully treated with the device. Other clinical placements with long-term follow-up studies, including intravascular ultrasound to assess neovascular wall formation, are planned.

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## Arterial Volume Flow by a New Ultrasonic Measurement Technique

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The direct measurement of the velocity profile of blood flowing in a vessel yields a volume flow estimate that is more accurate than single-point Doppler Ultrasound. A volume flow estimate is made by integrating the velocity profile measured along a diameter. The many velocity measurements made contribute to higher precision in the integrated velocity estimate. Also, the velocity profile furnishes the functional diameter of the vessel at many points through the cardiac cycle. This algorithm, as implemented in the Philips CVI™ system, was tested theoretically by numerical modelling, and experimentally with a flow simulator. The effect of beamwidth, vessel size, and measurement position misalignment on the volume flow estimate were studied. Experimental and theoretical results agreed well and showed that the flow estimation algorithm can produce precise and accurate ( $\pm 15\%$ ) volume flow estimates. Beamwidths of 1.0 to 1.5 mm are a good match to axial resolution and yield accurate volume flow estimates in vessels over 3 mm in diameter. Larger beamwidths give lower volume flow estimates, but are not as sensitive to misalignment.

To validate the clinical use of this new technique, twenty three investigators from fourteen institutions in Europe and the United States of America have continuing experiments and clinical studies. Over ten papers have been published or presented since the effort began two years ago. In vitro experiments with computer controlled flow test objects and string test objects have been used. Flow meters, timed fluid volume collection and accurate motion sensing have been used to establish the accuracy, and correlation of velocity and flow volume in the CVI™ method. Multiple studies have shown that

when a velocity at a site is chosen from within a CVI™ velocity image and compared to the spectral Doppler estimate of velocity for that same site, excellent agreement is found. This new measurement technique therefore extends a small region velocity estimate to a quantitative description of velocity over a wide area.

Since pulsed Doppler has not been found clinically reliable as a method of volume flow measurement, all comparisons of the CVI™ volume flow measurements were made to accepted flow meter and timed fluid collection techniques. Again, excellent results were found, which provided the basis for animal model and human in vivo studies. Animal models have shown excellent agreement between CVI™ and flow meter or timed fluid collection techniques.

Human studies of normals have been used to identify the range of blood velocity and volume flow rates for various anatomic sites and population variables (sex, age, etc). Initial studies of flow affected by pathology or treatment are promising and will continue to provide more information and understanding in the future. Within the constraints of the methodology, a new ultrasonic technique is available to accurately identify velocity and volume flow rates for diagnostic, therapeutic, and patient monitoring tasks.

## TIPSS

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### STENT TYPES FOR TIPSS

To date there is a variety of vascular stents on the market which might offer promise for its use in transjugular intrahepatic portosystemic stent shunt (TIPSS). Vascular stents are implanted as permanent devices. Choice of material and design properties, therefore, must be directed towards a maximum of intracorporeal durability. In this regard, factors such as metal corrosion, structural weak points or histologic inertness are of utmost importance. In the long history of medical applications stainless steel 316 L has shown an excellent biocompatibility without any known corrosion problem. By contrast, Tantalum alloys - the material used for the Strecker stent- are prone to relatively rapid intravascular corrosion. In addition to possible corrosion, specific design problems -or weak points- may add to failure in longterm durability. Therefore, in the design of a permanent vessel implant crossing points with metal riding over metal ("fretting corrosion") should be decreased to a minimum.

For the transjugular intrahepatic portosystemic stent shunt (TIPSS) two different stent types are widely used: the balloon expandable PALMAZ iliac stent (Johnson & Johnson Interventional Systems, Warren, NJ) and the selfexpandable WALLSTENT (Schneider, Zurich, Switzerland). The Palmaz stent consist of a single segment tubular stainless steel mesh, surgical grade 316 L with a wall thickness of  $150\ \mu\text{m}$  ( $=0.004''$ ) and an unexpanded length of 30 mm. Around its circumference, each stent has 4 staggered, offset slots that are 4.5 mm long. Its diameter in the non expanded state measures 3.1 mm. Radial compression of the stent over a balloon catheter decreases this diameter by closure of the slots and, thus, firmly attaches the crimped stent to the catheter (7F shaft). The Palmaz stent is a typical malleable stent type that, when expanded, employs plastic forces to withstand hoop stress. As it has no expansible force by itself, expansion of the coaxial balloon used for stent delivery will impose a force beyond the elastic limit of the metal mesh and, thus, radially open the slots to quadrangles. It is

because of this mode of actin that such a stent type has a wide range of possible luminal diameter which is solely dependant on the balloon size used for delivery. The Palmaz type is the only stent that allows such a variability. The size of the balloon applied for stent deployment determines the final expansion ratio. By contrast, the Wallstent (Schneider) is a selfexpandable vascular endoprosthesis the design of which closely resembles a "chinese finger". The basic metallic material from which the stent is manufactured is a commercial secret. Because of the spring load of the design a special delivery system is provided which consist of a 7F catheter at the distal end of which the stent is both hold in place and flattened down by a plastic membrane that covers the whole set. A side-arm is connected to the narrow interspace between shaft and membrane. As soon as contrast medium is injected at a limited pressure of 4 atm max. through this side-arm friction between membrane and catheter shaft is minimized and the membrane can be pulled back. This pulling or rolling back of the membrane will set free the stent from distal to proximal as it pops up from its springload. The stent is very flexible will open even when heavily bended or curved without kinking.

In addition to the previously mentioned two other stent types the Gianturco-Stent (Cook) in the modified version by Röach (28) has been advocated for TIPSS. This stent is also categorized as self-expanding and is made from spring load stainless steel wire which is bent in a zigzag pattern and soldered at the ends to form a closed circle. The thickness of the wire, its inherent spring load, the number of the turns and the angulation of the turns determine the final diameter and the resistance to hoop stress. Usually, a single stent cylinder is 2.5 cm in length. For long segment stenting there are double or triple cylinders available. For deployment the sent can be pushed trough a delivery cartridge in a compressed state. A specific feature of this stent is its availability in large diameters particularly useful in venous stenting.

## HISTORIC REMARKS

In 1969 Rösch et al, published initial results of a mainly nonsurgically created portosystemic connection between the inferior vena cava and the portal circulation in a canine model (27). During the following decade similar models have been described in literature none of which, however, achieved acceptable clinical relevance for potential treatment of portal hypertension (2,12,21). The first realization of nonsurgical portosystemic shunting in man was reported by Colapinto in 1982 (4). By means of longterm balloon dilatation he tried to keep patent a connection between liver veins and the portal circulation previously established by transjugular puncture. Patency, however, was very poor. In larger series, the concept proved unsatisfactory due to high rates of tract occlusion and rebleeding (1,10). A substantial improvement of the concept was contributed by the development of balloon expandable metallic endoprostheses, particularly, the so-called Palmaz stent (Johnson & Johnson, Warren, New Jersey, USA): in experimental dog models in 1985 and in 1987, respectively, Palmaz demonstrated sufficient long-term patency of portosystemic shunts bridging the inferior vena cava and the portal circulation by scaffolding the intrahepatic tract with his stent (17,18). Rösch, too, tried to scaffold artificial intrahepatic portosystemic tracts in a swine model with a different stent type: he used the modified Gianturco stent, a self expandable metallic endoprosthesis (28).

Longterm patency rates, however, were less favorable than in Palmaz series due to shunt occlusion from parenchymal overgrowth. The question whether stent design problems or a peculiar response of swine liver were the reason of the unfavorable results could not be settled (28). Palmaz achieved 50% long-term patency in a canine model without portal hypertension (17) and 100% patency when the animals had portal hypertension (18). From his experience it was justified to seek for a reproducible and reliable technique of transjugular intrahepatic portosystemic stent shunting to treat patients with severe portal hypertension. In 1987 we submitted to the Institutional Revision Board of the University Hospital of Freiburg ("Ethink-Komitee") a pilot study application for first clinical use of TIPSS. The first successful procedure was accomplished in January 1988 (23). From then on, we developed and improved the technique of TIPSS from the experience gathered in more than 100 patients to

date (24-26). Initially, the study protocol restricted the use of the procedure to patients in whom conservative management with repeat sclerotherapy and vasoactive drugs failed to control bleeding and who were at prohibitive risk for surgical shunts. Subsequent accumulation of technical and clinical experience encouraged a wider and more deliberate use of TIPSS to include younger patients and patients at milder stages of liver disease.

## INDICATIONS

To date, 6 years have elapsed since the first successful application of the TIPSS concept in man. It seems uncritical to us to accept such a time span as sufficient to define "absolute" indications. We rather propose to consider indications for the procedure as relative in respect to well established methods for the treatment of portal hypertension. Before TIPSS can be indicated a statement from a well experienced endoscopist as to the state and stages of oesophageal and gastric varices is mandatory. In particular, a reasonable prospect of success by continuation for sclerotherapy should be ruled. This following list of indications seems to be appropriate:

- chronically recurring variceal bleeding despite continuing sclerotherapy.
- recurring variceal bleeding and severe ulcerative or erosive disease from repeat sclerotherapy.
- repeat bleeding episodes from major gastric varices inaccessible for sclerotherapy.
- recurring variceal bleeding from occluded surgical shunts.

When patients are admitted for TIPSS because of unacceptable risk for surgery the patient presentation spectrum changes to a rather negative case selection profit. For this particular subset of patients no therapeutic choice other than TIPSS exists. For patients fit for surgery in stage A or B of the Child's and Turcotte's classification of liver disease (3) the classical therapeutic option has been the distal splenorenal shunt (DSRS, or Warren-shunt). In such patients TIPSS seems to be competitive with surgery (34).

It is our belief that more time and experience on the basis of a much wider clinical background will be necessary to enable stage related and risk stratified analysis of patency rates, risk of encephalopathy, and both of morbidity and mortality of the TIPSS method in comparison with

shunt surgery.

However, to date it seems safe to state that TIPSS can be recommended for patients already enrolled in a liver transplantation waiting list who are threatened by a high bleeding risk while waiting for the life-saving organ. In these patients the performance of a TIPSS procedure leaves the main vascular structures untouched in contradistinction to shunt surgery. Previous shunt surgeries well known to increase surgical difficulties and morbidity upon liver transplantation.

A still unclear issue is whether TIPSS should be offered to patients with untractable ascites. Our longterm results in this particular subset of patients, still, is too limited to allow more than preliminary conclusions. From our experience it appears that patients in whom ascites develops very rapidly and ascites is significantly associated with a high portosystemic pressure gradient TIPSS may be very beneficial. By contrast, in patients with a long history of liver cirrhosis bordering on liver failure, and small organ size without a very pronounced pressure component, TIPSS may lead to rapid liver failure from deprivation of portal blood nutrition. Therefore, it seems very critical to us to measure the venous occlusion pressure, a forehand, in patients referred to TIPSS for treating intractable ascites. This may help to sort out patients that will not benefit from the procedure and should either be transferred to a liver transplantation program or be left alone.

### CONTRAINDICATIONS

The contraindications for TIPSS are not necessarily the same as those of shunt surgery because the radiological intervention is much less invasive. However, there are four absolute contraindications to be mentioned:

- right heart failure or other cardiopulmonary factors contributing to substantial elevation of right ventricular pressure (chronic or acute left heart failure, or pulmonale, etc.)
- sepsis. Special attention must be paid to pulmonary infection as this may easily develop from aspiration pneumonia during bleeding episodes and sclerotherapy. Also, infected ascitic fluid can be a pertinent feature in long standing liver cirrhosis
- significant acute liver failure not attributable to active bleeding.

- presence of hepatocellular carcinoma (HCC) engorging or infiltrating the vascular structures or the parenchyma of the liver neighboring the proposed shunt tract.

A relative contraindication is portal vein occlusion. With wider clinical application of the TIPSS concept more experience with this problem has emerged. In a recent update the San Francisco group reported a 70% initial success rate in such patients (E. Ring, personal communication). Another relative contraindication is peripheral small HCC in patients unfit for surgical resection.

### TECHNIQUE

There were several complications during the first part of our learning curve with the procedure. Many of these early problems have been published in detail (11-14). To avoid unnecessary overlap we like to concentrate on our up-to-date methodology which reduced the procedural time from 7 hours to a little more than 2 hours in the majority of our cases.

#### *Patient Preparation:*

A variety of appropriate clinical and laboratory tests should be performed before TIPSS to allow for application of the Child's and Turcotte classification of liver cirrhosis (3) in the modified version according to Conn (7). Subclinical hepatic encephalopathy can be diagnosed applying the Number Connection Test according to Conn (6) in an age correlated version by Schomerus (41). Imaging procedures are performed to rule out malignant disease, infection, portal vein occlusion and major anatomic abnormalities preventing successful performance of the procedure. Hence, abdominal ultrasound including Doppler sonography of the portal vein, chest radiography and abdominal angiography are required. If hepatocellular carcinoma (HCC) or portal vein occlusion are suspected additional abdominal CT studies should be performed. If the hepatic vein are well seen on CT a good appreciation of the anatomic relationship between them and the portal vein is possible.

It is important to try to improve the clinical state of each individual patient before TIPSS. The measures taken to achieve this include correction of hematocrit, protein and coagulation deficits, bowel cleaning and prophylactic broad spectrum antibiotic therapy. Immediately before the procedure 3-6



blood units should be cross-matched.

*Anatomic Situation:*

We consider the creation of a wide, central and more or less straight intrahepatic shunt tract as crucial for early and longterm success. In view of this consideration, the puncture tract should bridge the proximal part of either the right or the middle hepatic vein with the upper wall of either one of the main portal vein branches. Therefore, exact knowledge of the anatomic relationship between the portal bifurcation and the hepatic vein radicles is mandatory. In most patients the portal bifurcation is located anterior to the main stems of the hepatic veins, but there are several anatomic variants. Of particular interest is the finding of the origin and termination of the liver capsule in the course of the portal vein.

Any inadvertent puncture of the extracapsular part of the portal vein carries a high risk of life-threatening intraabdominal bleeding. Both, CT and ultrasound scanning are extremely helpful in identifying and defining the individual anatomic situation in each patient.

*Transjugular Access:*

For sterility reasons the procedure is performed in the angiography suite on the already completely draped patient after careful skin preparation. After sonographic documentation of the course of the right internal jugular vein and skin anaesthesia a beveled 18 gauge cannula with a 5 ml syringe connected to the hub and filled with normal saline is introduced 4-5 cm cranial to the upper aspect of the clavicle at a very shallow angle to provide easy access for the large bore instruments to follow. When blood is drawn easily a 0,035" J-guide wire is inserted and manipulated down to the inferior vena cava under fluoroscopic control to allow insertion of a long 9F sheath (Terumo). If the wire does not advance to the inferior vena cava a selective catheter is used.

*3-D Orientation:*

Most of the difficulties associated with punctures aimed centrally and medially towards the portal bifurcation have been eliminated by guiding the puncture simultaneously by fluoroscopy and ultrasound. However, it has to be understood that the sonographic appearance of cirrhotic liver is significantly changed and vascular structures are much less visible as compared to normal liver.

Hence, only state-of-the-art ultrasound technology displays the relevant structures, particularly, when equipped with color Doppler mode.

Sonographic guidance from an intercostal lateral view visualizes both the bifurcation of the portal vein and the hepatic veins. By directing the beam anteriorly or posteriorly the length and angulation of the shortest shunt tract between both venous systems may be easily determined. In connection with CT studies this also helps to determine the orientation of the portal bifurcation in relation to the hepatic veins.

*Puncture and Shunt Tract Creation:*

The first step after the establishment of transjugular access is catheterization of the hepatic veins. Usually, we use a 5F multipurpose catheter (Terumo) and start with the right hepatic vein which in the vast majority of the cases will be the one to use. Angiographic documentation with the catheter tip positioned distally in the vein demonstrates the morphologic situation. Particular attention is drawn to the size of the vein at its inflow into the inferior vena cava. The diameter should be at least 10 mm. In some instances there is retrograde sinusoidal flow which quickly identifies the portal bifurcation. After this, a superstiff wire is inserted which helps to visualize the vein by ultrasound. Then, an 8F guiding catheter from the TIPSS set designed by us (Angiomed) is introduced over the wire. With the set comes a puncture device that has been specially designed for the TIPSS procedure. It features a blunt 50 cm long cannula that has a 15 G shaft tapered to 17.6 G at the tip. The cannula is pre-bent to 30° and is stiff for sufficient torque control. To function as a needle an inner mandril made from nitinol is inserted into the cannula which is extremely sharp and highly flexible with a smooth transition from its tip to the cannula. The cannula can be inserted in the liver over the wire already in place or by using a blunt obturator which is also part of the set and also made from nitinol. In most of the cases we change the curve of the needle to adopt the angle at which the hepatic vein enters the inferior vena cava. If this happens to be a near 90° angulation heavy manual bending is required. Then, the cannula can only be inserted over a superstiff wire positioned with its stiff part well in the peripheral hepatic vein covered by the guiding catheter in order to minimize the risk of catheter perforation.

With the aid of ultrasound and fluoroscopy the

optimal puncture site is chosen. Usually, ultrasound helps to identify a position in the hepatic vein from which a short and straight course to the portal bifurcation is achieved. Then the cannula is securely held in place and the sharp mandril inserted and locked to the hub of the cannula. The needle is rotated according to the pre-determined puncture direction.

Again, applying simultaneous ultrasound and fluoroscopy the intrahepatic advancement of needle is conveniently monitored. As soon as the needle reaches the portal vein wall significant resistance is felt which has to be overcome with some pushing force. Correct portal access is confirmed as soon as the sharp mandril is removed and blood returns upon aspiration. Then contrast medium injection should confirm the situation. During this highly critical step the needle must be perfectly held in place. With the needle tip pointing medially a superstiff 0.035" wire is passed which, in the majority of the cases travels down to the superior mesenteric vein. Over both the wire and the cannula the guiding catheter is pushed into the portal vein which, usually, requires considerable force. After this stable portal access is achieved. However, in some cases the wire lodges in peripheral portal branches instead of going centrally. Then a selective catheter has to be used to direct the wire centrally. In these cases a Terumo wire is more useful than a superstiff wire.

Once stable portal access is achieved the shunt tract is pre-dilatated to 8 mm utilizing low profile, 5F angioplasty catheters. Typically upon inflation a balloon waist forms at the entry site of the puncture tract into the portal wall. In most cases several minutes of inflation are needed for complete balloon expansion and effacement of the waist.

#### *Stenting and the Hemodynamic Concept:*

It is generally accepted that an absolute portal pressure of higher than 20 mm Hg or a portosystemic gradient of greater than 15mm Hg increase the risk of variceal bleeding. This holds true both for spontaneously occurring bleeding episodes and for reoccurrence of bleeding after surgical shunts. Conversely, a low portosystemic gradient accompanied by high volume shunt flow may significantly increase the risk of hepatic encephalopathy. Therefore, we prefer to lower the portosystemic gradient down to approximately 12 mm Hg. Careful measurement of the portal pressure conditions is a crucial point in the procedure. Typically, before pre-dilation of the

shunt tract, as described above, we monitor the portosystemic gradient. Then, the tract is completely scaffolded from its entry site at the portal system up into the liver vein using a technique identical to arterial stenting. Stenting is performed through a special 35 cm long sheath which is also part of the TIPSS set. We use as many Palmaz stents as necessary with an overlap of several millimeters and an initial diameter of 8 mm. The flow conditions in the hepatic vein are very important. This "outflow" part must be wide enough to accept the shunt flow. Therefore, it may become appropriate to stent the whole of the hepatic vein if it turns out to be too small. Upon completion of the stent shunt with an initial diameter of 8 mm the portosystemic gradient is monitored again. Unless substantial portal decompression within a range of 10-13 mm Hg is measured stepwise increase of the shunt diameter is performed by applying balloon dilation in 1 mm increments until the desired pressure level is reached. At any event, the stented segment within the liver vein is flared to a trumpet shape by dilating it with a 12 mm balloon. This is done to allow easier follow-up catheterization.

#### *Variceal Embolization:*

The need for embolization of varices as an adjunct to a successful TIPSS procedure in an acutely bleeding patient is a major concern and is a somewhat unclear issue. In our opinion, simultaneous embolotherapy (by whatever means) in addition to creation of a well functioning shunt can potentially help to speed patient recovery. Usually, acutely bleeding patients in whom medical treatment failed to control variceal hemorrhage present in a poor or critical clinical state resulting from substantial intestinal blood loss, coagulopathy, possibly worsened by mass transfusion, and hepatic encephalopathy. Failed vasopressor intravenous therapy and prolonged inflation times of gastroesophageal balloons add to the life threatening situation.

#### *Patient Monitoring during TIPSS:*

Significant pain usually accompanies dilation of the shunt tract and stent deployment which should be alleviated by appropriate intravenous analgesics under oxygen saturation monitoring. Inadvertent catheter or wire passage into the right ventricle frequently happens at a variety of stages during the procedure possibly activating severe arrhythmia. Therefore, constant ECG monitoring is required with antiarrhythmic medication ready at hand.

***Specific Medication for TIPSS and Postprocedural Care:***

**In elective procedures** 3-6 units of whole blood are prepared depending on coagulation status, hemoglobin level and total blood count. In cases of significant coagulation abnormalities (prolongation of the PTT by more than 30% and/or the decrease of the PT level to below 50% of the normal) 4-8 units of fresh frozen blood are ordered to allow instantaneous infusion if necessary to treat any bleeding complication (see also below). Broad spectrum antibiotic therapy is started on the day of the procedure and continued for two more days. Immediately prior to stent deployment heparin is given according to the coagulation status of the patient. Patients with PT levels of >60% of the control receive 5000 units, patients with PT < 60% and >45% receive 2500 units. Therapeutic heparinization is maintained for two more days in patients with more or less normal coagulation. In these antiplatelet medication is established for 3 months.

**In emergency shunting** the full range of conservative methods to control variceal hemorrhage should have been established and functioning before (occlusion tubes, beta blockers and vasoconstrictor infusions). General anaesthesia is required in patients in danger of aspiration or those with severe encephalopathy with failure to cooperate during the procedure. In acutely bleeding patients broad spectrum antibiotic therapy should have been already initiated at least one day before the procedure as well as medication and measurements for mechanical and biologic clearing of the bowel from blood and bacteria.

**After successful completion** of TIPSS the patients are kept in the intensive care unit until a stable clinical situation is guaranteed without signs of gastrointestinal hemorrhage, exclusion of pulmonary infection and renal and hepatic malfunction. The length of hospital stay depends on the general clinical presentation status.

**In the early postprocedural phase** endoscopic reevaluation of variceal filling is confirmed to obtain baseline information as to visible changes in comparison with the situation before TIPSS. If no variceal reduction is observed and no signs of hepatic encephalopathy are present the patient may be re-scheduled for shunt redilatation which is easily performed by simple re-expansion of the stent shunt with bigger balloons.

**Normal nutrition** is allowed for patients with near normal liver function. In patients with abnormal liver function are instructed a low protein diet is instituted.

**Direct portography** is performed as part of our routine follow-up examination program 3 months, 6 months and 12 months after TIPSS to examine the healing pattern of the stent shunt and observe possible onset of intimal hyperplasia (see below).

## **RESULTS**

### ***Changes in Study Population and Success Rates***

To date, more than 6 years have elapsed since our first TIPSS (23). Initially, only very high risk patients were selected for TIPSS. Nevertheless, with promising results emerging from our first year of clinical application (24,25). TIPSS was expanded to include patients with failed surgical shunts and to patients refractory to medical treatment for severe ascites and for patients enrolled in a waiting list for liver transplantation. With broadening of the inclusion criteria during our study difficulties arise in the interpretation of clinical benefit and long-term success of the procedure. In our first 24 patients, as recently reported (26), the technical success rate was 75%. Failures resulted from inability to puncture the portal vein because of equipment problems as detailed above. Since then, there has been only one more failure. Presently, the total success rate is 92% and the technical success rate in the year 1992 is 97%.

### ***30-Day Mortality and Complications***

The 30-day mortality rate in the first 13 (out of 18) successful cases was 15% (24-26).

This has completely changed since the advent of better material and better puncture techniques (see above). Among almost 200 hundred procedures completed by simultaneous fluoroscopic and sonographic guidance four early deaths occurred directly related to the procedure resulting from inadvertent puncture of extracapsular portal vein and onset of immediate exsanguination. The overall early mortality rate was 6,7%. Other deaths resulted from disease related problems such as sepsis or hepatic failure. Minor complications included transient elevation of bilirubin and transaminases without unfavorable sequelae. Two patients showed mild signs of hemolysis which could not be sufficiently explained. In 6 patients

balloon rupture upon stent placement required sophisticated stent correction methods all of which were successful.

### *30-Day Clinical Success*

The total early clinical success rate is 93.3% while rebleeding was encountered in 6.7% during the 30-day period. Five patients rebled from pre-existing severe ulcerative and erosive oesophageal and gastric mucosal disease probably caused by extensive sclerotherapy trials accompanied by prolonged inflation times of occlusion balloons. Our very first patient of the series started widespread mucosal bleeding two days after TIPSS most likely due to disseminated intravascular coagulopathy from accelerated absorption of ascites with imbalance of fibrinolysis. This was controlled by blood transfusions and fresh frozen plasma (23). In two other patients bleeding events continued approximately 2 weeks.

### *30-Day Encephalopathy*

In addition to mortality and shunt occlusion hepatic encephalopathy is one of the main problems of shunt surgery (9,11,13,15,16,32-34) and it is also a crucial issue in TIPSS. In non-selective shunts the postoperative rate of hepatic encephalopathy may rise up to 50% (18,26,32). Even in selective shunts an incidence of hepatic encephalopathy as high as 20% is reported (26,32). In our series, 6 patients developed a "de novo" hepatic encephalopathy during the first 30 days which was controlled by appropriate medical treatment in each. More importantly, in none of our 39 stage C patients who almost invariably presented with hepatic encephalopathy before TIPSS worsening of symptoms occurred thereafter. Conversely, those patients who had hepatic encephalopathy attributable to severe acute bleeding and significant intestinal protein uptake improved as soon as the shunt was functional. These findings are reflected more or less by variations in ammonia levels. In most of the patients with normal values before TIPSS they did not exceed the critical threshold during follow-up. In patients with increased values due to active bleeding ammonia levels decreased after TIPSS in most of the cases. At the present time, the study volume appears to be still too small for final conclusion as to the definitive risk of hepatic encephalopathy should be considered as a positive trend favouring the concept of partial diversion of portal flow volume with small calibre interposition shunting TIPSS.

### *Late Results*

The actuarial 1-year survival rate is 70%, and the 3-year survival rate 50%. In addition to the previously mentioned early deaths another 36 patients died with an average survival time of 10 months. Death was unrelated to the procedure in 13 cases while in two patients late shunt occlusion occurred 9 and 18 months, respectively, after TIPSS resulting in lethal variceal haemorrhage.

Four patients were referred for liver transplantation because of progressive decrease of liver function during a period of 5 months (4.1%).

"De novo" encephalopathy was seen in 4.7% of patients during late follow-up mostly attributable to failure to respond to protein uptake restriction. In all adequate hydration and re-establishment of correct dietary schedules retained normal brain function.

Intimal hyperplasia during the first 6 months after TIPSS is an important feature in patients with good liver function and normal or close-to-normal coagulation. In almost all patients in a Child's A stage of liver disease major intimal hyperplasia was seen both within the stented shunt segments and in the free area of the hepatic vein. It was never seen in the portal vein. When hemodynamically necessary, as determined by portosystemic pressure gradient monitoring, correction by either re-dilation or additional stenting of hepatic vein segments may be easily achieved. By contrast, in patients with reduced liver function such intimal hyperplasia is rare. This underscores the thrombogenic property of metallic stents particularly in areas without functioning endothelium and well functioning blood coagulation.

Thrombogenicity and inadvertent intimal hyperplasia appear to be some of the unsolved problems of the TIPSS procedure which warrant further research.

### **THE RATIONALE OF STENT CHOICE FOR TIPSS**

In theory, the flexible WALLSTENT can stent more peripheral and curved shunt tracts allowing a "take-what-you-can-get policy" after less than ideal transjugular punctures. With the use of the more rigid PALMAZ stent a more central and straight intrahepatic shunt course is mandatory requiring a more accurate puncture. Nevertheless, we believe

that a central and straight shunt tract facilitates an undisturbed flow pattern leading to as little as possible thrombus formation and intimal hyperplasia. This is important during shunt maturation and stented tract healing. Most of the difficulties associated with punctures aimed centrally and medially towards the portal bifurcation have been almost completely eliminated by the use of direct ultrasound guidance in addition to fluoroscopy. The radiopacity and expansion mechanism of the PALMAZ stent allows precise positioning within the target area. Excessive intrusion of the stent inside portal venous or hepatic venous structures may be avoided. Conversely, the WALLSTENT substantially extends into both the portal and the hepatic veins because of its unpredictable deployment and site of foreshortening. Presently, it is unknown if such protrusion of metallic stents inside portal or hepatic veins may have adverse effects. Protrusion into the main stem of the portal vein, typically found with the WALLSTENT, may cause impaired flow to the portal branches with decrease of liver function or even may increase the risk of intimal hyperplasia. The use of the PALMAZ stent allows adaptation of the shunt dimensions to the particular hemodynamic situation in each patient because a range of diameters between 7 and 16 mm is obtained by choosing the appropriate balloon size for deployment and, possibly, further expansion.

Notwithstanding the above mentioned theoretical considerations the choice of stent will be determined by the personal preference of the operator in the short term and the results of larger scale clinical trials in the long term.

#### References:

1. Abecassis M., Gordon J.D., Colapinto R.F. et al.: The transjugular intrahepatic portosystemic shunt (TIPSS): an alternative for the management of life-threatening variceal hemorrhage. *Hepatology* 1985; 5: 1032A
2. Burgener F.A., Gutierrez O.H.: Non-surgical production of intrahepatic portosystemic venous shunts in portal hypertension with the double lumen balloon catheter. *Fortschr Röntgenstr* 1979; 130: 686-688
3. Child C.G., Turcott J.G.: Surgery and portal hypertension. In: Child C.G.(ED.): *The liver and portal hypertension*. Saunders, Philadelphia 1964
4. Colapinto R.F., Stronell R.D., Birch S.J. et al: Creation of an intrahepatic portosystemic shunt with a Grüntzig balloon catheter. *Can Med Assoc. J* 1982; 126: 267-126.
5. Coldwell D.M., Moore A.D.A., Ben-Menachem Y. Johansen K.H.: Bleeding gastroesophageal varices: gastric vein embolization after partial decompression. *Radiology* 1991; 178: 249-251
6. DeLacy A.M., Nevasa M., García -Pagan J.C., et al: Reversal of portal flow after distal splenorenal shunt (DSRS). Relationship to hepatic encephalopathy and impaired liver function. *J. Hepatology* 1989; 9(Suppl): S 142.
7. Fischer J.E., McCinley J.: Comparative randomized study: Proximal versus distal splenorenal shunt. *Policlinico Sez Chir* 1985; 92: 592-596.
8. Foster J.H., Ellison L.H., Donovan Th., Anderson A.: Quantity and quality of survival after portosystemic shunts. *Am J. Surg* 1971; 92: 592-596.
9. Galambos J.T: Portal hypertension. *Seminars in Liver Disease* 1985; 5: 277-290
10. Gordon J.D., Colapinto R.F., Abecassis M. et al: Transjugular intrahepatic portosystemic shunt: A nonoperative approach to life-threatening variceal bleeding. *Can J. Surg* 1987; 30: 45-49.
11. Johansen K.: Partial portal decompression for variceal hemorrhage. *Am J. Surg* 1989; 157: 479-482.
12. Koch G., Rigler B., Tentzeris . et al: Der intrahepatische porto-cavale Shunt. *Langenbecks Arch Chir* 1973; 333: 237-244.
13. Lafortune M., Patriquin H., Pomier G. et al: Hemodynamic changes in portal circulation after portosystemic shunts: Use of duplex sonography in 43 patients. *A.J.R.* 1987; 149: 701-706.
14. Millikan W.J., Warren W.D., Henderson J.M. et al: The Emory prospective randomized trial: selective versus non-selective shunt to control variceal bleeding. *Ann Surg* 1985; 201: 712-722.
15. Murray J.F., Mulder D.G., Nebel L.: The effect of retrograde portal venous flow following side-to-side portocaval anastomosis. *J. Clin Invest* 1961; 40: 1413-1420.
16. Ohnishi K., Satto M., Sato S. et al: Direction of splenic venous flow assessed by pulsed Doppler flowmetry in patients with large splenorenal shunts. Relation to spontaneous hepatic encephalopathy. *Gastroenterol* 1985; 89: 180-189.
17. Pagliaro L., Burroughs A.D., Sorensen T.I.A., Lebrech D., Morabito A., D'Amico G., Tine F.: Therapeutic controversies and randomized controlled trials (RCTs): prevention of bleeding and rebleeding in cirrhosis. *Gastroenterol International* 1982; 2: 71-84.
18. Palmaz J.C., Sibbitt R.R., Reuter S.R., García F., Tío F.O.: Expandable intrahepatic portocaval shunt stents: early experience in the dog. *AJR* 1985; 145: 821-825.
19. Palmaz J.C., García F., Sibbitt S.R., Tío F.O., Kopp D.T., Schwesinger W., Lancaster J.L., Chang P.: Expandable intrahepatic portocaval shunt stents in dogs with chronic portal hypertension. *A.J.R.* 1986; 147: 1251-1254.
20. Rector W.G., Hoefs J.C., Hossack K.F., Everson G.T.: Hepatofugal portal flow in cirrhosis: Observation of hepatic hemodynamics and the nature of the arteriportal

#### IV INTERNATIONAL COURSE ON VASCULAR AND INTERVENTIONAL RADIOLOGY AS THERAPEUTIC ALTERNATIVE

- communications. *Hepatology* 1988; 8: 16-20.
21. Redeker A.G., Geller H.M., Reynolds T.B.: Hepatic wedge pressure, blood flow, vascular resistance and oxygen consumption in cirrhosis before and after end-to-side portocaval shunt. *J. Clin Invest* 1958; 37: 606-618.
  22. Reich M., Olumide F., Jorgensen E., Eiseman B.: Experimental cryoprobe production of intrahepatic portocaval shunt. *J. Surg Res* 1977; 23: 14-18.
  23. Reichle F.A., Fahmy W.F., Golsorkhi M.: Prospective comparative clinical trial with distal splenorenal and mesocaval shunts. *Am J. Surg* 1979; : 137:13-21.
  24. Richter G.M., Palmaz J.C., Noeldge G., Rössle M., Siegerstetter V., Franke M., Wenz W.: Der Transjuguläre intrahepatische portosystemische Stent-Shunt (TIPSS). *Radiologe* 1989; 29: 406-411.
  25. Richter G.M., Noeldge G., Palmaz J.C., Roessle M., Siegerstetter V., Franke M., Gerok W., Wenz W., Farthmann E.: Transjugular Intrahepatic Portocaval Stent Shunt: Preliminary Clinical Results *Radiology* 1990; 174: 1027-1030.
  26. Richter G.M., Noeldge G., Palmaz J.C., Roessle M.: The transjugular intrahepatic portosystemic stent-shunt (TIPSS): results of a pilot study. *Cardiovasc Intervent Radiol* 1990; 13: 200-207.
  27. Richter G.M., Noeldge G., Palmaz J.C., Roessle M.: Evolution and clinical introduction of TIPSS, the transjugular intrahepatic portosystemic stent-shunt. *Sem Intervent Radiol* 1991; 8: 331-340.
  28. Rösch J., Hanafee W.N., Snow H.: Transjugular portal venography and radiologic portocaval shunt: an experimental study. *Radiology* 1969; 92: 1112-1114.
  29. Rösch J., Uchida B.T., Putnam J.S. et al: Experimental intrahepatic portocaval anastomosis: use of expandable Gianturco stents. *Radiology* 1987; 162: 481-485.
  30. Rössle M., Haag K., Noeldge G., Richter G., Wenz W., Farthmann E., Gerok W.: Hämodynamische Konsequenzen der portalen Decompression: Welches ist der optimale Shunt?. *Z. Gastroenterol* 1990; 28: 630-634.
  31. Rypins E.B., Mason G.R., Conroy R.M., Sarfeh I.J.: Predictability and maintenance of portal flow pattern after small-diameter portocaval H-grafts in man. *Ann Surg* 1984; 200: 706-710.
  32. Sarfeh I.J., Rypins E.B., Conroy R.M., Mason G.R.: Portocaval H-graft: relationships of shunt diameter, portal flow patterns and encephalopathy. *Ann Surg* 1983; 197: 422-426.
  33. Sarfeh I.J., Rypins E.B., Raiszadeh M., Milne N., Conroy R.M., Lyons K.P.: Serial measurement of portal hemodynamics after partial portal decompression. *Surgery* 1986; 100: 52-58.
  34. Spina G.P., Galeotti F., Opocher E., Santambrogio R., Cucchiario G., López C., Pezzuoli G.: Selective distal splenorenal shunt versus side-to-side portocaval. Clinical results of a prospective controlled study. *Am J. Surg* 1988; 155: 564-571.
  35. Warren W.D., Millikan W. J. Jr. Henderson J.M et al: Ten years portal hypertensive surgery at Emory: results and new perspectives. *Ann Surg* 1982; 195: 530-542.



## Tratamiento Medico de la Hipertension Renovascular

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" Accordingly, any decision for medical management of a patient with a potentially correctable renal artery lesion requires close continued observation as to blood pressure control and function of the individual kidneys....."

Hunt JC et al. Arch Intern Med 1974 (1)

Se le ha reconocido al riñón un papel importante como determinante en la génesis de la hipertensión arterial (HTA) a través de su influencia entre otros en el metabolismo del agua y electrólitos. El daño renal y las lesiones renovasculares pueden producir HTA no solo en el laboratorio experimental, sino también en el hombre.

Es costumbre incluso en los libros de texto el clasificar la HTA como primaria o secundaria representando esta última un porcentaje poblacional del 1-5% según el medio donde se realice el estudio y el tipo de población analizada (2,3).

La hipertensión renovascular (HTARV) esta causada por 2 tipos de lesiones bien diferenciadas con orígenes y consecuencias bien distintas. Por un lado, la displasia fibromuscular con buenos resultados terapéuticos, control de la HTA y no progresión de la lesión anatómica. Por otro la lesión ateromatosa, la cual todavía no sabemos si es causa o consecuencia de la HTA y que en muchas ocasiones puede ser un componente sobreañadido a la HTA esencial, situación generalmente conflictiva por el hecho de una afectación bilateral y por tendencia a la progresiva oclusión arterial en distintos tiempos (4).

El tratamiento médico de la HTARV con las modernas drogas hipotensoras debería considerarse como manejo terapéutico inicial y de elección, representando la mejor alternativa si consiguiera el control de la HTA y evitara el fenómeno evolutivo de la lesión anatómica preservando la función renal.

El tratamiento farmacológico actual de la HTA ha permitido reducir sensiblemente el número de complicaciones vasculares y renales. Nos referimos

concretamente a los betabloqueantes, calcioantagonistas e inhibidores de la enzima convertidora de la angiotensina.

A pesar de esta euforia inicial no debemos olvidar el acuerdo generalmente aceptado por todos los "hipertensiólogos" del carácter evolutivo de determinadas lesiones de la arteria renal responsables de HTA.

No existen estudios randomizados para evaluar la eficacia del tratamiento médico versus revascularización (ya sea cirugía o dilatación transluminal percutánea-PTA). La mayoría de los estudios sobre el tema presentan como referencia el trabajo de Hunt JC y col (1), donde se compararon retrospectivamente dos grupos similares aunque no randomizados de aproximadamente 100 pacientes con HTARV tratados médicamente o por cirugía revascularizadora (todavía no disponíamos de PTA). De los 114 pacientes tratados médicamente de forma continuada por 6 o mas meses, 16 fueron transferidos a cirugía tras 1 a 5 años. Entre los 98 pacientes que continuaban con tratamiento médico se consiguió un control de la HTA (TAD < 100 mmHg) por 1 año en 92, durante 5 años en 63, por 7 o mas años en 52.

Al final del estudio de seguimiento (10 años) el 16% de los pacientes tratados quirúrgicamente habían fallecido comparados con el 40% de los tratados médicamente. Además, en 4 de los pacientes tratados médicamente la función renal se deterioró precisando diálisis. Este grado de deterioro de función renal no se llegó a observar en el grupo tratado mediante cirugía.

El grupo tratado mediante cirugía revascularizadora tuvo una menor y muy significativa tasa de mortalidad y morbilidad vascular y renal así como un mejor control de la presión arterial que el grupo tratado médicamente.

Un análisis actual de este estudio pone de inmediato en evidencia que el tipo de fármacos utilizados no son los que actualmente disponemos, que además carecen

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de efectos adversos sobre metabolismo glucídico y lipídico. Hay que ser cautos en la extrapolación de estos resultados a la actualidad. En este trabajo ya se da a entender que el control de la hipertensión arterial es solo un aspecto parcial de la enfermedad vasculorenal y que el control adecuado de las cifras de presión arterial con tratamiento farmacológico, no significa necesariamente un buen control de la enfermedad (5).

El tratamiento médico se ha establecido como el preferido inicialmente en la fibrodisplasia de la media. En esta situación parece ser que la lesión anatómica es la única causa de HTA (4), siendo poco frecuente el deterioro de la función renal como consecuencia de la progresiva obstrucción de la arteria renal (6) siempre que se controle la HTA. Se opta por dejar el tratamiento revascularizador para aquellos casos en que el control de la HTA sea difícil de conseguir después del uso de mas de dos drogas.

Hay que admitir incluso en las esferas mas conservadoras, que la aparición de la Radiología Vasculosa Intervencionista y la introducción de la Angioplastia transluminal percutánea (PTA) (7) modificó sustancialmente la actitud ante un paciente con HTARV.

Múltiples publicaciones han aparecido en los últimos años sobre la eficacia de la PTA en la HTARV que no voy a referir, abarcando pacientes que hasta ese momento se encontraban en tratamiento médico o con indicaciones límites para la revascularización quirúrgica, afectos de HTARV.

Aunque en la práctica médica un tratamiento farmacológico agresivo puede conseguir mejoría de la función renal en un paciente con HTARV, es mas frecuente el deterioro del filtrado glomerular, pudiendo ocurrir incluso en presencia de cifras normales de presión arterial en el 40-50% de los pacientes (5,8).

Incluida como la mas frecuente de las lesiones fibrodisplásicas, la fibroplasia de la capa media (75-80%) es la que mas se puede beneficiar del tratamiento médico (9), reservando el tratamiento revascularizador (cirugía y/o PTA) para aquellos casos que cursan con cifras de presión arterial difíciles de controlar con mas de dos drogas, y/o ante la presencia de un deterioro de la función renal y/o complicación cardiovascular, o afectación de ramas intrarenales.

El resto de las lesiones fibrodisplásicas son mas difíciles de diagnosticar, y aunque no son tan frecuentes está demostrado que el tratamiento

farmacológico por si solo no es capaz de frenar la evolución de la enfermedad vascular por lo que su corrección precisará de una actitud intervencionista ya sea cirugía o PTA.

Una mención especial requiere la lesión ateromatosa de la arteria renal (60-70%) en donde el tratamiento médico tiene una mayor justificación (6), ya que estos pacientes suelen ser añosos y presentar otro tipo de patología vascular extrarenal que les sitúa como de alto riesgo intervencionista. Sin embargo, debemos recordar que aquellos pacientes con estenosis severa (>75%) de la arteria renal, la disminución de las cifras de presión arterial con tratamientos farmacológicos agresivos podría reducir sensiblemente la presión de perfusión renal y facilitar la isquemia y atrofia renal.

No haré diferencias en este capítulo entre las diversas formas de presentación de la enfermedad ateromatosa ya que van a ser tratadas mas específicamente en otro lugar.

#### Referencias:

1. Hunt J.C., Sheps S., Harrison E. Jr, Strong C., Bernatz Ph: Renal and renovascular hipertensión. A reasoned approach to diagnosis and management. Arch Intern Med 1974; 133: 988-999.
2. Davis B., Crook J., Vestal R., Oates J.: Prevalence of renovascular hipertensión in patients with grade III or IV hypertensive retinopathy. New Engl J Med 1979; 301: 1273-1276.
3. Keith T.A.: Renovascular hipertensión in black patients. Hipertensión 1982; 4: 438-443.
4. Pickering T.G.: Diagnosis and evaluation of renovascular hipertensión. Circulation 1991; 83 (suppl D): 147-154.
5. Dean R., Kieffer R., Smith B., Oates J., Nadeau J., Hollifield J., DuPont W.: Renovascular hipertensión. Anatomic and renal function changes during drug therapy. Arch Surgery 1981; 116: 1408-1415.
6. Novick A.: Management of renovascular disease. A surgical perspective. Circulation 1991; 83 (suppl D): 167-171.
7. Gruntzig A., Kuhlmann U., Vetter W., Lutolf U., Meier B., Siegenthaler W.: Treatment of renovascular hipertensión with percutaneous transluminal dilatation of a renal-artery stenosis. Lancet 1978; 1: 801-802.
8. Novick A., Pohl M., Schreiber M., Gilford R. Jr, Vidt D.G.: Revascularization for preservation of renal function in patients with atherosclerotic renovascular disease. J Urol 1983; 129: 907-912.
9. Isorna S., Maynar M., Rodríguez Pérez J.C.: Patología vascular renal, en Jiménez-Cruz J.F. y Rioja-Sanz L.A., ed. Tratado de Urología. Spain: JR Prous; 1993: vol 2, 1729-1746.



## Technical Developments and Instrumentation

# Coaxial Catheter-Needle System for Transjugular Portal Vein Entrance<sup>1</sup>

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**P**ROPER entry into a large intrahepatic portal venous branch is the crucial part of successfully performing a transjugular intrahepatic portosystemic shunt (TIPS) procedure. The Brockenbrough or modified Ross transseptal needle, the Colapinto transjugular biopsy needle, and micropuncture coaxial needle have been used for liver puncture (1-4). In 1986, after traumatic experiences with the modified Ross needle in experimental work, we designed a coaxial catheter-needle for an "atraumatic" transjugular portal vein entry in swine (5). We modified this set for clinical use in performing TIPS and describe our experience with it in 50 patients.

### MATERIALS AND METHODS

The coaxial catheter-needle system consists of five components (Cook, Bloomington, Ind) (Figs 1, 2). The three outside components—a 10-F, 41-cm-long introducer sheath containing a 10-F Teflon, 52-cm-long catheter with a 51-cm-long, 14-gauge curved metal cannula inside—are used for catheterization of the hepatic

vein and orientation and stabilization of the needle for liver puncture. Portal vein entry is achieved with the two inner components—a 62-cm-long, 0.038-inch-diameter needle inside a well-tapered 5-F Teflon catheter.

The outside 10-F nylon sheath provides stability for the entire system and prevents buckling of the guide wire inside the heart during catheter exchanges. It may also be used for final stent placement, depending on which stent is selected. The sheath has a check flow valve that eliminates bleeding during catheter exchanges and a side port used for continuous pressure infusion of heparinized saline during the procedure to prevent clot formation inside the sheath. The sheath has a metal marker at its tip, which indicates its position in the vein. The well-tapered 10-F Teflon catheter and the 14-gauge, blunt-end metal cannula have a 15° curve over their distal 4 cm to facilitate hepatic vein catheterization and to orient the needle for liver puncture. An arrow at the needle hub indicates the direction of the curve. The curve on the metal cannula can be made more acute by carefully bending it. This facilitates catheterization of a horizontally oriented hepatic vein. However, the needle and 5-F catheter must be inside the cannula during bending to avoid compromising its lumen. Proximally, the 0.038-inch needle is a rigid, 50-cm-long metal cannula and, distally, a 12-cm-long segment of a guide wire. Its end is sharpened to a point that extends about 2 mm over the well-tapered tip of the 5-F Teflon catheter.

The coaxial set is introduced into an internal jugular vein, usually on the right side, after dilation with a 12-F dilator. It is guided down to the inferior vena cava and into the right hepatic vein with use of a J guide wire. In case of an acute angle between the hepatic vein origin from the inferior vena cava, a preshaped 5-F catheter is used for hepatic vein catheterization. After manual injection of contrast material to visualize the hepatic vein anatomy, the metal 0.038-inch needle is introduced and locked to the 5-F catheter and the 14-gauge cannula is locked to the 10-F catheter. For puncture, the set is

withdrawn to the proximal 3 cm of the right hepatic vein, cephalad to the expected portal vein bifurcation. The system is then rotated anteriorly and the 10-F catheter wedged inferiorly against the hepatic vein wall. Wedging is essential for successful liver puncture, because if the tip of the 10-F catheter is free in the hepatic vein, the needle slides along the vein wall rather than puncturing it. A sharp thrust into the hepatic parenchyma at a distance of 3-6 cm, depending on location and anatomy of the portal vein bifurcation, is necessary for puncture. The needle and 5-F catheter are locked together and advanced as a unit during puncture. The needle is then removed and suction applied to the 5-F catheter during its slow withdrawal. When blood is freely aspirated, contrast material is injected to confirm that a suitable portal branch has been entered. A 0.038-inch Bentson guide wire is advanced into the portal and splenic veins, and over it the 5-F catheter is advanced deeper into the portal vein. The floppy guide wire is then exchanged for a Superstiff Amplatz-type guide wire.

While keeping the cannula wedged, the 10-F Teflon catheter is unlocked and advanced into the portal vein, dilating the liver tract for introduction of further catheters. With a hard liver, the 14-F cannula can be advanced together with the 10-F catheter to stiffen it and overcome the resistance of the liver parenchyma and portal vein wall. The 10-F sheath is then advanced to the point at which its opaque tip is resting against the hepatic vein wall at the puncture site and the 10-F catheter and metal cannula are withdrawn. A pigtail catheter is then introduced for pressure measurement and portal venography. The distance between the metal marker at the sheath tip and the puncture site in the portal vein branch indicates the length of the intrahepatic tract and helps in selecting the proper length of stent(s) for TIPS (Figs 3, 4).

For placement of the Z stent (Gianturco-Rösch biliary Z stent; Cook), the sheath is advanced into the portal vein. After balloon dilation of the liver puncture tract, the sheath is first advanced to

**Index terms:** Catheters and catheterization, technology, 957.123 • Liver, interventional procedure, 761.1299, 957.123 • Portal vein, 957.123 • Shunt, portosystemic, 959.453.

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the upper end of the partially deflated balloon. Connected together, both are then advanced simultaneously into the portal vein. The balloon is then withdrawn and the stent deployed.

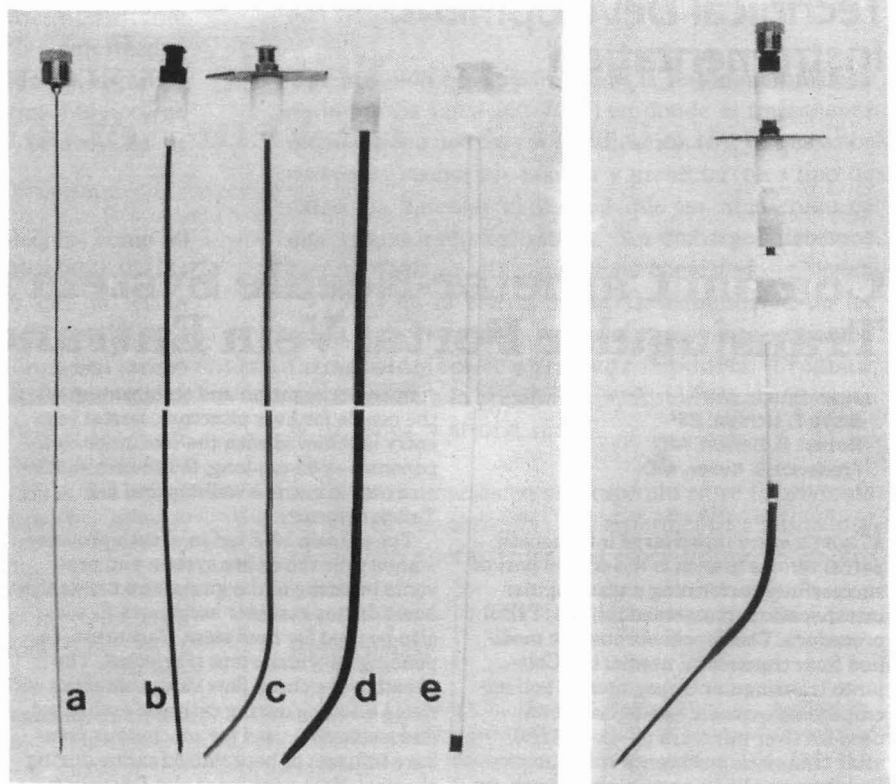
## RESULTS

With this coaxial catheter-needle set we were able to enter the portal system and establish a well-functioning TIPS in all 50 consecutive patients with a patent portal vein at arterial portography. The standard curve on the 14-gauge cannula was sufficient in 39 patients in whom the distance between the hepatic and portal vein was 3 cm or longer (Fig 3). In 11 patients with small livers and a high position of the portal bifurcations, we had to increase the cannula curve to about 30°–45° (Fig 4). Viewed in the frontal projection, the portal bifurcation was just below the hepatic vein in these patients. However, viewed in the lateral projection, the portal vein was 2–3 cm anterior to the hepatic vein and therefore an increased curve on the cannula was necessary to reach and enter a main portal vein branch. The main right portal branch was entered 1–5 cm from the portal bifurcation in 27 patients; the main left portal branch, 1–4 cm from the bifurcation in 19 patients; and the portal bifurcation, in four patients.

During attempts at portal vein puncture we entered an intrahepatic branch of a hepatic artery in three patients and an intrahepatic bile duct in eight patients. In two patients with massive ascites and small atrophic livers, we penetrated the liver capsule and aspirated ascites. The catheter was withdrawn back into the cannula in all of these cases and additional puncture(s) performed until the portal system was entered. No complications occurred from these inadvertent punctures.

## DISCUSSION

Several needle access systems for liver puncture and portal vein entrance are available and all have their advocates (1–4). We cannot compare the efficacy of these systems with that of our coaxial catheter-needle set, which we have used exclusively for the last 7 years. We developed it and began using it extensively for experimental work in young swine when use of a modified Ross needle resulted in



**1.** Components of the coaxial catheter-needle system: 0.038-inch-diameter point-sharp needle (a); 5-F well-tapered Teflon catheter (b); 14-gauge metal cannula (c); 10-F Teflon catheter (d); and 10-F introducer sheath with check flow valve, side port, and metal marker at its tip (e). **2.** Assembled coaxial catheter-needle system.

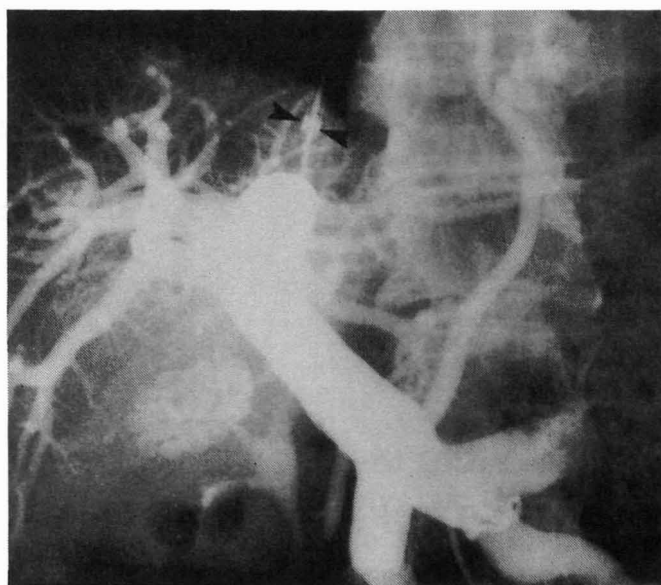
liver injuries (5). In 1990 we modified the set for clinical application and used it for successful TIPS creation in 50 patients. We consider it an effective and safe system. The rigidity of the 14-F metal cannula allows precise needle orientation and stabilization for liver puncture. The sharp small-diameter needle can penetrate even the most cirrhotic liver, and it is relatively atraumatic. No complications occurred despite penetration of the liver capsule (4% of procedures) and entry into the hepatic artery branches (6% of procedures) or intrahepatic bile ducts (16% of procedures). The 10-F outer sheath provides stability for the system, and the metal marker at its tip allows marking the puncture site in the hepatic vein on the portogram obtained prior to TIPS. The length of the liver puncture tract thus can be measured for selection and proper positioning of the expandable stent.

## References

1. Richter GM, Noeldge G, Palmaz JC, Roessle M. The transjugular intrahepatic portosystemic stent-shunt (TIPSS): results of a pilot study. *Cardiovasc Intervent Radiol* 1990; 13:200–207.
2. Zemel G, Katzen BT, Becker GJ, Benenati JF, Sallee S. Percutaneous transjugular portosystemic shunt. *JAMA* 1991; 266: 390–393.
3. Ring EJ, Lake JR, Roberts JP, et al. Using transjugular intrahepatic portosystemic shunts to control variceal bleeding before liver transplantation. *Ann Intern Med* 1992; 116:304–309.
4. Cope C, Burke DR, Meranze SG. Atlas of interventional radiology. Philadelphia: Lippincott, 1990.
5. Uchida BT, Putnam JS, Rösch J. "Atraumatic" transjugular needle for portal vein puncture in swine. *Radiology* 1987; 163:580–581.



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4.

**Figures 3, 4.** (3) Portal venogram obtained after entry into the right portal branch. The standard cannula curve was used for portal branch entrance. The metal marker on the 10-F introducer sheath (arrowheads) indicates puncture site in the hepatic vein wall. The puncture tract is 6.5 cm long (corrected for magnification). (4) Portal venogram obtained after entry into the left portal branch. An increased cannula curve to about 30° was used for portal branch entry. The metal marker on the 10-F introducer sheath (arrowheads) indicates puncture site in the hepatic vein wall. In frontal projection, the puncture tract is 1.5 cm long (corrected for magnification). It is 2.5 cm long in lateral projection.

## **Covered Self-expandable Z-stents in Treatment of Malignant Esophageal Obstructions and Esophagorespiratory Fistulae**

JOSEF RÖSCH, M.D.<sup>1</sup>

**Self-expandable Gianturco-Rösch type Z-stents (GRZ stents) covered with silicone or polyethylene membrane were used for treatment of 60 patients, in 48 with malignant esophageal obstructions and in 12 with esophagorespiratory fistulae. Fluoroscopically guided stent placement was well tolerated. With malignant obstructions immediate relief of dysphagia was achieved in 95% of patients and sustained relief in 80% of patients with an average follow up of 3.2 months and range from 1 to months**

**With esophagorespiratory fistulae aspiration was completely relieved in 75% of patients and partially relieved in 17% of patients. No complications occurred during stent placement. Late stent migration into the stomach occurred in 3 patients where stents extended into the gastric fundus and their course was uneventful. Severe delayed complications developed in 4 patients including esophageal perforation (1 patient) and significant bleeding (3 patients).**

**Conclusion: The covered GRZ stents are effective and relatively safe means of palliating patients with severe malignant esophageal obstructions and esophagorespiratory fistulae.**

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## Vascular Applications: Veins

# Gianturco-Rösch Expandable Z-Stents in the Treatment of Superior Vena Cava Syndrome

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**Abstract.** Gianturco-Rösch expandable Z-stents were used in 22 patients with superior vena cava syndrome (SVCS). Stents were placed in all patients in the SVC and in 17 patients, also into the innominate veins. Stent placement resulted in complete relief of symptoms in all patients. Twenty-one patients had no SVCS recurrence from 1 to 16 months, to their death, or to the present time. SVCS recurred only in 1 patient 9 months after stent placement due to tumor ingrowth and secondary thrombosis. Based on ours and on other reported experiences, expandable metallic stents are effective devices for treatment of the SVCS which is difficult to manage by other means.

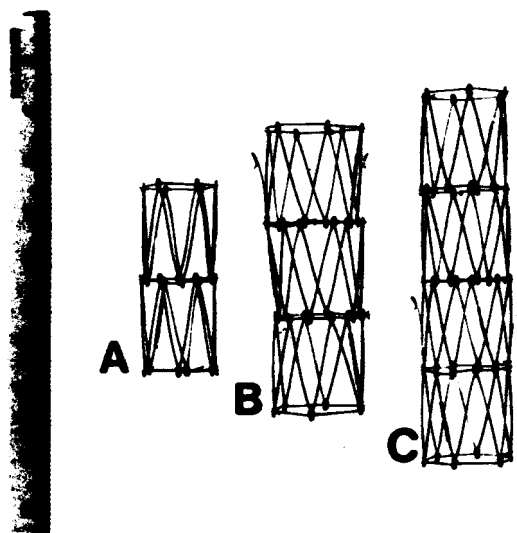
**Key words:** Superior vena cava syndrome— Venous obstruction—Lung carcinoma—Expandable metallic stent—Gianturco-Rösch Z-stent

Treatment of the superior vena cava syndrome (SVCS) is difficult, particularly in patients with obstructions caused by malignant tumors and/or post-radiation fibrosis. Patients are usually treated medically with minimal relief of their severe congestive symptoms. After our successful treatment of SVCS with modified Gianturco self-expandable Z-stents (Gianturco-Rösch Z-stents) (Fig. 1) in 2 patients [1], we used them in another 20 patients and summarize our experience.

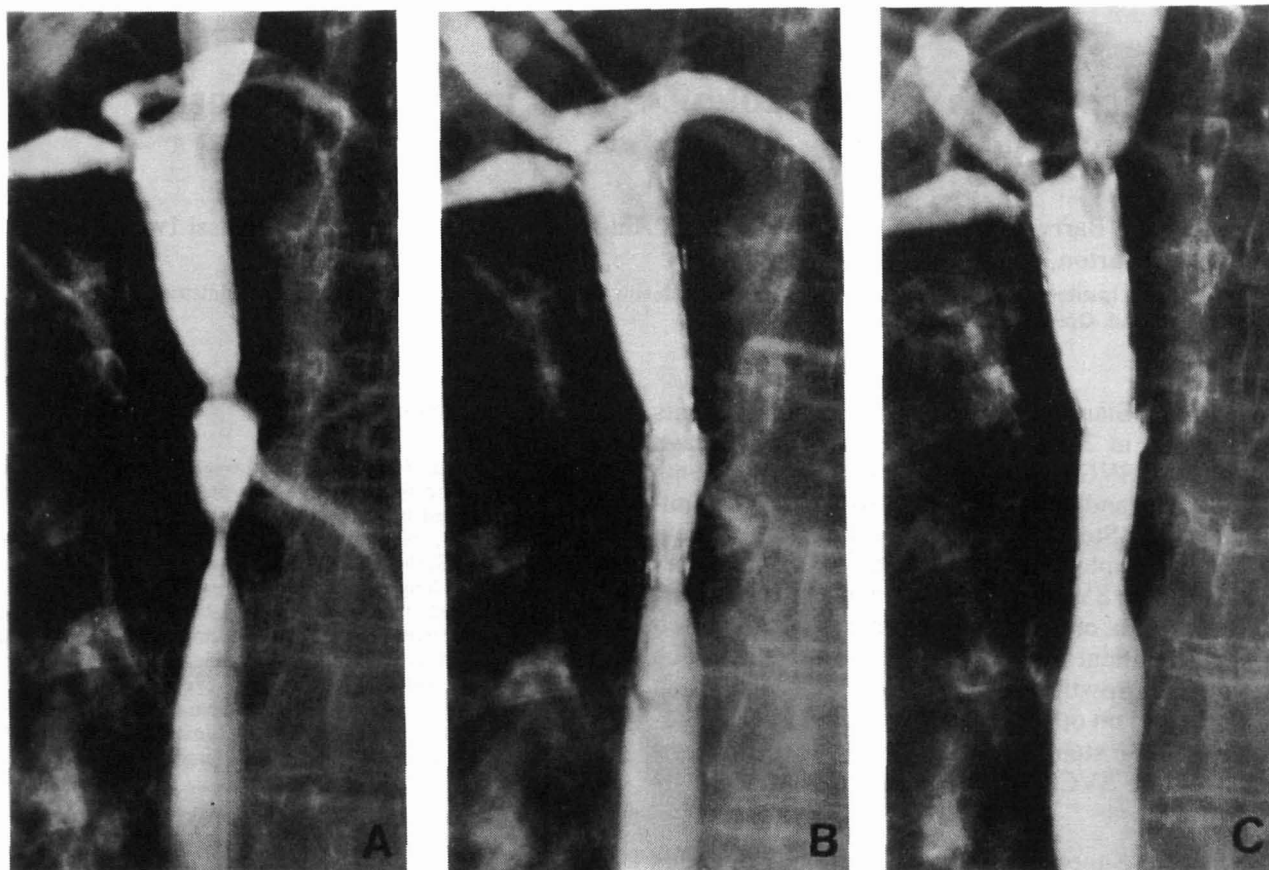
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## Material and Methods

Of the 22 patients, 16 were men and 6 were women; their ages ranged from 28 to 68 years (median, 56 years). In 2 patients, SVCS was caused by a benign process, post-radiation fibrosis and mediastinitis, respectively. Of the 20 patients with malignant etiology of SVCS, the SVC obstruction was caused by mediastinal extension of lung carcinoma in 14 patients and mediastinal metastases of abdominal tumors in 6 patients. All patients had advanced SVCS with face, neck, and arm swelling and venous engorgement. Twelve patients had significant facial cyanosis, 5 had hoarseness, and 7 complained of severe headaches. In 12 patients, SVCS symptoms developed from 5 months to 4 years following maximum radiation of their tumors; in 1 patient the SVCS occurred during radiation of his lung carcinoma.



**Fig. 1.** The Gianturco-Rösch self-expandable Z-stents hand made in our research laboratory. A A double-body 15-mm diameter stent without barbs. B A three-body 18-mm diameter stent with barbs at its upper body. C A four-body 18-mm diameter stent with barbs at its second lower body.



**Fig. 2.** A 58-year-old woman with severe superior vena cava syndrome secondary to postradiation fibrosis. After stent placement she was asymptomatic for 11 months until her death of a stroke. **A** Initial superior vena cavagram reveals two concentric stenoses and retrograde filling of collaterals. **B** Superior vena cavagram immediately after placement of a double-body stent with a skirt shows good flow through the stent which expanded to about 60% of its diameter. **C** Superior vena cavagram 2 months after stent placement reveals further expansion of the stent and improved flow.

Control venograms in 2 patients with benign obstructions exhibited tight, concentric, smoothly outlined stenoses of the SVC (Fig. 2). With malignant lesions, venography revealed a wide range of findings from irregular stenoses secondary to compression, and/or direct invasion of the tumor ( $n = 13$ ) to complete SVC occlusion ( $n = 7$ ) (Figs. 3–7). In 17 patients the obstructive process extended into the innominate vein(s). The right innominate vein was involved together with the SVC in 9 patients and in 1 of them the stenosis extended also on the subclavian vein. The left innominate vein was involved in 3 patients and both innominate veins in 5 patients, usually in their proximal portions. In 7 patients with complete SVC occlusion and 5 patients with significant thrombi in the SVC or its branches above the obstruction, selective infusion of urokinase (12–72 h) was used to achieve complete thrombolysis prior to stent placement (Figs. 4, 5, 7).

We used the Gianturco-Rösch self-expandable Z-stents made in our research laboratory from stainless steel wire 0.014–0.016 inches in diameter (Fig. 1). Their legs were connected with monofilament line to control their expansion to a diameter of 1.5–1.8

cm. The single body stents were 2–3 cm long. Depending on the length of the obstructive lesion, two to five single body stents were connected together with a monofilament line to form a stent 5–10 cm long. The SVC stents in the first of 10 patients had on their distal ends a 1.5 cm long wire skirt containing small hooks to prevent stent migration. Two stents had a skirt, but without hooks, also on their proximal ends. The SVC stents in the latter 12 patients did not have skirts at their ends but had two barbs at one of their bodies. The barbs were attached to the body placed in the center of stenosis. The stents placed in the innominate veins above the SVC stents did not have skirts or barbs.

The institutional Human Research Committee gave approval for stents to be used on a compassionate basis and the patients signed an informed consent for the procedure.

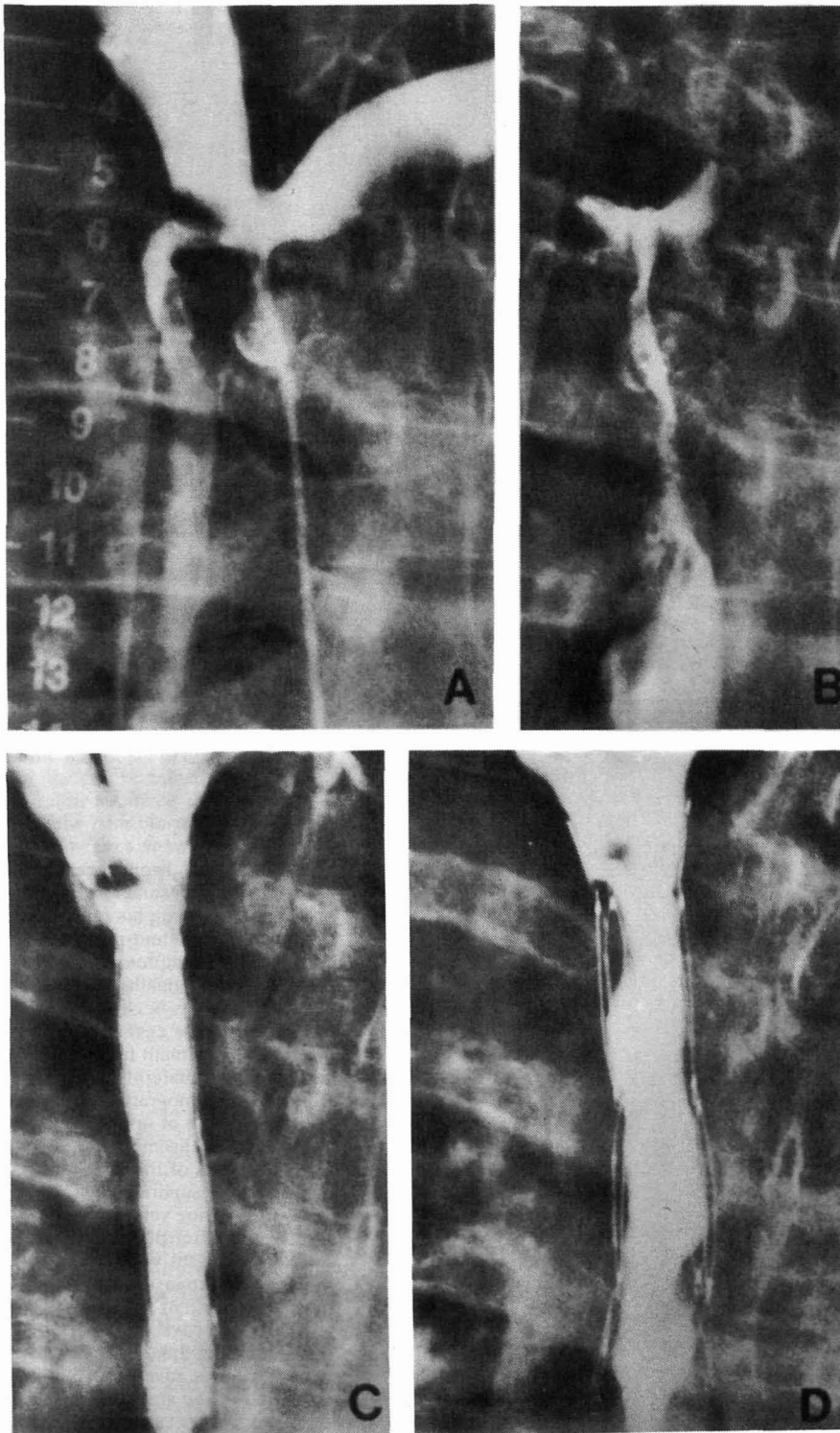
The right transfemoral approach was used in all patients for stent placement into the SVC, right innominate vein, and in 3 patients, into the left innominate vein. In 5 patients, the left innominate stent was placed by the left transjugular approach (Fig. 6). A 12-mm balloon catheter was introduced first and inflated inside the obstructed region. This was done more for delineation of the extent of the obstruction rather than for dilation, because in the majority of patients the obstruction relapsed after balloon deflation. The extent of the lesion was then marked for exact stent positioning.

A 12–14 French Teflon sheath with an introducing catheter inside was then exchanged for the balloon catheter. The stent was then loaded over a guidewire, introduced into the sheath, and pushed to its end. Still inside the sheath, it was carefully positioned just above the marked upper end of the stenotic lesion.

The stent was then deployed by withdrawing the sheath while holding the stent in position with a pusher. Extruded from the sheath, the stent expanded and dilated the narrowed venous lu-



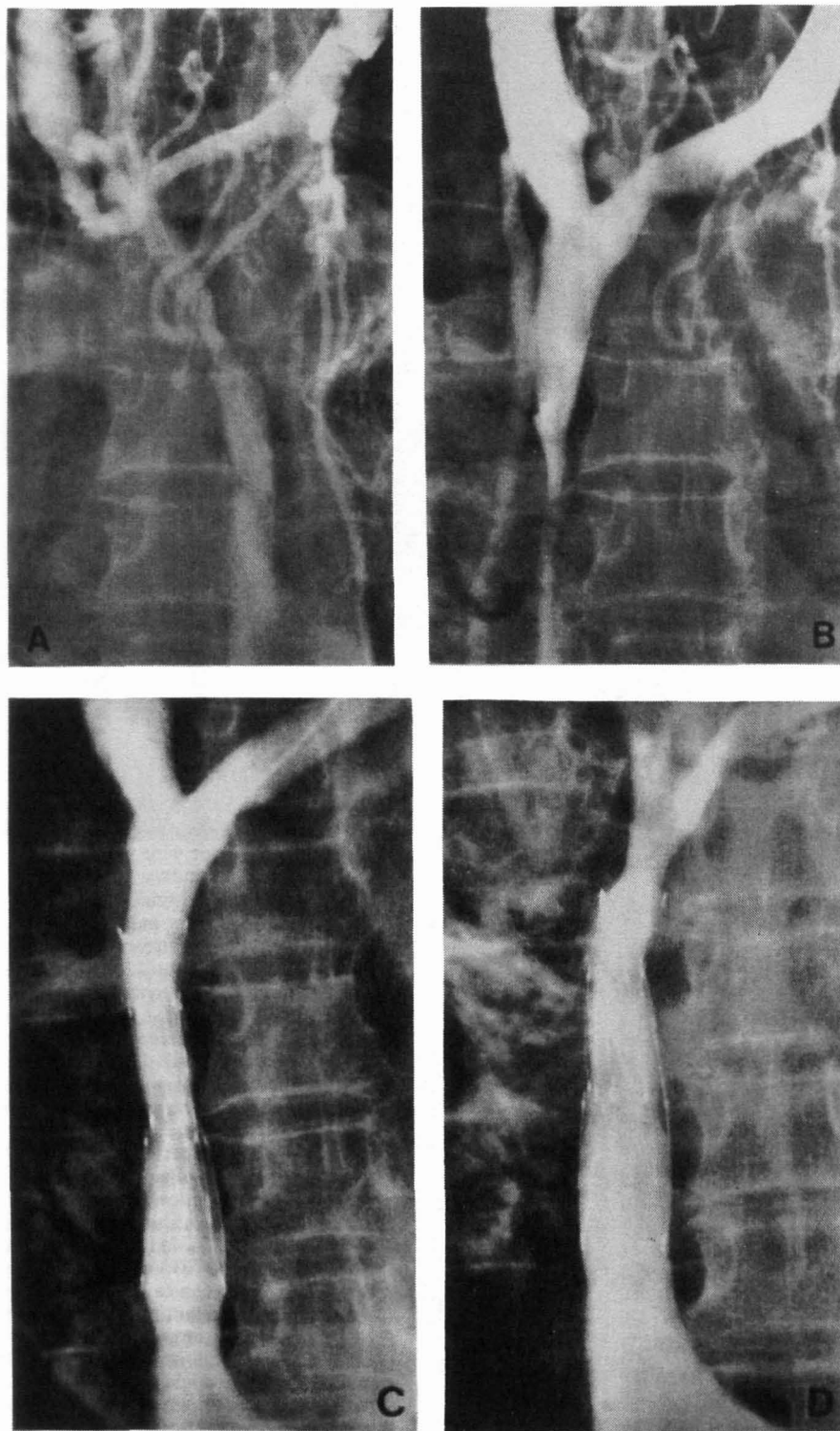
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**Fig. 3.** A 64-year-old man with severe superior vena cava syndrome secondary to squamous cell carcinoma extending into the mediastinum. The patient was already treated by maximum dose radiation. After stent placement he was asymptomatic for 11 months until his death due to tumor metastases. **A & B** Initial venograms show severe obstruction of the superior vena cava secondary to direct tumor ingrowth. **C** Superior vena cavagram immediately after placement of a double-body stent with skirts shows good stent expansion and excellent superior vena cava patency. **D** Superior vena cavagram 2 months after stent placement demonstrates further stent expansion. Filling defects at the stent outline could be due to tumor ingrowth and/or intimal hyperplasia.

men. When using several stents, the more distal stent was placed first, followed by more proximal stent(s). The obstructive lesions were often long and complex and required placement of multiple body stents or stent combinations for expansion of the entire narrowed area(s). A double-body stent was sufficient in only 4 patients. The others received three to five body stents or stents

in combination, with 1 patient receiving one four-body and two double-body stents. The majority of the stents usually expanded only to 10–12 mm; however, they continued to expand in the next few days. In 1 patient with a focal concentric narrowing, a tight stenosis milked the stent cranially. A second stent placed partially into the first one satisfactorily expanded the stenosis. Localiza-



**Fig. 4.** A 68-year-old man with severe superior vena cava syndrome due to bronchogenic carcinoma. He became symptomatic during his radiation treatment. After stent placement he remained asymptomatic for 6 months until his death. **A** Initial venogram reveals occlusion of the superior vena cava, thrombus in its main tributaries, and filling of collaterals. **B** Follow-up venogram after 48 h of local infusion of urokinase shows lysis of thrombi and severe stenosis of the distal portion of the superior vena cava. **C** Superior vena cavagram immediately after placement of a double-body stent with a skirt shows good expansion of the stent and excellent flow in the superior vena cava. **D** Superior vena cavagram 2 months after stent placement reveals further expansion of the stent and improved flow.

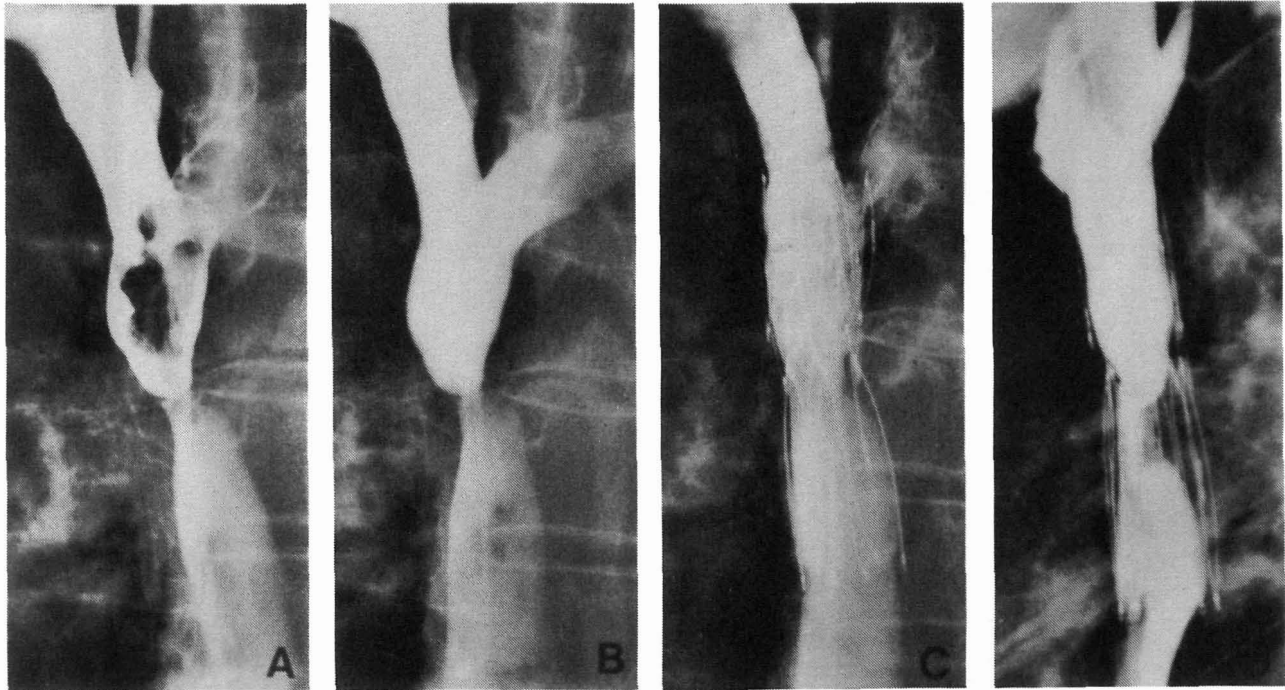
tion of the stent placement is detailed in Table 1. In all patients, a follow-up venogram was done after stent placement.

Patients were heparinized during the procedure and for 3–4 days afterwards, then they were switched to coumadin. The anticoagulation therapy continued for 2 or 3 months in patients with benign strictures and indefinitely in patients with malignant tumorous obstructions.

All patients were followed clinically at 1 month intervals (at least). A chest radiograph was done 1 month after stent placement to evaluate stent position and diameter. Follow-up venograms were performed in 4 asymptomatic patients 2 months after stent placement to evaluate the anatomy of the stented vessel. One of these patients also had a follow-up venogram 9 months after stent placement at the time of recurrent symptoms.



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**Fig. 5.** A 62-year-old man with severe superior vena cava syndrome secondary to mediastinal metastases of a colon carcinoma. The patient remained asymptomatic for 9 months when his syndrome recurred due to tumor ingrowth into the stent and secondary thrombosis. He died 1 month later. **A** Superior vena cavagram reveals severe focal stenosis with large thrombi in the superior vena cava, and left innominate vein. **B** Follow-up venogram after 24 h of local infusion of urokinase reveals lysis of all thrombi and persistent severe stenosis of the superior vena cava. **C** Follow-up cavagram 2 months after placement of a double-body stent shows complete stent expansion and excellent flow in the superior vena cava. **D** Superior vena cavagram 9 months after stent placement demonstrates a large defect at the posterior wall of the stent which intravascular biopsy proved to be ingrowing tumor.

**Table 1.** Localization of Stent Placement in 22 Patients  
SVC-superior vena cava, RI-right innominate vein, LI-left innominate vein, RSCL-right subclavian vein

Stent placement	No. of patients
SVC	5
SVC + RI	8
SVC + RI + RSCL	1
SVC + LI	3
SVC + RI + LI	5

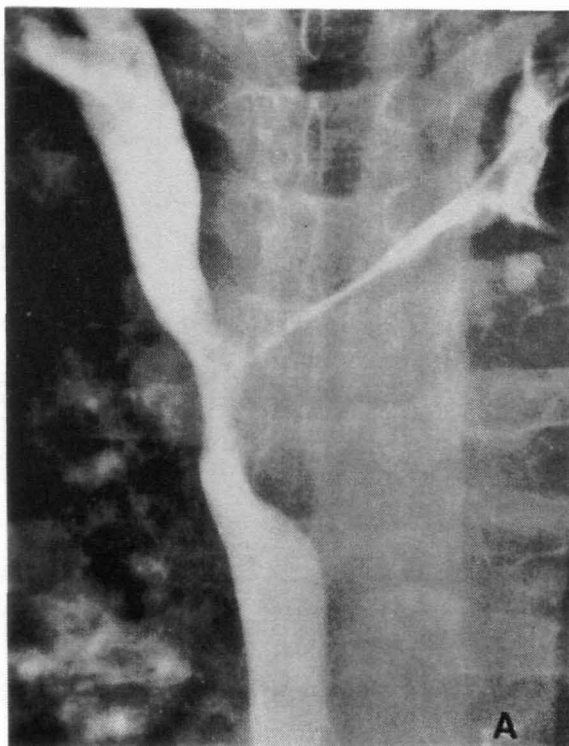
## Results

Stent placement proceeded smoothly and without complication in 21 patients. In 1 patient with simultaneous placement of stents in the SVC and both innominate veins, a prolonged procedure was complicated by thrombosis of the innominate vein stents, even with the patient fully heparinized. The patient received selective urokinase infusion for 6 h, which lysed the clots and opened a passageway through the stent. A few patients felt chest pain during balloon inflation of the stenotic region and after stent placement. The pain, however, disappeared within a few minutes and did not recur.

In all patients, stent placement resulted in relief of their symptoms of SVCS. Patients with a cyanotic

face usually returned to normal complexion shortly after stent placement, and facial edema and headaches regressed by the next day. Truncal and arm edema resolved in 2–3 days. Hoarseness persisted.

On 1 month follow-up chest radiographs, all stents remained in place and further expanded another 2–4 mm, almost to their full diameter. Follow-up venograms 2 months after stent placement showed excellent patency in all 4 patients studied (Figs. 2–5). In 1 patient with postradiation fibrosis, the outline of the stent lumen was smooth without intimal hyperplasia (Fig. 2). In 2 patients with malignant lesions, small filling defects were seen inside the stent, which we attributed to tumor ingrowth or intimal hyperplasia (Fig. 3). The patient with SVCS recurrence 9 months after stent placement, which developed shortly after he discontinued coumadin treatment, showed complete thrombosis of the stented SVC and both proximal innominate veins. After local urokinase infusion dissolved the thrombus, a venogram demonstrated gross filling defects



**Fig. 6.** A 64-year-old man with mediastinal carcinoma and neck, face, and left arm swelling. Swelling completely resolved in 2 days after stent placement and the patient did not have any recurrence until his death 4 months later. **A** Initial venogram reveals severe stenosis of the left innominate vein and moderate stenosis of the proximal right innominate vein and upper superior vena cava. **B** Follow-up venogram immediately after placement of a three-body stent into the superior vena cava and right innominate vein and a four-body stent into the left innominate vein show good expansion of the stenotic veins.

in the stent lumen (Fig. 5D). Intravascular biopsy of the mass revealed tumor ingrowth.

Of the 2 patients with benign lesions, 1 died 11 months after stent placement of unrelated cause (stroke) without recurrence of her SVCS. The other patient was asymptomatic for 12 months after the procedure. Later he moved out of the state and has been lost to follow-up.

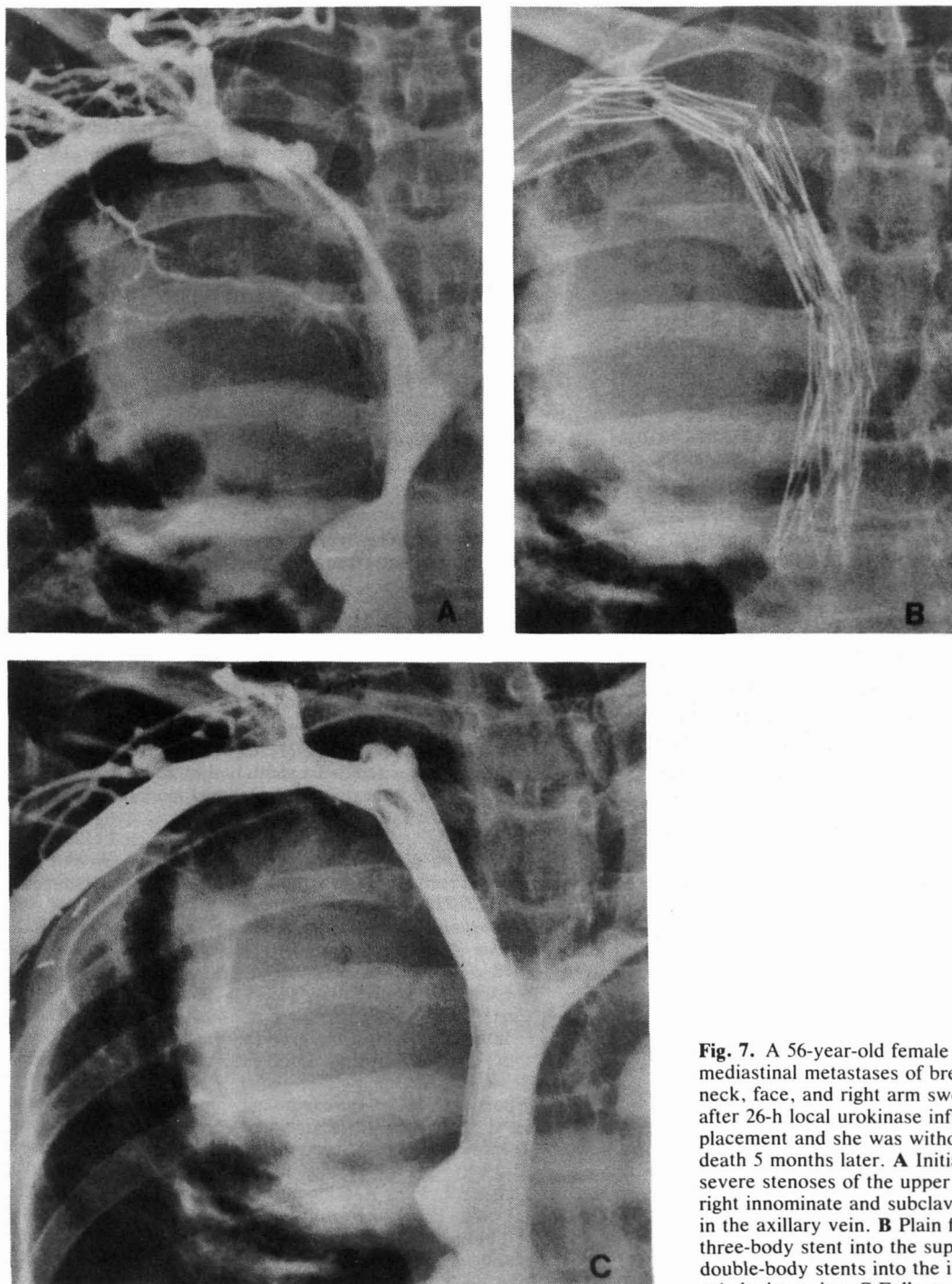
Seventeen patients with malignant lesions died from 1 month to 11 months after stent placement (median 5.2 months) from progression of their disease. Sixteen died without recurrence of their SVCS. In the patient who had SVCS recurrence 9 months after stent placement due to tumor ingrowth and thrombosis, placement of another stent was considered, but was not performed because of the patient's poor general condition; the patient died 1 month later. Three patients are alive and without

SVCS recurrence. One patient with mediastinal metastases is alive 16 months after stent placement and 2 recent patients are alive 1 and 2 months, respectively.

### Discussion

Including our series, expandable metallic stents have been used for treatment of SVCS in more than 70 patients. Z-stents were used in the majority of the patients [1–10]. Balloon expandable Palmaz stents and self-expandable Wallstents were used in only a few cases [5, 11–13]. In the majority of the reported cases SVCS was caused by malignancies, mainly lung cancer extending into the mediastinum. Stents were used in only a few patients to treat benign lesions, usually—including in 2 of our patients—after unsuccessful treatment by balloon angioplasty [5]. Stent placement resulted in complete relief of the SVCS symptoms in the majority of patients shortly after the procedure. The relief was of long-term duration, usually for the rest of the patient's life. In our patients with malignancies, relief ranged from 1 to 16 months. In only a few reported patients, SVCS recurred due to secondary thrombosis and/or tumor ingrowth inside the stent. Secondary thrombosis was successfully treated with selective thrombolysis [11] (and in one of our pa-

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**Fig. 7.** A 56-year-old female with lung and mediastinal metastases of breast carcinoma and neck, face, and right arm swelling. Swelling resolved after 26-h local urokinase infusion and stent placement and she was without recurrence until her death 5 months later. **A** Initial venogram reveals severe stenoses of the upper superior vena cava, right innominate and subclavian veins, and thrombi in the axillary vein. **B** Plain film after placement of a three-body stent into the superior vena cava and two double-body stents into the innominate and subclavian veins. **C** Follow-up venogram demonstrates good expansion of the stenotic veins.

tients). Tumor ingrowth could be managed, at least temporarily, by placing another stent. An optimal solution for this problem should be a stent with a cover strong enough to prevent tumor ingrowth into the stent.

Benign stenoses have a better prognosis for long-term patency than the malignant ones. The recur-

rence-free follow-up period in our 2 patients with benign SVC obstruction is only 11 and 12 months, respectively. Our more than 2 years recurrence-free follow-up in 4 patients with benign stenoses of the inferior vena cava and iliac veins is very promising in this context [10].

The best lesions for stent placement are SVC

stenoses 2 or more cm long and not completely obstructing the lumen, so that the balloon dilation catheter and introductory sheath can be passed freely through the stenosis. In a short, tight stenosis it is difficult at times to place the stent exactly across its center because the stent is pushed ("milked") by the stenosis, usually in a cranial direction. A multiple-body stent should be used in this case because it is more stable. A removable suture line looped around the monofilament line connecting the stent legs and held from the outside could also be used to hold the stent in place during its deployment and during its expansion. When a stenosis milked a stent in our patient, we simply put another stent inside the first one and achieved satisfactory expansion of the stenosis.

Stents can also be used in patients with complete occlusion of the SVC, particularly when the patients' recently accelerating symptoms indicate a superimposed thrombotic occlusion of the stenosed SVC. Fibrinolytic agents selectively infused directly into the clot can reopen the SVC for stent placement. The infusion in these patients usually has to be carried for a longer time. In one of our patients a 72-hr urokinase infusion was necessary to reopen the SVC lumen and to enable successful stent placement.

Stent placement into the SVC and right innominate vein is technically simple and easily accomplished. Placement of an additional stent into the left innominate vein is more difficult, particularly when its proximal portion has a short stenosis and the vein runs in a right angle to the SVC. The transjugular approach should be used in this case for the innominate vein stent placement. It is important that the left innominate veins stent be placed first, followed by the SVC stent. With this technique, a close contact between the SVC and innominate stents can be accomplished.

Stents remain in place. Stent migration was reported only in 1 case with use of an early model Z-stent, prior to the use of fixing barbs [3]. The stent migration into the right ventricle was without consequences. The Z-stents typically do not expand fully immediately after placement, usually to only about 50%–60% of their diameter. However, their expansion continues, and in our series 3–4 days after stent placement, the stents expanded to approximately 70%. On 1 month follow-up films, the stents were usually nearly fully expanded.

The published or presented reports did not go into detail on the use of anticoagulants. In our opinion, use of anticoagulation therapy is very important for long-term stent patency. We fully heparinized patients during and shortly after stent placement, then switched to coumadin. We feel it is essential

that all patients with malignancies and those with benign stenoses requiring fibrinolysis before or after stent placement remain on long-term anticoagulation. This is underlined by our experience in the patient with SVCS recurrence after 9 months. As long as he remained anticoagulated, the stent remained patent, even when ingrowing tumor narrowed its lumen. However, the stent clotted in a few days after coumadin was discontinued. Patients with simple benign stenoses are anticoagulated for 2–3 months prior to the time the stent is fully endothelialized [14]. When the follow-up venograms confirm good stent patency without formation of intimal hyperplasia, coumadin is discontinued.

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## References

1. Rösch J, Bedell JR, Putnam J, Antonovic R, Uchida B (1987) Gianturco expandable wire stents in the treatment of superior vena cava syndrome recurring after maximum-tolerance radiation. *Cancer* 60:1243–1246
2. Putnam JS, Uchida BT, Antonovic R, Rösch J (1988) Superior vena cava syndrome associated with massive thrombosis: Treatment with expandable wire stents. *Radiology* 167:727–728
3. Charnsangavej C, Carrasco CH, Wallace S, Wright KC, Ogawa K, Richli W, Gianturco C (1986) Stenosis of the vena cava: Preliminary assessment of treatment with expandable metallic stents. *Radiology* 161:295–298
4. Carrasco CH, Charnsangavej C, Wallace S, Wright KC, Gianturco C (1990) Gianturco stent in vena cava stenoses. 15th Annual Meeting Interventional Radiology, Miami Beach, FL, March 26–29
5. Wholey MH (1990) Interventional approach to superior vena caval syndromes. II International Congress Laser and Stent Therapy in Vascular Surgery, Scottsdale, Arizona, February 10–15
6. Yamada R, Yang R, Sonomura T, Tsuda M, Sato M, Rösch J, Uchida B (1990) Usefulness of expandable metallic stents in treatment for venous obstruction. 3rd Intl Symp of Interventional Radiology and New Vascular Imaging, Nara Japan, May 10–12
7. Sano A, Masada T, Murakami M, Kuroda Y, Huaang CL, Kitano T, Ichijima K (1990) Modified Gianturco expandable wire stents for SVC stenosis in a patient with lung cancer. 3rd Int Symp of Interventional Radiology and New Vascular Imaging, Nara, Japan, May 10–12
8. Castañeda-Zúñiga WR (1990) Clinical experience placing Gianturco vascular stents in the venous system. 2nd Congr Intl of Interventional Radiology in Cardiovascular Diseases, Toulouse, France, February 28–March 2
9. Fernando CC, Rösch J, Hunter DW, Yedlicka JW Jr., Castañeda-Zúñiga WR, Amplatz K (1992) Gianturco stents in the venous system. In: Castañeda-Zúñiga WR, Tadaworthy SM (eds) *Interventional Radiology*, 2nd ed. Williams & Wilkins, Baltimore

J. Rösch et al.: Superior Vena Cava Syndrome

10. Rösch J, Keller FS, Uchida BT, Barton RE (1992) Interventional treatment of large vein obstructions. In: Strandness DE, Jr., Van Breda A (eds) *Vascular Disease: Surgical and Interventional Therapy*. Churchill Livingstone, New York, in press
11. Zollikofer C, Antonucci F, Stuckmann G, Irving D, Salomonowitz E (1990) Self-expanding stents in veins, grafts and dialysis shunts: Long-term follow up. II Intl Course on Vascular-Interventional Radiology As A Therapeutic Alternative. Las Palmas-Canary Island, February 23-24
12. Elson JD, Becker GJ, Wholey MH, Ehrman KO (1991) Vena caval and central venous stenoses: Management with Palmaz balloon-expandable intraluminal stents. *JVIR* 2:215-223
13. Dondelinger RF, Goffette P, Kurdziel JC, Roche A (1991) Expandable metal stents for stenoses of the venae cavae and large veins. *Semin Interven Radiol* 8:252-263

## Long Term Results of Gianturco Z-stents in Treatment of Large Vein Obstructions

JOSEF RÖSCH, M.D.

**T**he modified Gianturco Z-stents with their leg connected with monofilament suture to limit stent diameter and to connect individual stents- the Gianturco-Rösch type Z-stents (GRZ stents) were used in 64 patients for treatment of large vein obstructions. The biliary GRZ stents, 10 and 12 mm in diameter (Cook, Inc.) were used for branches of IVC and SVC. The larger stents, 15 to 25 mm in diameter, used in most patients for the IVC and SVC, were handmade in our research laboratory. For a few recent IVC patients we used the Gianturco tracheobronchial stents 25 mm in diameter (Cook Inc.). The stents were 3 to 10 cm long and were placed by percutaneous femoral or internal jugular approach using 10 to 14 French Teflon introductory catheters. A 10 to 12 mm balloon catheter was introduced first and inflated inside the obstructive lesion. This was done more for delineation of the extent of the obstruction rather than for dilation, because in most patients obstruction relapsed after balloon deflation. In 20 patients with superimposed thrombosis local fibrinolysis was used prior to stent placement.

The patients were heparinized during the procedure. The majority of patients, except those with hemodialysis shunts, received continued anticoagulation therapy after stent placement. The anticoagulation continued for 2 months until the stent was fully endothelialized. After that it was discontinued in patients with benign structures and with high flow through the stent. Patients with malignancies and those requiring fibrinolysis before or after placement received long term anticoagulation.

Stent placement resulted in expansion of even tight obstructive lesions, establishment of normal blood flow and rapid regression of patients' congestive symptoms.

With SVC syndrome, the patients' cyanotic face returned to normal complexion shortly after stent placement, facial edema and headache regressed the next day, and truncal and upper extremities edema resolved in 2 to 3 days. Distal extremity swelling

and ascites in IVC obstructions usually resolved slower, 3 to 7 days after stenting. Stent placement also gave long term relief because expandable stent prevented recoil and recurrence of the obstruction in the majority of cases. Fifty-six (87%) of our patients have been without recurrence of their symptoms to the present time (2 to 48 months, mean 23.2 months) or until their death (8 days to 26 months, mean 8.5 months) after stent placement.

Obstruction in our 64 patients were related to malignant tumors in 46 patients, to benign lesions in 12 patients and to hemodialysis shunt in 6 patients.

### NEOPLASTIC VENOUS OBSTRUCTIONS

**T**he lung and mediastinal tumors caused obstruction of the SVC and its branches and severe SVC syndrome in 22 patients. Stents in these patients were placed in SVC but in many patients extended also into the innominate and subclavian veins. In 20 patients with metastatic liver hepatomegaly causing IVC stenosis and severe distal extremities edema and ascites, stents were placed into the narrowed intrahepatic IVC portion, and in one patient also in hepatic veins. Four patients with pelvic tumors and unilateral distal extremity edema had stents placed into the iliac veins.

Stent placement provided excellent palliation for all stented patients. Their congestive syndromes rapidly regressed and the majority (42 patients) did not have recurrence until their death or to the present time (from 8 days to 16 months, mean 6.8 months). Recurrence was observed in only 4 patients, at one, three, four or nine months, respectively, after stent placement, who discontinued anticoagulation therapy. Their stent exhibited tumor ingrowth complicated by secondary thrombotic occlusion which was dissolved with local thrombolysis.



## **BENIGN VENOUS OBSTRUCTION**

**I**n 12 patients with benign lesions, large vein obstruction was caused by post radiation fibrosis (4 patients), idiopathic mediastinal or retroperitoneal fibrosis (2 patients), indwelling catheter (1 patient), trauma (1 patient), cirrhosis (2 patients) and Budd-Chiari syndrome (2 patients). Stents were placed into the SVC, IVC and innominate subclavian, hepatic and iliac veins. Stent placement was the definitive treatment for patient with benign venous obstructions and resulted in long term palliation with complete regression of their symptoms. None of 12 patients had recurrence of the congestive symptoms to the present time or to their death. Six patients are still living from 8 to 48 months (mean 35.6) after stent placement. Six died from 9 to 26 months after stent placement secondary to their basic disease or of unrelated causes.

## **OBSTRUCTIONS RELATED TO DIALYSIS SHUNTS**

**S**ix of our patients had obstructions related to arteriovenous dialysis shunts with severe stenoses involving the innominate or subclavian vein. Stent placement extended life of the fistula but did not exhibit such a long patency as in patients with benign stenoses. Only 2 patients had normal diameter lumen 16 months after placement. In the other four patients, the stent lumen narrowed due to intimal hyperplasia, from 8 months to 14 months after stent placement. The obstructions were treated by an atherectomy, angioplasty, or by other stent placement.

## **COMPLICATIONS**

**E**arly in our series, when we did not use heparinization during the procedure, stents in two patients clotted during a prolonged placement. Their thrombosis was successfully treated by short (2 to 3 hours) local urokinase infusion; they remained open afterwards.

One patient with local fibrinolytic therapy prior to stent placement had a large neck hematoma at the area of catheter introduction.

## **CONCLUSION**

**T**he GRZ stents are effective devices for treatment of obstructions of larger veins, whether caused by

malignant tumors, benign processes or related to dialysis shunts. They are easy to place, alone or in combination, dilate venous obstruction by expanding to their given diameter, do not migrate, and exhibit long term patency.

Stent placement immediately results in a rapid resolution of patients' congestive syndromes. In patients with malignancies, stent placement is an excellent palliative treatment and gives long term relief in the majority of patients. In patients with benign lesions, stent placement, according to the present experience, gives promise to be a definitive treatment of large vein obstructions. Patients with obstruction related to their arteriovenous dialysis shunt can benefit from stent placement because it can significantly prolong the life of the shunt. Local fibrinolysis is an important adjunct therapy prior to stent placement in patients with superimposed thrombosis.

## Technical Evolution of Tips

JOSEF RÖSCH, M.D.

**E**arly stages, technical evolution of TIPS was focused mainly on finding ways for a consistent and safe transjugular puncture of the portal vein and for establishing a well functioning shunt. Recently TIPS development has been focused on finding techniques for keeping the established shunt open and functioning well on a long term basis.

A suitable transjugular liver puncture needle and delineation of the targeted portal vein are the primary components of a consistent and safe entrance into the portal vein. The Colapinto transjugular liver biopsy needle was used for the first TIPS patients and is still preferred by some, particularly in combination with complementary catheters and guide wires (Ring set, Cook Inc.). To avoid potential liver damage with multiple puncture attempts, a small coaxial puncture needle system was introduced (Cope, Hawkins). We prefer a coaxial catheter needle set (RUPS-100, Cook, Inc.) which was less traumatic and easier to use during our extensive animal TIPS work. This system has been simple and safe and provides excellent results also in our clinical work. The combination of the 10 Fr sheath, which has a metal marker at its end, a 10 Fr Teflon catheter and a metal cannula gives the set rigidity with good torque control and enables one to select a suitable site for liver puncture. The puncture is done with a .038 inch diameter sharp point needle inside a well tapered 5 Fr Teflon catheter and is relatively atraumatic. In our 160 TIPS patients we did not have any complications related to the liver puncture even when we accidentally entered the hepatic artery, bile ducts, gallbladder or peritoneal cavity.

For delineation of the portal vein targeted for puncture, transhepatic portal vein catheterization with introduction of a wire basket was used in a few early patients, but this was abandoned because of serious complications. Several other techniques have been suggested to facilitate portal vein puncture including sonographic placement of platinum microcoils adjacent to the intended site of portal vein entry (Harman) or of a platinum tip guidewire into the right portal vein via a subxyphoid puncture of the left portal vein (Rivera,

Teitelbaum) or percutaneous catheterization of the paraumbilical vein (Wenz). Other prefers to perform the portal vein puncture completely under ultrasonographic guidance (Richter). We puncture the portal vein under fluoroscopic control based on its expected location. In our early experience, we defined anatomy of the portal and hepatic veins by arterial portography and hepatic venography in AP and occasionally also in lateral projection. After gaining more experience with anatomy of both systems, we now perform a free and wedged hepatic venography only. With a balloon occluding a medium size hepatic vein, a satisfactory retrograde visualization of the portal vein is usually obtained. Using this approach we were able to perform TIPS in an average time of 60 minutes. Our only failure was in a patient with chronic occlusion of the portal vein extending deep into its intrahepatic branches.

Selection of a suitable expandable stent and its proper placement are important factor in establishment of a well functioning shunt. Most of the available stents are suitable for TIPS but have their pros and cons and range of their use. The self-expandable Wallstent (Schneider, Inc.) is the most user friendly stent with the widest range of applications for TIPS. It has a low profile catheter, and because of its flexibility and good expansible force, it can be used in all circumstances, whether for a centrally or peripherally located shunts. It also conforms well even to a sharp curve in the hepatic or portal vein. The Wallstent, however, also has disadvantages, mainly its lower opacity, less predictable placement because it shortens during placement and sometimes continues to shorten after placement. The balloon expandable Strecker stent (Boston Scientific) which was recently introduced for TIPS, has mechanical properties similar to Wallstent but has better opacity (Maynar). More experience, however, will be needed for its detailed evaluation. The balloon expandable Palmaz stent (Johnson & Johnson) and self-expandable Z-stent (the biliary GRZ stent, Cook, Inc.) are rigid or exhibit only slight flexibility (GRZ stent) and are suitable only for centrally placed shunts. The Palmaz stent can be expanded to a desired diameter for optimal shunting



and its placement is predictable with only minor shortening. Its opacity is less than optimal. The GRZ is opaque and easy to follow on fluoroscopy, and its placement is predictable because it does not shorten after delivery. Its incomplete expansion of about 2 mm less than the original lumen, after being pushed through the introductory sheath, is of certain advantage. When needed, it can be expanded to the original size by balloon catheter. We found that only the 12 mm diameter GRZ stent is suitable for TIPS. Need for introduction of 10 Fr delivery sheath into the portal vein and uneven expansible force are disadvantages of the GRZ stent. When a connection between the individual stent bodies is placed at the level of portal vein, the site of maximum resistance, the stent sometimes does not expand completely or can kink. If cost is taken into consideration, the GRZ stent is at the lower and the Palmaz stent at the higher range of the scale, particularly because of need for more than one Palmaz stent for TIPS in the majority of cases. The Wallstent and Strecker stent are in the middle of this scale.

For proper placement the stent has to cover the entire liver parenchymal tract and, in our opinion, it should protrude about 2 to 3 cm into both the portal and hepatic veins, and its lumen should be oriented along the lumen of the veins. Direct contact of the stent ends against the vein wall should be avoided, because resulting continuous irritation leads to higher occurrence of intimal hyperplasia, particularly in the hepatic vein.

World wide experience shows that TIPS can be done with consistency and safety and is effective in decreasing portal hypertension and in controlling its complications, particularly bleeding and ascites. It controls active bleeding practically in all cases and prevents hemorrhage recurrence in 70 to 90% of patients. As for ascites, TIPS controls or significantly decreases ascites, except in patients with advanced liver failure. TIPS has become an important bridge to liver transplantation, giving patients with acute bleeding or massive ascites sufficient time to be stabilized and prepared for surgery.

Shunt maintenance, however, is necessary for long term patency and adequate function of TIPS. Follow up ultrasonographic and venographic studies reveal progressive shunt stenoses in a high percentage of patients, particularly in the first year. Exuberant formation of pseudointimal tissue in the parenchymal tract and intimal hypertrophy in the draining vein are causes of stenoses. Pseudointimal

hypertrophy in the shunt occurs predominantly with use of the Wallstent, while hepatic vein intimal hypertrophy is more common with the Palmaz and Z-stents. Clinically some stenoses lead to bleeding recurrence. The majority of shunt stenoses diagnosed on our routine follow up portograms, however, have been asymptomatic and probably detected early in their development. We have intervened on stenoses greater than 50% of the stent diameter with a portosystemic gradient higher than 15 mm Hg. Follow up venographic studies of our 40 patients from 6 to 40 months after TIPS showed occurrence of stenoses at some time in 85% of the patients. All responded well to balloon dilation or new stent placement with an overall 98% secondary shunt patency. In some patients only one intervention was sufficient to keep the shunt well open. However, in the majority of patients, however, repeated interventions every 6 or 12 months were necessary in the first two years after TIPS. Repeated balloon dilations seemed to stabilize hypertrophic tissue formation in our patients.

The future TIPS evolution should focus on prevention of stenoses. Changes in stent construction, with adding a partial membrane cover or stent coating with drugs, early local treatment of the established shunt or new treatment techniques of shunt stenoses should be explored for prevention and treatment of the shunt stenoses. Further expansion of TIPS indications, particularly as an alternative to elective portosystemic surgical shunt in patients with well preserved liver function will mainly depend on our ability to achieve long term shunt patency without multiple secondary interventions.

## Tips: Results

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### PURPOSE

To establish the safety and long-term results of the use of Wallstents and Nitinol-Strecker stents for TIPS and to document the incidence of stent stenosis or occlusion by venographic study.

### MATERIAL AND METHODS

During the last two years, a total of 58 patients underwent TIPS procedure at our Institution following precise indications: recurrent bleeding (27), refractory ascites (9), waiting for liver transplant (13), randomized trial (TIPS versus sclerotherapy) (9). Eleven patients were Child's A, 26 were Child's B, 21 were Child's C. In total, 91 stents were used: 54 Wallstents, 24 Nitinol-Strecker stents, and 13 Palmaz stents, with an average of 1.5 stents per patient.

### TECHNIQUE

**a. Venous approach:** right internal jugular (50); left internal jugular (7); external jugular (1).

**b. Hepatic vein:** the shunts were performed from the right hepatic vein in 46 cases, and from the median hepatic vein in 12 cases.

**c. Portal vein:** the puncture of the vein was in the right portal branch in 21 cases and in the left portal branch and bifurcation in 27 cases.

**d.** All the intraparenchymal tracts were dilated with a 10-mm balloon and kept open with metallic stents.

### RESULTS

The procedure was technically successful in all patients (100%).

In 1-24 months follow-up after shunt creation, 38 patients were alive (65.5%), 14 are dead (24.2%), six are transplanted (10.3%). Variceal bleeding recurred in five patients (8.7%). Temporary encephalopathy, after shunting, occurred in 19 patients (33%). Eight patients improved ascites signs: only one patient unchanged. Shunt stenosis and occlusion developed in 19 patients (32.7%): 13 were stenosis (either stent or hepatic vein) and six were stent occlusion. Five patients had partial mural thrombosis and five had partial portal thrombosis. Among stent occlusions, four were Wallstent and one was Nitinol-Strecker.

### CONCLUSIONS

Nitinol-Strecker stents and Wallstents can be used to perform TIPS and are effective in reducing the portosystemic gradient. Both types of stents are quite similar with a major advantage for Wallstent because of the availability in different sizes and measures, whereas Nitinol-Strecker stent is only 6 cm in length and 10 mm in diameter. The incidence of obstruction or stenosis is almost identical.

## Midterm Results of Renal Artery Stenting

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**Abstract.** Percutaneous transluminal angioplasty (PTA) has become the treatment of choice for major renal artery stenosis. Nonetheless, about 10% of renal artery stenoses cannot be properly dilated, and among the patients successfully dilated, 10%–15% had a recurrence. Renal artery stenting was used in 21 patients in cases of insufficient results after PTA: persisting significant stenosis after a primary or several PTAs (15 cases), recurrences (9 cases). Follow-up in all patients was from 12 months to 4 years. Implantation was performed without any problems but the low radioopacity of the stent makes placement difficult in obese patients, particularly for ostial lesions. There was no major complication except occlusion of a segmental branch of the renal artery in 1 case. Radiological controls have shown a preserved patency in all cases except 2, which present restenosis inside the stent by intimal hyperplasia. A significant clinical improvement was obtained in 90% of cases. These results suggest that the endovascular prosthesis represents an important adjunct to renal PTA.

**Key words:** Vascular endoprostheses—Stents—Renovascular hypertension—Renal artery stenosis—Renal angioplasty

Since Gruntzig's pioneering work on renal percutaneous transluminal angioplasty (PTA) [1], numerous publications have attested to the efficacy of this method, which has become the treatment of choice for significant renal artery stenoses [2–6]. Nonetheless, this technique has its limitations in that certain stenoses are resistant to dilatation and others restenose some weeks after dilatation [7, 8].

Percutaneous endoprostheses have recently

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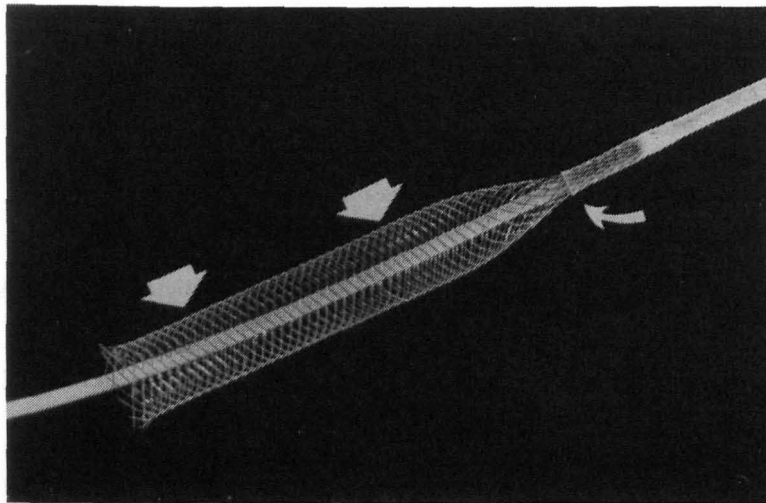
been proposed as a potential means of improving angioplasty results [9, 10]. We report here the preliminary experience of 17 implantations in renal arteries of 16 patients with a follow-up greater than 6 months.

### Materials and Methods

This study was undertaken using Wallstent endovascular prostheses of different dimensions. Experimental studies have already demonstrated the excellent tolerance, adaptation to the vasculature, and the endothelial covering of the device, integrating it totally into the vessel wall within 3–4 weeks [11].

The Wallstent endoprosthesis is composed of 20 stainless steel monofilaments 80  $\mu\text{m}$  in diameter. These monofilaments are woven into a cylindrical braided structure, the intersections being free to pivot over each other. This latter characteristic combined with the spring qualities of the stainless steel provides an elastic structure which can be stretched into a small diameter format that spontaneously recoils to its unconstrained large diameter format when released from the tip of its specially designed delivery catheter (Fig. 1). This coaxial device is designed for passage over the guidewire used for conventional PTA procedures. Even with the constrained prosthesis mounted on the catheter tip, this part of the instrument remains flexible, allowing easy access to the target zone. Prosthesis release is effected by the progressive retraction of the rolling membrane that constrains and covers the stent. Low hydraulic pressure (4 atm) applied to the annular space between the coaxial catheters permits the operator to displace the slide mechanism which controls membrane retraction. By removing the hydraulic pressure, the operator can stop the process of deployment at any time, even if the slide mechanism is accidentally displaced. This enables angiographic check of the part already deployed, and adjustment by pulling back the device or even extracting the prosthesis through the sheath if deemed necessary. Moreover, all these procedures can be performed without obstructing arterial flow.

In all 16 patients, conventional PTA via the retrograde femoral approach was performed with a balloon catheter and under local anesthesia. A moderate overdilation with a pressure of 10 atm was used according to the technique described by Sos et al. [3]. Leaving the guidewire in place after the procedure, the arterial pressure and the radiographs were carefully assessed. Arterial opacification was undertaken with an aortic catheter introduced via the contralateral femoral artery or via the same sheath. This opacification is an indispensable aid to positioning the stent with respect to the renal artery ostium. The 7F delivery catheter was always passed via a valved introducer and over the 0.035" (0.88 mm) guidewire. Implantation was made under fluoroscopy, the



**Fig. 1.** Macroscopic view of the delivery catheter during release of the stent. The distal part is released and has assumed its normal diameter (arrows). The proximal part is stretched on the catheter and is retained by a membrane, the withdrawal of which allows the release (curved arrow).

**Table 1.** Clinical data and general results

Cases	Age	Sex	Hypertension (HT)	Renal failure (RF)	Anat type	Post-PTA recurrence	Post-PTA bad result	Follow-up	Late anat result	Clinical result HT	Clinical result RF
1	49	M	+	+	Post op		+	12 M	+	I	I
2	62	F	+	+	ATH	+		34 M	+	I	U
3	68	F	+	+	ATH	+		36 M	+	I	I
4	41	F	+		FMD	+		36 M	+	C	
5	40	F	+		FMD	+		24 M	+	C	
6	60	M	+		ATH	+		24 M	Stenosis	I	
7	47	F	+		TAKA	+		24 M	+	I	
8	62	M	+	+	ATH		+	20 M	+	I	I
9	63	F	+		ATH	+		18 M	+	C	
10	68	F	+		ATH	+		3 M	Bad position	F	
11	Same patient							16 M	+	I	
12	66	M	+	+	ATH		+	18 M	+	I	U
13	56	M	+		ATH	+		9 M	+	C	
14	62	F	+		ATH		+	7 M	+	I	
15	60	M	+		ATH	+		8 M	+	I	
16	46	M	+		ATH		+	6 M	+	C	
17	22	M	+	+	post op (RT)	+	+	6 M	Partial thrombosis	F	F

Abbreviations: U = unchanged, I = improvement, C = cure, F = failure, Taka = Takayashu, FMD = fibro muscular dysplasia, ATH = atheroma, RT = renal transplantation

stent being placed across the lesion with the aid of cutaneous radioopaque markers. The implanted prostheses were 5–6 mm in diameter and 20–30 mm long. In general, unconstrained stent diameters were chosen to be 15% greater than that of the artery upstream of the stenosis.

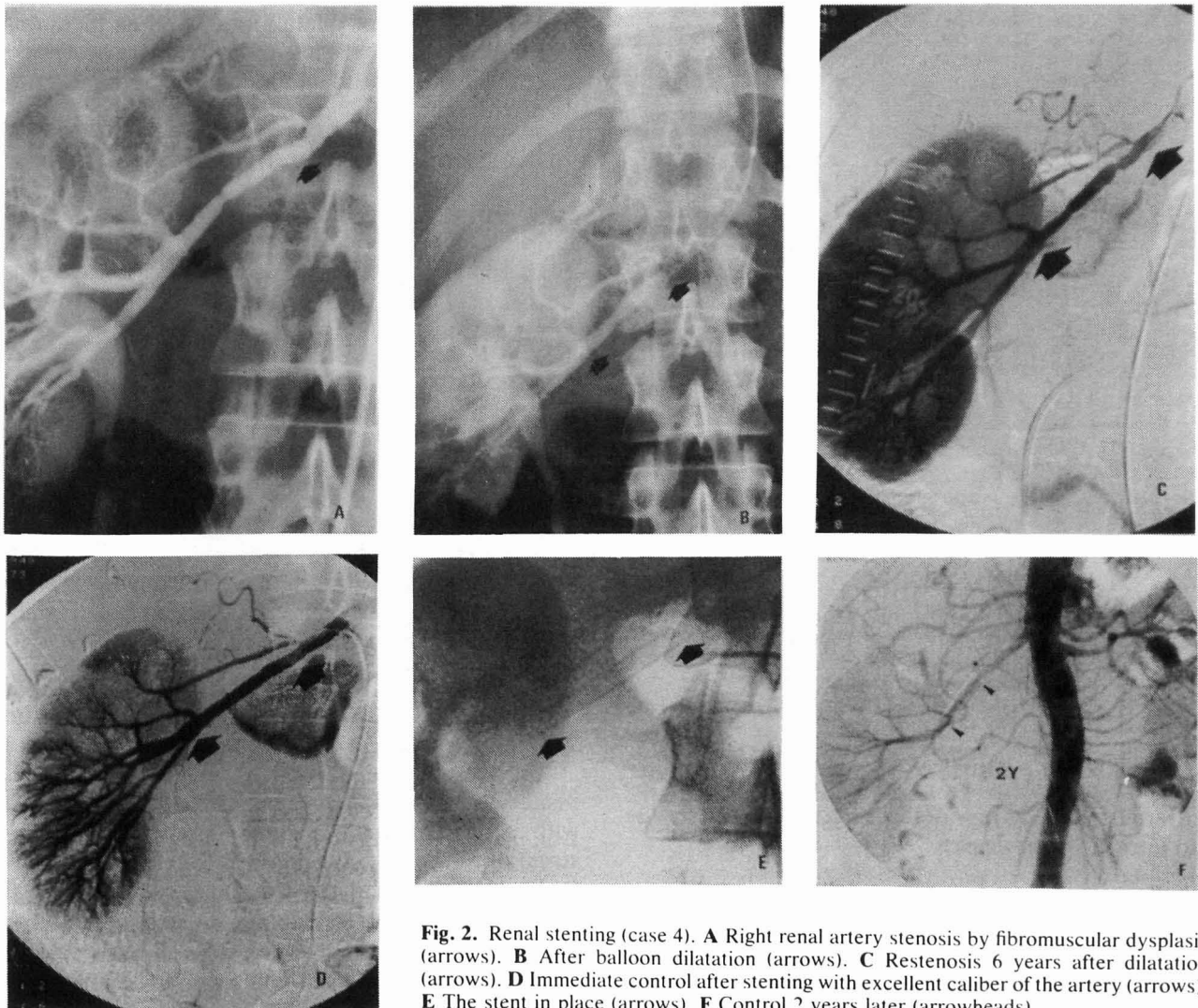
Radiographic and pressure studies were repeated after stent placement, and the arterial pressure and pulse were monitored throughout the examination. Given the important hemodynamic disturbances concomitant to the relief of renal artery stenosis, we maintained a rigorous pharmacologic protocol, combining 1) platelet antiaggregating agents, 24 h preimplant and for 6 months postoperatively; acetyl salicylic acid, 300 mg/day and dipyridamol 3 × 75 mg/day. 2) Injection of 5,000 IU heparin and 500 ml dextran during the procedure. 3) Intravenous heparin infusion for 24 h (keeping partial thromboplastin time at twice control)

followed by oral anticoagulation with Acenocoumarol for 2 weeks with prothrombin adjusted at 2.5 times control.

Patient follow-up was based on clinical, laboratory, and angiographic studies. All patients underwent intravenous digital subtraction angiography at 1 month and renal arteriography at 6 months. All patients were given a prosthesis and 7 of these were followed up for 6–36 months (Table 1). One patient had a second prosthesis implanted 2 months after the first one which was badly positioned initially (case 10, 11).

Clinical indications for stent placement were recurrent hypertension after previous angioplasty in 11 cases. In 6 cases the indication was improvement of renal function after recurrence of stenosis in a solitary functional kidney, stenosis of an aorto-renal bypass of a solitary kidney, transplant artery stenosis and uni- or bilateral stenosis with hypertension associated with renal failure.

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**Fig. 2.** Renal stenting (case 4). **A** Right renal artery stenosis by fibromuscular dysplasia (arrows). **B** After balloon dilatation (arrows). **C** Restenosis 6 years after dilatation (arrows). **D** Immediate control after stenting with excellent caliber of the artery (arrows). **E** The stent in place (arrows). **F** Control 2 years later (arrowheads).

Pathologically, the cause of the stenosis was atheromatous in 10 cases, fibrous dysplasia in 2 cases, Takayasu disease in one case, and postoperative stenosis in 2 cases. In 3 of the cases, the stenosis was located at the ostium.

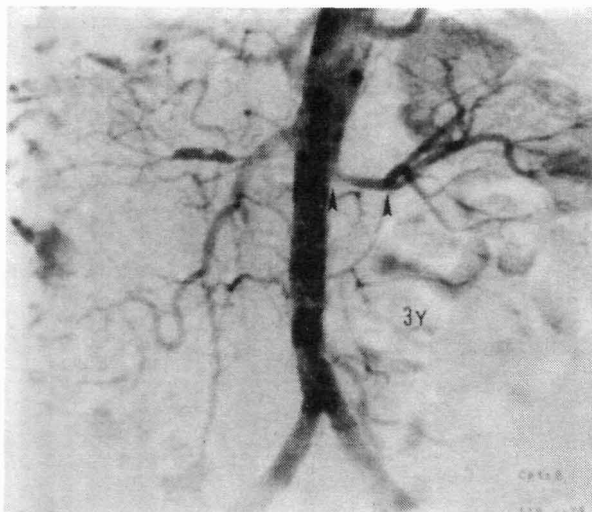
In all cases, the problem was that of incomplete result of dilatation, as defined by the criteria of Martin et al. [2], Sos et al., [3] and Kuhlmann et al. [5]: a residual stenosis of more than 30% associated with an increased pressure gradient. In 10 cases, there was a recurrence after angioplasty and in 1 of these cases this was a third recurrence. A new attempt at angioplasty gave only an incomplete result: persistence of a residual stenosis between 30% and 50% and a pressure gradient greater than 20 mm Hg. In 6 cases, the prosthesis was inserted after the first angioplasty attempt was inadequate.

### Results (Figs. 2, 3, and 4)

#### *Technical and Anatomic Results (Table 1)*

Implantation was successful in all cases and relatively easy to perform. The only problem was the

low radioopacity of the prosthesis, rendering the exact positioning of its proximal end with respect to the renal artery ostium somewhat difficult. In 1 patient, the prosthesis was placed too distally, not entirely covering the stenotic zone and leaving a moderate residual stenosis of 30% at the level of the ostium. The recurrence of hypertension necessitated a second implantation. In all the other cases the normal caliber was reestablished with perfect regularity of the vessel wall and lumen. Pressure gradients across the lesion were found to have disappeared in nearly all cases, with a marked improvement compared with the angioplasty results. One patient complained of lumbar pain for a few days associated with a small perirenal hematoma secondary to catheter manipulation during PTA. Another patient (case 17) had thrombosis of a branch of the renal artery of his graft.



**Fig. 3.** Renal stenting-late results (case 3). Follow-up aortogram at 3 years. Arrowheads indicate stent position.

Consecutive angiographic studies were undertaken between 1 month and 3 years and demonstrated preservation of artery patency in the stenotic region. There was no prosthesis migrations and all collaterals crossed by the prosthesis remained patent except in 1 patient (case 17).

In 1 patient (case 6), a reduction of 50% of the arterial diameter was noted within the prosthesis due to intimal hyperplasia.

### Clinical Results

At present, all the patients but one are normotensive, 5 without treatment and 10 with moderate doses of calcium antagonists (Nicardipine 40–80 mg; Verapamil 240 mg). Six patients had renal failure on presentation. We observed functional improvement in 3 cases and stabilization in 2 cases after a 1-year follow-up. The implantation in the transplanted patient is considered a failure (persistent hypertension and worsening renal failure).

### Discussion

Numerous studies on large series of patients have confirmed the efficacy of renal PTA [2–6]. Its simplicity and low morbidity with practically similar results to surgery, often make it the method of choice. The utility of radiologic interventional therapy continues to be debated only by Brawn and Ramsey [12] who consider that the clinical benefit does not justify angioplasty.

Renal angioplasty nevertheless has its limita-

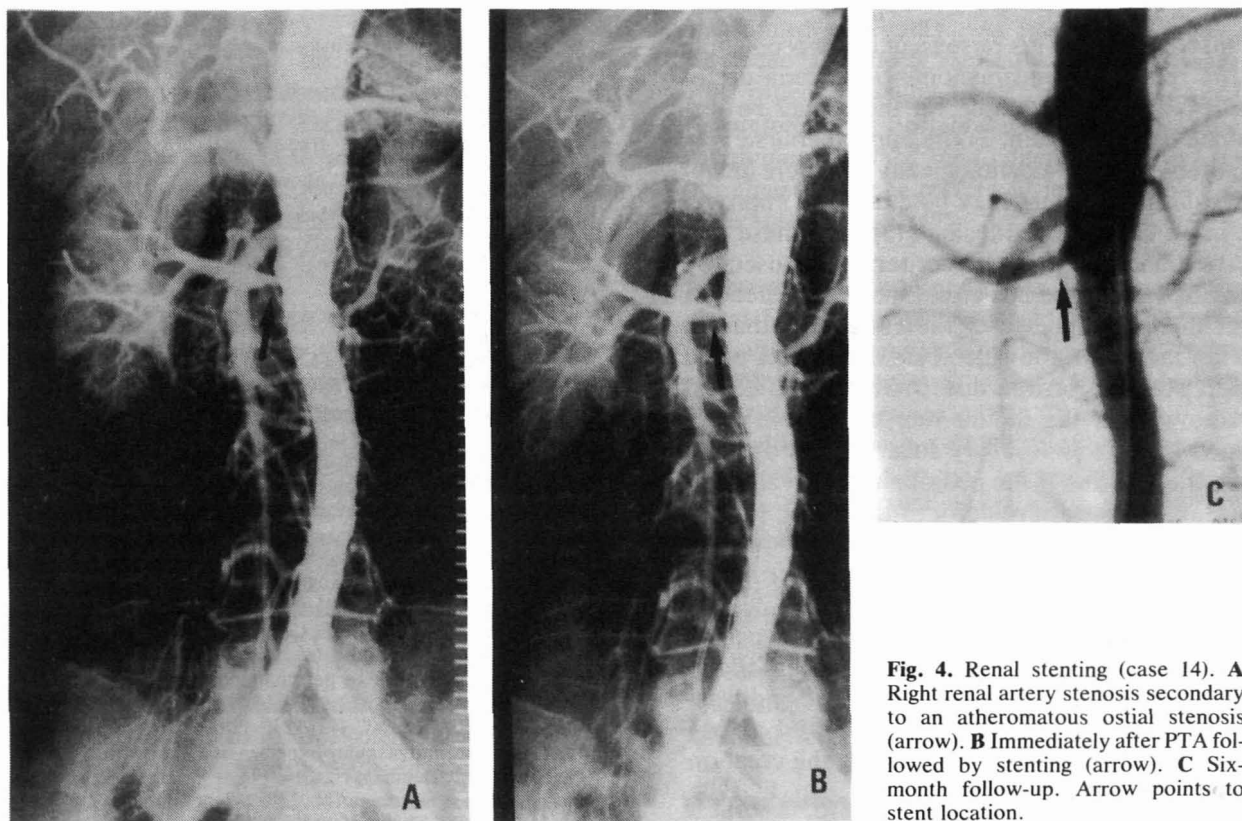
tions. The majority of major clinical studies report immediate failure of dilatation as well as medium-term restenoses. The immediate failures correspond to an unsatisfactory dilatation, with a residual stenosis greater than 50% associated with an elevated residual transstenotic pressure gradient [5]. These failures are encountered preferentially in certain etiologies: ostial atheromatous lesions [7], fibromuscular dysplasia lesions with a marked elastic component [13], and stenoses associated with Takayasu or von Recklinghausen's diseases [14–16]. Post-PTA restenoses are encountered in 5%–20% of cases. The conclusions of many studies done on restenosis after coronary PTA can be applied to the renal arteries. Experimental studies have led to the hypotheses of Faxon et al. [17] and Essed et al. [18] that restenosis has its origin in the parietal trauma induced in the dilated artery. Dilatation leads to a phenomenon of fissuring and partial dehiscence of the plaque with exposure of the media. The parietal irregularities are then filled by thrombus and fibrous tissue proliferation, culminating in healing of the lesion and smoothing of the arterial lumen. The proliferation can, however, extend to hyperplasia and thus to restenosis. The extent of this hyperplastic scarring appears to be directly related to the severity of the parietal injury engendered by the dilatation. Such restenoses are much more frequent if the residual stenosis is associated with elements of parietal dissection and with persistent hemodynamic disturbance or obstruction. The logic of using an autoexpandable scaffolding device to seal mural fissures, reduce residual stenoses, eliminate dissections, as well as minimizing hemodynamic disturbances thus appears clear.

Experimental studies using the Wallstent endoprosthesis [11] or with other models of endoprosthesis [9, 19] have shown excellent tolerance of these materials by the arterial wall. The endoprosthesis is rapidly covered with fibrin and then with endothelium; in a few weeks the stent is incorporated into the wall and covered by a hyperplastic intima the growth of which stabilizes at around 3 months. Experimental studies undertaken in the atheromatous rabbit model with the Wallstent showed that the device was not the source of trauma sufficient to stimulate atheroma proliferation. On the contrary, when placed at sites of atheroma previously induced by balloon trauma and hypercholesterol diet, the stent induced a fibrous proliferation covering and apparently protecting the arterial wall from further lipid infiltration [20]. Despite this remarkably interesting result, its extrapolation to human clinical application remains tenuous and one must be guarded in its interpretation.

The first clinical implantations reported were those in coronary and peripheral arteries [10]. Other



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**Fig. 4.** Renal stenting (case 14). **A** Right renal artery stenosis secondary to an atheromatous ostial stenosis (arrow). **B** Immediately after PTA followed by stenting (arrow). **C** Six-month follow-up. Arrow points to stent location.

reports concerned mainly iliac arteries [21] and femoral arteries [22].

The implantation of the Wallstent endoprostheses in renal arteries did not reveal any particular problem in this preliminary series. The diameter of the stents was chosen to be 15% greater than that of the artery so that the stent exerted a permanent radial force, preventing stent migration and dilating the residual stenosis. The only difficulty encountered was that of poor stent radioopacity, making implantation difficult in obese subjects, particularly if the prosthesis was close to the ostium. In 2 such patients, the prosthesis was placed too distally without totally covering the lesion; the need therefore, for a second aortic catheter in order to visualize the aorta and renal ostium is imperative. It is always possible, should radiographic study demonstrate malposition, to place a second prosthesis if necessary.

Experience obtained from our work in the femoropopliteal arteries highlighted the need for an optimal anticoagulant therapy and careful postoperative care. Early thromboses had occurred in 15% of our femoropopliteal series leading to energetic anticoagulation and the use of the drug regimen described above [22]. This resolved the problem of early femo-

ral occlusion, and there were no such occlusions in this series of renal implantations. Femoral restenoses after prosthesis implantation in our series occurred in 12% of cases and were difficult to predict [22]. Follow-up studies in our renal series have shown only 1 case of restenosis due to hyperplasia within the prosthesis. However, we plan long-term follow-up in these patients and define precise criteria for patient selection.

In general, the indications for renal PTA are identical to those for stent implantation, the prosthesis merely representing a supplementary technique aimed at improving the result. Restenosis after PTA appears to be the indication of choice. Although good results can undeniably be obtained from a second dilatation in many cases, the benefit does not appear to be greater than that of the first [4]. The implantation of a prosthesis does not seem necessary if the result of redilatation appears satisfactory, but on the other hand, if there are clear radiologic and manometric signs of residual stenosis, stent implantation is justified. This is particularly true for lesions with a high risk of restenosis, that is, ostial stenosis or stenosis secondary to von Recklinghausen's disease.

The question of prosthesis implantation after a

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first unsatisfactory PTA raises a delicate problem in that the radiologic and manometric criteria are of limited value. For example, 1) there may be discordance between them, 2) the clinical course may be favorable despite a radiologically mediocre immediate result, or 3) there may be a delayed improvement in an underdilated lesion [13]. For these reasons we maintain that the criteria proposed by Kuhlman et al. [5] and Sos et al. [3] (residual stenosis greater than 50%, pressure gradient greater than 20 mm Hg) should be the rule. This discussion could also apply to ostial lesions due to aortorenal plaques. It is known that the failure rate of PTA in these lesions is high (over 70%) [7]. Inserting a prosthesis may be the solution if the positioning is precise and if the stent does not project into the aortic lumen. This approach would be justifiable, given that the results of surgery are not significantly better in this type of patients [23].

Although preliminary, our results appear encouraging. A larger series of patients with longer follow-up is necessary, but in our experience the endovascular prosthesis can already be considered an important adjunct to renal PTA, allowing the creation of a true endoluminal bypass.

## References

1. Gruntzig A, Kuhlmann V, Vetter W, Lutolf U, Meier B, Siegenthaler W (1978) Treatment of renovascular hypertension with percutaneous transluminal dilatation of renal artery stenosis. *Lancet* 1:801-802
2. Martin LG, Casarella WJ, Alspaugh JP, Chuang UP (1986) Renal artery angioplasty: Increased technical success and decreased complication in the second 100 patients. *Radiology* 159:631-634
3. Sos T, Pickering TG, Sniderman DPK, Saddekni S, Case DB, Saline MF, Vaughan ED, Laragh JH (1983) Percutaneous transluminal renal angioplasty in renovascular hypertension due to atheroma or fibromuscular dysplasia. *N Engl J Med* 309:274-279
4. Geyskes GG (1988) Treatment of renovascular hypertension with percutaneous transluminal renal angioplasty. *Am J Kidney Dis* 4:253-265
5. Kuhlmann U, Greminger P, Gruntzig A, Schneider E, Pouliadis G, Lüscher T, Steurer J, Siegenthaler W, Vetter W (1985) Long-term experience in percutaneous transluminal dilatation of renal artery stenosis. *Am J Med* 79:692-698
6. Millan VG, McCauley J, Kopelman RI, Madias NE (1985) Percutaneous transluminal renal angioplasty in nonatheromatous renovascular hypertension. Long-term results. *Hypertension* 7:668-674
7. Cicuto KP, McLean GK, Oleaga JA, Ring EJ, Freiman DB, Grossman RA (1981) Renal artery stenosis: Anatomic classification for percutaneous transluminal angioplasty. *AJR* 137:599-601
8. Kremer Hovinga TK, de Jong PE, de Zeeuw D, Donker AJ, Schuur KH, van der Hem GK (1986) Restenosis prevalence and long-term effects on renal function after percutaneous transluminal renal angioplasty. *Nephron* 44:64-67
9. Palmaz JC (1988) Balloon-expandable intravascular stent. *AJR* 150:1263-1269
10. Sigwart U, Puel J, Mirkovitch V, Joffre F, Kappenberger L (1987) Intravascular stents to prevent occlusion and restenosis after transluminal angioplasty. *N Engl J Med* 316:701-706
11. Rousseau H, Puel J, Joffre F, Sigwart U, Duboucher C, Imbert C, Knight C, Kropf L, Wallsten H (1987) Self-expanding endovascular prosthesis: An experimental study. *Radiology* 164:709-714
12. Brawn LA, Ramsey LE (1987) Is "improvement" real with percutaneous transluminal angioplasty in the management of renovascular hypertension? *Lancet* 1:1313-1316
13. Srur MF, Sos TA, Saddekni S, Cohn DJ, Rozenblit G, Wetter EB (1985) Intimal fibromuscular dysplasia and Takayasu arteritis: Delayed response to percutaneous transluminal renal angioplasty. *Radiology* 157:657-660
14. Dong ZJ, Li SH, Lu XC (1987) Percutaneous transluminal angioplasty for renovascular hypertension in arteritis: Experience in China. *Radiology* 162:477-479
15. Gardiner GA, Freedman AM, Shlansky-Goldberg R (1988) Percutaneous transluminal angioplasty delayed response in neurofibromatosis. *Radiology* 169:79-80
16. Baxi R, Epstein HY, Abitol C (1981) Percutaneous transluminal renal angioplasty in hypertension associated with neurofibromatosis. *Radiology* 139:583-584
17. Faxon DP, Sanborn TA, Haudenschild CC (1987) Mechanism of angioplasty and its relation to restenosis. *Am J Cardiol* 60:5B-9B
18. Essed CE, Van den Brand M, Becker AE (1983) Transluminal coronary angioplasty and early stenosis: Fibrocellular occlusion after wall laceration. *Br Heart J* 49:393-396
19. Duprat G, Wright KC, Charnsangavej C, Wallace S, Gianturco C (1987) Self-expanding metallic stents for small vessels: An experimental evaluation. *Radiology* 162:469-472
20. Rousseau H, Joffre F, Raillat C, Julian M, Pierragi MT, Chalabi F, Bouissou H (1989) Self-expanding endovascular prosthesis in experimental atherosclerosis. *Radiology* 170:773-778
21. Palmaz JC, Richter GM, Noeldt C (1988) Intraluminal stents in atherosclerotic iliac artery stenosis: Preliminary report of a multicenter study. *Radiology* 168:727-731
22. Rousseau H, Raillat C, Joffre F, Knight C, Ginestet MC (1989) Treatment of femoropopliteal stenoses by "wallstent" endoprosthesis. *Radiology* 172:961-964
23. Miller CA, Ford KK, Braun SD, Newman GE, Moore AV, Malone R, Dunnick NR (1985) Percutaneous transluminal angioplasty vs surgery for renovascular hypertension. *AJR* 144:447-450



## **Stents in Renal Arteries: Long Term Follow-up with the Wallstent.**

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### **PURPOSE**

**E**ndovascular stenting has been proposed and tested as an endovascular mechanical support that can be introduced percutaneously to compensate or to prevent the limitations of percutaneous transluminal renal angioplasty (PTRA). Stent provides an effective way for non-operative management of flow-compromising PTRA- included dissection and vessel reclosure during PTRA. Stent placement has also been proposed for treating delayed post-PTRA restenosis and preventing a second restenosis.

### **MATERIAL AND METHODS**

**We** report our experience with the placement of 25 stents during 23 procedures in 21 patients with a mean follow-up of  $32 \pm 15$  months. Twenty-five Wallstent endoprostheses were placed in 21 patients to treat delayed restenosis after previous balloon angioplasty (n=13) or inadequate immediate postangioplasty response (n=8). The stenosis nature was atheromatosis in 15 patients, involving ostium in 7 cases. Kidney was solitary in 5 patients.

### **RESULTS**

**T**echnical success was achieved in all patients. Early benign complication occurred in 4 patients. Follow-up

angiography (12 to 60 months) showed restenosis in 4 patients. Cumulative primary patency rate (Kaplan-Meier) was 95% at 7 months, 85% at 9 months and 77% at 15 months. At clinical follow-up ( $32 \pm 15$  months), hypertension was cured in 3 patients and improved in the other 18. Renal function remained normal in patients without renal failure before stenting. In 6 patients with renal failure, renal function improved in 1 patient, stabilized in 3 and slightly decreased in 2.

### **CONCLUSION**

**A**rterial stenting is beneficial in many patients with poor results from conventional renal angioplasty. It is also beneficial to those with delayed restenosis, especially with ostial lesion, with good clinical results and a high patency rate in long-term follow-up.

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## **Tratamiento Endovascular de Aneurismas de Aorta Abdominal, Fístulas A-V y Pseudoaneurismas con Stent-graft**

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Los aneurismas de aorta abdominal (AAA) son considerados como una patología letal. La incidencia de esta enfermedad es elevada variando entre 1.8% y 6.6% en estudios autopsicos y del 5.4% en pacientes entre 65 y 74 años de edad en screening. En los últimos años con el aumento de métodos de diagnóstico por imágenes no invasivos y la mayor expectativa de vida el número y la edad de los pacientes con AAA se encuentran en ascenso. La trombosis espontánea o provocada de los AAA no resulta una protección segura para los pacientes.

El tratamiento quirúrgico de los AAA fue descrito en Francia por DuBost en 1951, siendo actualmente el reemplazo con prótesis el tratamiento de elección para esta patología.

Si bien la tasa de morbimortalidad en cirugía electiva de los AAA en distintos centros se encuentra por debajo del 5%, los cirujanos vasculares se enfrentan cada vez mas frecuentemente con pacientes añosos con enfermedades asociadas graves en los cuales estas cifras se elevan a tal punto que muchos de estos enfermos son considerados con riesgo prohibitivo para la cirugía convencional.

Para este grupo especial de enfermos se pensó en una nueva alternativa terapéutica y desde el año 1976 se trabajó sobre la

idea de implantar una prótesis por vía endovascular. Distintos dispositivos fueron ensayados con resultado negativo hasta 1988 donde se demostró que los stents pueden reemplazar a la sutura quirúrgica para la fijación de la prótesis a la pared de la aorta.

El dispositivo consiste en un catéter del tipo de los utilizados para valvuloplastia aórtica con uno a dos balones de 60 mm de longitud por 30 mm de

diámetro, sobre el cual se monta un stent expandible por balón de 5 mm de diámetro y 3.5 cm de longitud al cual se sutura una prótesis de Dacron de pared fina; todos estos elementos se encuentran plegados en el interior de un catéter introductor de 21 french.

El procedimiento se efectúa por vía femoral, procediendo inicialmente a colocar un catéter a nivel de la aorta abdominal por encima de las arterias renales para realizar un estudio angiográfico para marcación de distintos reparos anatómicos. Luego se reemplaza el catéter angiográfico sobre guía metálica por el introductor que contiene en su interior el stent-graft ubicándolo bajo control radioscópico a nivel del cuello superior; con el catéter en la posición elegida se retira el introductor y se infla el

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balón, lo cual produce la expansión del stent fijando la prótesis de Dacron sobre la pared aórtica. A continuación se coloca otro stent para fijar el extremo distal de la prótesis a nivel del cuello inferior. De esta forma se obtiene la exclusión del aneurisma estableciendo el flujo a través de la prótesis. Los estudios de factibilidad de esta técnica se realizaron en animales de experimentación. Se reemplazó la aorta infrarenal de una serie de perros por una prótesis de Dacron con forma fusiforme. Luego de crear estos aneurismas artificiales, los animales fueron tratados implantando prótesis de Dacron tubulares fijadas en ambos extremos con stents metálicos logrando la exclusión satisfactoria del aneurisma.

Habiendo finalizado la etapa experimental, en el mes de Septiembre de 1990, se efectuó el tratamiento de un AAA por vía endovascular en un paciente con alto riesgo para cirugía convencional. Se implantó una prótesis de Dacron fijada con un stent a nivel del cuello proximal, evolucionando en forma favorable.

Entre septiembre 1990 y diciembre 1993 se realizaron 50 procedimientos endovasculares con stent-graft, 40 aneurismas de la aorta abdominal, 1 aneurisma ilíaco, 2 by-pass femorales, 2 fístulas A-V de arteria subclavia, 1 fístula A-V de arteria ilíaca-vena cava inferior, 1 fístula A-V de arteria femoral superficial, 1 pseudoaneurisma infectado de arteria femoral común y 1 pseudoaneurisma de la arteria carótida primitiva.

De los 40 aneurismas abdominales, 8 fueron tratados con prótesis aorto-aórtica fijada con stent proximal, 17 con prótesis aorto-aórtica con stent proximal y distal y 15 con prótesis aorto-ilíaca.

Se obtuvo un éxito inicial en un 80% (32/40), y entre los 8 pacientes con fallo, 4 pudieron corregirse ulteriormente.

Las complicaciones observadas: hematoma inguinal (2), apertura incompleta del stent proximal con migración (1), microembolización masiva (3), incorrecta posición del stent proximal (1), leak proximal (1). Los pacientes con fístulas A-V y pseudoaneurismas fueron tratados con stents cubiertos con prótesis de Dacron y en dos casos con stents cubiertos con vena (pseudoaneurisma femoral infectado y pseudoaneurisma carotideo en un paciente con SIDA). En estos pacientes el resultado fue satisfactorio y no se presentaron complicaciones. El seguimiento de estos pacientes incluye consultas clínicas periódicas, eco-doppler color, tomografía computada y angiografía.

## **Brachiocephalic PTA**

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**D**espite the success of angioplasty in virtually all arteries not carrying blood to the cerebral circulation, interventionalists have been reluctant to perform carotid PTA because of the known risk of cerebral embolization of debris and the perception that this will result in clinically significant neurological deficits. Before peripheral and coronary PTA became widely used and accepted, similar concerns about embolic complications were expressed.

A number of reports, including some substantial series indicating excellent anatomic and clinical results with PTA of craniocerebral vessels. Most of these reports have selected patients with angiographic anatomy that would minimize the risk of cerebral embolization, for example, subclavian PTA in the presence of a steal or PTA of "smooth" stenosis at the origin of the vertebral artery. Angioplasty of the carotid bifurcation using "protection balloons" has been reported, but bifurcation PTA using standard angioplasty catheter has also been reported. Most carotid bifurcation procedures have been performed on arteries that did not have angioplasty visible ulcerations.

Clearly patient selection has had an impact on the reported "good" results of extracranial brachycephalic PTA. No report has included blinding of angiographic interpretation - before or after PTA. Definitions of anatomic success have rarely been described. Essential objective follow-up has been lacking. If carotid/vertebral PTA is to be a viable option to surgical revascularization, we must know not only the procedure related stroke incidence, but the long term risk of stroke.

For almost three years the North American Cerebral PTA Register (NACPTAR) has been enrolling patients in a prospective cohort study to assess the safety and efficacy of brachycephalic PTA using objective criteria and clearly defined parameters. Inclusion in the study requires that the patient be symptomatic in the cerebral distribution of the target artery. The target artery must have a

stenosis equal to or greater than 75% on a selective 4 vessel angiogram. Prior to PTA all patients receive a detailed neurologic exam by a neurologist, a brain CT/MRI, doppler and when possible SPECT. Post-PTA, prior to discharge, these studies are repeated. During the first year post-PTA, clinical and imaging exams are repeated at quarterly intervals and the angiogram is repeated at the end of this year. Semiannual follow-up continues for an additional year.

All patients are placed on 325 mg ASA daily, and all are systemically heparinized for the PTA procedure. Beyond this the technical details of each angioplasty procedure is the choice of the individual interventionalist.

Extracranial angioplasty is most commonly performed without guiding with appropriate anatomy and symptoms who have failed medical therapy. Intracranial PTA is performed through guiding catheters designed for craniocerebral angioplasty.

The majority of patients enrolled in the NACPTAR to date have been contributed by two of the seventeen participating centers. More than 165 cerebral PTA procedures have been performed. The median patient age is 63 years. Thirty-five percent (35%) of the patients were female. The average pre-PTA stenosis was 80%. The average post-PTA residual stenosis was 30%. There were no embolic strokes. There was one stroke related to an extracranial PTA. There were seven strokes related to intracranial PTA. All of the strokes were confirmed by CT/MRI. There were 3 deaths, all in patients having an intracranial PTA.

The stroke/death rate for extracranial PTA is lower than the same event rate for carotid endarterectomy as reported in NASCET.

Cerebral PTA can produce a clinically and biologically significant decrease in arterial stenoses with an acceptable rate of central nervous system

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complications.

We will present technical information as time permits, and during the procedure demonstration. The principles of neurovascular rescue stroke salvage - will be included.

**References.**

1. Backman D.M., Kim R.M., Transluminal dilatation for subclavian steal syndrome. *Am. J. Radiol.* 1980; 135: 995-996.
2. Motarjeme A., Keifer J.W., Zuska A.J. Percutaneous transluminal angioplasty of the brachiocephalic arteries. *A.J.R.* 1982; 138 (3): 457-462.
3. Damouth H.D. Jr., Diamond A.B., Rappoport A.S., Renner J.W. Angioplasty of subclavian artery stenosis proximal to the vertebral origin. *Am. J. Neurolog. Radiol.* 1983; 4 (6): 1239-1242.
4. Bockenheimer S.A.M., Mathias K. Percutaneous angioplasty in arterio-sclerotic internal carotid artery stenosis. *A.J.N.R.* 1983; 4: 791-792.
5. Courtheous P., Theron J., Maiza D., Derlon J.M., Pelouze G.A., Henriot J.P., Evrard C., Commeau P. Endoluminal angioplasty for atheromatous stenosis of supra-aortic trunks. The brachiocephalica arterial trunk, subclavian arteries. *J. Radiol.* 1984; 65 (12): 845-851.
6. Theron J., Courtheous P., Henriot J.P., Pelouze G., Derlon J.M., Maiza D. Angioplasty of supra-aortic arteries. *J. Neuroradiol.* 1984; 11 (3): 187-200.
7. Gordon R.L., Haskell L., Hirsch M., Shifrin E., Weinman E., Romanoff H. Transluminal dilatation of the subclavian artery. *Cardiovasc. Intervent. Radiol.* 1985; 8 (1): 14-19.
8. Motarjeme A., Keifer J.W., Zuska A.J., Nabawi P. Percutaneous transluminal angioplasty for treatment of subclavian steal. *Radiology* 1985; 155 (3): 611-613.
9. Theron J., Melancon D., Ethier R. Subclavian steal syndromes and their treatment by angioplasty. Hemodynamic classification of subclavian artery stenoses. *Neuroradiology* 1985; 27 (3): 265-270.
10. Tsai F.Y., Matovich V., Hieshima G., Shah D.C., Mehringer C.M., Tiu G., Higashida R., Pribram H.F.W. Percutaneous transluminal angioplasty of the carotid artery. *Am. J. Neurolog. Radiol.* 1986; 7: 349-358.
11. Tsai F.Y., Matovich V., Hieshima G., Mehringer C.M., Tiu G., Higashida R., Shah D.C., Ashraf A., Pribram H.F.W. Practical aspects of percutaneous transluminal angioplasty of the carotid artery. *A.C.T.A. Radiol.* 1986; 369: 127-139.
12. Freitag G., Freitag J., Koch R.D., Wagemann W. Percutaneous angioplasty of carotid artery stenoses. *Neuroradiology* 1986; 28: 126-127.
13. Higashida R.T., Hieshima G.B., Tsai F.Y., Bentson J.R., Halbach V.V. Percutaneous transluminal angioplasty of the subclavian and vertebral arterial. *Am. J. Neurolog. Radiol.* 1986; 369: 124-126.
14. Tsai F.Y., Higashida R.T., Matovich V., Alfieri K., Kobayashi S. Seven year's experience with PTA of carotid artery. *Neuroradiology* 1991; 33 (Suppl): 397-398.
15. Courtheous P., Tournade A., Theron J., Henriot J.P., Maiza D., Derlon J.M., Pelouze G., Evrard C. Transcatheter angioplasty of vertebral artery atheromatous ostial stricture. *Neuroradiology* 1985; 27: 259-264.
16. Higashida R.T., Hieshima G.B., Tsai F.Y., Bentson J.R., Halbach V.V. Percutaneous transluminal angioplasty of the subclavian and vertebral arteries. *Acta. Radiol.* 1986; (Suppl) 369: 124-127.
17. Higashida R.T., Hieshima G.B., Tsai F.Y., Halbach V.V., Bentson J.R., Norman D., Newton T.H. Transluminal angioplasty of the vertebral and basilar artery. *Am. J. Neurolog. Radiol.* 1987; 8: 745-750.
18. Vitek J.J. Subclavian artery angioplasty and the origin of the vertebral artery. *Radiology* 1989; 170: 407-409.
19. Higashida R.T., Tsai F.Y., Halbach V.V., Hieshima G.B., Dowd C.F., Barnwell S.L. Percutaneous transluminal angioplasty of the subclavian, vertebral and basilar arteries: technical considerations. *Neuroradiology* 1991; 33 (Suppl.): 394-396.
20. DeMonte F., Peerless S.J., Rankin R.N. Carotid angioplasty with evidence of distal embolization. *J. Neurosurg.* 1989; 70: 138-141.
21. Theron J., Courtheous P., Alanchkar F., Bouvard G., Maiza D. New triple coaxial catheter system for carotid angioplasty with cerebral protection. *A.J.N.R.* 1990; 11: 869-874.
22. Tsai F.Y., Nakashima H., Wadley D., Myers T.V., Huang J.Y. IVT atherectomy with suction for treatment of carotid stenosis: experimental study. *Neuroradiology* 1991; 33 (Suppl): 53-56.
23. Tsai F.Y., Nakashima H., Medli C. Percutaneous Transluminal Angioplasty of Extracranial Arterial Stenosis in the Head and Neck. *Neuroimaging Clinics of North America* 1992; 2: 2.
24. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade stenoses. *New Eng. J. Med.* 1991; 325: 445-453.

## Percutaneous Transluminal Angioplasty in Renovascular Hypertension

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### SCREENING AND DIAGNOSIS OF RENOVASCULAR HYPERTENSION

Recent onset of rapidly accelerated difficult to control severe hypertension in young women or older smokers, especially if associated with generalized vascular disease, an abdominal bruit and an elevated plasma renin activity (PRA) are clinical characteristics of renovascular hypertension (1,2). The sensitivity of PRA can be increased by the diagnostic use of an angiotensin converting enzyme inhibitor (ACEI), in the peripheral captopril challenge test (3). Such patients can be further evaluated, often as outpatients, by selective renal vein blood sampling and assay for renin activity (RVR) (4). The sensitivity of RVR can also be enhanced by the use of ACEI (5). RVR may be followed by intravenous digital subtraction angiography (IVDSA) for anatomic information (6.-8). Other centers advocate radionuclide scanning especially following ACEI (9).

Patients with azotemia or following renal transplantation and clinical characteristics of renovascular hypertension should be evaluated by arteriography, since if a stenosis is identified, renal angioplasty (PTRA) in them should be attempted regardless of biochemical studies which are often unreliable and misleading (10) (Table I).

#### Arteriography:

At the New York Hospital, most patients are admitted for diagnostic arteriography and PTRA based on positive and congruent results of RVR and IVDSA or are referred to us with positive studies. In patients with azotemia and following renal transplantation, intraarterial digital subtraction angiography (IADSA) is the initial diagnostic technique of choice (10).

In hospitals where RVR are difficult to obtain or are not reliable and increasingly frequent even at the New York Hospital, and where there is a prior agreement between the referring clinicians and the

vascular radiologists, the initial screen may be IADSA. RVR may be obtained after the arteriogram but before PTRA for retrospective analysis.

### PATHOPHYSIOLOGY OF ANGIOPLASTY AND BALLOON DILATION

The mechanical aspect of renal angioplasty are related to the underlying histopathology and pathophysiology of the lesions. It was originally assumed that angioplasty worked by compression and remodeling of the atheromatous material (11,12). Block (13), Catañeda-Zuniga and Amplatz (14), and later Wolf (15), have shown that angioplasty works by controlled trauma. The atheroma is fragmented longitudinally and circumferentially, with the cracks also tearing into the intima and the media. The muscle fibers of the media are injured and paralyzed, the adventitia is overdilated, and the atheromatous material is redistributed radially and laterally into the overdilated adventitia, thus leaving a large patent central lumen which is kept open by the increased flow. In ostial atheromatous stenoses (16) the entire sheet of atheroma surrounding the renal artery ostium is acted upon by the dilating balloon. Soon after deflation of the balloon the stenosis recurs. Stenoses of the true origin of the renal artery respond well to PTRA. There origin stenoses account for only twenty-five percent of the ostial stenoses. It is likely that the mechanism of action of angioplasty in fibromuscular dysplasia is similar to that in atheroma in overdilation of local stenotic segments with healing in the dilated state. Most patients with the media form of fibromuscular dysplasia respond to a low pressure inflation of the balloons.

In intimal fibroplasia (17,18), in Takayasu's arteritis (17) and in neurofibromatosis (19), initial response is disappointing in spite of repeated inflations at high pressures. The initial dilation ruptures the large intimal fibrous cushions and dilates the vessel wall, but spasm and the large bulk

of tissue continue to produce a residual stenosis. With increased pressure and flow in the vessel, however, delayed continued expansion of the lumen to normal size has been observed. In these lesions, aggressive repeated overdilation can result in vessel rupture.

For this reason, in these patients we do not persist in trying to eliminate the "waist" of the angioplasty balloon. Infants and young children with the mid-aortic syndrome (20,21) have shown very poor response to angioplasty and aggressive dilation can produce severe complications including rupture of the artery in these small vessels.

#### Equipment:

The fundamenal techniques of percutaneous transluminal renal angioplasty (PTRA) have not changed over the last five years. The equipment however has undergone a tremendous amount of modification. Five years ago the standard angioplasty balloon catheter for renal vascular disease had a 7-French shaft; now balloons up to 8 mm in diameter on a 5-French and balloons up to 6 mm in diameter on 4.3 French catheter shafts are available. Even smaller "balloons on a wire" are approximately 3-Frech and also have balloons up to 6 mm in diameter. Balloon catheters have a lower profile transition zone between the balloon and shaft, and the tips of most catheters extend less than 1 cm beyond the balloon. Guidewires have become down-sized (.038" to 0.014"), more floppy at the tip, yet with a stiff shaft even in the .0014" size, more torquable and more visible. In addition, wires tapered from .035 inches t .016 inches with torquable soft platinum tips are available. These evolutions is catheter and guidewire design have permitted safer and more successful treatment of severe proximal renal artery disease as well as access to branch stenoses.

Advances in imaging have also greatly contributed to the safety and success of PTRA. Reduced doses of dilute conventional contrast media and the use

of CO<sub>2</sub> as a contrast agent for IADSA have diminished the incidence of renal failure.

#### **TECHNIQUE OF ANGIOPLASTY (22)**

Our standard procedure is to approach atheromatous renal stenoses from the femoral artery with a Sos Omni Selective catheter (AngioDynamics). An extremely floppy "Bentson" \*\* (Cook Inc.) guidewire extending several centimeter beyond the catheter tip is the least traumatic way to enter and cross atheromatous stenoses, especially those which are very proximal in the renal artery. Alternatively the TAD\*\*\* (ACS) floppy tip wire tapered from .035" to .018 with a rigid shaft can be used. Fibromuscular stenoses are also crossed with the aid of a Sos Omni Selective and Bentson for TAD guide wire. If the guide wire does not easily cross the stenoses, then these catheters can be manipulated across without the use of a guide wire while constantly injecting dilute contrast material. The use of inspiration and expiration to alter the course of the renal artery can be quite helpful.

Once the initial catheter has crossed the stenosis, the floppy wire is exchanged for the more rigid "TAD" guidewire. This has a short straight floppy tip which allows its introduction further into the segmental branches, thereby assuring that the junction of the renal artery and the aorta are subtended by the rigid rather than the more floppy transitional portion of the wire. This facilitates exchange of the initial for the balloon dilation catheter. This exchange wire is often preshaped to lie securely across the aorto renal angle, and the very tip of the balloon catheter (proximal to the balloon) is gently shaped into a beak to allow it to follow over the wire from the aorta into the renal artery even through severe rigid proximal stenoses. Balloons should be observed during inflation for elimination of the indentation, but equally as importantly during deflation for recurrence of the indentation, which indicates an inadequate dilation.

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## **COMPLICATIONS AND THEIR PREVENTION**

The most frequent major complications in PTRAs are initiated by thrombosis and spasm of the renal artery. The normal response of the vessel wall to trauma (even to the controlled trauma of angioplasty) is to release serotonin and thromboxane, especially if clot and atheromatous material are present (23). The renal artery responds to these substances by spasm and thrombosis. The manipulation of guide wires in arteries with spasm and/or thrombosis increases the risks of dissection, occlusion or perforation. Complications are reported to occur in 5-10% of patients (17,24-29). Adjunctive medications can prevent or diminish the incidence and severity of complications. These are: a) Antiplatelet: Aspirin, in a dose of 80-300 mg/day for a few days before angioplasty, and continued for at least three months following successful angioplasty. b) Spasmolytic and anti-spasmodic: Nifedipine, a calcium channel blocker can be given as little as twenty minutes before the procedure, in a dose of 10-20 mg, by mouth or sublingually (if the capsule is pierced), and it remains active for two to four hours. 100-200 mcg of intraarterial nitroglycerin is also very effective to reverse spasm. c) Anticoagulant: Once intraarterial position has been established 2000-5000 units of Heparin is given IV or IA. The Heparin is not reversed at the end of the procedure, but additional doses of Heparin or Coumarin are not used following PTRAs. Symptomatic distal embolization of dislodged large plaque is rare. However, as more patients with diffuse atheromatous disease are treated by PTRAs cholesterol or athero microemboli are an increasingly frequent though fortunately still rare complication (30, 31). Once it occurs it is untreatable and can cause ischemia or necrosis of the kidneys, bowel and extremities. PTRAs in patients with diffusely atheromatous abdominal aortas should be performed with the least manipulation, and possibly through long sheaths introduced to just below the renal arteries.

## **RESULTS**

Since 1978, we have attempted PTRAs in over five hundred renal arteries. Patients with fibromuscular disease of the main and/or branch renal arteries are the most suitable candidates for PTRAs.

Approximately ninety percent of the initial thirty-one patients in our series with fibromuscular

disease had a technically successful PTRAs, and of these, ninety percent showed a blood pressure benefit (cured and improved) (32). These results are similar to those reported by other angiographers (Table II) (32,33,30,34,35,36). Children form an interesting subset of patients in whom branch stenosis is very frequent. This presents important diagnostic and therapeutic problems. Selective renal vein blood sampling and assay for renin activity (RVR) are very important for identifying these patients. Intravenous DSA should not be used since even IADSA and conventional film screen arteriography may miss these branch lesions which require selective magnification arteriography. We successfully dilated 16 of 18 arteries in 14 children resulting in 9 cures and 3 improved (37).

In the initial fifty-one patients with atheromatous disease, the presence of an occluded renal artery (five patients), or a stenosis at the ostium of the renal artery at the aortic wall (five patients), heralded a low incidence of technical success (approximately twenty-five percent), whereas in twenty non-occluded and non-ostial atheromatous lesions, we achieved a technical success rate of seventy-five percent. Following a technically successful angioplasty, the blood pressure benefit was eighty-two percent in patients with unilateral atheromatous lesions. Our results are similar to those in the literature (Table III) (33,38,30,34,32,35,36). Of the twenty-one atheromatous patients with bilateral physiologically significant renal artery lesions in our series, all had severely advanced diffuse vascular disease: two-thirds of them had a total renal artery occlusion and renal atrophy, and half had an ostial stenosis.

Since, in bilateral disease, only total success in dilating each offending lesion can be expected to successfully alleviate hypertension, it is not surprising that blood pressure benefit was the least in this group (32).

We have also attempted renal angioplasty in fifty-five consecutive patients with renovascular hypertension and azotemia (defined as a serum creatinine above 1.7 mg/dl) (10). All patients had blood pressures above 160/95 mmHg and had arteriographically documented stenosis of one or both renal arteries of at least 75% of the luminal diameter.

Mean serum creatinine for the entire group was greater than 3 mg/dl. PTRAs were technically successful in 45 patients. At a two year (mean) follow up 38 of these patients were alive, and only 8 needed dialysis. The rest had improvement or



stabilization of their renal functions. In contrast, of the 10 patients where PTRA failed, only 4 were alive and 6 had needed dialysis.

Our results indicate that the use of angioplasty may be justified in such high-risk patients on the grounds that the risk (further deterioration of renal function in 9% are outweighed by the benefits. Similar results were obtained by Tegtmeier (33), Bell (39) and Martin (34).

### **RESTENOSIS**

Restenosis in ten to fifty percent of dilated arteries depending on anatomic location continues to be the chief long-term problem with percutaneous transluminal angioplasty (PTA) (40). There have been various mechanical and pharmacological attempts to diminish the incidence of restenosis as well as to reduce the incidence of immediate complications and failures of angioplasty.

In the first few months restenosis is often due to elastic recoil of and incompletely dilated artery, however, after six months it is most likely to be neointimal hyperplasia. After one year, recurrence of atheromatous disease at the dilatation site or elsewhere continues to jeopardize the long-term outcome.

Previous attempts to reduce restenosis by pharmacologic interventions such as heparin, warfarin, and aspirin (41) have not been successful. More recently, angiotensin converting enzyme inhibitors and long-term anti-spasmodic medications are being evaluated (42).

Mechanical means to reduce the restenosis rate are also being explored. The twenty-five to thirty percent recurrence rate in PTRA for atheroma reported by many authors is probably in part due to an inadequate initial dilation and rapid "recoil".

Preliminary data indicates that our re-stenosis rate in unilateral non-ostial atheromatous renal artery disease is only approximately fifteen percent. This is possibly a result of having used balloons at least 1 mm greater than the diameter of the vessel measured on the arteriogram.

### **STENTS**

Coil like intravascular stents to eliminate restenosis were first proposed by Charles Dotter (43). In the last five years several different stents (44, 45) have been developed and are undergoing clinical trials in the renal arteries.

A United States multicenter study to evaluate the use of the Palmaz stent in the renal arteries was initiated in 1988 and the preliminary results reports in 1991 (46). To date, over 170 patients have been entered, however only 53 patients with 54 stents have been analyzed. There were 43 ostial, seven non-ostial and three miscellaneous lesions. Stents were placed in 33 lesions for residual hemodynamically significant stenosis and in 21 for restenosis after prior angioplasty. Follow-up arteriography was performed in 29 patients at  $8.1 \pm 4.3$  months with a mean follow-up time of approximately four months. Forty-three percent of lesions demonstrated restenosis within the stent to a diameter of less than fifty percent. At six months and one year 51% and 47% of patients respectively demonstrated cure and improvement of their hypertension. There are also preliminary reports on experience with the Wallstent (47) and the Strecker (48, 49) stents in the renal arteries which are also encouraging.

We have placed Palmaz stents in 22 patients. Two patients received bilateral stents. Half the patients were stented following recurrence of an initially good angioplasty result whereas in the others, ostial lesions which did not respond to angioplasty were stented immediately. Clinical follow-up demonstrated improvement or stabilization in all patients up to a two-year follow-up period. Follow-up arteriography has already been performed at a minimum of six months in half the patients. Half of the patients restenosed had evidence of angiographic restenosis (> 50% reduction from the post stent diameter).

The recurrence rate following stenting of the renal artery seems clearly greater than that in the iliac artery. There are multiple factors responsible for this including greater turbulence of the aorta to renal artery bifurcation as well as the relatively smaller diameter and flow in the renal artery. A great deal of work remains to elucidate the exact mechanism of restenosis in the renal artery stents and their possible prevention.

Nevertheless, even if one were to take the most pessimistic (and I believe realistic) view that only half of the patients with an ostial renal artery lesion are successfully treated by stents, the number of patients who need surgical revascularization for ostial lesions would be halved.

TABLE I. PTR A IN AZOTEMIA

	GROUP I	GROUP II	GROUP III
NUMBER OF PATIENTS	26	19	10
MEAN AGE (YEARS)	62	62	65
SEX (% MALE)	65	63	80
PERCENTAGE WITH DIFFUSE ATHEROMA	65	63	100
PERCENTAGE WITH BILATERAL STENOSES	77	63	50
LENGTH OF ISCHAEMIC KIDNEY			
CM	8.0	8.3	10.3
RANGE	6.8-13.0	6.6-11.5	6.2-13.5
LENGTH OF CONTRALATERAL KIDNEY			
CM	11.8	10.2	11.8
RANGE	9.5-16.0	9.0-13.5	9.8-14.0
BLOOD PRESSURE (MMHG)			
PRE-ANGIOPLASTY	190/99	182/96	185/98
+1 DAY	147/87	160/86	178/89
+6 MONTHS	165/88	171/89	194/88
+1 YEAR	151/83	158/86	-
+ 2 YEARS	147/81	-	-
SERUM CREATININE (MG/DL)			
PRE-ANGIOPLASTY	3.1	2.7	3.4
+1 DAY	2.8	3.3	5.1
+6 MONTHS	1.9	3.4	4.1
+1 YEAR	1.9	3.1	-
+2 YEARS	1.9	-	-

**TABLE II. IMMEDIATE AND LONG-TERM CLINICAL RESULTS OF RENAL ANGIOPLASTY IN FIBROMUSCULAR DYSPLASIA**

AUTHOR	NUMBER OF PATIENTS	SUCCESSFULLY DILATED (%)	CURED (%)	IMPROVED (%)	FOLLOW-UP (MOS) MEAN (RANGE)
SOS (32)	31	27 (87)	16 (59)	9 (33)	16 (4-40)
TEGMEYER (33)	27	27 (100)	10 (37)	17(63)	NA (2-51)
GEYSKES (30)	21	21 (100)	10 (48)	10 (48)	NA (12-48)
MARTIN,L (34)	20	20 (100)	5 (25)	12 (60)	16 (3-36)
MARTIN,L (35)	11	8 (73)	5 (63)	1 (13)	13 (NA)
GRIM (36)	10	9 (90)	5 (56)	4 (44)	10 (1-14)
<b>TOTAL</b>	<b>120</b>	<b>112 (93)</b>	<b>51 (46)</b>	<b>53 (47)</b>	

NA= NOT AVAILABLE

**TABLE III. IMMEDIATE AND LONG-TERM CLINICAL RESULTS OF RENAL ANGIOPLASTY IN UNILATERAL NON-OSTIAL ATHEROMA**

AUTHOR (REF#)	NUMBER OF PATIENTS	SUCCESSFULLY DILATED (%)	CURED (%)	IMPROVED (%)	FOLLOW-UP (MOS) MEAN (RANGE)
TEGMEYER*,** (33)	65	61 (94)	15 (25)	129 (55)	NA (1-60)
SCHWARTEN (38)	54	49 (91)	23 (47)	25 (51)	NA (1-18)
GEYSKES * (30)	44	44 (100)	4 (9)	19 (43)	NA (12-48)
MARTIN,L** (34)	38	38 (100)?	10 (25)	19(47)	19 (3-36)
SOS (32)	20	15 (75)	4 (27)	9 (60)	16 (4-40)
MARTIN,L (35)	15	13 (87)	2 (15)	4 (31)	13 (NA)
GRIM (36)	16	16 (100)	1 (7)	8 (50)	10(3-24)
<b>TOTAL</b>	<b>252</b>	<b>236 (94)</b>	<b>59 (25)</b>	<b>130 (55)</b>	

\* INCLUDES SOME PATIENTS WITH BILATERAL DISEASE

\*\* INCLUDES SOME PATIENTS WITH OSTIAL STENOSES

NA= NOT AVAILBLE

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##### References.

1. Nicholson J.P., Teichman S.L., Alderman M.H., et al. Cigarette smoking and renovascular hypertension. *Lancet* 1983; 2: 765.
2. Maxwell M.H., Bleifer K.H., Franklin S.S., et al. Cooperative Study of Renovascular Hypertension. Demographic analysis of the study. *JAMA* 1972; 220: 1195.
3. Mueller F.B., Sealey J.E., Case C.B., et al. Theaptopril test for identifying renovascular disease in hypertensive patients. *Am J. Med.* 1986; 80: 633.
4. Vaughan E.D. Jr., Buhler F.R., Laragh J.H., et al. Renovascular hypertension: renin measurements to indicate hypersecretion and contralateral suppression, estimate renal plasma flow, and score for surgical curability. *Am J. Med.* 1973; 55: 402.
5. Pickering T.G., Sos T.A., Vaughan E.D. Jr., et al. Predictive value and changes of renin secretion in hypertensive patients with unilateral renovascular disease undergoing successful renal angioplasty. *Am J. Med.* 1984; 76: 398-404.
6. Clarke R.A., Alexander E.S. Digital subtraction angiography of the renal arteries - prospective comparison with conventional arteriography. *Invest. Radiol.* 1983; 18: 6.
7. Hillman B.J., Ovitt T.W., Nudelman S., et al. Digital subtraction angiography or renal vascular abnormalities. *Radiology* 1981; 138: 271.
8. Zabbo A., Novick A.C. Digital subtraction angiography for noninvasive imaging of the renal artery. *Urol Clin North Am* 1984; 11: 409.
9. Geysker G.G., Oei H.Y., Puylaert C.B., et al. Renovascular hypertension identified captopril-induced changes in the renogram. *Hypertension* 1987; 9 (5): 451-8.
10. Pickering T.G., Sos T.A., Saddekni S., Rozenblit G., James G.D., Orenstein A., Helseth G., Laragh J.H.: Renal angioplasty in patients with azotaemia and renovascular hypertension. *J. Hypertension* 1986; 4 (6): S667-S669.
11. Dotter C.T., Judkins M.P. Transluminal treatment of arteriosclerotic obstruction: Description of a new technique and a preliminary report of its applications. *Circulation* 1964; 30: 654.
12. Gruntzig A., Hopff H., Perkutane rekanalisation chronischer arterieller Verschlüsse mit einem neuen Dilatationskatheter. Modifikation der Dotter-Technik. *Deutsch Med. Wochenschr* 1974; 99: 2502-11.
13. Block P.C., Myler R., Sterzer S. Morphology after transluminal angioplasty in human beings. *n Engl. J. Med.* 1981; 305: 382.
14. Castañeda-Zuniga W.R., Formanek A., Tadavarthy M., et al. The mechanism of balloon angioplasty. *Radiology* 1980; 135 (3): 565-71.
15. Wolf G.L., LeVeen R.F., Ring E.J. Potential mechanism of angioplasty. *Cardiovasc. Intervent. Radiol.* 1984; 7: 11.
16. Cicuto K.P., McLean G.K., Oleaga J.A., et al. Renal artery stenosis: anatomic classification for percutaneous transluminal angioplasty. *A.J.R.* 1981; 137: 599-601.
17. Srur M.F., Sos T.A., Saddekni S., et al. Intimal fibromuscular dysplasia and Takayasu arteritis: delayed response to percutaneous transluminal renal angioplasty. *Radiology* 1985; 157: 657-60.
18. Archibald G.R., Beckman C.F., Libertino J.A.: Focal renal artery stenosis caused by fibromuscular dysplasia: Treatment by percutaneous transluminal angioplasty. *A.J.R.* 1988; 151: 593.
19. Gardiner G.A. Jr., Freedman A.M., Shlansky-Goldberg R. Percutaneous transluminal angioplasty: Delayed response in neurofibromatosis. *Radiology* 1988; 169: 79.
20. Stanley J.C., Renovascular Hypertension: The Surgical Point of View. In: Schilfgaard R.V., et al., eds. *Clinical Aspects of Renovascular Hypertension.* Boston: Martinus Nijhoff, 1983; 259.
21. Berkemen Y.M. The midaortic syndrome: Diagnosis and treatment. (Letter) *Radiology* 1989; 170: 571.
22. Tegtmeier C.J., Sos T.A. Techniques of renal angioplasty *Radiology* 1986; 161: 577-586.
23. Monsen C.H., Adams P.C., Badimon L., Chesebro J.H., Fuster V. Platelet-vessel wall interactions in the development of restenosis after coronary angioplasty. *Kardiologie* 1987; 76 (6): 23-28.
24. Saddekni S., Sniderman K.W., Hilton S., Sos T.A., Percutaneous transluminal angioplasty of non-atherosclerotic lesions. *A.J.R.* 1980; 135: 975-982.
25. Martin E.C., Diamond N.G., Casarella W.J. Percutaneous transluminal angioplasty in non-atherosclerotic disease. *Radiology* 1980; 135: 27-33.
26. Sos T.A., Saddekni S., Sniderman S.W., et al. Renal artery angioplasty: Techniques and early results. *Urol. Radiol.* 1982; 3: 223-231.
27. Sniderman K.W., Sos T.A. Percutaneous transluminal recanalization and dilatation of totally occluded renal arteries. *Radiol.* 1982; 142: 607-610.
28. Sniderman K.W., Sprayregen S., Sos T.A. Percutaneous transluminal dilation in renal transplant arterial stenosis. *Transplantation* 1980; 30: 440-4.
29. Tegtmeier C.J., Dyer R., Teates C.D., et al. Percutaneous transluminal dilatation of the renal arteries: techniques and results. *Radiology* 1980; 135: 589-99.
30. Geyskes G.G., Puylaert C.B.A., Oei H.Y., et al. Follow-up study of 70 patients with renal artery stenosis treated by percutaneous transluminal dilatation. *Br. Me. J.* 1983; 287: 333.

#### **IV INTERNATIONAL COURSE ON VASCULAR AND INTERVENTIONAL RADIOLOGY AS THERAPEUTIC ALTERNATIVE**

31. Martin L.G., Casarella W.J., Alspaugh J.P., et al. Renal artery angioplasty: increased technical success and decreased complications in the second 100 patients. *Radiology* 1986; 159: 631-4.
32. Sos T.A., Pickering T.G., Phil D., et al. Percutaneous transluminal renal angioplasty in renovascular hypertension due to atheroma or fibromuscular dysplasia *N.E.J.M.* 1983; 309: 274-279.
33. Tegtmeier C.J., Kellum C.D., Ayers C. Percutaneous transluminal angioplasty of the renal artery: results and long-term follow-up. *Radiology* 1984; 153: 77-84.
34. Martin L.G., Casarella W.J., Gaylord G.M. Azotemia caused by renal artery stenosis: treatment by percutaneous angioplasty. *A.J.R.* 1988; 150: 839-44.
35. Martin E.D., Mattern R.F., Baer L. Renal angioplasty for hypertension: Predictive factors for long-term success. *Am J. Radiol.* 1981; 128: 951.
36. Grim C.E., Luft F.C., Yune H.Y. Percutaneous transluminal dilatation in the treatment of renal vascular hypertension. *Ann. Intern. Med.* 1981; 95: 439.
37. Sos T.A., Saddekni S. Pediatric renovascular hypertension: the role of renal angioplasty. *Dialogues in Ped. Urol.* 1985; 8 (12): 7.
38. Schwarten D.E., Yune H.Y., Klatts E.C., et al. Clinical experience with percutaneous transluminal angioplasty (PTA) of stenosed renal arteries. *Radiology* 1980; 135: 601.
39. Bell G.M., Reid J., Buist T.A.S. Percutaneous transluminal angioplasty improves blood pressure and renal function in renovascular hypertension. *Quarterly J. Med.* 1987; 63 (241): 393-403.
40. Faxon D.P., Sanborn T.A., Haudenschild C.C. Mechanism of angioplasty and its relation to restenosis. *Am J. Cardiol.* 1987; 60: 5B-9B.
41. Thornton M.A., Gruentzig A.R., Hollman J., King S.B. III, Douglas J.S. Coumadin and aspirin in prevention of recurrence after transluminal coronary angioplasty: a randomized study. *Circulation* 1984; 69: 721-727.
42. Whitworth H.B., Roubin G.S., Hollman J., Meier B., Leimgruber P.P., Douglas J.S., King S.B. III, Gruentzig A.R. Effect of nifedipine on recurrent stenosis after percutaneous transluminal coronary angioplasty. *J.A.C.C.* 1986; 8: 1271-1276.
43. Dotter C.T. Transluminally-placed coilspring endarterial tube grafts: long-term patency in canine popliteal artery. *Invest. Radiol.* 1969; 4: 329-332.
44. Palmaz J.C., Kopp D.T., Hayashi H., et al. Normal and stenotic renal arteries: experimental balloon-expandable intraluminal stenting. *Radiology* 1987; 164: 705-708.
45. Sigwart U., Puel J., Mirkovitch V., Joffe F., Kappenberger L. Intravascular stents to prevent occlusion and restenosis after transluminal angioplasty. *N. Engl. J. Med.* 1987; 316 (12): 701-706.
46. Rees C., Palmaz J., Becker G., et al. Result of multicenter trial of renal stenting. *Radiology* 1991; 181: 507-514.
47. Wilms G.E., Peene P.T., Baert A.L., et al. Renal artery stent placement with use of the Wallstent endoprosthesis. *Radiology* 1991 May; 179 (2): 457-462.
48. Kuhn F.P., Malms J., Kutkuhn B., et al. Renal stent implantation. The current indications, 2-year results and the possibilities for percutaneous stent recovery. *Rofu Fortschr Geb Rontgenstr Neuen Bildgeb Berfahr* 1992 Jul; 157 (1): 65-71.
49. Kuhn F.P., Kutkuhn B., Torsello G., Modde U. Renal artery stenosis: preliminary results of treatment with the Strecker stent. *Radiology* 1991 Aug.; 180 (2): 367-372.

## Results of Thrombolytic Therapy in the Management of Renal Artery Occlusion

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Percutaneous renal angioplasty is a well accepted and widely used alternative to surgical revascularization in both fibromuscular dysplasia and atherosclerotic renal artery stenosis. Since 1978, we have performed renal angioplasties on over 500 patients. Thrombolysis has been used in only twelve cases. The patients fall in four categories:

- I. Total acute renal artery occlusion in patients with a solitary kidney (2 patients).
- II. Chronic renal artery occlusion in patients with two kidneys (2 patients).
- III. Chronic renal artery stenosis in patients with two kidneys (2 patients).
- IV. Iatrogenic stenosis/occlusions in patients during renal angioplasty (6 patients).

### TECHNIQUES

Urokinase was used exclusively in all twelve patients with local infusion into the renal artery in all but one patient. Most patients received an initial bolus of urokinase (50,000 - 500,000 units, usually 100 - 250,000 units were given). If necessary, the bolus was followed by an infusion of 1 to 36 hours (20 - 200,000 units/hour). Additionally, patients received nifedipine p.o. prior to angioplasty along with 3,000 - 7,000 units of heparin intravenously and 100 - 400 mcg of nitroglycerin intraarterially during the procedure. No complications related to thrombolytic therapy were encountered.

### RESULTS

#### Acute renal artery occlusion, solitary kidney:

Two patients with solitary kidneys presented with acute onset of anuria, flank pain and elevated creatinine (6.5 and 7.7 mg/ml). One patient had blood pressure elevation as well. Initial aortography

showed native renal artery occlusion in one patient and renal artery bypass graft occlusion in the second patient. Both were treated initially with urokinase, one with a bolus and infusion and the other with a suprarenal aortic high dose infusion (300,000 units/hour) because the catheter could not be stabilized in the renal artery orifice. Follow-up arteriography showed successful thrombolysis as well as an underlying main renal artery or graft stenosis in both cases. In one of the two patients, the stenosis was successfully dilated. Both patients, despite short term dialysis, regained renal function and stabilized their creatinine (2.1 and 3.7 mg/ml). Blood pressure elevation also resolved in the initially hypertensive patient.

#### Unilateral chronic renal occlusion, bilateral kidneys:

We have performed renal angioplasties in over fifty patients with chronic renal artery occlusion. Our criteria for performing angiography include residual function on radionuclide scan and a unilateral decrease in renal artery size. Angiographically, if the proximal stump of the occluded renal artery is evident and if good collateral flow with distal reconstitution is present, our approach has been to attempt renal angioplasty as the initial therapeutic step. In approximately one-half of our patients, we were successful in crossing the occlusion and in dilating the main renal artery. In all but two cases, the distal main and branch renal arteries were normal on post-angioplasty arteriography. In two patients the angioplasty was technically successful but thrombus was present either on the initial diagnostic angiogram or on an angiogram obtained just after crossing the occluded segment, suggesting pre-existent thrombus. The thrombus in both cases looked acute/subacute in appearance with irregularity and near-filling of the arterial lumen. Both patients were treated with urokinase after angioplasty. Successful thrombolysis was achieved in one patient after a one-hour infusion and in the second patient after a bolus and infusion. On follow-up, both patients maintained good renal function

and were normotensive, one on decreased medication and the other on no medication.

#### Chronic renal artery stenosis, bilateral kidneys:

In two patients with renal artery stenosis, the diagnostic arteriography showed a filling defect/thrombus which was unusually bulky and/or irregular distal to the stenosis. This appearance raised the question of acute or sub-acute thrombus possibly superimposed on a chronic lesion (organized thrombus vs. atheroma). Both patients underwent thrombolysis as their initial therapy. One patient had a proximal stenosis in an upper pole main renal artery with a filling defect distally at the renal hilum. Arteriography following thrombolytic therapy showed no change in the appearance of the distal thrombus. The proximal stenosis was not dilated since surgical bypass was decided upon for further management. In the second patient, thrombolysis had no effect on the appearance of the thrombus. Renal angioplasty was then attempted with follow-up arteriography showing occlusion of the proximal renal artery with distal reconstitution. The patient underwent renal artery bypass surgery and was discharged with improved blood pressure control on decreased medication and unchanged renal function.

#### Iatrogenic stenosis/occlusion during renal angioplasty:

In six patients with renal artery stenosis, post angioplasty arteriography showed the presence of stenosis or occlusion with acute thrombosis. All six patients underwent thrombolytic therapy after angioplasty. In three cases the thrombolysis was successful; in two of these, the angioplasty was also successful; in one (an FMD branch stenosis) blood pressure was improved while in the other (an ostial stenosis) the blood pressure was unchanged. In the third patient with successful lysis, angioplasty of a branch FMD stenosis was unsuccessful and blood pressure was unchanged. In three cases thrombolysis was unsuccessful. One of these patients had a 99% stenosis where the lesion was crossed with only a guide wire. Because multiple attempts were made to cross the lesion with a catheter, it is likely that a mechanical obstructive component from subintimal hematoma was present. This patient underwent a successful surgical bypass. In the second failed thrombolysis, the angioplasty was successful but post angioplasty arteriography revealed a dissection along with thrombus. Blood pressure was subsequently well controlled on medication. In the third failed lysis, there was

successful angioplasty of an FMD branch stenosis. Post angioplasty arteriography revealed thrombus, residual narrowing unresponsive to nitroglycerin, and a segmental infarct, findings which suggest a component of dissection. On follow-up, this patient was normotensive off medication.

#### **DISCUSSION**

Several individual case reports have appeared which describe the use of lytic agents in non-embolic renal artery thrombosis. Additionally, a recent abstract summarizes a twelve-patient series. In these cases, improvements in renal dysfunction and hypertension, if present, were achieved when thrombosis was acute or sub-acute.

In our series, thrombolytic therapy was successful in patients with a solitary kidney and acute renal occlusion. In both patients, an underlying stenosis was unmasked. We suggest that thrombolysis for renal salvage should be attempted even in sub-optimal circumstances. For example,

- a) If non-selective catheter position precludes local infusion, systemic thrombolysis may still be effective as illustrated by one of our cases.
- b) Even if oliguria/anuria is of long duration (days to several weeks) a significant number of patients will have a pre-existing stenosis with collateral protection of renal blood supply. In these patients, the actual renal artery occlusion time is probably less critical for predicting salvage of renal function by thrombolytic therapy than would be the case for occlusion by acute thromboembolus where no collateral flow exists.
- c) Although renal viability may be in question, thrombolysis in the absence of absolute contraindications should still be attempted since reperfusion of a non-viable small renal mass does not have significant clinical consequences in contrast to reperfusion of a non-viable extremity.

In our patients with chronic renal artery stenosis/occlusion with thrombosis (categories II & III), thrombolysis was effective when thrombus was acute/sub-acute but not when filling defects were chronic (organized clot versus atheroma).

With iatrogenic stenosis or occlusion (category IV), our results indicate that lesions without permanent

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mechanical damage respond well to thrombolytic therapy. In patients with permanent mechanical damage (as opposed to spasm), urokinase is not helpful.

In conclusion, based on our experience and the results of others,

1. renal angioplasty and thrombolysis should be attempted in all patients with acute/sub-acute anuria and a solitary functioning kidney where clinical and angiographic signs suggest thrombosis +/- stenosis. If the clinical history suggests thromboembolus, surgical embolectomy should be considered.
2. If thrombus is present following renal angioplasty, whether iatrogenic or pre-existent in nature, vasodilatation to relieve spasm and thrombolysis should be performed.

#### **References:**

1. Adler J. Combined thrombolysis with low-dose streptokinase and angioplasty in the treatment of renal artery occlusion. *Urol. Radiol.* 1983; 5: 113-116.
2. Cronan J.J., Dorfman G.S. Low dose thrombolysis: a non-operative approach to renal artery occlusion. *J. Urol.* 1983; 130: 757-759.
3. Hayes J.M., et al. Experience with percutaneous transluminal angioplasty for renal artery stenosis at the Cleveland Clinic. *J. Urol.* 1988; 139: 488-492.
4. LaCombe M. Surgical versus medical treatment of renal artery embolism. *J. Cardiovas. Surg.* 1979; 18: 281-290.
5. McCready R.A., et al. Renal revascularization in patients with a single functioning kidney. *J. Vas. Surg.* 1987; 6: 185-190.
6. McGilligan D.J. The occluded renal artery. *Surgery* 1975; 730-735.
7. Montes X.L.T., et al. Renal artery thrombosis occurring in an adult with idiopathic nephrotic syndrome: Results of local treatment with streptokinase. *Clin Nephrology* 1979; 12: 90-92.
8. Olin J.W., et al. Thrombolytic therapy for renal artery occlusions: A preliminary report. *Cleve Clin J. Med.* 1989; 56:432-438.
9. Rozenblit G., et al. Thrombolysis and renal angioplasty. *Radiology* 1986; 161 (P): 104. (Abstract).
10. Skinner R.E., et al. Recovery of function in a solitary kidney after intraarterial thrombolytic therapy. *J. Urol.* 1989; 141: 108-110.
11. Sniderman K.W., Sos T.A. Percutaneous transluminal recanalization and dilatation of totally occluded renal arteries. *Radiology* 1982; 142: 607-610.
12. Sos T.A., Pickering T.G. Pickering T.G. Percutaneous arterial dilatation for renovascular hypertension. In: *Current Therapy in Vascular Surgery.* Ernst C.B., Stanley J.C., (Eds), Decker B. (Pub). Philadelphia 1987; 358-363.
13. Van Breda A. Thrombolysis and percutaneous transluminal angioplasty in acute renal artery occlusion. *Radiology* 1989; 170: 1103 (Abstract).
14. Zajko A.B., et al. Percutaneous transluminal angioplasty and fibrinolytic therapy for renal allograft arterial stenosis and thrombosis. *Transplantation* 1982; 33: 447-450.
15. Zinman L., Libertino J.A. Revascularization of the chronic totally occluded renal artery with restoration of renal function. *J. Urol.* 1979; 118: 517-521.



## Tibial Angioplasty

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Since Dotter and Judkins published the first description of percutaneous transluminal angioplasty (PTA) in 1964, many atherosclerotic lesions of the iliac artery or superficial femoral artery (SFA) have been successfully treated. However, early results of popliteal artery angioplasty were not favorable. Zeitler initially stated that four out of nine procedures suffered from complications resulting in clinical deterioration due to mechanically induced spasm with resultant occlusion of the artery. However, such spasm can be effectively prevented with antispasmodic agents. Although Zeitler and others who were initially pessimistic about popliteal artery angioplasty have clearly changed their minds, and popliteal angioplasty today is a routine procedure, many still regard angioplasty of the infrapopliteal arteries as a technically difficult and relatively dangerous procedure with many potential complications, which should be reserved for limb salvage. Just as the initial results of popliteal angioplasty were disappointing, so were the early results of infrapopliteal angioplasty. Recent refinements in catheter technology, guidewire technology and angiographic techniques as well as imaging modalities have made infrapopliteal angioplasty easier and less dangerous to perform with fewer complications and therefore more widely applicable.

### PATIENT SELECTION

Up until a few years ago, only patients with rest pain or tissue damage and possible limb loss were considered to be suitable candidates for infrapopliteal angioplasty. The primary reasons for this were the widespread but mistaken impression that infrapopliteal angioplasty has poor results, the complication rate is high and the outcome of complications inevitably results in limb loss. Since infrapopliteal arteries are essentially end arteries and particularly in patients who have threatened limbs tend to have few collateral sources, this is indeed a possibility. On the other hand, the pioneering work of Schwarten demonstrated that infrapopliteal angioplasty is indeed safe and successful if one uses smaller and more flexible catheters and guidewires, and meticulous angiographic techniques. Our understanding of

pharmacology and complications related to spasm and thrombosis have also improved so that complications related to spasm and diminished flow have been markedly reduced. As improvements in infrapopliteal angioplasty occurred so have improvements been made in the techniques and results of distal bypass surgery. Both the non reversed in-situ vein grafts as well as the reversed bypass grafts have improved dramatically in initial technical success and long term patency. While complications of angioplasty such as distal spasm thrombosis dissection or occlusion are still relatively difficult to treat, however in many instances antispasmodic, spasmolytic and fibrinolytic drugs can be used percutaneously and if these fail, bypass procedures even emergently or urgently, can salvage these limbs. Because of these improvements many now consider patients with severe claudication in whom angioplasty or bypass of more proximal inflow vessels is not enough to relieve their symptoms, suitable candidates for angioplasty of infrapopliteal vessels particularly if these vessels are easily accessible and not technically extremely difficult.

### TECHNICAL CONSIDERATIONS

#### Puncture site:

In the vast majority of patients lesions of the infrapopliteal and popliteal arteries are approached by an antegrade ipsilateral femoral puncture. In a small number of patients, particularly those with popliteal or proximal infrapopliteal lesions these can be approached by a contralateral retrograde femoral puncture around the aortic bifurcation. This is particularly useful in very obese patients in whom an antegrade puncture would be difficult if not impossible.

#### Catheter Selection:

Most lesions of the infrapopliteal arteries can be dilated by 2-4 mm diameter and 2.4 cm log balloons. These are now widely available from several manufacturers on catheter shafts ranging from 3.7-4.7 inches in diameter and capable of accepting 0.018 inch guidewires. The balloons have a very low profile in their deflated and wrapped configuration. The 0.018 inch guidewires have very

soft steerable and highly radiopaque tips. The major limitation of the techniques is that all of these catheters have very thin and flexible shafts with low axial stiffness. If the infrapopliteal arteries are diffusely calcified or severely diseased, the balloon catheters may not follow the wires through the lesions. In many cases they accordion and cannot be advanced. In these cases balloons on a wire such as the "Tegwire" and others are used under these circumstances. The disadvantages of these balloons on a wire are that they consist of a single lumen hollow steel shaft which kinks, bends and breaks easily although it has great axial stiffness. The other major disadvantages of these balloons on a wire are that they do not have a second lumen for the guidewire and/or test injection of contrast, pharmaceuticals or pressure measurement. Their position in the vessels, whether intraluminal or dissecting is difficult to establish since the shaft stiffness gives relatively little tactile feedback and roadmapping from the femoral sheath is difficult unless a 6-French long sheath is used extending to the popliteal artery for their introduction. The catheter is currently under development which would combine the advantages of the double lumen, coaxial infrapopliteal balloon catheters with the stiffer shaft of the balloons on a wire. This catheter is made from a flexible synthetic material with a 3,7-French shaft diameter with axial stiffness close to that of the balloons on a wire but with a double lumen construction and a shaft which does not kink or break.

#### Pharmacological adjuncts:

We recommend 10,000 units of parenteral heparin, 100-300 mcgm aliquotes in intraarterial nitroglycerin as often as necessary. The use of an activated clotting time (ACT) unit to determine the level of anticoagulation is very useful. Adequate anticoagulation and spasmolytic medications prevent and diminish incidences of spasm and subsequent dissection and thrombosis.

#### **TECHIQUE OF INFRAPOPLITEAL ANGIOPLASTY**

1. Antegrade femoral puncture with a 4 or 5-French intraarterial sheath. (Rarely retrograde femoral approach around the aortic bifurcation as previously discussed).
2. If extremely tight critical SFA or popliteal stenoses are present these should be dilated prior to tibial angioplasty with conventional .035" guidewires and 5-Frech balloon catheters.

3. If no severe lesions of the SFA are present, a combination of the tibial balloon catheter and the 0.018" guidewire are introduced together through the arterial sheath and advanced to the popliteal artery with or without the use roadmapping as appropriate.
4. Roadmapping, if available, is extremely useful for entering the infrapopliteal arteries, particularly the anterior tibial artery. The guidewire is advanced either together with the catheter with at least 5 cm of the guidewire leading or the guidewire is advanced initially and then the catheter over it through the stenoses. If severe stenoses which prohibit further advancement of the catheter are encountered, then predilatation of the proximal lesions may be necessary in order to free up the catheter and to allow it to advance distally. If the catheter still cannot be advanced over the guidewire then a balloon on a wire system may be necessary. This system can either be used with a 6-French guiding catheter or by itself through the femoral sheath with roadmapping.
5. In occlusions of the infrapopliteal arteries, the standard .018" platinum tip soft guidewires may not be rigid enough to traverse the occlusions. In these cases a 0.018" glidewire may will be successful. This advantage of these 0.018" glidewires is that they are realtively soft and a combination of a soft guidewire and soft tibial balloon catheter may be difficult to advance through the occlusion.

#### **RESULTS OF INFRAPOPLITEAL ANGIOPLASTY**

While early reports of infrapopliteal angioplasty in a small number of patients, including our work in 1980 were cautiously optimistic, subsequent improvements in technology and techniques as well as our understanding of the possible complications and/or avoidance and the use of adjunctive medications has brought about much improved results.

The results of several recent large series of infrapopliteal angioplasty are summarized below.

Our experience at the New York Hospital has been one of rapid growth in the use of infrapopliteal angioplasty. In 1987 we treated five limbs whereas in 1991, thirty-six for infrapopliteal disease. In almost three-quarters of the patients, tandem lesions

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in the superficial femoral or popliteal arteries were

**INFRAPOPLITEAL PTA  
DEMOGRAPHICS**

	Pt/Limb/Vessel	Age (mean)	Sex M/F	Smoker %	Diabetes %	CAD%
Schwarten	98/114/145	37-86 (67)	64/34	95 (97)	59 (60)	NA
Bakal	53/ 57/ 76	36-96 (70)	30/23	25 (66)	45 (85)	35 (66)
Brown	11/ 12/ 16	42-89 (66)	7/4	7 (64)	10 (91)	NA

**RESULTS**

	1°Success %		1° Clinical Balloon Success %	1° Total Clinical Balloon Success %	Complications Major/Minor
	Catheter	Balloon			
Schwarten	45 (84)	141 (97)	141 (97)	100 (88)	2/?
Bakal	4 (29)	37 (86)	28 (78)	28 (67)	7/36
Brown		9 (75)	8 (89)	8 (67)	5/?

dilated "enroute". Our initial technical success was greater in single or focal stenoses as compared with infrapopliteal vessels with complete occlusion.

**RESULTS**

	Balloon Limited Salvage (%)	Length of follow-up (mos)
Schwarten	32/37 (88)	24
Bakal	28/42 (67)	?
Brown	6/9 (67)	1-22

**TABLE 1. INFRAPOPLITEAL PTA NYH-CUM 1/87-12/91**

	PATIENTS	LIMS	TECHNICAL SUCCESS	TANDEM VESSELS DILATED
1987	5	5	4	3
1988	2	3	1	3
1989	14	15	13	15
1990	14	18	13	10
1991	34	36	33	29

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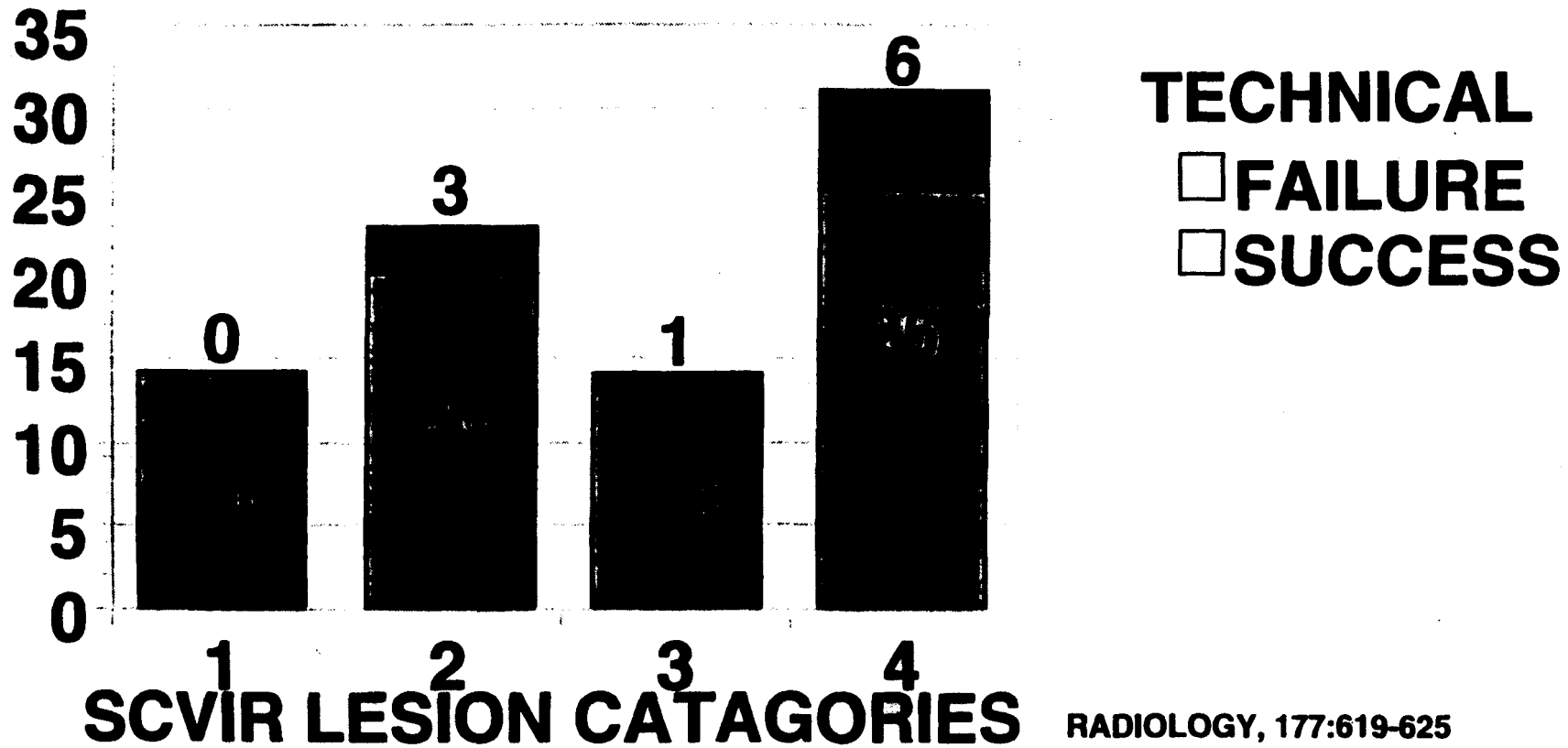
	69	77	67 (87%)	55 (71%)
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**TABLE 2. INFRAPLOPLITEAL PTA NYH-CUM 1/87-4/91**

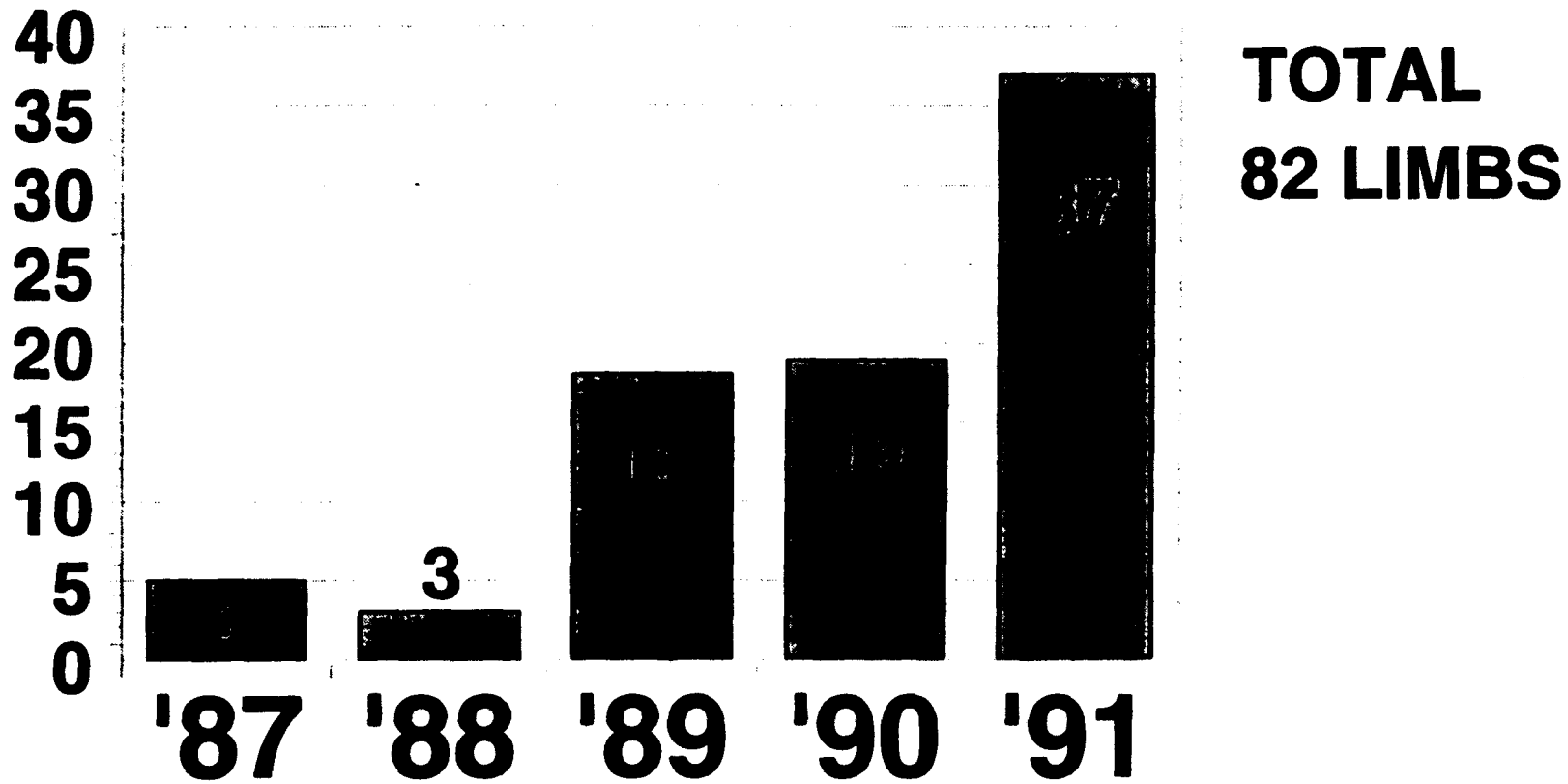
<b>\$ STENOSES</b>	<b>LIMBS</b>	<b>TECHNICAL SUCESS (%)</b>
1	13	13 (100)
2-3	15	15 (100)
>3	17	15 (88)
OCCLUSION	9	4 (44)
<b>TOTAL</b>	<b>54</b>	<b>47 (85)</b>

# INFRAPOPLITEAL PTA NYH-CUMC 1/87-12/91

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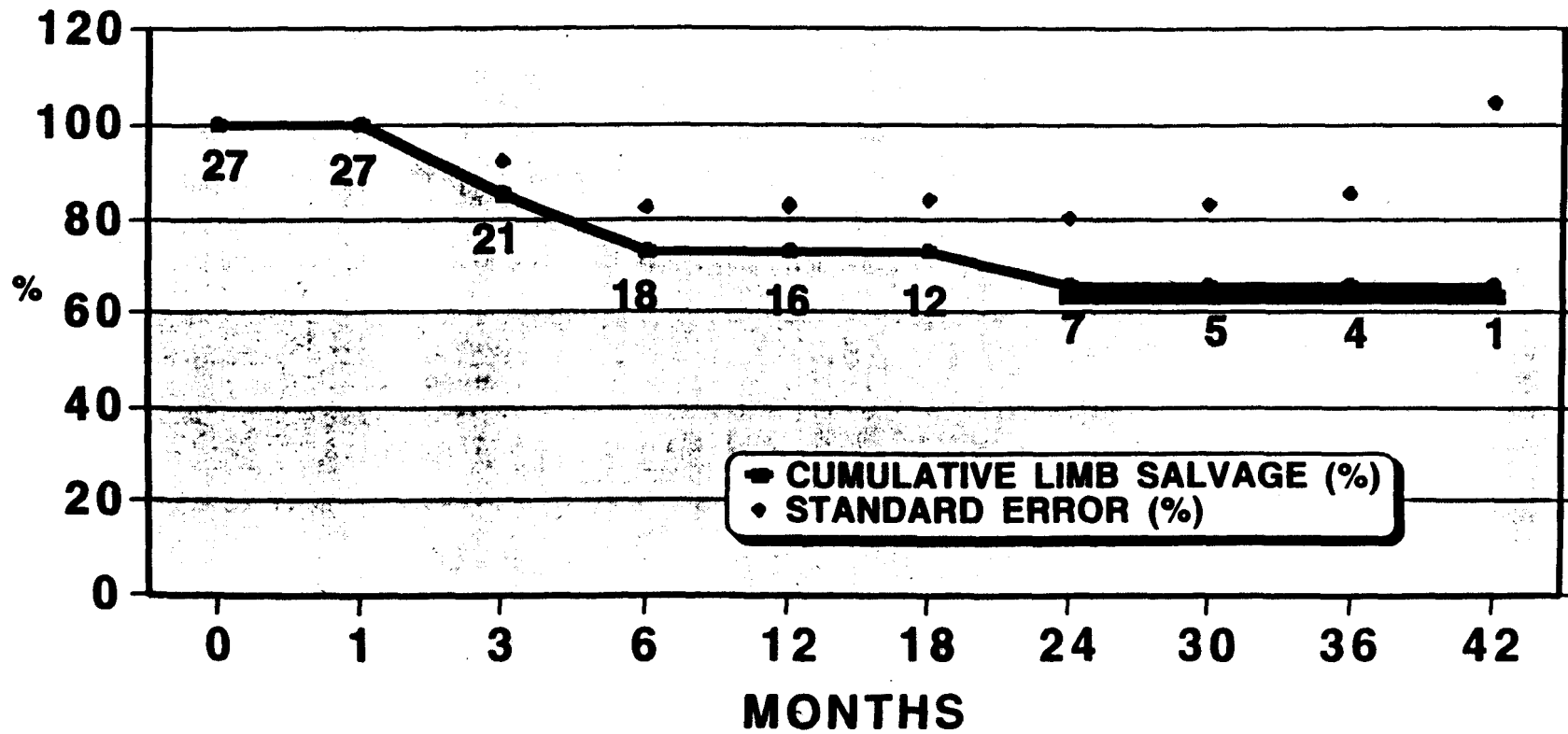


# INFRAPOPLITEAL PTA NYH-CUMC 1/87-12/91



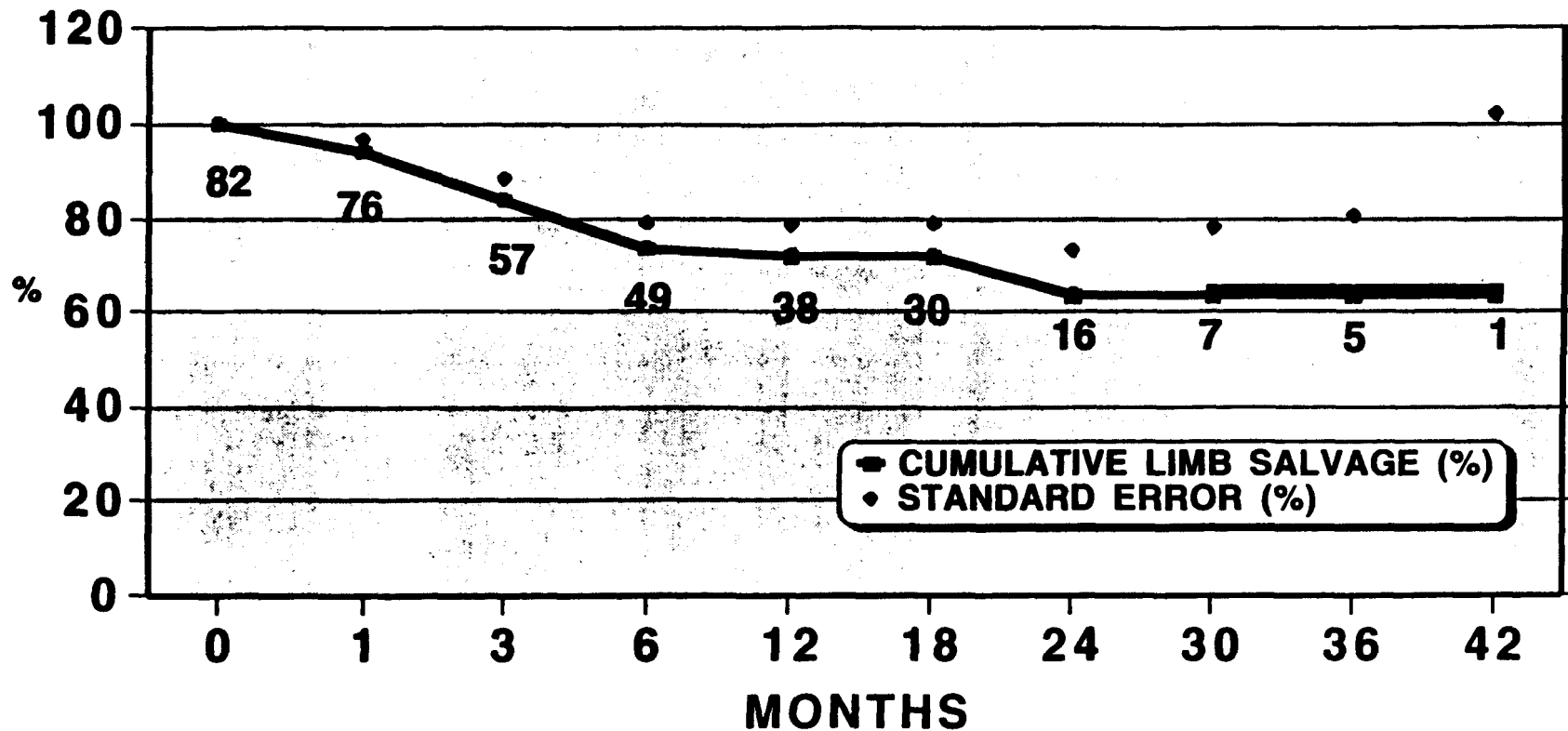
# INFRAPOPLITEAL PTA NYH-CUMC 1/87-12/91

## SUCCESSFUL TIBIAL PTA IN NONDIABETICS



# INFRAPOPLITEAL PTA NYH-CUMC 1/87-12/91

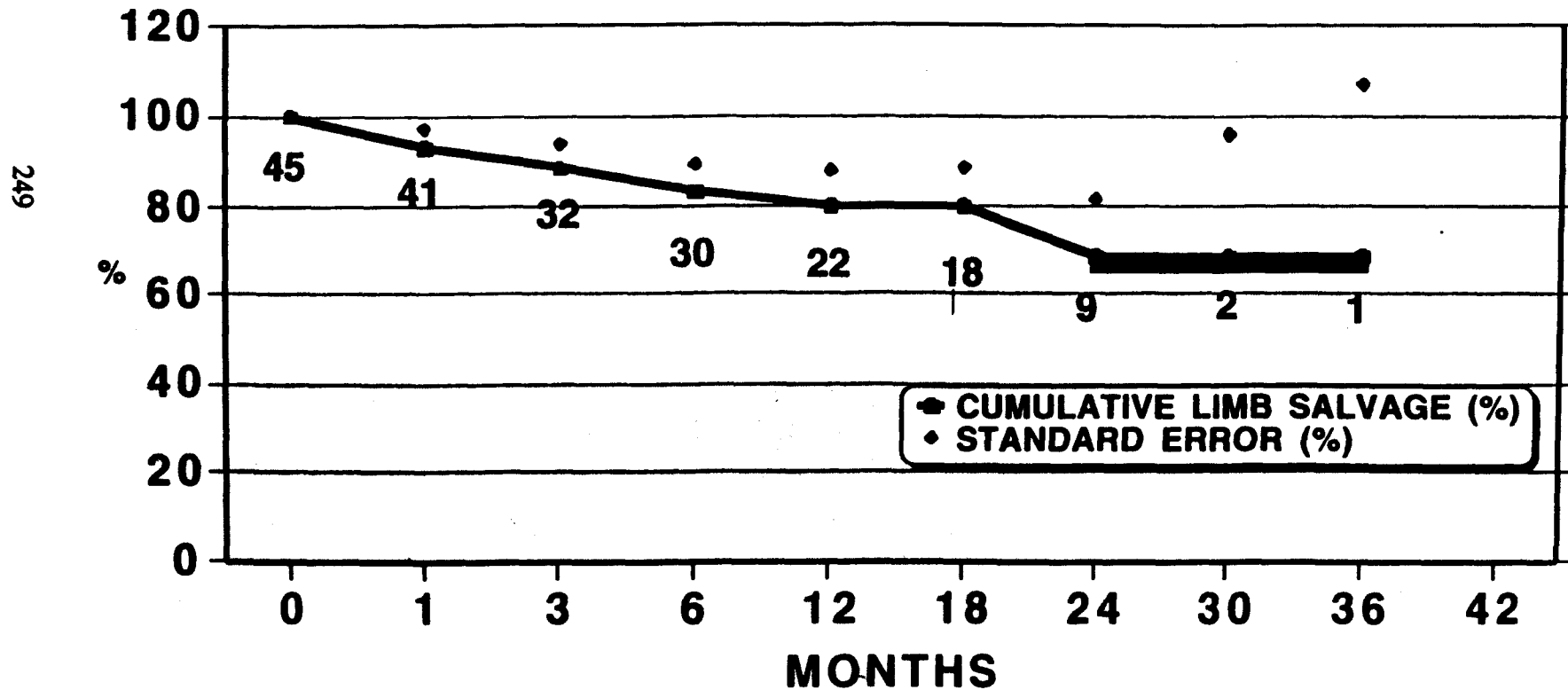
## ALL ATTEMPTED TIBIAL PTAS





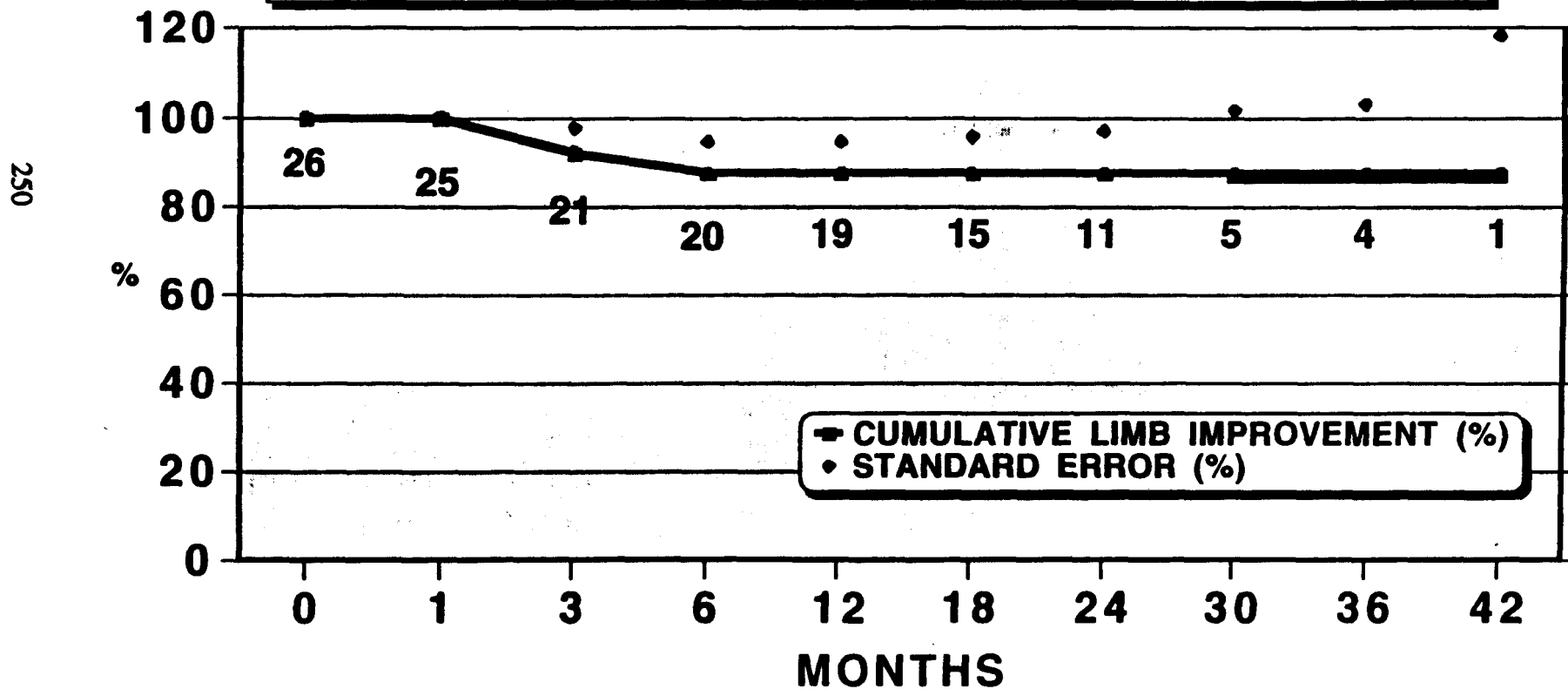
# INFRAPOPLITEAL PTA NYH-CUMC 1/87-12/91

## TIBIAL PTA IN DIABETICS



# INFRAPOPLITEAL PTA NYH-CUMC 1/87-12/91

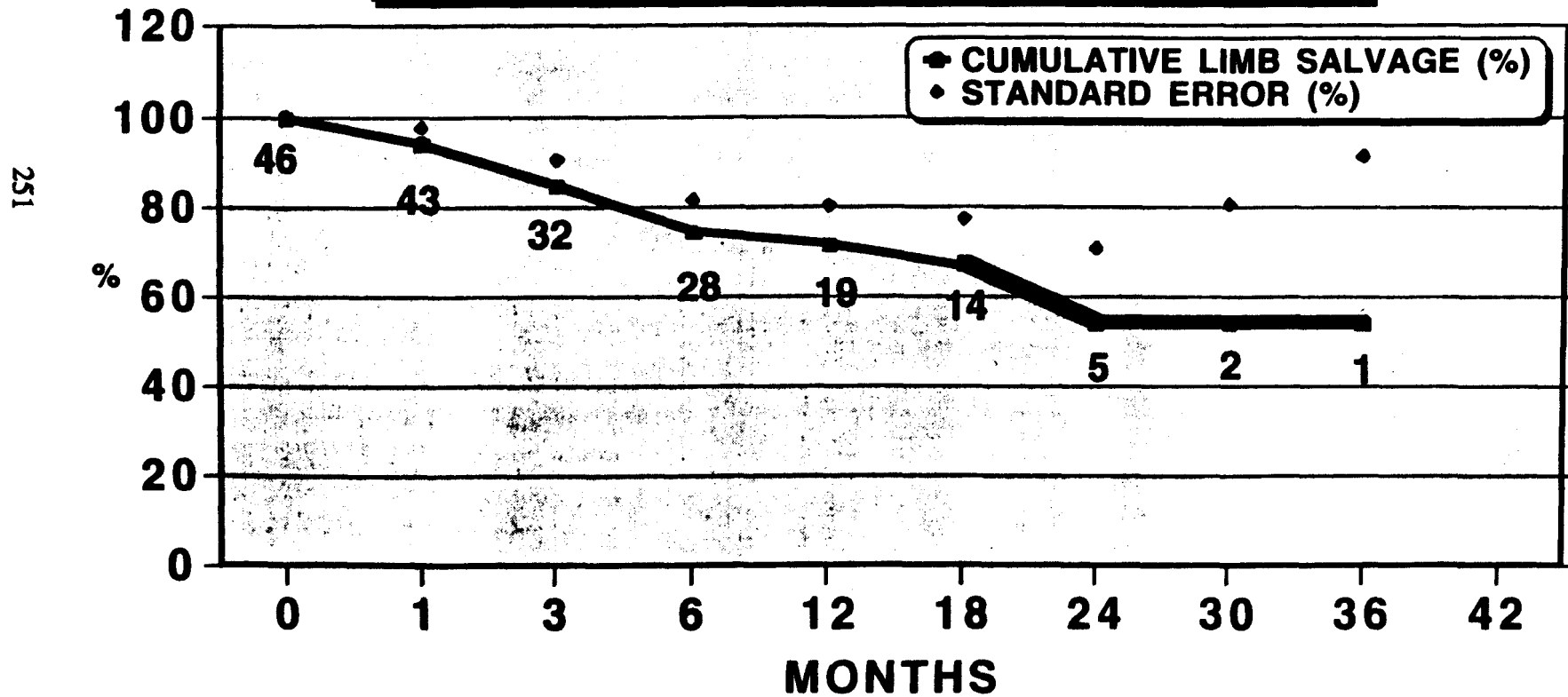
**SUCCESSFUL TIBIAL PTA IN PATIENTS  
WITH RESTPAIN, SEVERE CLAUDICATION, ACUTE ISCHEMIA**



# INFRAPOPLITEAL PTA

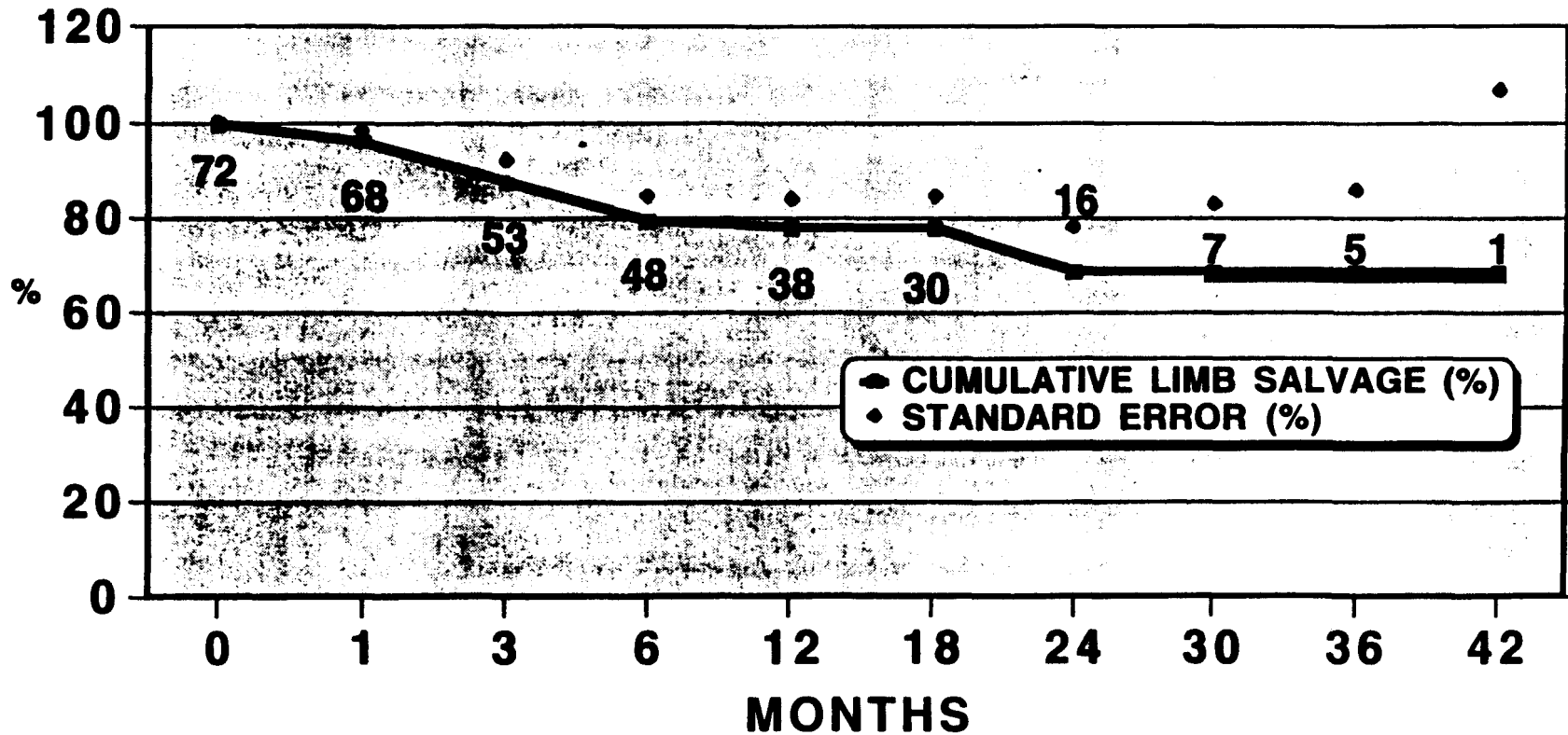
## NYH-CUMC 1/87-12/91

LIFETABLE OF SUCCESSFUL TIBIAL PTA  
IN PATIENTS WITH TISSUE LOSS



# INFRAPOPLITEAL PTA NYH-CUMC 1/87-12/91

## TECHNICALLY SUCCESSFUL TIBIAL PTAS



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##### References.

1. Tamura S., Sniderman K.W., Beinart C., Sos T.A. Percutaneous transluminal angioplasty of the popliteal artery and its branches. *Radiology* 1982; 143: 645-648.
  2. Brown K.T., Schoenberg N.Y., Moore E.D., Saddekni S. Percutaneous transluminal angioplasty of infrapopliteal vessels: preliminary results and technical considerations. *Radiology* 1988; 169: 75-78.
  3. Schwarten D.E., Cutcliff W.B. Arterial occlusive disease below the knee: treatment with percutaneous transluminal angioplasty performed with low profile catheters and steerable guide wires. *Radiology* 1988; 169: 71-74.
  4. Bakal D.W., Sprayregen S., Scheinbaum K., Cynamon J., Veith F.J. Percutaneous transluminal angioplasty of the infrapopliteal arteries: Results in 53 patients. *A.J.R.* 1990; 154: 171-174.
  5. Spreayregen S., Sniderman K.W., Sos T.A., Vieux U., Singer A., Veith F.J. Popliteal artery branches: percutaneous transluminal angioplasty. *A.J.R.* 1980; 135: 945-950.
  6. Krepel V.M., van Andel G.P., van Erp W.F.M., Breslau P.J. Percutaneous transluminal angioplasty of the femoropopliteal artery: initial and long-term results. *Radiology* 1985; 156: 325-328.
  7. Greenfield A.J., Femoral, popliteal, and tibial arteries: percutaneous transluminal angioplasty. *A.J.R.* 1980; 135: 927-935.
  8. Leather R.P., Karmody A.M. In-situ saphenous vein arterial bypass for the treatment of limb ischemia. In: Mannick J.A., ed. *Advances in Surgery*. Vol. 19, Chicago: Year Book Medical 1986; 175-219.
  9. Taylor L.M. JR, Edwards J.M., Phinney E.S., Porter J.M. Reversed vein by-pass to infrapopliteal arteries: modern results are superior to or equivalent to in-situ by-pass for patency and for vein utilization. *Am. Surg.* 1987; 205: 90-97.
  10. Wierny L., Plass R., Porstmann W. Long term results in 100 consecutive patients treated by transluminal angioplasty. *Radiology* 1974; 112: 543-548.
  11. Fontaine R., Kieny R., Gangloff J.M., et al. Long-term results of restorative arterial surgery in obstructive diseases of the arteries. *J. Cardiovasc. Surg. (Torino)* 1964; 5: 463-472.
  12. Sobbe A., Martin M., Trubestein G. Besondere Aspekte der transluminalen Katheterrekanalisation und ihrer Rezidivbehandlung. *R.O.E.F.O.* 1973; 118: 682-690(Ger).
  13. Dotter C.T. Transluminal angioplasty: a long view. *Radiology* 1980; 135: 561-564.
  14. Zeitler E. Complications in and after PTR. In: Zeitler E., Gruntzig A., Schoop W., eds. *Percutaneous vascular recanalization. Technique, Application, Clinical Results*. Berlin: Springer, 1978: 120-125.
1. New York Hospital-Cornell University Medical Center, New York, N.Y.

## Long-Term Results of Stent Treatment of Iliac Artery Stenoses and Occlusions with Flexible Tantalum Stent

E.P. STRECKER, B. HAGEN, D. LIERMANN, I. BOOS

### INTRODUCTION

Stent therapy of stenoses and occlusions of the iliac arteries has proved to be a valuable adjunct to percutaneous transluminal angioplasty (PTA), presenting long-term patency rates between 64 and 95% for balloon-expandable or self-expandable stents (1-4). Although long-term patency rates are favorable, the success of stent therapy is diminished by acute thrombotic stent occlusions and late restenoses.

Only a clinical long-term study will provide more information about the success of stent therapy; the knowledge of risk factors will enable the interventional radiologist to prevent complications. This prospective multicenter study discloses new information about the benefits of iliac artery stenting with special regard to complications and risk factors, especially for long iliac artery occlusions.

#### Patients and methods:

The technical properties of the balloon-expandable tantalum stent (Type Strecker, Boston Scientific Inc., Watertown, M.A., USA) and the insertion technique were described in detail before (5,6). Briefly, the stent, balloon expandable and implantable through an introducer sheath, consists of a cylindrical, electropolished tantalum filament knit with a wire diameter of 0.1mm. This prosthesis is flexible and elastic within certain limits in both the expanded and the non-expanded states. The loosely connected wire loops give the stent an inherent flexibility, allowing the introduction of the catheter stent assembly through curved arteries, and the implantation into arteries leading over the hip or knee joint (Fig. 1).

Correct positioning of the stent in the area to be treated is facilitated by the following facts: shortening of the stent during dilatation will be compensated by the design with longitudinal compression of the overlapping loops in the non-expanded state. The stent is well visible under

fluoroscopy, due to the radiodensity of tantalum, being in this regard superior to any stainless-steel prostheses.

Recently improved catheter-stent-assemblies with a 5F-PTA-catheter, of a balloon width up to 7mm can be introduced through a 6.5F-introducer-sheath (Peter von Berg, Kirchseeon, Germany), whereas wider stents (9mm) will need an 8F-sheath and stents with a diameter of 11mm will need a 10F-introducer-sheath. Stents available have a length of 4 or 8 cm.

239 patients with atherosclerotic disease treated with iliac artery stents were included in this study.

In 150 patients lesions of the common iliac artery were stented, including 25 patients having received kissing stents to treat atherosclerotic disease of the aorto-iliac bifurcation (Fig. 2). In 89 patients lesions of the external iliac artery were treated. There were 56 occlusions (maximum length 200.0 cm, mean 6.8 cm), and 183 stenoses (maximum length 16.0 cm, mean 2.7 cm). In 33 patients, 2 or more stents (maximally 4 stents) were implanted consecutively, from proximally to distally, with their ends overlapping by a few millimeters. Indications for iliac artery stenting were insufficient PTA results due to dissection (39%), elastic recoil (37%) or acute occlusion post PTA (2%). Long artery occlusions were regarded as primary indications (25%).

During the preceding PTA procedure, 5000 IU of heparin were applied intra-arterially as a bolus, and in the case of stent implantation, additional 2000-5000 IU were given, depending on the patient's weight, the duration of the procedure and the length of the treated arterial site. Immediately after stent implantation, heparin was applied intravenously by a perfusor to increase the partial thrombin time to two or three times the normal. Heparin treatment lasted up to three days; 100 mg of aspirin were given daily ad infinitum to prevent thrombus formation by platelet aggregation inhibition.

Clinical symptoms before and after treatment were determined on the basis of Fontaine's classification. The Doppler sonographic ankle-arm indexes (AAI) were evaluated immediately prior to treatment, two days after, six months later and, finally, at yearly intervals. DSA controls were performed when clinical symptoms or the AAI deteriorated during the follow-up period.

## **RESULTS**

### **Primary Results:**

In all cases, stents could be successfully implanted.

The Doppler AAI increased significantly immediately after stenting by more than 0.15 in all patients.

83% of the patients showed an improvement of their clinical symptoms. 16% of the patients remained in their clinical stage, and 1 patient showed a deterioration of clinical symptoms within the first weeks after stenting. Patients showing no improvement of their clinical symptoms required a therapy of additional lesions located more distally.

### **Complications:**

In 11 patients, thrombotic stent occlusion occurred within the first ten days after stenting.

Such occlusion could be treated with local fibrinolysis in 8 patients.

Distal embolization of thrombus material to distal arteries was seen in 7 patients, this could also be treated with local fibrinolysis or aspiration thrombectomy in 6 of them.

The incidence of early thrombotic stent occlusion and peripheral thromboembolism was especially high in patients with long arterial occlusions.

When there was an immediate partial stent compression due to a too strong arterial wall recoil effect, balloon dilation was repeated eventually with a shorter and wider balloon over a longer time period of 1 to 5 minutes. In one case, two stents were implanted in an overlapping fashion so as to obtain a higher stent stability.

Surgical intervention was required in 3 patients with acute thrombotic occlusion and in 1 of the patients with distal embolization. 9 patients developed sizable hematoma at the puncture site

with the result that they required prolonged bed rest. 3 patients developed a false aneurysm, which was treated by compression with the head of a color doppler in 2 cases. The remaining patient required surgical intervention.

One patient in whom arterial rupture occurred during balloon inflation underwent immediate bypass surgery, after the bleeding could be stopped by dilatation of a PTA balloon at the ruptured site.

A comparison between patients with iliac stenosis and those with iliac occlusion showed that the incidence of acute thrombotic stent occlusion and distal embolization was especially high in patients in whom iliac artery occlusion indicated stenting. Moreover, acute thrombotic stent reocclusion occurred significantly more often in patients with poor run-off vessels (more than 50% stenosis of the common or superficial femoral artery).

### **Follow-up and long-term results:**

The longest observation time was 72 months, the mean being 20 months.

In 41 patients, deterioration of the Doppler AAI was observed during the follow-up period. In 28 cases this was due to stent restenosis (more than 50% of the diameter) angiographically proven between 2 and 18 months after stent implantation. 7 patients were operated with bypass procedures, 19 were treated with a repeat PTA successfully, and 2 patients did not receive any therapy because of mild symptoms only.

In the remaining 13 patients, angiography revealed patent iliac stents, but additionally, a hemodynamically relevant atherosclerotic disease located more distally.

Life table analysis showed a 54-months cumulative patency rate of 81.5% for iliac artery stents, acute complications like thrombotic stent occlusion within the first ten days after stent placement being included. The separation of stented stenoses and stented occlusions reveals a 4-year primary patency rate of 82.9% for iliac stenoses, and of 76.3% for iliac occlusions, the difference between both patency rates being statistically significant ( $p < 0.01$ ) (Fig.4).

## **DISCUSSION**

**I**n the last few years stent therapy has proved to be

a valuable support for the interventional radiologist performing PTA of the aortic bifurcation and iliac arteries (1-4). Long occlusions of the iliac arteries of the aortic bifurcation have been added as new stent indication (5-9). As an endoluminal mechanical support the stent counteracts the mechanical recoil of the elastic components still remaining after PTA. Further, wall dissection or intimal flaps are able to reduce the lumen of the arteries or even to occlude them. Vascular stenting provides successful treatment of such complications followed by good long-term success rates. Our stent study including 239 patients reveals a 54-months primary patency rate of 81.5% (Fig. 3), even being superior to the success rate of 667 patients treated solely with PTA, as published by Johnston (10) (Fig.5). In this regard it must be emphasized that our cohort includes only patients suffering from very severe arterial lesions not responding to PTA treatment, thus representing a group even less favorable than the PTA group. Richter confirms this statement, describing superior patency rate for stented iliac arteries versus arteries solely treated with PTA (11).

Especially in long occlusions of the iliac arteries and the aortic bifurcation, solely performed PTA has revealed insufficient results because there is a high incidence of acute collapse of the thrombotic material into the previously opened arterial segment, accompanied by acute thrombosis. Therefore, because of early recurrence, iliac artery occlusions were not felt to be suited for PTA therapy. However, stent therapy offers a new modality for the treatment of long iliac artery occlusions (7,8,9), providing a long-term success rate of 76,3% in our study. This patency rate is not as favorable as in stenotic lesions (Fig.4), what can be mainly explained by the higher incidence of acute thrombotic occlusions within the first ten days after stent implantation. Occlusions are usually longer than stenoses resulting in a more extensive wall injury. Further, more metallic stent material being thrombotic is introduced. Also, the surface of thrombotic material fragmented beyond the stent meshes increases thrombogenicity. In addition, thrombotic material might protrude backwards into the lumen, causing thrombosis. Therefore, a thoroughly performed anticoagulation becomes necessary after stenting of iliac artery occlusions.

Local fibrinolysis presents another modality to reopen long iliac artery occlusions. Solely local fibrinolysis has been advocated for the treatment of these arteries. Mostly performed by using the cross-over technique, the fibrinolytic agent is

injected from the contralateral side through a catheter into the thrombus. However, for a complete dissolution of the thrombus 3 to 24 hours are necessary and control angiographies must be performed repeatedly. As there is a risk of systemic side effects causing severe hemorrhages, the patient must be taken care of thoroughly. Therefore, local fibrinolysis is stressing and time-consuming method and often, PTA or stent treatment has to be performed in addition in order to achieve success (12).

However, in combination with vascular stenting, local fibrinolysis might be helpful. As performed initially, it can provide a thin channel, enabling the radiologist to pass a guide wire through the obstructed segment (Fig.2). Then, after underdilatation with a 5F-PTA-balloon, a stent can be introduced and dilated. A complete predilatation with a PTA balloon would increase the risk of peripheral thromboembolism.

Peripheral thromboembolism and acute stent thromboses, representing the main complications occurring after stent implantation into long arterial occlusions, can be treated successfully with local fibrinolysis or aspiration thrombectomy.

In conclusion, stent therapy has established a new helpful additional treatment for insufficient PTA results within the same session. Especially after stent treatment of long iliac artery occlusions, acute thrombosis can occur; this can be prevented by thoroughly performed anticoagulation therapy. In order to prevent complicating peripheral thromboembolism, an only incomplete PTA should be performed before stent implantation. In conclusion, stent therapy offers new areas for vascular interventions like the reopening of long arterial occlusions in the area of the aortic bifurcation and the iliac arteries.

**References:**

1. Strunk H.M., Schild .H., Dueber C. et al. (1993) Resobstruction after iliac artery stent placement: frequency and methods of treatment. *Radiology, Supplement to Radiology*, Vol. 189 (P), Abstract N°. 1088,293.
2. Strecker E.P., Hagen B., Liermann D. et al. (1993) Iliac and femoropopliteal vascular occlusive disease treated with flexible tantalum stents. *Cardiovasc. Intervent. Radiol.* 16:158.
3. Palmaz J.C., Richter G.M., Noeldge G. et al. (1988) Intraluminal stents in atherosclerotic iliac artery stenosis: preliminary report of a multicenter study. *Radiology* 168:727.



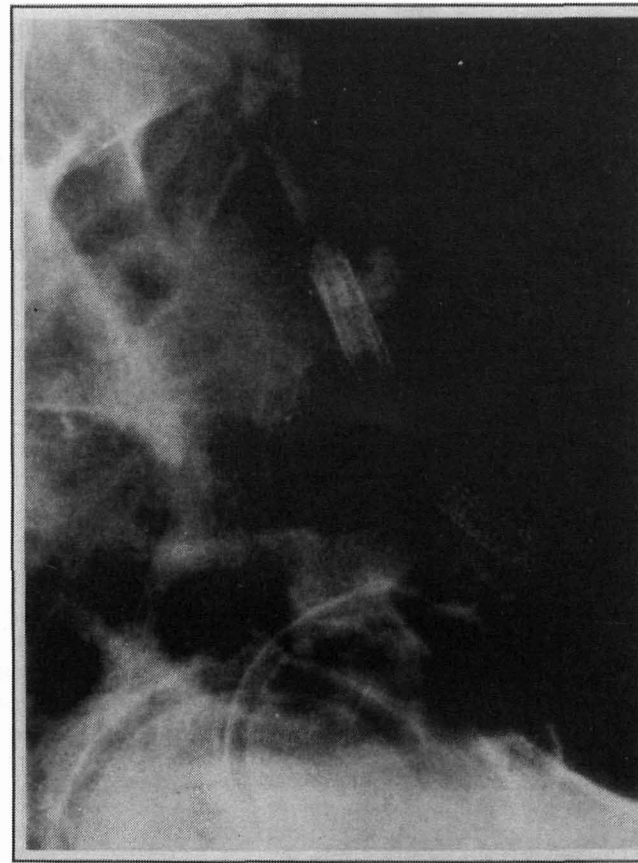
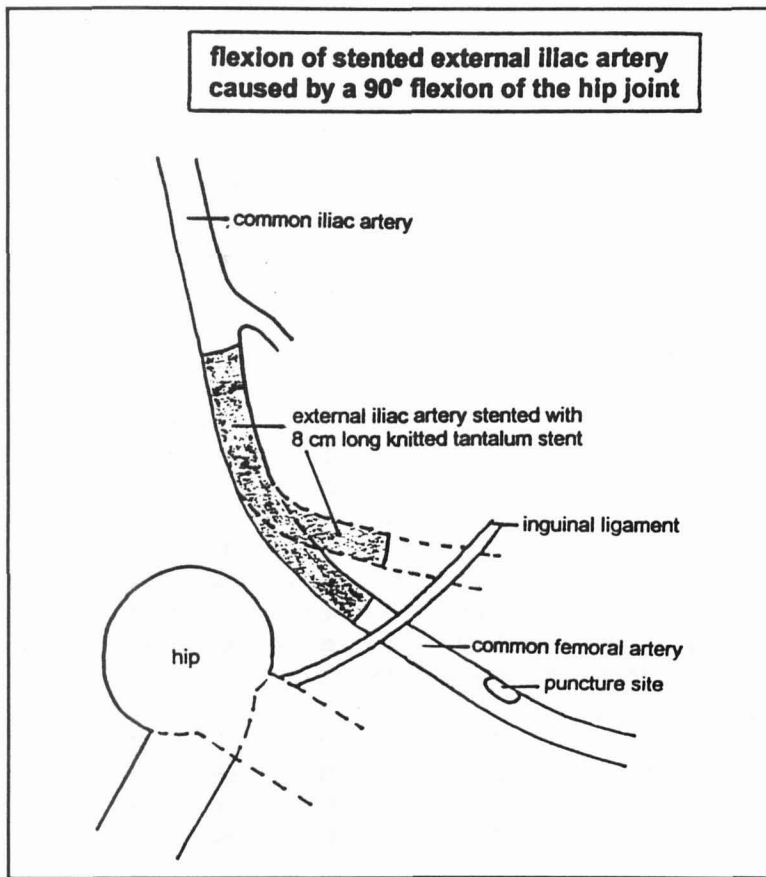
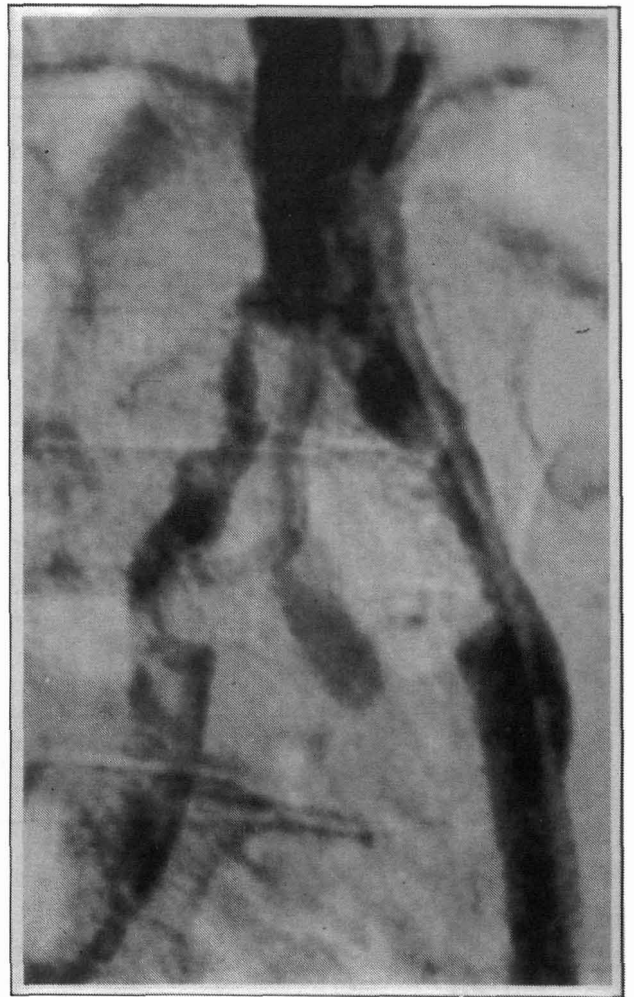
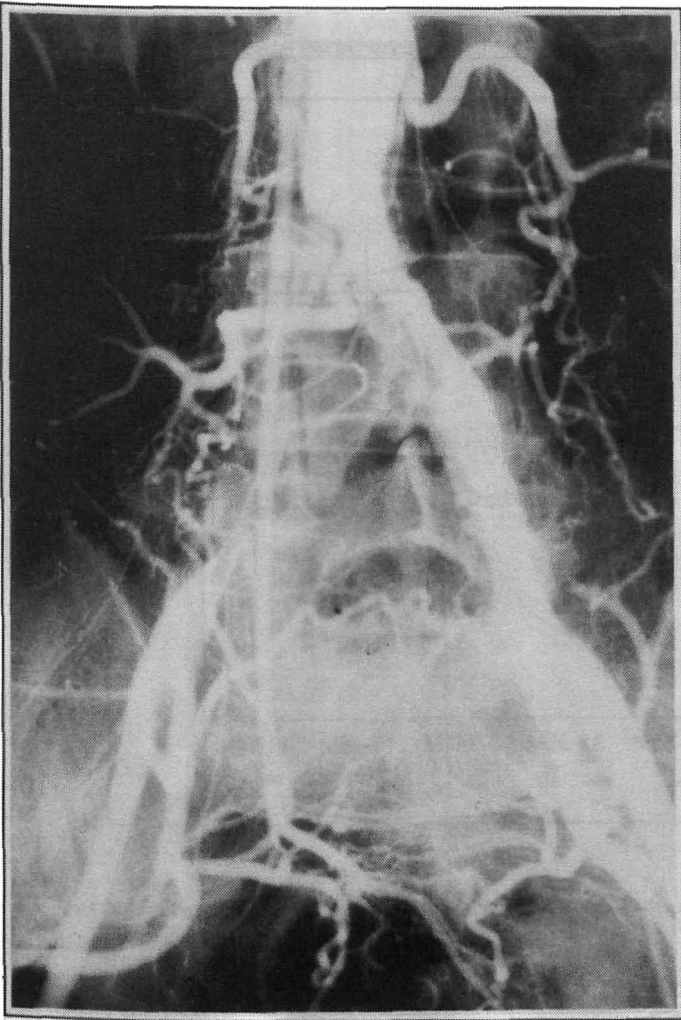


Fig. 1.a,b: Iliac arteries appear to have a straight course in the a.p. view. However, in the lateral view, the external iliac artery curves increasingly with hip flexion. Therefore, in case of a long (8cm) stent required, only flexible stents can be implanted into this anatomical site.

4. Zollikofer C.L., Antonucci F., Pfyffer M. et al. (1991) Arterial stent placement with use of the Wallstent: midterm results of clinical experiences. *Radiology* 179:449.
5. Strecker E-P., Kuhn F.P., Liermann D. et al. (1992) Strecker flexible tantalum stent. In: Kim D., Orron D.E. (eds) *Peripheral vascular imaging and intervention*. 1st ed. St. Louis Missouri: Mosby Year Book, Inc., pp 541-552.
6. Strecker E-P., Hagen B., Liermann D. et al. (1993) Treatment of atherosclerotic occlusive disease of the aortic bifurcation with balloon expandable tantalum stents. *Eur. Radiol.* 3: 536.
7. Palmaz J.C., Encarnación C.E., García O.J. et al (1991) Aortic bifurcation stenosis: treatment with intravascular stents. *JVIR* 2: 319.
8. Vorwerk D., Guenther R.W. (1990) Mechanical revascularization of occluded iliac arteries with use of self-expandable endoprosthesis. *Radiology* 175: 411.
9. Rees C.R., Palmaz J.C., García O., Roeren T., Richter G.M., Gardiner G. Jr, Schwarten D., Schatz R.A., Root H.D., Rogers W. (1989) Angioplasty and stenting of completely occluded iliac arteries. *Radiology* 172: 953.
10. Johnston K.W. (1993) Iliac arteries. reanalysis of results of balloon angioplasty. *Radiology* 186: 207.
11. Richter G.M. (1992) Erste Langzeitergebnisse der randomisierten 5-Jahres-Studie: Iliacale Stentimplantation versus PTA. *Vasa Suppl.* 35: 192.
12. Hausegger K.A., Lammer J., Klein G.E., Fluckiger F., Lafer M., Pilger E., Aschauer M. (1991) Percutaneous recanalization of pelvic artery occlusions - fibrinolysis, PTA, stents. *RoFo Fortschr. Geb. Röntgenstr. Neuen. Bildgeb. Verfahr.* 155: 550.



**Fig.2:** a) Patient with atherosclerotic disease of the aortic bifurcation: unilateral occlusion and contralateral stenosis of the common iliac artery.  
b) After local fibrinolysis of the occluded arterial segment, contrast media delineates a thin channel for the introduction of a guide-wire. After predilatation with a 5mm-PTA-balloon, a stent-catheter-assembly can be introduced.  
c) Two 8 cm long stents were implanted simultaneously on either side, according to the kissing-method.

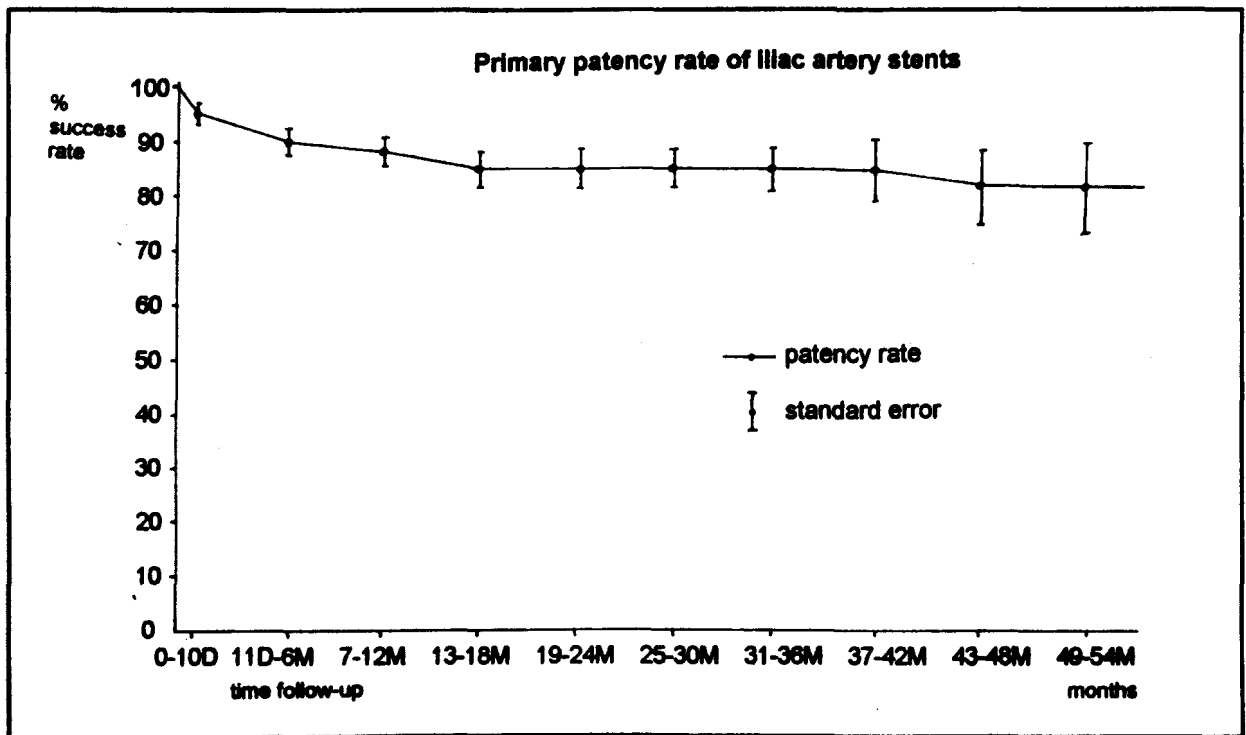


Fig.3: Cumulative patency rate (Kaplan-Meier-method) over a follow-up of 54 months for 239 stented iliac arteries including stenoses and occlusions.

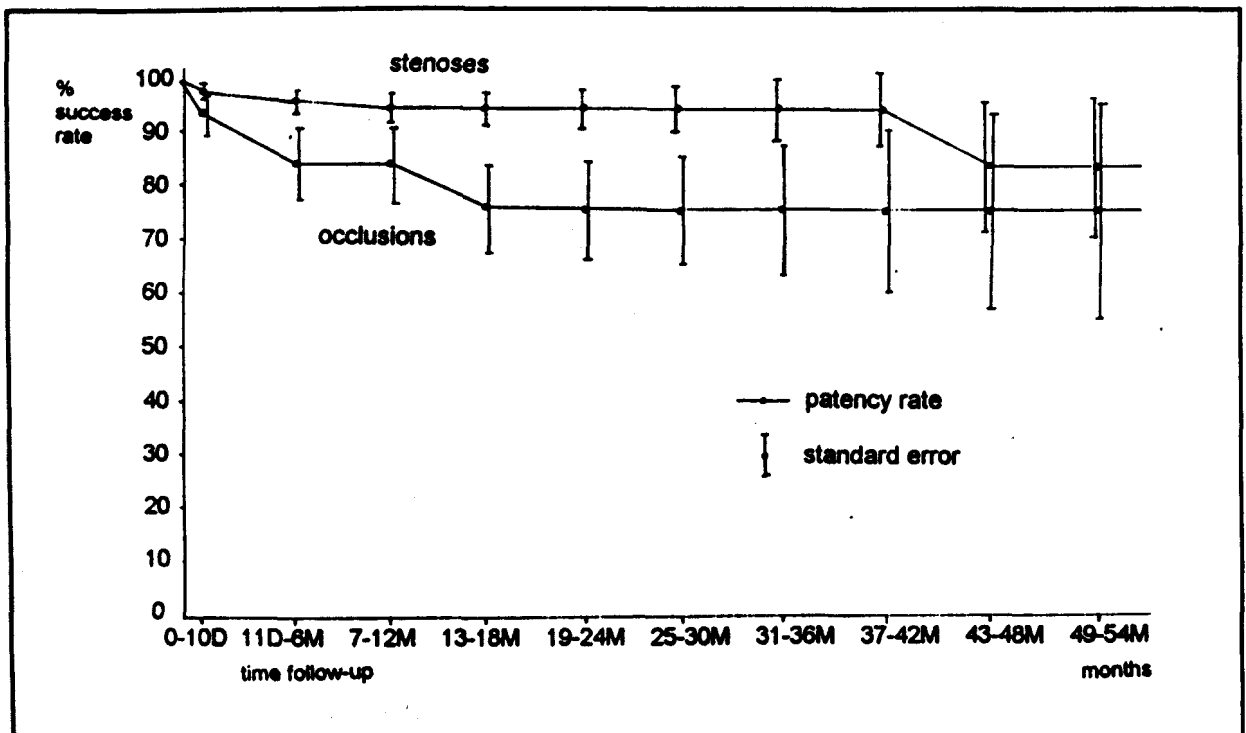
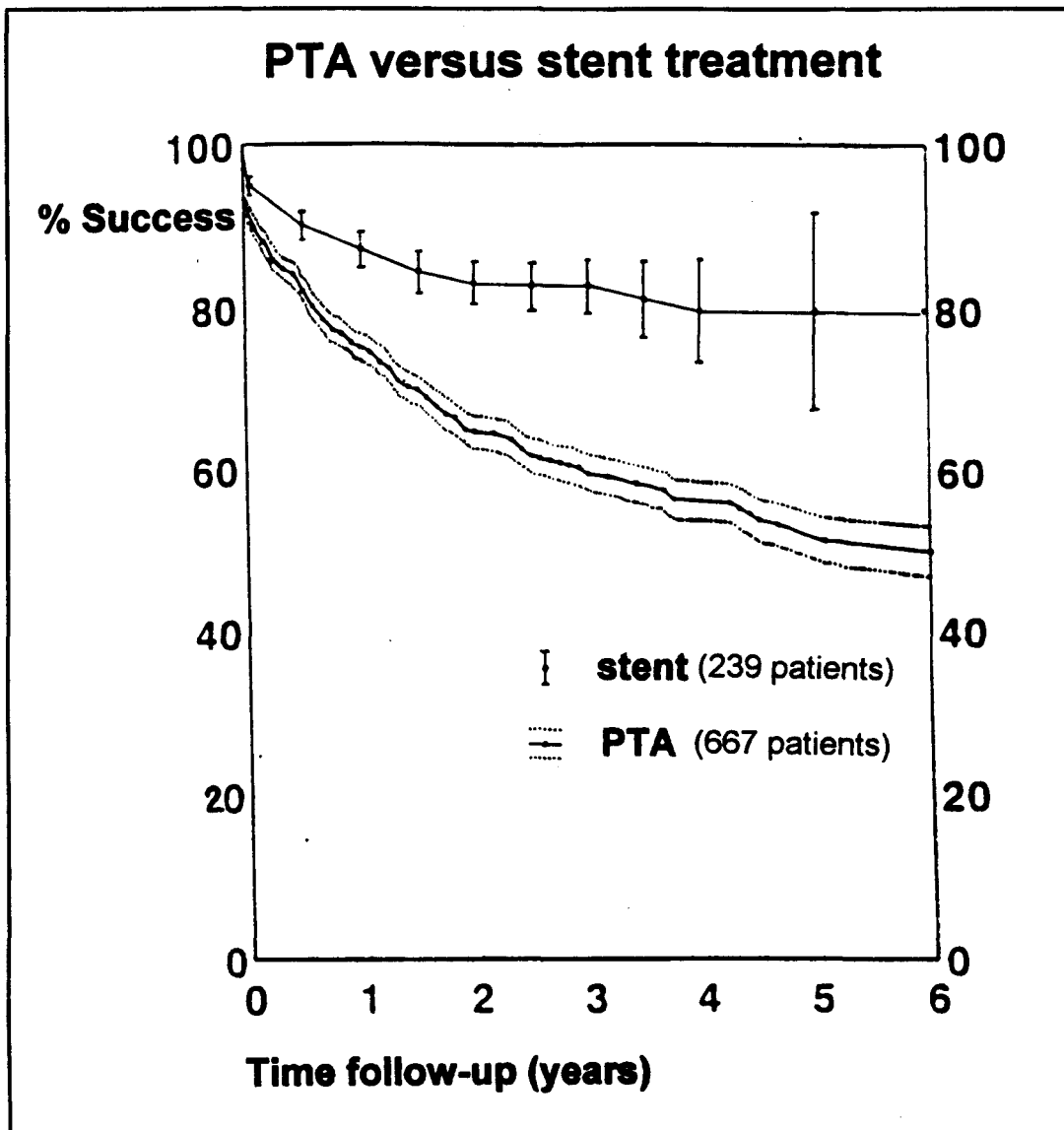


Fig.4: Cumulative patency rates over a follow-up of 54 months: stented stenoses (183 patients) versus stented occlusions (56 patients).



**Fig.5** Iliac artery occlusions and stenoses: Patency rate of iliac arteries having been stented primarily in artery occlusions or secondarily after failed PTA versus patency rate of iliac arteries solely treated with PTA (data of Johnston (10)) over an observation time of 6 years. There is a significant higher success rate for stented lesions.

## Interventional radiology

# Treatment of atherosclerotic occlusive disease of the aortic bifurcation with balloon expandable tantalum stents

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**Abstract.** Eighteen patients with atherosclerotic disease of the aorto-iliac bifurcation were treated with percutaneously implantable, balloon expandable tantalum stents. Stenting indications were percutaneous transluminal angioplasty (PTA)-resistant stenoses and long aorto-iliac occlusions. The vascular prostheses were inserted by bilateral femoral approach and dilated simultaneously, their proximal parts being deployed in parallel in the aorta and their distal parts anchored in the common iliac arteries (kissing-stent method). In aorto-iliac occlusions, local fibrinolysis was used to facilitate guide wire passage. Within 20 months (the duration of longest observation), no major acute or late complications occurred. Clinical symptoms improved immediately in all patients. Deterioration of clinical symptoms during the follow-up period was observed in five patients and was due to additional, newly developed atherosclerotic disease located distally, the bifurcation stents remaining patent.

This new type of non-invasive procedure can be regarded a safe and efficacious alternative to vascular surgery, especially in poor surgical candidates.

**Key words:** Arteries – Stents – Atherosclerotic occlusive disease – Aortic bifurcation – Percutaneous transluminal angioplasty

## Introduction

Stent therapy of atherosclerotic occlusive disease of the iliac arteries has proved to be a valuable adjunct to percutaneous transluminal angioplasty (PTA) in cases presenting insufficient results, like residual stenoses or acute re-occlusions caused by arterial wall dissections, recoil or intimal flaps [1–5]. In long iliac artery occlusions, stent therapy may be the primary therapy [20], without the necessity

of preceding balloon dilatation or fibrinolytic therapy [6–8]. A new anatomical site for non-surgical vascular interventions with stents should be the aortic bifurcation where serious atherosclerotic disease with severe stenoses or occlusions is often located.

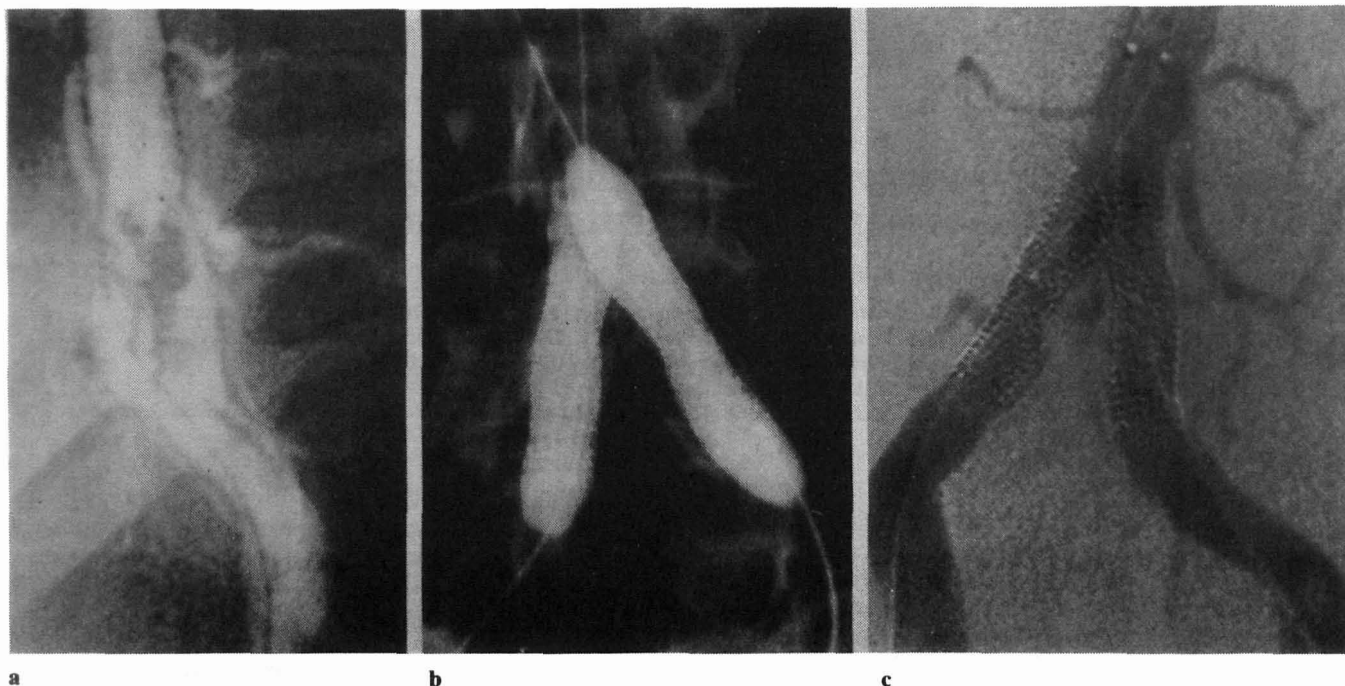
These lesions are commonly treated with major surgical procedures, (e.g. using bifurcation prosthesis) and, as these patients frequently have coexisting, complicating systemic disease, the mortality rises to 5% with a high rate of peri-operative morbidity [9–12]. Therefore, the development of a less invasive, non-surgical procedure would be beneficial, especially in poor surgical candidates.

Stenting of aortic bifurcation stenoses has been recently reported [13], but little information is currently available. Our study differs from that recently published in terms of patient population, type of disease, stent design and implantation procedure. In our patient series, with severe stenoses as well as long occlusions of the aortic bifurcation, a new method is described to treat this type of atherosclerotic disease with flexible tantalum stents forming a prosthetic bifurcation.

## Patients and methods

Eleven male and seven female patients (mean age of 61 years, range 44 to 78 years) with severe long-lasting claudication (16 patients) and rest pain (2 patients) secondary to stenoses or occlusions of the aortic bifurcation were treated (Figs. 1, 2). None of the patients had undergone previous PTA, nor previous surgical therapy at this site. All patients were current or previous smokers; additionally, 4 of them had diabetes mellitus, 10 had high blood pressure, 4 had coronary artery disease and 14 had elevated serum cholesterol (Table 1). The mean ankle-arm index was  $0.44 \pm 0.16$ .

Pre-therapeutic angiograms demonstrated bilateral common iliac artery stenoses in 10 of 18 patients; 4 patients had unilateral common iliac artery occlusion with contralateral stenosis at the aortic bifurcation and 4 patients had bilateral occlusion of the bifurcation and common iliac arteries. The mean length of stenoses was



**Fig. 1 a.** Lateral aortogram demonstrating severe stenoses due to calcified plaque formation in the distal abdominal aorta and the origins of the common iliac arteries. **b** 4-cm long stents with a diameter of 9 mm in the expanded state were inserted from either side using the femoral approach and dilated simultaneously. **c** The proximal stent ends were implanted into the lumen of the distal aorta, forming a new prosthetic aortic bifurcation located more proximally, whilst the distal parts were anchored in the common iliac arteries. A previous PTA was not performed in order to avoid distal embolization. The patient's symptoms, (severe claudication) disappeared completely

1.75 cm, with a maximum length of 3 cm. Occlusions were longer with a mean length of 14.2 cm, and a maximum length of 19 cm (Table 2). Occlusions of the aortic bifurcation extended from the distal aorta (at the origin of the inferior mesenteric artery or fifth lumbar arteries) into the common iliac arteries, down to the origin of the internal iliac arteries.

In 5 of the 18 patients, lesions were combined with calcified atherosclerotic wall disease directly located in the area of the aorto-iliac junction; in one case a large plaque was noted on the arterial divider of the bifurcation.

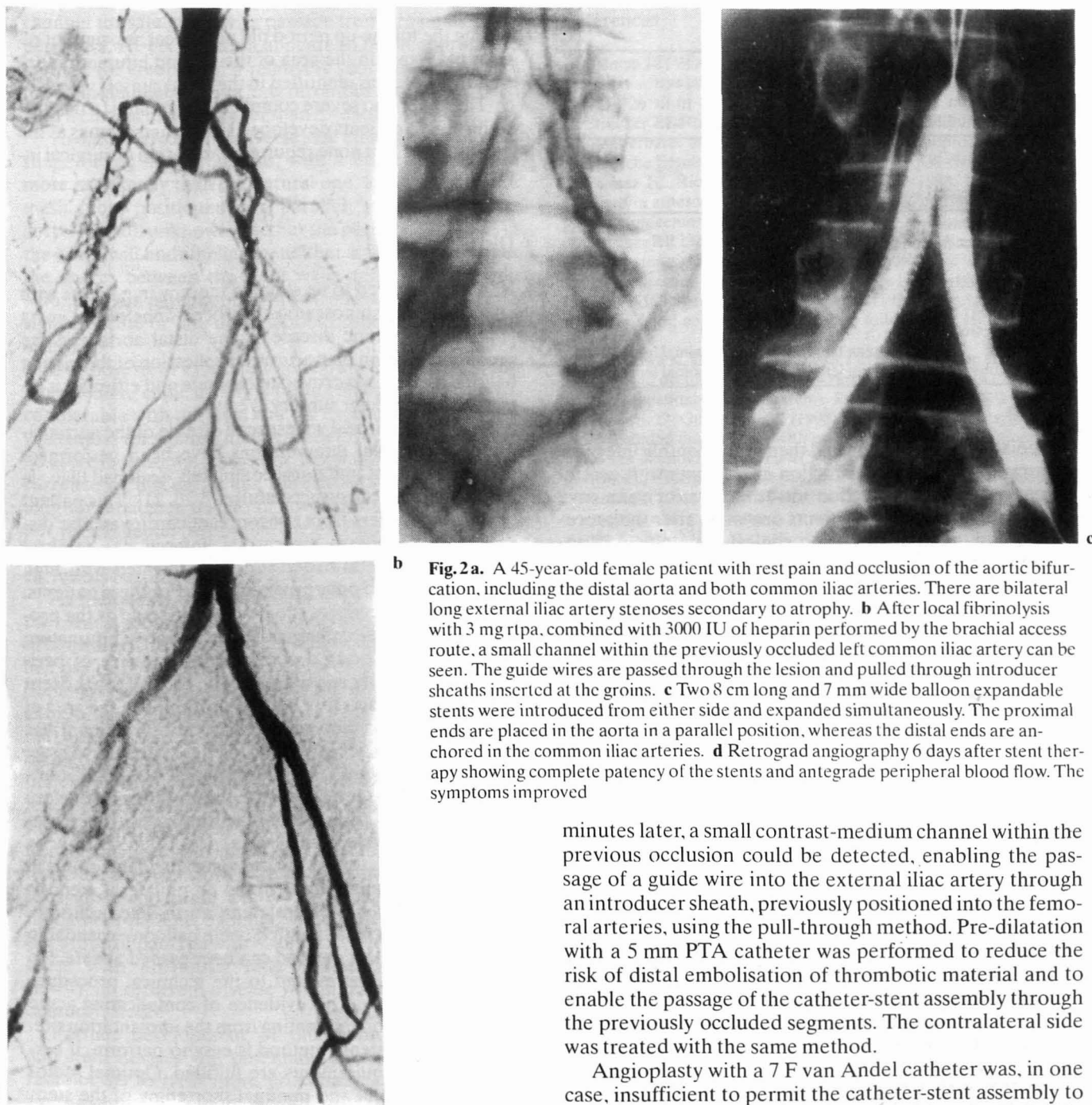
Four of the 18 patients had normal run-off vessels. Of the 14 patients with affected run-off vessels, 2 had stenoses of the external iliac arteries only, 2 had stenoses of the external iliac artery combined with occlusion and stenoses of the superficial femoral artery, popliteal and lower limb arteries; 10 patients had patent external iliac arteries, but disease of the femoro-popliteal and distal vessels.

The tantalum balloon expandable stents consist of a knitted, flexible, metallic tube and can be implanted percutaneously through an introducer sheath. The stent and the implantation method have been described previously in detail [2, 4, 8].

In the patients with stenoses, the femoral arteries were punctured bilaterally and introducer sheaths were in-

serted. For stents with a diameter of up to 8 mm, 8.5 F introducer sheaths were used. Stents of 9 mm diameter required a 10 F introducer sheath. Depending on the duration of the intervention, between 5000–10000 IU of heparin was given intra-arterially during the procedure. J-shaped guide-wires were advanced through the stenosed arterial sites into the aorta, using a digital subtraction (DS) road map procedure. Dilatation of the stenosed bifurcation was performed with 7 or 8 mm Ultrathin™ balloons, in cases of bilateral stenosis using a kissing balloon method (Tegtmeyer). In patients with a large amount of calcified plaque material, balloon dilatation was not performed in order to prevent distal embolization of the sclerotic material. In one of these cases stent insertion could only be achieved with careful rotating movements to prevent dislocation of the stent from the balloon. Then, the catheter stent assemblies were introduced bilaterally and a new DS road map (to confirm correct placement) was performed. The catheter stent assemblies were positioned in the aortic bifurcation, their cephalic ends lying in the aorta and their distal ends extending into the common iliac arteries (Fig. 1). In each case, the two introduced stents were lying in a parallel position with their proximal ends in the stenosed aorta. The proximal stent ends were of equal height, shaping the new bifurcation divider. The expanded stent diameter corresponded to the widths of the normal lumen of the common iliac arteries, which, in our patients, was 7–9 mm. Both balloons of the catheter-stent assemblies were manually and simultaneously dilated under fluoroscopic control. A control angiogram confirmed correct positioning and shape of the stents. In cases where elastic recoil or severe calcification of the arterial wall caused high wall resistance such that the stent could not be opened completely by a single balloon dilatation, repeat balloon dilatation was performed with the same balloon with a maximum inflation duration of 60 seconds. If this procedure was not sufficiently successful in complete opening of the stent, the original balloon





**Fig. 2a.** A 45-year-old female patient with rest pain and occlusion of the aortic bifurcation, including the distal aorta and both common iliac arteries. There are bilateral long external iliac artery stenoses secondary to atrophy. **b** After local fibrinolysis with 3 mg rtpa, combined with 3000 IU of heparin performed by the brachial access route, a small channel within the previously occluded left common iliac artery can be seen. The guide wires are passed through the lesion and pulled through introducer sheaths inserted at the groins. **c** Two 8 cm long and 7 mm wide balloon expandable stents were introduced from either side and expanded simultaneously. The proximal ends are placed in the aorta in a parallel position, whereas the distal ends are anchored in the common iliac arteries. **d** Retrograd angiography 6 days after stent therapy showing complete patency of the stents and antegrade peripheral blood flow. The symptoms improved

minutes later, a small contrast-medium channel within the previous occlusion could be detected, enabling the passage of a guide wire into the external iliac artery through an introducer sheath, previously positioned into the femoral arteries, using the pull-through method. Pre-dilatation with a 5 mm PTA catheter was performed to reduce the risk of distal embolisation of thrombotic material and to enable the passage of the catheter-stent assembly through the previously occluded segments. The contralateral side was treated with the same method.

Angioplasty with a 7 F van Andel catheter was, in one case, insufficient to permit the catheter-stent assembly to pass through the obstruction. The catheter-stent assembly was therefore pulled back into the introducer sheath and the stent removed by unravelling the knitting. After pre-dilatation with a 5 mm-balloon, stent insertion was successfully achieved. After stent placement, intravenous heparin was continuously given for 2 days by a perfusor to increase the partial thrombin time to 60–90 s. A dosage 100 mg aspirin daily was also prescribed.

Follow-up was possible in 15 patients. The mean observation time being 11 months with a range from 6 to 20 months. Three patients were treated recently and have not yet received a post-treatment examination.

The clinical symptoms and the Doppler ankle-arm indexes were evaluated in all patients before treatment, im-

catheter was withdrawn and replaced by a shorter balloon tolerating an inflation pressure up to 17 atm, the balloon diameter being 1 or 2 mm larger than the nominal stent diameter.

In the patients with long occlusions of the bifurcation, the guide wire could not be inserted from the femoral artery into the aorta. Thus, after puncture of the left brachial artery, a straight catheter was placed proximally into the aortic thrombus, and a maximum dose of 5 mg of recombinant tissue plasminogen activator (rtpa) with 3000 IU of heparin was injected into the thrombotic material. A further 5000 IU of heparin were given intra-arterially to prevent thrombus formation during the intervention. Several

**Table 1.** Risk factors

Smoking	18/18
Elevated serum cholesterol	14/18
Hypertension	10/18
Diabetes mellitus	4/18

**Table 2.** Stented aortic bifurcations

Bilateral stenoses	10
Unilateral occlusion with contralateral stenosis	4
Bilateral occlusion	4
Dimensions of stented lesions:	
Stenoses:	mean length 1.75 cm maximal length 3 cm
Occlusions:	mean length 14.2 cm maximal length 19 cm

mediately after stenting and then at six monthly intervals. Intravenous digital subtraction angiograms with central contrast medium injection into the superior vena cava were performed in all patients one week after the procedure and, additionally, when clinical symptoms deteriorated during the follow-up period. It occurred in one patient after 8 weeks, three patients after 6 months and one after 8 months. A patient with bilaterally implanted tandem-stents had a magnetic resonance angiogram of the aortic bifurcation and iliac arteries as a control examination at 12 months. In patients with additional atherosclerotic disease located more distally in the iliac arteries, PTA was performed during the stenting session. PTA for co-existing disease of limb arteries was performed at a second session.

## Results

In all patients stents were implanted correctly into the aortic bifurcation. Patients with stenoses of the aorto-iliac bifurcation received one 4 cm long stent on each side. There were 4 patients with aorto-iliac occlusion: one received 8 cm long stents bilaterally, 2 had two 8-cm tandem stents on each side; the fourth received two 8-cm long tandem stents on the one side and three 8-cm plus one 4-cm long stents on the other side to cover the lesion completely.

As the cephalic ends of the stents contact in the midline, they form a new endoprosthetic aorto-iliac junction with an arterial divider located more proximally than the natural one. The new prosthetic bifurcation was located from 0.5 to 4 cm more cranially than the natural one, covering the ilio-lumbar arteries, while sparing the inferior mesenteric artery.

Clinical symptoms improved immediately in all patients. In 15 patients, a stage without any limited walking distance (Fontaine I) was achieved by stent implantation and PTA of stenoses located more distally. Three patients improved their symptoms only to Fontaine IIa. In all patients, the Doppler ankle-arm index (AAI) increased after treatment (mean increase  $0.40 \pm 0.14$ , range 0.22–0.66). Doppler AAI remained unchanged during the follow-up period in 13 patients. The arteriograms performed in the 5 patients with deterioration of clinical symptoms

during the follow-up period did not reveal any signs of recurrent disease in the area of the stented bifurcation. Instead, disease was identified in the distal run-off vessels.

There were no severe complications related to the procedure. Two patients developed sizable hematomas at the puncture site, but none required transfusion or surgical intervention.

## Discussion

PTA is considered to be suitable therapy in patients with atherosclerotic stenoses or even occlusions of the aorta [14–18]. In stenotic disease of the distal aorta and the proximal common iliac arteries, application of the kissing balloon method has proved to be a safe and efficacious alternative to vascular surgery [17, 19].

For occlusions and widespread disease of the aortic bifurcation, surgical interventions have been performed, unless stenting of unilateral completely occluded iliac arteries was performed successfully [7, 20, 21]. This patient group often suffers from concomitant cardiovascular disease (e.g. previous myocardial infarctions, cerebral strokes and arterial hypertension) associated with high operative risk and poor prognosis [11]. For these patients, less invasive procedures should be considered as the procedure of first choice. Surgical implantation of bifurcation prostheses is associated with a high incidence of perioperative mortality and morbidity [9, 10, 12, 16, 17]. Stent therapy should therefore be also performed in the area of the aorto-iliac junction, since this form of treatment has proved to be a valuable adjunct to PTA especially in large diameter arteries with relatively poor PTA results. Stenting can be used as the primary therapy in long iliac artery occlusions [7, 22, 23]. Aorto-iliac stenting with percutaneously implantable stents has been reported recently by Palmaz et al. [13], using rigid balloon expandable stents in 7 patients with bilateral stenoses or unilateral stenosis combined with short contralateral aorto-iliac occlusion. According to our results with flexible balloon expandable tantalum stents, this method can be regarded as safe. No major complications related to the technical procedure occurred. There was no evidence of complicating acute distal embolization originating from the implantation site.

The "kissing stent" method is easy to perform, if certain technical requirements are fulfilled. Optimal radioopaque stent metal and minimal shortening of the stent during its dilatation facilitates correct stent placement. Furthermore, flexible tantalum stents accommodate themselves very well to the curve between the aorta and iliac arteries, even if their cephalic ends, lying in a parallel fashion, are ranging far into the distal aorta. In patients, in whom additional lesions of the distal aorta had to be treated, stent flexibility was very suitable.

Due to the bifemoral approach, the size of one arterial puncture will not be larger than for iliac artery stenting where the same introducer sheath and catheter-stent assemblies are used. The two stents are inserted separately and converging to form the new prosthetic aortic bifurcation.

Cases with stenoses are easily performed, whereas long aorto-iliac occlusions require prior fibrinolysis to find a



channel for safe guide wire passage from proximal to the periphery. Rt-PA injection appears to be an appropriate method for creating, in a relatively short time interval, a new thin channel through long thrombus formations, even with a clinical history longer than 3 years.

To date, there are no adverse side effects caused by the shape of the new prosthesis, with a bifurcation located more proximally than the natural one, and with two wire mesh tubes positioned in a parallel fashion within the aorta. It is still unknown whether the parallel stent walls in the aorta will endothelialize, neither is it known whether the spaces between the stent walls and the aortic wall would remain patent or fill with thrombus. There were no signs of acute or late distal embolization into peripheral arteries.

Our post stent examinations show excellent early and intermediate clinical results for aortic bifurcation stents, comparable with our own and the experience of other investigators for stented iliac arteries [5, 8, 22]. There were no acute thrombotic occlusions, nor was there any later recurrence of disease at the implantation site. Although long-term patency studies of aortic bifurcation stents are not yet available, favourable long-term results are likely, since it is known from other implantation sites that late recurrences become rare with time.

As the quality of run-off vessels is known to influence the clinical success of PTA or stent therapy, deterioration of clinical symptoms during the follow-up period occurred in 5 out of 18 patients. This was, in all cases, due to additional and newly developed vascular disease in the lower extremities and can be regarded as general progression of the atherosclerotic disease. All bifurcation stents remained patent, as was proven by angiography in the 5 patients. In the remainder, there was no evidence of recurrent, hemodynamically-significant stenoses or occlusions, since clinical symptoms and Doppler ankle-arm indexes were unchanged. However, the possibility of subclinical recurrent disease is not excluded. In this regard, our results are comparable with those of Palmaz [13], even though our patients had more extensive disease. Even in our group of patients with incomplete run-off (n = 14) and those with previously long bilateral iliac occlusions (n = 4), no recurrent disease within the stented area was demonstrated.

Further development of interventional procedures and stent devices will potentially enable interventional radiologists to treat more proximally located aortic lesions eventually combined with aortic aneurysms. With this new non-invasive procedure, the high-risk patient group may be treated more safely, with less severe complications and discomfort and more economically than with vascular surgery. However, further clinical studies including more patients with longer observation times are mandatory to establish the value of this new therapeutic method.

## References

1. Palmaz JC, Windeler SA, Garcia F et al. (1986) Atherosclerotic rabbit aortas: expandable intraluminal grafting. *Radiology* 160: 723-726
2. Strecker EP, Romaniuk P, Schneider B et al. (1988) Perkutan implantierbare, durch Ballon aufdehnbare Gefäßprothese: Erste klinische Ergebnisse. *Dtsch Med Wschr* 113: 538-542
3. Palmaz JC, Richter GM, Noeldge G et al. (1988) Intraluminal stents in atherosclerotic iliac artery stenosis: preliminary report of a multicenter study. *Radiology* 168: 727-731
4. Strecker EP, Liermann D, Barth KH et al. (1990) Expandable tubular stents for treatment of arterial occlusive disease: experimental and clinical results. *Radiology* 175: 97-102
5. Long AL, Page PE, Raynaud AC et al. (1991) Percutaneous iliac artery stent: angiographic long-term follow-up. *Radiology* 180: 771-778
6. Kichikawa K, Ushida H, Yoshioka T et al. (1990) Iliac artery stenosis and occlusion: preliminary results of treatment with Gianturco expandable metallic stents. *Radiology* 177: 799-802
7. Vorwerk D, Guenther RW (1990) Mechanical revascularization of occluded iliac arteries with use of self-expandable endoprostheses. *Radiology* 175: 411-415
8. Strecker EP, Kuhn FP, Liermann D et al. (1992) Strecker flexible tantalum stent. In: Kim D, Orron DE (eds) *Peripheral vascular imaging and intervention*. 1st ed. Mosby Year Book, St. Louis, Mo, pp 541-552
9. Rutherford RB, Patt A, Pearce WH (1987) Extra-anatomic bypass: a closer review. *J Vasc Surg* 6: 437-446
10. Braun L (1988) Early and late results following implantation of an aortic bifurcation prosthesis. *Zentralbl Chir* 113: 423-435
11. Kent KC, Skillman JJ (1992) *Operative therapy for aneurysmal and lower extremity occlusive diseases*. In: Kim D, Orron DE (eds) *Peripheral vascular imaging and intervention*. 1st ed. Mosby Year Book, St. Louis, Mo, pp 57-60
12. Givel JC (1990) Ischemic colitis as a complication after reconstruction of the aortic bifurcation. *Langenbecks Arch Klin Chir Suppl* 1990: 335-340
13. Palmaz JC, Encarnacion CE, Garcia OJ et al. (1991) Aortic bifurcation stenosis: treatment with intravascular stents. *JVIR* 2: 319-323
14. Ravimandalam K, Rao VR, Kumar S, Gypta AK, Joseph S, Unni M, Rao AS (1991) Obstruction of the infrarenal portion of the abdominal aorta: results of treatment with balloon angioplasty. *AJR* 156: 1257-1260
15. Yakes WF, Kumpe DA, Brown SB et al. (1989) Percutaneous transluminal aortic angioplasty. *Radiology* 172: 965-970
16. Odurny A, Colapinto RF, Sniderman KW, Johnston KW (1989) Percutaneous transluminal angioplasty of abdominal aortic stenoses. *Cardiovasc Intervent Radiology* 12: 1-6
17. Tadavarthy AK, Sullivan WA Jr, Nicoloff D et al. (1989) Aorta balloon angioplasty: 9-year follow-up. *Radiology* 170: 1039-1041
18. Becker GJ, Katzen BT, Dake MD (1989) Noncoronary angioplasty. *Radiology* 170: 921-940
19. Tegtmeier CJ, Kellum CD, Kron JL, Mentzer RM Jr (1985) Percutaneous transluminal angioplasty in the region of the aortic bifurcation. The two-balloon technique with results and long-term follow-up study. *Radiology* 157: 661-665
20. Rees CR, Palmaz JC, Garcia O, Roeren T, Richter GM, Gardiner G Jr, Swarten D, Schatz RA, Root HD, Rogers W (1989) Angioplasty and stenting of completely occluded iliac arteries. *Radiology* 172: 953-959
21. Hausegger KA, Lammer J, Klein GE, Fluckiger F, Lafer M, Pilger E, Aschauer M (1991) Percutaneous recanalization of pelvic artery occlusions - fibrinolysis, PTA, stents. *RoFo Fortschr Röntgenstr* 155: 550-555
22. Zollikofer CL, Antonucci F, Pfyffer M et al. (1991) Arterial stent placement with use of the Wallstent: midterm results of clinical experiences. *Radiology* 179: 449-456
23. Yang XM, Manninen H, Matsi P, Soimakillo S (1991) Percutaneous endovascular stenting: development, investigation and application. *Eur J Radiol* 13: 161-173

## **Human Experience Using the Amplatz Mechanical Thrombolysis Catheter**

**S. MURTHY TADAVARTHY, M.D.<sup>1</sup>**

**M**echanical thrombolysis has been shown to be a reliable method for prompt restoration of perfusion. Although chemical fibrinolysis has become an effective treatment for vascular thrombosis, there are several potential drawbacks. A 24-to-48-hour trial of lytic therapy is often required, whereas mechanical clot removal can restore perfusion within minutes. Prolonged infusions of thrombolytic agents eventually lead to a systemic lytic state with increased risk of bleeding. In addition, when a femoral approach is used, patients must maintain a supine position for up to 48 hours. Thrombolytic therapy may fail to open the occluded vessel. Finally, currently used thrombolytic agents and the required stay in the intensive care unit during infusion are expensive.

Because of these limitations, several investigators have studied percutaneous mechanical rotational thrombectomy devices for clot removal or dissolution. Theoretically, these devices can reduce or even completely remove thrombus from grafts or vessels, thus eliminating or reducing the need for thrombolytic infusion. Mechanical thrombectomy may also be useful in patients with thrombus that is refractory to fibrinolytic therapy and should reduce overall patient morbidity as well as the cost of chemical thrombolysis.

Several mechanical thrombectomy devices have been developed, including the transluminal extraction catheter, the Amplatz mechanical thrombolysis catheter, the Ponomar transjugular clot-trapper device, the Gunther aspiration thromboembolotomy catheter, the rotational thrombectomy catheter, Starck aspiration thromboembolotomy, the ultrasonic thrombolysis catheter, and the rheolytic thrombectomy catheter. This presentation focuses on preliminary data in 14 humans treated with the Amplatz mechanical thrombolysis catheter.

### **AMPLATZ CATHETER**

**T**he Amplatz catheter has a special housing enclosing a tiny spinning propeller. It works by recycling cut particles over and over again, thereby progressively reducing their size. The propeller is connected to a drive shaft that can be connected to either a high-speed electric or air-driven motor.

Rotational speeds of 150,000 rpm can be achieved.

A "Y" connector at the proximal end of the catheter allows the infusion of saline, contrast medium, or fibrinolytic solutions, which can also lubricate and cool the drive shaft. The catheter is 100 cm long, 8 Fr in size, and made of polyurethane. The distal tip is a 1 cm long open-ended metal capsule with two side holes, which houses the propeller.

### **METHODS**

**T**he Amplatz mechanical thrombolysis catheter was used in 14 patients. Ten procedures were carried out in arterial Gortex grafts, two in native arteries, and two with venous thrombosis. Duration of thrombosis was one or two days to three weeks. In 12 patients mechanical thrombectomy was carried out as a primary procedure, and in two patients after failure of urokinase infusion.

Under local anesthesia, a catheter was introduced percutaneously at the site of thrombosis and the status of the runoff vessels was demonstrated angiographically. Usually a downstream puncture of the ipsilateral femoral artery had to be performed, because passage around the iliac bifurcation proved to be too difficult. A blood sample was drawn for baseline free hemoglobin, haptoglobin, and hematocrit determination. Five thousand units of heparin were given intravenously.

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A blood pressure cuff was applied distally to the graft and inflated to 20 mm Hg above arterial pressure prior to thrombectomy. This was to prevent peripheral embolization and to aspirate particles and hemolyzed blood after the procedure.

The device was connected to the air outlet in the special procedure room, where the centrally regulated air pressure ranged from 47 to 50 lbs/square inch. This resulted in a rotational speed of the air motor of 150,000 rpm. The device was activated by a foot switch.

Contrast medium was injected through a side arm of the sheath into the thrombosed vessel. The device was activated and slowly advanced through the thrombosed graft or native artery with a gentle back-and-forth motion.

The blood pressure cuff remained inflated after thrombectomy, and a wide-lumen 8 Fr catheter was introduced over a guide wire through the recanalized graft or artery beyond the distal anastomotic site. Under fluoroscopic guidance, the now-liquid content of the graft was forcefully aspirated by syringe suction and ejected onto a towel or gauze. Only a few small fragments of red or white thrombi were observed.

Contrast medium was injected through the same catheter to demonstrate residual thrombotic material. If found, the thrombectomy catheter was re-introduced and re-activated.

After the last application and aspiration, the blood pressure cuff was released, and a standard angiogram was recorded. Underlying stenoses were treated by balloon angioplasty or atherectomy. Another blood sample was drawn after the procedure for determination of plasma free hemoglobin, haptoglobin, and hematocrit.

## RESULTS

Complete success (defined as complete clearing of thrombolytic material in the treated segment of native artery or graft without the need for adjunctive lytic therapy or surgical thromboembolectomy) was achieved in ten patients (71%). Partial success (defined as partial or incomplete clearing of thrombus with the device and requiring additional intervention such as lysis or surgery to achieve complete clearing of clot) was achieved in two patients (14%). In two patients (14%) the procedure was considered a failure, and

the device did not significantly contribute to or shorten the time toward complete resolution of the clot.

The mean plasma free hemoglobin was 41 mg/dl (range 7 to 150 mg/dl) 30 to 60 minutes after activation of the device. The mean absolute decrease in haptoglobin was 35 mg/dl (range 0 to 63 mg/dl). The mean decrease in hemoglobin was 1.0 mg/dl (range 0.2 to 1.8 mg/dl), and the mean decrease in hematocrit was 2.4% (range 0 to 4.6%).

Eleven of the 14 (78%) treated arteries or grafts were patent at one week follow-up. Three acute occlusions occurred within 24 to 72 hours. Although long-term follow-up is incomplete, six patients' vessels remained patent from one to six months. One patient (7%) presented with reocclusions six weeks after the initial procedure.

Complications occurred in four (28%) of patients and included distal embolization of debris in two, significant hematomas in two (14%). In one patient with a hematoma, the procedure could not be completed because of mechanical failure of the device with no back-up available. Both patients with embolic problems were easily managed with additional use of the device or intra-arterial urokinase.

## DISCUSSION

Surgical thrombectomy with Fogarty catheters has been largely replaced by the intravascular infusion of thrombolytic agents in recent years, but both techniques have significant morbidity and a small mortality rate and require prolonged hospitalization. Therefore, it is understandable that investigators have searched for devices to mechanically destroy or aspirate thrombi. Unfortunately, none of the mechanical devices used for thrombectomy has been uniformly successful, and chemical thrombolysis remains the procedure of choice for the treatment of thromboembolic disease.

Mechanical thrombectomy devices have several advantages over chemothrombolysis. They can achieve patency promptly (mean activation time is 2 minutes, 45 seconds, whereas chemical thrombolysis may take several days). Also, the thrombus is usually destroyed in a single session, so the patient does not have to undergo multiple examinations as with chemothrombolysis. It is not necessary to monitor patients for several days in

the intensive care unit, and expensive, repeated laboratory tests are not required. The patient does not have to lie immobile for several days and can leave the hospital soon after a successful procedure. Complications of chemotherapeutic therapy may include external and internal bleeding, pseudoaneurysms, distal embolization, pericatheter thrombosis, allergic reactions, and strokes.

The Amplatz device has several advantages over other devices. It is powered by a disposable air turbine that can be connected to the hospital air outlet, eliminating the need to purchase an expensive high-speed motor. A speed of 150,000 rpm is achieved at a pressure of 50 lb/square inch, and because of the high speed, even fibrin is macerated and the number and size of residual particles is smaller. A strong vortex provides recirculation and remaceration of particles. In addition, the catheter is flexible, facilitating passage around curves, and the propeller is recessed in the housing, minimizing vascular perforation.

Although the Amplatz thrombectomy catheter is overall very effective, there are some drawbacks. An antegrade 8 Fr sheath has to be placed into the common femoral artery in a downstream fashion with the present system, and this may not be possible in all patients, especially those who are obese and have a short common femoral artery. The catheter also lacks steerability and tends to hug the greater curvature of a vessel, which may leave thrombus along the lesser curvature. To broaden the clinical applicability of this otherwise promising device, an over-the-wire system and improved steerability will be necessary.

### CONCLUSION

The Amplatz device proved very powerful and effective in thrombosed grafts that contain nonadherent unaltered thrombus. We anticipate that this device will be less successful in native vessels where the thrombus tends to adhere to the vessel in 12 to 14 hours and becomes organized in a few weeks.

Based on our preliminary experience, this technique may play an important role in the treatment of thromboembolism, and it will be the treatment of choice for occluded Gortex bypass grafts. Investigations are underway to evaluate its use in venous thrombosis and thrombosed native arteries.

### References

1. McNamara T.O., Fischer J.R. Thrombolysis of peripheral arterial and graft occlusions: improved results using high dose urokinase. *AJR* 1985; 144: 769-775.
2. McNamara T.O., Bomberger R.A., Merchant R.F. Intra-arterial urokinase as the initial therapy for acutely ischemic lower limbs. *Circulation* 1991; 83(Suppl D): 1-106 - 1-119.
3. Becker G.J., Holden R.W. Fibrinolytic therapy. In Castaneda-Zuniga W.R., Tadavarthi S.M. (eds): *Interventional Radiology*. Baltimore: Williams & Wilkins 1992, pp. 599-634.
4. Bildsoe M.C., Moradian G.P., Hunter D.W., Castaneda-Zuniga W.R., Amplatz K. Mechanical clot dissolution: New concept. *Radiology* 1989; 171: 231-233.
5. Nazarian G.K., Qian Z., Bjarnason H., Rengel G., Castaneda-Zuniga W.R., Hunter D.W., Amplatz K. Hemolytic effect of Amplatz thrombectomy device. Presented at the 18th Annual Meeting of the Society of Cardiovascular and Interventional Radiology in New Orleans, L.A., 1993.
6. Yedlicka J.W., Carlson J.E., Hunter D.W., Castaneda-Zuniga W.R., Amplatz K. Thrombectomy with the TBC endarterectomy system: An experimental study and case report. *JVIR* 1991; 2: 343-347.
7. Gunter R.W., Vorwerk D. A new aspiration thrombectomy catheter with propeller-tipped rotating wire: An in-vitro study. *J Intervent Radiol* 1990; 1: 17-20.
8. Starck E.E., Wagner H.J. Rotations-Aspirations-thromboembolotomie. *Deutsche Medizinische Wochenschrift* 1990; 116: 1-6.
9. Starck E.E., Wagner H.J. Percutaneous aspiration thromboembolotomie. In Castaneda-Zuniga W.R., Tadavarthi S.M. (eds): *Interventional Radiology*. Baltimore, Williams & Wilkins 1992; pp. 652-659.
10. Kensey K.R., Nash J.E., Abrahams C., Zarins C.K. Recanalization of obstructed arteries with a flexible, rotating tip catheter. *Radiology* 1987; 165: 387-389.
11. Schmitz-Rode T., Gunther R.W. Oscillating probe aspiration thrombectomy: Comparative in-vitro evaluation of two concepts. *Cardiovasc Intervent Radiol* 1992; 15: 151-153.
12. Drasler W.J., Jenson M.S., Wilson G.J., et al. Rheolytic catheter for percutaneous removal of thrombus. *Radiology* 1992; 182: 263-267.
13. Yasui K., Qian Z., Nazarian G.K., Hunter D.W., Castaneda-Zuniga W.R., Amplatz K. Recirculation-type Amplatz clot macerator: Determination of particle size and distribution. *SCVIR* 1993; 4: 275-278.
14. Snook T.L., Wise R.K., Miller C.A., Pae W.E., McCoach R.M., Williams D.R. A comparison of three pumps with respect to their effects on fibrinogen, platelets, and plasma hemoglobin during cardiopulmonary bypass. *Proc. Am Acad of Cardiovasc Perf*, Jan 1992; 3: 54-57.

## **Takayasu's Vasculitis: PTA Results**

LENNY K. A. TAN, M.D.<sup>1</sup>

**Takayasu's disease is an arteritis of unknown etiology affecting the aorta and the proximal segments of its major branches. More recent reports also show that the pulmonary vessels are affected in a similar fashion.**

**Whilst the commonest presentation is that of vessel stenosis and ischaemia, other manifestations include diffuse dilatation of vessels and aneurysm formation. Most, however, present with a mixture of the above. In view of the multiplicity of vessels that may be involved, the pathological possibilities and the end organ, the disease can present in many ways. Other factors include the rapidity of progression of the disease process, severity of obstruction, collateral development and the additional effects of hypertension.**

**It is therefore no surprise that over the years, the disease has acquired numerous names, including that of the person who described it originally - a Japanese ophthalmologist called Takayasu.**

**Significant clinical features include the following:**

- 1) Affects the young, usually in the second to fourth decade of life;**
- 2) Far more common in females.**

**We have had the opportunity to study a substantial number of these patients and to follow them up. Amongst the important factors that influence outcome is the**

**severity of hypertension and its control. This is one of the most important prognostic factors as causes of death or severe morbidity are almost entirely related to the cardiovascular and central nervous systems.**

**When balloon angioplasty first became available in Singapore in the early '80s, the technique was applied to several of these patients as surgery was found not to be of much help.**

**Although the histological findings in Takayasu's disease is that of fibrotic change together with superimposed arteriosclerosis, the stenotic lesions have responded well to balloon angioplasty. Self-expandable metallic stents were not available at that time.**

**We have performed balloon angioplasty on a wide range of vessels, including the aorta, renal arteries, brachycephalic vessels, iliac vessels with good results. Many of these patients have been followed up between 3 to 8 years and have remained well.**

**The results of angioplasty on the individual vessels will be discussed.**

**Aortic lesions and renal lesions appear to respond particularly well. Symptomatic relief from dilatation of brachycephalic vessels are quite remarkable although the cosmetic appearance may not be as impressive. Iliac arteries do extremely well with balloon angioplasty.**

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In our experience, only two patients did not respond very well. One was a lesion in the renal artery where stenosis recurred. I am sure a metallic stent would have been useful here. The other was a very extensive disease of the aorta. After dilatation, its lumen could not be maintained although flow improved. I am sure stents would have been very useful for this patient too. In spite of this, the patients are still very well symptomatically after nearly 8 years.

Patients who do not respond well at those with "active" disease manifesting.

- 1) Raised ESR
- 2) Progression of the disease process

Balloon angioplasty is a very useful method of treating stenotic lesions from Takayasu's disease. Not only is there relief of symptoms but the benefits appear to last a long time. I am sure self-expandable metallic stents would be another great source of benefit to these patients.

Unfortunately in Singapore, in recent years, there have hardly been any fresh cases of Takayasu's disease. This is yet another mystery and is probably related to the very rapid advances we have made in public health measures in Singapore. It might be a clue to the etiology of the disease.

## **Atherectomy and Renal Artery Stenosis**

**C. J. TEGTMEYER, M.D.<sup>1</sup>**

The first thing that should be started is that there are no definitive indications for renal atherectomy in the United States. This is because no good device has been approved by the FDA for use in the United States. This study was undertaken as a FDA protocol.

Eleven patients have undergone a modified renal atherectomy procedure at the University of Virginia Hospital. The TEC atherectomy device was used. There were six men and five women patients. The mean age was 62.9 years. Two patients were treated primarily for renal

insufficiency, two patients for hypertension, and seven patients were treated for a combination of hypertension and renal insufficiency. Fourteen lesions were dilated in these patients and seven of those lesions were estial lesions. Two of the lesions undergoing atherectomy were due to recurrences following previous balloon angioplasties. A contralateral renal artery was occluded in three patients and a nephrectomy had been performed in one additional patient. The patients have been followed for up to eight months. The technical aspects and results of renal atherectomy will be discussed.

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## **Long-Term Results of PTA in Peripheral Vascular Disease**

**C. J. TEGTMEYER, M.D.<sup>1</sup>**

Percutaneous transluminal angioplasty was originally developed by Charles Dotter in 1964 for the treatment of atherosclerotic peripheral vascular disease. Then in 1974, Andreas Gruntzig revolutionized the technique when he developed a soft flexible double lumen balloon catheter. In the three decades since its inception, percutaneous transluminal angioplasty has evolved into a versatile and reliable therapeutic procedure. If the patients are carefully selected, good results can be anticipated.

Long-term results of peripheral angioplasty are dependent on many factors. The best results are obtained in the distal aorta or in the iliac vessels because of the large size of the arteries and the high flow. Technically, iliac stenoses are the easiest angioplasties to perform and tibioperoneal angioplasties are the most difficult. Stenoses are easier to dilate than occlusions. Morphology is a critical factor.

Best results with angioplasty are obtained in short, focal stenoses and in arteries of large diameter. Longer stenoses, occlusions and smaller vessels do not achieve the same long-term patency rates. The long-term benefit of angioplasty is enhanced in patients with good run-off. Therefore, patients with multiple lesions and poor run-off do not have as good a long-term patency rate as patients with good run-off and short focal stenoses. The overall clinical condition of the patient is also a major factor in assessing the long-term patency rates. The long-term results of peripheral angioplasty in the aortiliac, femoropopliteal, and tibioperoneal regions will be discussed.

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## CHAPTER 13

# Angioplasty of Brachiocephalic Vessels

Jacques Thérion

Percutaneous transluminal angioplasty (PTA) has become a well-accepted technique for treating arterial stenosis throughout the body. It was first described by Dotter and Judkins (1). Gruntzig and Hopff (2) introduced the double-lumen balloon dilatation catheter. The application of this technique to disease involving the supra-aortic vessels has lagged behind its application in other regions because of the potential risks of complications related to untoward embolization of the central nervous system.

Originally described as a preoperative technique (3-5), angioplasty was first used in inflammatory lesions (6-8) and in selected cases of atherosclerotic stenosis involving the origin of brachiocephalic vessels (9,10) and of the external carotid, the innominate, and the internal carotid arteries (11-18).

Because of the frequent ulceration of atheromatous plaques in the common carotid artery bifurcation, I have developed a technique of cerebral protection during angioplasty, occluding the internal carotid artery with a latex balloon positioned distal to the site of the therapeutic procedure (19-21).

### MATERIALS AND METHODS

Balloon angioplasty consists of stretching of the media of the diseased artery as well as breaking atherosclerotic plaque by inflating a balloon at the site of the stenosis (22). The plaque is split and not compressed (23). This is followed by remodeling of the arterial lumen (23,24). The angioplasty balloon should be large enough to obtain dilatation of the artery without any effort upon balloon inflation. We currently use 8- to 10-mm-diameter balloons for angioplasty of brachiocephalic arteries. A 4- to 5-mm balloon is used to angioplasty the ostium of the

vertebral artery, and a 6- to 7-mm balloon is used for the origin of the internal carotid artery. A "kissing" technique may be useful for the stenoses involving both common and internal carotid arteries. This technique allows efficient dilatation of the common carotid artery without overdistending the internal carotid artery (21). We do not use pressure gauges to perform angioplasty, and we prefer manual inflation of the angioplasty balloon. We do not keep the balloon inflated, but instead we inflate and deflate the balloon repeatedly. This maneuver appears to be as effective as keeping the balloon inflated for several seconds.

The patient is given antiplatelet medication (aspirin 250 mg or ticlopidine 500 mg), every day for 1 week before the procedure and for 2 months after the angioplasty. Atropine is injected intravenously immediately before balloon inflation in stenoses involving the common carotid artery bifurcation. Atropine decreases the bradycardia due to compression of the carotid body (glomus). This is not necessary for angioplasty of postendarterectomy recurrent stenoses. Heparin is only given (7000-10,000 I.U.) before the inflation of a latex protective balloon in carotid angioplasty. Anticoagulants are not given for angioplasty in other arteries.

### ANGIOPLASTY OF SUBCLAVIAN AND VERTEBRAL ARTERIES (144 CASES)

Atherosclerotic stenosis of the subclavian artery is more frequently observed involving the ostium of this artery. The angioplasty of the subclavian artery is relatively simple; in addition, untoward distal migration of plaque debris into the vertebral artery is infrequent, due to the frequently associated "steal" (reversal of flow) or "pre-steal" syndromes (25). Catheterization of the left subclavian artery from the femoral approach is simple when compared with that from the axillary approach

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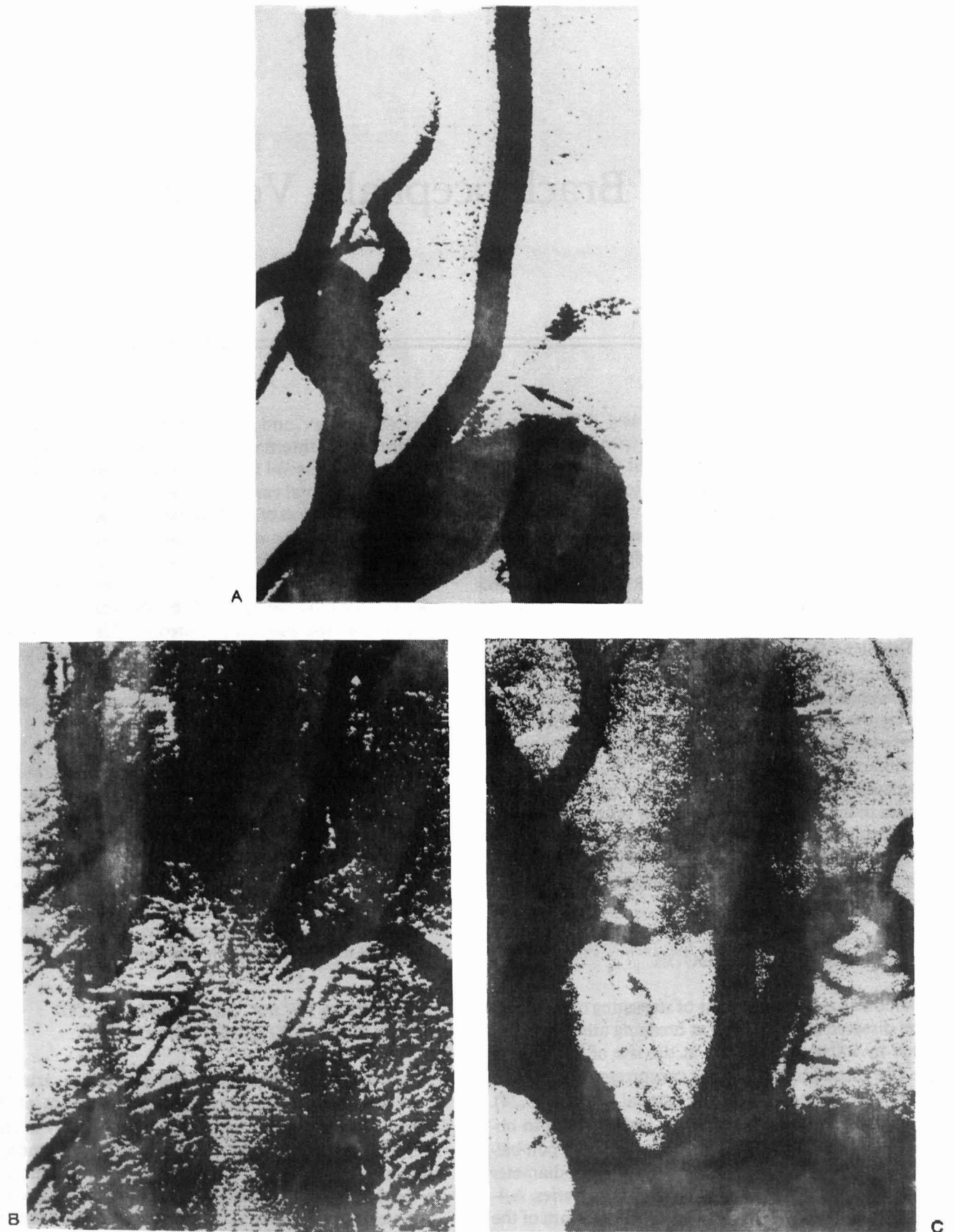


FIG. 1. Stenosis of the left subclavian steal syndrome. A: Before angioplasty. Early phase. Narrow stenosis of the origin of the left subclavian artery (*arrow*). B: Same procedure. Late phase. Retrograde filling of the left vertebral artery. C: After angioplasty. Early phase. Normal diameter of the subclavian artery (*arrow*), with filling of the left vertebral artery (*double arrow*).

(the weakness of the arterial pulse in the arm makes its percutaneous catheterization more difficult).

The most significant risk of angioplasty in this region is the inadvertent occlusion of the vertebral artery. This is more common when there is a plaque on the superior and medial side of the subclavian artery close to the origin of the vertebral artery. During dilatation with a short balloon, the compressed plaque may occlude the vertebral artery. We currently use a 4-cm-long angioplasty balloon for this type of situation. This balloon not only covers the plaque but also covers the ostium of the vertebral artery.

Reversal of a subclavian steal syndrome was first reported by Bachman and Kim (26) and by others (9,10) (Fig. 1). We have developed a hemodynamic classification of stenoses of the subclavian artery covering the various locations of stenoses and their hemodynamic consequences in the vertebral artery (25). A complete anatomical angioplasty of an atherosclerotic subclavian artery is rare, but this good result may be achieved in inflammatory lesions (27).

Asymptomatic, angiographic subclavian steal syn-

drome is often considered benign, and it is often not treated. We believe that angioplasty should be used in those cases because it allows us to save not only the stenosed subclavian artery but also the homolateral and contralateral vertebral arteries. The vertebral arteries may become essential to the development of intracranial collateral circulation in patients with severe atherosclerosis.

Stenoses of the origin of the vertebral artery is frequently smooth, and it rarely has an ulcerated plaque (28). Angioplasty of this type of lesion may be safely performed without cerebral protection (Fig. 2). We did not observe any embolic complications related to the angioplasty of 55 cases of subclavian artery stenoses.

Selection of patients for angioplasty in patients with vertebrobasilar insufficiency remains difficult because of the nonspecificity of the clinical presentation. One may even hesitate to perform a diagnostic angiogram because of potential, though infrequent, complications related to this procedure. Doppler ultrasonography provides valuable information that helps the patient selection for angioplasty (29). It is important to remember that athero-

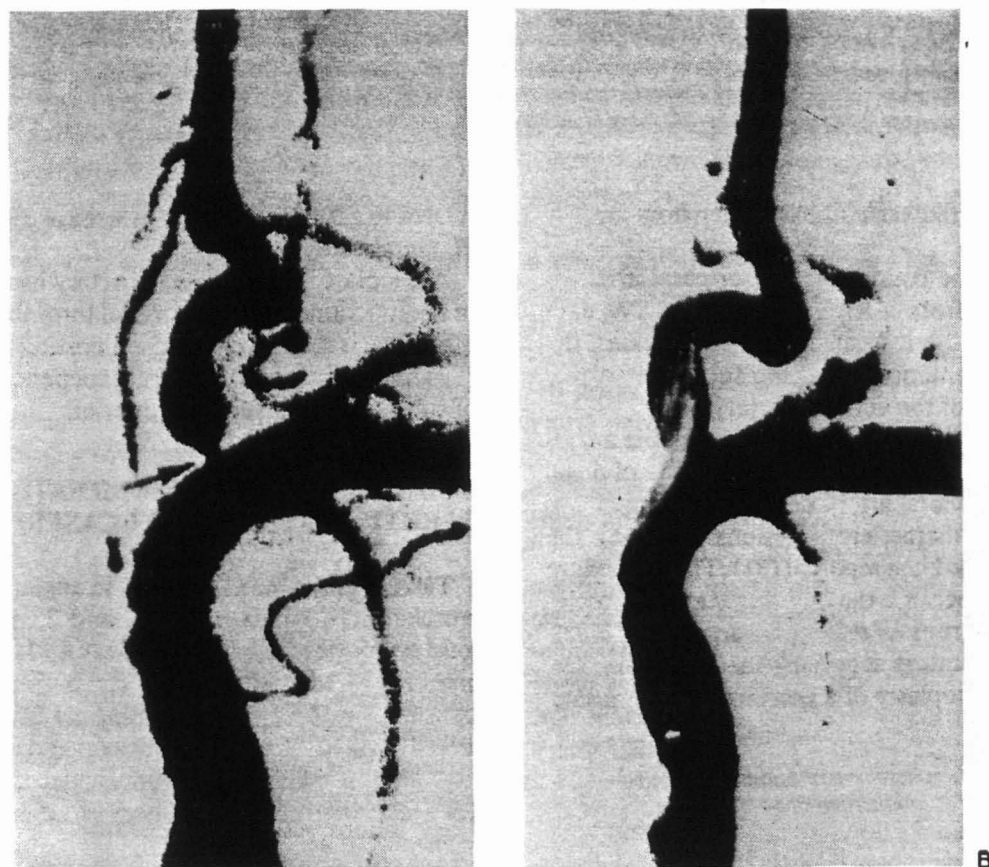


FIG. 2. Vertebrobasilar insufficiency symptoms. Stenosis of the left vertebral artery. A: Before angioplasty. Narrow stenosis of the origin of the left vertebral artery (arrow). B: After angioplasty. Note successful dilatation of the stenosis, as well as impaired filling of the vertebral artery. The patient is now symptom-free.

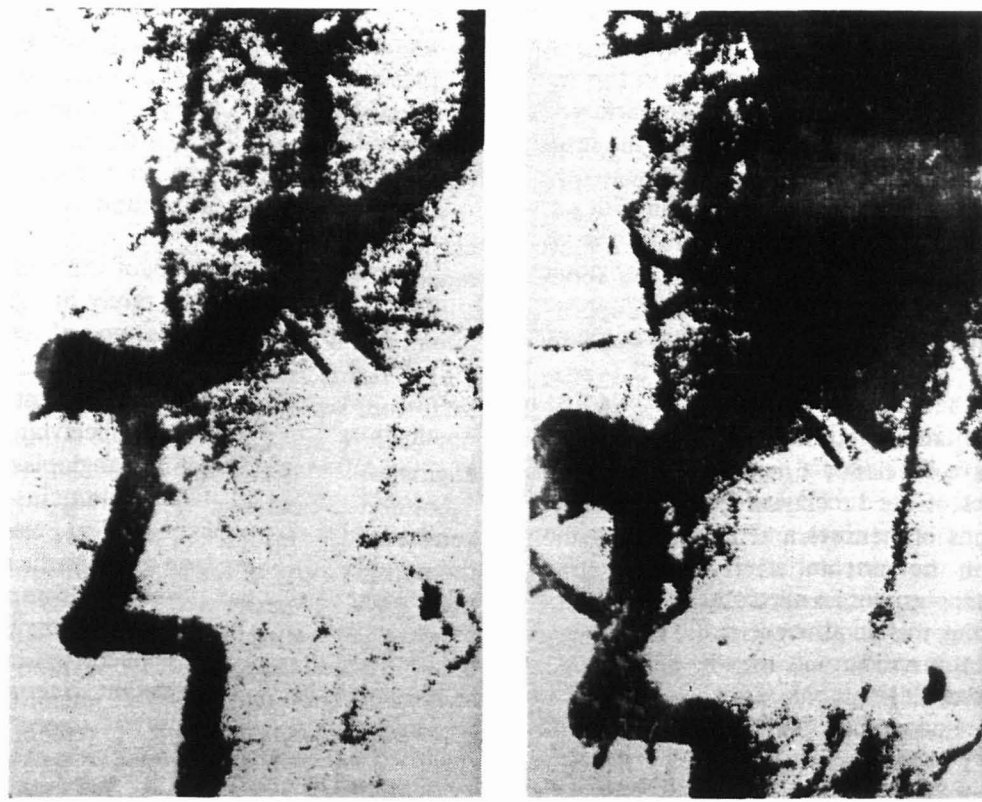


FIG. 3. Right cerebellar infarction and progressive cerebellar symptoms. Secondary to stenosis of the distal segment of the right vertebral artery. A: Before angioplasty. Stenosis of the vertebral artery (arrow) before the origin of the posterior inferior cerebellar artery. B: After angioplasty. Dilatation of the stenosis (arrow). Better opacification of the basilar artery and retrograde filling of the contralateral vertebral artery.

sclerotic lesions in the vertebrobasilar territory carry a poor prognosis (30).

Angioplasty of the ostium of the vertebral artery is usually performed from a femoral approach. The axillary approach may be indicated in selective cases, depending on the orientation of (a) the subclavian artery and (b) the ostium of the vertebral artery.

The development of smaller and more flexible angioplasty systems allows the intravascular therapy of distal segments of the vertebral artery (Fig. 3).

Angioplasty of atherosclerotic lesions involving the basilar artery has also been reported (31). This procedure is more dangerous because the dilation of an atherosclerotic plaque in the trunk of the basilar artery may block the ostium of perforators supplying the brainstem. On the other hand, angioplasty of a narrowed basilar artery

related to a subarachnoid hemorrhage carries lesser risks of complications (32).

Angioplasty of the vertebral artery has also been used in conjunction with intra-arterial thrombolysis when the thrombus is distal to a stenosed portion of the artery.

Table 1 describes our overall experience in angioplasty of subclavian and vertebral arteries.

#### ANGIOPLASTY OF INNOMINATE AND CAROTID ARTERIES (123 CASES)

Table 2 shows our experience in angioplasty of 83 atherosclerotic, 13 inflammatory, and 27 postsurgical carotid artery stenoses. Inflammatory and postsurgical stenoses were treated without cerebral protection. No transient or permanent neurological changes were observed in this group. Successful angioplasty of inflammatory lesions related to fibromuscular dysplasia (6-8), Takayasu arteritis (27), and neck radiotherapy were performed (Fig. 4).

Intimal hyperplasia is responsible for early recurrent postendarterectomy stenoses. This type of lesion may be easily dilated by balloon angioplasty if the diagnosis is

TABLE 1. Complications of subclavian and vertebral angioplasties (144 cases)

Asymptomatic:	1.4% (axillary aneurysm) (axillary occlusion)
Transient:	0.6% (subclavian fissure)
Permanent:	0%

TABLE 2. Complications of innominate and carotid angioplasties (123 cases)

Stenosis	Neurological changes		
	Asymptomatic	Transient	Permanent
Inflammatory and postsurgical (40 cases; no cerebral protection)	0%	2.5% (femoral occlusion)	0%
Atherosclerotic (32 cases; no cerebral protection)	6% (aneurysms)	6% (carotid occlusion)	12% { 3% dissecting 9% embolic
Atherosclerotic (51 cases; cerebral protection)	0%	2% (femoral aneurysm)	2% { 2% dissecting 0% embolic

made early with Doppler ultrasonography (Fig. 5). If the diagnosis is made late, the thickening of the arterial wall makes the lesion more resistant to angioplasty; in addition, more than one session may be indicated to achieve a satisfactory anatomical result (20,31).

Angioplasty of atherosclerotic stenoses of the carotid bifurcation was first performed in the external carotid artery (14) because of the reduced risk of potential untoward embolization into the cerebral circulation with plaque debris. There are some reports in the literature describing angioplasty of internal carotid artery without

cerebral protection (12,18). In our experience of 82 cases of atherosclerotic stenoses involving the common carotid artery bifurcation, 32 were performed without cerebral protection. A 10% morbidity was noted in this group. No untoward embolization of the intracranial circulation was observed in 23 patients treated with the first method of cerebral protection and in 28 patients treated with the new technique of cerebral protection during carotid angioplasty.

The effects of innominate artery angioplasty are shown in Fig. 6.

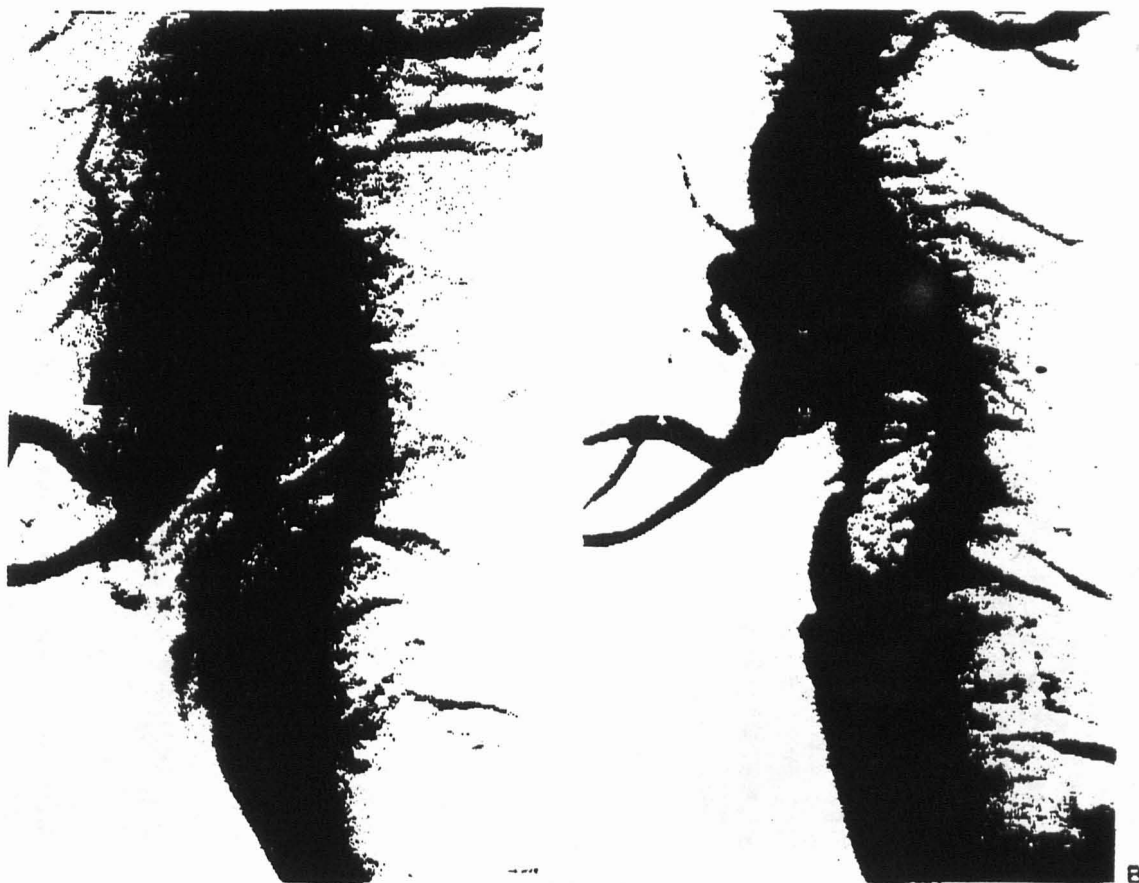


FIG. 4. Post-radiation stenosis of the internal and external carotid arteries. A: Before angioplasty. B: After angioplasty of the internal carotid artery.





FIG. 5. Postendarterectomy recurrent stenosis of the internal and common carotid arteries. A: Before angioplasty. B: After angioplasty.

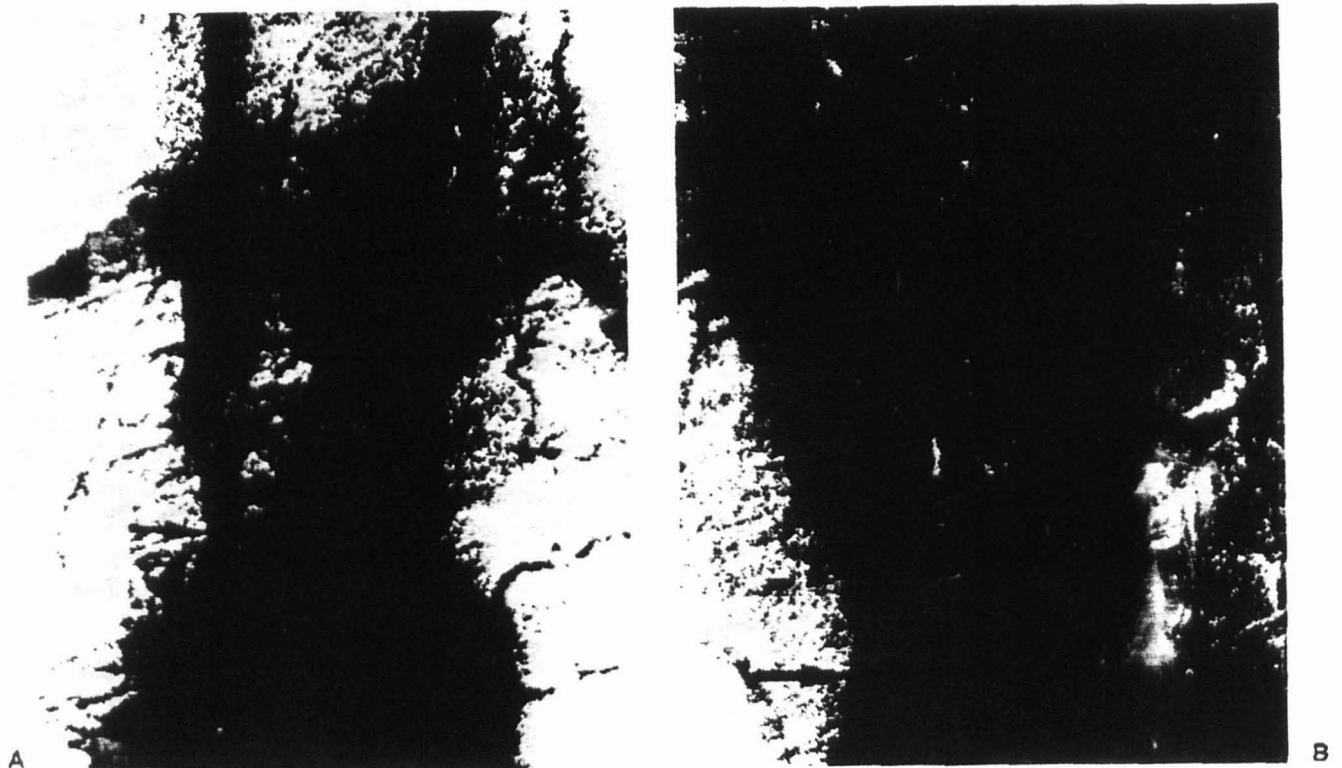


FIG. 6. Stenosis of the innominate artery with subclavian steal syndrome. A: Before angioplasty. Early phase. Narrow stenosis of the innominate artery (*arrow*). The right vertebral artery is retrogradely opacified on the late phase. B: After angioplasty. Early phase. Normal diameter of the innominate artery (*arrow*). Early filling of the right vertebral artery (*double arrow*).

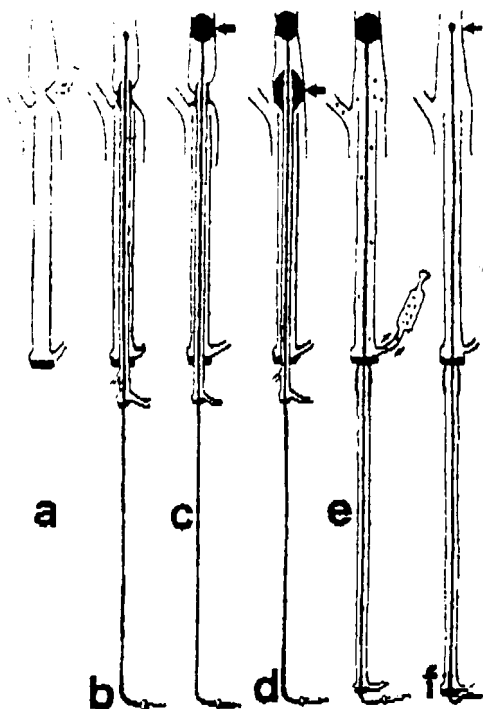


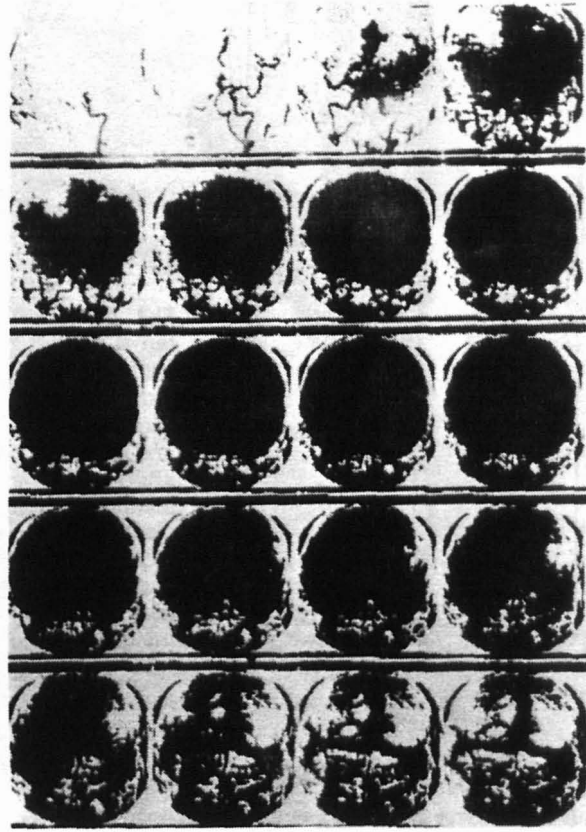
FIG. 7. Angioplasty of an atherosclerotic stenosis of the internal carotid artery. Schematic drawing of the procedure with cerebral protection using a triple coaxial catheter system. A: The guiding catheter is positioned in the common carotid artery close to the stenosis (arrow). B: Angioplasty and occlusive catheters are positioned in the internal carotid artery. C: Inflation of the occlusive latex balloon (arrow). D: Inflation of the angioplasty balloon (arrow). E: The angioplasty catheter is deflated and withdrawn. The occlusive balloon remains inflated. Atherosclerotic particles or clots are aspirated via the valve connector of the guiding catheter (arrows). F: The occlusive balloon is then deflated (arrow) and the system is withdrawn.



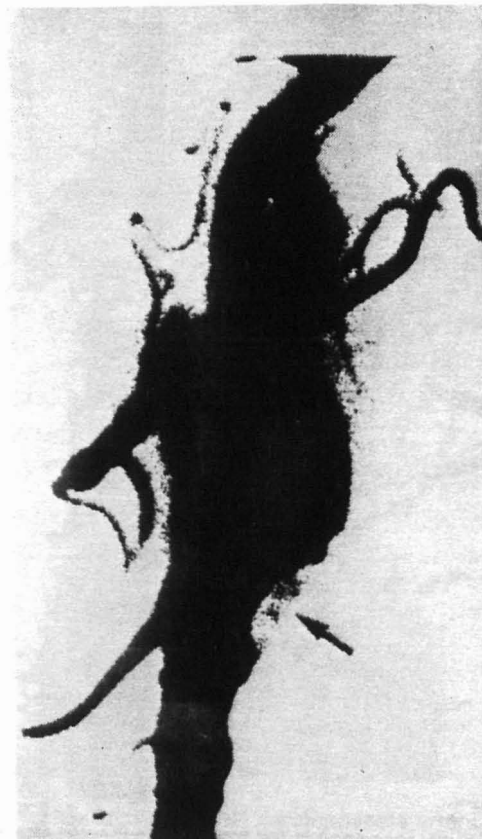
FIG. 8. Atherosclerotic stenosis of the internal carotid artery responsible for transient ischemic attacks. A: Before angioplasty. B: After angioplasty.



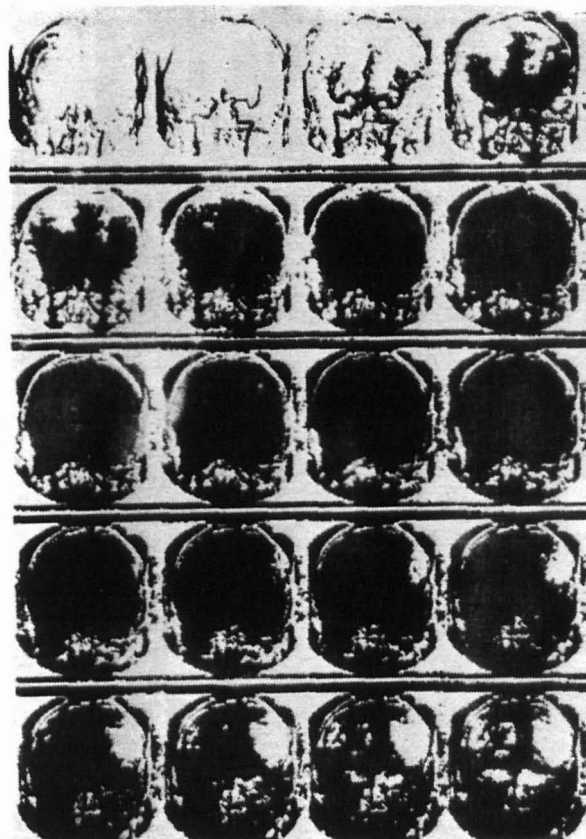
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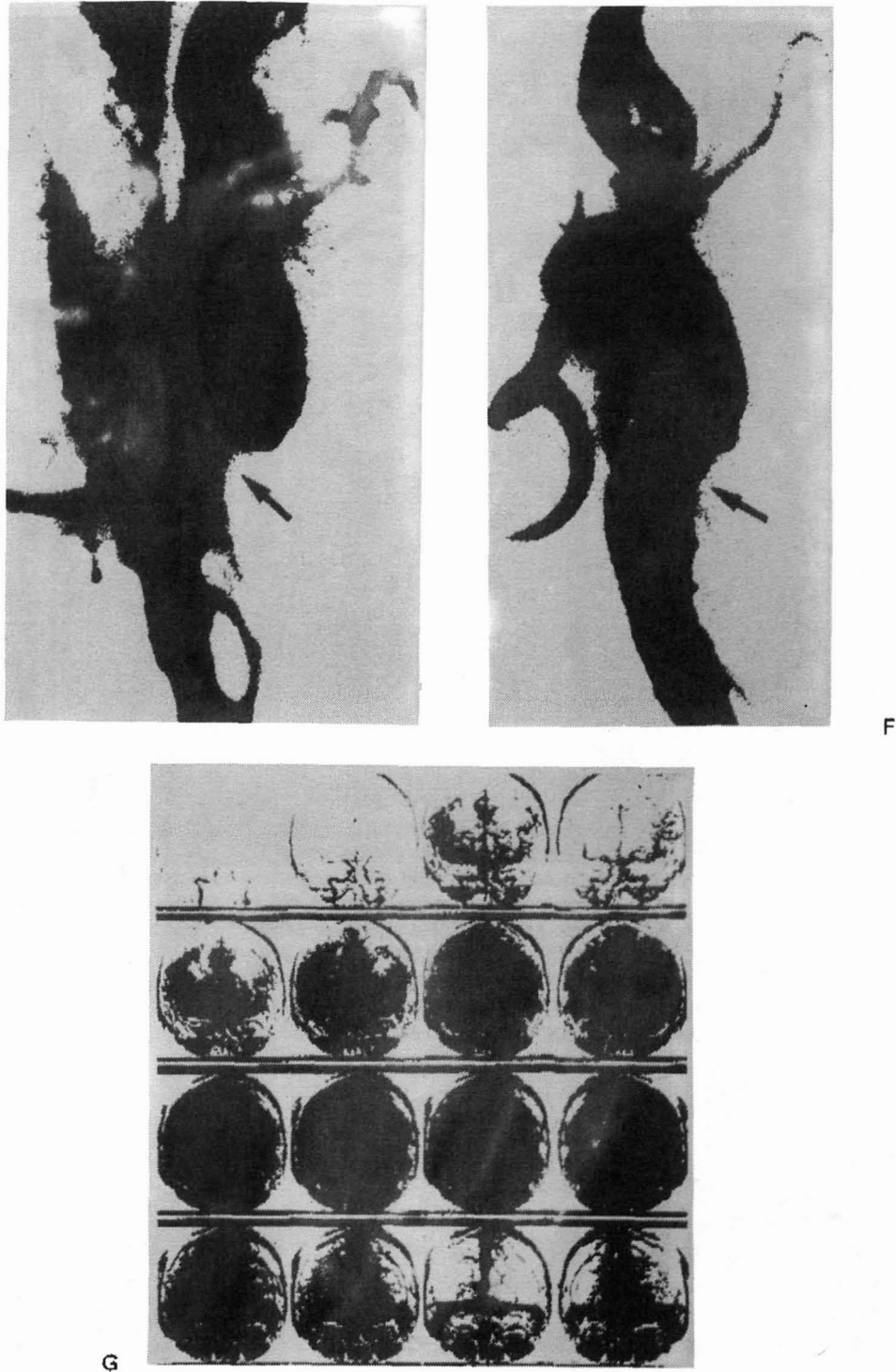


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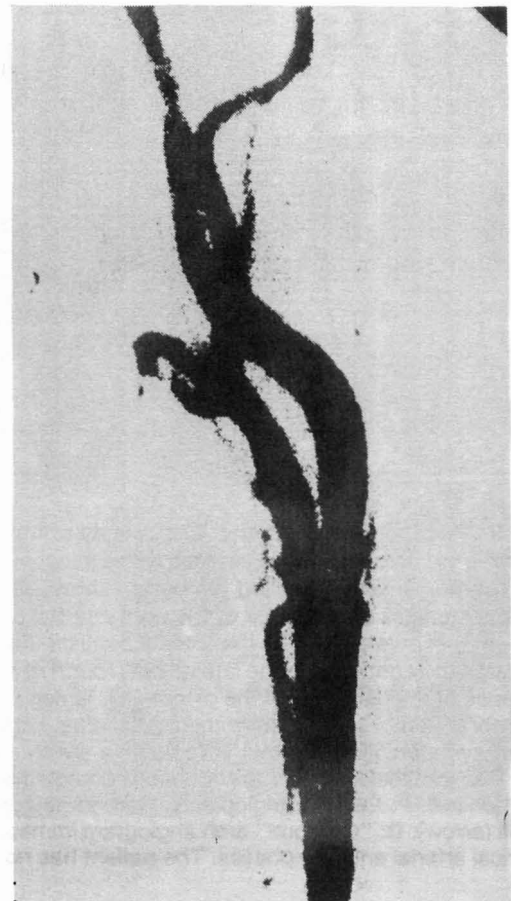
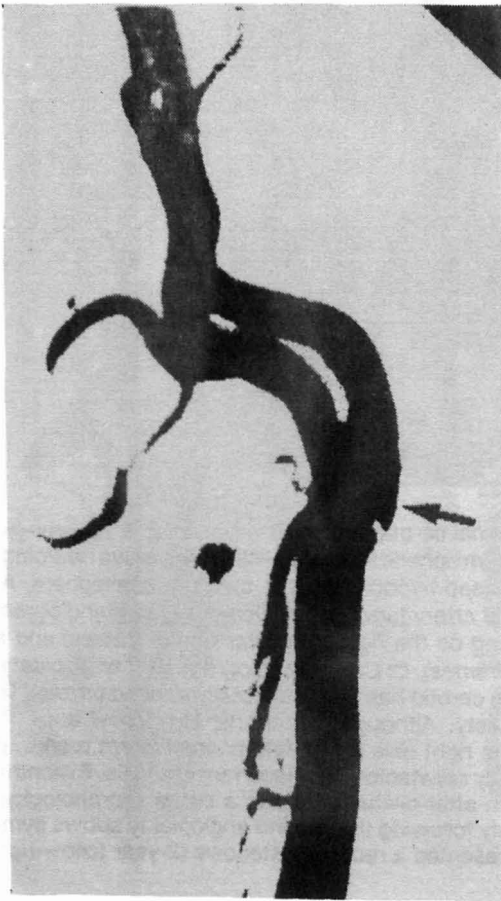
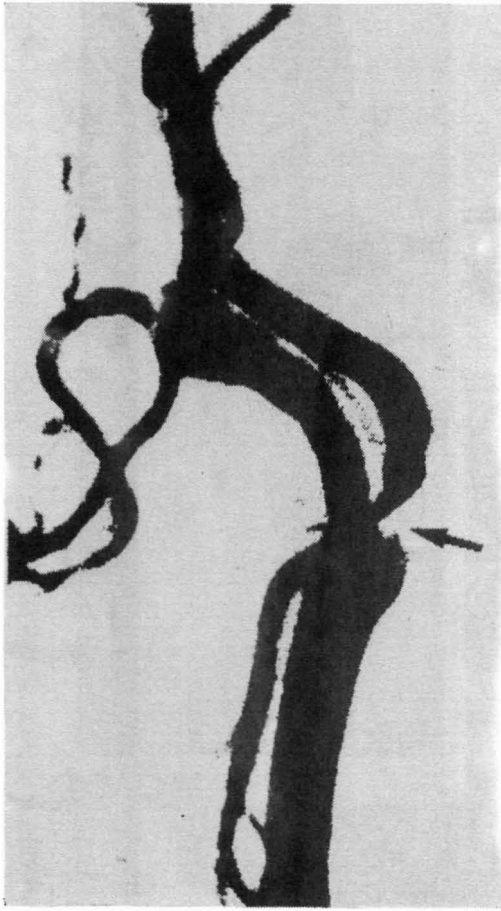


D





**FIG. 9.** Recurrent stenosis after angioplasty of the internal carotid artery. The patient is a 75-year-old female who has previously presented with a minor right hemispheric infarction with progressive neurological symptoms. Computerized tomogram shows small deep hypodensities in the right hemisphere. **A:** Narrow stenosis of the origin of the right internal carotid artery (*arrow*). **B:** "Cerebral" arch angiogram (see text) shows a delay in the arterial hemispheric filling on the right side (*first row of frames*) and a stasis on the right side on the late phase (*fourth row of frames*). **C:** Carotid angiogram after angioplasty. Because of the hardness of the plaque, the lumen of the carotid has not been totally opened (*arrow*). **D:** "Cerebral" arch angiogram immediately after angioplasty. Although the arterial filling now appears rather symmetrical (*first row*), it remains a stasis on the right side at the late phase (*fourth row*) that indicates that the dilatation has not been hemodynamically satisfactory. **E:** Recurrent stenosis, 6 months later (*arrow*). **F:** Second angioplasty. Carotid angiogram after dilatation shows a better morphological result (*arrow*). **G:** "Cerebral" arch angiogram immediately following the second angioplasty shows symmetrical arterial and late phases. The patient has not presented a recurrent stenosis (2-year follow-up).





**FIG. 11.** Multistenotic patient who has presented a left hemispheric infarction with residual moderate deficit of the right arm. Computed tomogram shows a left fronto-rolandic hypodensity. **A:** Angiogram prior to angioplasty shows an occlusion of the left internal carotid artery (*arrow*), a narrow stenosis of the right internal carotid artery (*double arrow*), and a narrow stenosis of the left subclavian artery (*open arrow*). There is no filling of the left vertebral artery on the arterial and late phases. The left external carotid artery does not participate in the cerebral supply. **B:** Angiogram after angioplasty of the right internal carotid artery (*double arrow*) and subclavian artery (*open arrow*). The left vertebral artery (*arrow*) now fills during the arterial phase. This patient has three efficient arteries participating in the supply of the brain instead of one before angioplasty, and has not presented recurrent neurological symptoms (4-year follow-up).

The triple coaxial system of carotid angioplasty has several components and requires special manipulations (Fig. 7):

1. The guiding catheter is positioned in the common carotid artery, by the transfemoral approach.
2. Occlusive and angioplasty catheters are introduced. The occlusive latex balloon is positioned at approximately the C2-C3 level, and the center of the angioplasty balloon is situated at the level of the stenosis.
3. Intravenous injection of heparin (7000-10,000

I.U.) is given, immediately before inflation of the angioplasty balloon. The clinical tolerance to the carotid occlusion by balloon inflation is then tested.

4. The angioplasty balloon is now inflated. The guiding catheter should be at the inferior edge of the angioplasty balloon to prevent its longitudinal displacement during maneuvers of inflation and deflation.

5. The angioplasty balloon is deflated and the catheter is withdrawn while the occlusive balloon remains inflated, occluding the internal carotid artery. The tip of the angioplasty catheter remains at the valve connector

**FIG. 10.** Angioplasty of the internal carotid. Use of stents. This 58-year-old female had previously presented with a left hemisphere infarction with neurological symptoms which have cleared. Computerized tomogram shows a left parietal limited hypodensity. **A:** Carotid angiogram prior to angioplasty shows a narrow stenosis of the internal carotid artery (*arrow*). **B:** Angiogram immediately following angioplasty shows a suboptimal morphological result (*arrow*). **C:** Positioning (in the same session) of two endovascular strecker stents. One is in the internal carotid artery. The second is in the common carotid artery. **D:** Follow-up angiogram (6 months) after treatment. Good morphological results. The patient has not presented any new neurological symptom.

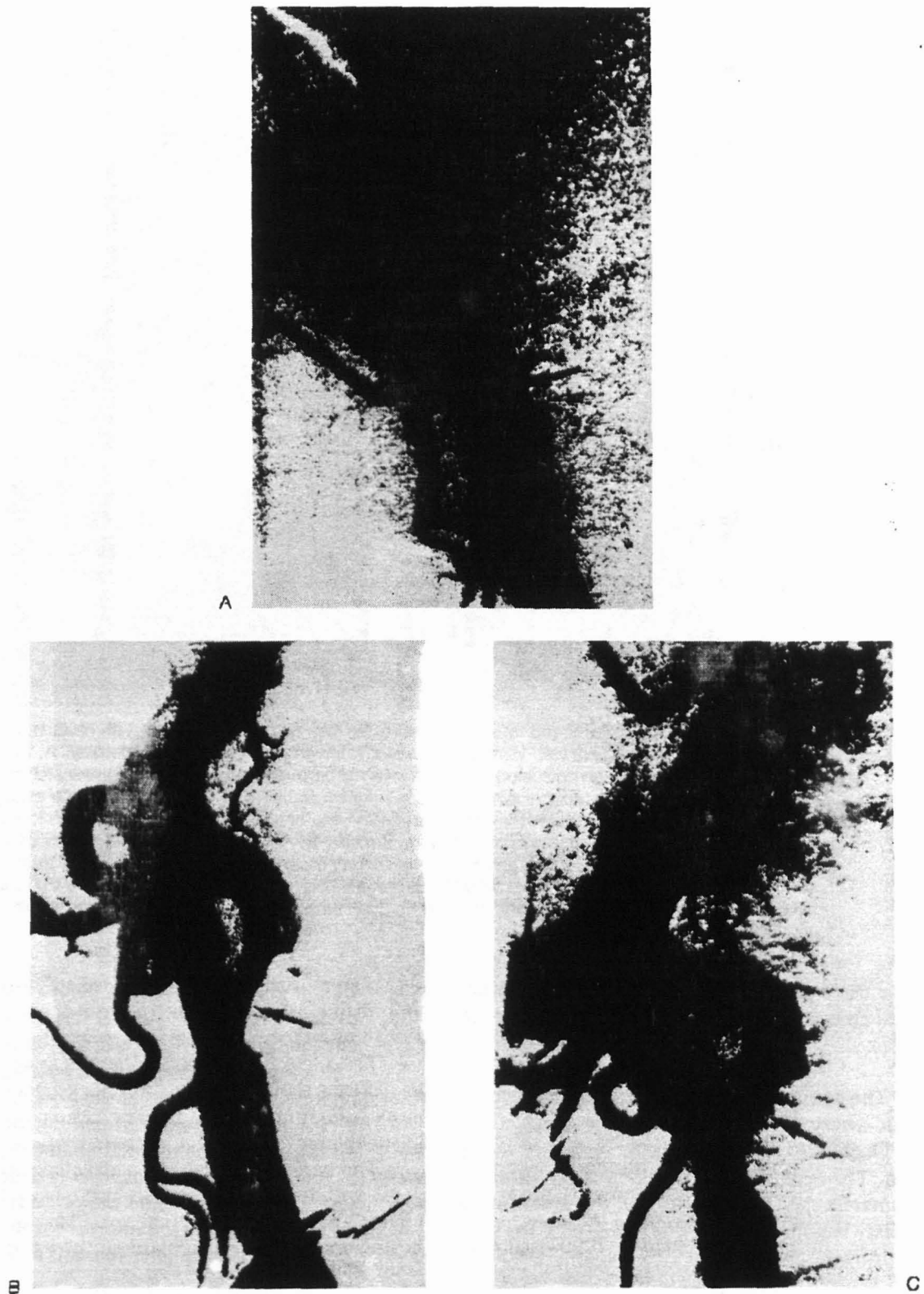


FIG. 12. Left internal carotid thrombosis with occlusion of the middle cerebral artery. Acute onset. This 54-year-old patient presented with a complete left hemiplegia. Intra-arterial thrombolysis and internal carotid angioplasty were performed. A: Carotid angiogram prior to treatment shows a complete occlusion of the internal carotid artery (arrow). Intracerebral series do not show any revascularization from the opposite side of the vertebral system. Presumed bad prognosis. B: Intra-arterial thrombolysis (urokinase) performed 4 hr after the onset of symptoms. Carotid angiogram shows the narrow internal carotid stenosis responsible for the occlusion (arrow) and deemed to thrombose again. C: Minimal internal carotid angioplasty performed in a course of thrombolysis to keep open the internal carotid artery (arrow). The patient keeps a moderate deficit on the right side (1-year follow-up).

of the guiding catheter. The plaque debris and clots potentially dislodged during the inflation of the angioplasty balloon are aspirated with a 20-ml syringe through the guiding catheter. The blood aspiration is followed by manual, high-pressure injection of normal saline order to direct residual debris into the lumen of the external carotid artery.

6. The occlusive latex balloon is then deflated and withdrawn. An immediate postangioplasty angiogram is always performed. When the morphological results are unsatisfactory, the same or larger angioplasty balloon is repositioned in the stenosis and a new angioplasty is performed.

7. The heparinization is reversed and the femoral sheath is removed.

We believe that a cerebral protection system is mandatory in angioplasty of atherosclerotic stenoses involving the common carotid artery bifurcation. This conclusion is based upon (a) our higher rate of embolic complications in the nonprotected group and (b) the frequent presence of cholesterol crystals observed in the blood aspirated immediately after the balloon angioplasty.

The immediate angioplasty results are usually good when the atherosclerotic plaque is partially calcified (Fig. 8). When the plaque is completely calcified, a complete dilatation of the artery is not always successful and there is an increased risk of recurrence of the stenosis (Fig. 9).

We now study the pre- and postangioplasty brain hemodynamic changes using a modified windowing on digital angiography. The injection of contrast material in the aortic arch allows a better understanding of the vascularization of the brain parenchyma if the computer windows and levels are low and narrow (Fig. 9). Before angioplasty, two signs may be observed in the involved brain hemisphere: (i) delay in arterial visualization and (ii) parenchymal stasis on the later phase of the angiogram. When the carotid dilatation has been suboptimal (Fig. 9), symmetrical arterial filling is observed but the stasis in the brain parenchyma remains.

A postangioplasty recurrent stenoses can be suspected on Doppler ultrasonography. A second angioplasty is usually simpler, allowing visualization of symmetrical arterial and late filling on the cerebral arch angiogram (Fig. 9). This technical complication has occurred in 10% of our carotid angioplasties in atherosclerotic lesions.

#### FUTURE DEVELOPMENTS IN INTRAVASCULAR ANGIOPLASTY

In two patients, the risk of recurrent stenosis has led us to position endovascular Strecker stents in the carotid artery (32). These patients presented with a transient stroke and hypodensities in the head computerized tomogram. Angioplasty of the carotid artery had been sub-

optimal in the two cases (Fig. 10). In one case a stent was positioned in the internal carotid artery, and in the other the stent involved the common and internal carotid arteries. Satisfactory anatomical and clinical results were observed in the two cases. A Strecker stent has been recently used in the ostium of a vertebral artery in a patient presenting with recurrent, severe vertebrobasilar symptoms, 2 years after the first angioplasty.

Angioplasty has the advantage of treating stenosis of multiple arteries in one session. This simultaneous angioplasty of the carotid and vertebrobasilar circulation may potentially improve the cerebral circulation in patients with severe, diffuse atherosclerosis (Fig. 11).

Angioplasty may be part of the emergency treatment in stroke patients. This is especially true in those cases related to occlusion of the vertebral or internal carotid arteries distal to a stenosis at their origins. Fibrinolysis of the internal carotid and middle cerebral arteries may be performed, depending upon the time of the clinical onset of stroke (33). An incomplete angioplasty of the internal carotid artery will be enough to keep this artery opened (Fig. 12).

We strongly believe that angioplasty will be incorporated among several therapeutic modalities to be used in cervical and intracranial vascular ischemic pathology. One may imagine systems allowing the introduction of intravascular instruments to visualize, thrombolize, and dilate stenoses or remove atherosclerotic plaques in a single session.

#### REFERENCES

- Dotter CT, Judkins MP. Transluminal treatment of arteriosclerotic obstruction: description of a new technique and a preliminary report of its applications. *Circulation* 1964;30:654-670.
- Gruntzig A, Hopff H. Perkutane Rekanalisation chronischer arterieller Verschlüsse mit einem neuen Dilatationskatheter. Modification des Dotter Technik. *Dtsch Med Wochenschr* 1974;99:2502-2505.
- Kerber CW, Cromwell LD, Leohdenol. Catheter dilatation of proximal stenosis during distal bifurcation endarterectomy. *AJNR* 1980;1:348-349.
- Hodgins GW, Dutton JW. Subclavian and carotid angioplasty for Takayasu's arteritis. *J Can Assoc Radiol* 1982;33:205-207.
- Derauf BF, Hunter DW, Erickson DL, Castaneda-Zuniga WR, Cardella JF, Amplatz K. "Wash-out" technique for brachiocephalic angioplasty. *AJR* 1985;146:849-851.
- Hasso AN, Zinke DE, Thompson JR. Fibromuscular dysplasia of the internal carotid artery: percutaneous angioplasty. *AJNR* 1981;2:175-180.
- Garrido E, Montoya J. Transluminal dilatation of internal carotid artery in fibromuscular dysplasia: a preliminary report. *Surg Neurol* 1981;16:469-471.
- Belan A, Vesica M, Vanek I, Weiss K, Peregram JJ. Percutaneous angioplasty of fibromuscular dysplasia of the internal carotid. *Cardiovasc Intervent Radiol* 1982;5:79-81.
- Motajeme A, Keifer JW, Zuska AJ. Percutaneous transluminal angioplasty of the brachiocephalic arteries. *AJR* 1982;138:457-462.
- Théron J, Courtheoux P, Henriet JP, Pelouze G, Derlon JM, Maiza D. Angioplasty of supraaortic arteries. *J Neuroradiol* 1984;11:187-200.



**IV INTERNATIONAL COURSE ON VASCULAR AND INTERVENTIONAL RADIOLOGY AS THERAPEUTIC ALTERNATIVE**

11. Vitek JJ. Percutaneous transluminal angioplasty of the external carotid artery. *AJNR* 1983;4:796-799.
12. Mathias K. Perkutane transluminale katheterbehandlung suprasonticr Arterienobstruktion. *Angiology* 1981;3:47-50.
13. Wiggli V, Gratzl O. Transluminal angioplasty of stenotic carotid arteries: case reports and protocol. *AJNR* 1983;4:793-795.
14. Vitek JJ, Raymond BC, Oh SJ. Innominate artery angioplasty. *AJNR* 1984;5:113-114.
15. Bockenheimer SA, Mathias K. Percutaneous transluminal angioplasty in arteriosclerotic internal carotid artery stenosis. *AJNR* 1983;4:791-792.
16. Tsai FY, Matovitch V, Hieshima G, et al. Percutaneous transluminal angioplasty of the carotid artery. *AJNR* 1986;7:349-358.
17. Kachel R, Eadert G, Basche S, Grossman, Glaser F. Percutaneous transluminal angioplasty (dilatation) of carotid, vertebral and innominate artery stenoses. *Cardiovasc Intervent Radiol* 1987; 10:142-146.
18. Freitag J, Koch RD, Wagemann W. Percutaneous angioplasty of carotid artery stenoses. *Neuroradiology* 1986;28:126.
19. Imperato AM, Riler TS, Ecorstein F. The carotid bifurcation plaque: pathologic findings associated with cerebral ischemia. *Stroke* 1979;10:238-245.
20. Théron J, Raymond J, Casasco A, Courtheoux P. Percutaneous angioplasty of atherosclerotic and post-surgical stenosis of carotid arteries. *AJNR* 1987;8:499-500.
21. Théron J, Courtheoux P, Alachkar F, Maiza D. New triple coaxial catheter system for carotid angioplasty with cerebral protection. *AJNR* 1990;11:869-874.
22. Castaneda-Zuniga WR, Formanek A, Tadavarthy M, et al. The mechanism of balloon angioplasty. *Radiology* 1980;135:565-571.
23. Block C, Fallon JT, Elmer D. Experimental angioplasty: lessons from the laboratory. *AJR* 1980;135:907-912.
24. Zöllhoffer CL, Salomonovitz E, Sibley R, et al. Transluminal angioplasty evaluated by electron microscopy. *Radiology* 1984; 153:372-374.
25. Théron J, Melancon D, Ethier R. "Pre" subclavian steal syndromes and their treatment by angioplasty. *Neuroradiology* 1985;27:265-270.
26. Bachman DM, Kim RM. Transluminal dilatation for subclavian steal syndrome. *AJR* 1980;136:995-996.
27. Théron J, Tyler JL. Takayasu's arteritis of the aortic arch: endovascular treatment and correlation with positron emission tomography. *AJNR* 1987;8:621-626.
28. Fischer CM, Gore I, Okabe N, White P. Atherosclerosis of the carotid and vertebral arteries: extracranial and intracranial. *J Neuropathol Exp Neurol* 1963;24:455-476.
29. Franceschi C. *L'investigation vasculaire par ultra-sonographie Doppler*. Paris: Masson, 1980.
30. George B, Laurian C. Vertebrobasilar ischemia. Its relation to stenosis and occlusion of the vertebral artery. *Acta Neurochir* 1982;62:287-295.
31. Sundt TM, Smith HC, Campbell JK, Vliestra RE, et al. Transluminal angioplasty for basilar artery stenosis. *Mayo Clinic Proc* 1980;55:673-680.
32. Hieshima GB, Higashida RT, Wapenski J, Halbach VV, Cahan L, Bentson JR. Balloon embolization of a large distal artery aneurysm. *J Neurosurg* 1986;65:413-416.
33. Tievsky AL, Drury EM, Mardiat JG. Transluminal angioplasty in post-surgical stenosis of the extracranial carotid artery. *AJNR* 1983;4:800-802.
34. Strecker EP, Liermann D, Barth KH, Wolf HR, et al. Expandable tubular stents for treatment of arterial occlusive diseases: Experimental and clinical results. *Radiology* 1990;175:97-102.
35. Théron J, Courtheoux P, Casasco A, Alachkar F, Notari F, Ganem F, Maiza D. Local intra-arterial fibrinolysis in the carotid territory. *AJNR* 1989;10:753-755.

## **Cardio-Pulmonary Collapse: A Sequelae of Ethanol Embolotherapy**

WAYNE F. YAKES, M.D.

### **PURPOSE**

**T**o retrospectively evaluated the cause for cardio-pulmonary collapse in three patients undergoing ethanol embolization of their extremity vascular malformations.

### **MATERIAL & METHODS**

**F**our patients, one male and three females (aged range: 22-35 yrs; mean: 27 yrs), presented for ethanol endovascular management of their lower extremity vascular malformations (1-AVM, 3-venous malformations). All patients were treated while under general anesthesia. Late towards the end of the procedure, all patients demonstrated an increase in  $p\text{CO}_2$ , followed by a decrease in  $p\text{O}_2$  saturation, followed by a brady-arrhythmia and cardiopulmonary collapse.

### **RESULT**

**T**hree patients were resuscitated and had no adverse outcome. One patient failed resuscitative efforts and died.

### **CONCLUSION**

**I**ntravascular ethanol may have secondary effects which should be planned for. These include suppression of the myocardial conduction system and increased pulmonary artery pressures secondary to pre-capillary vascular spasm. Later efforts include peripheral hypotension and

myocardial ischemia with resultant arrhythmias. Because our patients were intubated under general anesthesia, they were perfectly controlled and three patients had no adverse outcome. If patients are not as controlled, delays in intubation and managing C-P collapse could lead to cerebral anoxia and other sequelae, and death, as occurred in one patient despite rapid resuscitative efforts.

## Ethanol Endovascular Ablation of Brain AVMs; Initial Results

WAYNE F. YAKES, M.D.

### PURPOSE

To determinate the safety and efficacy of undiluted absolute ethanol in the management of intra-axial brain AVMs as a primary mode of therapy.

### MATERIAL & METHODS

Six patients (3 males, 3 females) underwent transcatheter embolization of their brain AVMs (age range: 19-54 yrs; mean: 38 yrs). Clinical presentations included SAH, headache, seizure, and vertigo. Superselective catheterizations were performed. Amytal testing preceded all ethanol embolizations (8 procedures). Follow-up consisted of MR and arteriography. Two patients were lost to follow-up after 3 months (follow-up range: 3-13 months; mean: 6 months). Neuroleptic anesthesia was used in 5 patients; general anesthesia 1 patient.

### RESULTS

No recanalization or neovascular recruitment in any patient were observed at follow-up. When comparing the immediate post-embolization arteriograms to follow-up arteriograms, progressive AVM thrombosis routinely occurs. One patient with an occipital lobe hemorrhage and resultant homonomous hemianopsia recovered his visual field deficit on the angiographic table. A 50% complication rate was observed (1 patient episode of dizziness totally resolved; 1 patient with reading difficulty, totally resolved; 1 patient with bilateral upper outer quadrant visual field deficits, resolving).

### CONCLUSIONS

Ethanol can safely be used in the brain to treat AVMs. Progressive AVM occlusion is a common finding at follow-up. Our initial results indicate that ethanol has a permanence seldom encountered in AVM therapy.



## Management of Vascular Malformations

WAYNE F. YAKES, M.D.<sup>1</sup>

125 patients with vascular malformations in all anatomic locations were treated by ethanol endovascular ablation as a primary mode of therapy. 40% (50 patients) of these patients had prior failed therapies to include surgery, radiation therapy, and embolotherapy with other agents (IBCA/NBCA, PVA, coils, detachable balloons, sotradecol). All these treatment modalities failed in that recanalizations, neovascular recruitment and usually worsening of the initial symptoms at follow-up was demonstrated in this group of patients. Fifty-two patients presented with high-flow malformations (AVM, AVF) and 73 patients presented with low-flow malformations (venous and/or lymphatic malformations).

The results of ethanol endovascular ablation were extremely gratifying, of significance, the phenomena of neovascular recruitment, vascular recanalizations and vascular malformation recurrence were not observed in any patient. Further, in high-flow malformations, unembolized arterial feeders regressed as the AVM was ablated. In high-flow malformations and low-flow malformations cures were achieved in 53% of patients. Not all patients required cure to totally relieve the presenting symptoms. This group of patients who still have portions of their vascular malformations remaining, although asymptomatic, are being followed and would undergo follow-up therapy if symptoms recur. Follow-up in all patients is: range 3 months-9 years; mean 3 years.

Follow-up consist of, where appropriate, MR, ultrasound, arteriography and venography. An overall complication rate of 10% was observed.

Our conclusions are that ethanol ablation of vascular malformations is efficacious in their management as a primary form of therapy. Ethanol consistently provides a level of permanent occlusion that is seldom achieved by other agents. For the first time, long-term cures are possible by endovascular means alone. Consistent improvement in patient symptoms routinely occurs. A total complication rate of 10% is acceptable in light of the problems encountered by these malformations, the complication rates of alternative therapies, the rarity of curing these lesions by these alternatives therapies, and the permanence that can be achieved by the use of ethanol.

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Denver, C.O. U.S.A.

## Recent Advances in Chemoembolization of Liver Cancer

RYUSAKU YAMADA, M.D.

### INTRODUCTION

Since 1977 we have performed transcatheter arterial chemoembolization (TAE) and related therapies in more than 1,500 patients with liver cancer. In this session I would like to discuss our experience and approach to TAE for liver cancer.

Transcatheter therapies, including hepatic artery chemoembolization and arterial infusion, are currently considered first for treatment of unresectable liver cancer. However, the strategy selected should be based on knowledge of concurrent or traditional therapies: it is essential for clinical transcatheter therapists, who confront every type of difficulty, to have some alternative modalities. Systemic chemotherapy for the treatment of liver cancer is largely unsuccessful.

### CHEMOEMBOLIZATION WITH VARIOUS EMBOLIC MATERIALS

For tumor embolization, embolic materials are commonly mixed with anticancer drugs. Injection of embolic material should be carefully monitored under fluoroscopy to avoid unexpected reflux which tends to occur when the blood flow becomes slow.

#### Gelatin sponge (Gelfoam) particles

Gelatin sponge particles are a commonly used embolic material which is less inflammable and non antigenic. It is available as Gelfoam, Spongel or Spongostan. A gelatin sponge block is sliced with a blade and cut with scissors into particles 1 mm in size. The particles are then soaked in a mixed solution of anticancer agents and low osmolarity water-soluble contrast medium. Adriamycin 20 mg and mitomycin C 10 mg are mixed together to form an embolic material. The TAE for hepatoma brings about selective coagulation necrosis of a tumor without damage to surrounding liver parenchyma. Absorption of a gelatin sponge occurs within 2 weeks.

#### Gelatin sponge powder

Gelatin sponge powder is usually used in combination with gelatin sponge particles. The powder causes more peripheral arterial occlusions because of its smaller size, varying from 30 to 600  $\mu$  m, with stronger ischemic effect on the tumor tissue.

Therefore, inadvertent embolization with gelatin sponge powder can cause infarction of not only peripheral liver parenchyma but also gallbladder, bile duct, pancreas and small intestine.

#### Lipiodol (ethiodol) injection therapy

Lipiodol is said to retain selectively in the tumor, a fact that has led to its therapeutic use in hepatoma patients, in which it is used as a carrier of anticancer agents. SMANCS/Lipiodol, a mixture of lipiodol and styrene maleic acid neocarzinostatin, has been recently applied with fairly good results for the treatment of unresectable hepatomas.

Lipiodol infusion followed by gelatin sponge TAE ("Lp-TAE") enhances the effects of TAE; however, it may cause liver infarction. In our animal experiments with Lipiodol infusion followed by gelatin sponge TAE. The incidence of liver infarction was 100% in those embolized with 0.2 ml per kg of Lipiodol and gelatin sponge particles. Our conclusion was that the dosage of Lipiodol infusion followed by gelatin sponge TAE should be limited to below 0.1 ml/kg for the normal part of the liver.

#### Ivaron particles

Ivaron, a polyvinyl alcohol foam which is commercially available in the United States, reaches smaller arteries because of its smaller diameter, and occludes more permanently due to its nonabsorbable and inflammatory nature.

#### Degradable starch microsphere infusion

Degradable starch microsphere (Pharmacia) is 40  $\mu$  m in diameter. Within half an hour, this

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temporarily occludes blood flow at the precapillary arterioles during degradation by the serum amylase. DSM is used as a mixture with anticancer agents, injected through a catheter into the hepatic artery, and attains a higher local concentration during the quarter of half an hour of the blockade. However, the tumor vessel of the hepatoma is usually more coarse than that of the secondary tumor, and the reported dosage for complete occlusion varies wildly from 150mg to 2,700mg. If the tumor vessel is too large to be occluded by the usual administration of DSM, the balloon catheter technique may be helpful.

#### **Steel coil embolization**

Similar to arterial ligation therapy, the steel coil usually has a weak ischemic effect on the tumor because the coil occludes only larger arteries and keeps peripheral vascular lumen open. This coil is also used in hepatic blood flow redistribution when the arterial flow needs to be changed prior to embolization or arterial infusion.

#### **ARTERIAL INFUSIONS, ALTERNATIVES TO TAE**

Various Arterial injection chemotherapies, such as bolus injection of anticancer agents, continuous injection, sometimes with implantable porto and balloon occluded artery infusion, are carried out when the main portal vein or its first order branch is occluded by a tumor thrombus or invasion because TAE can not be indicated in such cases.

US guided direct injection chemotherapy combined with TAE and percutaneous ethanol injection after TAE are going to be the effective alternatives to chemoembolization.

#### **EFFECTS OF TRANSCATHETER ARTERIAL CHEMOEMBOLIZATION**

TAE usually shows a marked decrease in density of tumor without any change in density of the surrounding liver parenchyma, which indicated selective necrosis of the tumor. Furthermore, sequential CT examinations revealed that the contrast medium containing anticancer agents had remained selectively in the tumor tissue at a high concentration until 48 hours after TAE, although the liver parenchyma surrounding the tumor showed no accumulation of contrast medium.

These observations on the dynamics of anticancer agents were also confirmed by measurement of the

drug concentration in a surgical specimen of the tumor tissue obtained 48 hours after TAE.

Sequential CT examination also disclose a selective accumulation of contrast medium in the tumor tissue for 48 hours or more after TAE, these facts may mean that TAE is a good drug delivery system; this is regarded as a targetting chemotherapy known as "chemoembolization". For this reason, TAE for liver cancer can be expected to produce both ischemic and chemical necrosis of the tumor.

Side affects and complications of TAE and long term results of TAE will be also discussed in this session.

## Use of the Wallstent in the Venous System Including Hemodialysis-Related Stenoses

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**Abstract.** Eighteen patients with a total of 23 venous stenoses or occlusions were treated with the Wallstent. In 5 patients treated for malignant stenosis there was one primary failure due to insufficient stent expansion. The other 4 patients showed rapid relief of their inflow obstruction, all remaining asymptomatic despite later stent occlusion in 1 patient. Four patients were treated for benign postoperative stenoses of the iliac or femoral vein. All stents remained patent for a period of 6 weeks–58 months. Nine patients were treated for one or multiple stenoses along the venous outflow tract of hemodialysis fistulas. Of 14 lesions that were eventually stented, 12 are still patent after 3–27 months (mean 19). However, 10 secondary interventions (eight percutaneous transluminal angioplasty (PTA), two stents) and three additional stent procedures for new lesions were necessary. Although our experience is limited, we believe that patients with tumor compression or postoperative strictures of large veins benefit from treatment with stents. Stenting of venous outflow stenoses in hemodialysis fistulas can significantly prolong stent function, however, PTA should always be the first treatment of choice.

**Key words:** Dialysis shunts—Veins, transluminal angioplasty, grafts and prostheses

Treatment of venous stenoses with percutaneous transluminal angioplasty (PTA) has a high rate of recurrence [1, 2]. Moreover, the fibrotic nature of many benign venous stenoses or external malignant

compression often preclude a primary effective dilatation. Endovascular stents have been successfully used in the arterial system mainly for atherosclerotic disease. It therefore seems logical that the technique of an inner mechanical support to prevent collapse or compression of a lumen as well as recurrences should also be applied in the venous system [3–9]. Of special interest are obstructions of the vena cava, large caliber central veins, and the venous outflow tract of dialysis shunts. Herein we report our experience in 18 patients using self-expanding endovascular stents of the Wallstent type.

### Material and Methods

Eighteen patients with a total of 23 venous stenoses or occlusions were treated with the Wallstent. There were 12 women and 6 men, aged 26–78 years (mean 47 years). According to localization and etiology of the lesions the patients were divided in three groups: 1) Palliative treatment of tumor compression in 5 patients: superior vena cava (2); inferior vena cava (1); iliac vein (1); confluence of basilic and axillary veins in 1 patient with breast carcinoma. 2) Postoperative recurrent stenoses in 4 patients: 1 had recurrent stenosis of the common iliac vein after operation of a venous spur. 2 had stenoses of the common femoral vein after crosssection of the greater saphenous vein, and 1 had a long segment stenosis after surgical reconstruction for traumatic severance of the superficial femoral vein. 3) Stenoses of the venous outflow tract were treated in 9 patients on chronic hemodialysis (Table 1): Five patients had peripheral lesions stented (cephalic vein, basilic vein) for recurrent stenoses (4) or primarily insufficient result after PTA (1); 2 of these patients developed additional proximal stenoses 6 and 16–24 months later. Four patients had isolated central lesions of the innominate vein.

The characteristics and releasing mechanism of the Wallstent have been described previously in this issue. Most stents were implanted inguinally with the exception of the peripheral stents in patients on hemodialysis where the draining vein was directly punctured. For the superior vena cava (SVC), stents of 14-mm diameter were used in 2 patients. Two overlapping 25-mm prototype stents were used in 1 patient with metastatic obstruction of the inferior vena cava (IVC). For stents in the femoral vein and

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**Table 1.** Hemodialysis-related stenoses

Location	No. of patients	No. of lesions	Stent diameter (mm)
(a) Isolated proximal lesion (brachioceph. vein)	4	4	10–12
(b) Isolated peripheral lesions	3	3	6–8
(c) Peripheral lesion with consecutive proximal lesion	2	2/4	8–10

(a) Stenosis in the brachiocephalic vein; (b) lesions with a few cm of the AF fistula or at or near a brachial anastomosis of an autologous or xenograft; (c) 2 patients from (b) who later developed additional lesions

iliac vein, stent sizes were 10–14 mm. For the brachiocephalic vein we used diameters of 10 or 12 mm and for peripheral veins, such as the basilic or cephalic veins, 6 or 8 mm. Stents up to a diameter of 10 mm can be introduced through a 7 French introducing sheath, stents from 12–16 mm through a 9 French sheath, and for the prototype of 25 mm diameter stents, a 20 French introducer sheath was used. Most obstructing lesions were dilated with a balloon catheter before stent application. In cases where the endoprosthesis would not spontaneously open to a sufficient degree, balloon dilatation was performed within the implanted stent. In such cases the dimensions of the balloons were selected 1 mm smaller than the diameter of the completely expanded stent. If needed, two stents were placed in tandem overlapping each other to cover the entire length of the stenosis.

Analogous to conventional PTA, 5,000 U of heparin were given intravenously during the procedure. The patients were then treated with intravenous heparin of 800–1,000 U/h for 2–4 days, starting peroral anticoagulation with coumadin the same day. Unless contraindicated, peroral anticoagulation was continued for at least 6 months. The patients were followed clinically at regular intervals at 1, 3, 6, and 12 months and yearly thereafter. If feasible, color duplex flow studies were done at these follow-ups and phlebography, if possible, at 6 and/or 12 months.

## Results

### Tumor Stenosis

The results are summarized in Table 2. Three of the 5 patients treated for malignant obstruction died after 6 weeks–9 months. None of these patients showed recurrent inflow obstruction at the time of death. One patient, however, with SVC syndrome showed reocclusion of the stented segment on phlebography 4 weeks after stent placement. In this patient, recanalization with thrombolysis and thrombus aspiration had to be performed 3 days after implantation of the stent. Of the 2 surviving patients, 1 with two stents in the IVC has been asymptomatic for 1 year (Fig. 1). In the other patient with a very tight stenosis at the inflow of a double basilic vein into the axillary vein, the two implanted stents did not open sufficiently in spite of vigorous addi-

tional balloon dilatation, and stent occlusion occurred 2 days after placement. The patient was taken to surgery where a hard tumor stenosis was found and the entire vein segment was bridged with a graft.

### Benign Stenosis after Previous Operation

All patients currently have patent stents and are asymptomatic after a follow-up time of 6 weeks–58 months (Table 3). One patient, who was stented for a recurrent stenosis after surgery of a traumatically severed superficial femoral vein, showed moderate intimal hyperplasia of about 25% of the luminal diameter after 6 weeks. The intimal thickening, however, remained stable and the patient has been asymptomatic for more than 4 years (Fig. 2). The patient who was stented for a recurrent venous iliac spur has been free of symptoms for almost 5 years. The 2 other patients with stents in the common femoral vein are also asymptomatic but have only a limited follow-up of 6 weeks and 4 months (Fig. 3).

### Hemodialysis Shunts

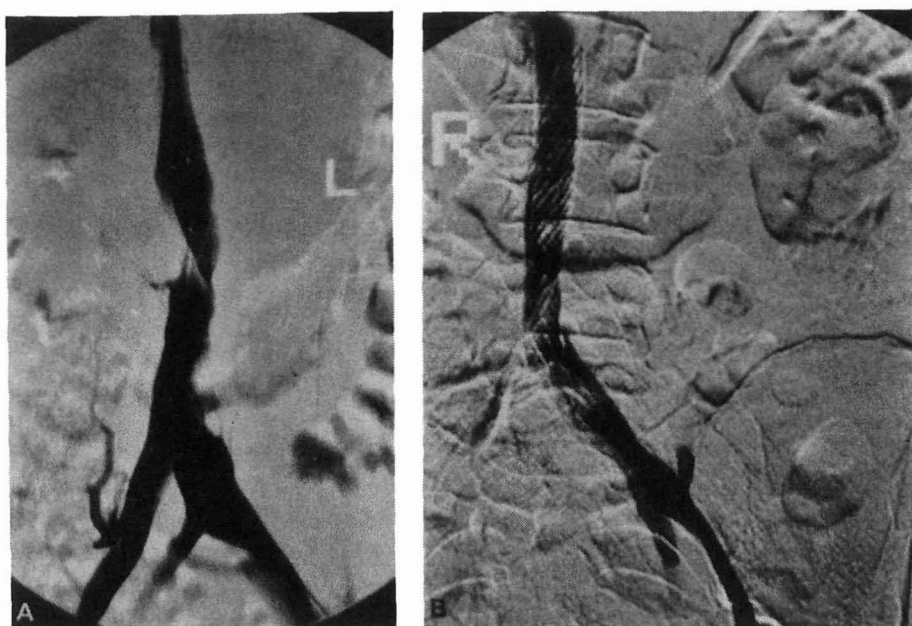
Nine patients were treated for one or multiple stenoses developing in the course of chronic hemodialysis along the venous outflow tract of the AV-fistula. These patients can be classified into two groups according to the localization of the stenotic lesion (Table 4). The first group (a) comprises patients with stenoses centrally in the region of the brachiocephalic vein, and the second group (b) are patients who had lesions either within a few centimeters of the AV-fistula or at or close to a brachial anastomosis of an autologous or xenograft. A subgroup (c) consists of 2 patients from group (b) who later developed additional proximal lesions (Table 1).

**Proximal Lesions.** These were found in 6 patients. In 2 of these, proximal lesions in the region of the subclavian and axillary vein developed between 7 and 24 months after previous stenting of peripheral lesions (Tables 1 and 4, group (c)). In the other 4 patients, the innominate vein was stented for recurrent stenoses after PTA in 3 and as primary treatment in 1 patient. One patient died 5 months after stenting, dialysis was possible until death. However, the patient suffered from arm swelling and phlebography 1 month prior to death, when a new stenosis 3 cm proximal to the stented lesion was found. The stent itself was completely patent without any signs of recurrent stenosis (Fig. 4). The follow-up of the other 3 patients ranges from 3½ to

**Table 2.** Tumor stenoses and results

Location	No. of patients	No. of stents	Living (mos)	Deceased (mos)	Free of symptoms	Stent patency	Stent diameter (mm)
V. cava	3	4	1 (12)	2 (1½)	3/3	2/3	14 + 25
Iliac V.	1	1	—	1 (9)	1/1	1/1	14
Basilic/axill. V.	1	2	1	—	0/1 <sup>a</sup>	0/1 <sup>a</sup>	8
Total	5	7	2/5	3/5	4/5	3/5	

<sup>a</sup> Patient free of symptoms after surgical bypass



**Fig. 1.** A 47-year-old female with metastatic ovarian carcinoma postsurgery and irradiation. **A** Inferior vena cavography shows severe infrarenal stenosis and excentric tumor compression of the left common iliac vein. **B** Cavography 6 months after placement of two overlapping Wallstents 25 mm in diameter shows complete patency. The patient has been asymptomatic for 1 year.

39 months. The patient with the longest follow-up received a kidney transplant 8 months after stenting, with resection of the AV-fistula at the time of operation. A control phlebogram 18 months after stent implantation showed a widely patent stented innominate vein. However, a new high-grade stenosis had developed at the orifice of the subclavian vein. As the patient has remained asymptomatic with good collateral drainage via the internal jugular vein, no further treatment has become necessary.

*Peripheral Lesions.* Follow-up of the various stented lesions in the 5 patients vary from 7 to 34 months (Tables 1 and 4). There was one acute occlusion one day after stenting of a long segment stenosis of the basilic vein after undue sportive activity the day following stent placement. Percutaneous recanalization was successful, however, 4½

months later the patient had severe intimal hyperplasia of the stented segment as well as of the native vein proximal to the stent. A secondary intervention was denied and the graft occluded.

A second patient received a kidney transplant 5 months after stenting of a graft anastomosis and phlebography 1 month later showed a distinct intimal hyperplasia. The patient was last seen 11 months after stenting with a clinically open stent. An additional patient had to be redilated 7 and 13 months after stenting with a functioning stent after a total of 17 months (Fig. 5). Two other patients (subgroup c) received kidney transplants 22 and 27 months after the first stenting. Recurrent intimal hyperplasia in these 2 patients, however, required 10 secondary interventions, the first starting between 3 and 9 months after stenting. Seven of these secondary interventions were necessary in 1 patient, re-

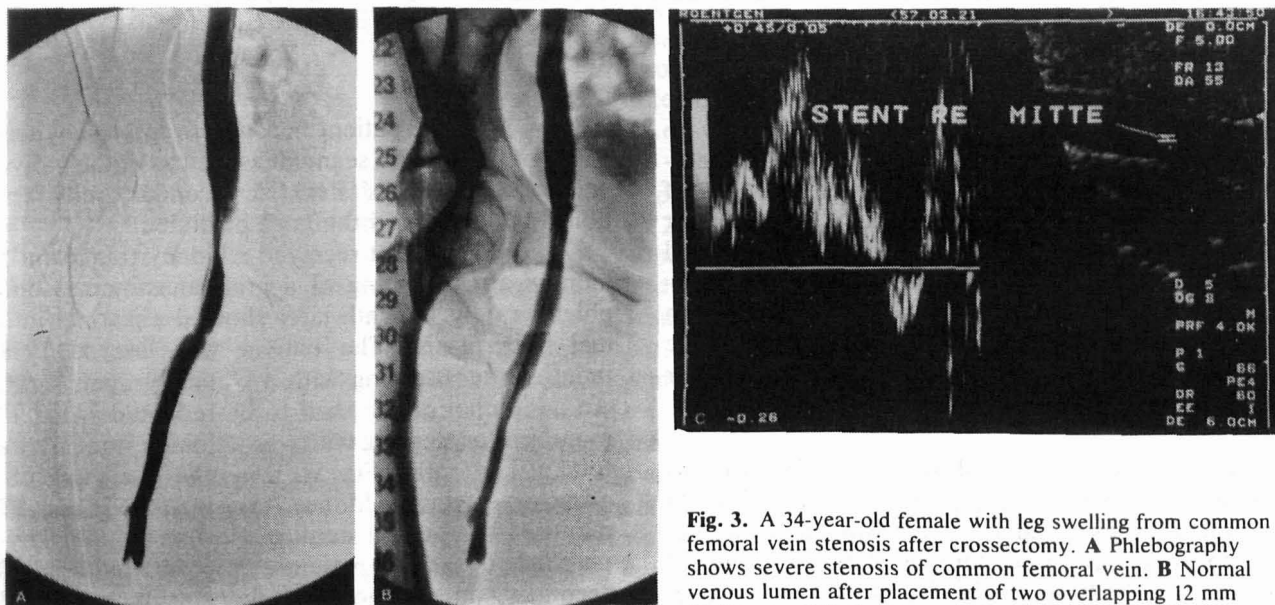
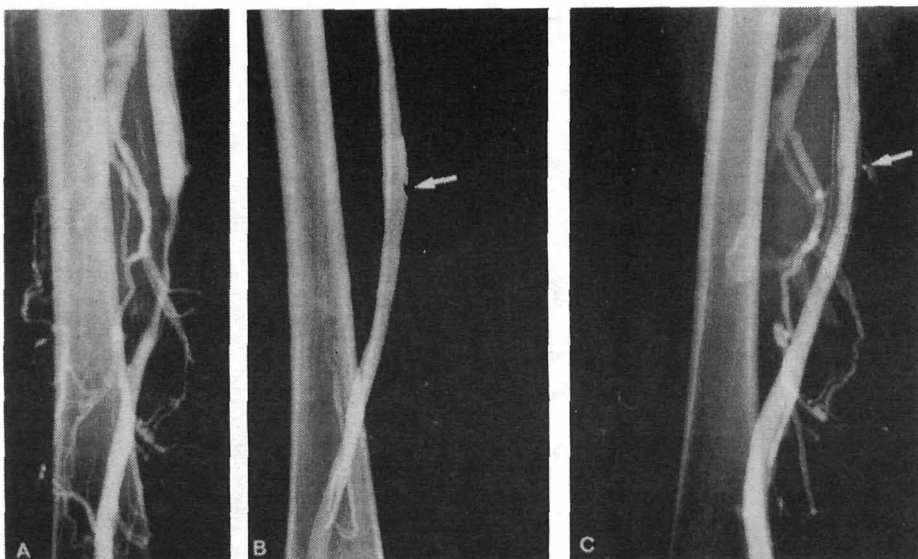


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**Table 3.** Benign postoperative stenoses and results

Location	No. of patients	No. of stents	Follow-up (mos)	Free of symptoms	Stent patency	Stent diameter (mm)
Iliac vein	1	1	58	Yes	Yes	14
Common femoral vein	2	3	1½ + 4	Yes	Yes	10 + 12
Superficial femoral vein	1	2	52	Yes	Yes	12 + 14
Total	4	6	m 29	All	All	

**Fig. 2.** A 33-year-old female with surgical reconstruction of the superficial femoral vein after traumatic severence. **A** Antegrade phlebography shows severe stenosis of the superficial femoral vein 4 weeks after surgery. **B** Immediately after placement of a 10 mm and a 12 mm Wallstent in overlapping fashion from a retrograde femoral approach there is a well-established lumen of the superficial femoral vein. Note inflow from sidebranch (arrow). **C** Follow-up venogram after 2½ years shows adequate patency in spite of moderate intimal reaction. Note patency of venous side-branch (arrow). Patient has been asymptomatic for 4½ years.



**Fig. 3.** A 34-year-old female with leg swelling from common femoral vein stenosis after crosssectomy. **A** Phlebography shows severe stenosis of common femoral vein. **B** Normal venous lumen after placement of two overlapping 12 mm Wallstents. **C** Patient has widely patent common femoral vein on color Doppler at 4 months follow-up.

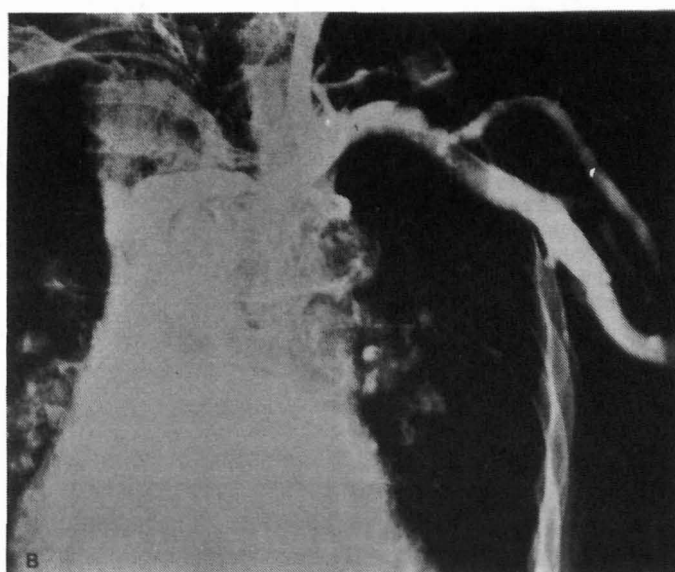
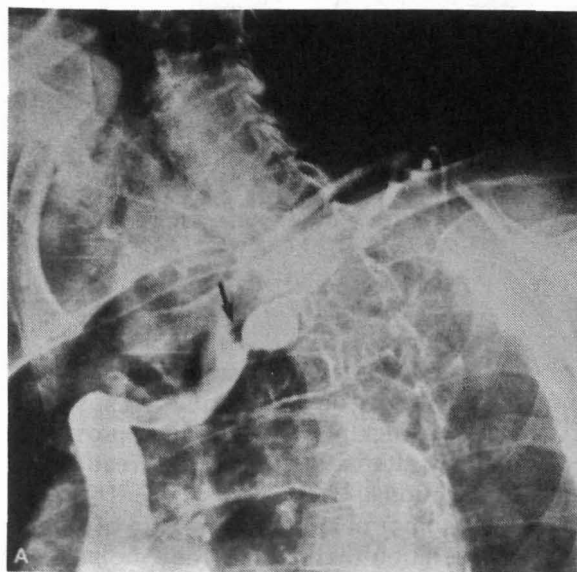
**Table 4.** Hemodialysis shunt-related stenoses and results

Location and no. of patients	No. of original lesions	Follow-up months (mean)	Patency <sup>a</sup>	Recurrence (mos)	2° Interventions	Occlusion <sup>a</sup>	No. of new lesions
(a) 4	4	3-39 (18)	4/4	0/4	—	—	1
(b) 2	3	7-17 (12)	2/3	3/3 (4-7)	1	1	—
(c) 2	1	24-34 (27)	6/6	2/2 (7-24)	2 PTA 3 <sup>b</sup> 1 Stent 6 PTA 7 <sup>c</sup> 5 Stents	—	1 <sup>b</sup>
	1					—	3 <sup>c</sup>
<b>Total</b>	<b>9</b>		<b>12/13</b>	<b>5/9</b>	<b>10</b>	<b>1/13</b>	<b>5</b>

Number of stented lesions

Pt 1: PTA of new lesion in subclavian vein, stented for recurrence. PTA of stented cephalic vein after 9 months

Pt 2: 6 × PTA of stented graft-basilic vein anastomosis and second stent. Segmental stenting of three additional proximal lesions



**Fig. 4.** A 78-year-old female on hemodialysis with left-sided arm swelling. **A** Retrograde phlebography shows severe stenosis at the confluence of the subclavian and internal jugular veins (arrow). **B** Four months after placement of a 12 mm Wallstent, the previous stenotic area is widely patent. No intimal reaction. However, a new stenosis has developed in the innominate vein. Dialysis was still possible, therefore no treatment was performed. The patient died 1 month later of underlying disease.

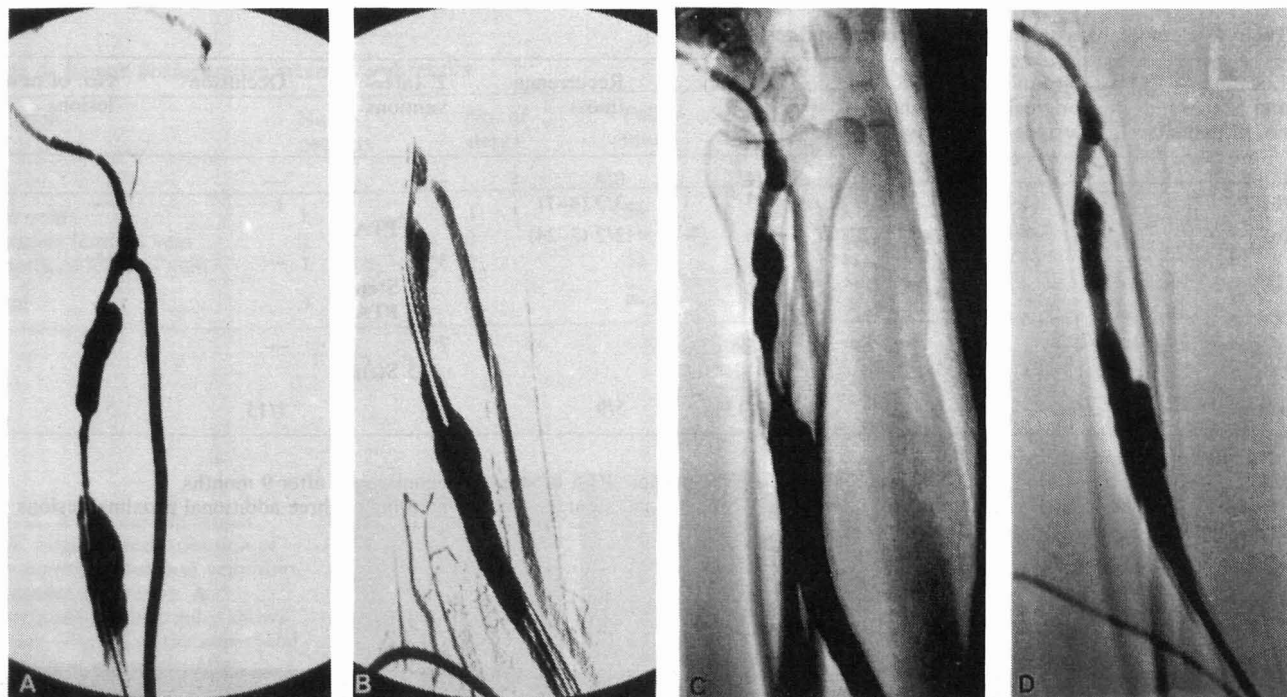
quiring six repeat PTAs at decreasing intervals of 5-2½ months, finally requiring a second stenting of the same lesion (Fig. 6).

In addition, both patients developed central lesions as mentioned above. In one of these a tight

stenosis at the origin of the cephalic vein developed after 6 months. After successful PTA of the stenosis, a recurrence developed 10 months later which could not be sufficiently dilated and was therefore stented. Because of significant intimal hyperplasia, redilatation of the stented segment was necessary another 10 months later. The second patient needed stenting of two short segments in the subclavian and axillary vein 14 months after stenting a proximal graft stenosis in the basilic vein (Fig. 6). Nine months later, the nonstented intermediate segment of the axillary vein also had to be stented because of a new highgrade, nondilatable stenosis. The patient now has a continuous set of stents from the graft anastomosis in the basilic vein to the junction of the



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**Fig. 5.** A 74-year-old female patient with Cimino shunt for hemodialysis. PTA had been performed twice previously for stenosis of the venous outflow tract. **A** Highgrade stenosis at the venous outflow in the forearm. **B** After placement of an 8 mm wallstent, there is only a slight residual stenosis. **C** There is a second recurrence 13 months after stenting. **D** Widely patent lumen after repeat PTA with 8 mm balloon catheter. Patient has been free of symptoms for 5 months.

subclavian and internal jugular vein. The stented venous outflow has thereby been kept functioning for a total of 3 years so far.

In summary, all patients with peripheral stents in the region of the basilic or cephalic vein developed recurrent stenoses by intimal hyperplasia at various intervals from 4 to 9 months after stenting.

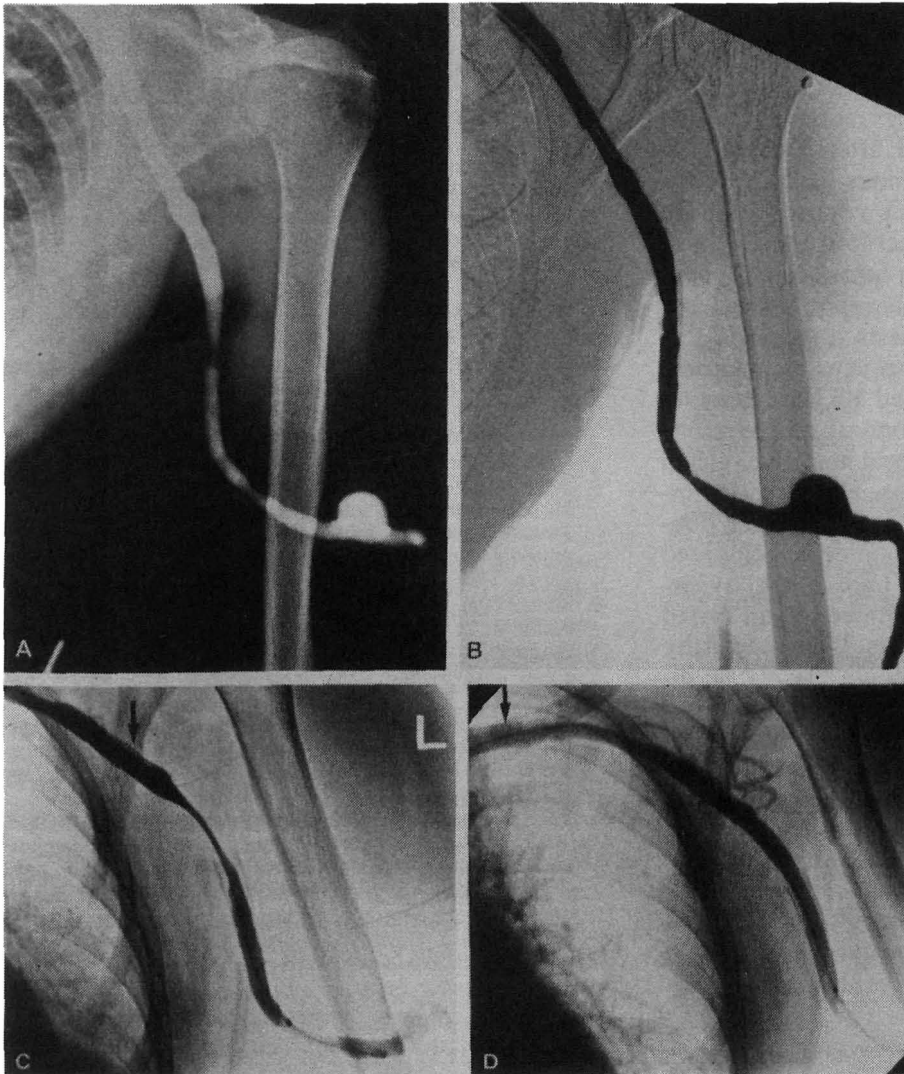
### Complications

In the whole group of 18 patients there were three acute occlusions, two of which were successfully treated percutaneously by fibrinolysis and thrombus aspiration. Otherwise we did not experience any serious complications, particularly no signs of stent migration or infection.

### Discussion

Most frequently, percutaneous angioplasty in the venous system is applied for hemodialysis-related stenoses. These are found in the venous outflow tract of AV-dialysis fistulas or in the region of the brachiocephalic veins [2, 10, 11]. So far, there are only a few reports on percutaneous treatment of other benign or tumor-induced venous stenoses [12, 13]. The use of flexible endovascular prostheses opens up a new alternative to surgical treatment of venous obstructions.

Persisting tumor obstructions in spite of chemotherapy or radiation therapy may now be palliated successfully with endoluminal stents. The symptoms of SVC obstruction that occur in about 3–4% of patients treated for bronchial carcinoma can be rapidly palliated [9]. Our own experience shows that in obstructions of the SVC, even in the event of a later occlusion by tumor or thrombosis, bridging of the acute obstruction gains time for the development of collaterals. Endoluminal vascular prostheses are also suitable for treating tumor compression or fibrotic stenoses after radiation therapy in the region of the pelvic veins and the IVC. If an occluded segment can be recanalized with the guidewire, there is a good chance that the occlusion may be kept open with a stent. If necessary, fibrinolytic agents can be used to reopen an occluded caval segment as suggested by Rösch's article in this is-



**Fig. 6.** A 34-year-old female on hemodialysis with stenosis at the proximal anastomosis of a xenograft to the basilic vein. This stenosis had been dilated four times prior to stenting. **A** Recurrent stenosis 4 months after first Wallstent at the site of the previous anastomotic stricture between the graft and the basilic vein. There is an incidental graft aneurysm. **B** After dilatation with an 8 mm Gruntzig balloon catheter, the lumen is well restored. **C** The sixth recurrence 18 months after stenting. Note that the stenosis is now mainly at the distal end of the stent. An additional stent in the axillary vein (arrow) had to be placed 2 months earlier for a new proximal lesion. **D** After a second stent in the basilic vein there is now good patency. Also note a third stent placed in the subclavian vein 2 months earlier for a second new proximal lesion (arrow).

sue. However, the limitations of the technique were shown in one of our patients where a tight tumor stenosis of the cephalic-axillary region could not be sufficiently improved with stenting. Stent placement should never preclude a potentially necessary surgical procedure.

Stenoses after surgical venous reconstructions, surgical venous anastomoses, or treatment of a venous spur are other important indications for stent implantation in the venous system. Our 2 cases with long-term follow-up of now approximately 4 and 5 years show that late complications (i.e., symptomatic recurrent stenoses or occlusions of large caliber veins) are extremely unlikely. Therefore, we believe that patients with May-Thurner syndrome should primarily be treated by balloon dilatation combined with stent implantation instead of sur-

gery. Our preliminary results are also encouraging in the region of the common femoral vein, a critical region for surgical reconstruction. So far, bending of the hip joint seems not to have any adverse effects when using a flexible stent.

Apart from strictures of the AV-anastomosis itself, stenoses along the venous outflow tract are the most common cause for dialysis shunt dysfunction [2, 14]. As for Cimino shunts, the stenoses are commonly found in the draining vein; in the case of shunt grafts, in the region of the proximal anastomosis. Furthermore, stenoses of the brachiocephalic veins are bothersome complications in hemodialysis shunts [10, 15–17]. Various possible causes for these lesions are discussed, such as damage of the vascular wall by catheterization or central venous lines, turbulence from increased flow particularly in

the region of valves, and finally, abnormal function of platelets damaged by hemodialysis [17, 18]. Conventional balloon dilatation, even with high-pressure balloons, of all such venous lesions yields unsatisfactory long-term patency rates of 35–50% after 1 year and only 10–32% after 2 years [19]. Intimal hyperplasia and perivenous fibrosis are the main causes for these recurrent stenoses after angioplasty [3], and endoluminal prostheses have been used to improve the long-term results. However, our experience shows that in spite of mechanical support by the endoprosthesis, restenosis secondary to intimal hyperplasia remains a continuing problem. The need for repeated PTA or percutaneous arterectomy [20] as secondary interventions to preserve shunt function are almost the rule in case of peripheral venous lesions. However, the intervals between repeated PTA can usually be doubled or tripled after stent implantation as compared with PTA alone. The time span until intimal hyperplasia became manifest in the stented region varied from 4 to 10 months in our patients. It is noteworthy that these recurrent stenoses could usually be dilated easily and a better lumen was achieved when compared with the immediate poststenting result. Careful evaluation of the whole venous outflow tract is always necessary if clinical signs of reduced fistula function occur, as 2 of our patients illustrate. Proximal lesions at a great distance from the primary stent placement may develop. The central lesions in the region of the brachiocephalic vein seem less prone to restenosis than peripheral stenoses. However, new lesions adjacent to the stented segments may develop as well.

Though repeated interventions may be necessary, we believe that stenting of complicated shunt-related lesions in dialysis patients provides a significant therapeutic improvement as it prolongs shunt function without surgical intervention. Furthermore, the secondary interventions are usually successful and technically simple. Such stent procedures are indicated both for patients in a transplant program as well as for patients on lifelong hemodialysis. However, we use stents only for patients in whom angioplasty is primarily unsuccessful or for those requiring repeat angioplasty in decreasing intervals (less than 2 months). Endoprosthesis should not be used in patients whose AV-shunts have never functioned properly or whose available shunt segment for venous puncture after stent placement would be too short, as the stented segment can no longer be used for venous puncture.

#### References

1. Wilms G, Baert AL, Nevelsteen A, Suy R, Verbrugge H, Hauglustaine D, Michielsen P (1989) Balloon angioplasty of venous structures. *JBR-PTR* 72:273–277
2. Hunter DW, Castaneda-Zuniga WR, Coleman CC, Young AT, Salomonowitz E, Mercado S, Amplatz K (1984) Failing arteriovenous dialysis fistulas: Evaluation and treatment. *Radiology* 152:631–635
3. Zollikofer CL, Largiadèr I, Brühlmann WF, Uhlschmid GK, Marty AH (1988) Endovascular stenting of veins and grafts: Preliminary clinical experience. *Radiology* 167:707–712
4. Charnsangavej CH, Carrasco CH, Wallace S, Wright KC, Ogawa K, Richli W, Gianturco C (1985) Stenosis of vena cava: Preliminary assessment of treatment with expandable metallic stents. *Radiology* 161:295–298
5. Günther RW, Vorwerk D, Bohndorf K, Klose KC, Kistler D, Mann H, Sieberth HG, el-Din A (1989) Venous stenoses in dialysis shunts: Treatment with self-expanding metallic stents. *Radiology* 170:401–405
6. Moradian GP, Hunter DW, Castaneda F, Castaneda-Zuniga WR, Amplatz K (1989) Clinical experience with placement of Gianturco vascular stents in the venous system. 75th Annual Meeting of the Radiological Society of North America, Chicago
7. Antonucci F, Zollikofer CL, Salomonowitz E, Hugentobler M, Stuckmann G (1990) Stenotic veins, grafts and dialysis shunts: Treatment with self-expanding prostheses. Presented at the 76th Scientific Assembly and Meeting of the RSNA, Chicago
8. Rousseau H, Morfaux V, Joffre F, Tor That H, Goudable C, Partoli P, Yong MY (1989) Treatment of hemodialysis arteriovenous fistula stenosis by percutaneous implantation of a new intravascular stent. *Interventional Radiology* 4:161–169
9. Putman JS, Uchida BT, Antonovic R, Rösch J (1988) Superior vena cava syndrome associated with massive thrombosis: Treatment with expandable wire stents. *Radiology* 167:727–728
10. Vanherweghe JL, Cabolet P, Dhaene M, Goldman M, Stolar JC, Sabot JP, Waterlot Y, Marchal M (1986) Complications related to subclavian catheters for haemodialysis. *Am J Nephrol* 6:339–345
11. Schwab SJ, Quarles LD, Middleton JP, Cohan RH, Saeed M, Dennis VW (1988) Haemodialysis-associated subclavian vein stenosis. *Kidney Int* 33:1156–1159
12. Werner WR, Sievers KW, Serdarevic M (1988) Ueber eine perkutane transluminale Angioplastie (PTA) der Vena brachiocephalica bei Sarkoidose mit oberer Einflusstauung. *Radiologe* 28:429–432
13. Capek P, Cope C (1989) Percutaneous treatment of superior vena cava syndrome. *AJR* 152:183–184
14. Glanz S, Gordon D, Butt KMH, Hong J, Adamson R, Sclafani SJA (1984) Dialysis access fistulas: Treatment of stenoses by transluminal angioplasty. *Radiology* 152:637–642
15. Glanz S, Gordon DH, Lipkowitz GS, Butt KMH, Hong J, Sclafani SJ (1988) Axillary and subclavian vein stenosis: Percutaneous angioplasty. *Radiology* 168:371–373
16. Ingram TL, Reid SH, Tisnado J, Cho SR, Posner MP (1988) Percutaneous transluminal angioplasty of brachiocephalic vein stenoses in patients with dialysis shunts. *Radiology* 166:45–47
17. Barrett N, Spencer S, McIvor J, Brown EA (1988) Subclavian stenosis: A major complication of subclavian dialysis catheters. *Nephrol Dial Transplant* 3:423–425
18. Gaux JC, Bourgelot P (1990) Percutaneous transluminal angioplasty of arteriovenous shunts. In: Dondelinger RF, Rossi P, Kurdziel JC, Wallace S (eds) *Interventional Radiology*. Stuttgart, Georg Thieme, pp 662–669
19. Glanz S (1991) Dialysis fistula stenosis. Treatment by angioplasty. In: Kadir S (ed) *Current practice of interventional radiology*. B.C. Decker Inc, Philadelphia, pp 280–287
20. Vorwerk D, Guenther R (1990) Removal of intimal hyperplasia in vascular endoprosthesis by atherectomy and balloon dilatation. *AJR* 154:617–619

## Stents in the Venous Vessel System

CH. L. ZOLLIKOFER, M.D.<sup>1</sup>

**M**alignant as well as benign venous obstructions are usually refractory to balloon angioplasty alone. Therefore percutaneous stent placement with selfexpanding stents like the Gianturco and modified Roesch-Z-type or Wallstents have been advocated as a low risk procedure to treat such lesions.

Mediastinal or retroperitoneal tumor involvement and post irradiation fibrosis are the most common causes of the superior and inferior vena cava syndrome. Stent placement results in rapid relief of the symptoms in 70 to 100 %. Severe complications are rare. Early thrombotic stent occlusion (5 to 15 %) can be treated with fibrinolysis and possible stent migration seen with Gianturco-stents remains generally without sequelae and can be managed by placing additional stents. Tumor ingrowth into the stent has not been a major problem.

Benign strictures in the venous system occur most frequently in the outflow tract of hemodialysis arteriovenous fistulae. Stenting of these lesions may significantly prolong Shunt patency, however high recurrence rates of 50 % and more are seen. But the secondary patency rates are significantly better when compared to PTA as a stand-alone procedure (one year 84 % versus 38 %).

Other causes for benign obstruction of the vena cava and large veins of the pelvis, thigh and brachycephalic region are mediastinal and retroperitoneal fibrosis, post-thrombotic occlusion, pelvic venous spur (May-Thurner-syndrome), previous surgery, trauma and central catheter placement. So far, no larger series have been published but the experience today shows, that rapid relief of symptoms can be expected in 90 to 100 % with excellent long term patency in the majority of patients.

In conclusion stenting offers a highly successful and low risk mode of treatment for benign and malignant venous obstruction.

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**IV INTERNATIONAL COURSE ON VASCULAR AND INTERVENTIONAL RADIOLOGY AS THERAPEUTIC ALTERNATIVE**

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