

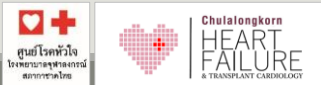
30 July 2016

Right Heart Catheterization

เอกราช อริยะชัยพาณิชย์

Heart Failure and Transplant Cardiology

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Disclosure

- Speaker, CME service:
Merck, Otsuka, Servier
- Consultant, non-CME service:
Novartis, Menarini



Agenda: Right heart cath

- How to perform
- Measuring pressure and CO
- Common calculations
- Common mistakes and pitfalls
- ~~PA guided therapy~~



Cardiac Catheterization: The use of a catheter(tube) into the heart.

Right heart catheterization

1. Measure "hemodynamics"
 - Pressure
 - Cardiac outputs
 - Resistant
2. Shunt study (step up O2Sat)
3. Vasoreactivity test
4. Others
 - Drug / treatment delivery
 - Trans-septal approach
 - Hepatic wedge
 - Comprehensive RHC+LHC



Contemporary Reviews in Cardiovascular Medicine

Hemodynamics in the Cardiac Catheterization Laboratory of the 21st Century

Rick A. Nishimura, MD; Blase A. Carabello, MD

There has been a striking evolution in the role of the cardiac catheterization laboratory over the past decades.¹ In the 1950s and 1960s, hemodynamic assessment in the cardiac catheterization laboratory was essential for understanding the physiology and pathophysiology of patients with cardiovascular diseases. With the development of surgical interventions to treat patients with valvular and congenital heart disease, it became necessary for the cardiac catheterization laboratory to provide an accurate hemodynamic assessment, laying out a therapeutic road map. Nearly all patients who had open heart surgery underwent a complete hemodynamic catheterization before surgery.

In the 1980s and 1990s, the evolution of 2-dimensional echocardiography and Doppler echocardiography provided an alternative noninvasive approach for the assessment of both cardiac anatomy and hemodynamics in patients with structural heart disease.² By measuring blood flow velocities noninvasively, Doppler echocardiography was able to provide information on volumetric flow, intracardiac pressures, pressure gradients, and valve areas, as well as diastolic filling of the heart. Furthermore, noninvasive studies could be repeated easily, allowing the practitioner to follow the progress of his/her patient's condition longitudinally. At the same time, there was growing emphasis on coronary angiography for defining epicardial coronary disease with the subsequent development of interventional approaches for coronary disease with catheter-based therapies. As the major focus in the catheterization laboratory shifted to the diagnosis and treatment of the patient with acute and chronic coronary artery

disease, hemodynamic evaluation has become of great importance in the evaluation of the patient with congenital heart disease.³ In addition, the noninvasive hemodynamic evaluation has inherent limitations, now recognized by clinicians who take care of the increasing number of patients who present with complex cardiovascular problems. The catheterization laboratory in the current era has become the place to solve the difficult diagnostic challenges that arise in patients with structural heart disease when answers are not apparent through the clinical examination and noninvasive testing.

Implications of the New Cardiac Catheterization Laboratory in the 21st Century

The changes that have occurred in patient evaluation throughout the last 2 decades have important implications for the new cardiac catheterization laboratory. Patients now coming for hemodynamic assessment have already had a thorough noninvasive evaluation. Thus, the remaining questions are complex and pose difficult diagnostic dilemmas. It is **no longer possible for the patient to leave an invasive hemodynamic assessment without a definitive answer about his/her condition. Thus, hemodynamic assessment in the cardiac catheterization now requires meticulous attention to detail. There is no longer such a procedure as routine cardiac catheterization.** The operator should be constantly evaluating the accrued data, ready to perform additional diagnostic interventions if necessary such as exercise or other provocative maneuvers.

Circulation. 2012;125:2138-2150

Right heart cath : Indication

- DDx types of shock
 - DDx type of pulmonary edema
 - Dx PH
 - Dx PAH, evaluate response to CCB
 - Dx L → R shunt
 - Hemodynamic tailored therapy in HF
 - Prognosticate severe HF and transplant candidacy
- No benefit shown in RCTs and should not be routinely use



Multiple studies confirmed no benefit (survival or days in hospital) from PACs in any medical or surgical population.

Table 1 Studies of the benefits and risks of PAC

| Study, date | Type of study | Patients | Benefits | Risks |
|---|---------------|----------|----------|---------------------------------|
| Acute coronary syndrome | | | | |
| Gore, 1987 ⁴ | Observ | 3623 | None | Increased mortality |
| Zion, 1990 ⁵ | Observ | 5481 | None | Increased mortality |
| Cohen, 2004 ⁷ | Observ | 26 437 | None | Increased mortality |
| Critically ill ICU patients | | | | |
| Connors, 1996 ⁸ | Observ | 5735 | None | Increased mortality |
| Rhodes, 2002 ¹⁵ | RCT | 201 | None | None |
| Patients undergoing major noncardiac surgery | | | | |
| Polanczyk, 2001 ¹³ | Observ | 4059 | None | Increased cardiac complications |
| Sandham, 2003 ¹⁶ | RCT | 1994 | None | Increased PE |
| Refractory congestive heart failure | | | | |
| Shah, 2004 ¹⁹ | RCT | 433 | None | None |
| ARDS/shock | | | | |
| Richard, 2003 ¹⁷ | RCT | 676 | None | None |

Observ = observational trial; RCT = randomized clinical trial; PE = pulmonary embolism.

Am J Med 2005 118, 449



Planning

Pre-procedure

- Indication, contraindication
- Consent

Procedure

- Technique
 - Position/ Site
 - Equipment
 - Imaging guide
 - Anesthesiology
- Vascular access (+/- ultrasound guide)
 Right IJ, left SubCl, Fem, brachial
 Swann-Ganz cath, MPA + 0.025 wire + wedge cath (Berman)
 Fluoroscopy, echo
 Local

Post-procedure

- Care
- Complication



PA cath placement

Femoral approach

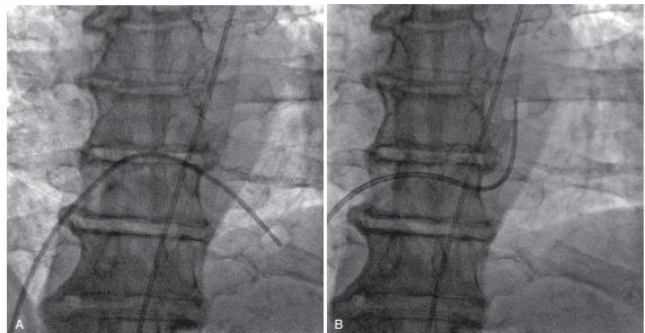
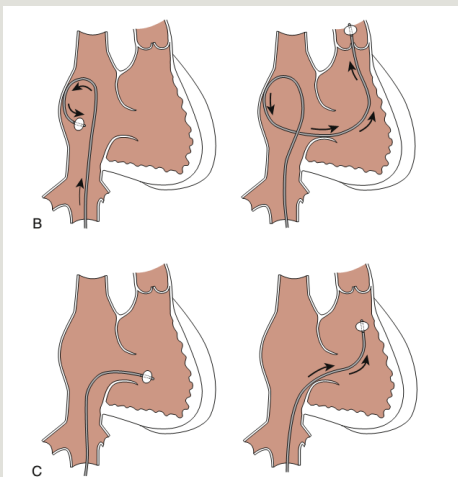


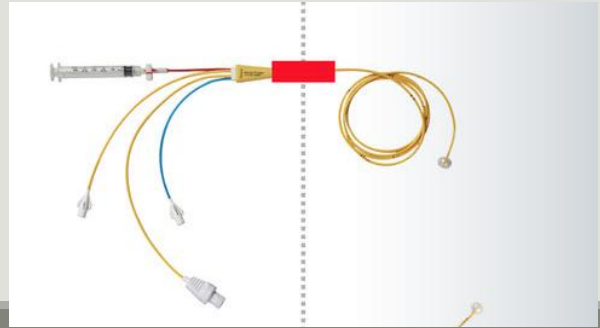
FIGURE 4-2. With the balloon inflated, the Swann-Ganz catheter is advanced across the tricuspid valve and positioned to the left of the spine in the outflow tract (A). The catheter should not be advanced to the apex of the right ventricle because this will make it more difficult to turn into the outflow tract. From this position, the catheter is aggressively turned in a clockwise manner with the balloon inflated until the tip points up and into the pulmonary outflow tract (B).

The cardiac catheterization handbook / edited by Morton J. Kern. -- 5th ed 2011
 Cardiac catheterization : an atlas and DVD / Michael Ragosta. -- 1st ed. 2010



Right heart cath : PA Catheter or Swan Ganz Catheter

- A 120-cm long, multi-lumen, balloon-tipped catheter
- Usually 7.5 fr
- Connected to a pressure transducer and temperature sensor
- Fluid-filled catheter
- 1.5 ml air syringe
- 4-5 lumens



Transducer

Changing mechanical to electrical signal

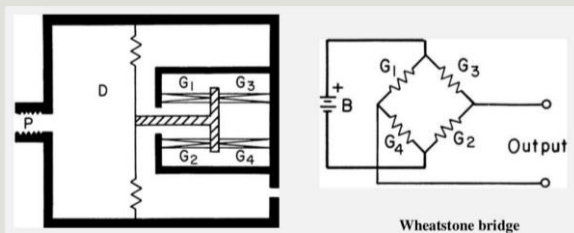
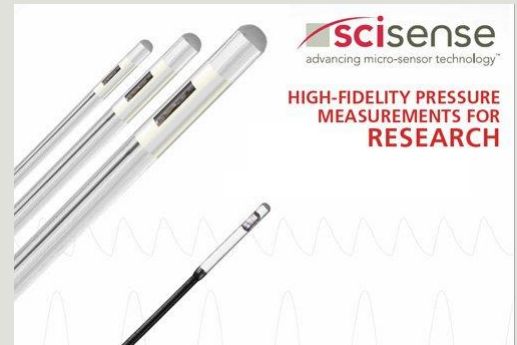


Figure 7.7 Schematic representation of a strain gauge pressure transducer. Pressure is transmitted through port P and acts on diaphragm D, which is vented to atmospheric pressure on its opposite side. Pressure causes the diaphragm to stretch, in turn stretching and therefore increasing the resistance of wires G_1 and G_2 , while having the opposite effect on wires G_3 and G_4 . The wires are electrically connected as shown in Fig. 7.8.



Micromanometer (Catheter-tip pressure manometer)

- High fidelity transducer catheter
- \uparrow frequency response, \downarrow artifact
- Research setting
- Measurement of myocardial mechanics (e.g. dP/dT)
- “Pressure wire”



Obtaining sloppy, poor-quality information or misinterpreting waveforms can lead to major errors in diagnosis and management.



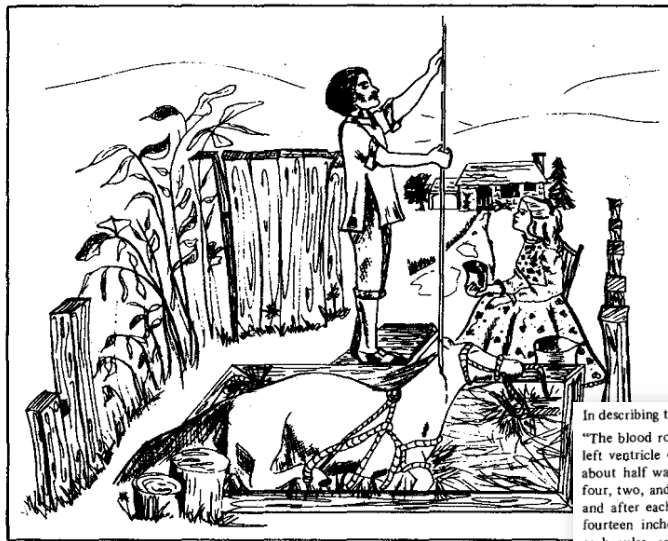


Figure 1. First Arterial Pressure Measurement

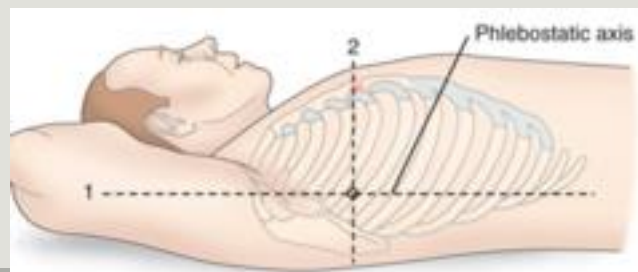
In describing this first experiment, Hales reported the following:

"The blood rose in the tube eight feet three inches perpendicular above the level of the left ventricle of the heart, but it did not attain to its full height at once; it rushed up about half way in an instant, and afterwards gradually at each pulse twelve, eight, six, four, two, and sometimes one inch. When it was at its full height, it would rise and fall at and after each pulse two, three, or four inches; and sometimes it would fall twelve or fourteen inches, and for a time have the same vibrations up and down at and after each pulse, as it had, when it was at its full height, to which it would rise again, after forty and fifty pulses.

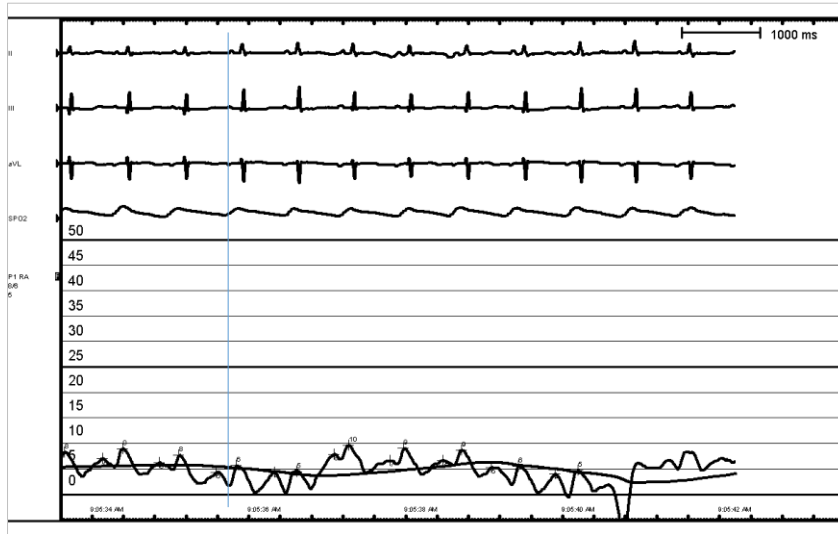


Zero and Level

- **Zero:** Open transducer to air and “zero”
 - Physiologic measurements are made relative to atmosphere
 - Make the transducer to read zero while exposed to the atmosphere
- **Level:** adjust transducer to “Phlebostasis axis”
 - Intracardiac measurements are referenced to mid chest position
 - Mid-chest from sternal angle (or Mid axillary line x 4th ICS)

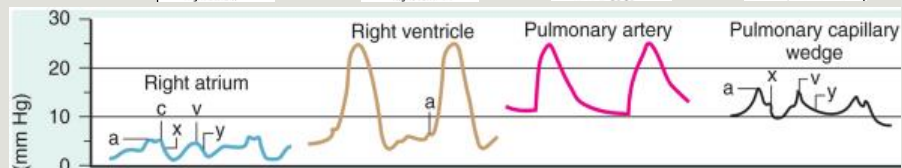
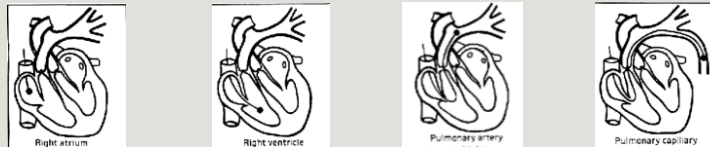


Time the wave with the ECG.



- Pressure increase if
- ↓ Chamber size
- ↑ Volume
- ↓ compliance

Pressure

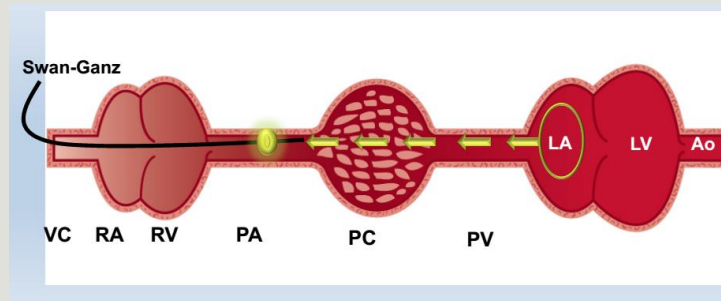


| RA (6 mmHg) | RV (24/6 mmHg) | PA (24/12 mmHg) | PCWP (12 mmHg) |
|---|---|--|--|
| <ul style="list-style-type: none"> • Venous waveform (2 up, 2 down per cardiac cycle) A: Atrial systole <ul style="list-style-type: none"> - Increased in RV infarct, PS, PE, Pul HTN - Giant Cannon a waves in A-V dissociation, 3°AV block, VT X: atrial relaxation <ul style="list-style-type: none"> - Increase in restrictive and constrictive disease - Decrease in severe tricuspid regurgitation C: bulging of the AV valve V: filling of atrium (atrial diastole) <ul style="list-style-type: none"> - large C-V waves in TR Y: emptying of the RA into RV <ul style="list-style-type: none"> - Increase in early restrictive, severe TR - Blunted in TS, RV infarct and frank tamponade. | <ul style="list-style-type: none"> • Rapid upstroke followed by a rapid downstroke. Rasing during diastole | <ul style="list-style-type: none"> • Rapid upstroke with dicrotic notch on down slope, down rending during diastole Pul Hypertension: mPA > 25 mmHg | <ul style="list-style-type: none"> • Venous waveform <ul style="list-style-type: none"> - confirm by O2sat > 95% - Surrogate of LV filling pressure |



PCWP

- Balloon inflation → obstructs blood flow
- End hold lumen connect with LA (surrogate of LV filling pressures)
- Verify waveform, fluoroscopy, and oximetry (>95%)



Flow

:Cardiac output measurement (indirect measure)

CO by thermodilution:

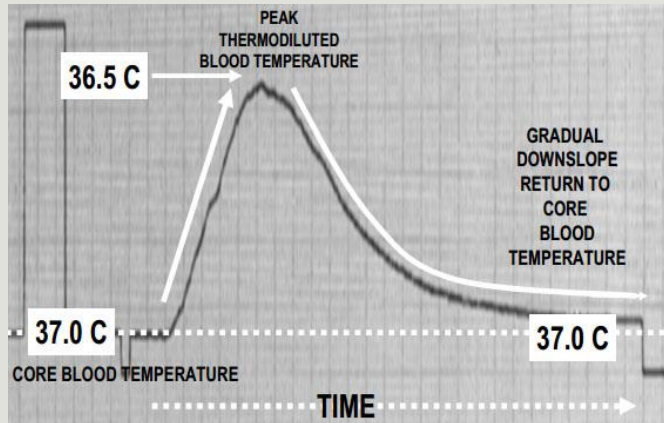
- Indicator dilution method
- Technique: Injecting 10 ml of known temp NS to a proximal port and measure Δ temp at distal port.
- Calculation = Reverse area/time under the curve
- Limit in TR, shunt, low CO, rhythm disturbances, incorrect constant number. (Crit Care Med 1993; 21:586)

CO by Fick: "Gold standard"

- Constant of mass.
 - Technique: Collect mixed venous and arterial blood to calculate O₂ content (O₂Sat, Hb)
 - Calculated = Product of O₂ contents and extraction.
- $$CO = \frac{(VO_2)}{10 \times 1.34Hb(SaO_2 - MvO_2Sat)}$$
- Limit in shunt
 - Most cath lab use assumed VO₂ → inaccurate assumption of VO₂ (circ 2014;129:203)



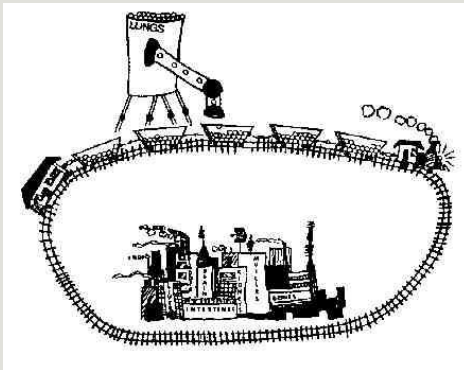
Flow : CO by thermodilution



Single entry
Known volume
No re-circulate
No contaminate
Correct constant number



Right heart cath : CO by Fick



CO is calculated as oxygen consumption divided by the arteriovenous oxygen concentration difference

$$CO = \frac{VO_2}{10 \times 1.34 \times Hb \times (SaO_2 - MvO_2Sat)}$$



Estimates of resting $\dot{V}O_2$ derived from conventional formulae are inaccurate, especially in severely obese individuals.

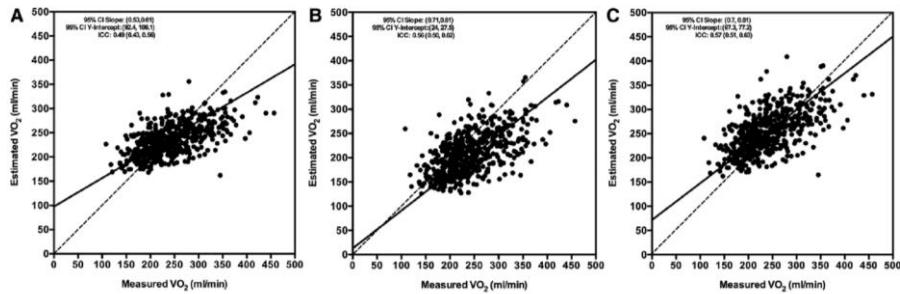


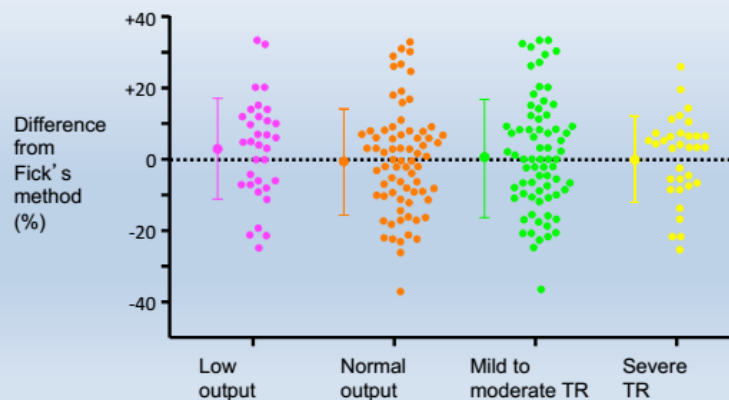
Figure 1. Comparative plot of measured $\dot{V}O_2$ vs. estimated $\dot{V}O_2$ for overall cohort using (A) Dehmer, (B) LaFarge, and (C) Bergstra formulae. Dashed line indicates line of equality; solid line, ordinary least products regression line. CI indicates confidence interval; ICC, intraclass correlation coefficient; and $\dot{V}O_2$, oxygen uptake.

Circulation. 2014;129:203-210.



Cardiac Output: Thermodilution vs. Fick

comparison of 105 CO by Fick + TD in 35 PH patients
 ****TD good with low CO and with Severe TR****



Hoeper MM et al. *Am J Respir Crit Care Med.* 1999;160:535-541.



Calculation

- SVR
- PVR
- TPG
- SV
- CI



Right heart cath : calculation

| Parameter and relations | Normal value and unit |
|---|----------------------------------|
| $V = I R$ | $\Delta BP = CO \times SVR$ |
| CO | = 5 L/min |
| BSA | = 2 m ² |
| $CI = \frac{CO}{BSA}$ | = 2.5 L/min/m ² |
| HR | = 70 bpm |
| $SV = \frac{CO}{HR}$ | = 70 ml/beat |
| $SVI = \frac{SV}{BSA}$ | = 35 ml/beat/m ² |
| $SVR = \frac{(MAP - CVP) \times 80}{CO}$ | = 1300 dynes.sec/cm ⁵ |
| $PVR = \frac{(mPA - PCWP)}{CO}$ | = 1 wood unit |
| TPG = mPA - PCWP | = 5 mmHg |
| Ao | = 120/80 mmHg |
| A O ₂ sat | = 95-100 % |
| Mixed V O ₂ sat | = 75 % |
| A - V O ₂ content difference | = 20 - 15 = 5 ml/dL |
| $LVSWI = SVI \times (MAP - PCWP) \times 0.0136$ | = 50 - 62 g/m ² /beat |
| $RVSWI = SVI \times (mPA - CVP) \times 0.0136$ | = 5-10 g/m ² /beat |



Right heart cath : Shunt study (O₂ step up)

- Diagnosis of L → R shunt
- Blood sample at many location

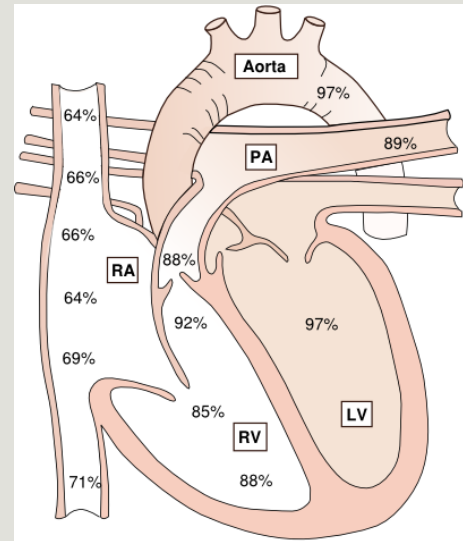
Table 3-7

Oxygen Saturation Values for Shunt Detection

| Level of Shunt | Significant Step-Up Difference* O ₂ % Saturation |
|---------------------------------|--|
| Atrial (SVC/IVC to right aorta) | ≥7 |
| Ventricular | ≥5 |
| Great vessel | ≥5 |

IVC, Inferior vena cava; PA, pulmonary artery pressure; SVC, superior vena cava.

*Difference between distal and proximal chamber. For example, for atrial septal defect: MVO₂ = (3 SVC + 1 IVC)/4 and difference from PA should be ≤7% normally.



The cardiac catheterization handbook / edited by Morton J. Kern. -- 5th ed 2011



CO measurement in patient with shunt

Shunt Calculation

- Shunt Calculation, $QP/QS = \frac{AO_{sat} - MV_{sat}}{PV_{sat} - PA_{sat}}$
(normal QP/QS = 1)
- Resting mix venous (MV), calculation = $\frac{(3SVC + 1IVC)}{4}$
- Some authors use SVC alone as Mix venous.

* Abraham Rudolph, *Congenital Diseases of the Heart, Second Edition*



Inaccurate measurement due to

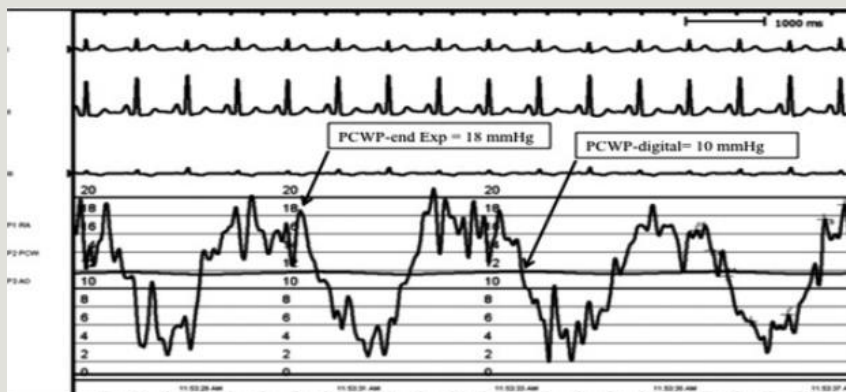
- Improper zero level reference
- Influence of respiratory pressure
 - End expiratory “Sunrise and Valley”
 - Do not use computer reading number
- Partially wedge
- Dampening / overdamp



Inaccurate measurement: Digital PCWP vs End-expiratory PCWP

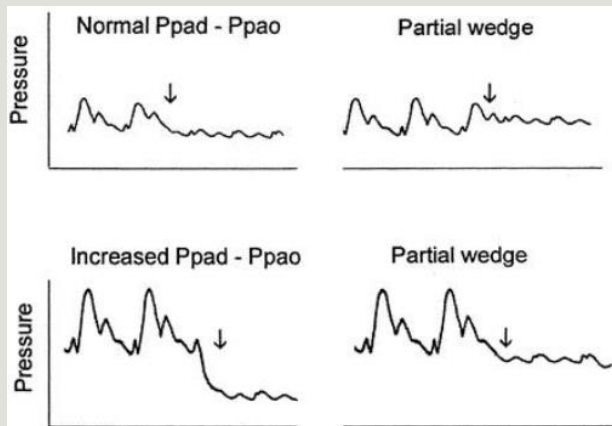
Prospective 61 PH patients

- mean bias of -4.4 mm Hg (95% limits of agreement of -11.3 to 2.5 mmHg)



Ryan JJ. Am Heart J. 2012;163(4):589-94.

Inaccurate measurement: :Partially wedge



When suspect

1. Check waveform
2. Check position
3. Check wedge O2Sat

Tonelli A, et al CHEST 2009;136(1):37-43



Important of PCWP in PH

- WHO Category 2 –PH due to heart heart disease
 - mPA ≥ 25 mmHg
 - PCWP > 15 mmHg

Table 3 Haemodynamic definitions of pulmonary hypertension^a

| Definition | Characteristics ^a | Clinical group(s) ^a |
|---|---|---|
| PH | PAPm ≥ 25 mmHg | All |
| Pre-capillary PH | PAPm ≥ 25 mmHg PAWP ≤ 15 mmHg | 1. Pulmonary arterial hypertension 3. PH due to lung diseases 4. Chronic thromboembolic PH 5. PH with unclear and/or multifactorial mechanisms |
| Post-capillary PH | PAPm ≥ 25 mmHg PAWP > 15 mmHg | 2. PH due to left heart disease 5. PH with unclear and/or multifactorial mechanisms |
| Isolated post-capillary PH (Ipc-PH) | DPG < 7 mmHg and/or PVR ≤ 3 WU ^b | |
| Combined post-capillary and pre-capillary PH (Cpc-PH) | DPG ≥ 7 mmHg and/or PVR > 3 WU ^b | |

2015 ESC/ERS Guidelines for PH



Table 8 Suggested Definitions for Pulmonary Hypertension Due to Left Heart Disease

| Nomenclature | Description | Physiologic definition | Hemodynamic criteria |
|-----------------|--|---|--|
| Passive PH | PH with elevated left cardiac filling pressure | Post-capillary (passive congestion) eg, pulmonary venous hypertension | Mean PAP \geq 25 mm Hg and PCW, LAP, LVEDP $>$ 15 mm Hg and TPG \leq 15 mm Hg or PVR \leq 3.0 WU |
| Mixed PH | PH with elevated left cardiac filling pressure and increased pulmonary vascular resistance | Pre- and post-capillary (passive congestion with excessive arterial vasoconstriction \pm vascular remodeling), eg, pulmonary arterial and venous hypertension | Mean PAP \geq 25 mm Hg and PCW, LAP, LVEDP $>$ 15 mm Hg and TPG $>$ 15 mm Hg or PVR $>$ 3.0 WU |
| Reactive PH | Component of mixed PH that is acutely or chronically responsive to pharmacologic (diuretics, vasodilators, inodilators) and/or mechanical circulatory support device therapies | With vasodilators and/or inodilators: TPG \leq 15 mm Hg or PVR \leq 3.0 WU | |
| Non-reactive PH | Component of mixed PH that is not responsive to above strategies | Despite vasodilators and/or inodilators: TPG $>$ 15 mm Hg or PVR $>$ 3.0 WU | |



Vasoreactivity Test

- To identification patient who is CCB “responders”
 - Mean PA fall \geq 10 mmHG and to \leq 40 mmHg
 - Unchanged or increased CO
- Most data from iPAH
- Only 5-10% of patient response
- Not recommend for gr 2, 3, 4, and 5
 - May be harmful and misleading



Table 7. Agents for Acute Vasodilator Testing

| | Epoprostenol | Adenosine | Nitric Oxide |
|-------------------------|-----------------------------------|-------------------------------|---|
| Route of Administration | Intravenous infusion | Intravenous infusion | Inhaled |
| Dose Titration | 2 ng/kg/min every 10 to 15 min | 50 mcg/kg/min every 2 min | None |
| Dose Range | 2 to 10 ng/kg/min | 50 to 250 mcg/kg/min | 10 to 80 ppm |
| Side Effects | Headache, nausea, lightheadedness | Dyspnea, chest pain, AV block | Increased left heart filling pressure in susceptible patients |

AV indicates atrioventricular.

Web Table IV Route of administration, half-life, dose ranges, increments, and duration of administration of the most commonly used agents for pulmonary vasoreactivity tests

| Drug | Route | Half-life | Dose range ^d | Increments ^e | Duration ^f | Class ^a | Level ^b | Ref ^c |
|--------------|-------|-----------|-------------------------|-------------------------|-----------------------|--------------------|--------------------|------------------|
| Nitric oxide | Inh | 15–30 sec | 10–20 ppm | - | 5 min ^g | I | C | 4, 5 |
| Epoprostenol | i.v. | 3 min | 2–12 ng/kg/min | 2 ng/kg/min | 10 min | I | C | 4, 6 |
| Adenosine | i.v. | 5–10 sec | 50–350 µg/kg/min | 50 µg/kg/min | 2 min | IIa | C | 7 |
| Iloprost | Inh | 30 min | 5–20 µg | - | 15 min | IIb | C | 8 |

2009 ACC/AHA Guidelines for PH
2015 ESC/ERS Guidelines for PH



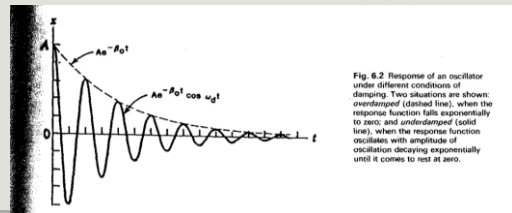
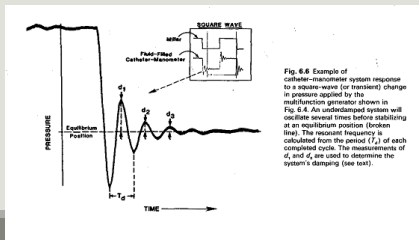
Inaccurate measurement due to

- Improper zero level reference
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- Dampening



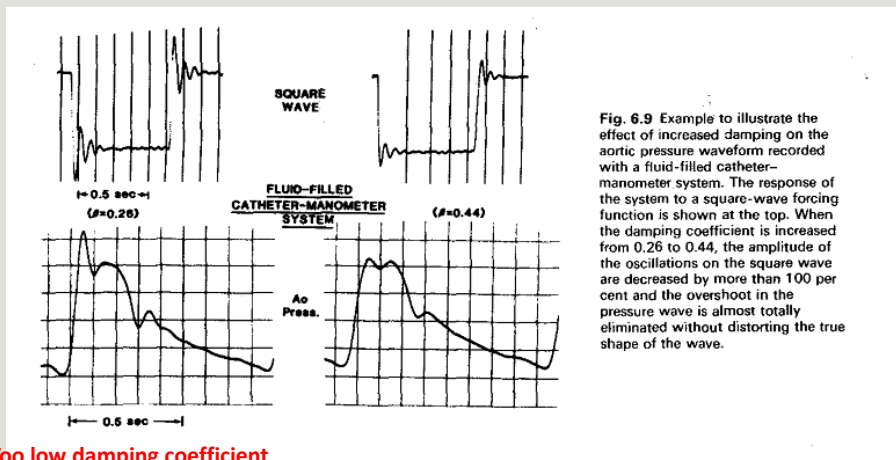
Dynamic frequency response

- Specific property of each fluid filled system
 - A pressure change at the end of a catheter will cause a system to oscillate at its natural frequency and will decay accordance with the damping coefficient
 - Depend upon radius, length, fluid density, viscosity
- Critically damp
 - It would be ideal if the pressure variations at the catheter tip were exactly reproduced into transducers.



Measuring principle of arterial wave. McDonalds

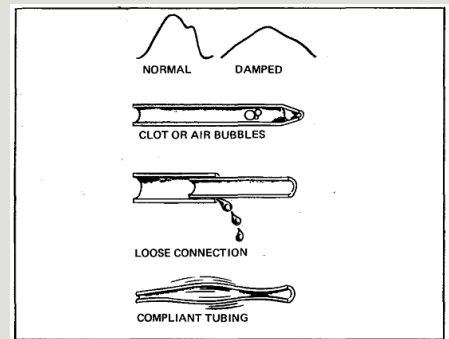
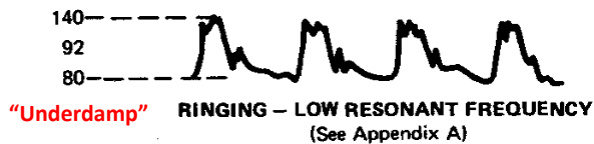
Effect of damping coefficient



Too low damping coefficient
"Underdamp"
Cause resonate

Practical "Damping"

MONITOR



To low reading "Damping"

- Thrombus formation
- Air bubbles
- Blood
- Loose connection
- Too small tube
- Against vessel wall
- Kinking



Right heart cath : Complication

- Vascular access
 - Bleeding, pneumothorax, hemothorax, air embolism
- Arrhythmia
 - PVC, VT (3%)
 - RBBB (3rd degree AV block in preexisting LBBB) (5%)
- Knotting
- Balloon
 - PA rupture (Over wedge)
 - Pulmonary infarct
- Wrong data is worse than no data.

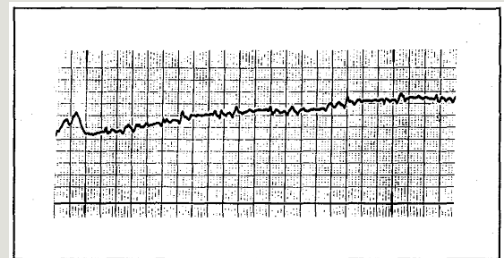


Figure 35. Progressive Elevation of Wedge Waveform Indicating Balloon Overinflation

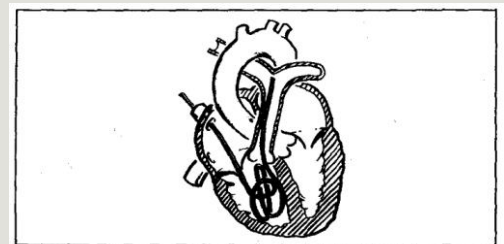
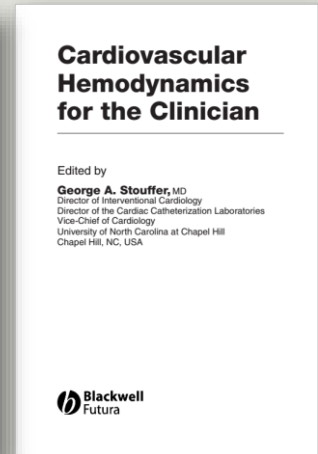
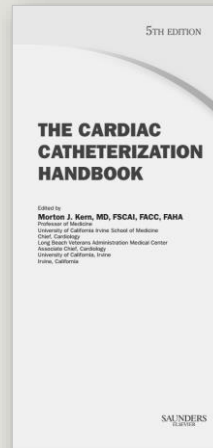
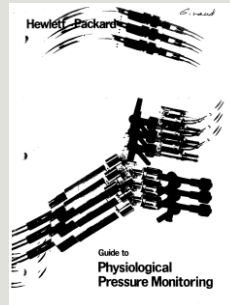


Figure 36. Kinking in Right Ventricle



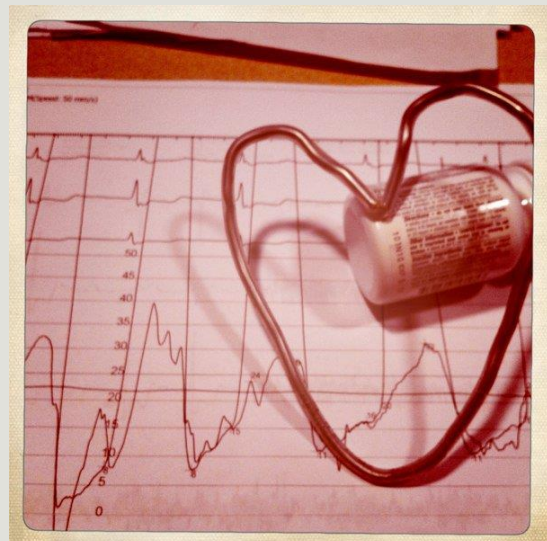
Recommend readings

- Kern's hand book
- CV hemodynamics for clinician
- McDonalds
- Uptodate



Thank you

aeakarach.a@chula.ac.th



Back up slide



Whipping artifact

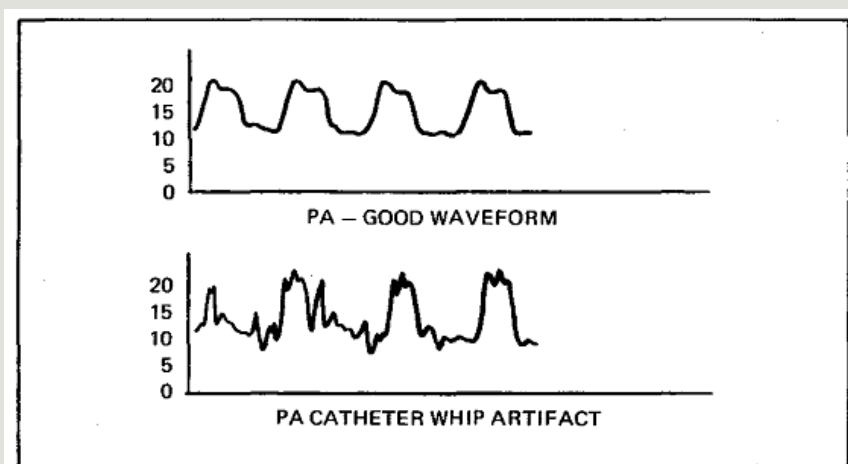
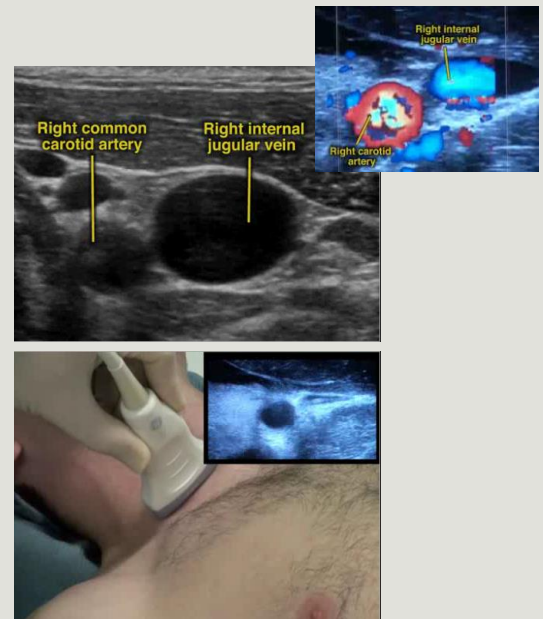


Figure 38. Artifact Generated by Catheter Motion



Ultrasound guide

- Linear vascular probe
- Place in a sterile sheath
- Vein = Thin wall, compressible, continuous color
- Needle is “bright” – echogenic
- Look at the tip of the needle
- Learning curve
 - ↑ success, ↓ complication
 - ↓ procedure time



RVSP from Doppler echocardiogram

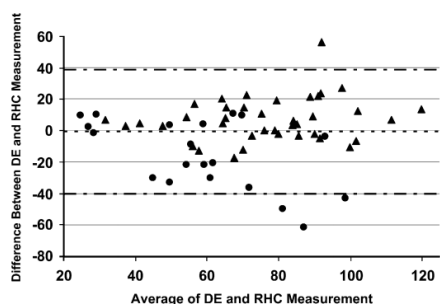


Figure 1. Bland-Altman plot of Doppler echocardiographic estimates of pulmonary artery pressure and right-heart catheterization measurements. The bias was -0.6 mm Hg and the 95% limits of agreement were $+38.8$ and -40.0 mm Hg. *Triangles* represent excellent- and good-quality Doppler signal; *circles* = fair- and poor-quality Doppler signal; *dotted line* = bias; *dash/dotted line* = upper and lower limits of agreement. Abbreviations: DE = Doppler echocardiography; PASP = pulmonary artery systolic pressure; RHC = right-heart catheterization.

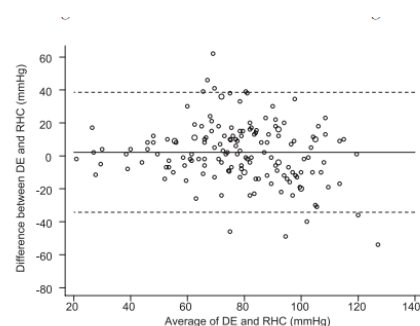
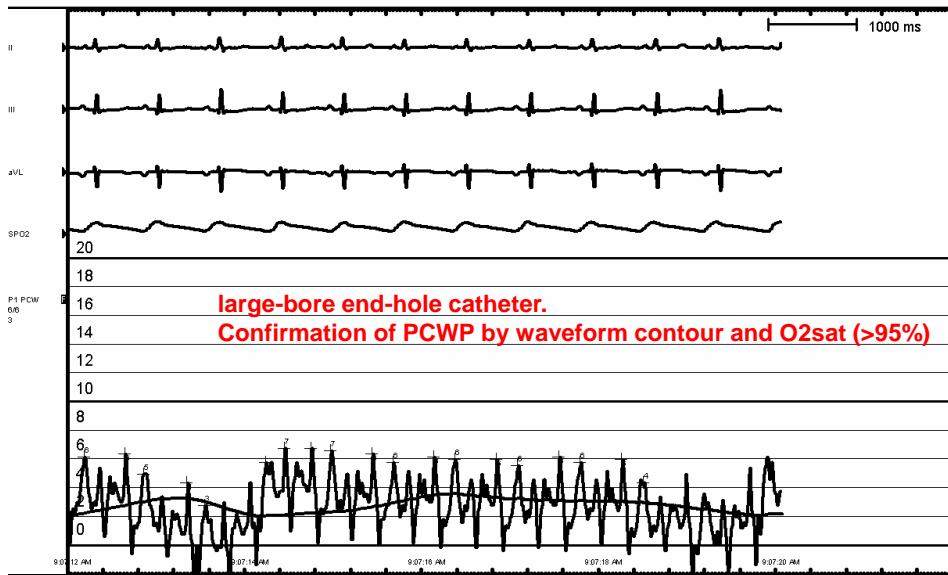


FIGURE 1. Bland-Altman analysis demonstrating a lack of agreement between DE estimates of pulmonary artery systolic pressure (PASP) and PASP determined during RHC (solid line), as highlighted by the 95% limits of agreement, ranging from -34.2 mm Hg to 38.6 mm Hg (dashed lines). Larger circles represent identical observations among multiple patients. The inaccuracy of DE estimates of PASP is particularly apparent at higher PASP. DE = Doppler echocardiography; RHC = right-sided heart catheterization.

Am J Respir Crit Care Med Vol 179. pp 615–621, 2009
CHEST 2011; 139(5):988–993

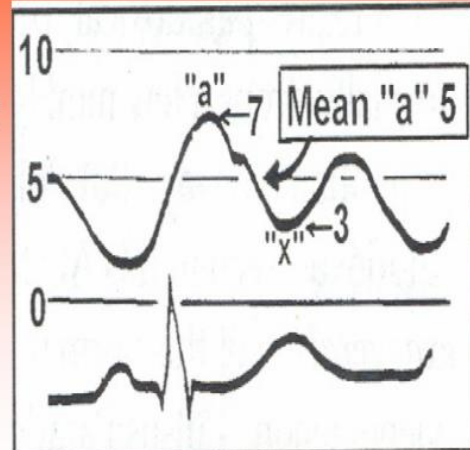


Choosing vascular approach : Depend upon expertise, anatomy, risk

| Location | Advantage | Disadvantage |
|------------------|---|---|
| Internal Jugular | Easy to control bleeding Less pneumothorax Straight shot to RA Compressible Excellent US target | Difficult in large neck, intubate. Poor landmark Carotid a inj Difficult dressing |
| Subclavian | Most comfortable for pt. Easy dressing Less DVT, less arterial inj, less infection Bony landmarks in obesity | Higher pneumothorax Cannot compress malposition No not do it lung, coagulopathy |
| Femoral | Fast, easy, high success rate Not interfere with intubation, CPR No pneumothorax Compressible No need for trendelenburg | Dirty / infect High rate of arterial inj High rate of DVT Pt cannot mobile Cannot monitor CVP |

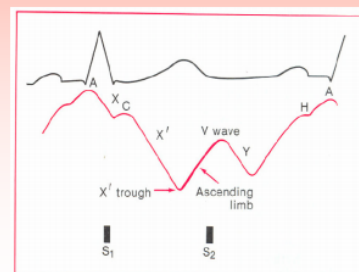
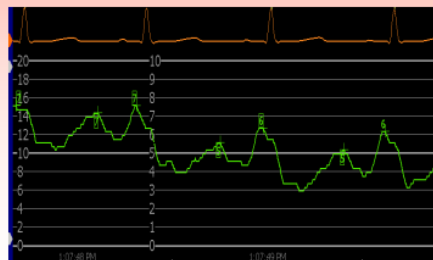
Getting the mean Atrial pressure

- Mean atrial pressure also known as mean 'a' wave.
- Equal to ventricular end diastolic pressure.
- Half way between 'a' and "x"



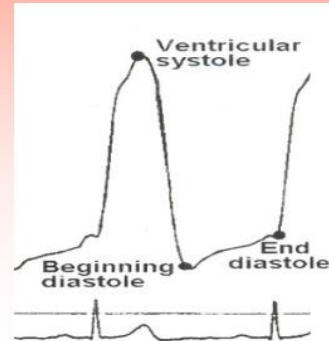
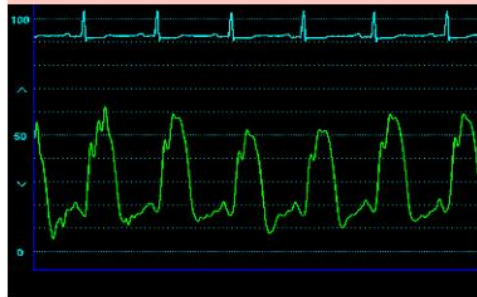
Atrial Pressure Waveform

- RA, CVP, LA and PCW, known as sine wave.
- To be reported as A,V, and Mean.
- Normal Mean RA = 2- 6 mmHg.



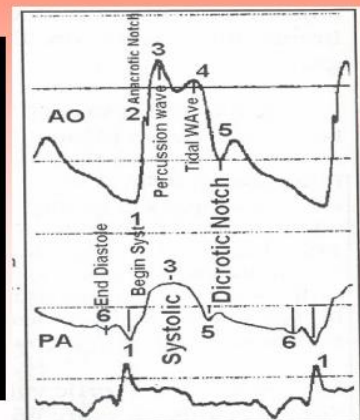
Ventricular Pressure Waveform

- Known as square or rectangular wave.
- To be read as Systole and EDP,
- **no mean pressure.**



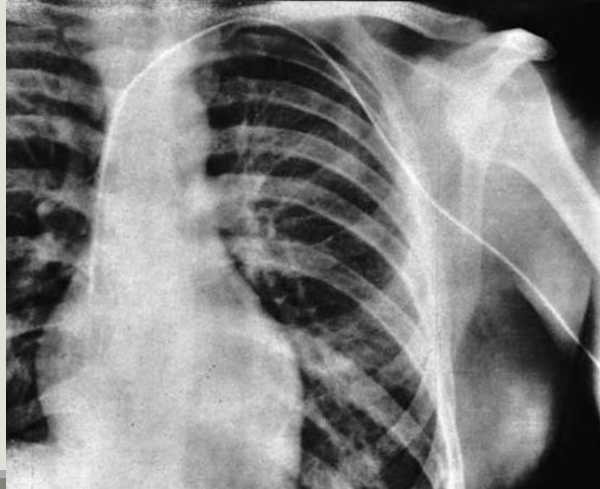
Arterial Pressure Waveform.

- AO and PA, triangular in shape.
- To be read as systolic, diastolic and mean.



Nobel prize in medicine

Werner Forssmann - First central line 1929, at that time a surgical Intern



Pop test

