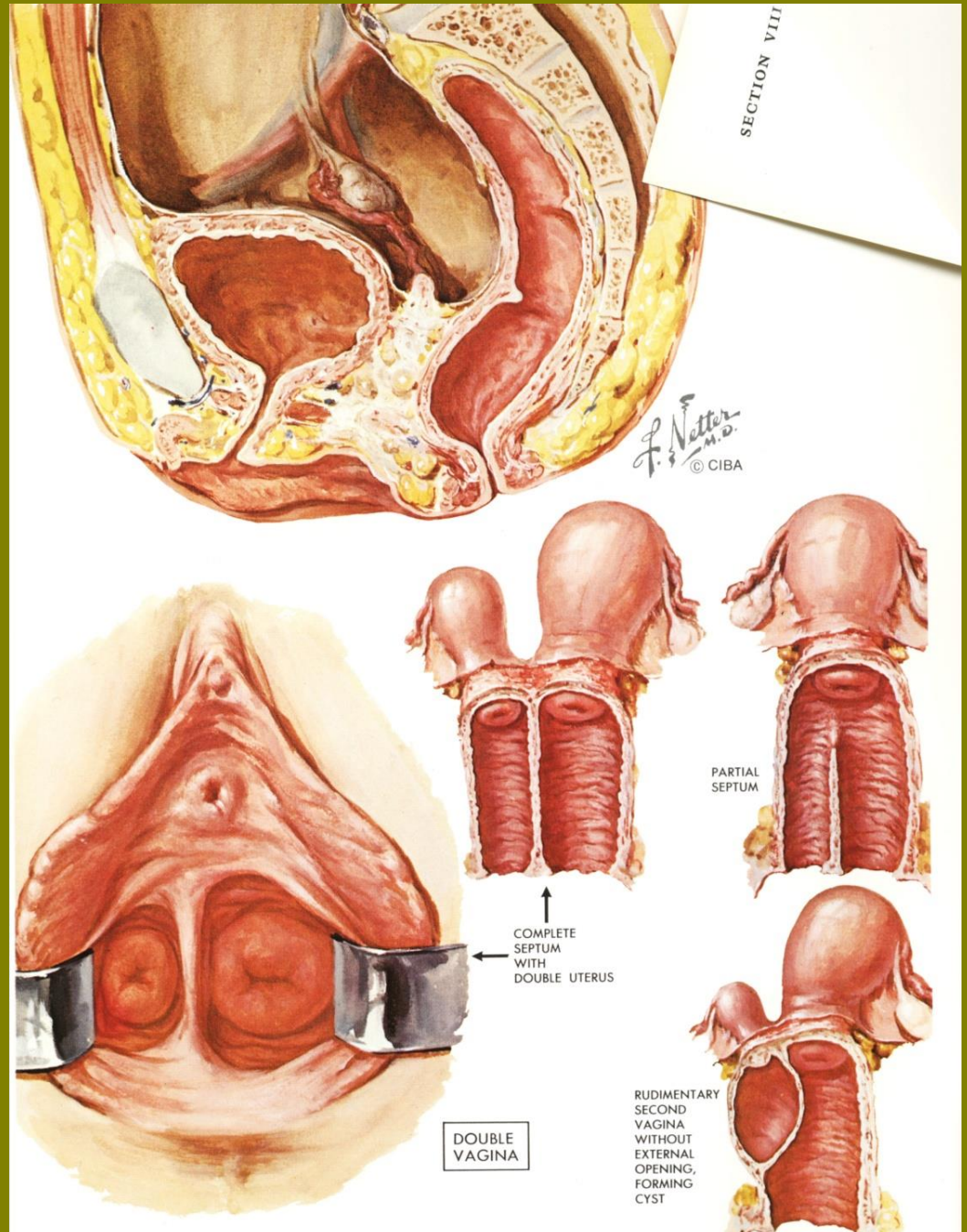


LECTURE 20-21



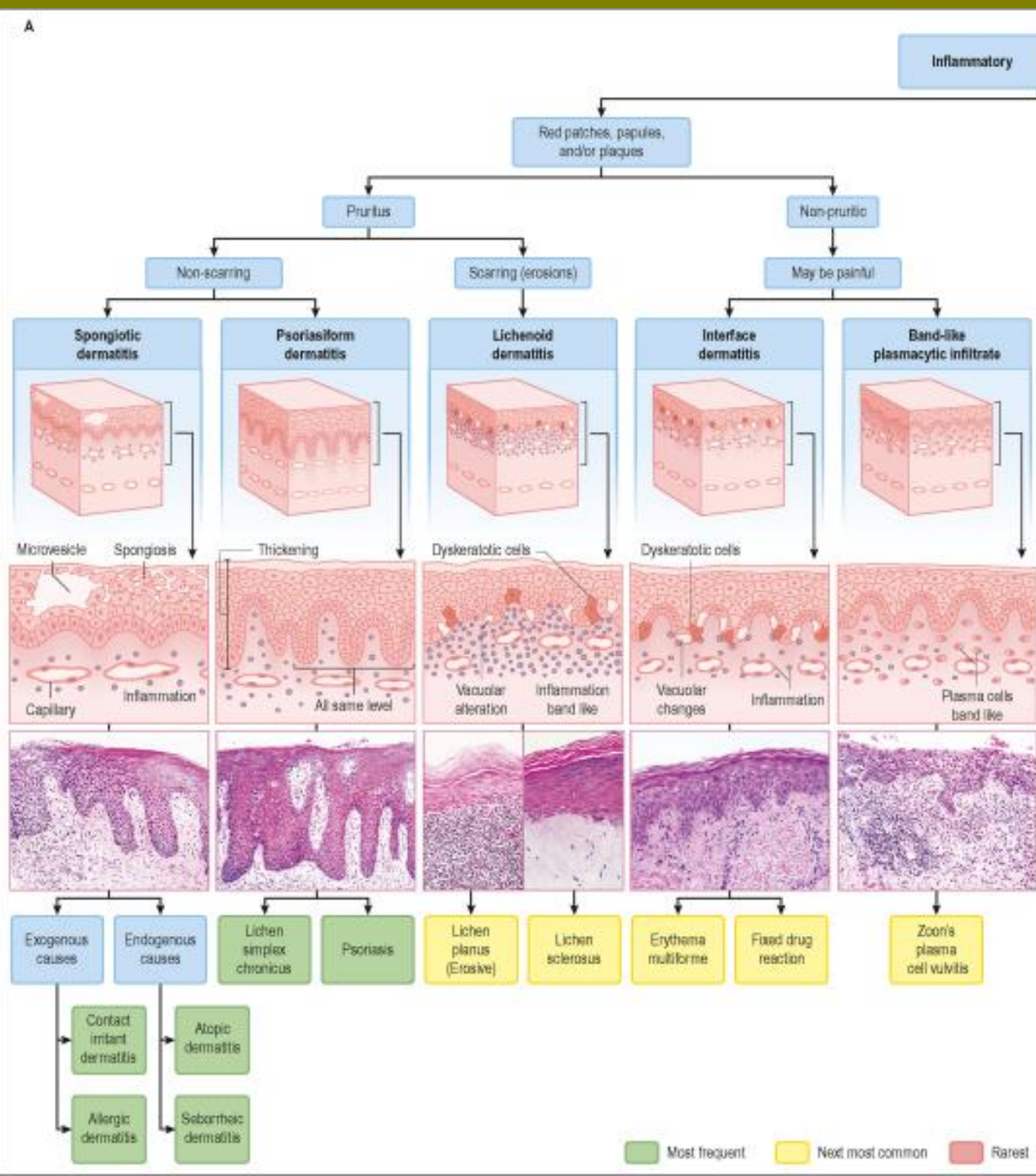
DEVELOPMENTAL DISTURBANCES

**UTERINE AND VAGINAL
AGENESIA**
**DUPLICATED UTERUS AND
VAGINA**

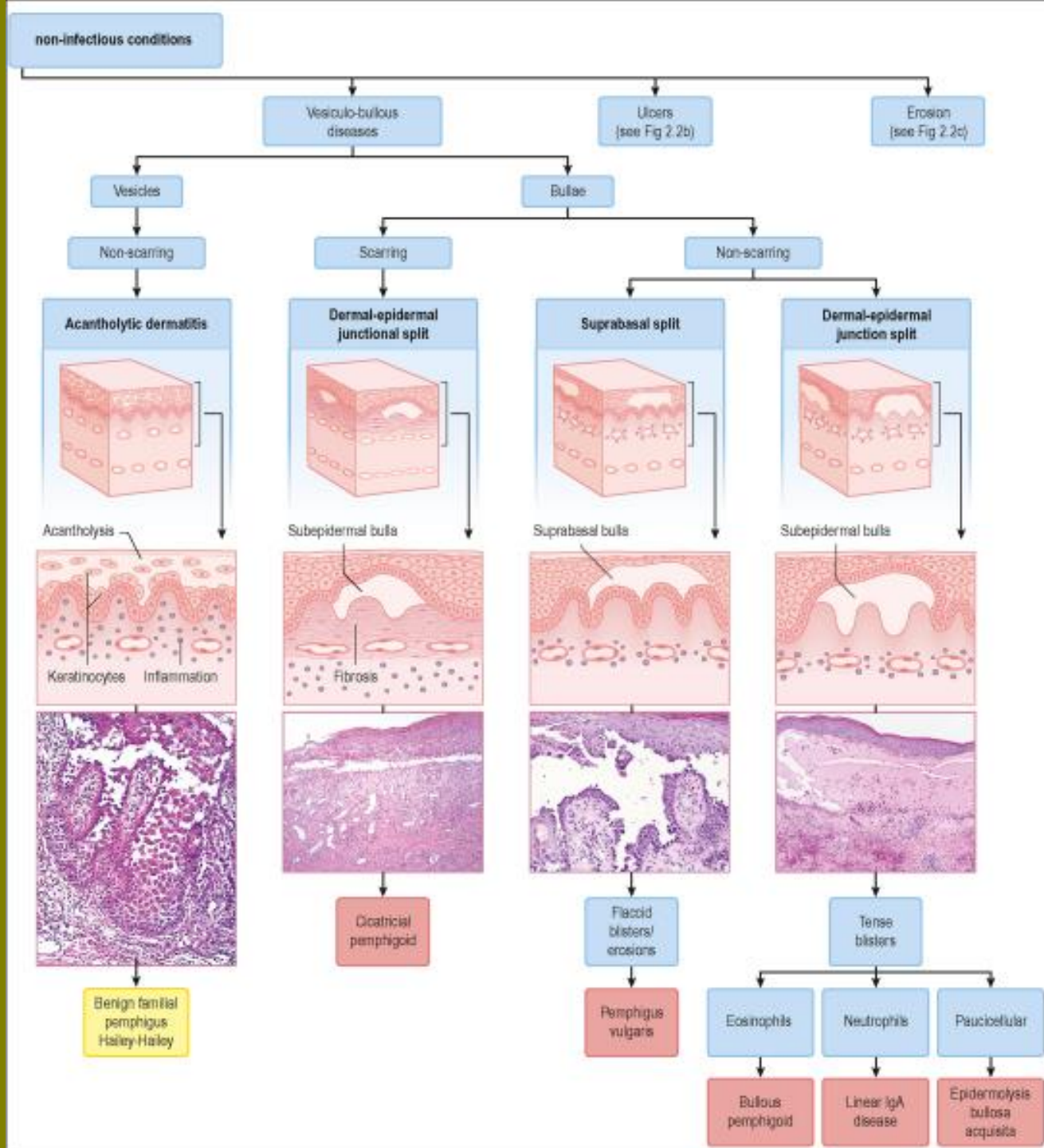


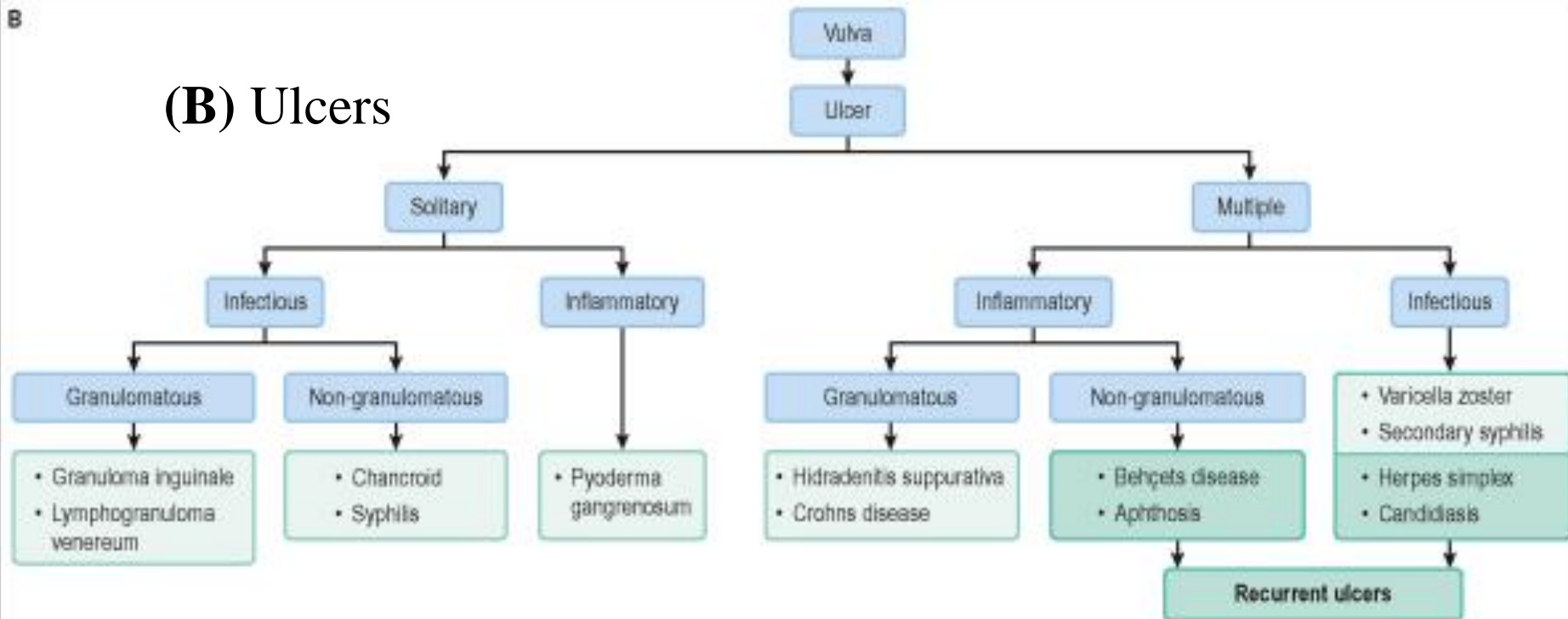
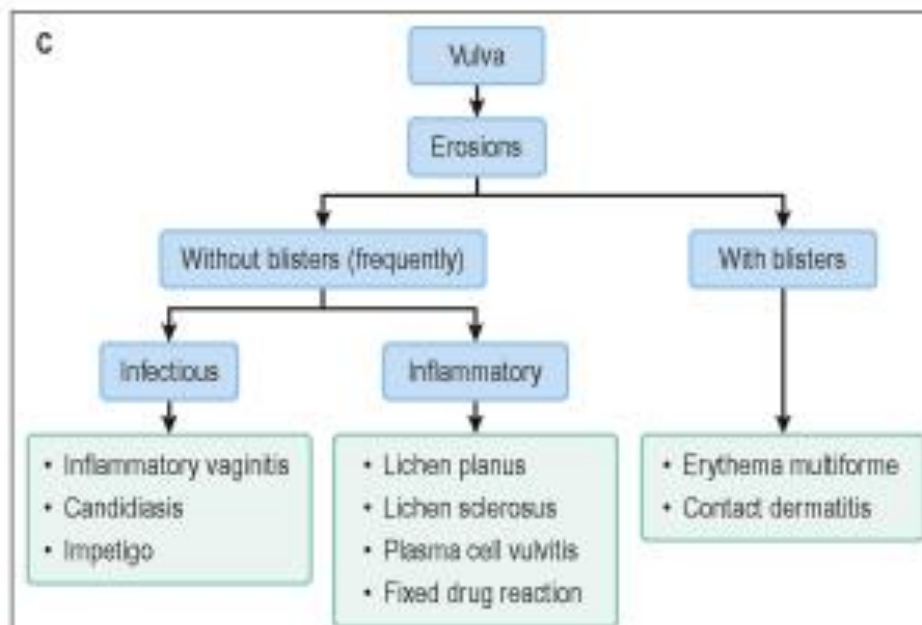
Differential diagnoses of vulvar dermatoses.

(A) Non-infectious Inflammatory conditions.



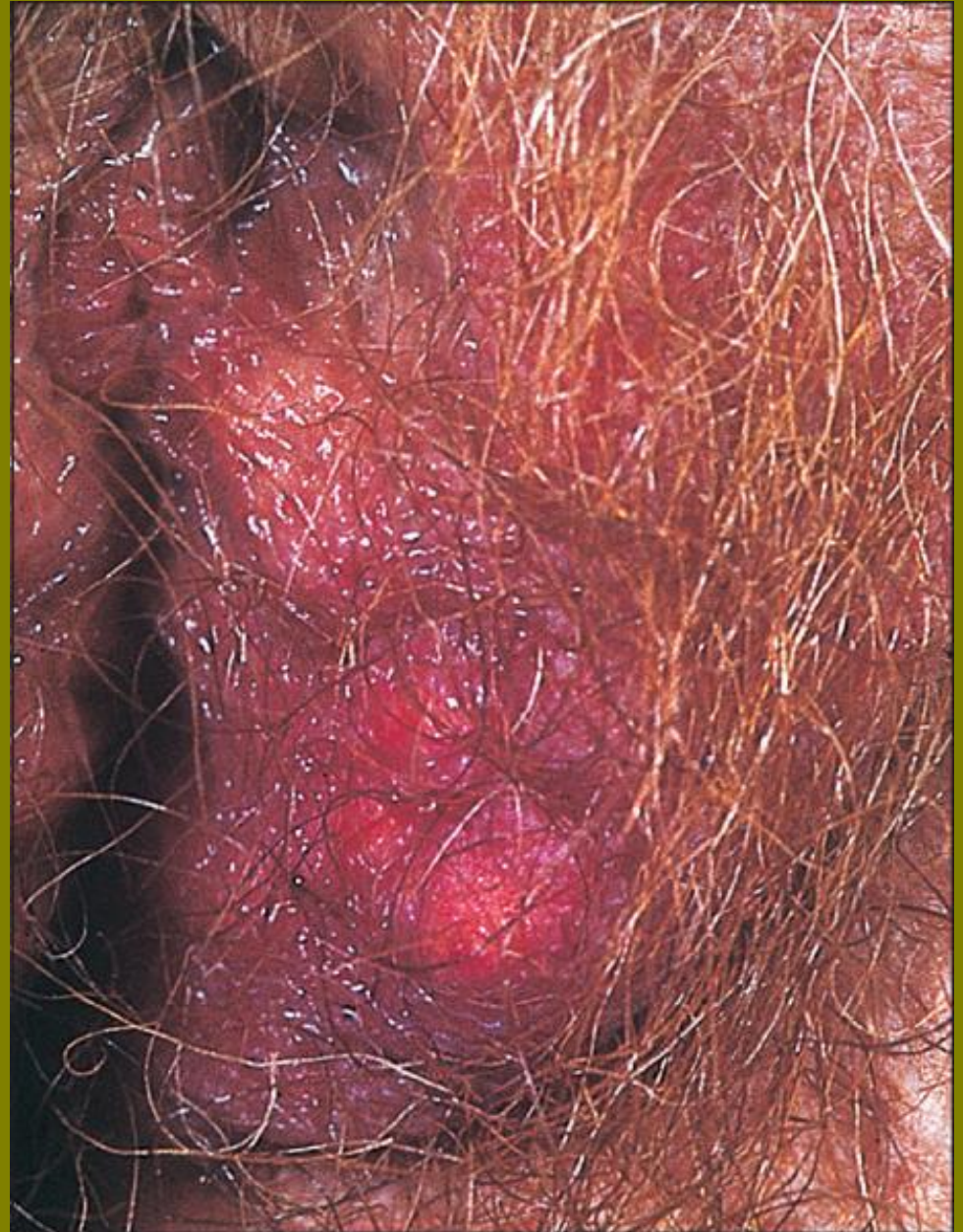
Non-infectious inflammatory conditions



B**(B) Ulcers****(C) Erosions**

Follicular cysts ('epidermal inclusion cysts').

**Ruptured cysts become inflamed as a result of foreign body
giant cell reaction to
keratin leakage.**



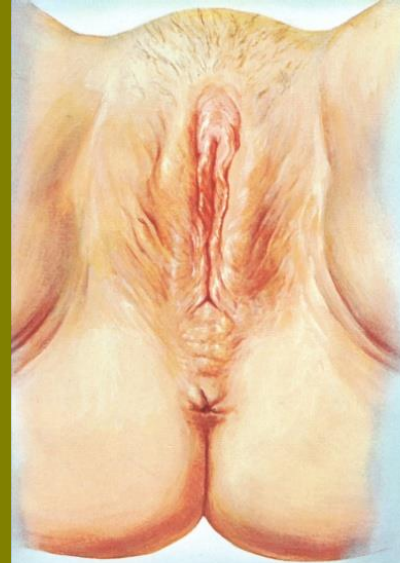
PATHOLOGY OF VULVA

LICHEN SCLEROSUS ET ATROPHICUS

LEUKOPLAKIA

LICHEN
SCLEROSUS
ET
ATROPHICUS

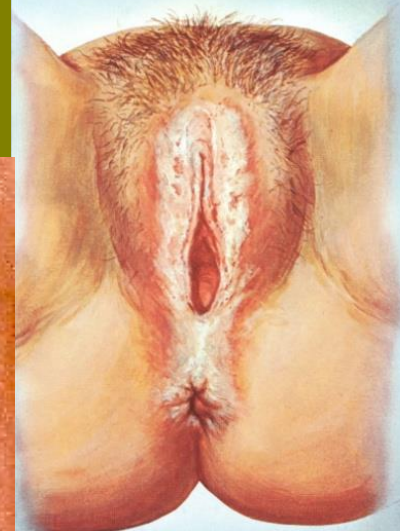
CANCER
DEVELOPING
FROM
ATROPHIC
LESION



SENILE ATROPHY



KRAUROSIS VULVAE



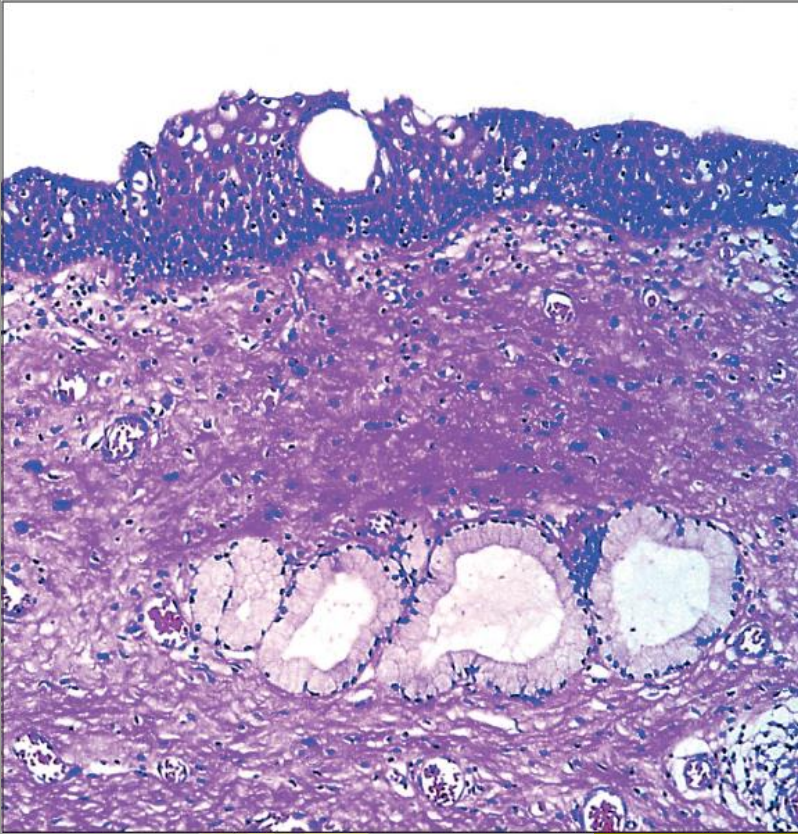
LEUKOPLAKIA



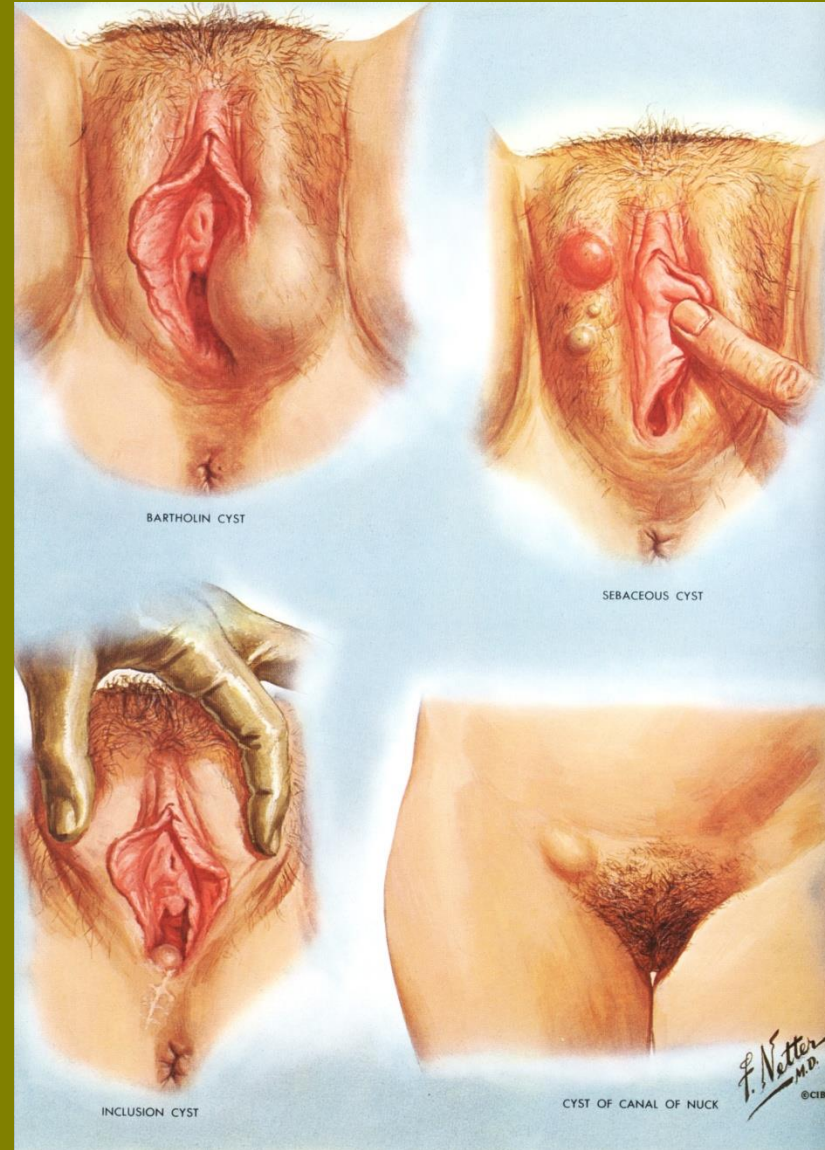
LICHENIFICATION

PATHOLOGY OF VULVA

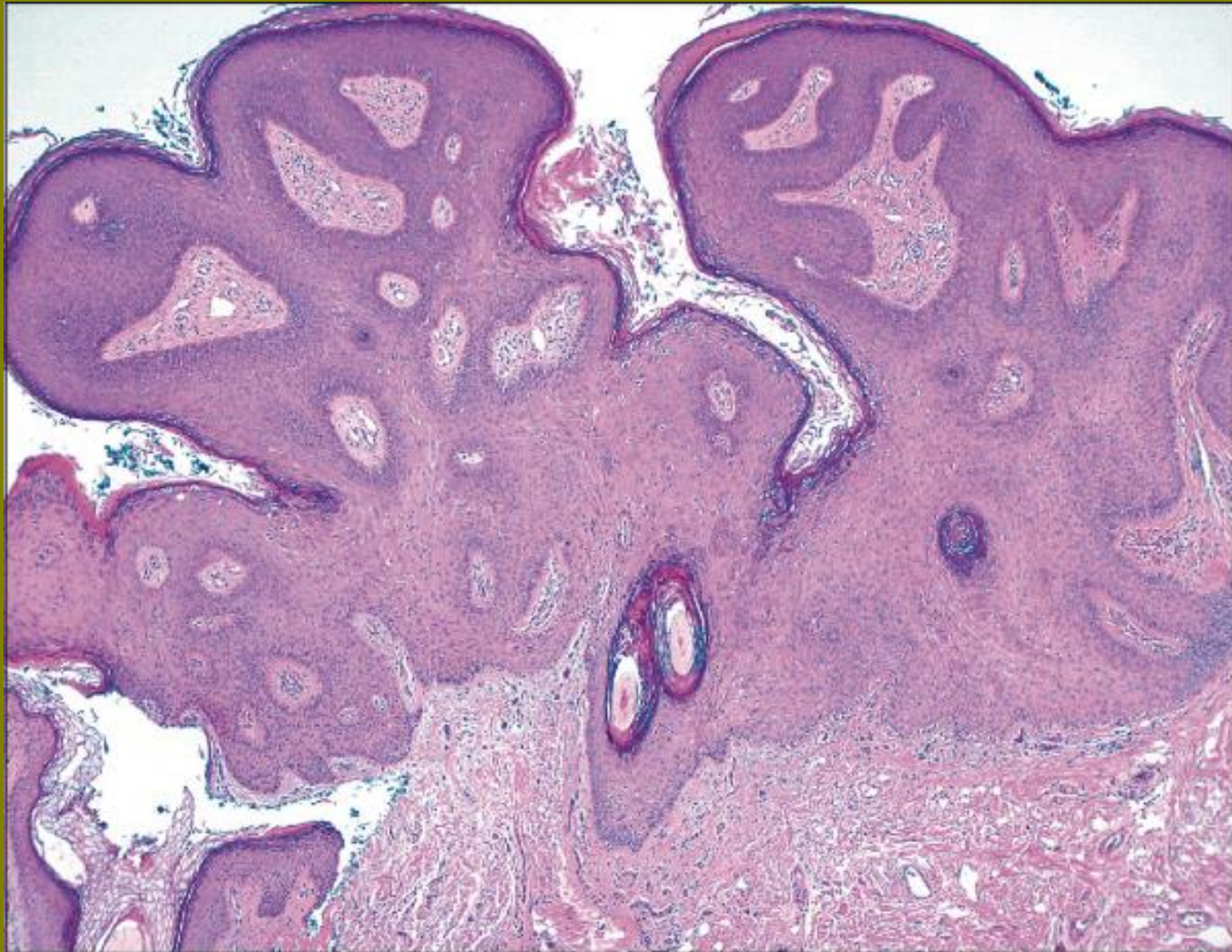
NON-CANCEROUS CYSTS OF VULVA



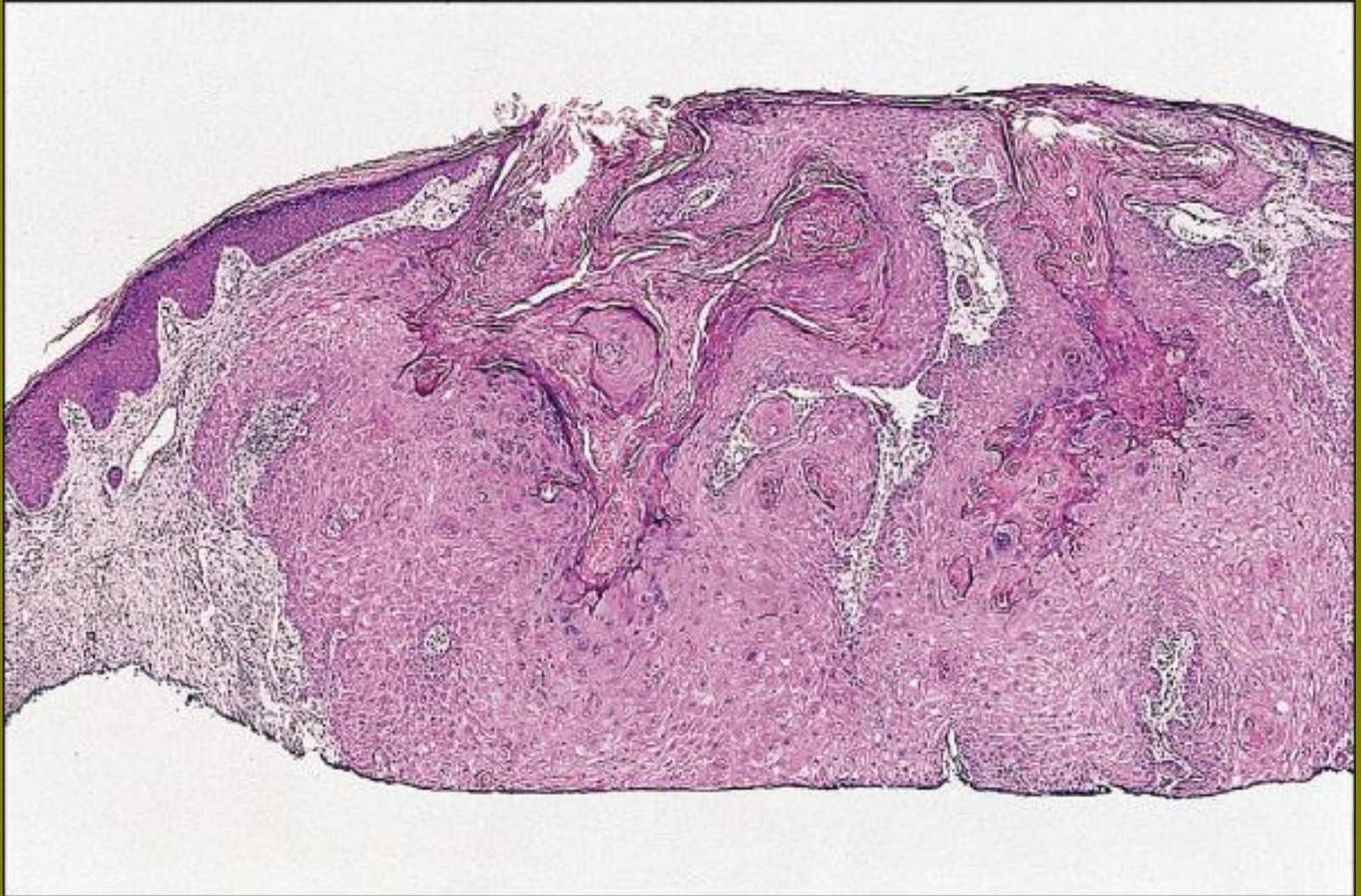
Bartholin cyst. A thick transitional epithelium with focal squamous metaplasia lines the cyst wall (top). Normal mucus glands are frequently seen in the wall.



Seborrheic keratosis. Invaginations lead to keratin-filled cysts.



Keratoacanthoma.



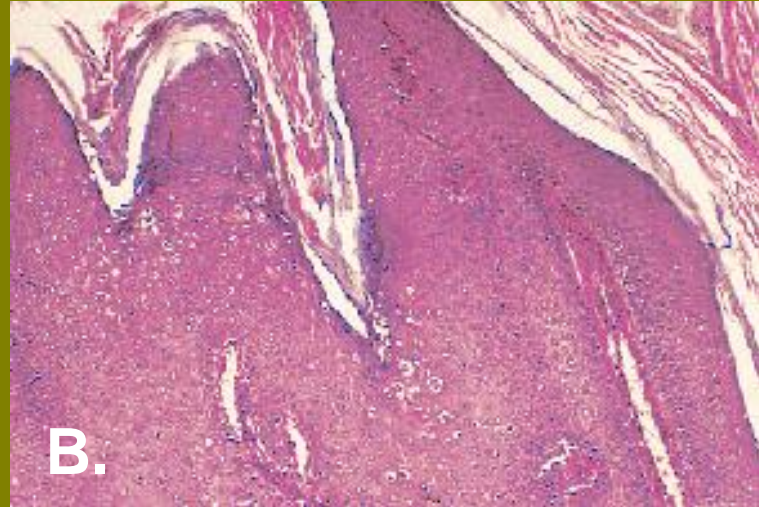
PATHOLOGY OF VULVA

CONDYLOMA ACUMINATUM



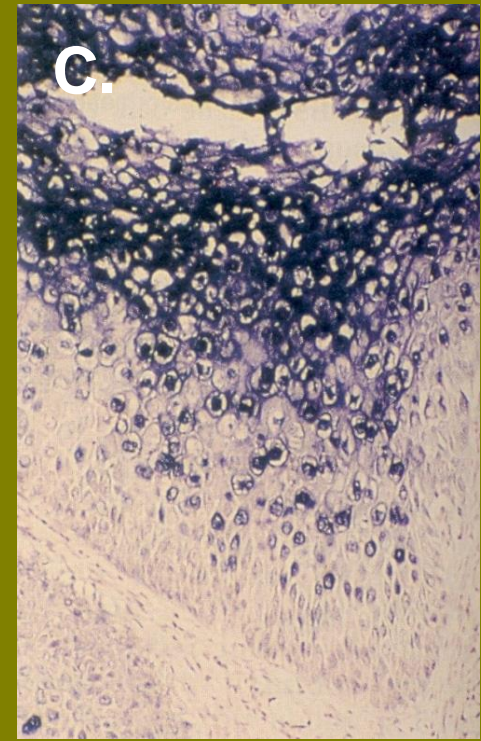
A.

**A. GROSS PATHOLOGY
OF CONDYLOMA
ACUMINATUM**



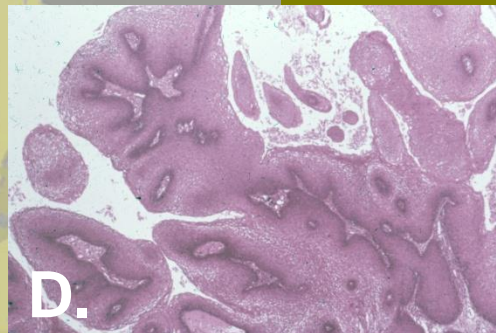
B.

**B. MICROSCOPIC
PICTURE**



C.

**C. VIRAL INCLUSIONS IN
EPITHELIUM**

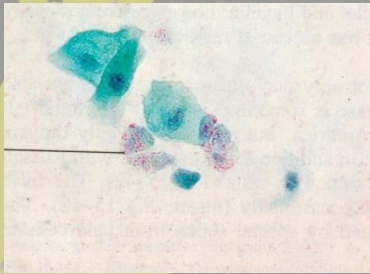


D.

**D. MICROSCOPIC
PICTURE OF
CONDYLOMA
ACUMINATUM**

PATHOLOGY OF VAGINA AND UTERINE CERVIX COLPITIS AND CERVICITIS

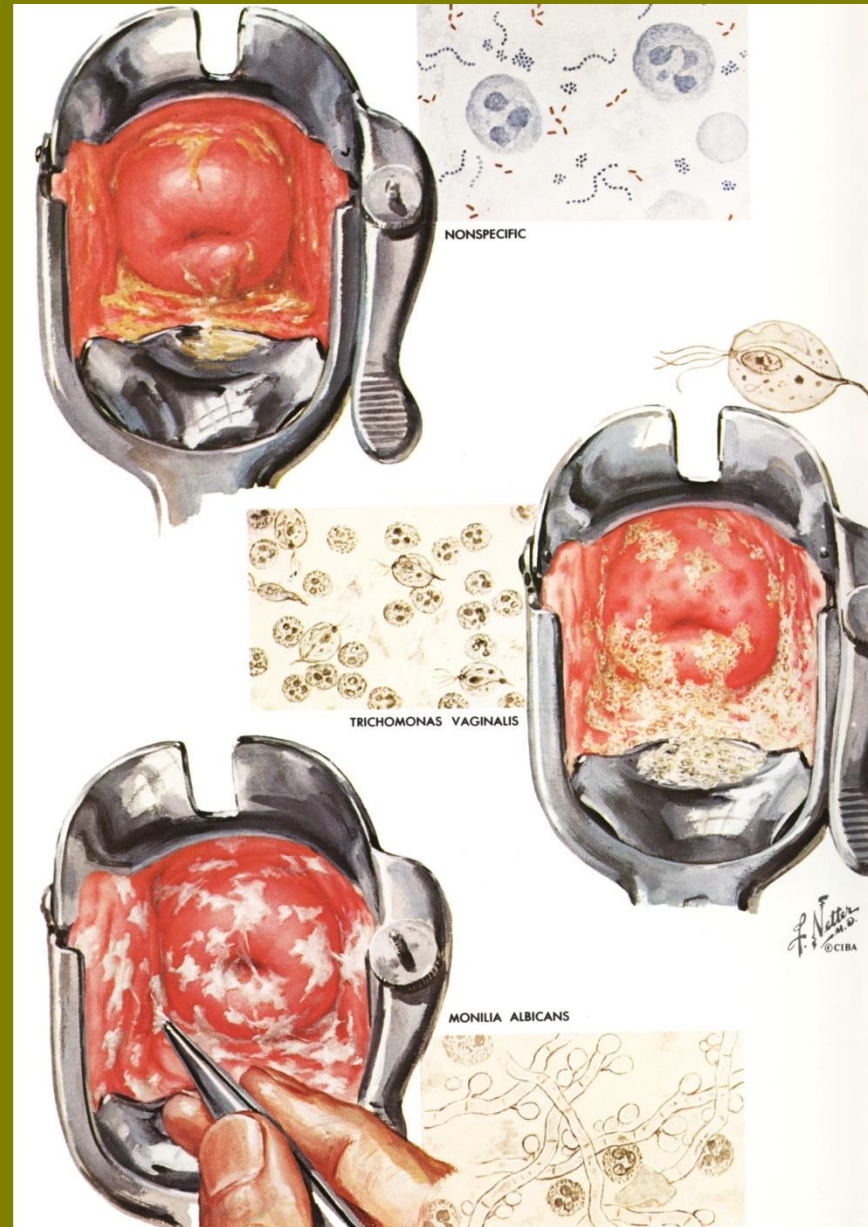
NON-SPECIFIC



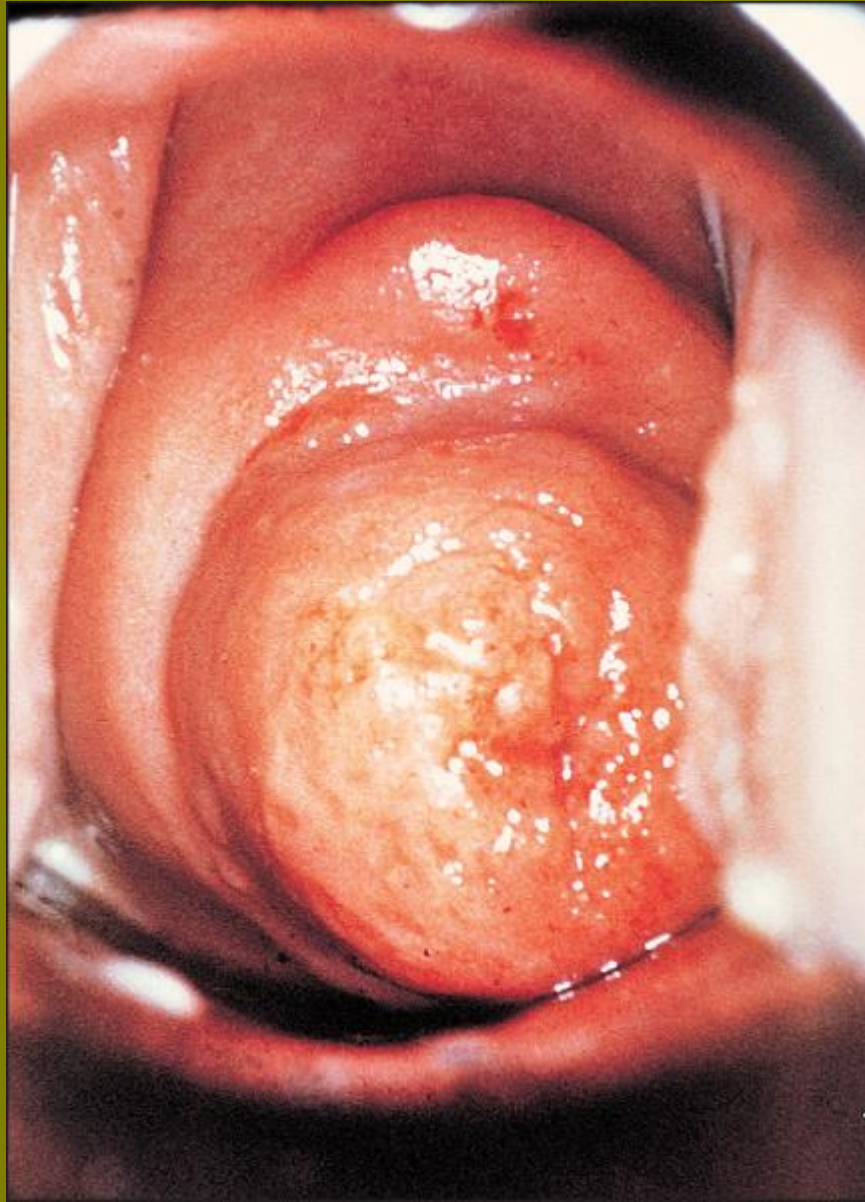
TRICHOMONIASIS



MONILIASIS



DES-associated cervical abnormalities. A coxcomb (upper) and pseudopolyp (middle) are present.



DEVELOPMENTAL DISTURBANCES

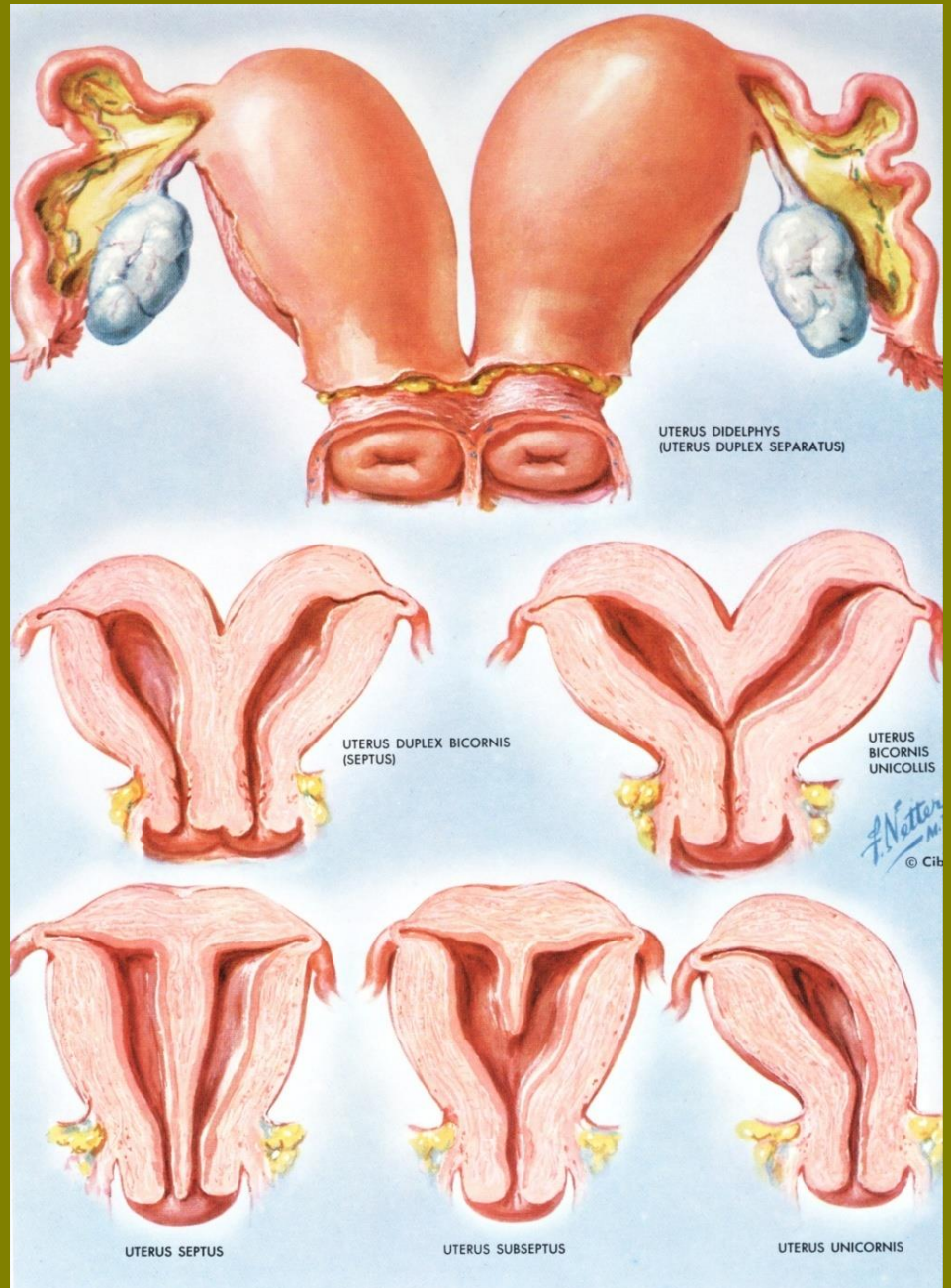
UTERUS DUPLEX

UTERUS ARCUATUS

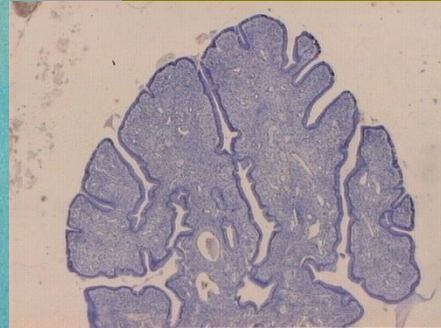
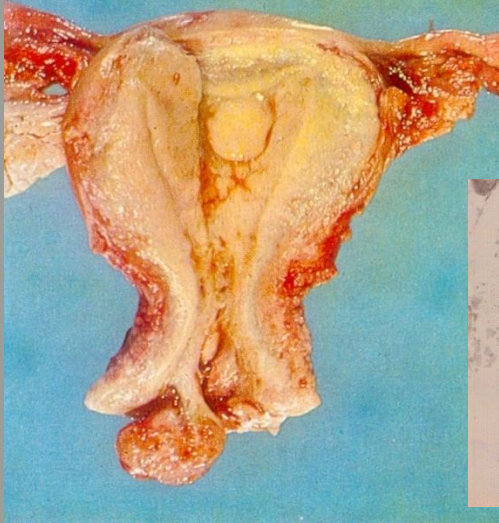
UTERUS BICORNIS

UTERUS UNICORNIS

UTERUS SUBSEPTUS

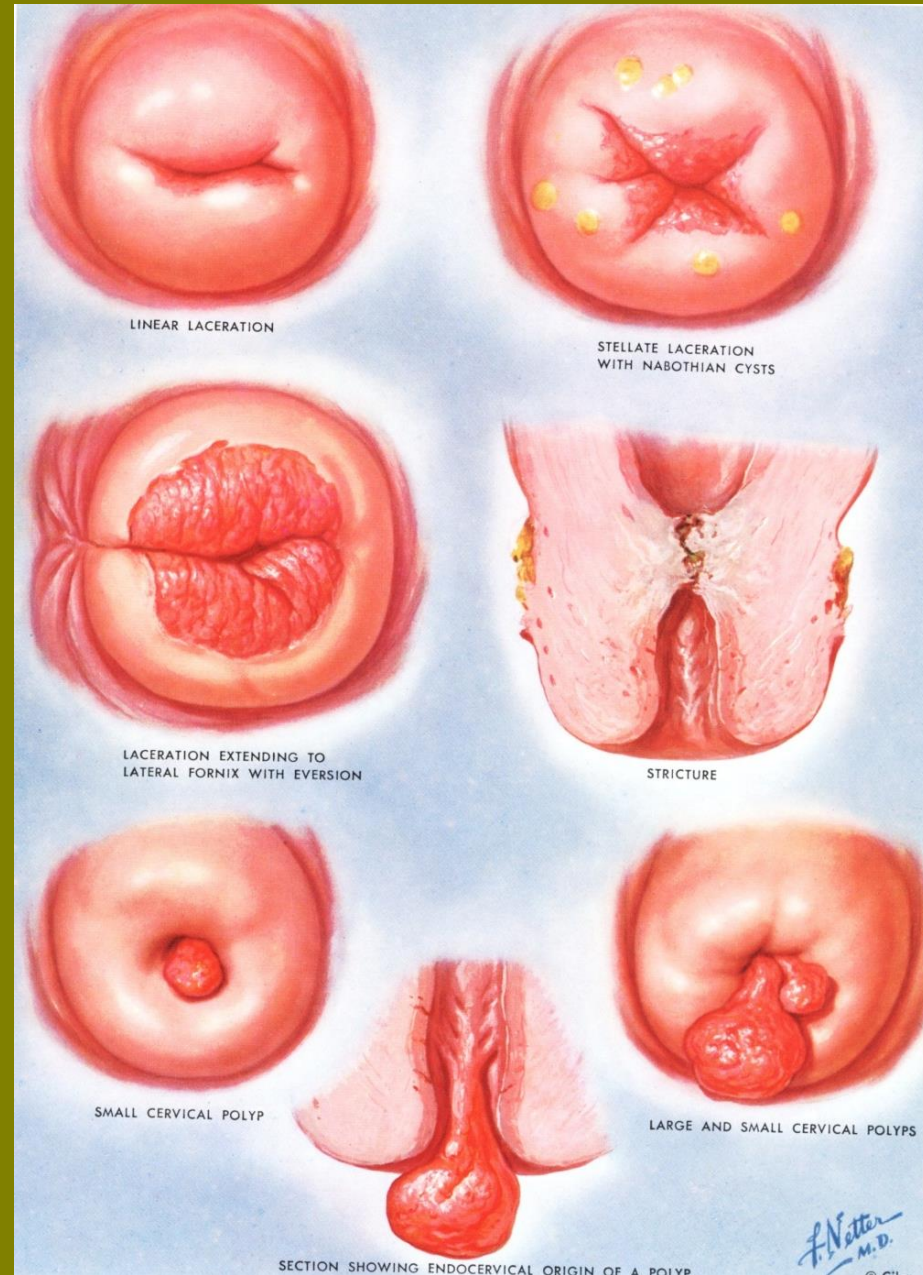


POLYPS AND LACERATION

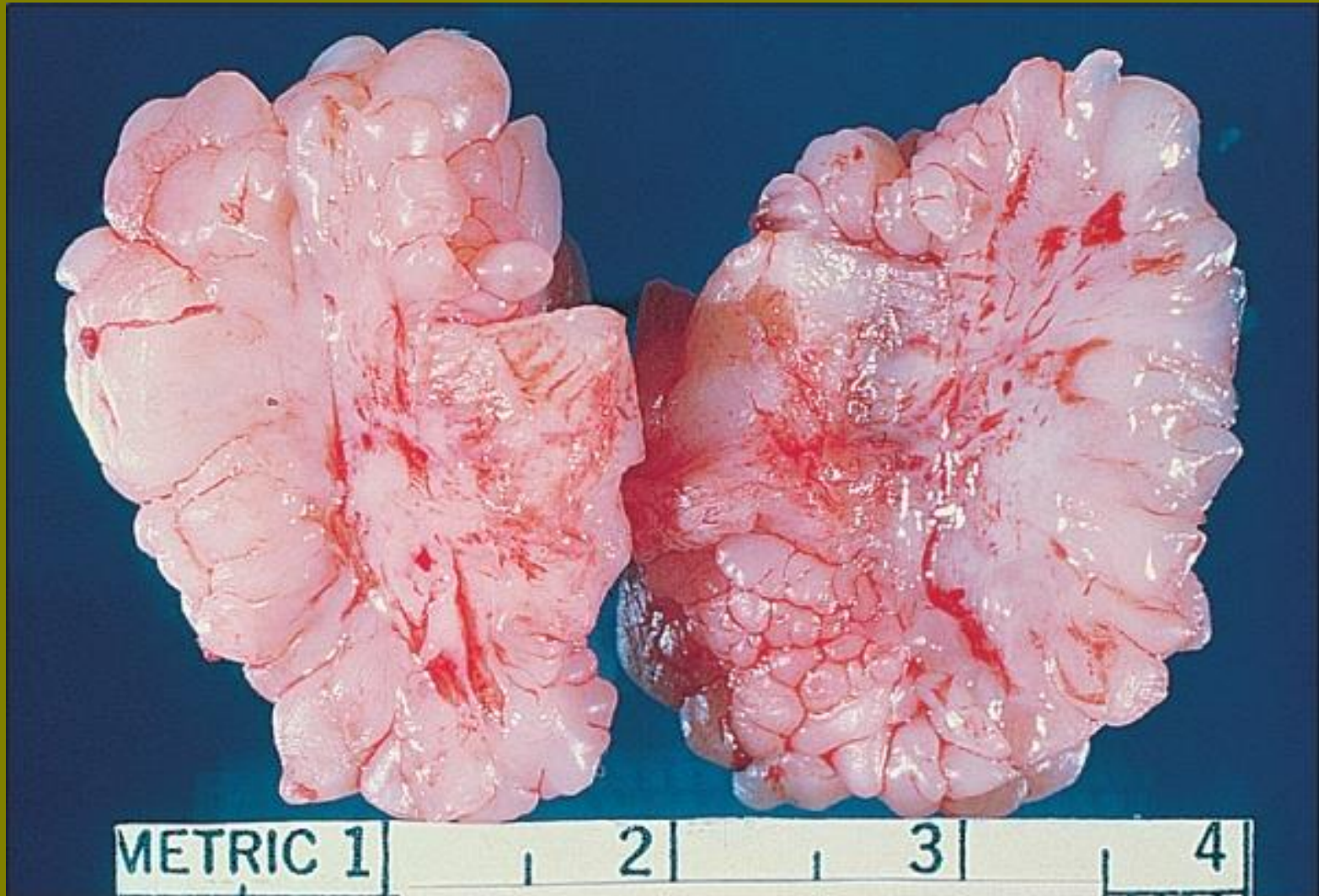


POLYP OF UTERINE CERVIX

POLYPS OF CERVIX ARE COMMON, OF DIFFERENT SIZES, BUILT FROM DIFFERENT PROPORTIONS OF FIBROUS STROMA, GLANDS AND SQUAMOUS EPITHELIUM



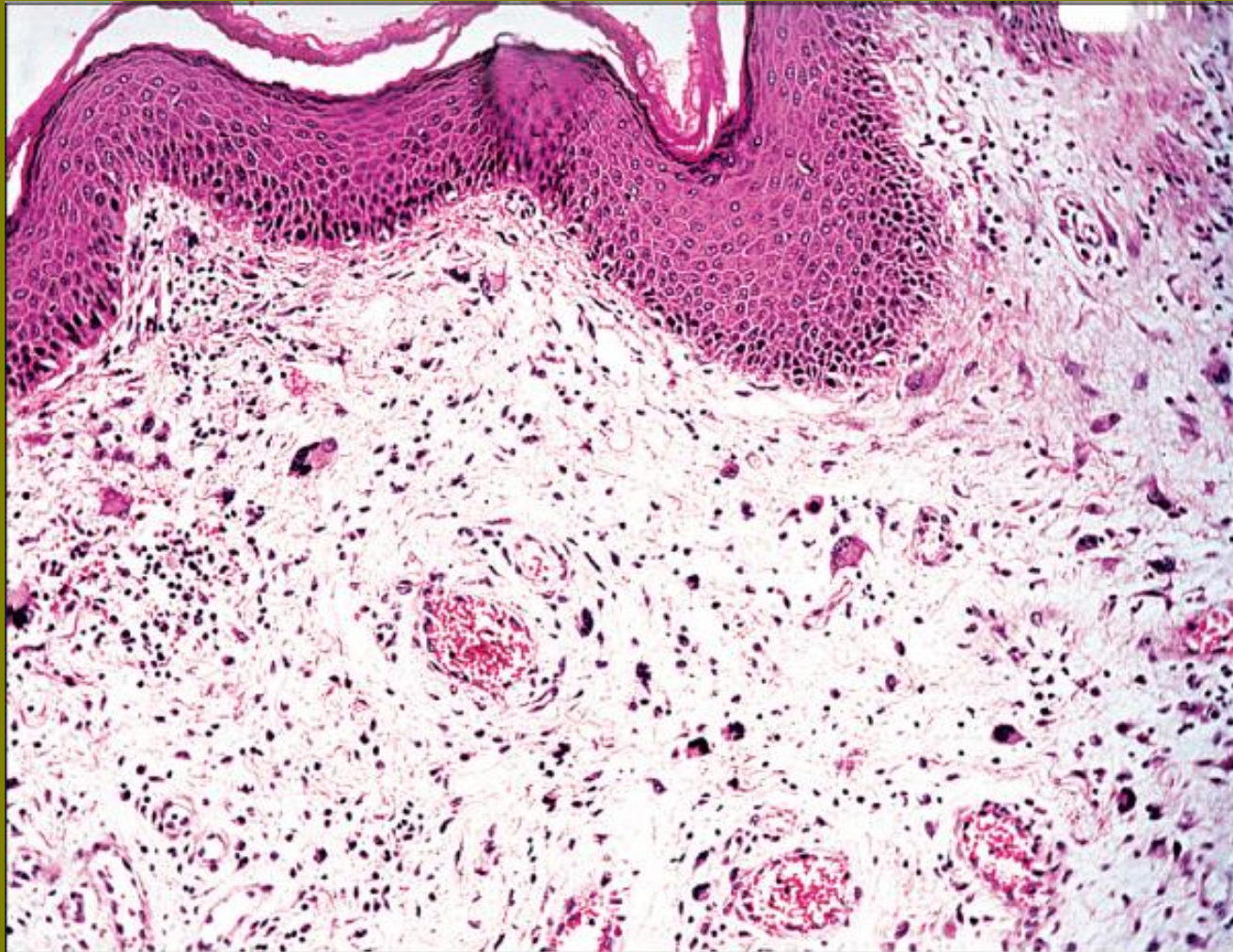
Fibroepithelial polyp, solid.



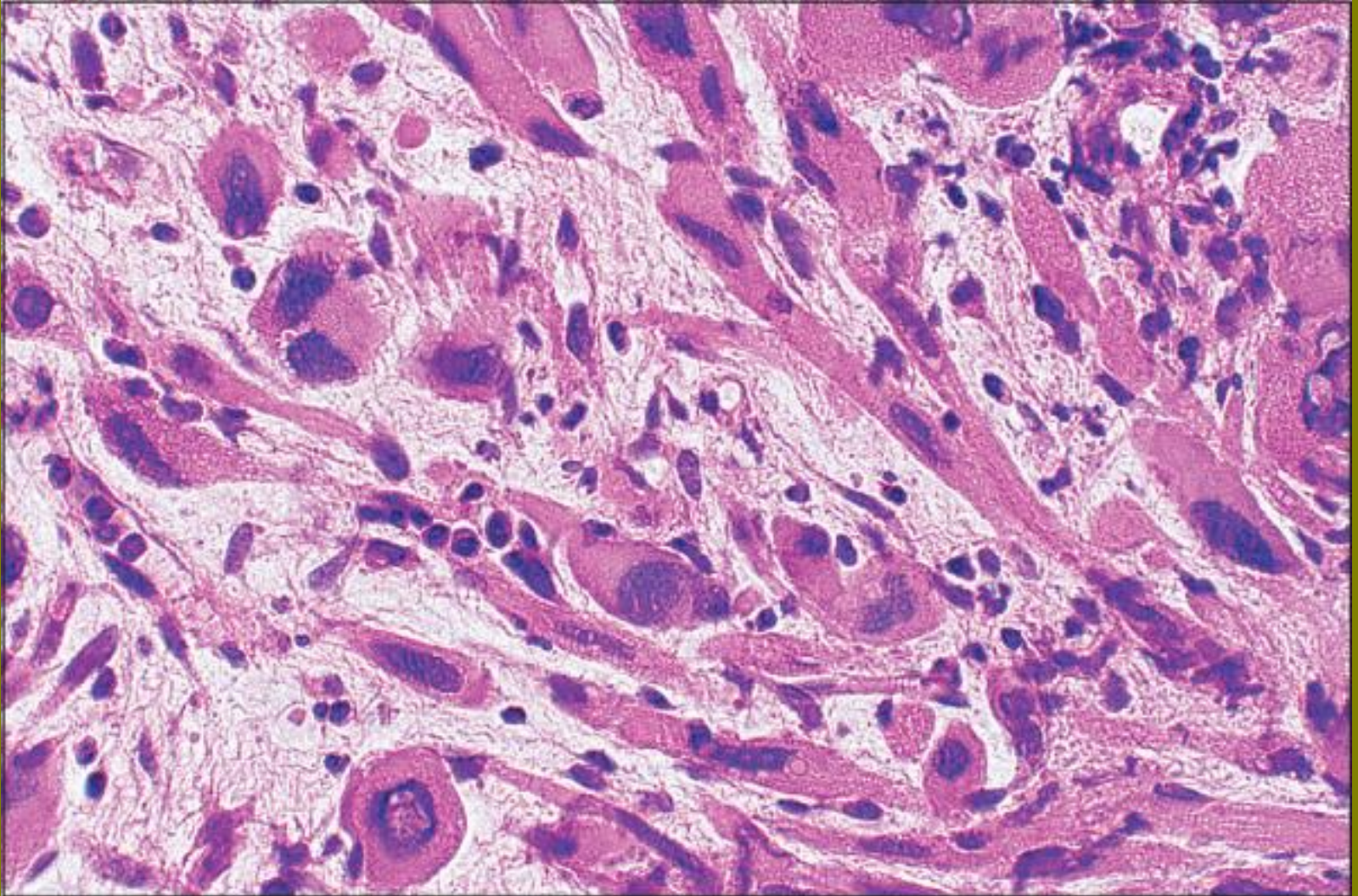
Fibroepithelial polyp, villiform. The tumor has numerous finger-like projections.



Fibroepithelial polyp. Large atypical stromal cells with delicate, pointed cytoplasmic processes are conspicuous.



Rhabdomyoma of vagina.



HUMAN PAPILLOMAVIRUS (HPV) INFECTION

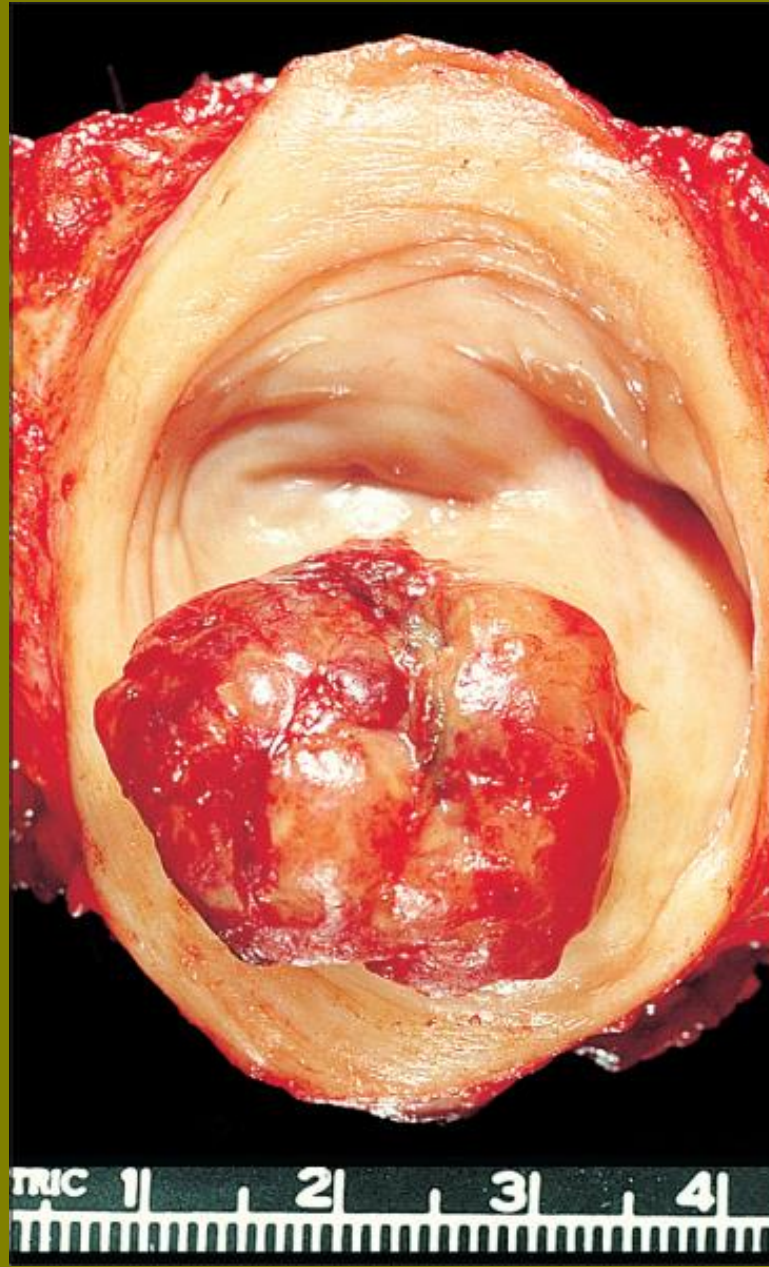
Vaginal mucosa is frequently the site of genital papillomavirus infections, hosting approximately one-fourth of genital condylomata.

Lesions may be flat, slightly raised, or verrucous with stromal papillae (asperities). While frequently small and virtually invisible without colposcopic visualization, they usually become more easily identified following application of 3% acetic acid.

Biopsy usually discloses cells with a characteristic perinuclear halo (koilocytes).

Condylomata acuminata are predominantly HPV 6 or 11 positive. In one study of 71 patients with premalignant changes, 15 different HPV DNA types were found (HPV 16, 18, 30, 31, 35, 40, 42, 43, 51, 52, 53, 54, 56, 58 and 66)

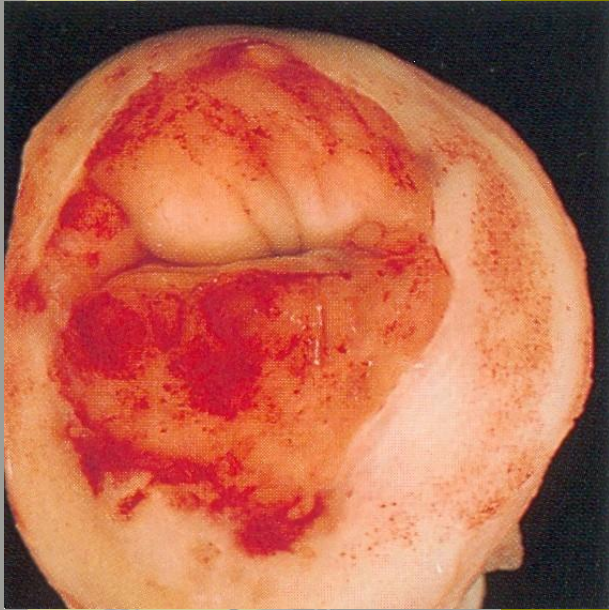
Verrucous carcinoma (vagina).



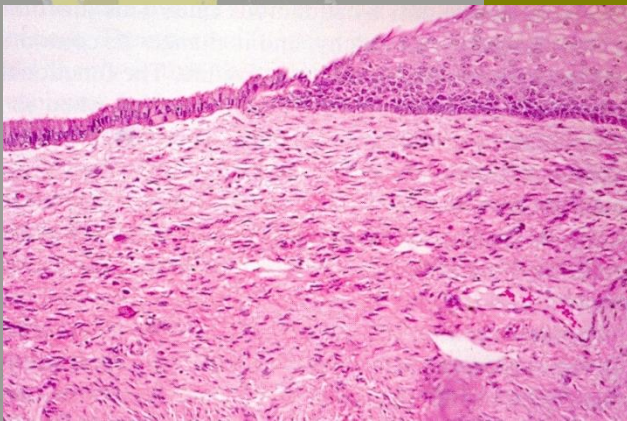
Verrucous carcinoma. The deep margin discloses no invasion (vagina)



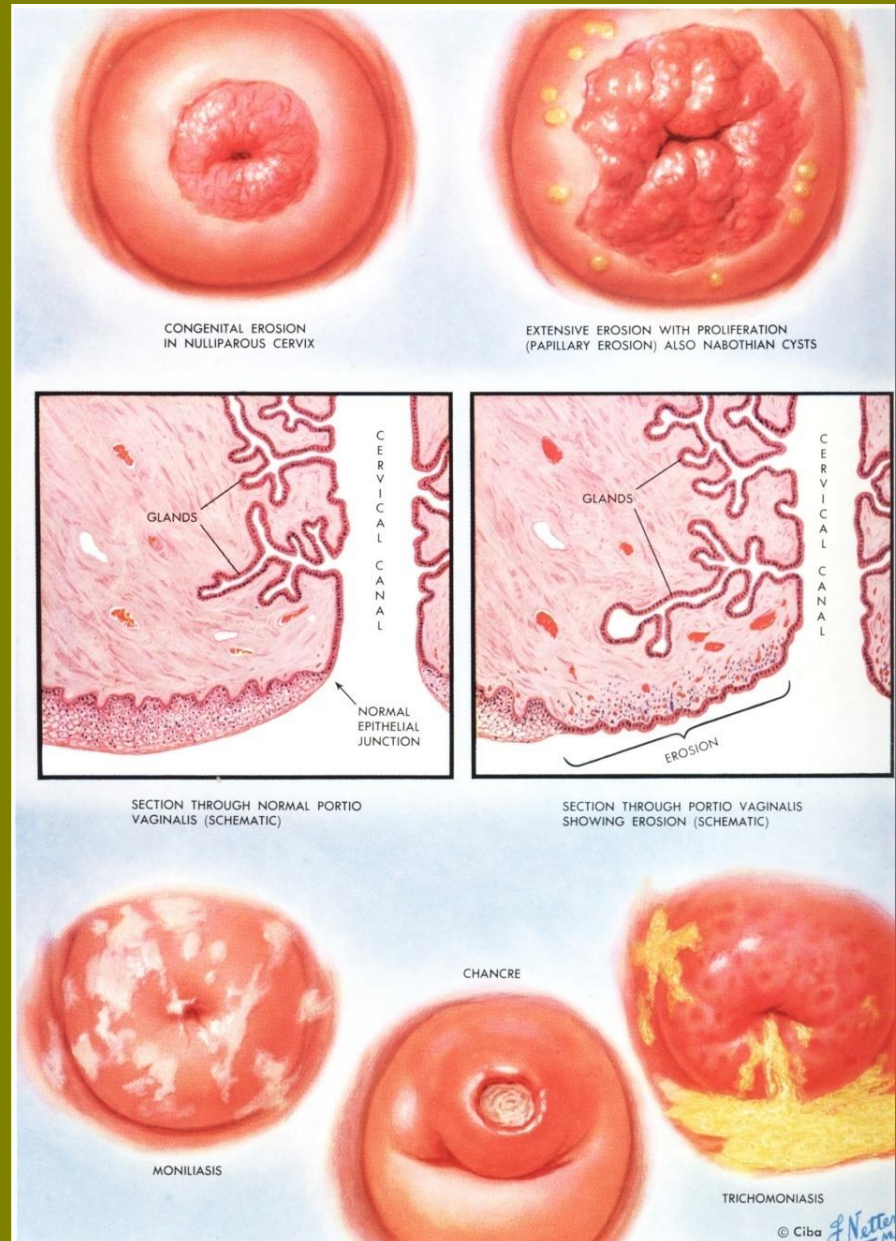
CHANGES IN THE CERVIX



**GLANDULAR EROSION,
ECTROPION**



TRANSITIONAL ZONE



CONGENITAL EROSION
IN NULLIPAROUS CERVIX

EXTENSIVE EROSION WITH PROLIFERATION
(PAPILLARY EROSION) ALSO NABOTHIAN CYSTS

GLANDS

CERVICAL
CANAL

NORMAL
EPITHELIAL
JUNCTION

GLANDS

CERVICAL
CANAL

EROSION

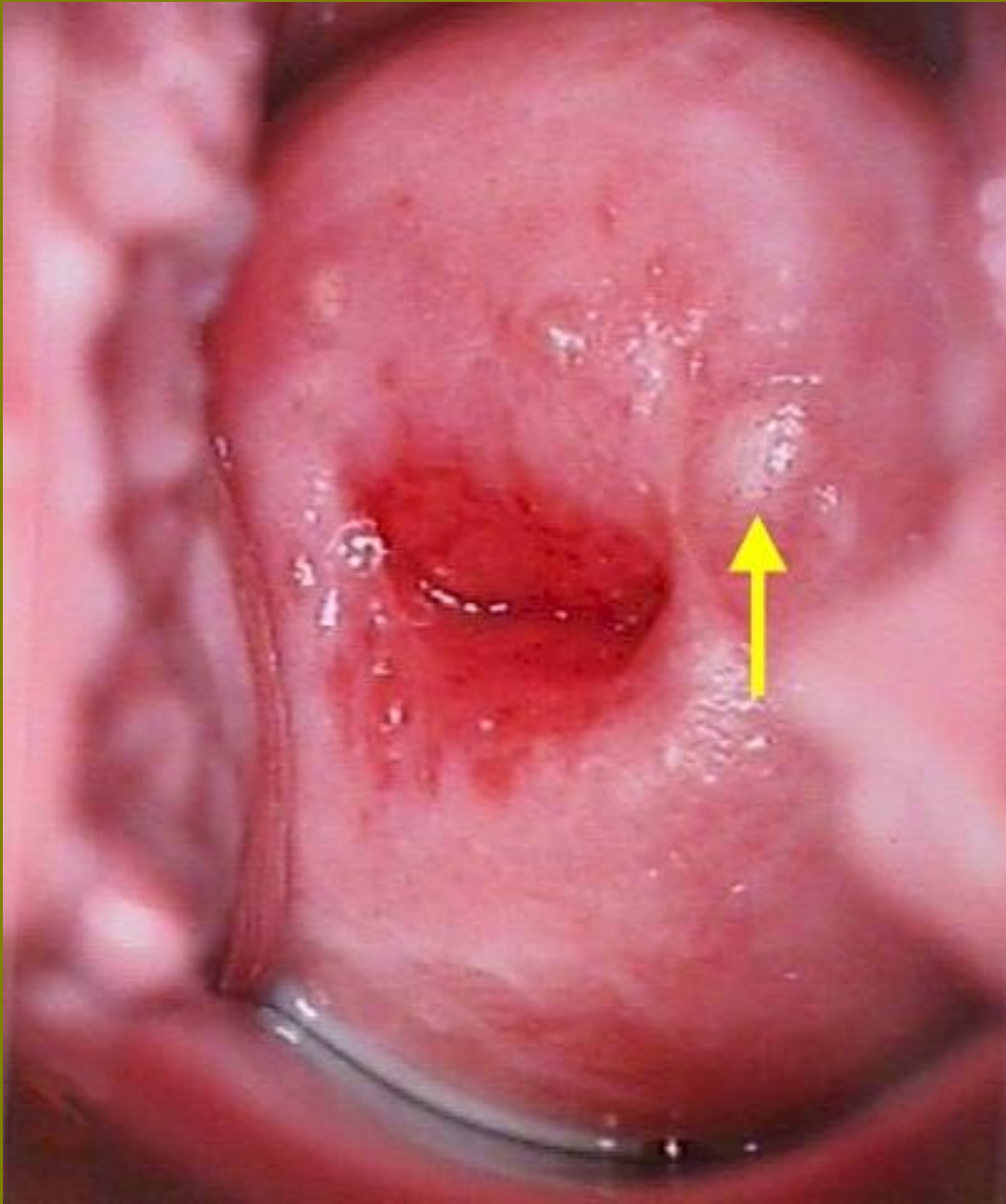
SECTION THROUGH NORMAL PORTIO
VAGINALIS (SCHEMATIC)

SECTION THROUGH PORTIO VAGINALIS
SHOWING EROSION (SCHEMATIC)

MONILIASIS

CHANCRE

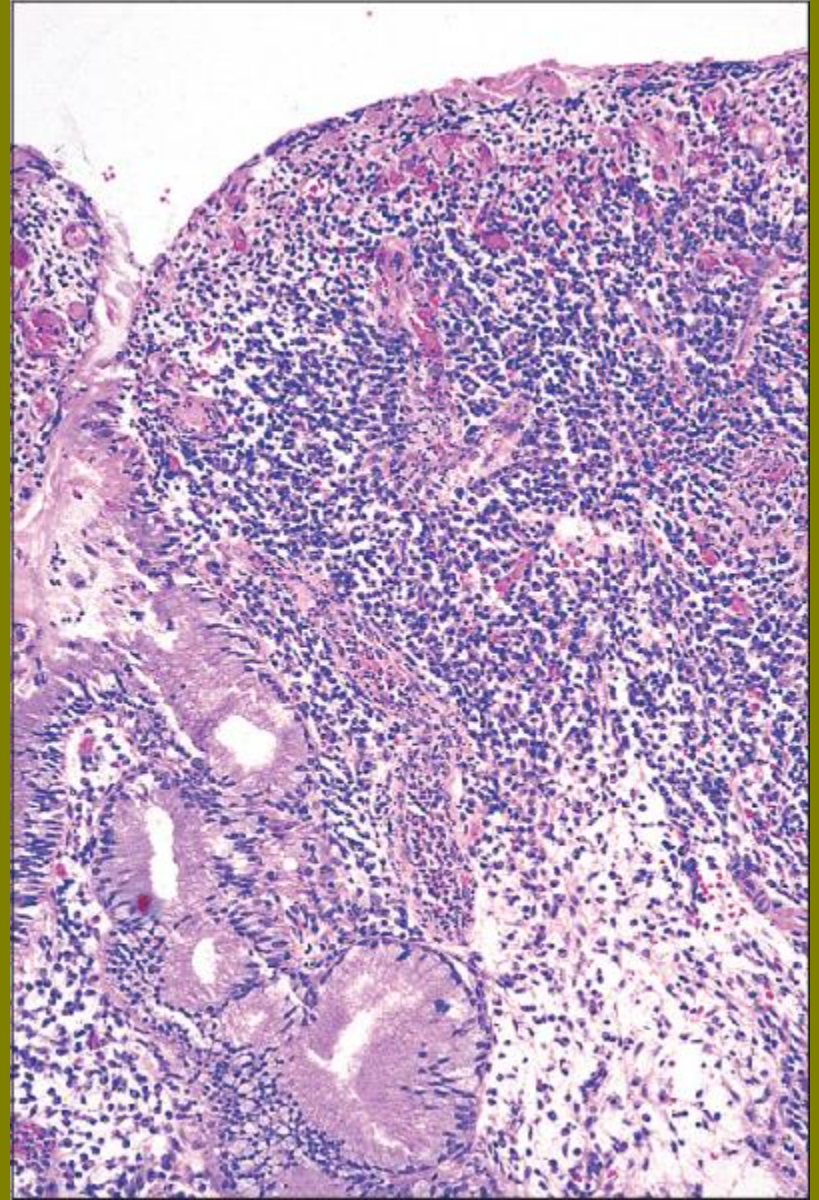
TRICHOMONIASIS



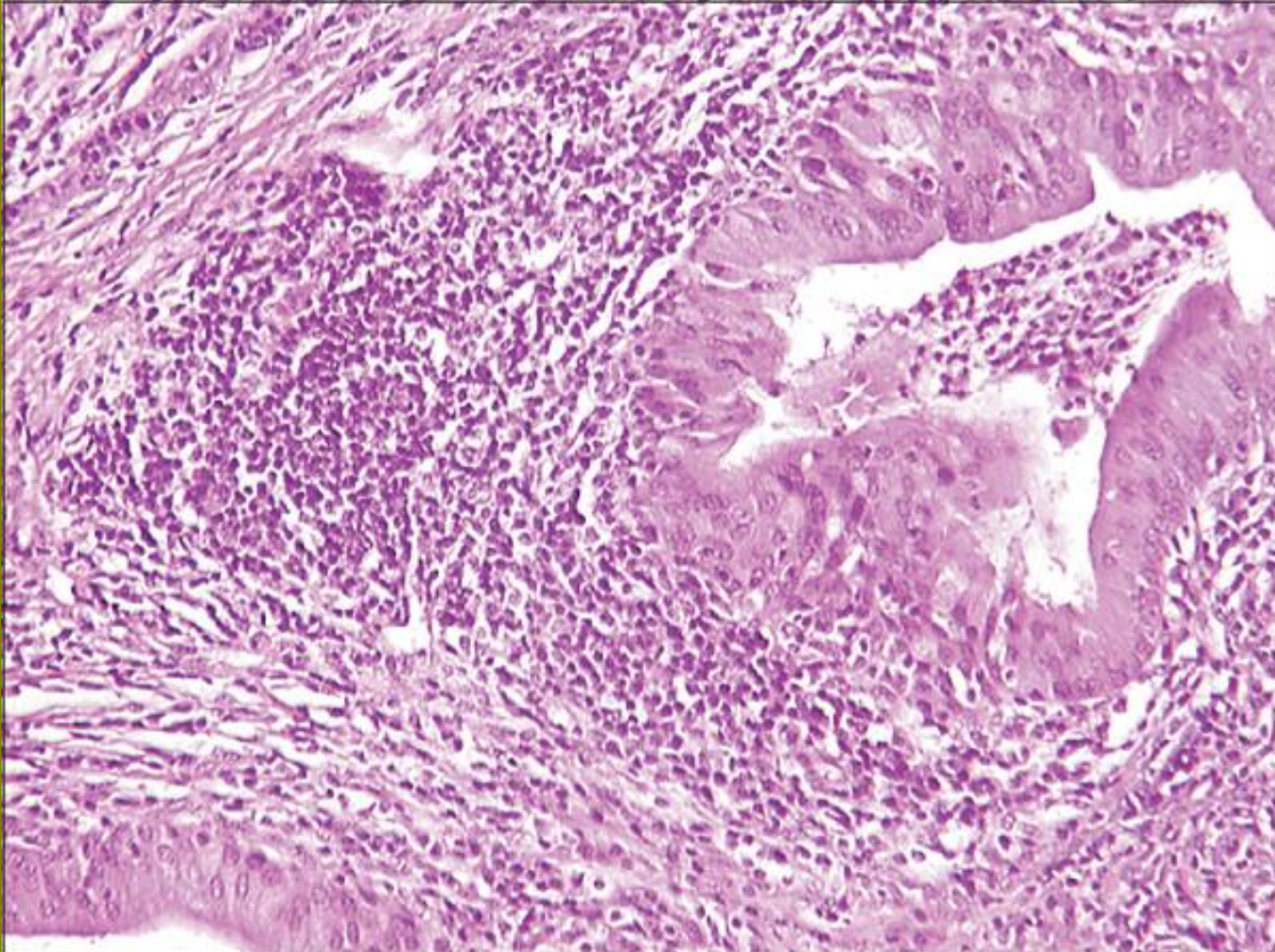
CERVICAL ECTROPION

**([www.womanh
ealth](http://www.womanhealth))**

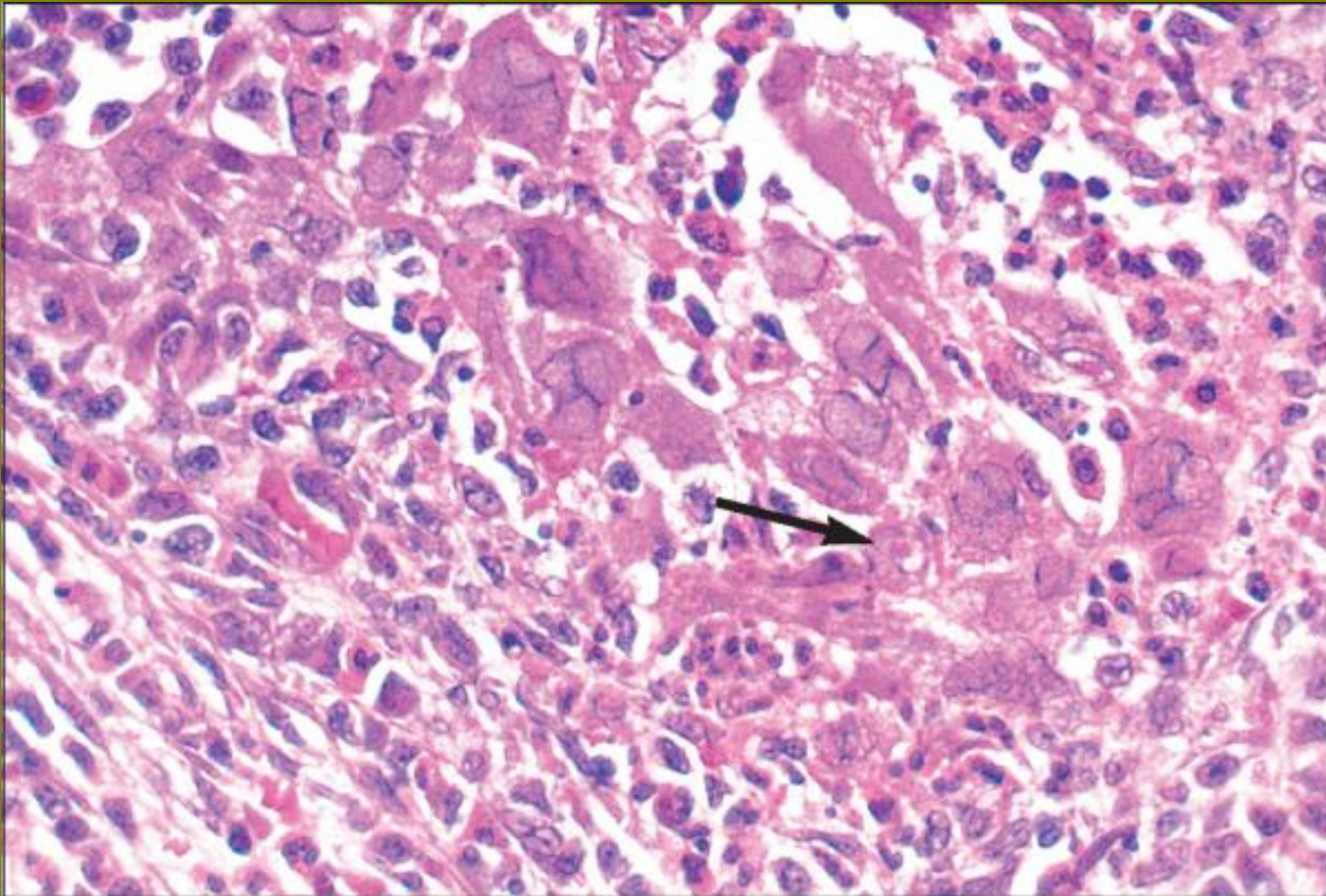
Chronic cervicitis. There is a dense infiltrate of lymphocytes and neutrophils, with new blood vessel formation and dilated vessels packed with neutrophils.



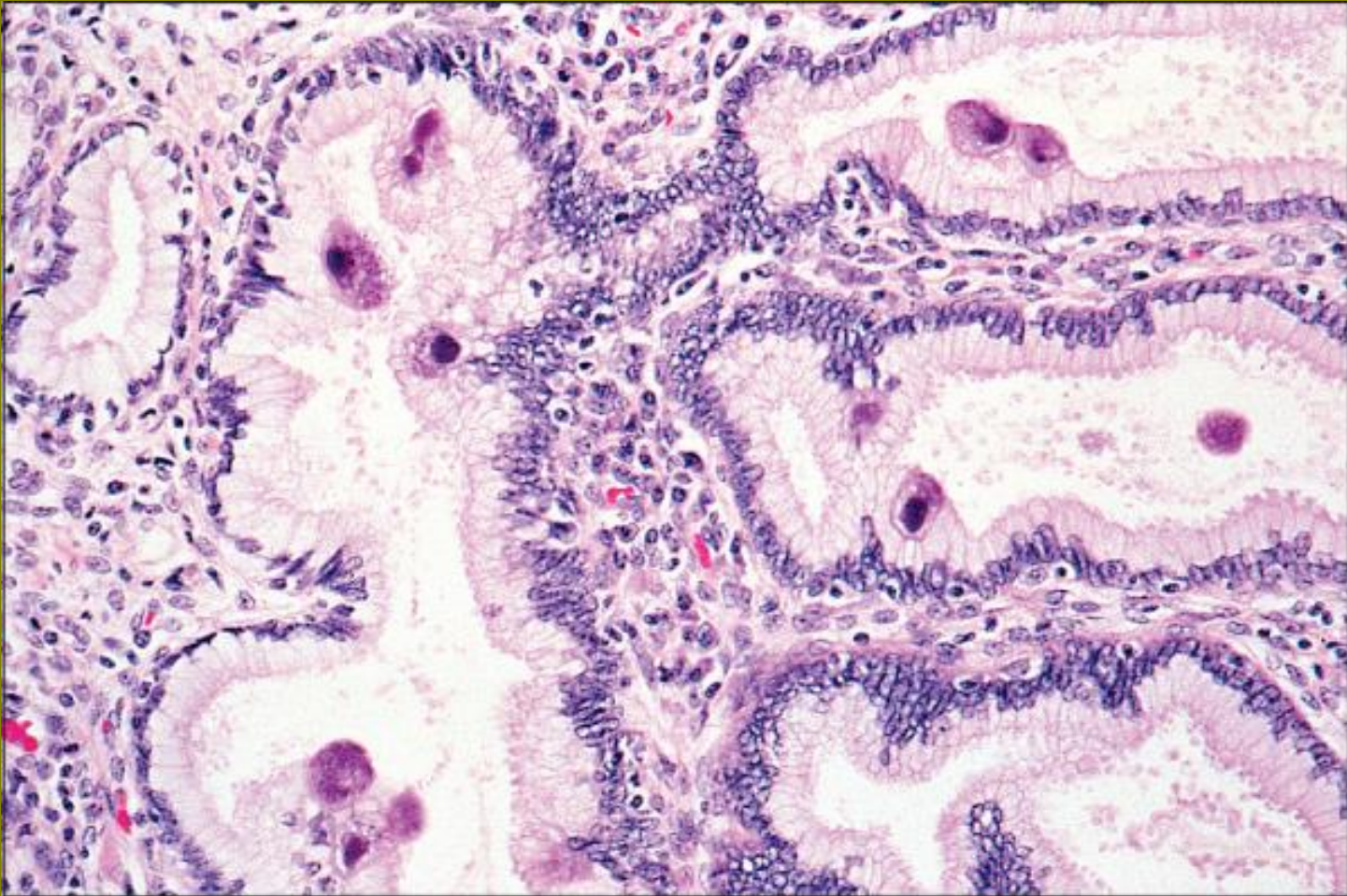
Chlamydial cervicitis. A gland crypt surrounded by a dense infiltrate of lymphocytes and plasma cells. The infiltrate also affects the epithelium and is present in the gland lumen.



Herpes simplex infection. There is lysis of the epithelial tissue. Multinucleated giant cells are present (arrow), showing margination of chromatin and a 'ground-glass' appearance of the nuclei.



Cytomegalovirus infection. Several endocervical cells within the crypt epithelium show large, rounded, basophilic inclusions.



CERVICAL CARCINOMA

Comparison of HPV frequency detected by laboratory techniques

	DNA hybridization (%)	Koilocytosis (%)	Immunocytochemistry (%)
Condyloma	100	80	80
CIN 1	100	89	61
CIN 2	86	57	29
CIN 3 (severe dysplasia)	100	33	17
CIN 3 (carcinoma <i>in situ</i>)	100	20	0

1. THE DEVELOPMENT OF CERVICAL CANCER IS PRECEDED BY CHANGES IN THE EPITHELIUM.

2. IN THE PATHOMECHANISM OF CANCER

TRANSFORMATION THE KEY ROLE IS PLAYED BY HSV AND HPV VIRUSES

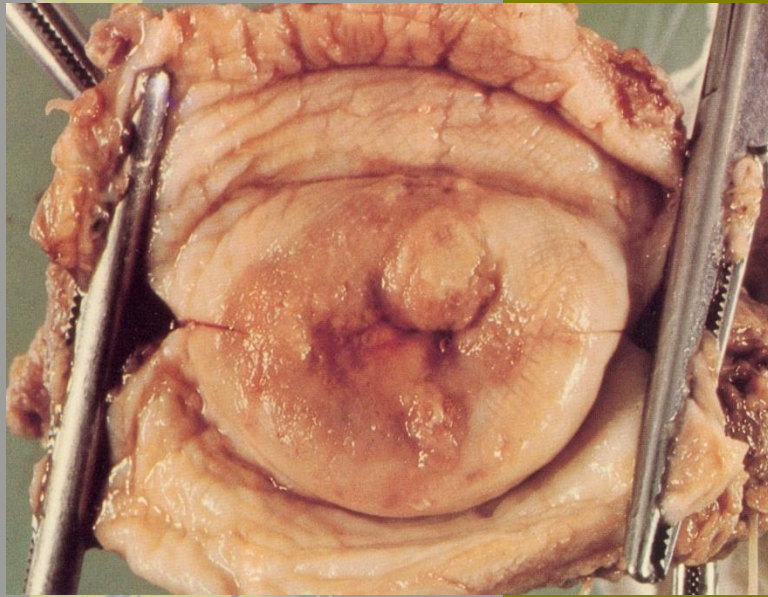
3. CERVICAL CANCER IS ONE OF THE MOST COMMON MALIGNANT CANCERS IN WOMEN

4. MOSTLY DEVELOPS IN THE REPRODUCTIVE PERIOD

5. RISK GROUPS: MANY PREGNANCIES, PROSTITUTES

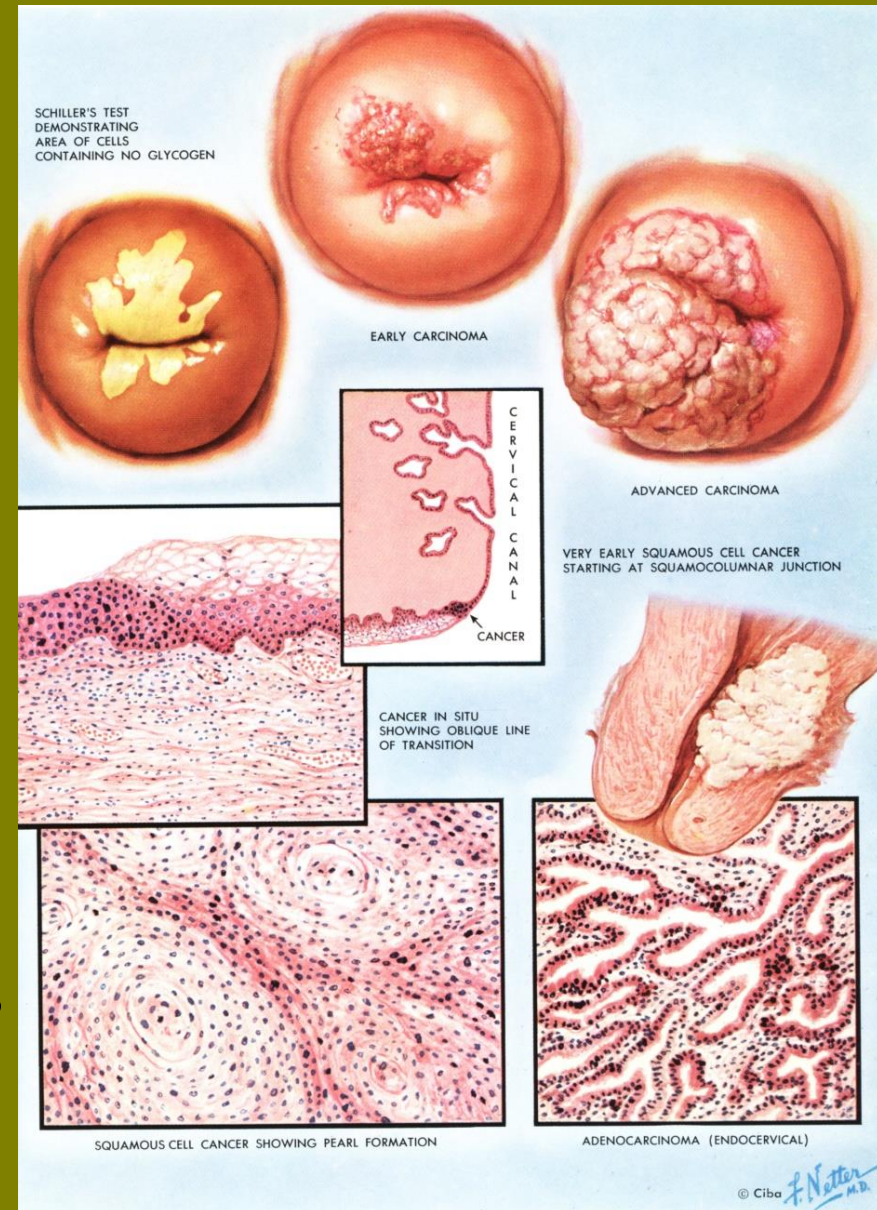
EPIDEMIOLOGY AND PATHOMECHANISM OF CERVICAL CANCER

CERVICAL CARCINOMA – MACROSCOPIC PICTURE HISTOLOGICAL FORMS



**THE MOST COMMON FORM OF CANCER
IN THE CERVIX IS SQUAMOUS CELL
CARCINOMA (SCC) – 95%.
5% - ADENOCARCINOMA**

**Cancer of the cervix is the second
most common cancer in women
worldwide after cancer of breast. It is
a single most common female genital
cancer in developing countries.**



Squamous cell carcinoma. Tumor protrudes through the external os and involves the exocervix.

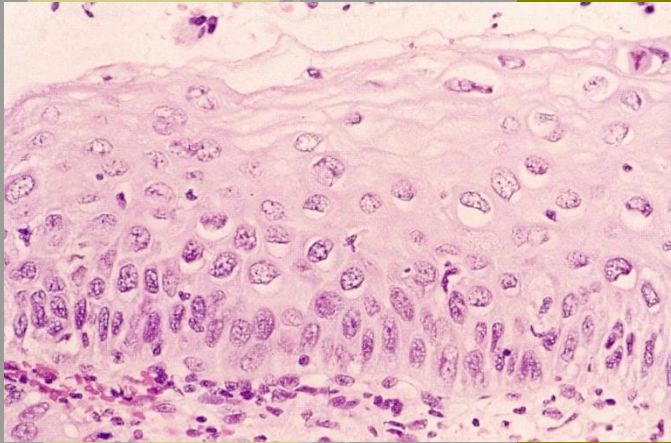


Squamous cell carcinoma. The uterus, cut in cross-section, discloses an extensive tumor infiltrating throughout the wall of the endocervix (white)

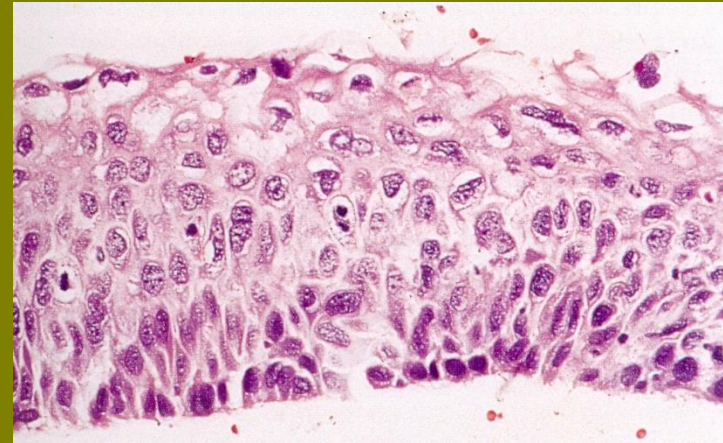


CERVICAL CARCINOMA – CARCINOMA PLANOEPITHELIALE (SCC)

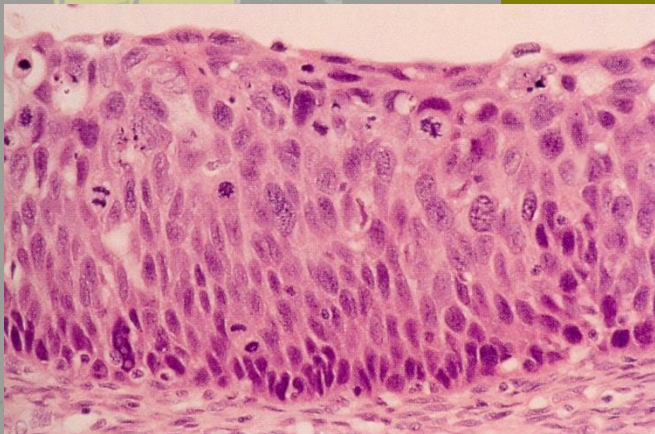
DEVELOPMENT OF SCC IS PRECEDED BY MANY DYSPLASTIC
CHANGES IN THE EPITHELIUM



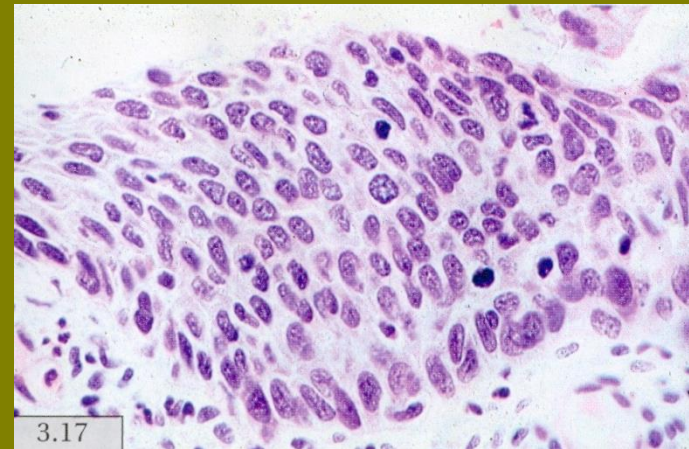
DYSPLASIA LEVIS (CIN 1)



DYSPLASIA MEDII GRADUS (CIN 2)

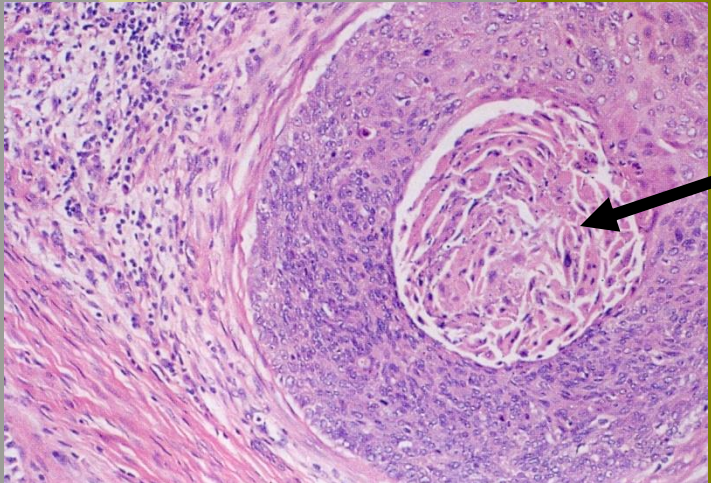


DYSPLASIA MAIORIS GRADUS (CIN 3)

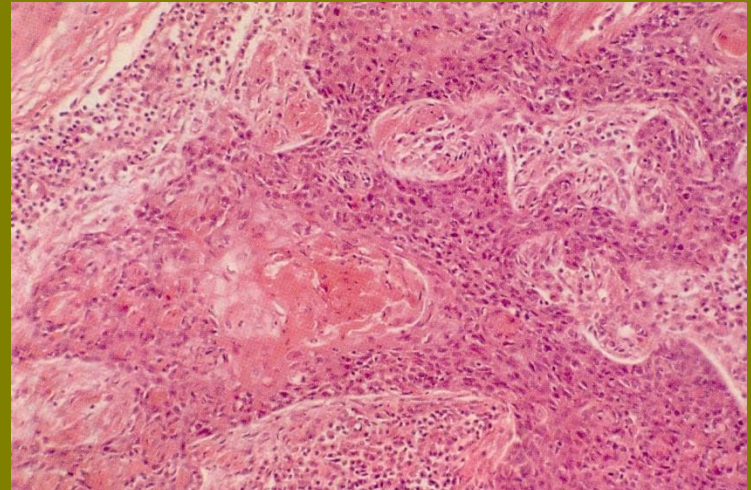


PREINVASIVE CARCINOMA (IN SITU) (CIN 3)

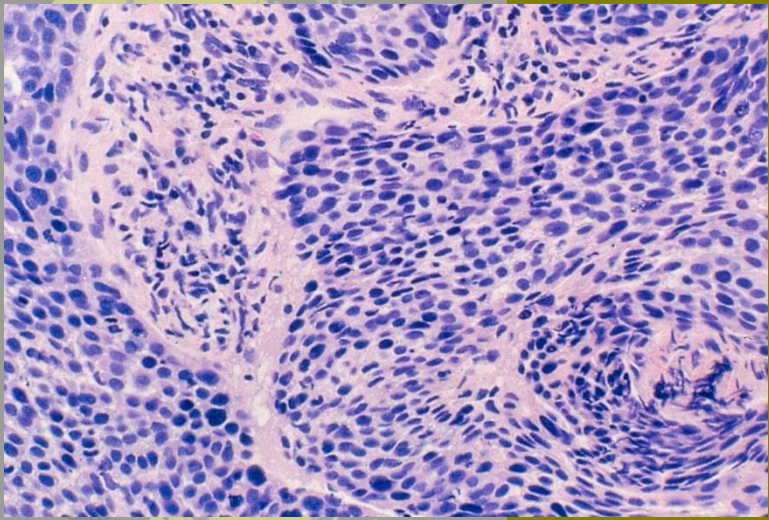
CERVICAL CARCINOMA – CARCINOMA PLANOEPITHELIALE (SCC)



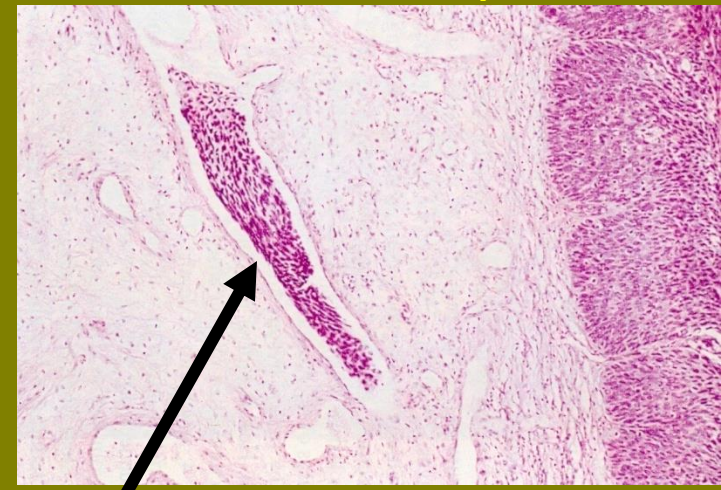
KERATINIZING SCC



KERATINIZING SCC (WITH LARGE CELLS)

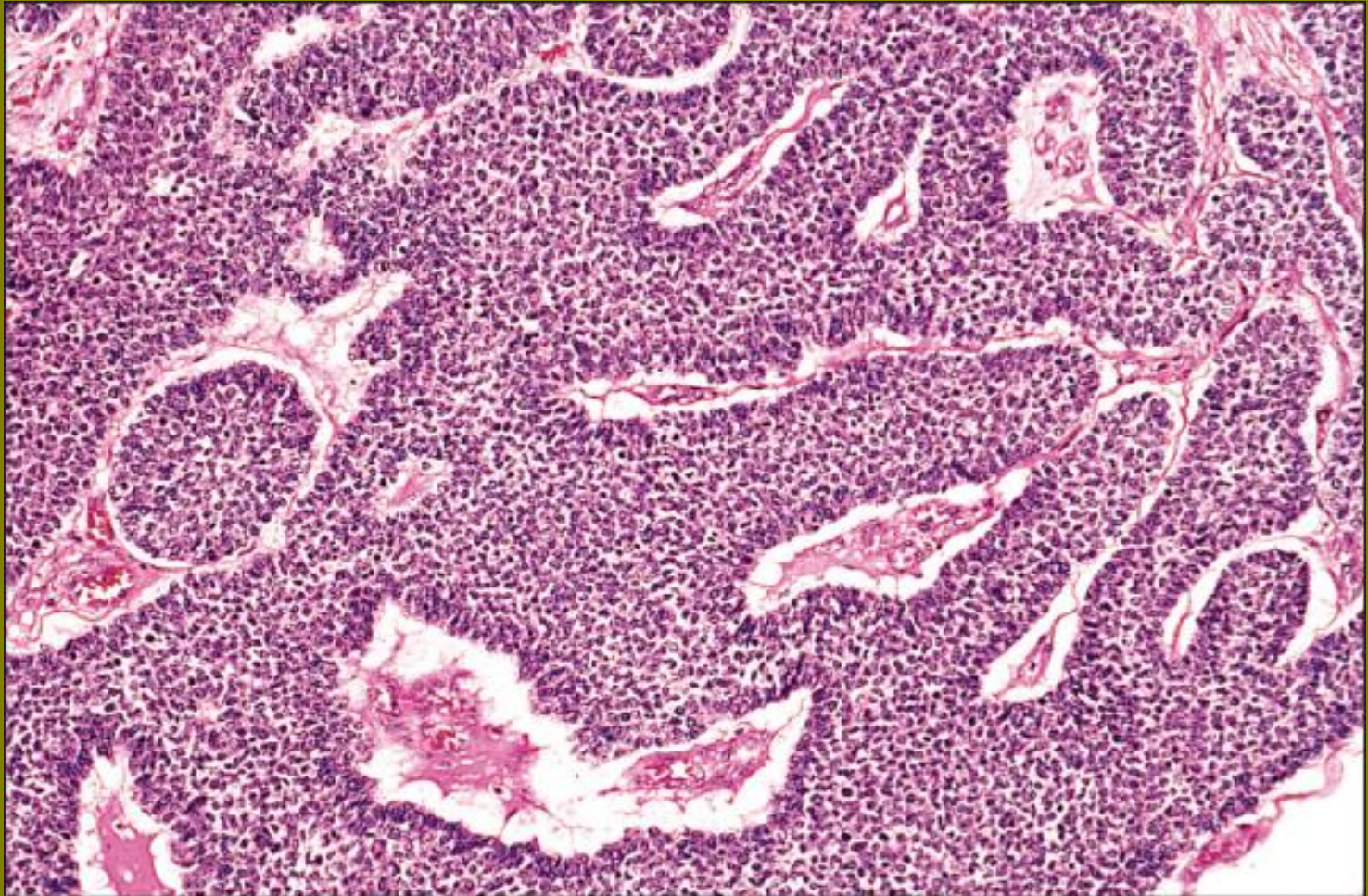


NON-KERATINIZING SCC (WITH SMALL CELLS)



**INVASION OF LYMPHATIC VESSELS
IN CERVICAL CANCER**

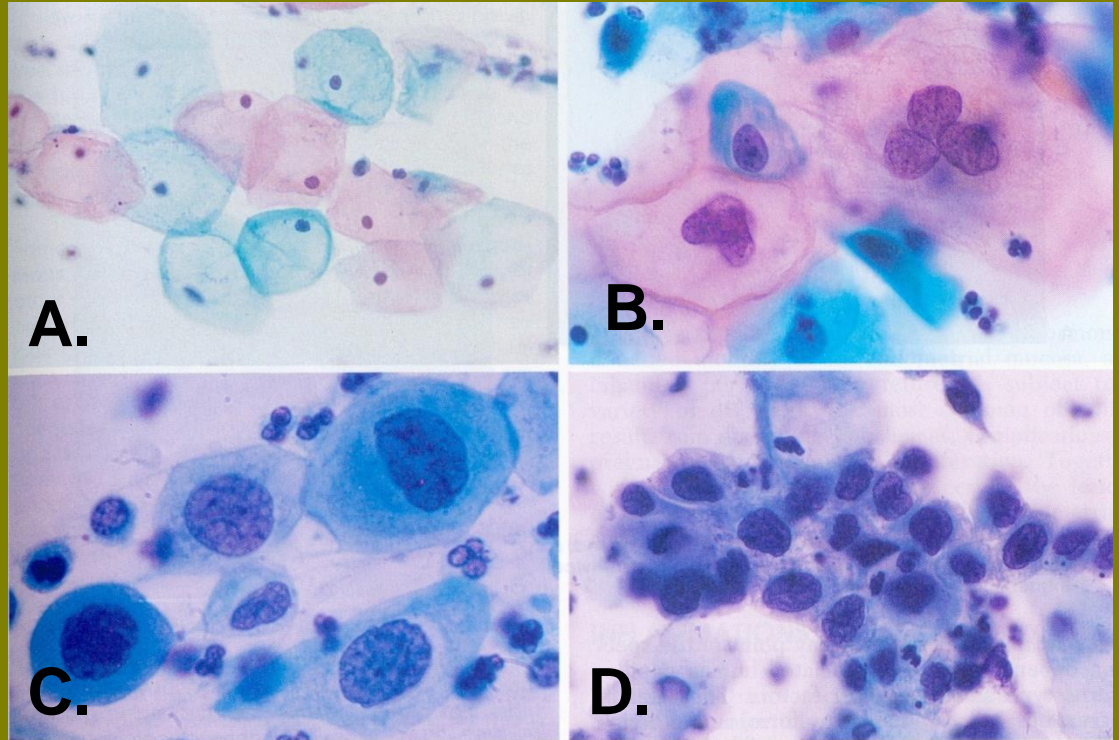
**Basaloid (small cell) squamous cell carcinoma.
The cells contain minimal cytoplasm.**



CYTOLOGY



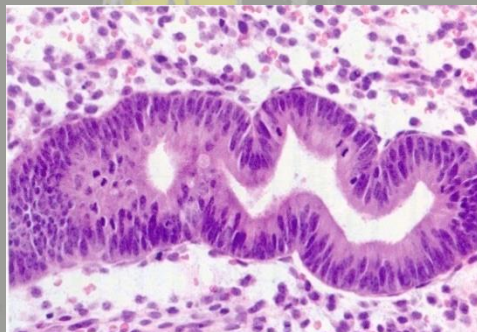
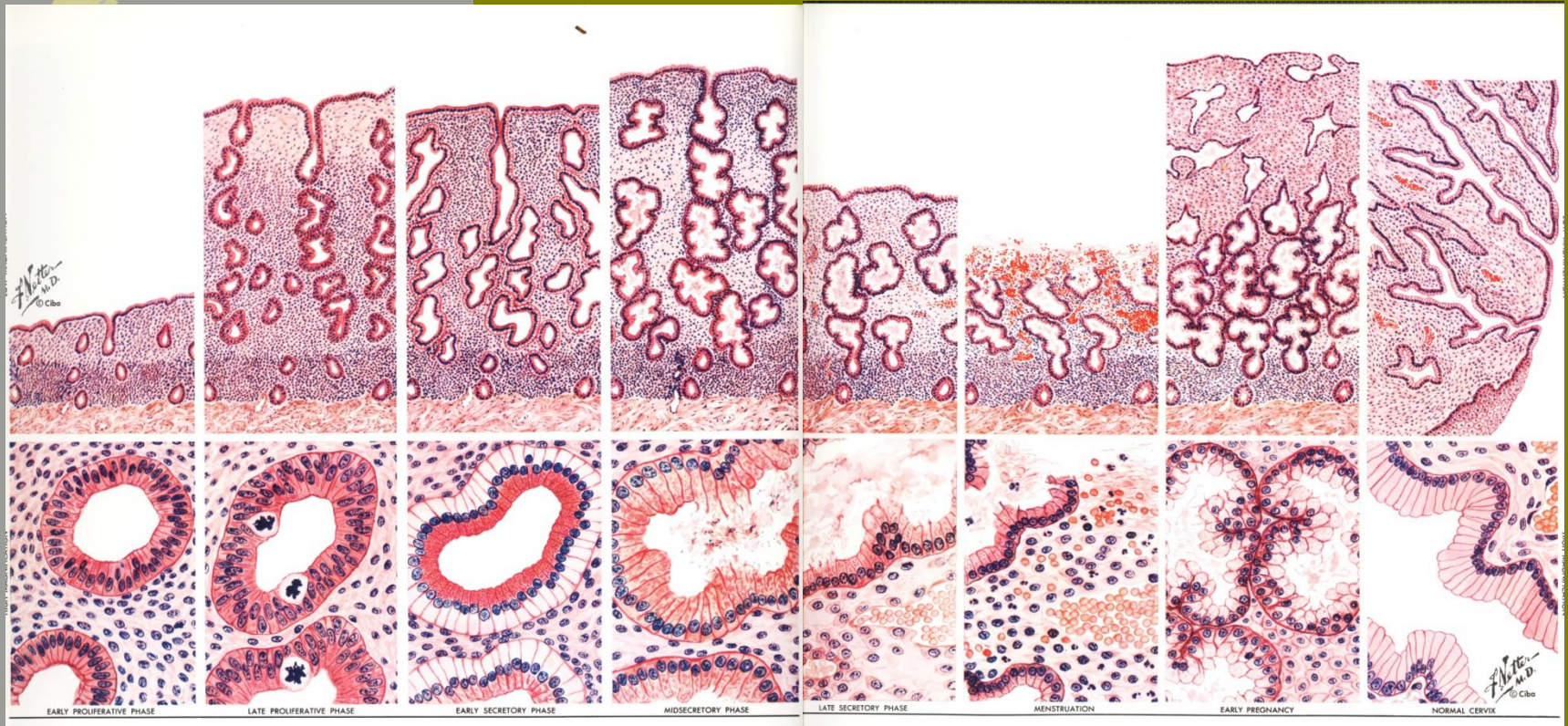
**INVENTOR OF CYTOLOGY
GEORGE NICOLAS PAPANICOLAOU
(1883-1962)**



- A. NORMAL SMEAR
- B. SMEAR WITH CIN 1
- C. SMEAR WITH CIN 2
- D. SMEAR WITH CIN 3

CIN = cervical intraepithelial neoplasia

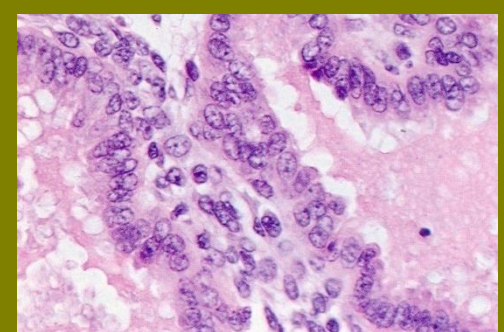
PHASES OF MENSTRUAL CYCLE – ENDOMETRIUM



PROLIFERATION



EARLY SECRETION



LATE SECRETION

BETHESDA SYSTEM

- **Based on workshop held in April / May 2001 at National Cancer Institute (JAMA 2002;287:2114)**
- **By 2003, was implemented by 85% of labs participating in College of American Pathologists' Interlaboratory Comparison Program in Cervicovaginal Cytology (Arch Pathol Lab Med 2004;128:1224)**

BETHESDA SYSTEM

- Interpretation/result
- **1. Negative for Intraepithelial Lesion or Malignancy (NILM):**
- **A. Organisms**
- **Trichomonas vaginalis**
- **Fungal organisms morphologically consistent with Candida species**
- **Shift in flora suggestive of bacterial vaginosis**
- **Bacteria morphologically consistent with Actinomyces species**
- **Cellular changes associated with Herpes simplex virus**

BETHESDA SYSTEM

- **B. Other nonneoplastic findings (optional to report, list is not inclusive)**
- **Reactive cellular changes associated with:**
 - **Inflammation (includes typical repair)**
 - **Radiation**
 - **Intrauterine contraceptive device (IUD)**
 - **Glandular cells status post hysterectomy**
 - **Atrophy**

BETHESDA SYSTEM

- **C. Other**
- **Endometrial cells (in a woman older than or equal to 40 years of age; specify if "negative for squamous intraepithelial lesion")**

BETHESDA SYSTEM

- **2. Epithelial Cell Abnormalities A. Squamous cell**
- **Atypical squamous cells: of undetermined significance (ASC-US); cannot exclude HSIL (ASC-H)**
- **Low grade squamous intraepithelial lesion (LSIL): encompassing HPV/mild dysplasia / CIN I**
- **High grade squamous intraepithelial lesion (HSIL): encompassing: moderate and severe dysplasia / CIN2 / CIN3 / CIS; with features suspicious for invasion (if invasion suspected)**
- **Squamous cell carcinoma**

BETHESDA SYSTEM

- **B. Glandular cell**

- **Atypical:**

Endocervical cells (NOS or specify in comment)

Endometrial cells (NOS or specify in comment)

Glandular cells (NOS or specify in comment)

BETHESDA SYSTEM

- **Atypical:**
- **Endocervical cells, favor neoplastic**
- **Glandular cells, favor neoplastic**
- **Endocervical Adenocarcinoma in situ**

BETHESDA SYSTEM

- **Adenocarcinoma:**

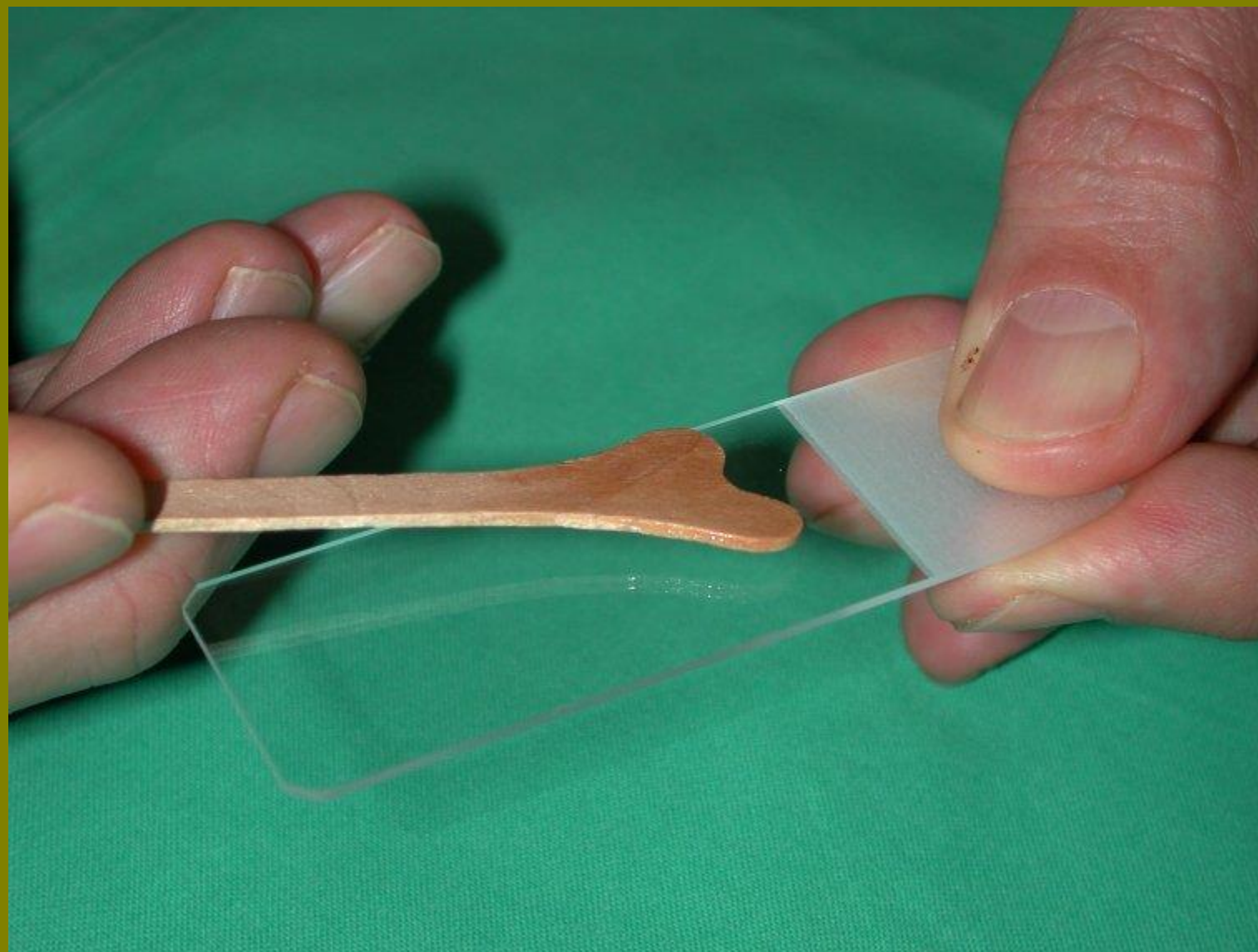
 - Endocervical**

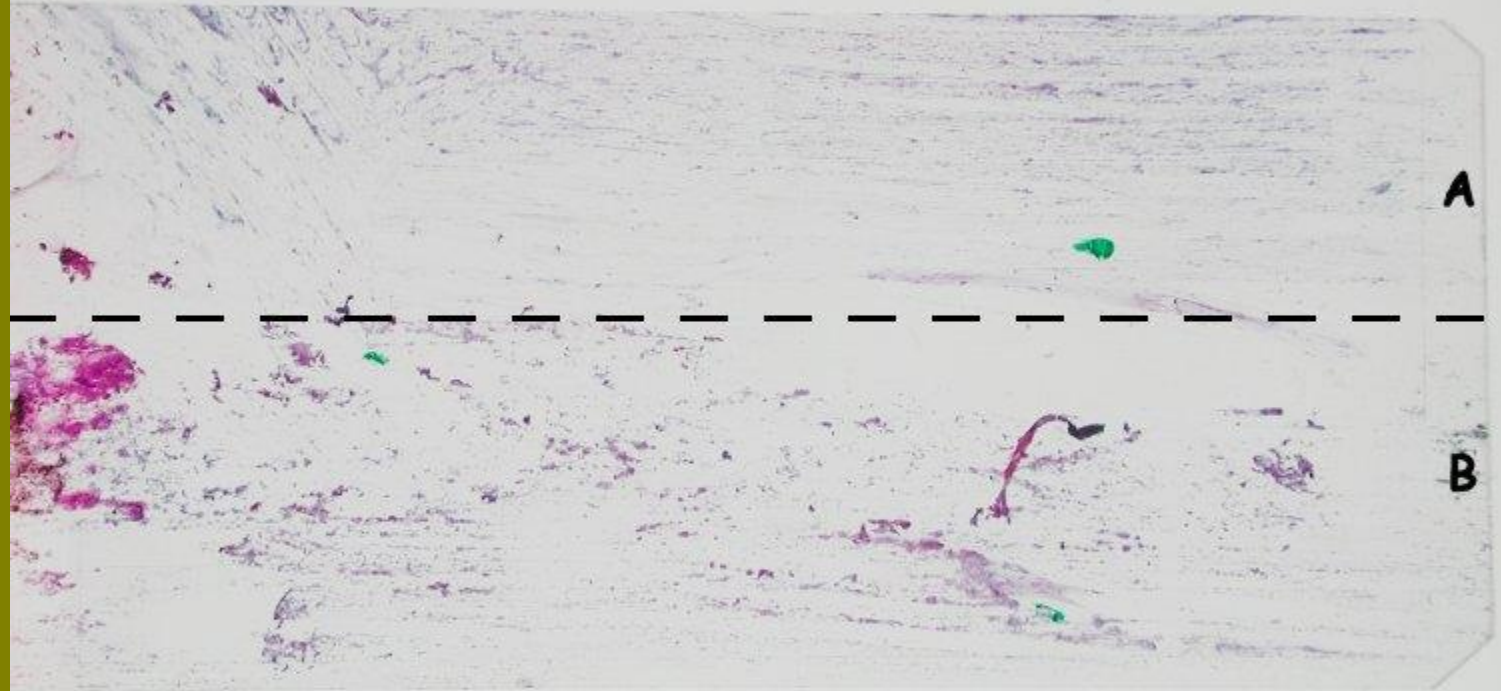
 - Endometrial**

 - Extrauterine**

 - Not otherwise specified (NOS)**

- C. Other malignant neoplasms (Specify)**

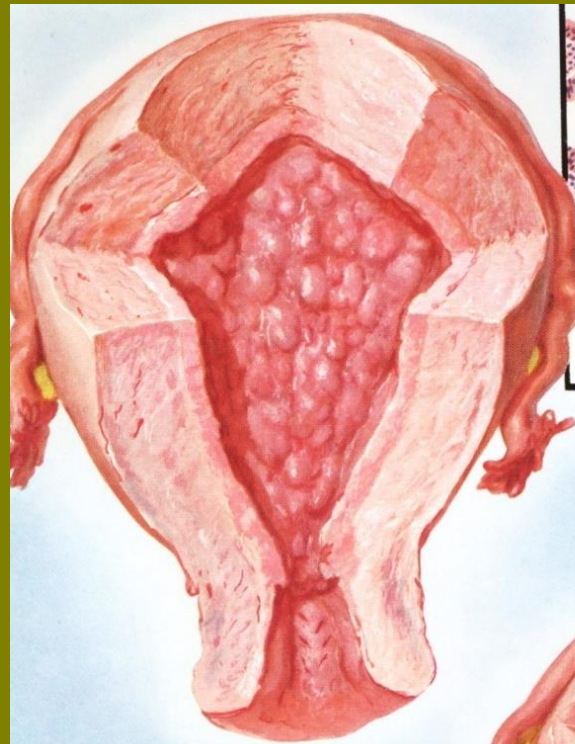
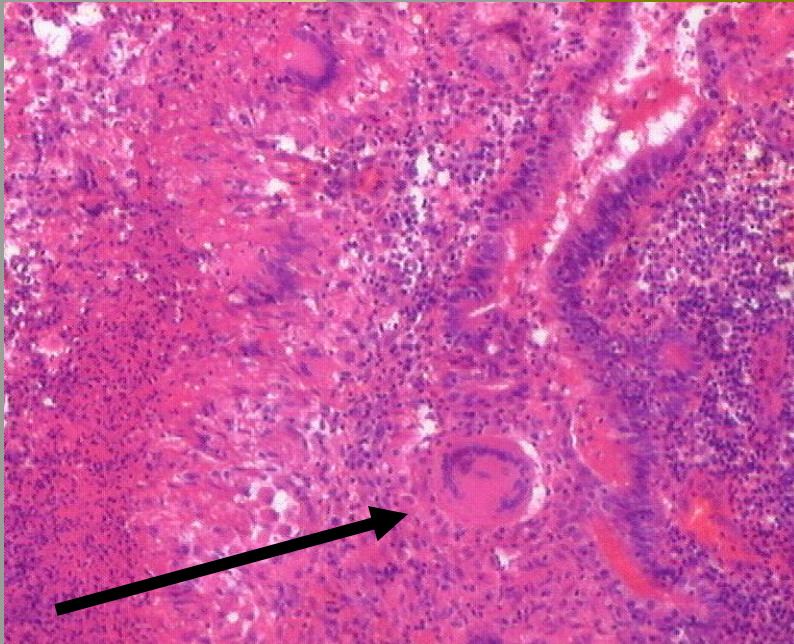




ENDOMETRITIS

NON-SPECIFIC INFLAMMATION

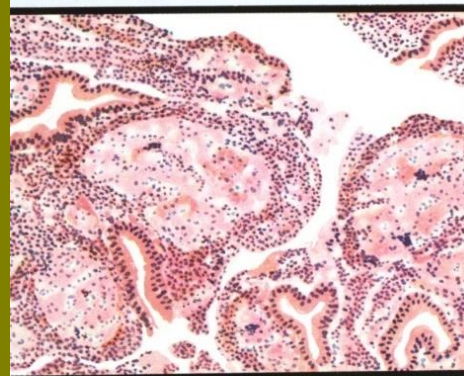
SPECIFIC INFLAMMATION



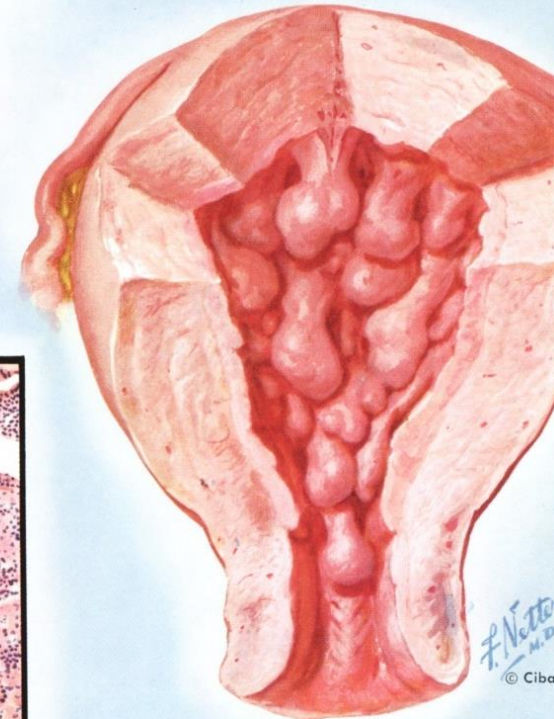
EXTENSIVE, DIFFUSE ENDOMETRIAL HYPERPLASIA WITH POLYPOID TENDENCY



ENDOMETRIAL HYPERPLASIA (MICROSCOPIC APPEARANCE)



TUBERCULOUS ENDOMETRITIS



MULTIPLE ENDOMETRIAL POLYPS

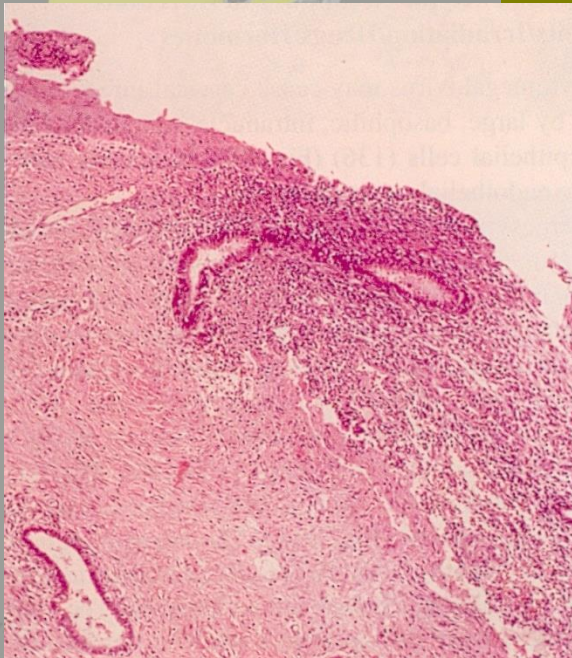
F. Netter M.D.
© Ciba

TUBERCULOSIS OF ENDOMETRIUM

ENDOMETRIOSIS



ENDOMETRIOSIS OF MYOMETRIUM (ADENOMYOSIS)



ENDOMETRIOSIS OF CERVIX

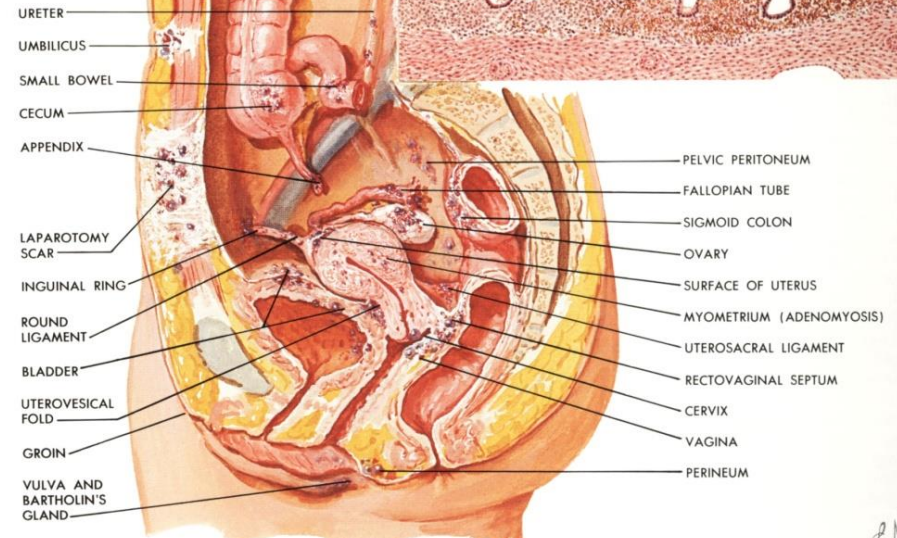
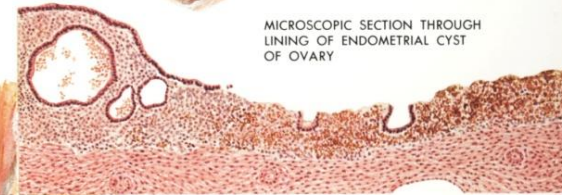
DIFFUSE PELVIC ENDOMETRIOSIS: RUPTURED ENDOMETRIAL (CHOCOLATE) CYST



HEMISECTION OF OVARY WITH ENDOMETRIAL CYSTS AND CORPUS LUTEUM

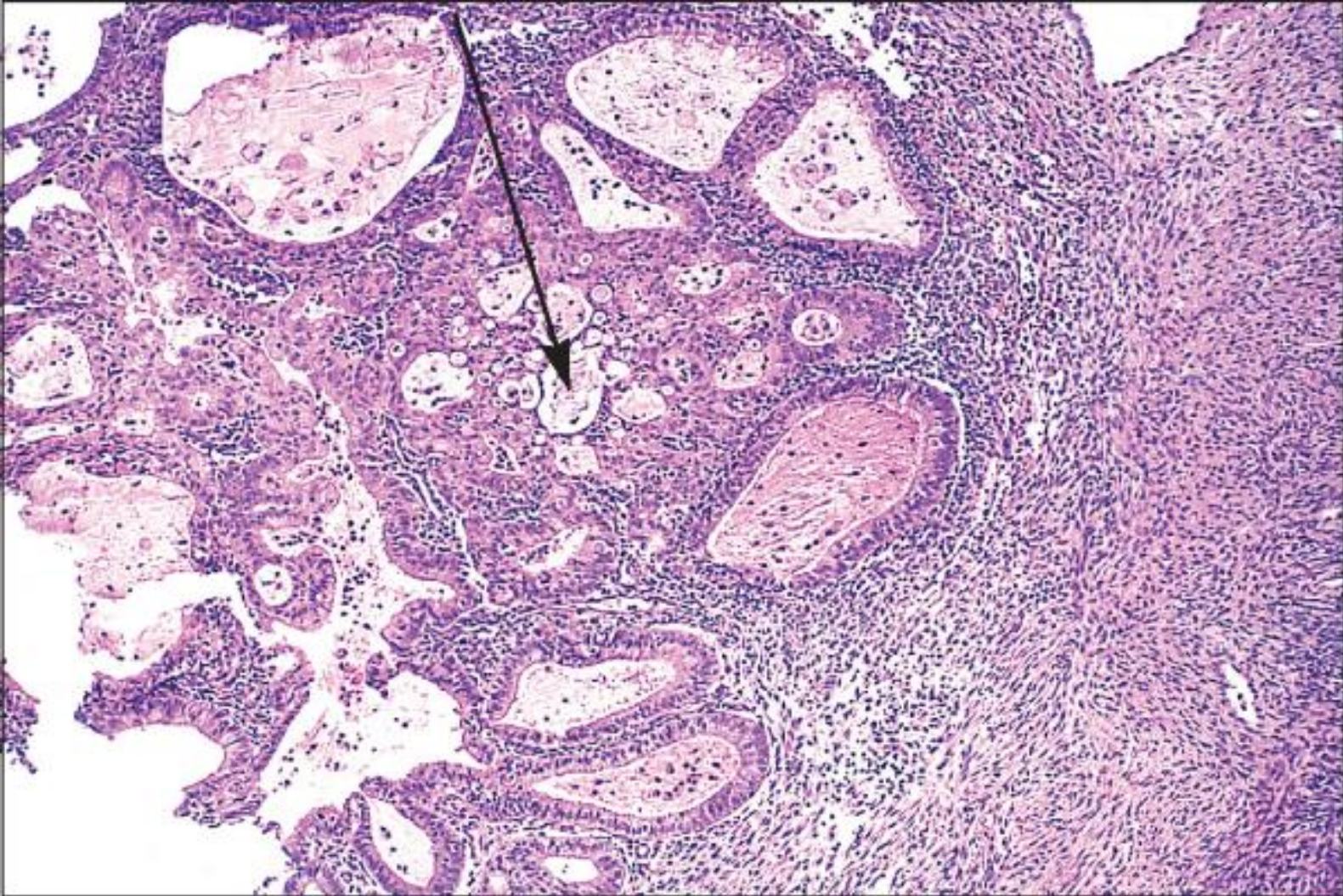


MICROSCOPIC SECTION THROUGH LINING OF ENDOMETRIAL CYST OF OVARY



POSSIBLE SITES OF DISTRIBUTION OF ENDOMETRIOSIS

**Endometrioid adenocarcinoma (arrow)
arising in atypical endometriosis.**



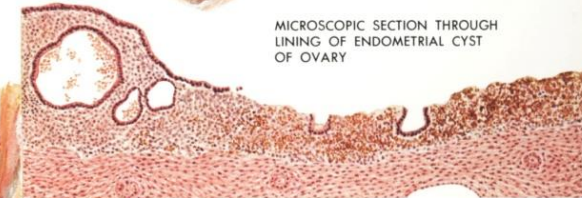
Clinical location of endometrial implants

Location	Frequency
Uterosacral ligaments	63%
Ovaries	
Superficial	56%
Deep (endometrioma)	20%
Ovarian fossae	33%
Anterior vesicle pouch	22%
Pouch of Douglas	19%
Intestines	5%
Fallopian tubes	5%
Uterus	5%

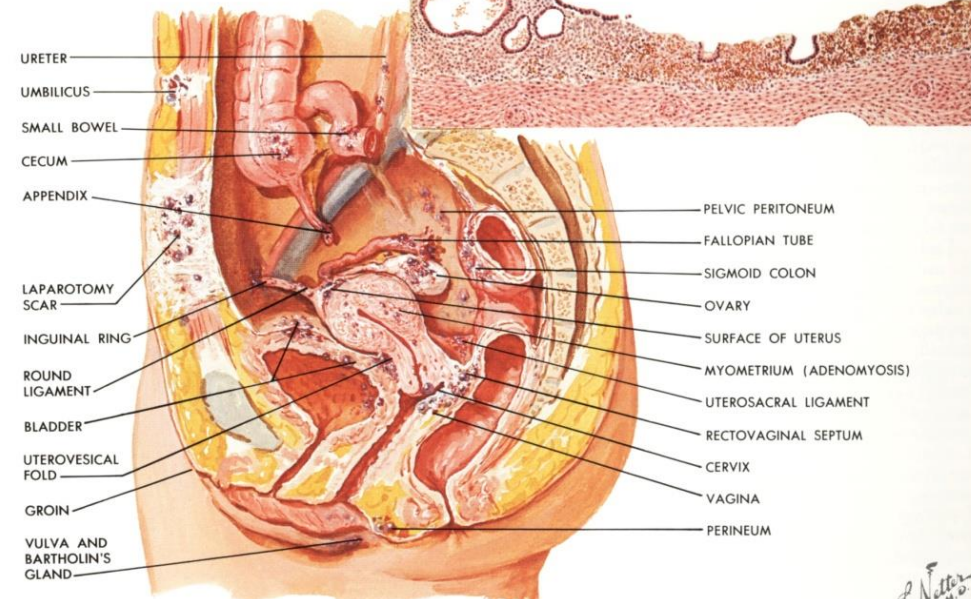
DIFFUSE PELVIC ENDOMETRIOSIS: RUPTURED ENDOMETRIAL (CHOCOLATE) CYST



HEMISECTION OF OVARY WITH ENDOMETRIAL CYSTS AND CORPUS LUTEUM



MICROSCOPIC SECTION THROUGH LINING OF ENDOMETRIAL CYST OF OVARY



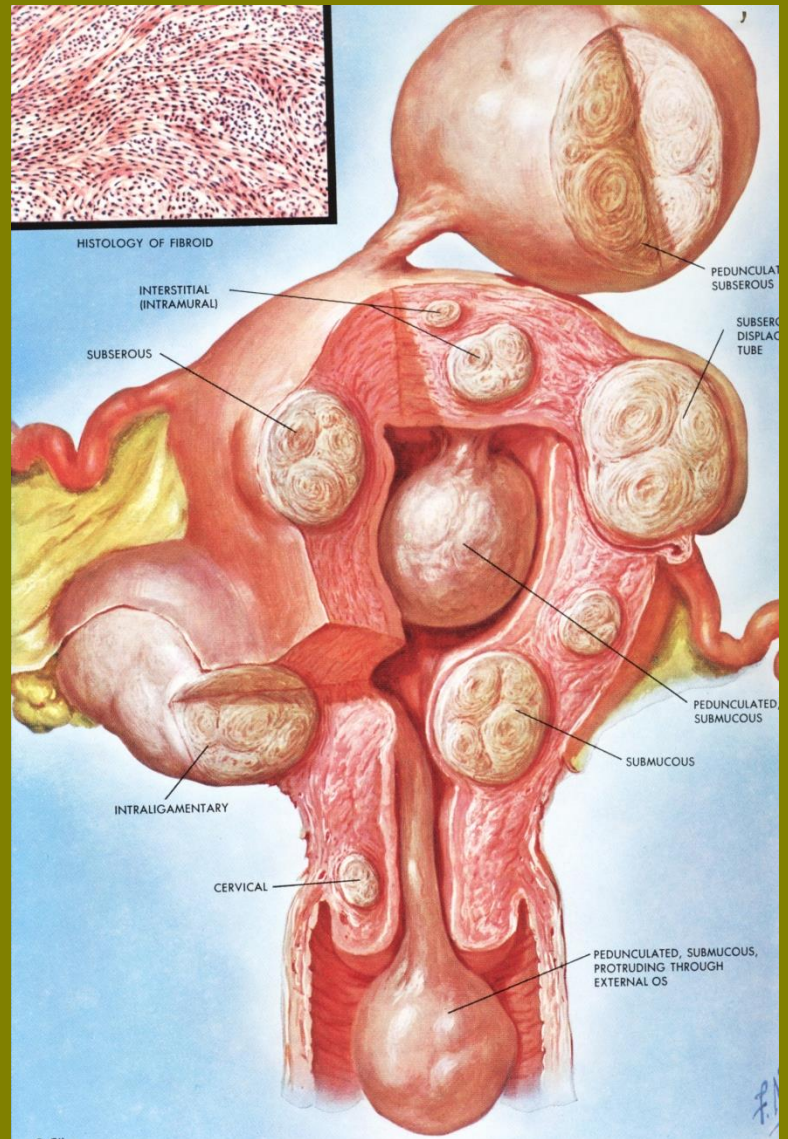
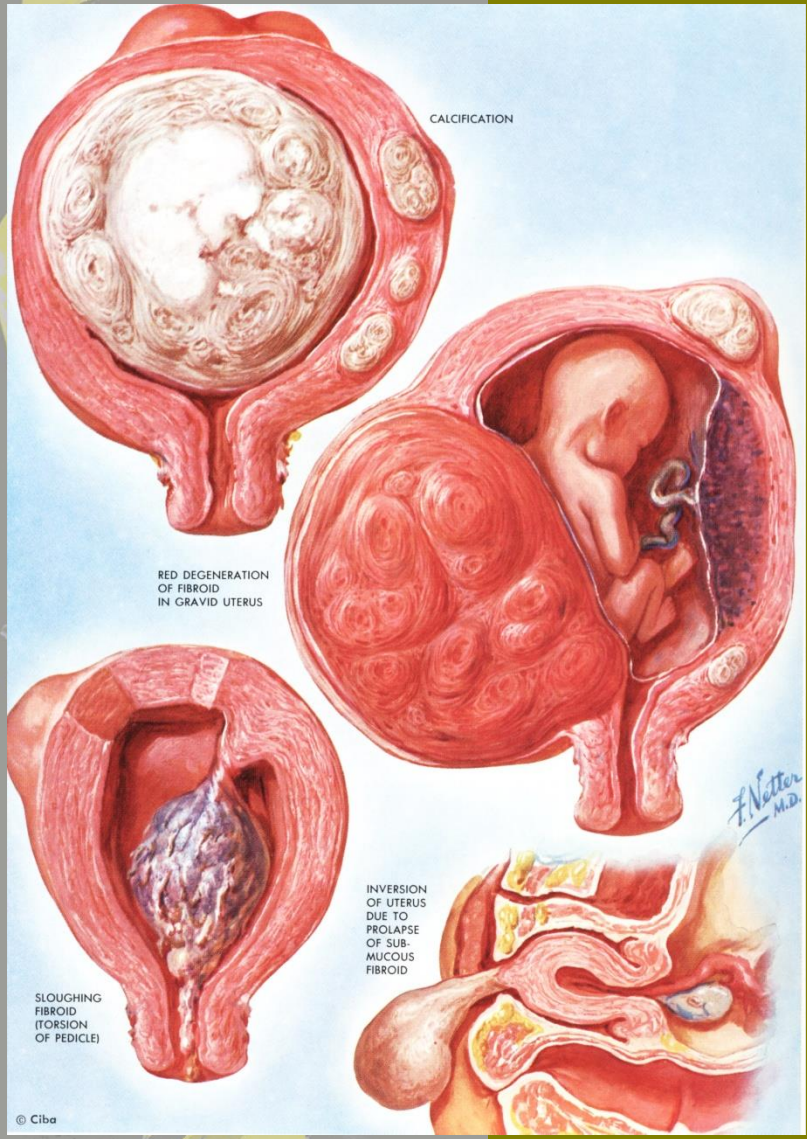
POSSIBLE SITES OF DISTRIBUTION OF ENDOMETRIOSIS

Location of endometriosis based upon biopsy findings

Location	Frequency
Ovary	36%
Fallopian tube	14%
Uterine serosa	12%
Cul-de-sac	6%
Cervix	3%
Colon	3%
Peritoneum	3%
Appendix	2%
Broad ligament	2%
Pelvis	2%
Uterosacral ligament	2%
Vagina	2%
Abdominal wall	1%
Bladder	1%
Fibrous tissue	1%
Parametrium	1%
Rectum	1%
Small intestine	1%
Other sites (>20)	7%

END OF PART I

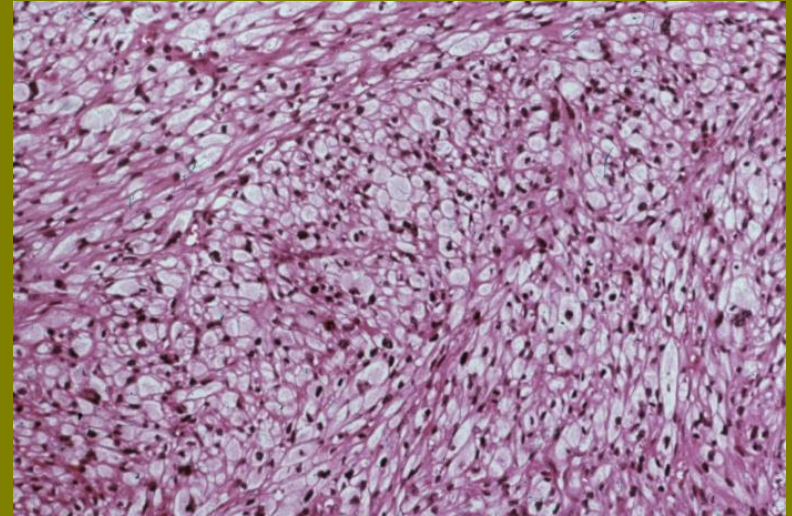
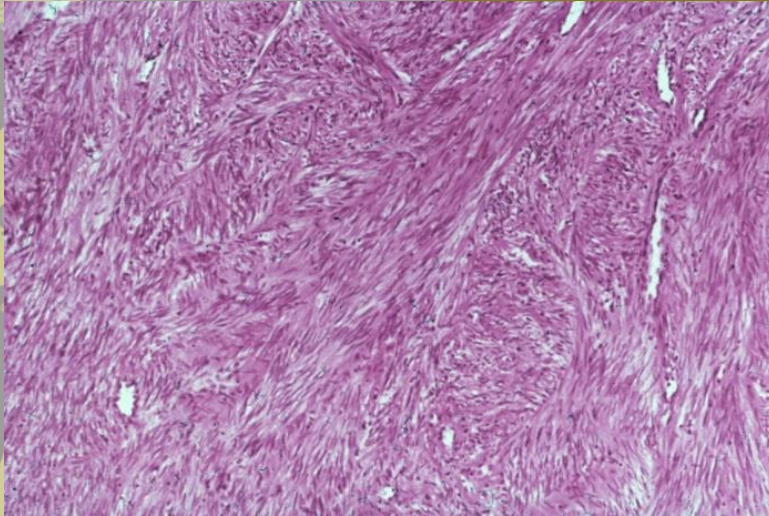
LEIOMYOMAS



MOST COMMON LOCALIZATION OF LEIOMYOMA IN THE UTERUS

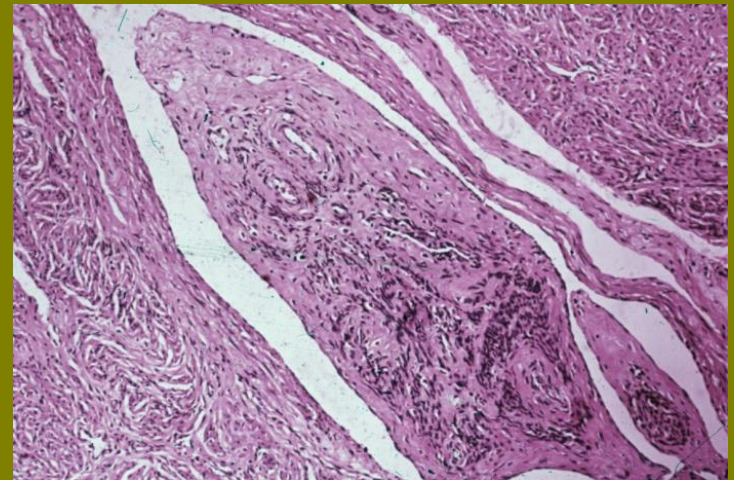
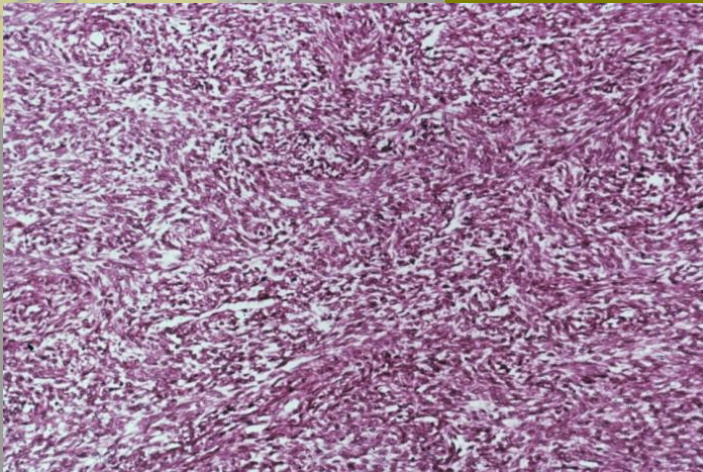
LEIOMYOMA

HISTOLOGICAL FORMS



LEIOMYOMA

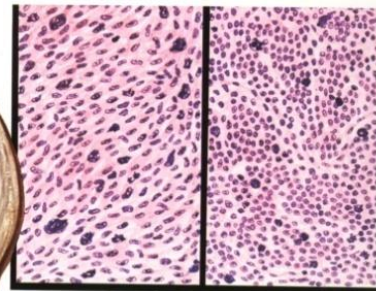
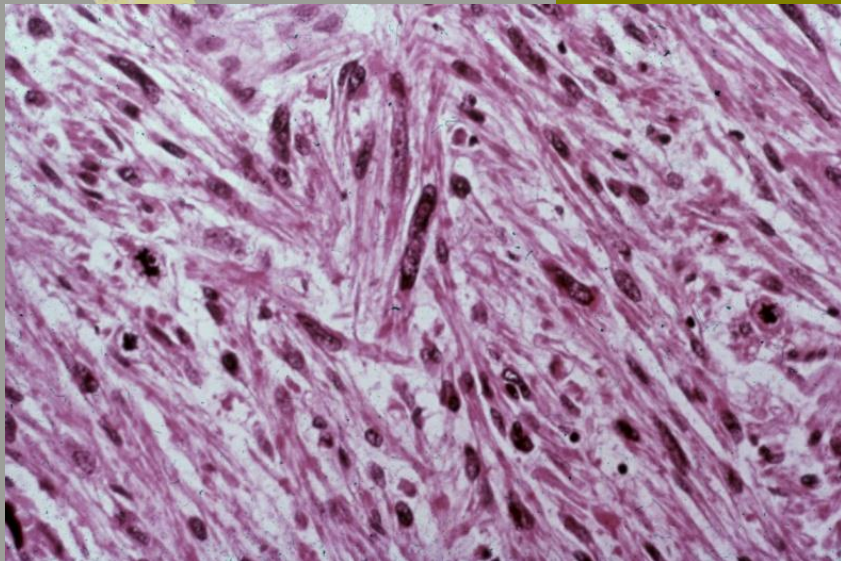
LEIOMYOBLASTOMA



CELLULAR LEIOMYOMA

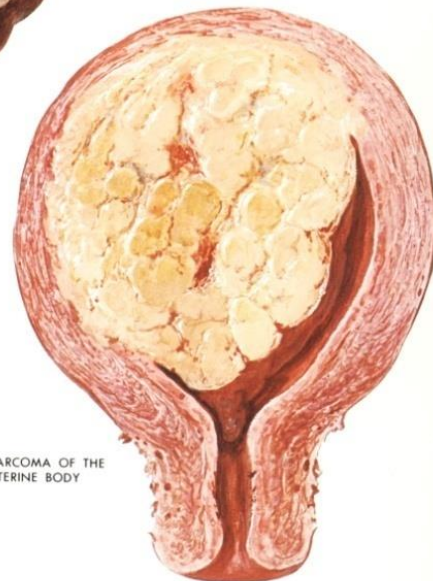
INTRAVASCULAR LEIOMYOMATOSIS

LEIOMYOSARCOMA

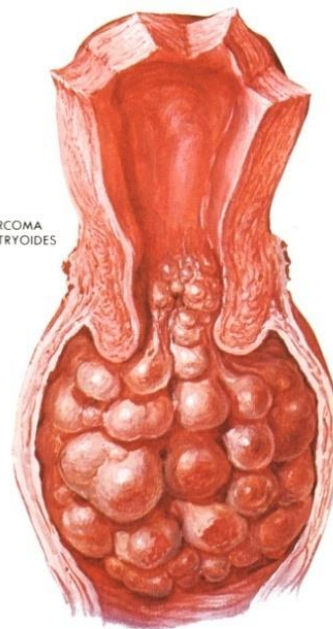


SPINDLE-CELL SARCOMA ROUND-CELL SARCOMA

SARCOMA IN A FIBROID



SARCOMA OF THE UTERINE BODY

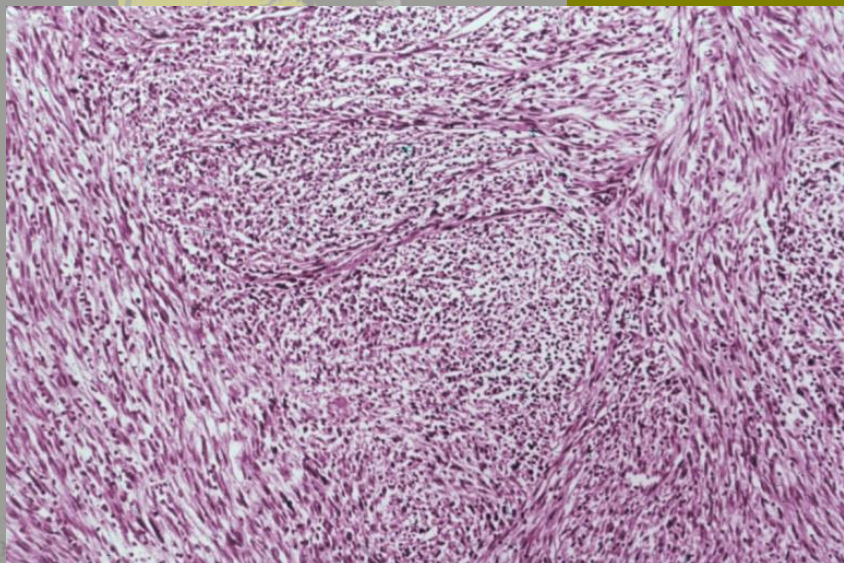


SARCOMA BOTRYOIDES

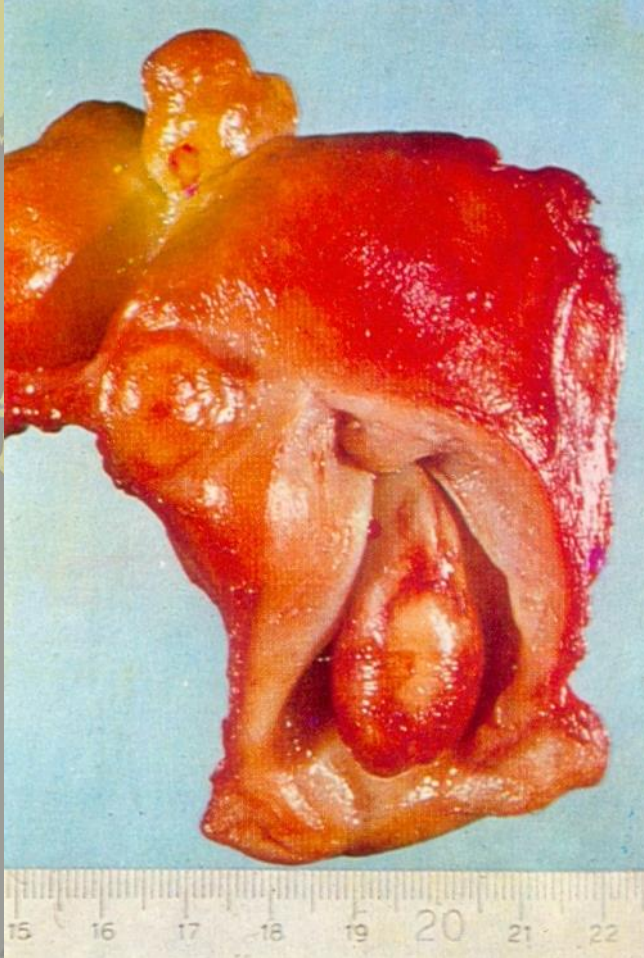


SARCOMATOUS POLYP PROLAPSING THROUGH CERVIX

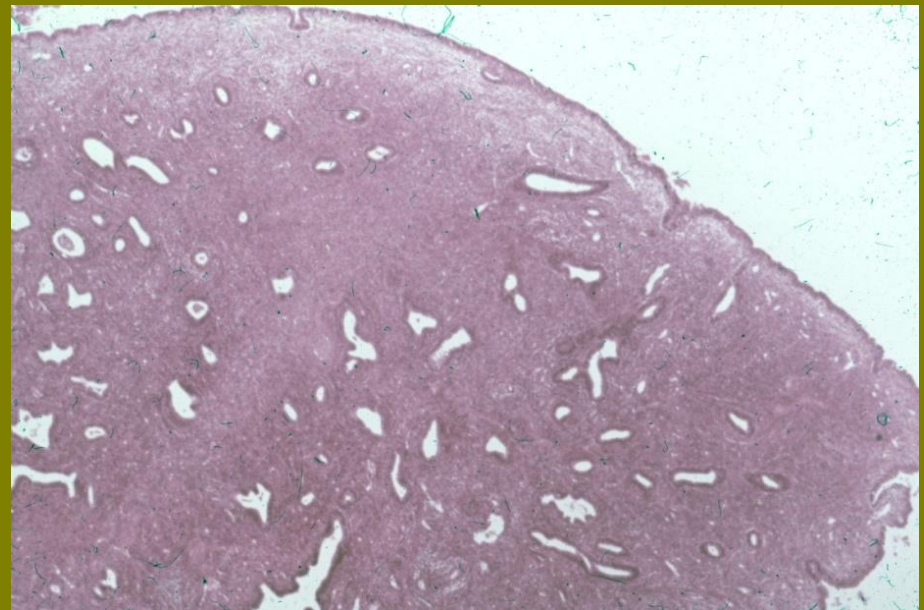
LEIOMYOSARCOMA



ENDOMETRIAL POLYP

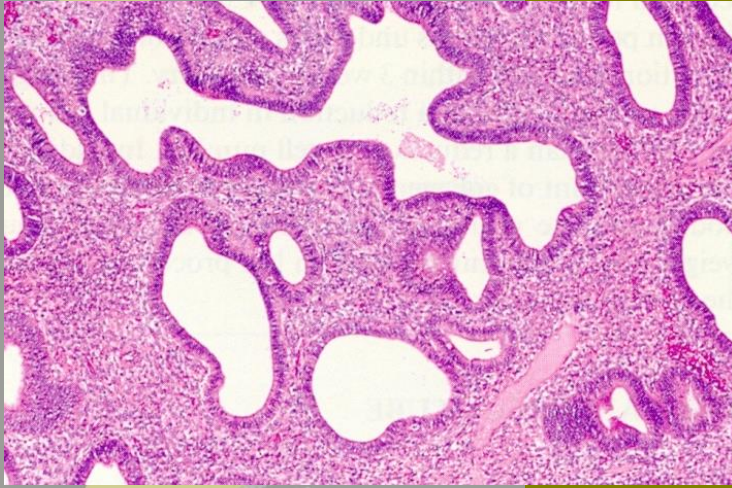


CYSTIC ENDOMETRIAL POLYP

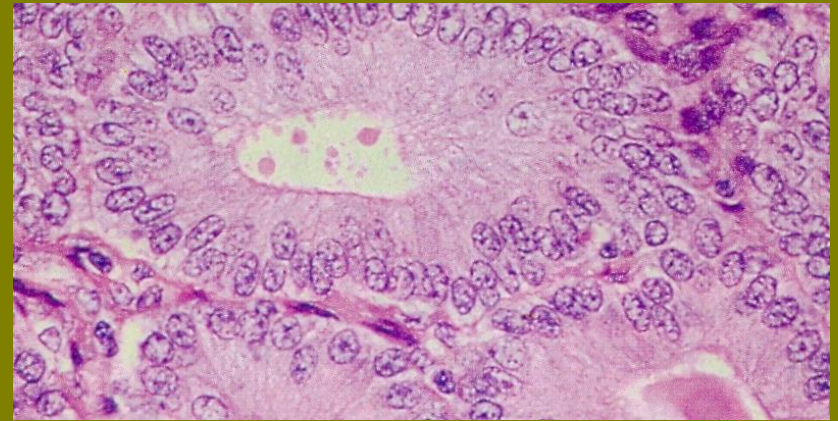


ENDOMETRIAL GLANDULAR POLYP

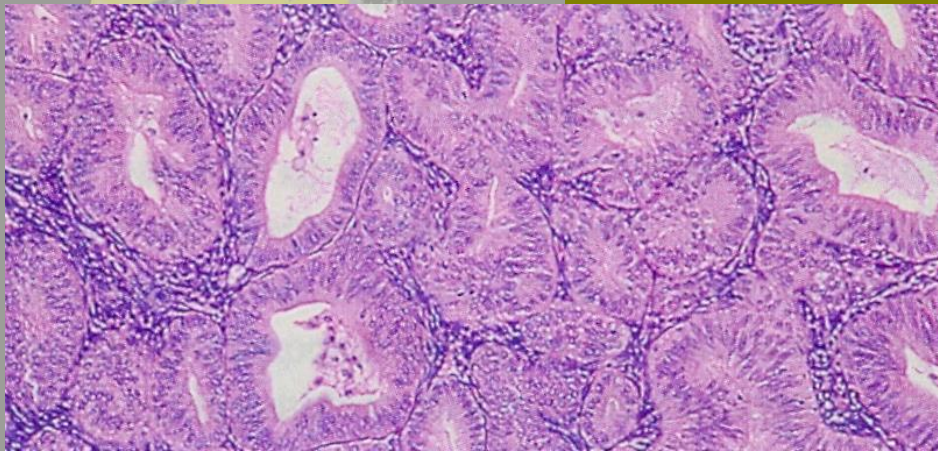
ENDOMETRIAL HYPERPLASIA



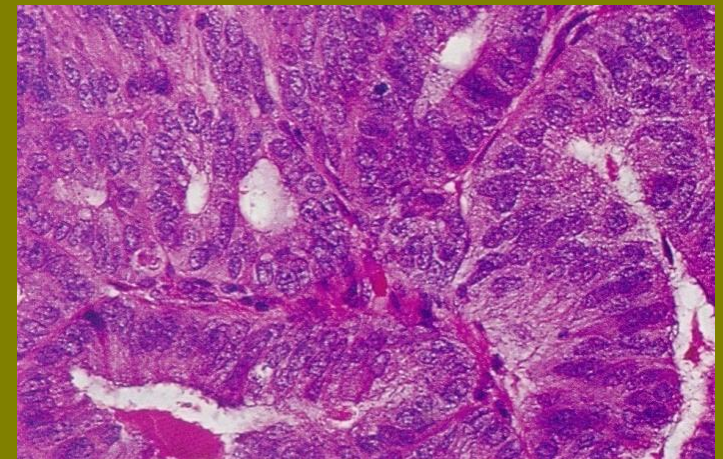
SIMPLE HYPERPLASIA WITHOUT ATYPIA



SIMPLE HYPERPLASIA WITH ATYPIA



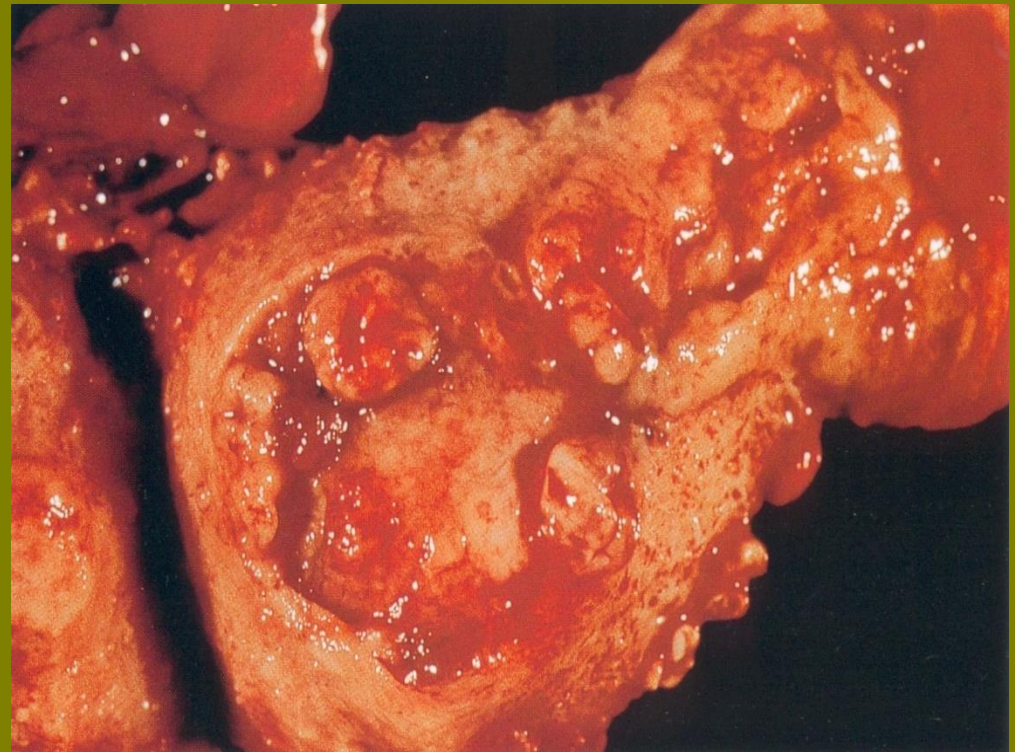
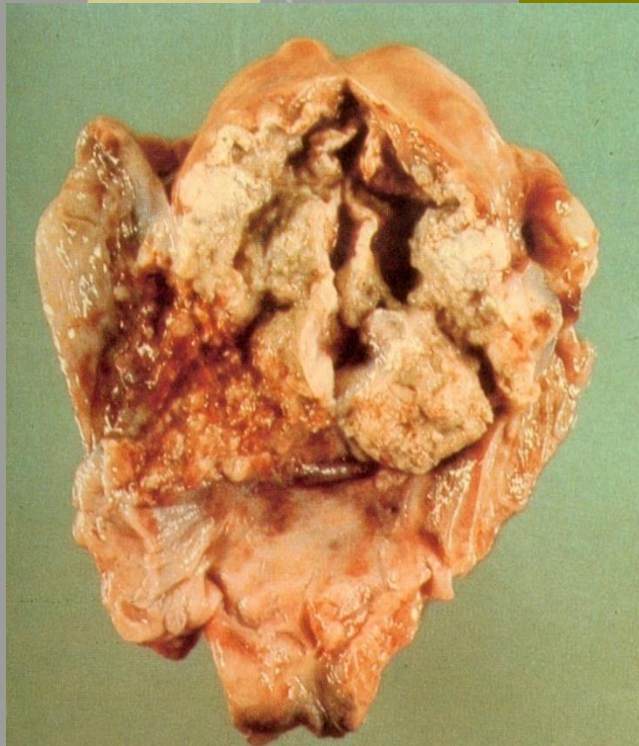
COMPLEX HYPERPLASIA WITHOUT ATYPIA



COMPLEX HYPERPLASIA WITH ATYPIA

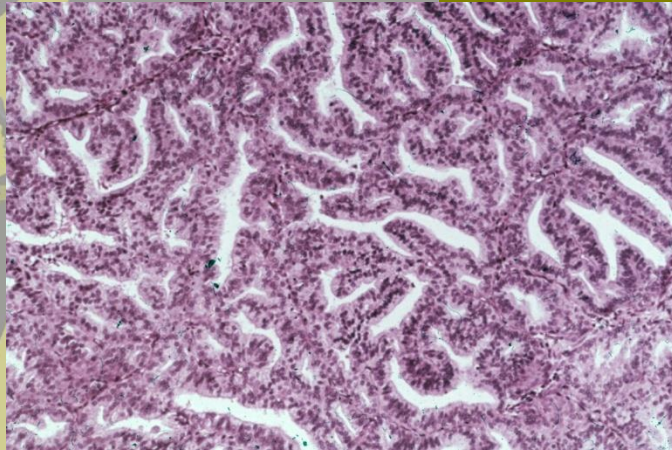
ENDOMETRIAL CARCINOMA

APPROX. 7% OF MALIGNANT TUMORS IN WOMEN. MORE FREQUENT THAN CERVICAL CANCER. RARE BEFORE 40, MOST COMMONLY BETWEEN THE 55TH AND 65TH YEAR OF LIFE. INVOLVED WITH THE OVERPRODUCTION OF ESTROGEN; OTHER FACTORS INVOLVED:
1. OBESITY 2. DIABETES 3. HYPERTENSION 4. LACK OF PREGNANCY.

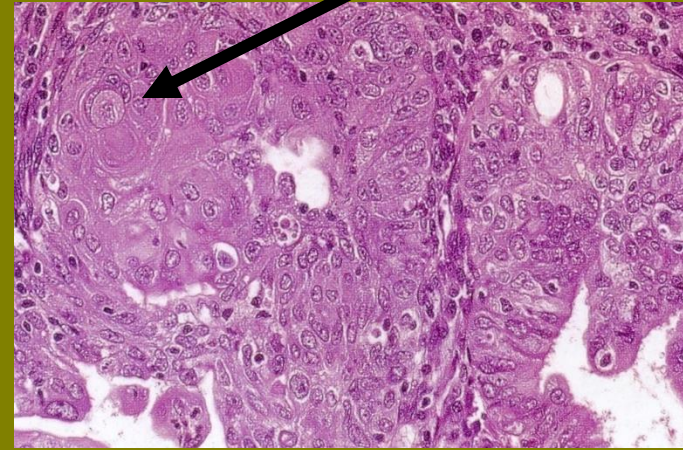


ENDOMETRIAL CARCINOMA

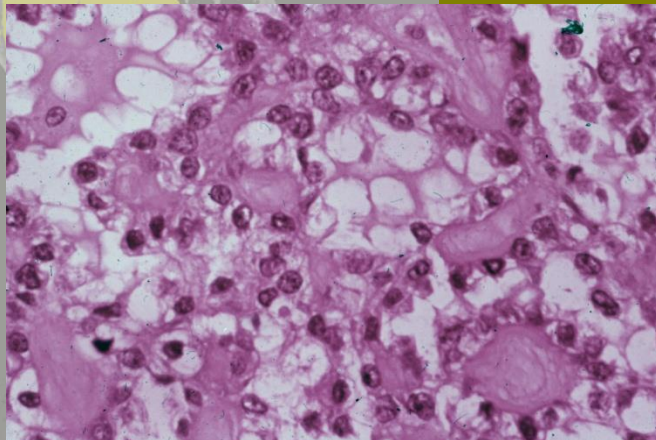
HISTOLOGICAL FORMS



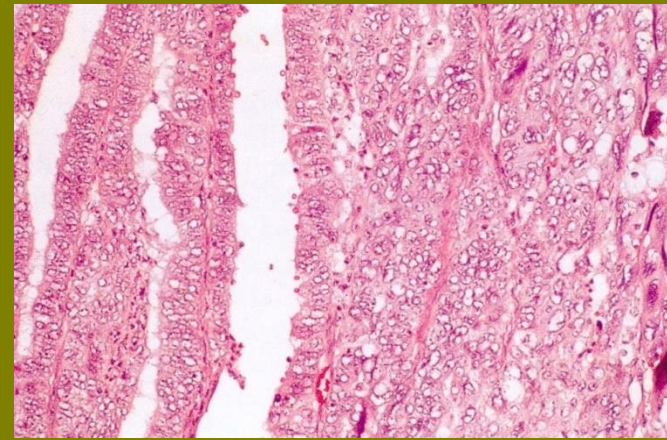
**WELL-DIFFERENTIATED
CARCINOMA**



ADENOACANTHOMA



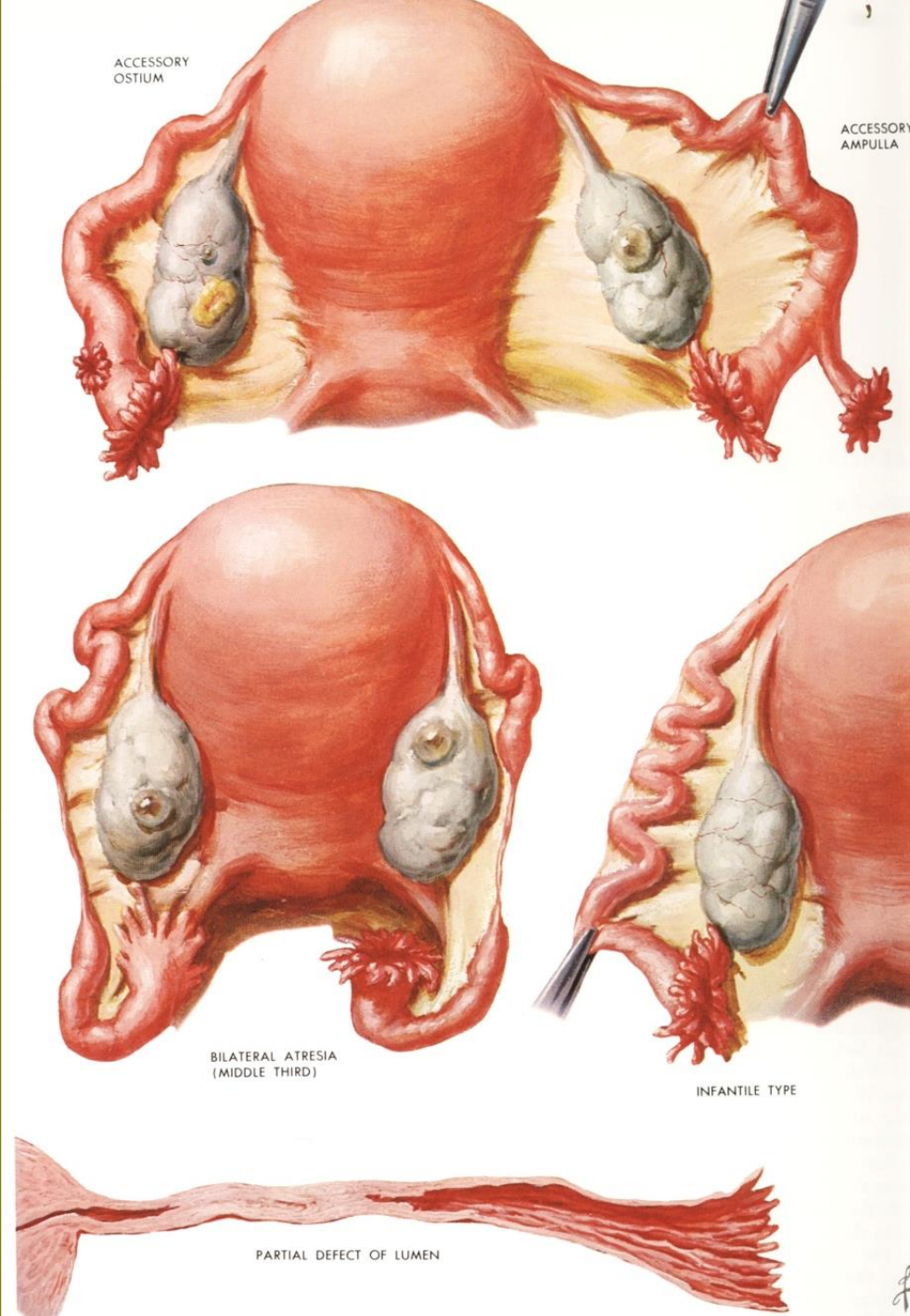
**CLEAR CELL CARCINOMA
(MESONEPHROID)**



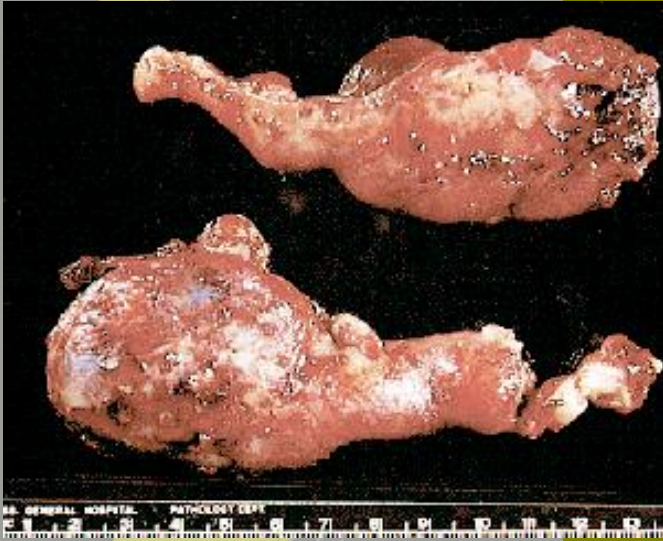
POORLY-DIFFERENTIATED CARCINOMA

PATHOLOGY OF OVIDUCTS; FALLOPIAN TUBES

DEVELOPMENTAL DISTURBANCES



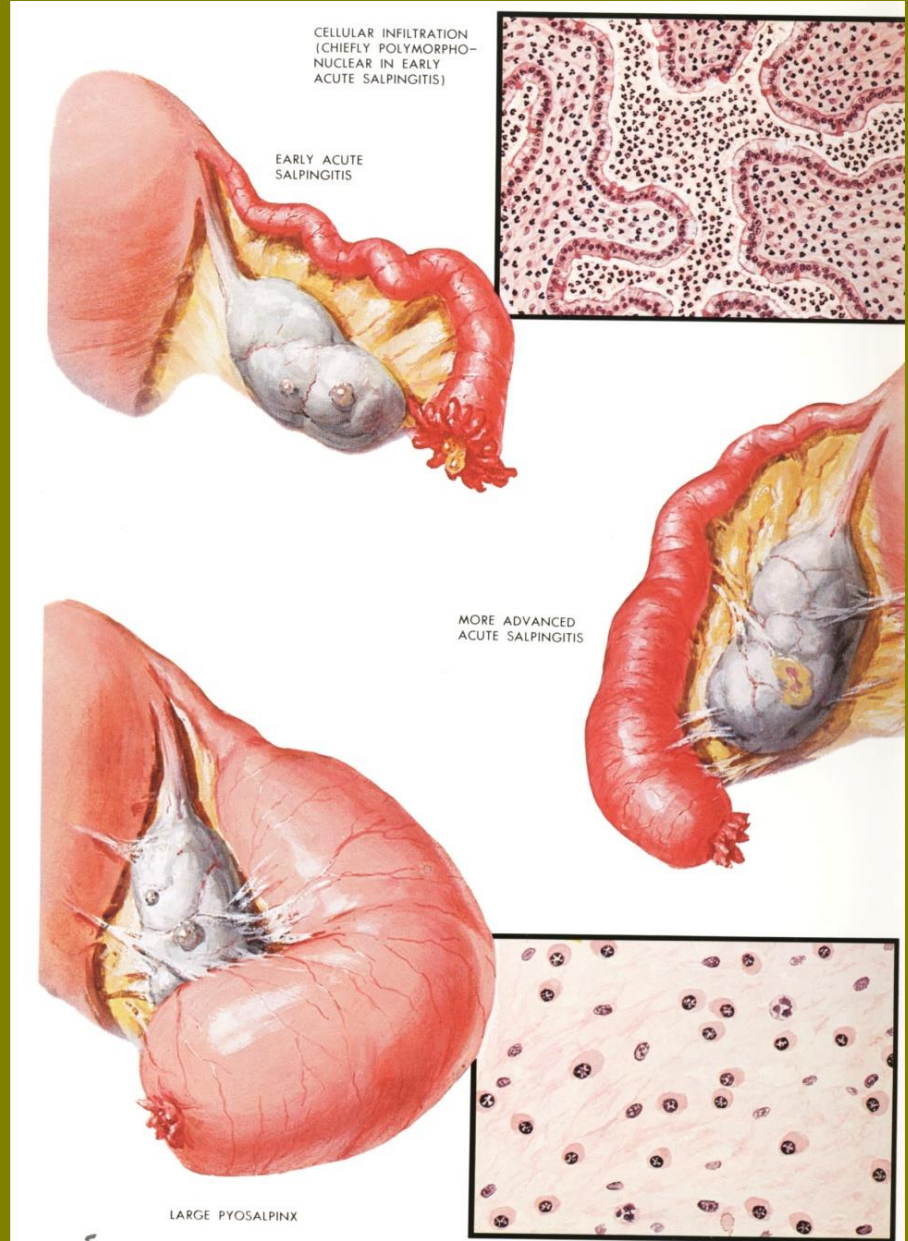
PATHOLOGY OF OVIDUCTS



ACUTE SALPINGITIS

PYOSALPINX

SACTOSALPINX



CELLULAR INFILTRATION
(CHIEFLY POLYMORPHO-
NUCLEAR IN EARLY
ACUTE SALPINGITIS)

EARLY ACUTE
SALPINGITIS

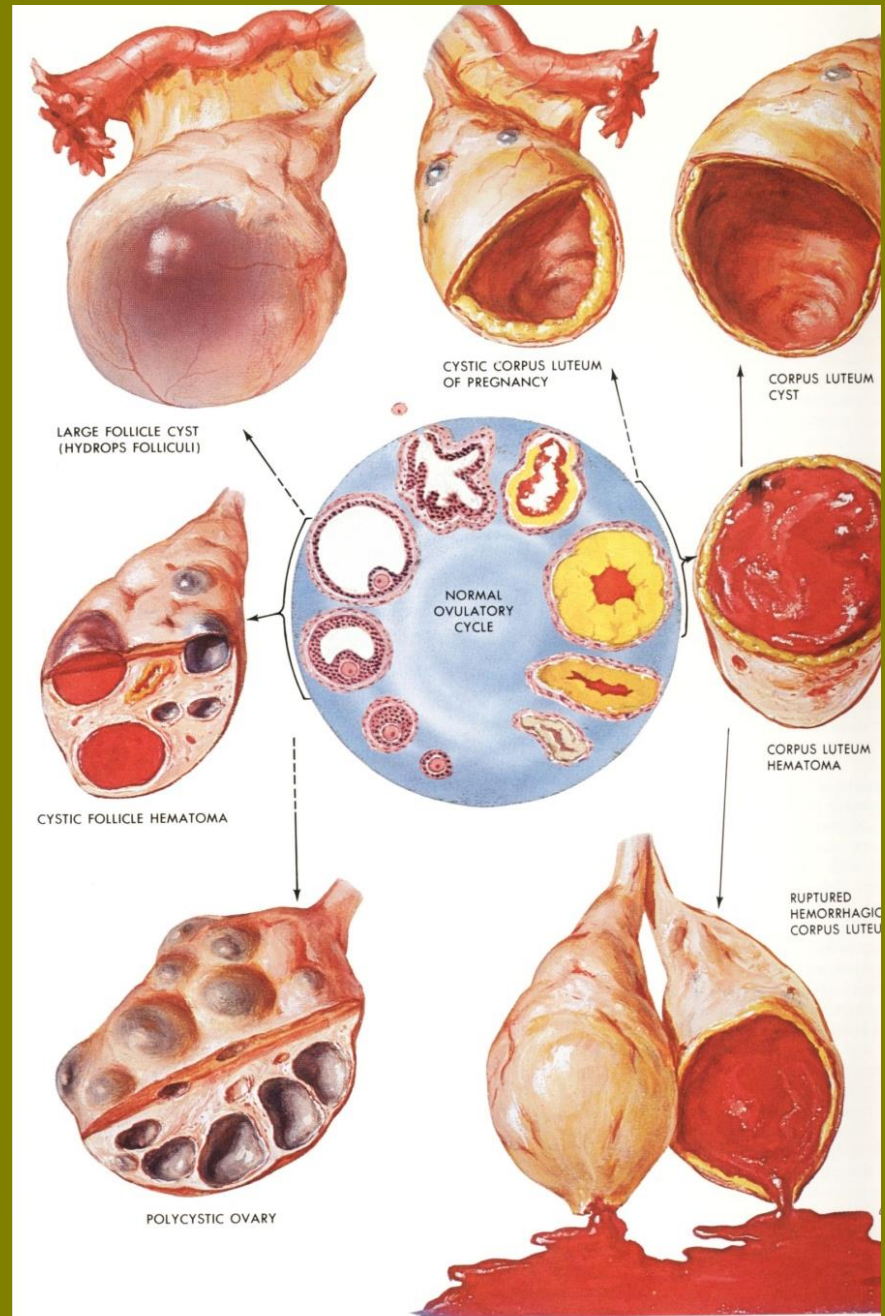
MORE ADVANCED
ACUTE SALPINGITIS

LARGE PYOSALPINX

PLASMA CELL INFILTRATION
CHARACTERISTIC OF SUBACUTE
AND CHRONIC SALPINGITIS

F. Netter
M.D.
© CIBA

PATHOLOGY OF OVARIES



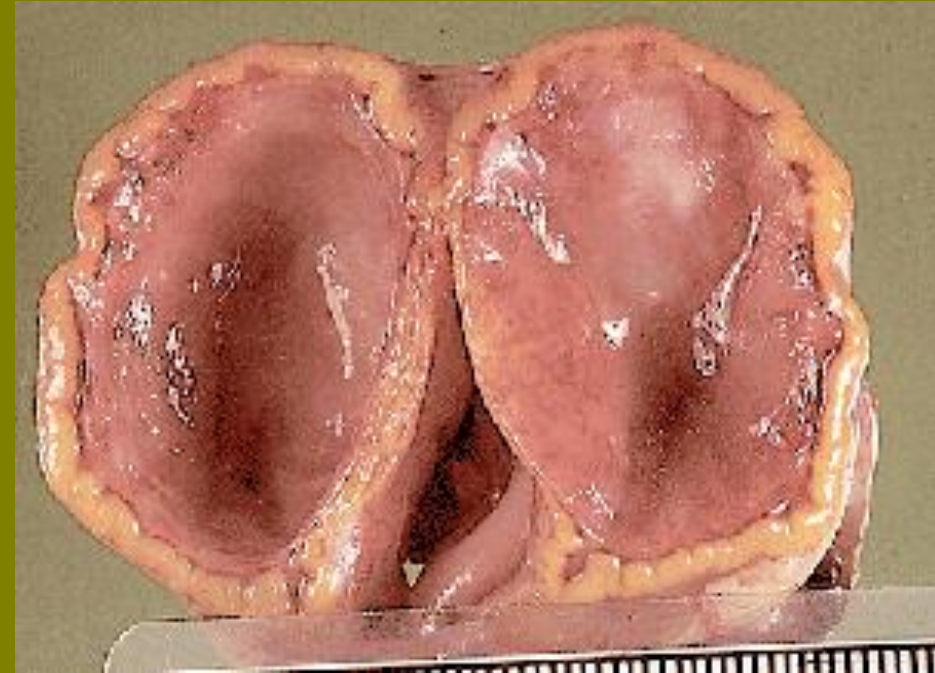
**CYSTIC CHANGES IN OVARIES !
SOME NORMAL PARTS OF
OVARY ARE CYSTIC BUT THEY
TEND TO INCREASE
SIGNIFICANTLY IN SIZE**

PATHOLOGY OF OVARIES

CYSTS



FOLLICULAR CYST



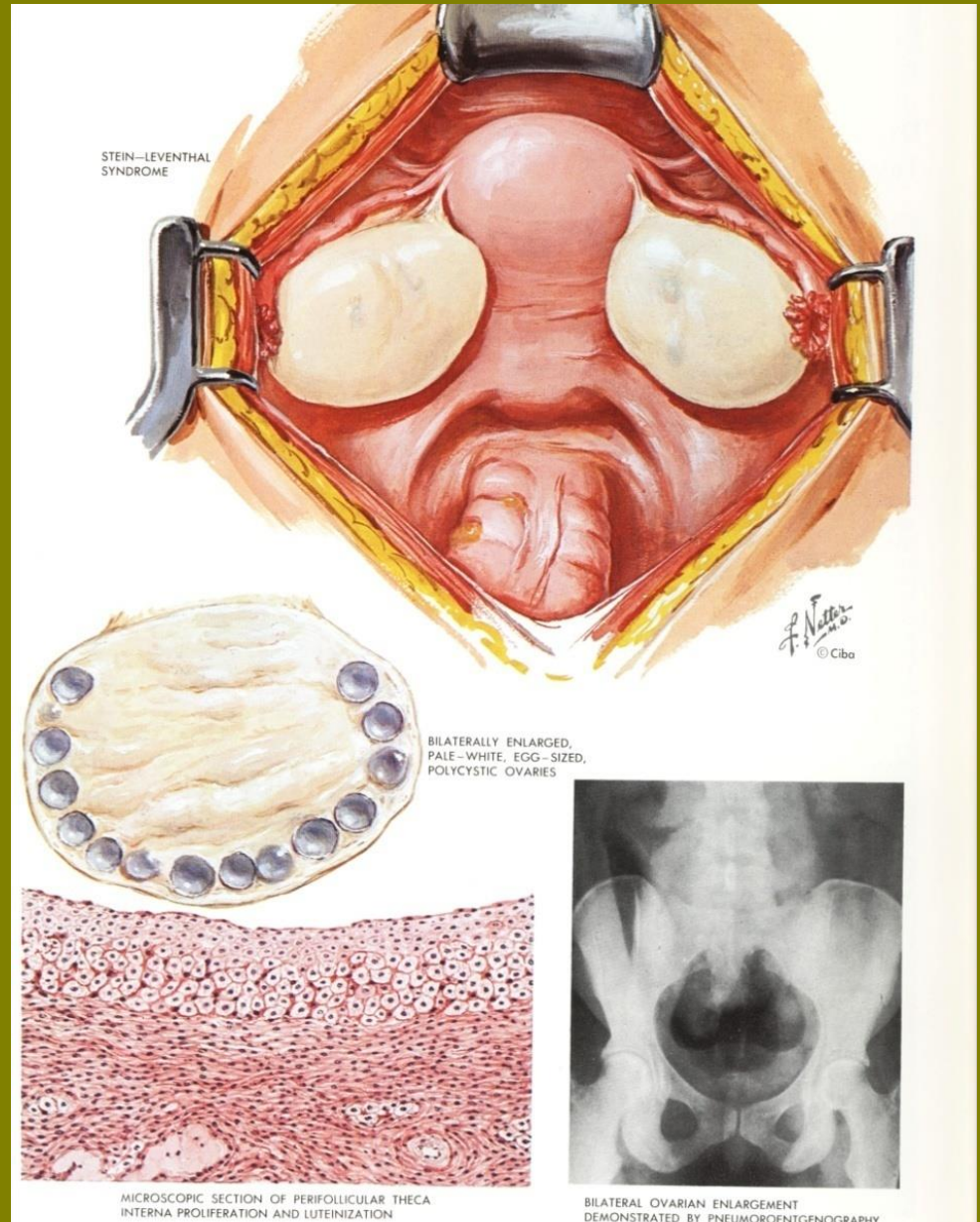
LUTEIN CYST (CORPUS LUTEUM CYST)

PATHOLOGY OF OVARIES

STEIN-LEVENTHAL SYNDROME, PCO SYNDROME

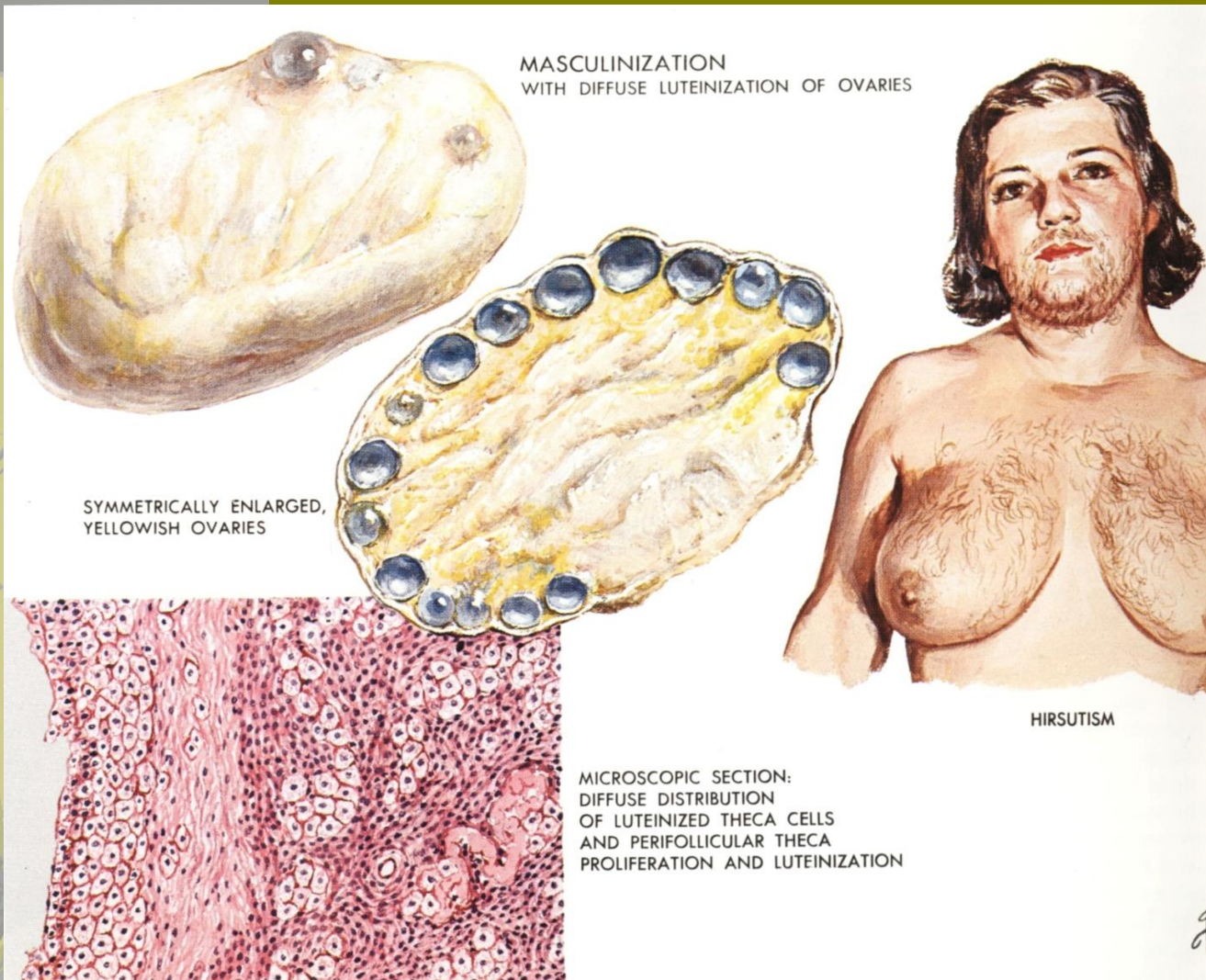


NUMEROUS CYSTS + OLIGOMENORRHEA, STEIN LEVENTHAL SYNDROME. THESE PATIENTS HAVE AN ANOVULAR CYCLE, THEY ARE OBESE (40%), WITH HIRSUTISM (50%) AND RARELY VIRILISM



PATHOLOGY OF OVARIES

(STEIN-LEVENTHAL SYNDROME, PCO SYNDROME)



THE ETIOLOGY OF PCO IS NOT CLEAR

PATHOLOGY OF OVARIES

CLASSIFICATION OF OVARIAN TUMORS (ACCORDINGLY TO WHO)

I. COMMON EPITHELIAL

- A. SEROUS TUMORS
- B. MUCOUS TUMORS
- C. ENDOMETRIAL TUMORS
- D. MESONEPHROID TUMORS
- E. BRENNER TUMOR
- F. UNDIFFERENTIATED CARCINOMA
- G. MIXED MESODERMAL MÜLLERS' TUMOR

II. FROM SEX CORDS AND STROMAL TUMORS

- A. FOLLICULOMA (GRANULOSA CELL TUMOR)
- B. XANTHOFIBROMA THECOCELLULARE AND FIBROMA
- C. ANDROBLASTOMA
- D. SERTOLI-LEYDIG TUMORS

III. LIPID-CONTAINING TUMORS

IV. GERMINAL CELL TUMORS

- A. DYSGERMINOMA
- B. YOLK-SAC TUMOUR (ENDODERMAL SINUS TUMOR)
- C. EMBRYONAL CARCINOMA
- D. POLYEMBRIOMA
- E. CHORIOCARCINOMA
- F. TERATOMA

V. GONADOBLASTOMA

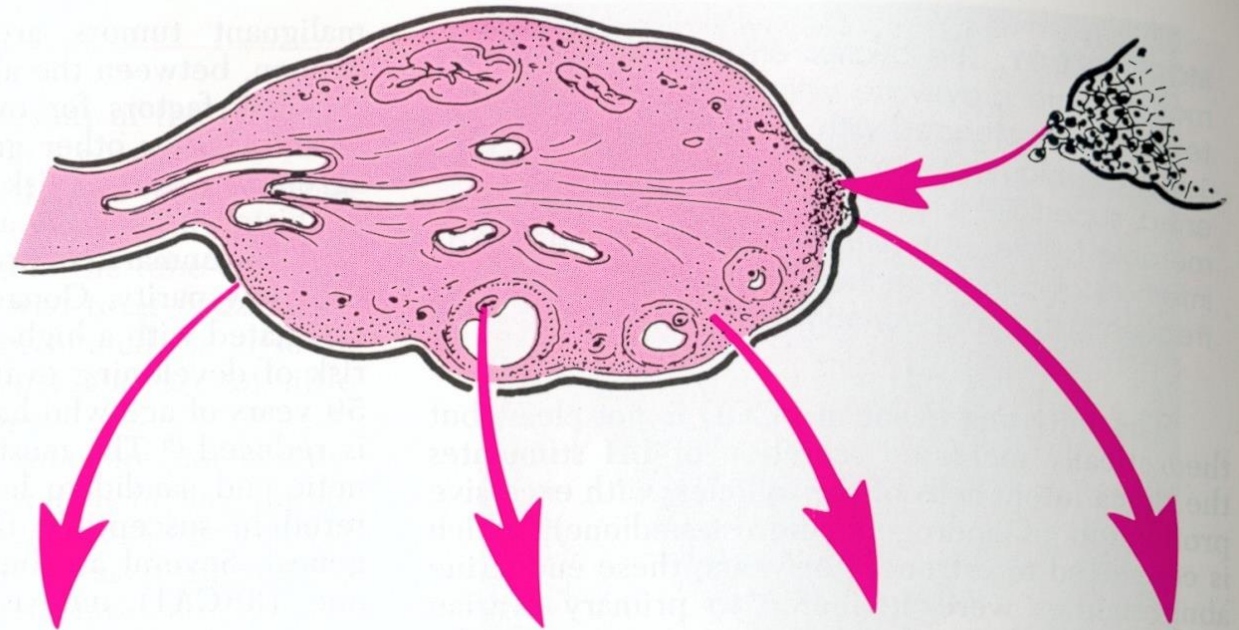
VI. NON-SPECIFIC CONNECTIVE TISSUE TUMORS

VII. NON-CLASSIFIED TUMORS

VIII. METASTATIC TUMORS

PATHOLOGY OF OVARIES

HISTOGENESIS OF OVARIAN TUMORS



Origin	Surface epithelial cells (common epithelial tumors)	Germ cell	Sex cord–stroma	Metastasis to ovaries
Frequency	65–70%	15–20%	5–10%	5%
Age group affected	20+ years	0–25+ years	All ages	Variable
Types	<ul style="list-style-type: none"> • Serous tumor • Mucinous tumor • Endometrioid tumor • Clear cell tumor • Brenner tumor 	<ul style="list-style-type: none"> • Teratoma • Dysgerminoma • Endodermal sinus tumor • Choriocarcinoma 	<ul style="list-style-type: none"> • Fibroma • Granulosa–theca cell tumor • Sertoli–Leydig cell tumor 	

PATHOLOGY OF OVARIES

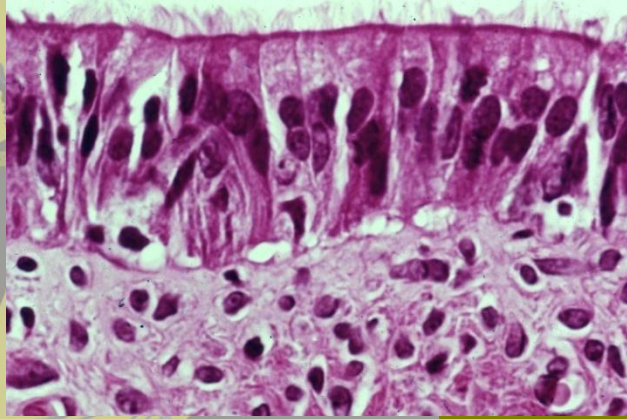
OVARIAN TUMORS

APPROX. 65% OF SEROUS CYSTS ARE BENIGN, AT EVERY AGE, MOST COMMON IN THE 5TH DECADE. IN ABOUT 25% - BILATERAL.

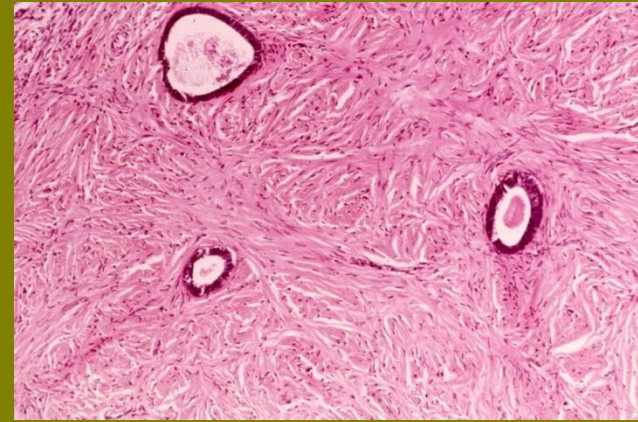


SEROUS TUMORS

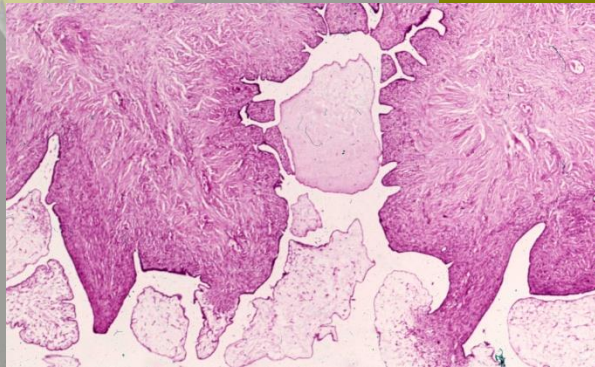
BENIGN



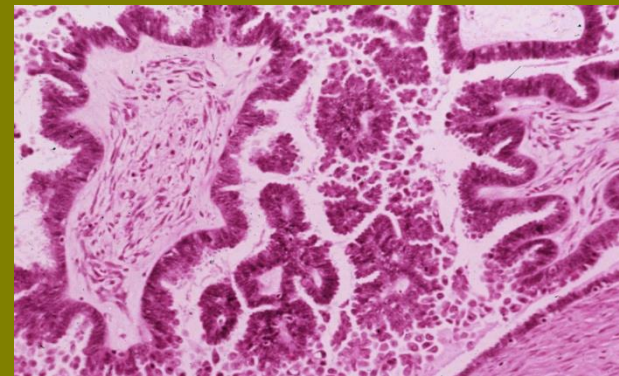
**CILIATED EPITHELIUM OF
SEROUS CYST**



SEROUS ADENOFIBROMA



**SEROUS PAPILLARY
KYSTADENOMA**

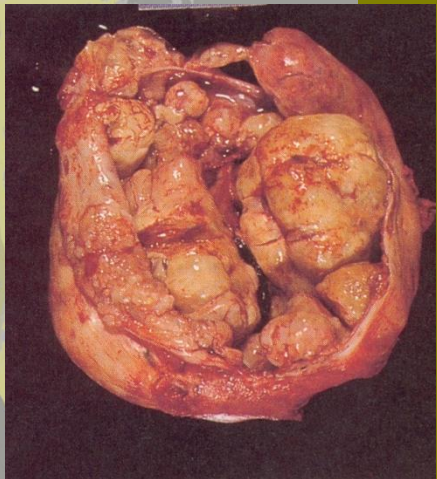


**SEROUS PAPILLARY KYSTADENOMA
(BORDERLINE TYPE) – OMENTAL IMPLANTS**

SEROUS TUMORS BENIGN



SEROUS CYST OF OVARY



PAPILLARY SEROUS KYSTADENOMA



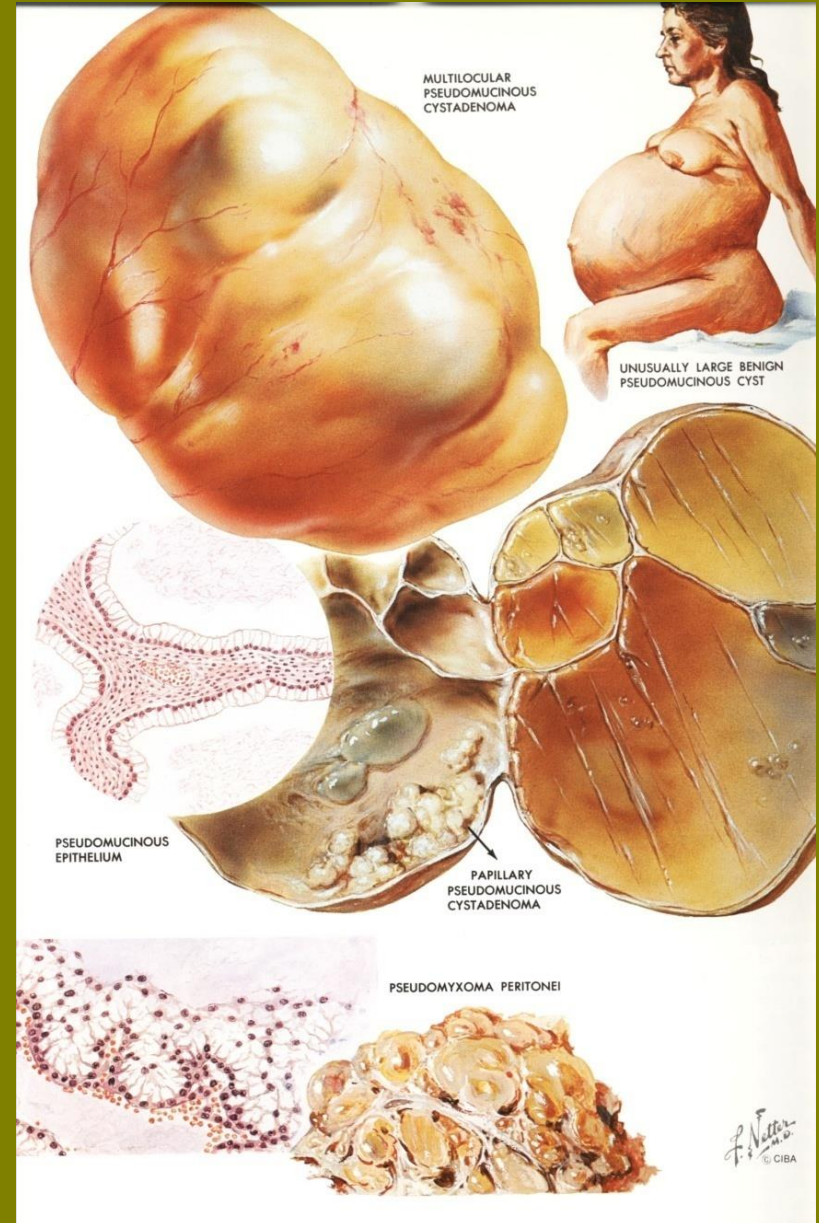
PAPILLARY SEROUS KYSTADENOMA

OVARIAN TUMORS

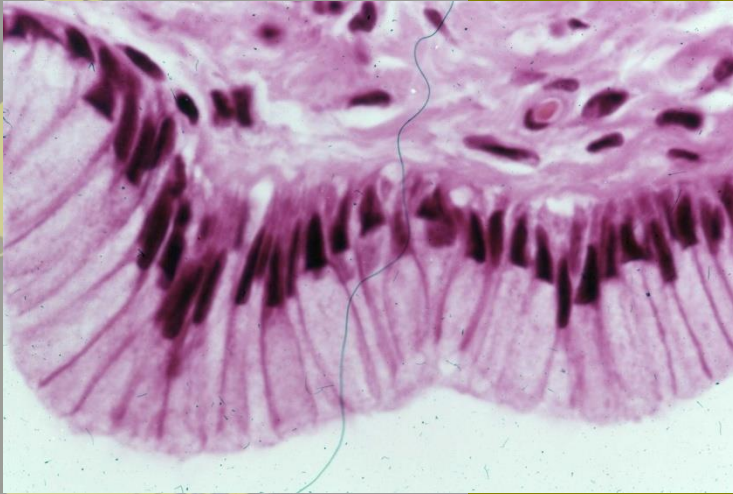
MUCINOUS CYSTS ARE THE BIGGEST OF ALL OVARIAN TUMORS. 85% ARE BENIGN TUMORS



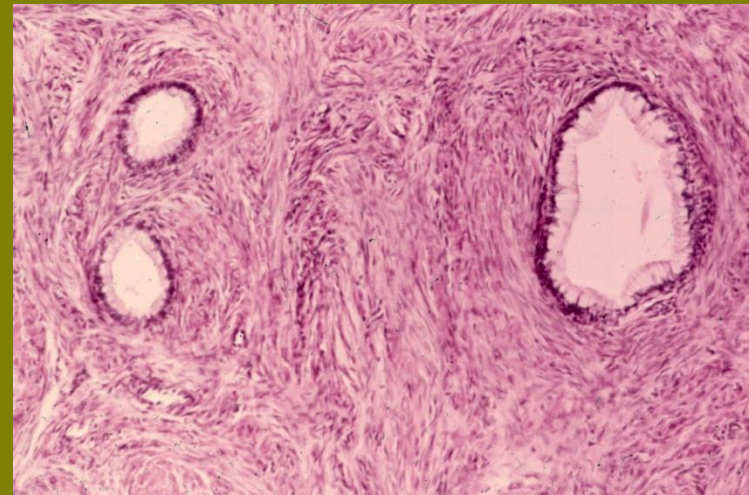
MUCINOUS MULTILOCULAR KYSTADENOMA



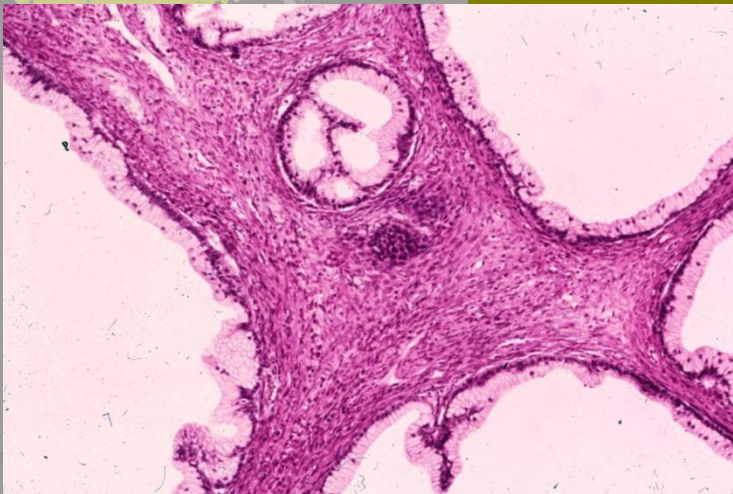
MUCINOUS TUMORS BENIGN



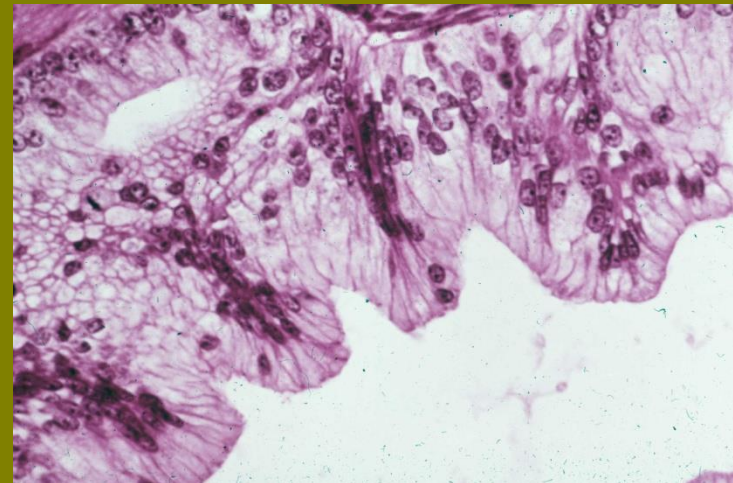
**MUCINOUS KYSTADENOMA
(TYPICAL EPITHELIUM)**



MUCINOUS ADENOFIBROMA

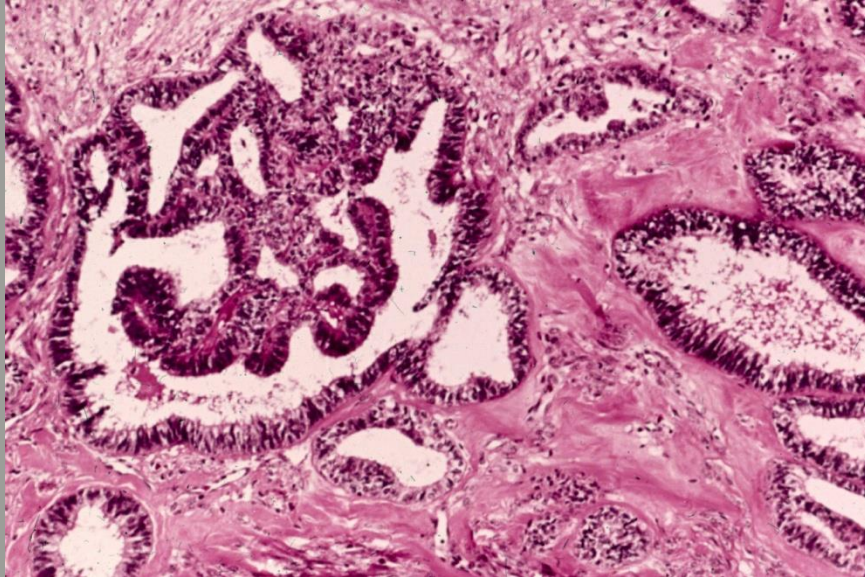


**MUCINOUS MULTILOCULAR
KYSTADENOMA**

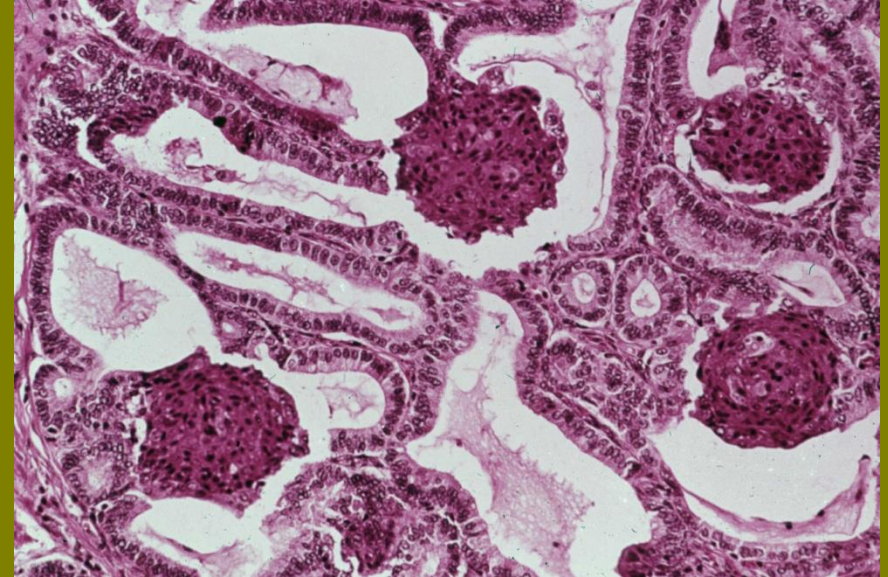


**MUCINOUS KYSTADENOMA
(BORDERLINE)**

ENDOMETRIOIDAL TUMORS



**ENDOMETRIOIDAL
KYSTADENOCARCINOMA**

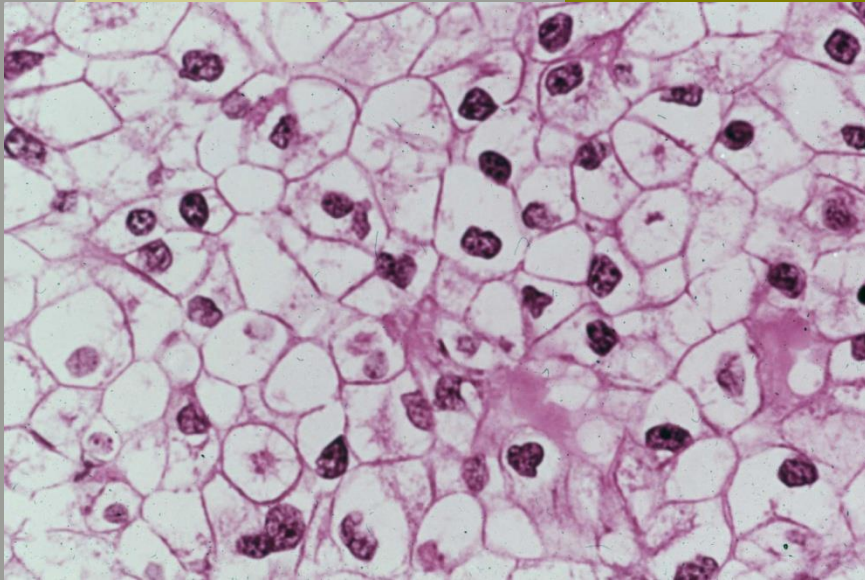


**KYSTADENOCARCINOMA WITH
SQUAMOUS METAPLASIA
(ADENOACANTHOMA)**

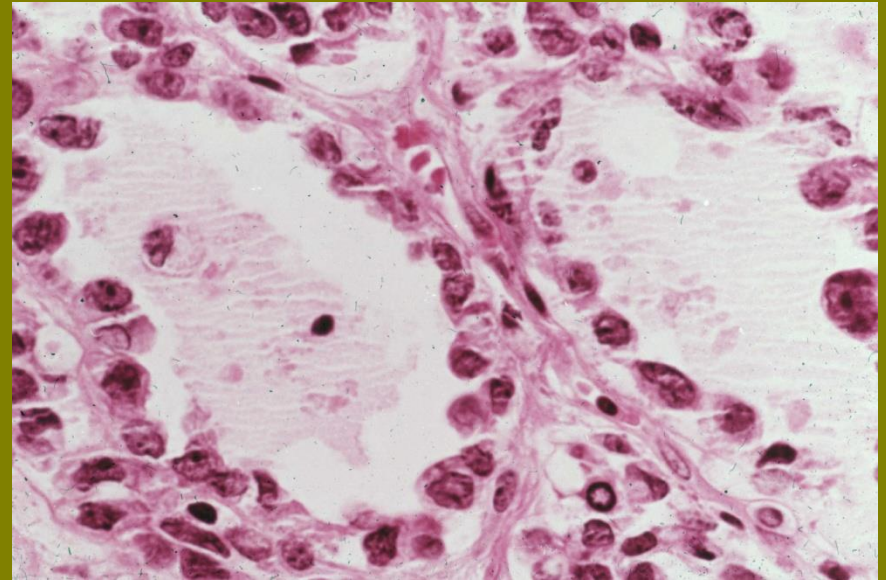
**THEY ARE MALIGNANT (ABOUT 15% OF ALL
MALIGNANT OVARIAN TUMORS)**

CLEAR CELL CARCINOMA

CARCINOMA CLAROCELLULARE



CLEAR CELLS



GLANDS

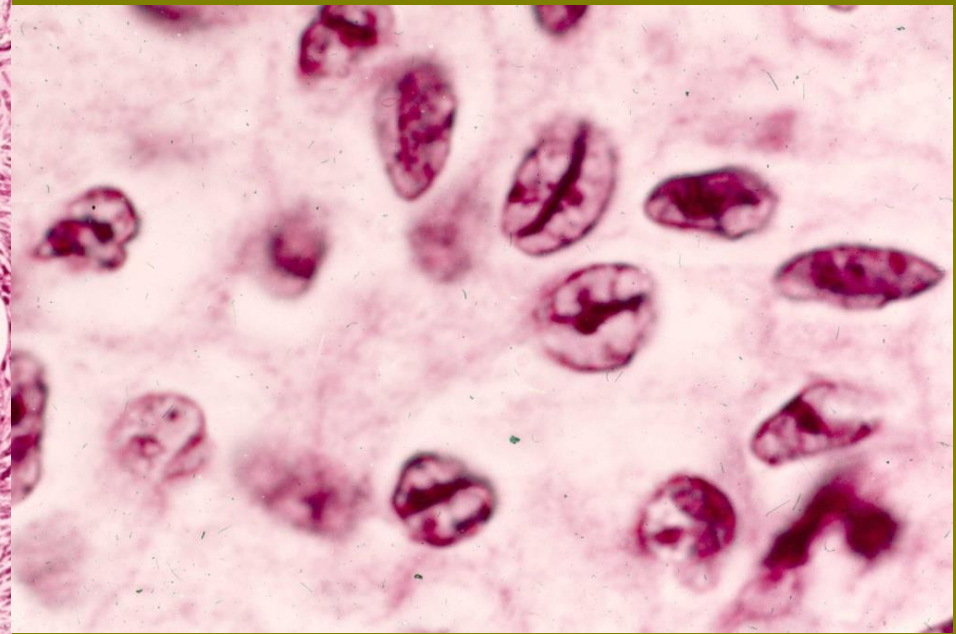
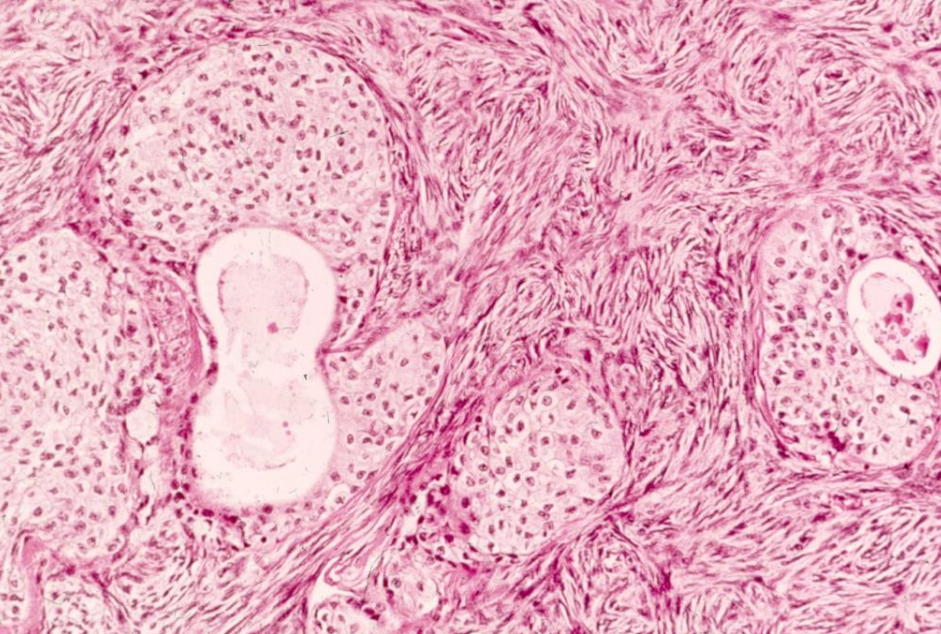
BRENNER TUMOR - general

- One of two types of transitional cell tumors - also transitional cell carcinoma; 1 - 2% of ovarian neoplasms
- Usually age 40+; median age 50 years
- Slow growth, rarely ascites
- Adenofibromas in which epithelial component consists of sharply demarcated nests of urothelial-like cells
- Associated with hyperestrinism (endometrial hyperplasia and uterine bleeding), mucinous cystadenoma, struma ovarii, urothelial carcinoma of bladder
- Has true urothelial differentiation based on immunostains
Epidermoid cysts may originate from epithelial cell nests of Brenner tumor (Am J Clin Pathol 1980;73:272)

EPITHELIAL TUMORS

BRENNER TUMOR

BENIGN



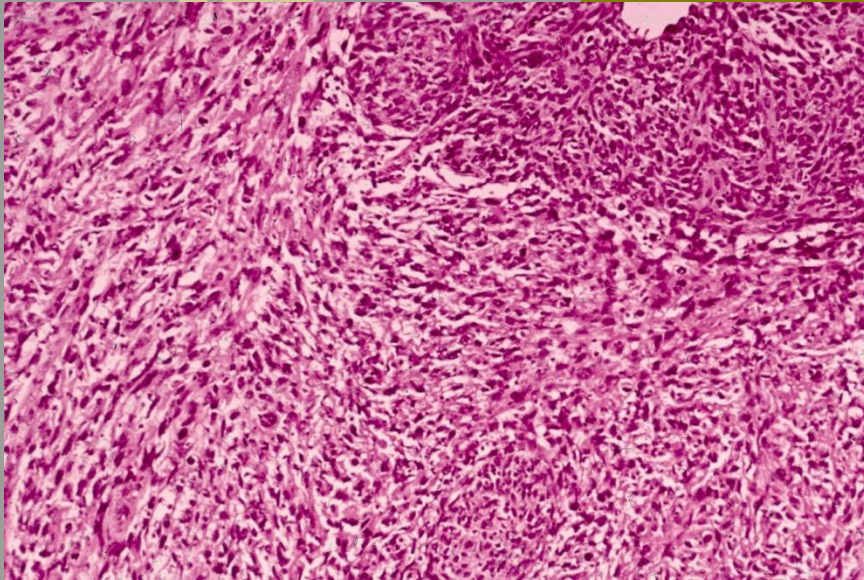
TYPICAL MICROSCOPIC PICTURE

COFFEE BEAN-LIKE NUCLEI

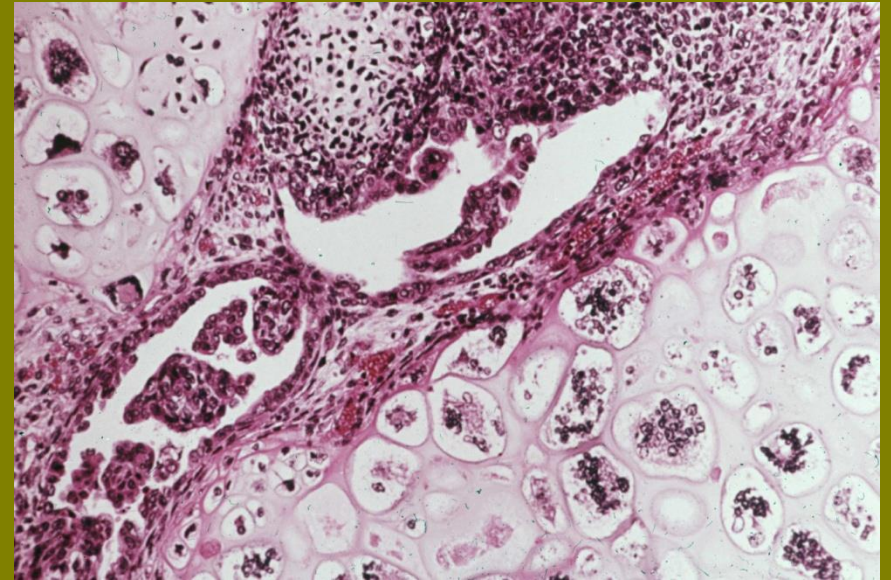
**MOST COMMONLY BILATERAL, COMMONLY CYSTIC.
SOMETIMES PRODUCES ESTROGENS**

EPITHELIAL TUMORS

MIXED MESODERMAL TUMOR (MÜLLER)



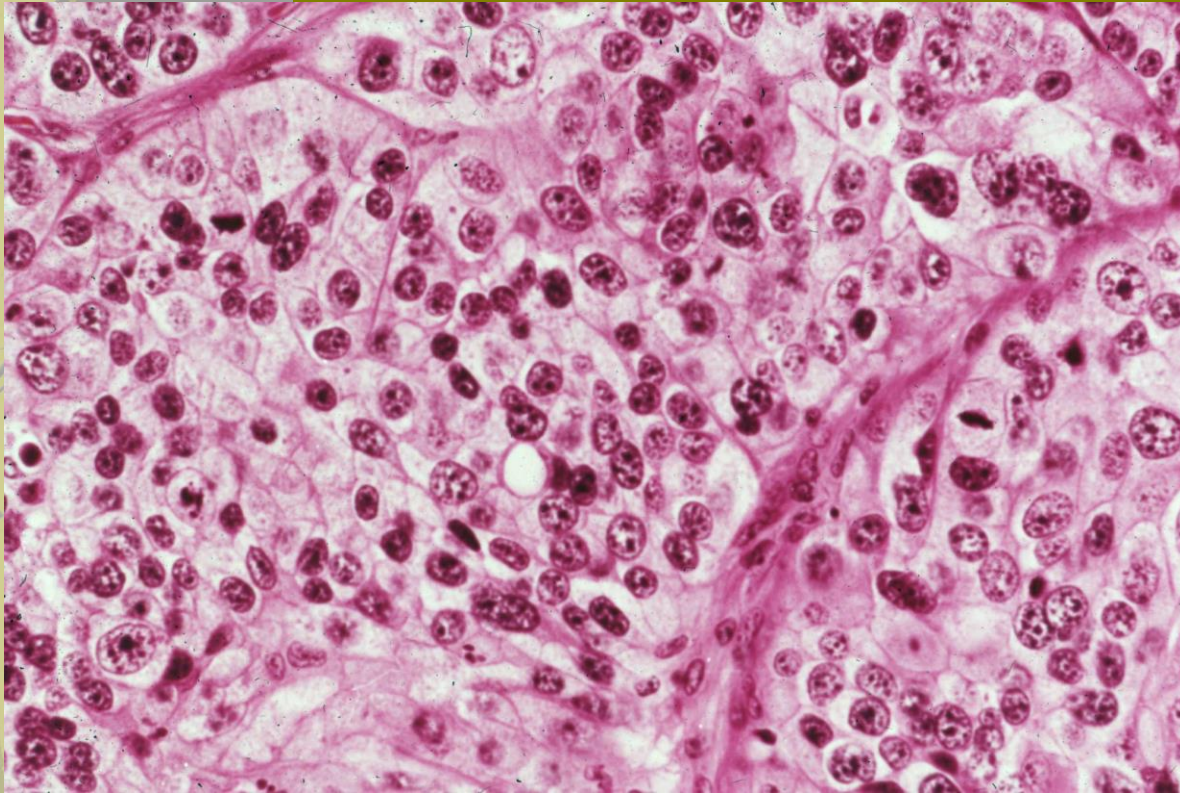
HOMOLOGOUS



HETEROLOGOUS

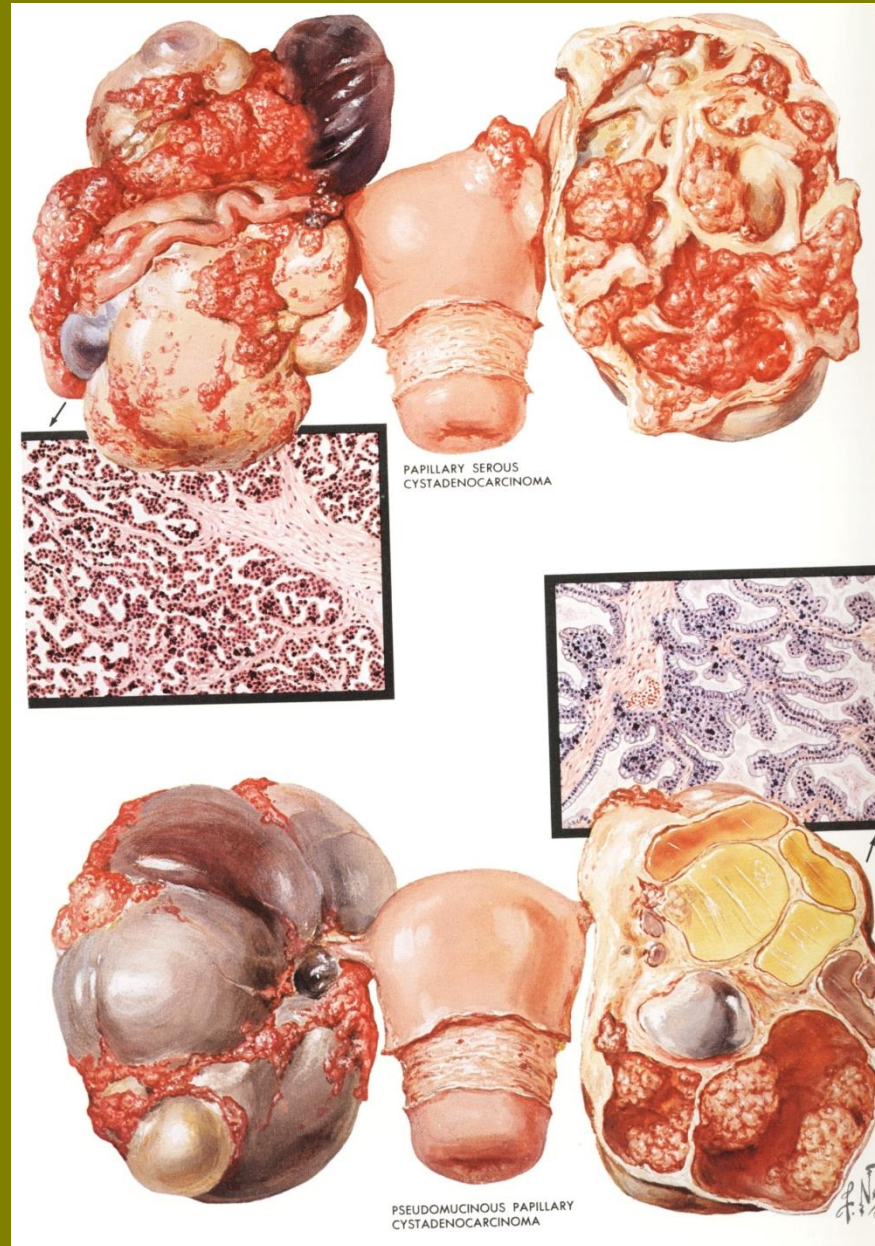
EPITHELIAL TUMORS

UNDIFFERENTIATED CARCINOMA



**TYPICAL MICROSCOPIC
PICTURE**

**GROSS PATHOLOGY OF
COMMON OVARIAN
MALIGNANT TUMORS
APPROX. 25% OF THESE
TUMORS OCCUR BILATERALLY**

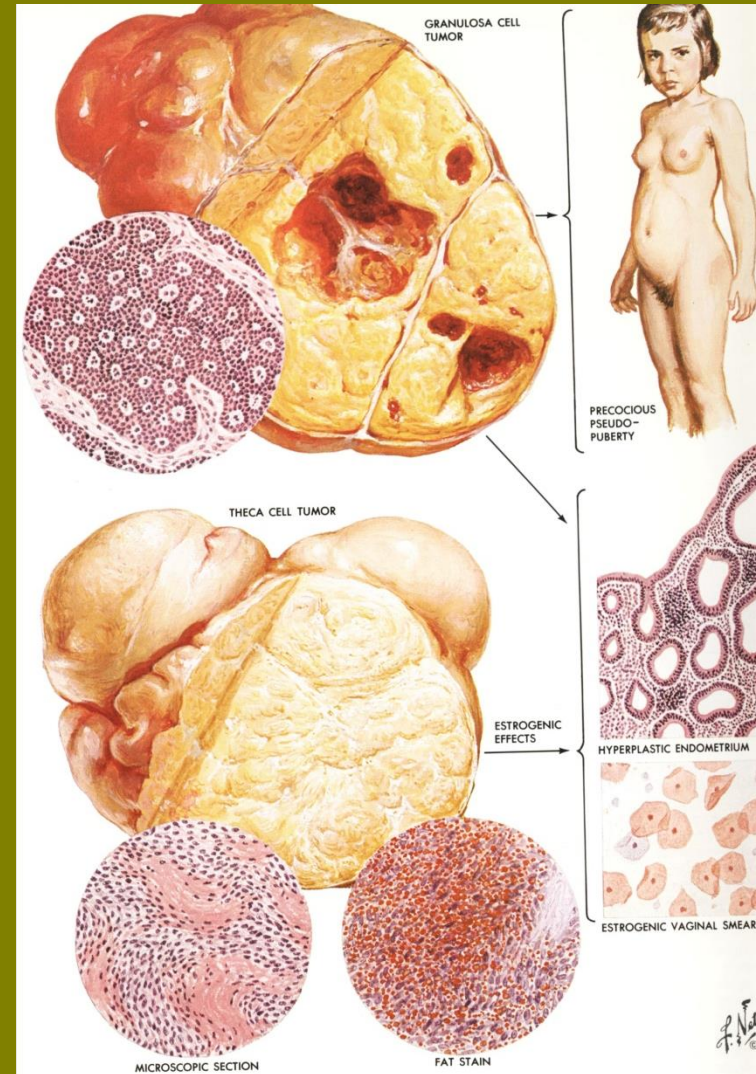


SEX CORDS-STROMAL TUMORS

FROM GRANULOSA CELLS



**FOLLICULOMA – ADULT TYPE
FROM THE GRANULOSA CELLS OF
GRAAFIAN FOLLICLE. TUMOR IS
SOLID, SOMETIMES CYSTIC,
USUALLY UNILATERAL. PRODUCES
ESTROGENS →
HYPERPLASIA OF ENDOMETRIUM.**

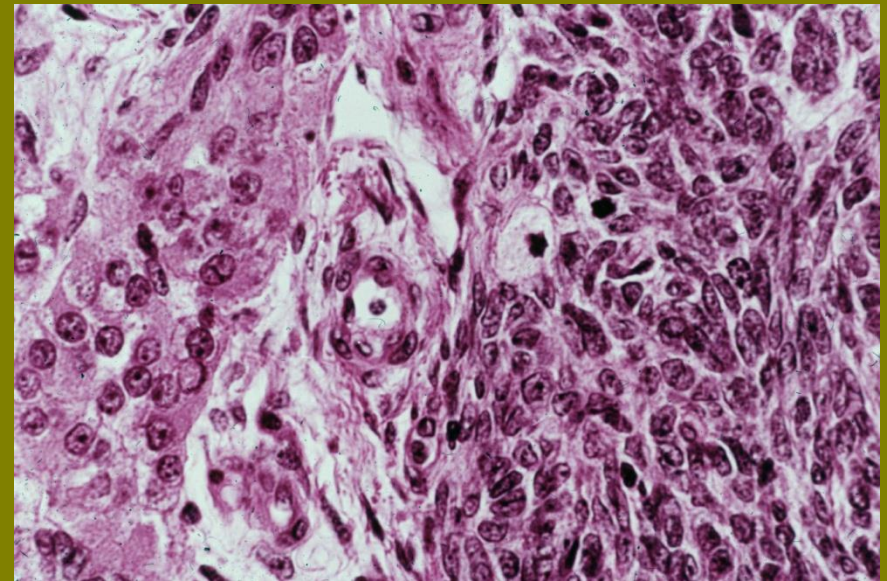
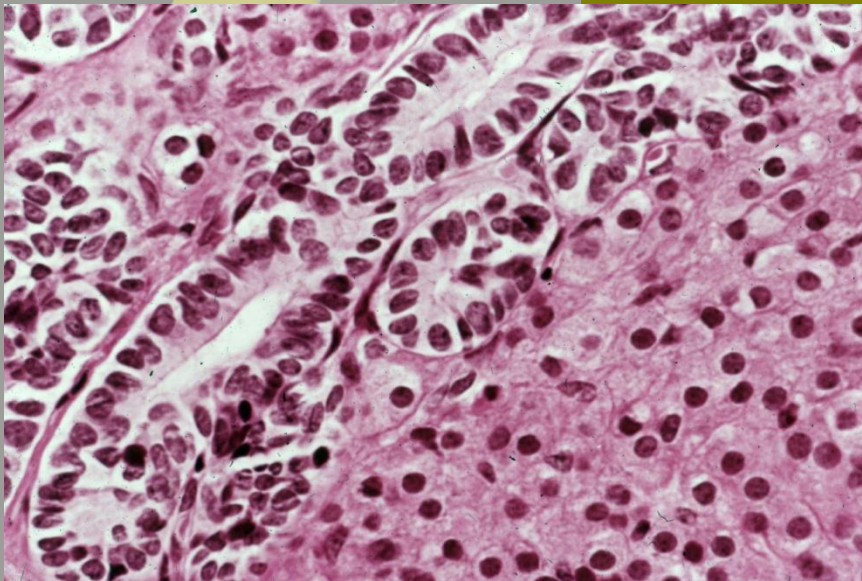




SEX CORDS-STROMAL TUMORS

FROM SERTOLI AND LEYDIG CELLS – ANDROBLASTOMA,
ARRHENOBLASTOMA

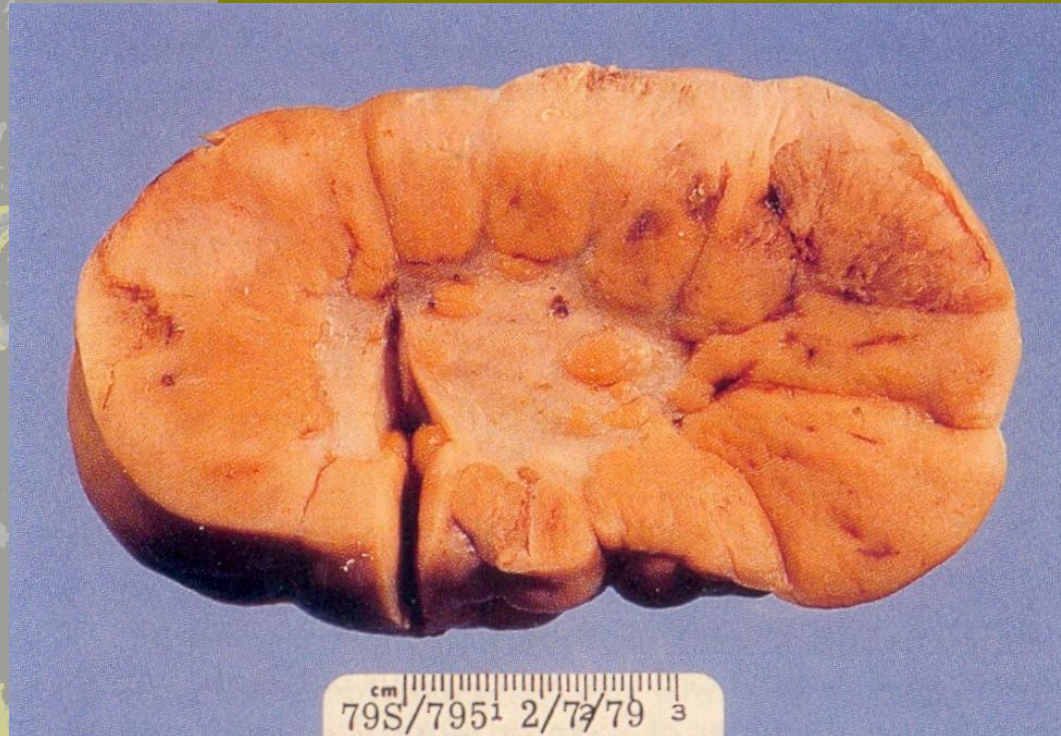
SERTOLI-LEYDIG TUMOR



BUILT FROM CELLS TYPICAL FOR TESTIS IN DIFFERENT PHASES OF DEVELOPMENT. USUALLY OCCURS IN THE 2ND AND 3RD DECADE OF LIFE, UNILATERAL. MASCULATION, ALTHOUGH SOMETIMES HYPERESTROGENISM. GROSS: YELLOW TO BROWN TUMOR.

GERMINAL CELL TUMORS

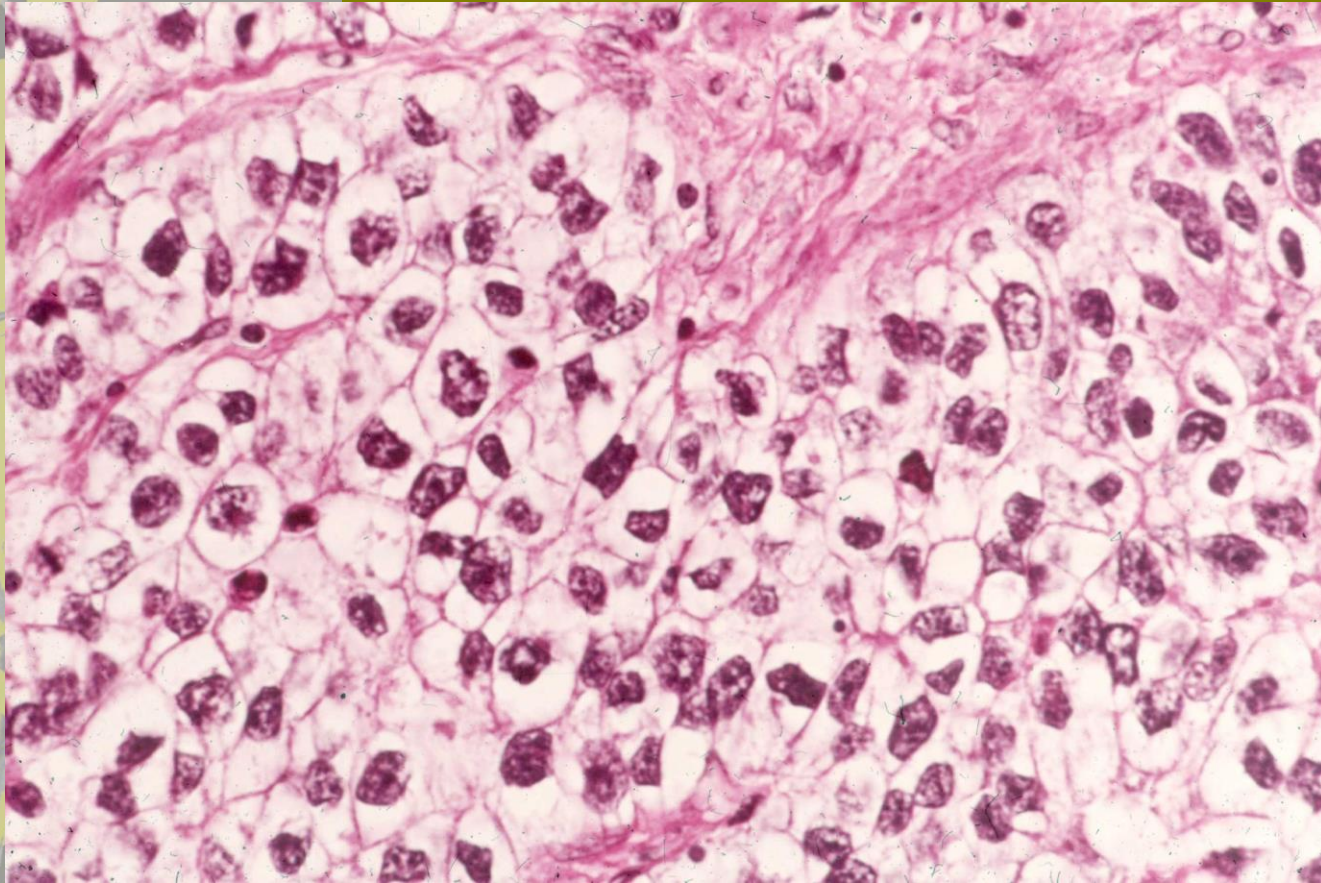
DYSGERMINOMA



UP TO 1% OF MALIGNANT OVARIAN TUMORS, AND AS MUCH AS 5-10% OF MALIGNANT TUMORS IN THE 1ST – 2ND DECADE OF LIFE. AVERAGE AGE OF WOMAN: 21

GERMINAL CELL TUMORS

DYSGERMINOMA



**BUILT FROM CELLS RESEMBLING PRIMARY SEX CELLS;
ABUNDANT IN GLYCOGEN, WITH LYMPHOCYTES**

GERMINAL CELL TUMORS

YOLK SAC TUMOR (ENDODERMAL SINUS TUMOR)

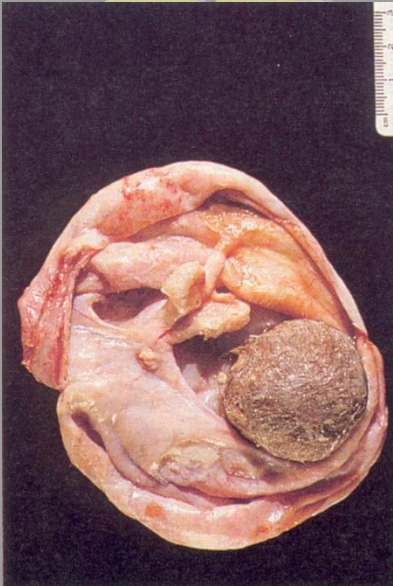


UP TO 20% OF MALIGNANT GERMINAL TUMORS. AVERAGE AGE IS 19 YEARS !! EXTREMELY INCREASED LEVELS OF AFP IN SERUM.

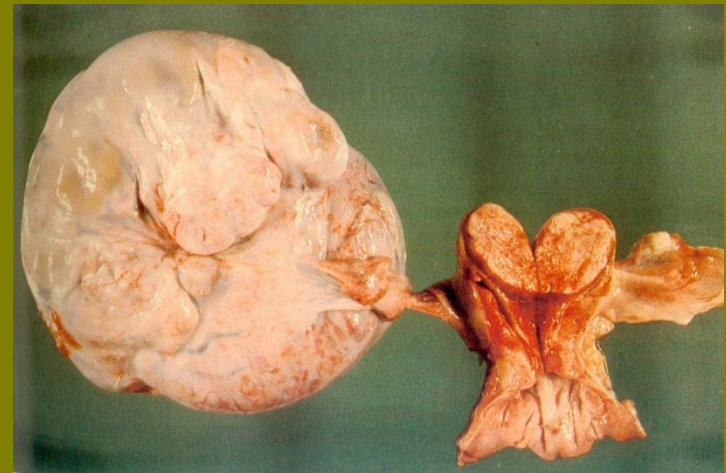
GERMINAL CELL TUMORS

TERATOMA

MATURE (ADULT) TERATOMA



DIFFERENT MACROSCOPIC PICTURES OF MATURE TERATOMA

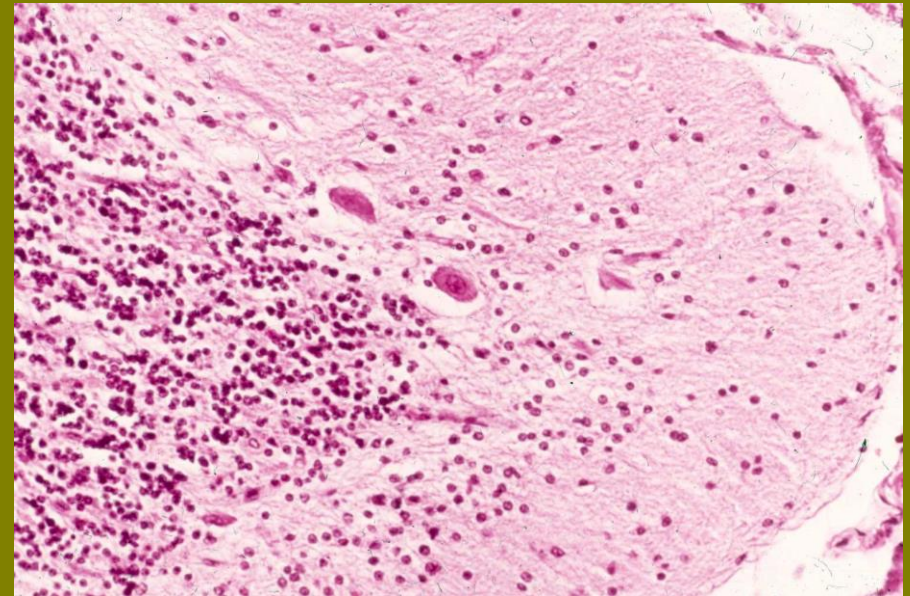


GERMINAL CELL TUMORS

TERATOMA



**SKIN WITH ADNEXA IN
DERMOID CYST**

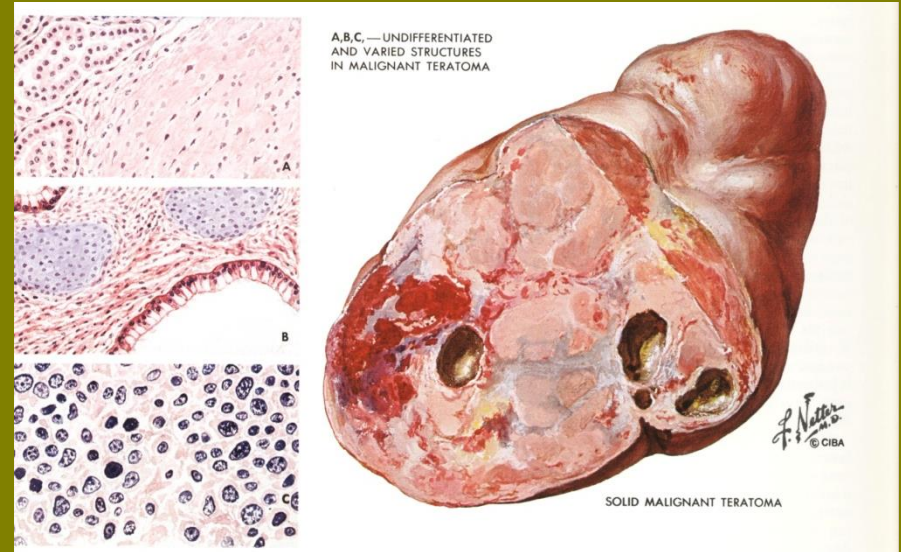


**CEREBELLAR CORTEX IN
ADULT TERATOMA**

**UP TO 25% OF ALL OVARIAN TUMORS; MAINLY IN
YOUNGER WOMEN**

GERMINAL CELL TUMORS

IMMATURE EMBRYONAL TERATOMA

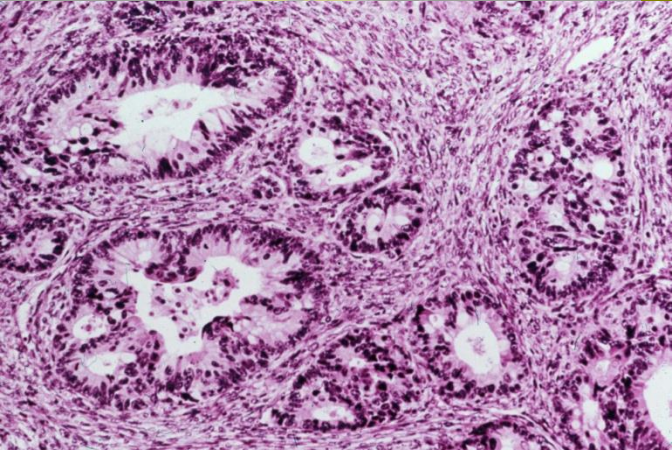
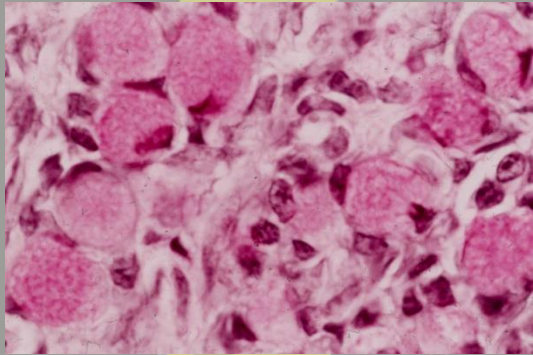
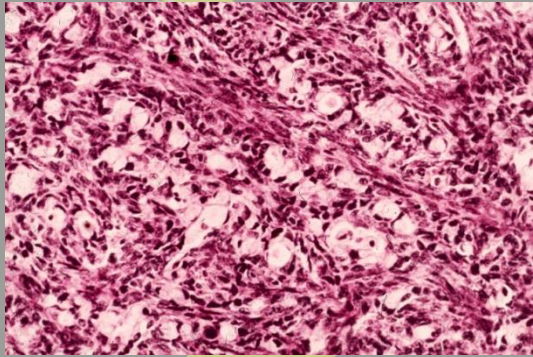


EMBRYONAL TERATOMA

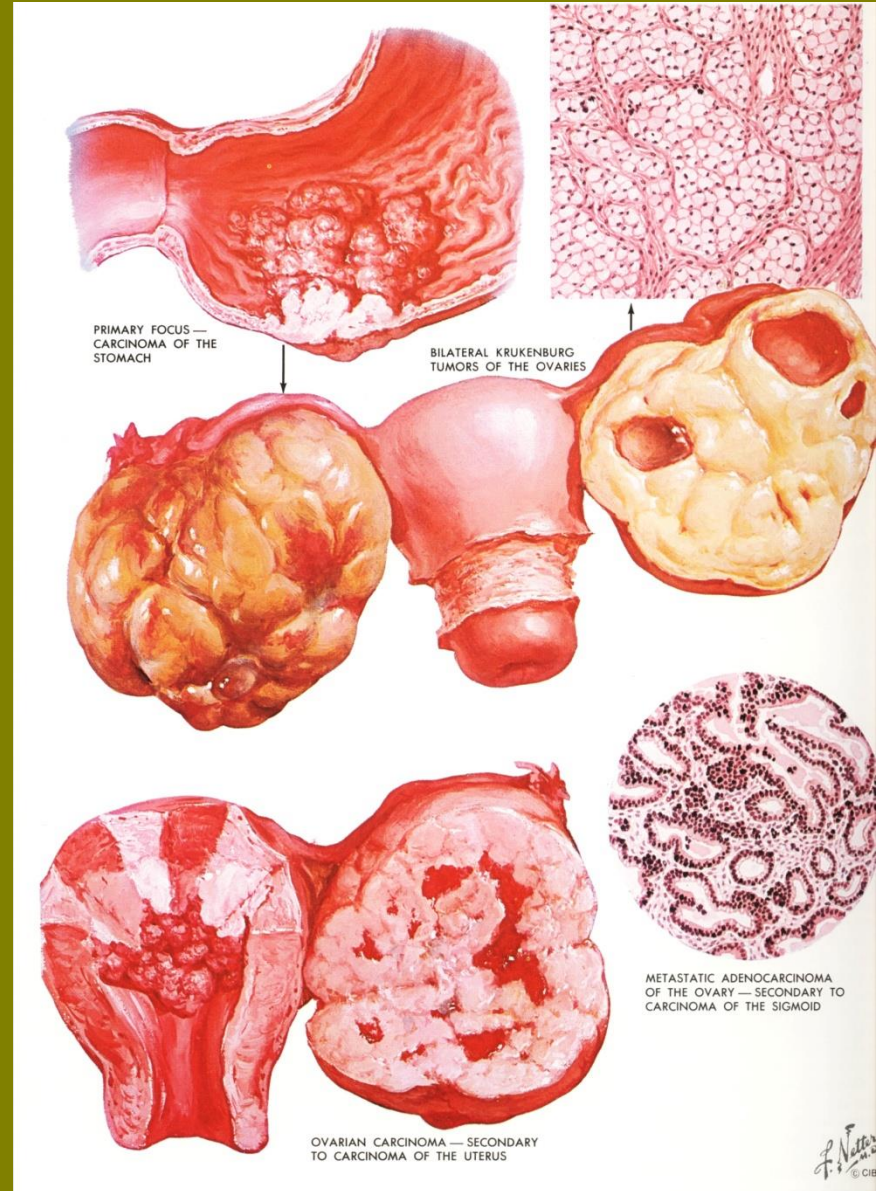
1% OF MALIGNANT OVARIAN TUMORS AND AS MUCH AS 10-20% OF MALIGNANT TUMORS IN THE FIRST AND SECOND DECADE OF LIFE.

PATHOLOGY OF OVARIES

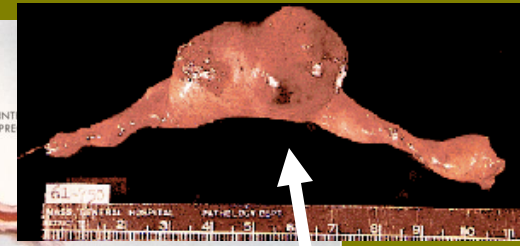
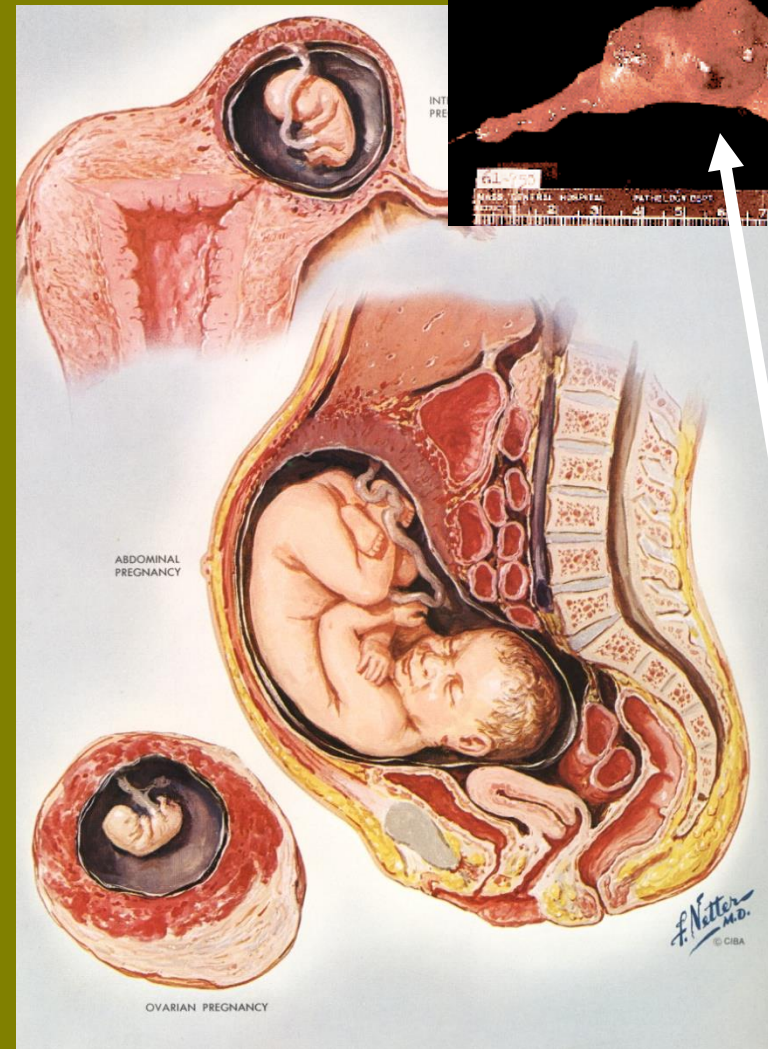
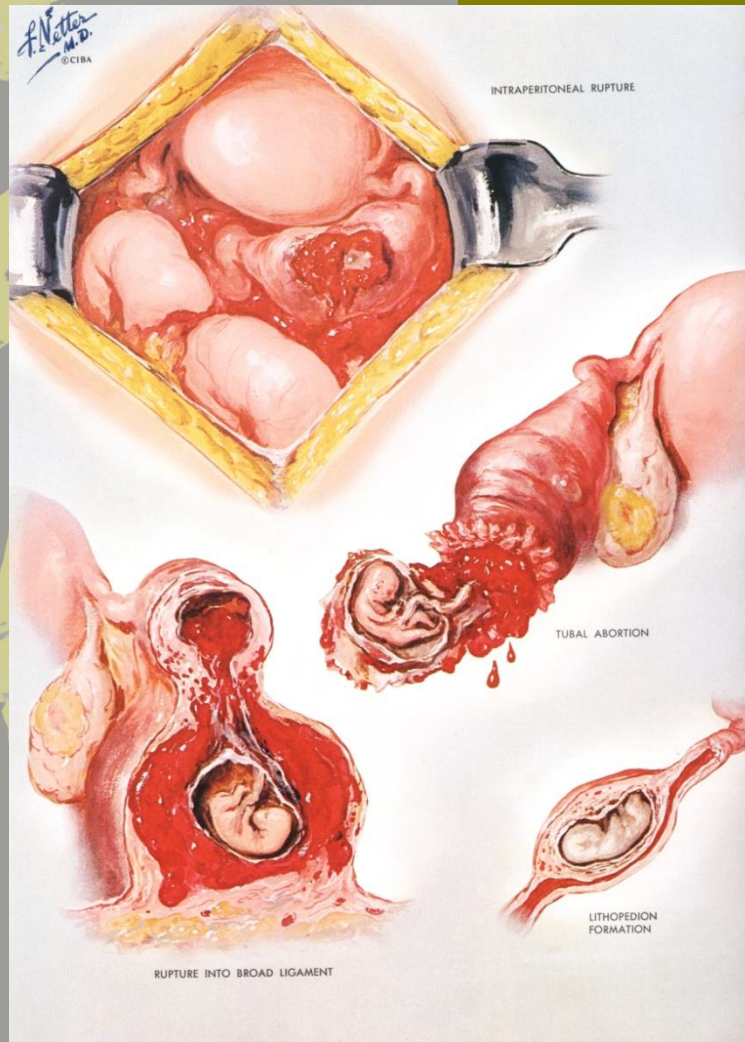
METASTATIC TUMORS



**KRUKENBERG
TUMOR –
USUALLY THE
METASTASIS
TO THE
OVARY FROM
MUCINOUS
CARCINOMA**



ECTOPIC PREGNANCY

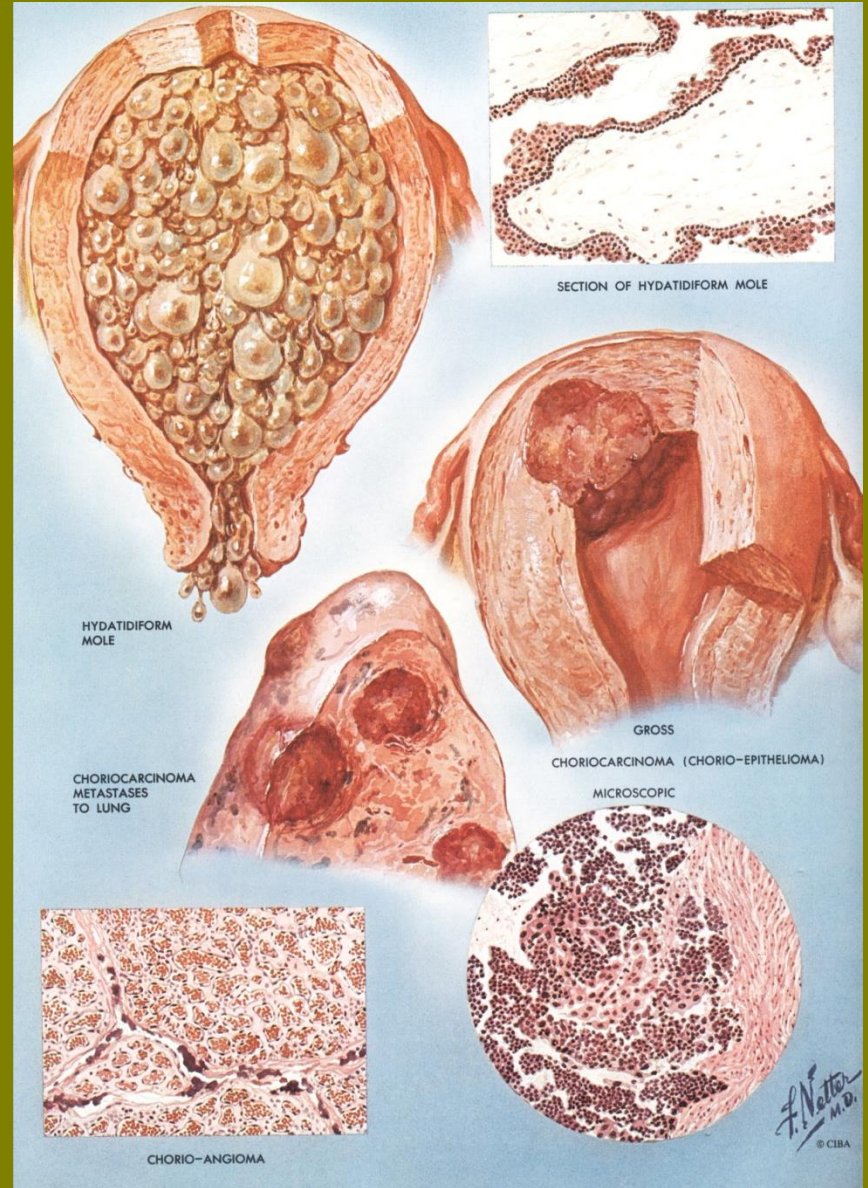


TUBAL PREGNANCY, TUBAL ABORTION, HEMASCOS, ABDOMINAL PREGNANCY, OVARIAN PREGNANCY

TROPHOBLASTIC DISEASE



**HYDATID MOLE
CHARACTERIZED BY THE EDEMA
OF THE CHORIONIC VILLI AS
WELL AS PROLIFERATION OF
TROPHOBLAST.**



HYDATIDIFORM MOLE

SECTION OF HYDATIDIFORM MOLE

CHORIOCARCINOMA METASTASES TO LUNG

CHORIOCARCINOMA (CHORIO-EPITHELIOMA)

CHORIO-ANGIOMA

GROSS

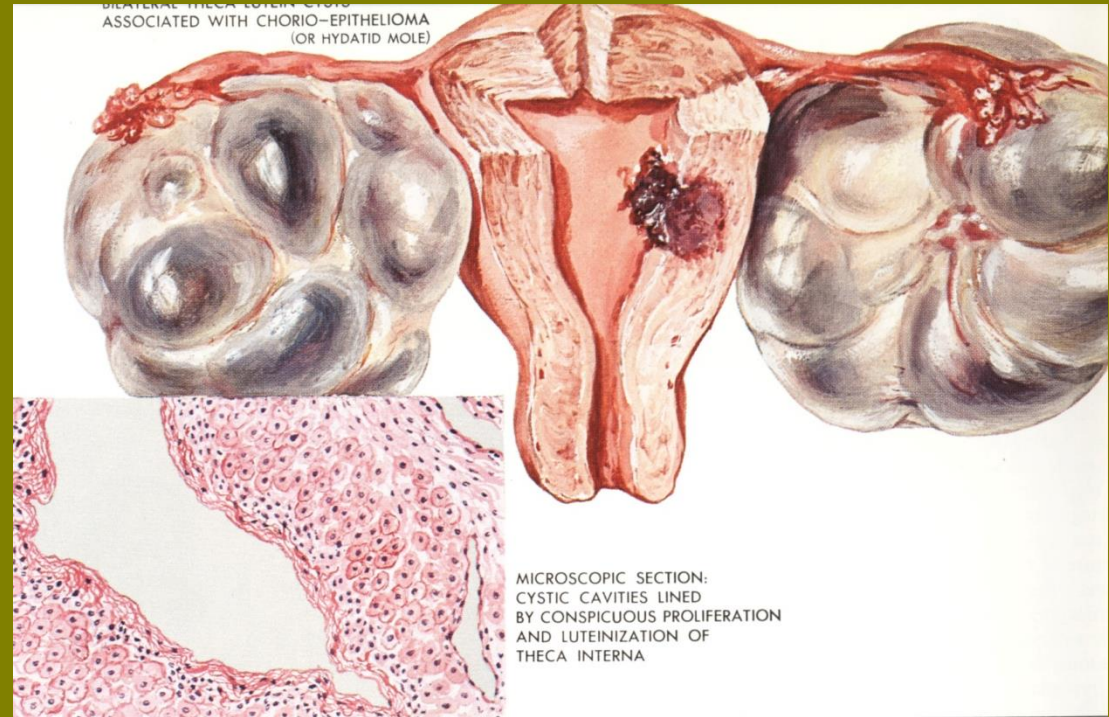
MICROSCOPIC

TROPHOBLASTIC DISEASE

CHORIOEPITHELIOMA



**METASTASIS OF
CHORIOEPITHELIOMA TO
LIVER**

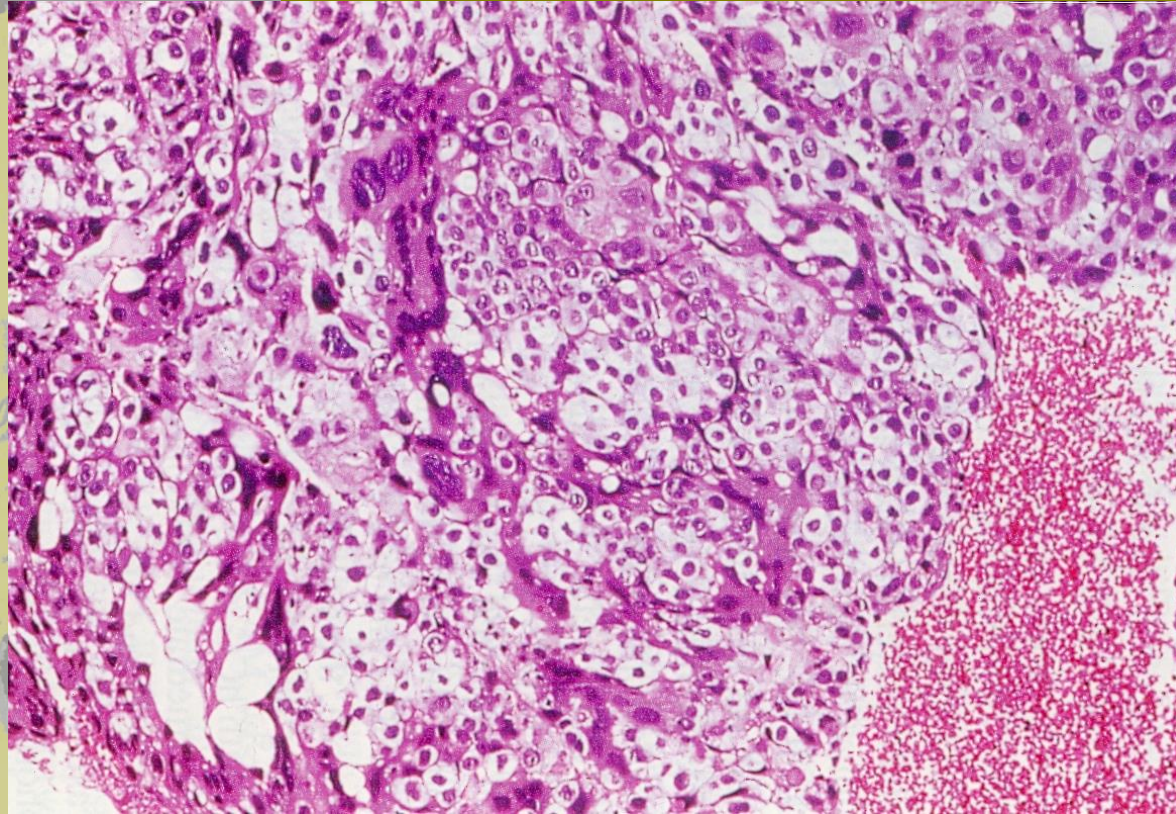


**THECALUTEIN CYSTS DEVELOP IN THE
OVARIES**

INVASIVE TUMOR BUILT FROM TROPHOBLAST (MAINLY CYTO- AND SYNCYTIOTROPHOBLAST), EXTREME DIMORPHISM AND LACK OF CHORIONIC VILLI. DEVELOPS DURING OR AFTER PREGNANCY. EXTREMELY AGGRESSIVE, AND INFILTRATIVE GROWTH, HEMORRHAGIC, METASTATIC. PRODUCES LARGE AMOUNTS OF hCG.

TROPHOBLASTIC DISEASE

CHORIOEPITHELIOMA



**CHORIOEPITHELIOMA (CHORIOCARCINOMA)
SYNCYTIO- AND CYTOTROPHOBLAST AND HEMORRHAGE**

