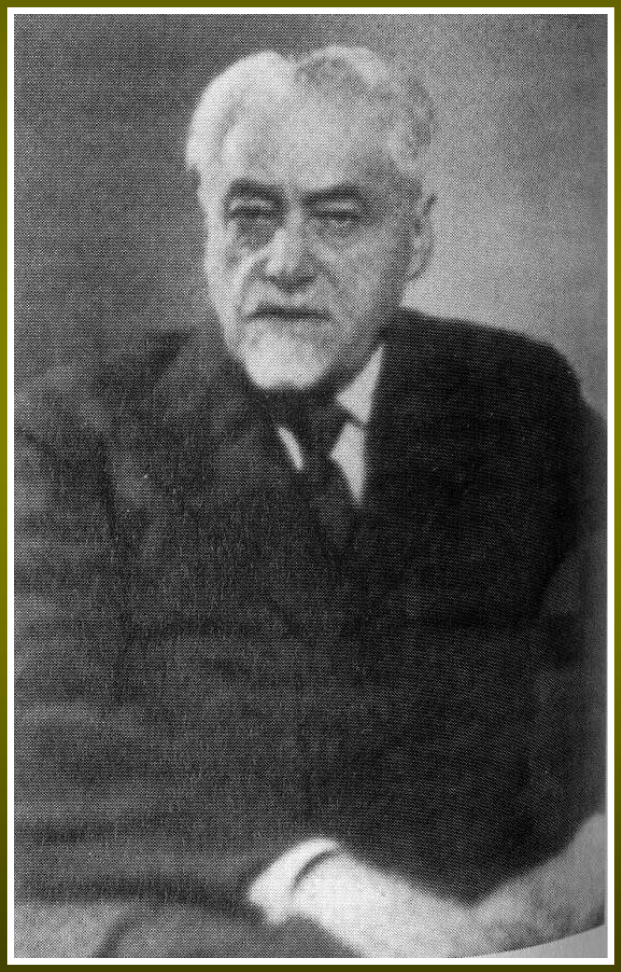
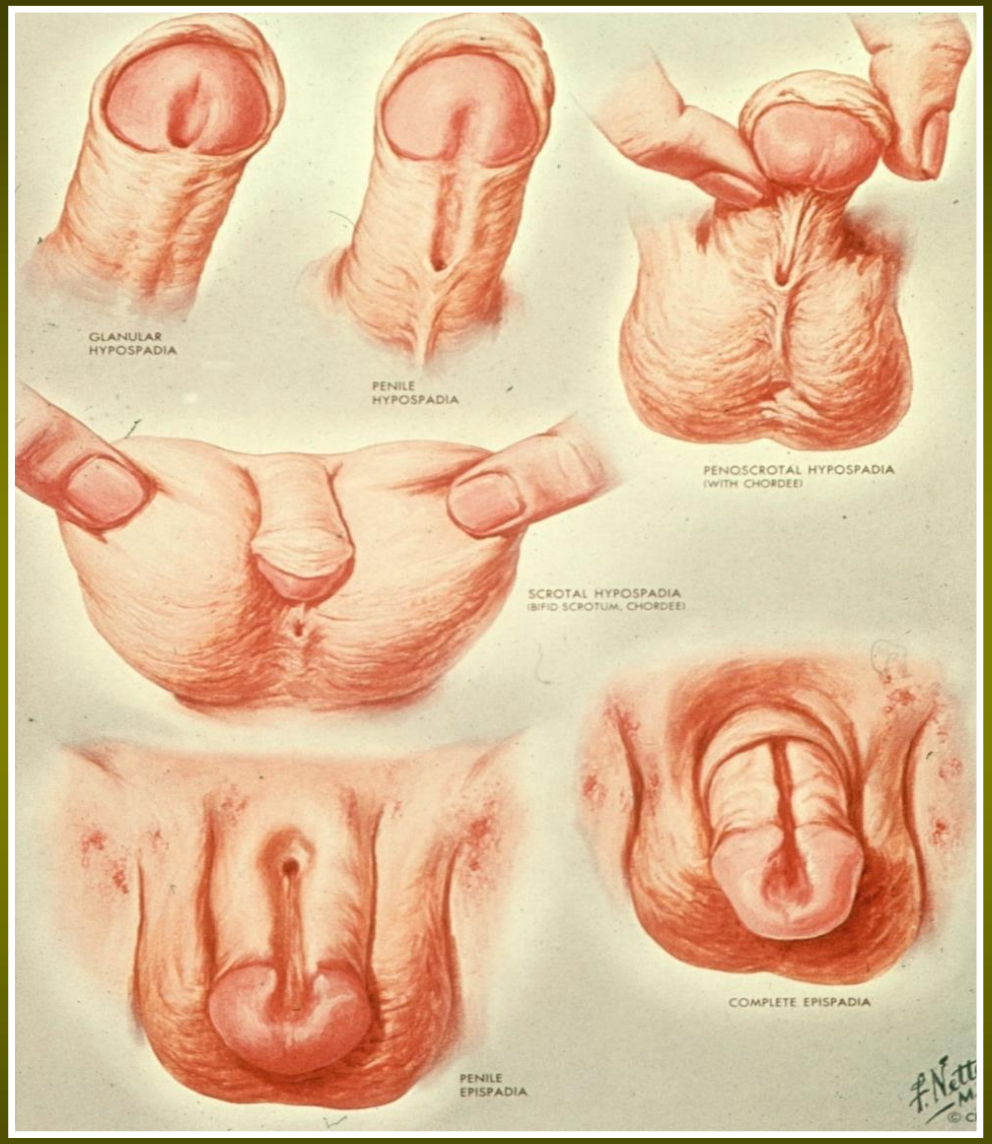
LECTURE 20



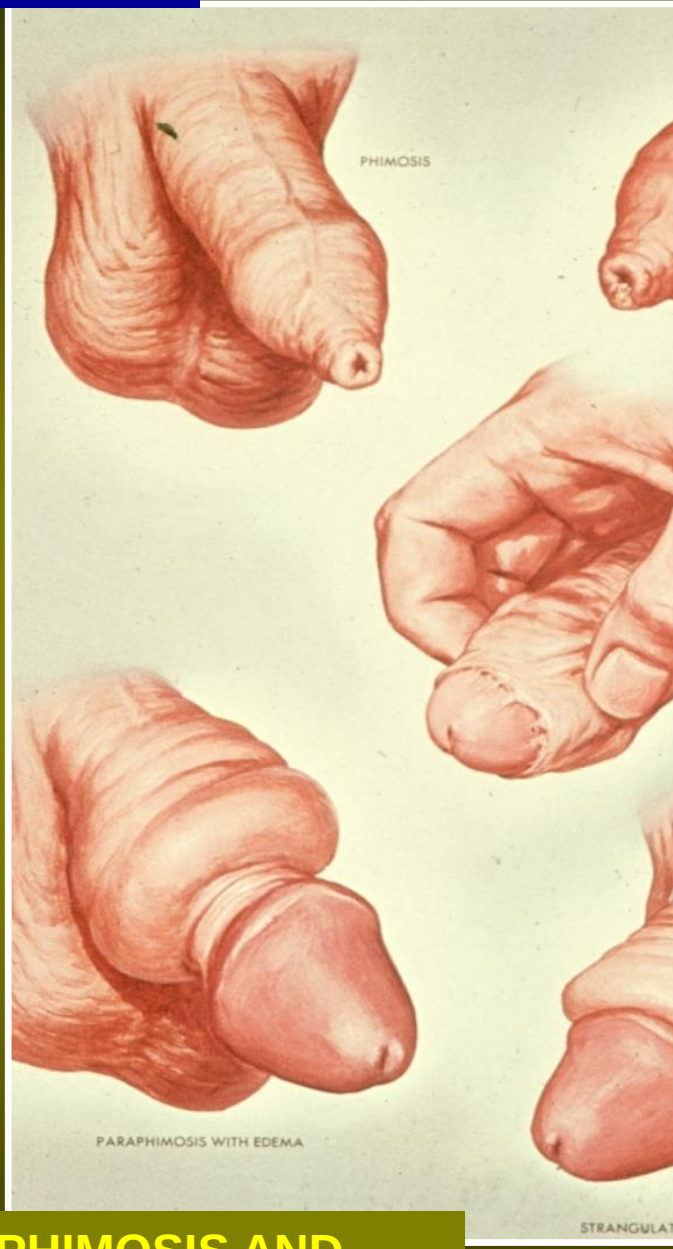
SAMUEL GOLDFLAM
(1852-1932)

MALE REPRODUCTIVE SYSTEM

ANOMALIES OF URETHRA AND PENIS

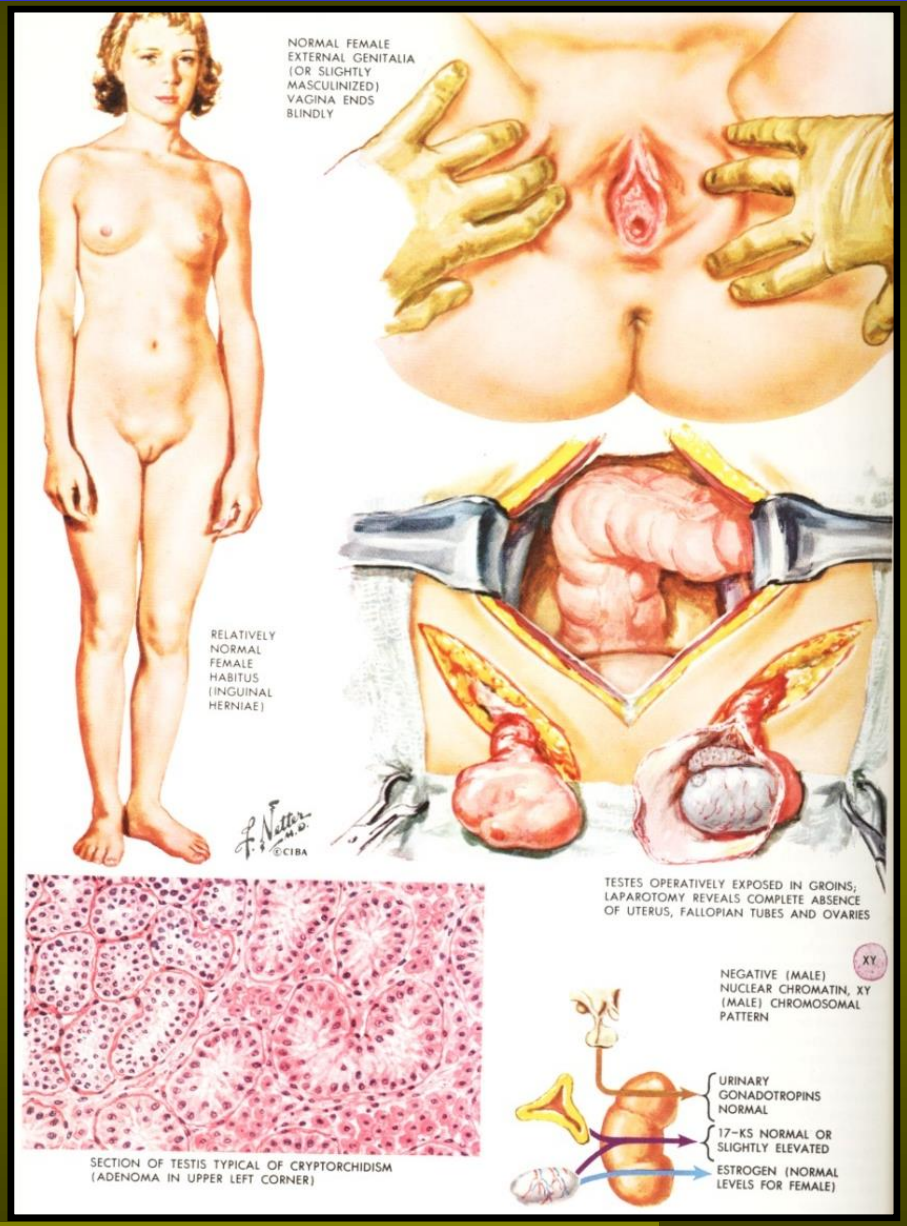


EPISPADIASIS AND HYPOSPADIASIS



PHIMOSIS AND PARAPHIMOSIS

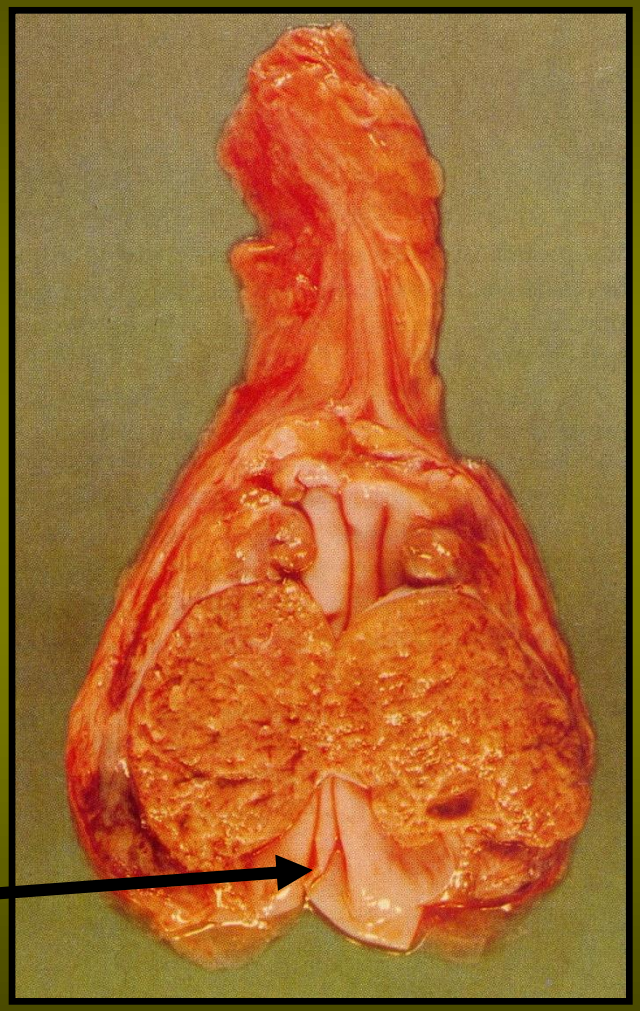
PATHOLOGY OF TESTIS - DEVELOPMENTAL DISTURBANCES



CRYPTORCHIDISM

PATHOLOGY OF TESTIS

CYSTS



TESTICULAR HYDROCELE

HEMATOCELE – HYDROCELE WITH BLOOD

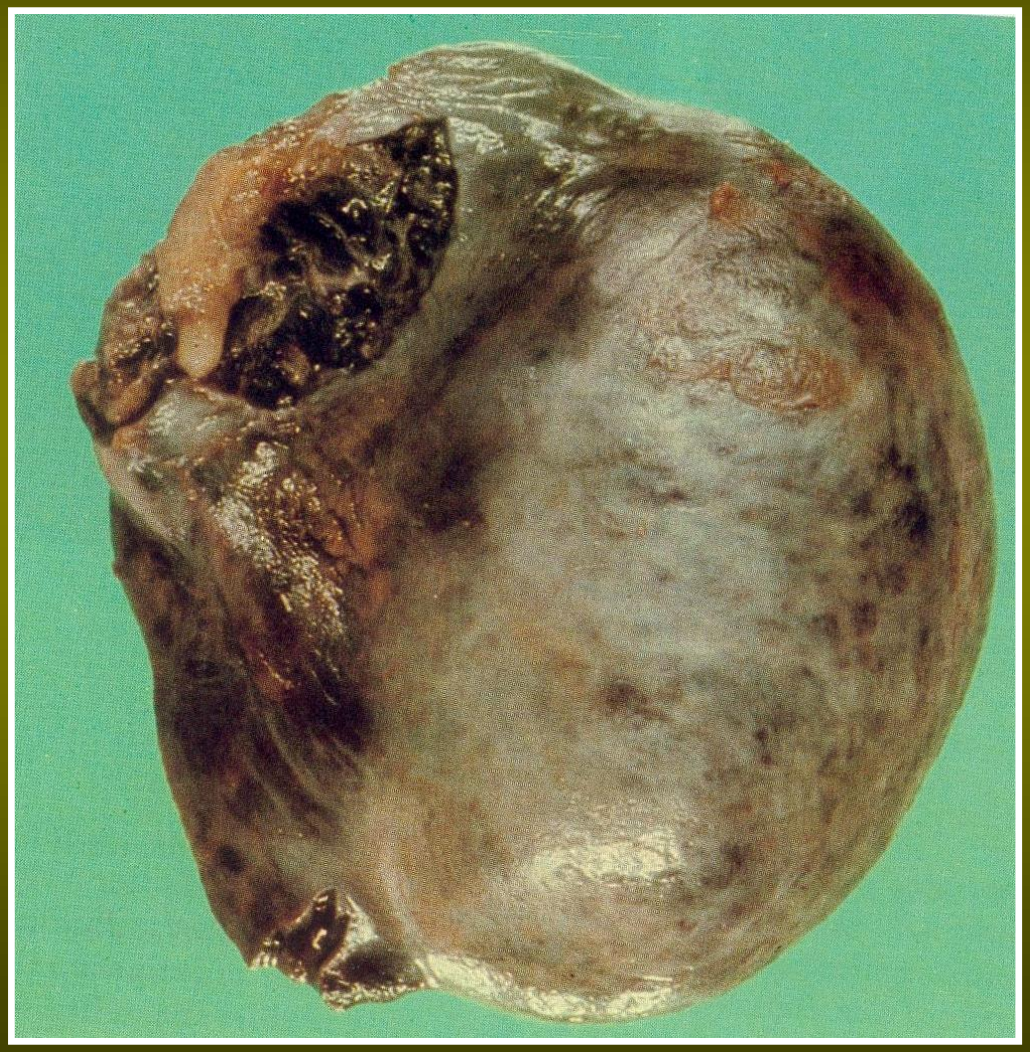
VARICOCELE OF SPERMATIC CORD – DILATED VEINS OF THE PAMPINIFORM PLEXUS

SPERMATOCELE – CYST OF THE EPIDIDYMIS CONTAINING SPERMATOZOA

!!! CYST OF THE MORGAGNI APPENDIX IN BOYS TENDS TO CONVOLUTE

PATHOLOGY OF TESTIS

DISTURBANCES IN CIRCULATION

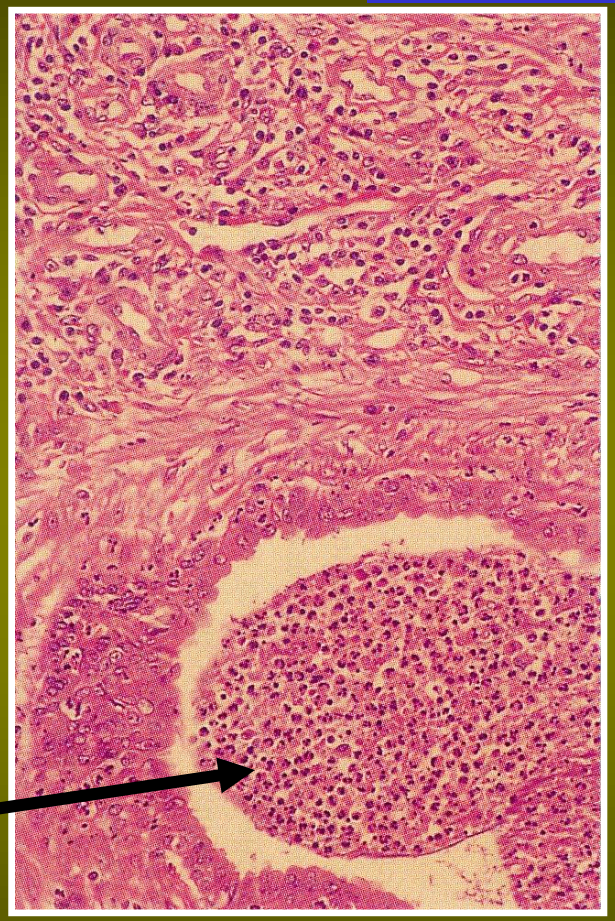


PALE AND HEMORRHAGIC INFARCT

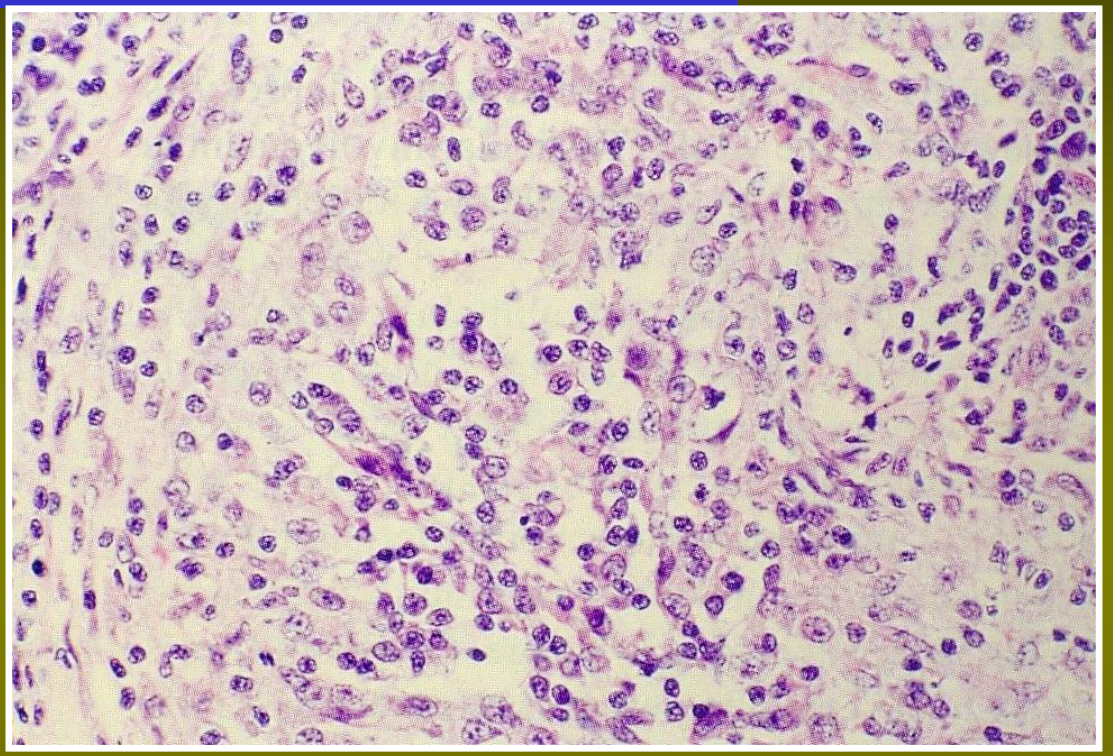
DUE TO EMBOLUS (SPERMATIC INTERNAL ARTERY IS ANATOMICALLY AN END ARTERY); MOSTLY OCCURS AFTER TRAUMA OR TORSION OF THE TESTICULAR PEDUNCLE

PATHOLOGY OF TESTIS

INFLAMMATION OF TESTIS AND EPIDIDYMIS



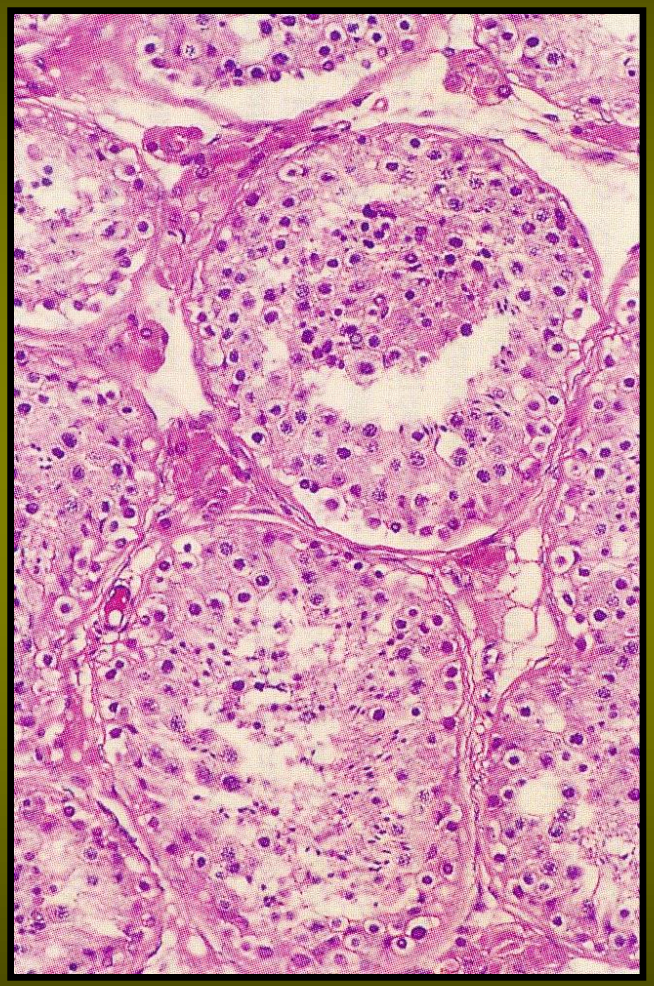
PURULENT EPIDIDYMITIS



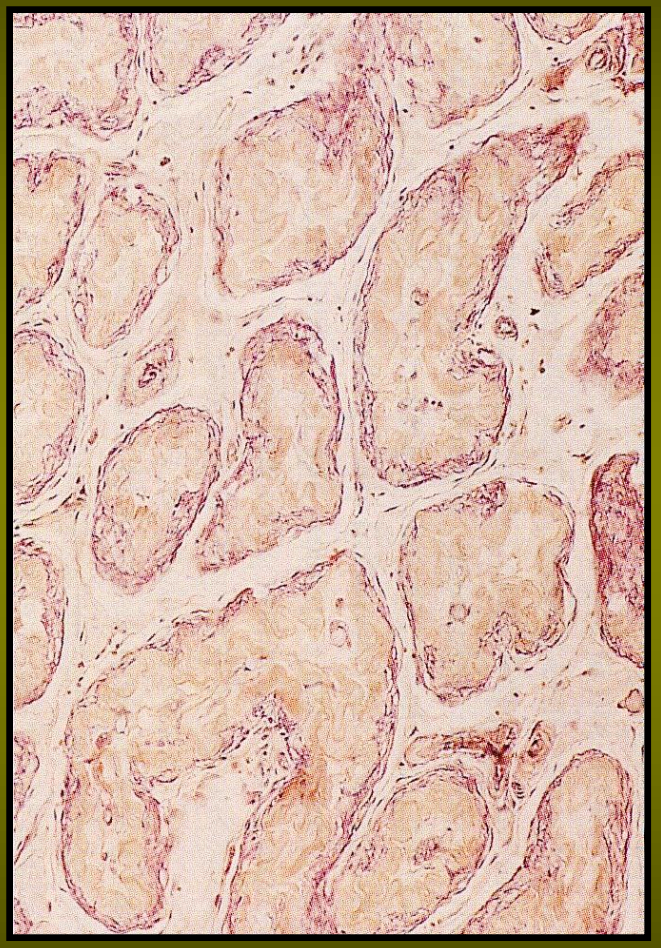
GRANULOMATOUS ORCHITIS – AUTOAGGRESSIVE DISEASE. INFLAMMATION OF PARENCHYMA E.G. IN SYPHILIS. LEADS TO THE DESTRUCTION OF THE PROCREATIVE EPITHELIUM

PATHOLOGY OF TESTIS

ATROPHY



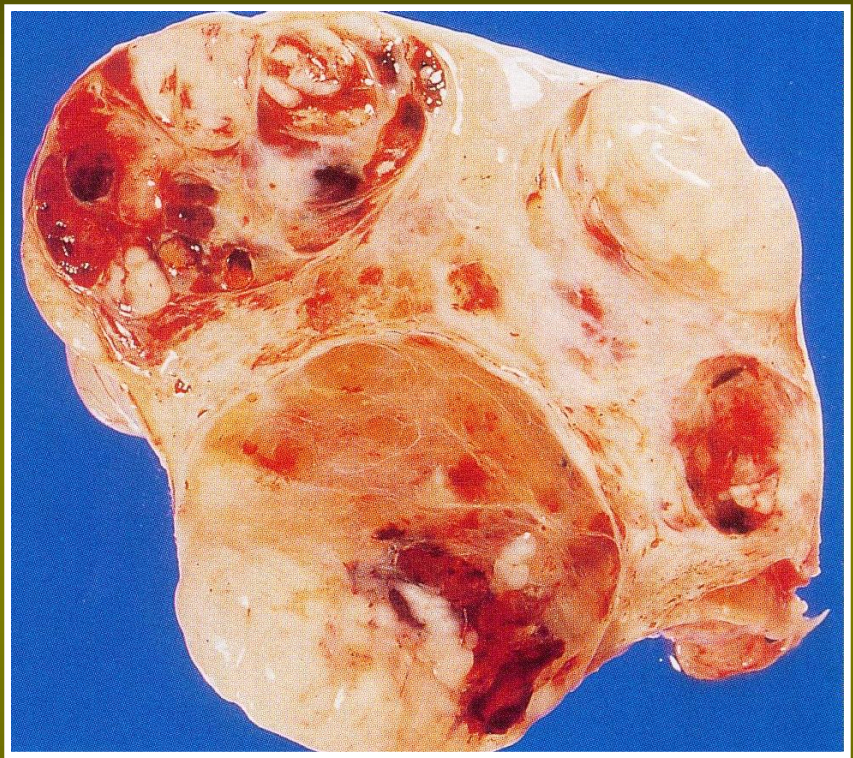
NORMAL TESTIS – ACTIVE SPERMATOGENESIS

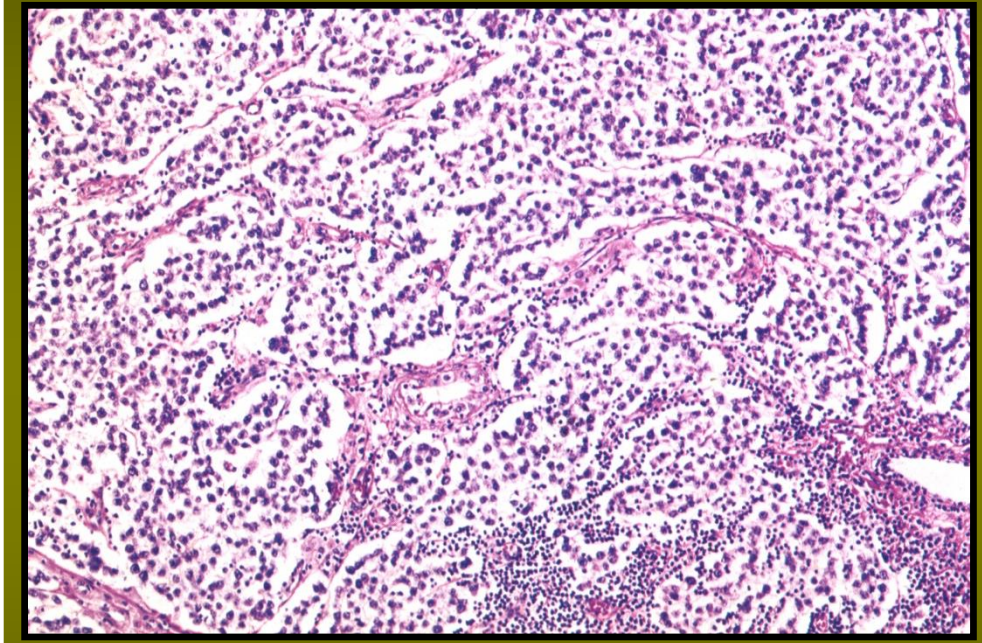


ATROPHY OF TESTIS. HYALINIZATION OF CANALICULI , COMPLETE LACK OF PROCREATIVE EPITHELIUM.

PATHOLOGY OF TESTIS

TUMORS





SEMINOMA

MOST COMMON TESTICULAR TUMOR, DERIVED FROM PRIMARY SEX CELLS – HOMOLOGUE OF DYSGERMINOMA IN OVARIES. CELLS ACCUMULATE GLYCOGEN. LYMPHOCYTIC INFILTRATIONS

SEMINOMA

- Also called classic seminoma
- 30 - 50% of testicular germ cell tumors
- Mean age 40 years versus 25 years for nonseminomatous germ cell tumors (NSGCT); rare in infants
- In ovary, called dysgerminoma
- Also present in mediastinum, pineal gland (germinoma), retroperitoneum

SEMINOMA

- 70% have stage 1 disease
- May metastasize to lymph nodes or bone; late hematogenous spread may occur
- Presence of elevated serum hCG does not change classification and has no clinical significance; however elevated AFP indicates a nonseminomatous germ cell component (or liver disease), even if not seen histologically
- 40% have increased serum PLAP (placental alkaline phosphatase)
- 95% cure rate for stages 1 or 2

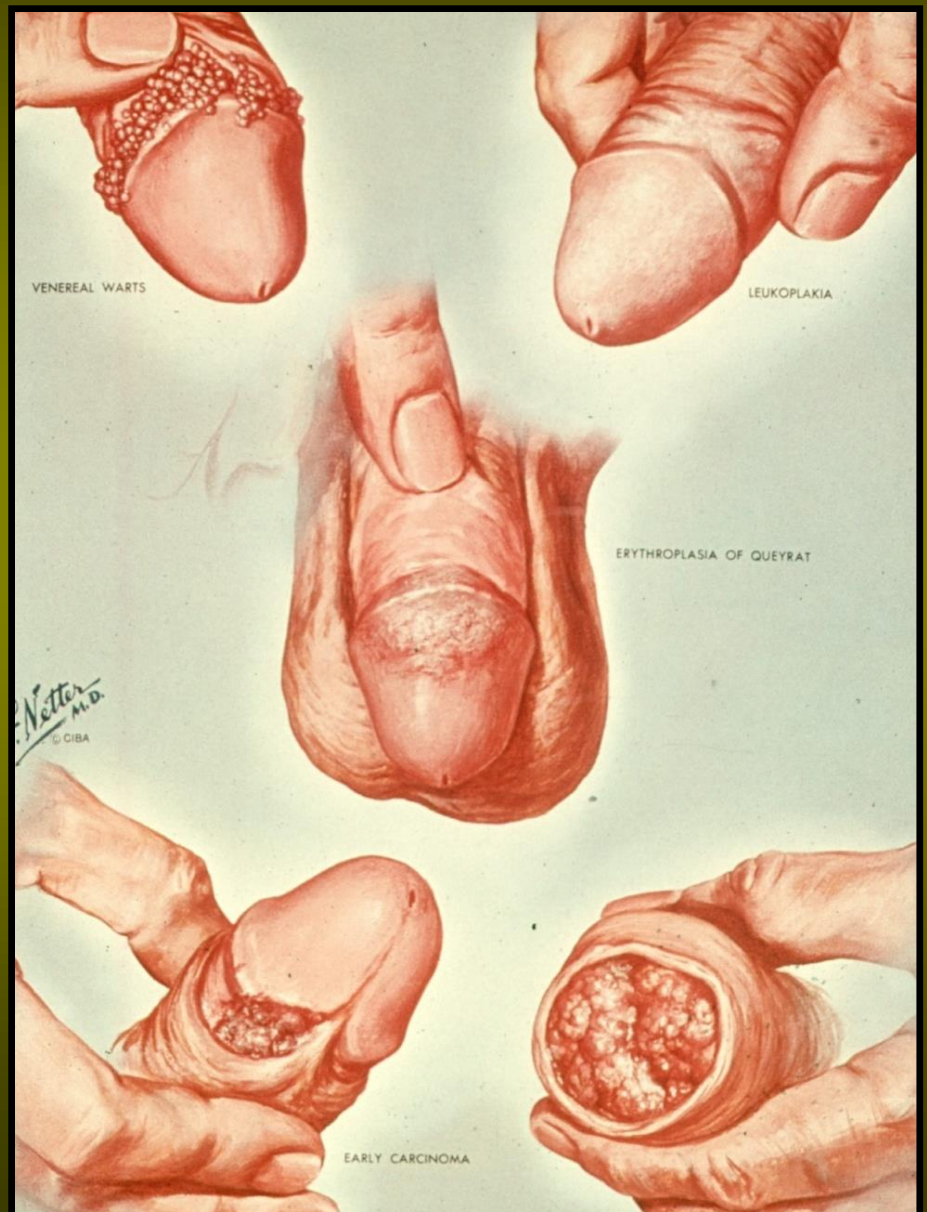
SEMINOMA

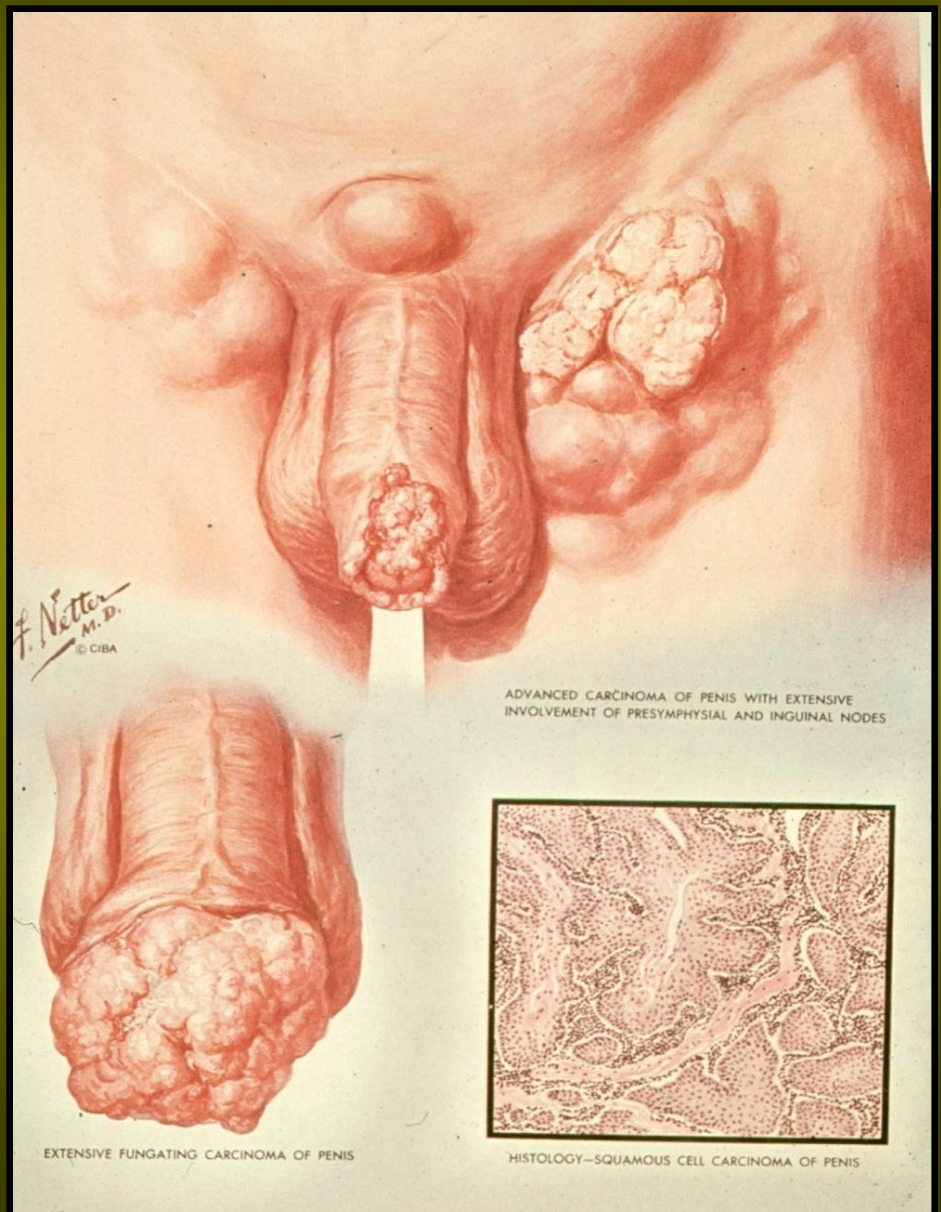
- **Sheets of relatively uniform tumor cells divided into poorly demarcated lobules by delicate fibrous septa with T lymphocytes and plasma cells**
- **Cells are large, round polyhedral with distinct cell membranes, abundant clear/watery cytoplasm (glycogen), large central nuclei, 1 - 2 prominent often elongated and irregular nucleoli**
- **Usually minimal mitotic figures; Tubular preservation may occur at periphery of tumor**
- **10% have significant NSGCT component**


CREDIT
LYONNAIS
CL

TUMORS OF THE EXTERNAL MALE REPRODUCTIVE ORGANS

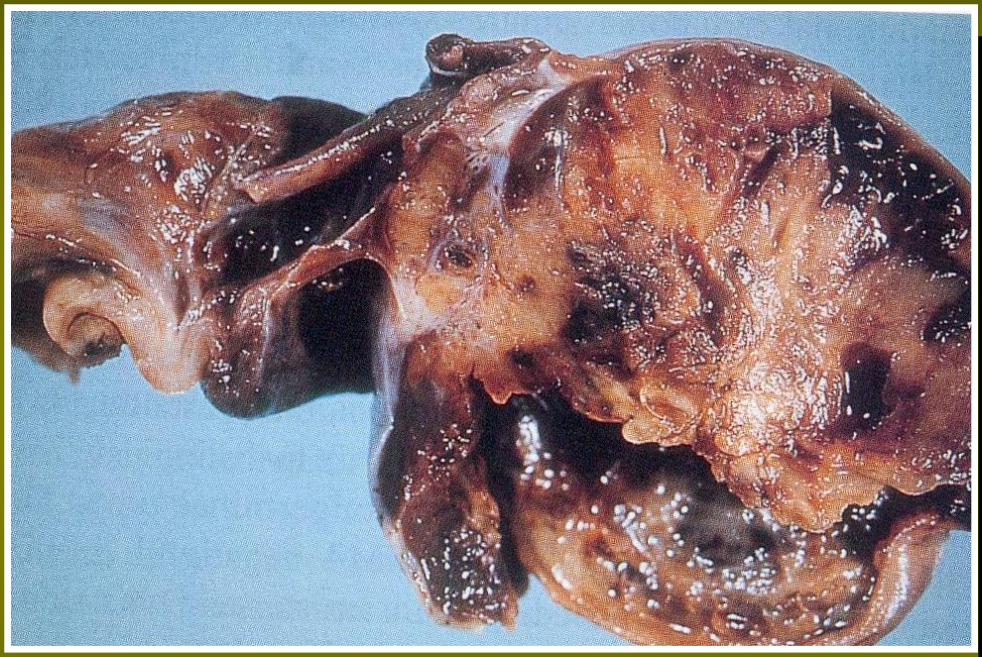
CONDYLOMATA ACUMINATA, PAPILLOMAS, SCC, MELANOMA

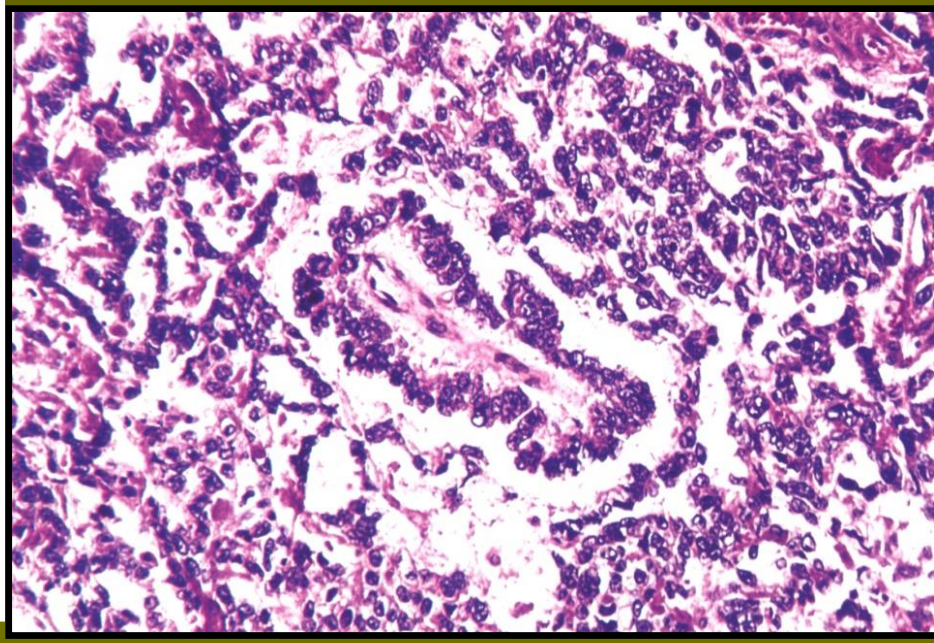




PATHOLOGY OF TESTIS

OTHER TUMORS





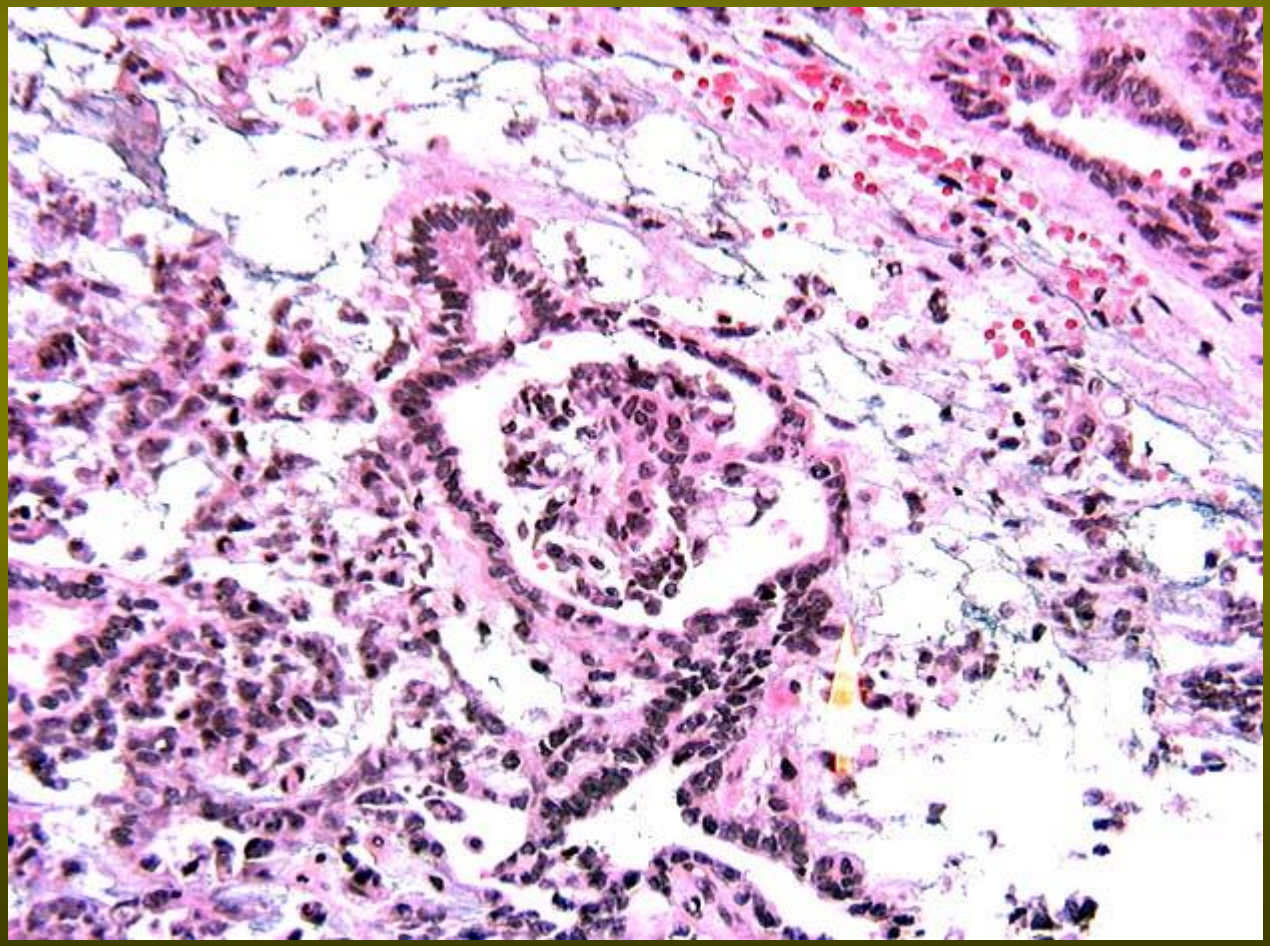
ENDODERMAL SINUS TUMOR – YOLK SAC TUMOR

VERY PRIMITIVE, PRODUCES SIGNIFICANT AMOUNTS OF ALPHA-FETOPROTEIN. ITS STRUCTURE RESEMBLES SCHILLER-DUVAL SINUS IN RAT PLACENTA

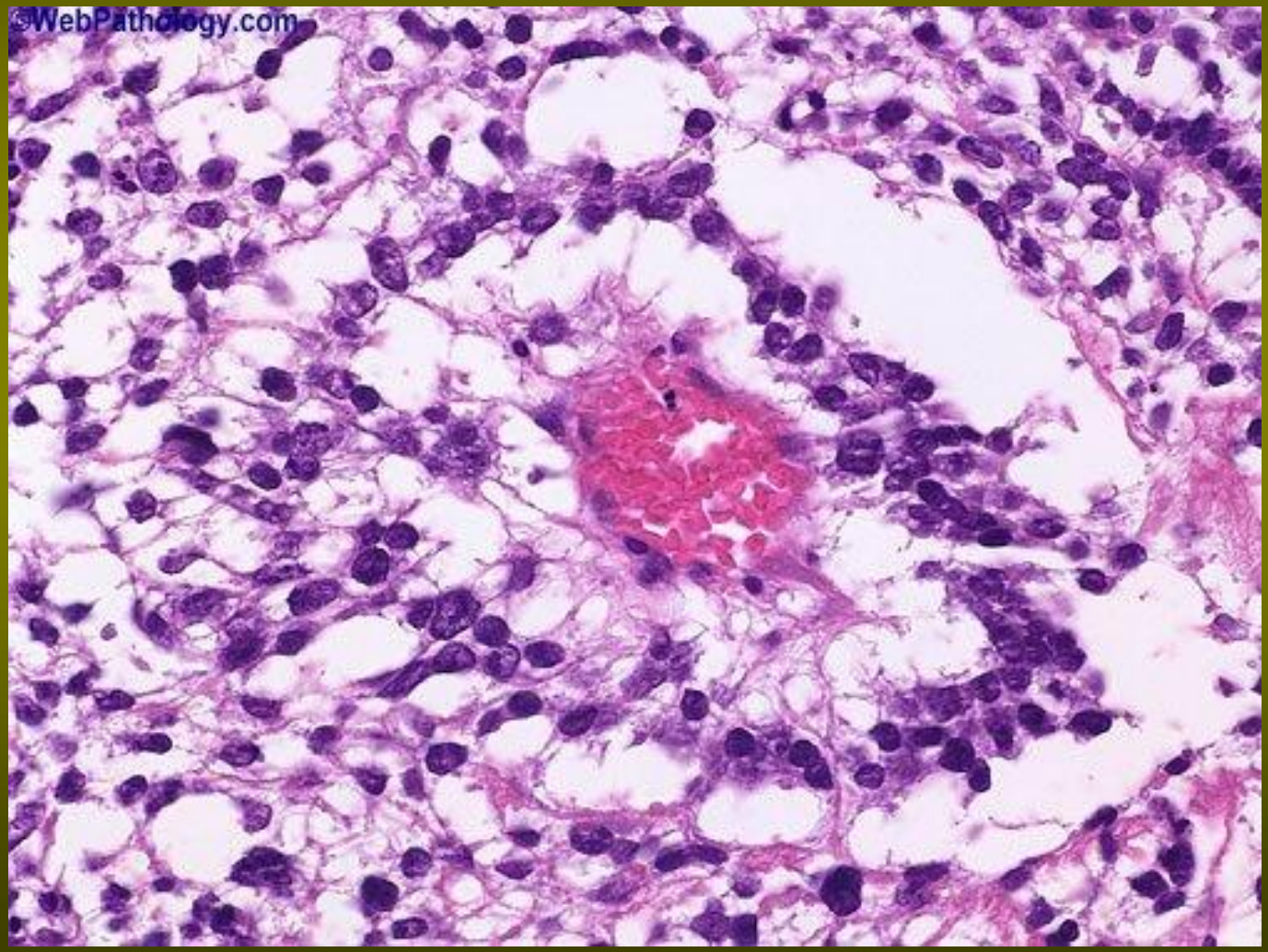
YOLK SAC TUMOR

- Also called endodermal sinus tumor of Teilum (endoderm is embryonic layer closest to yolk sac)
- Numerous patterns that recapitulate the yolk sac, allantois and extra-embryonic mesenchyme
- Most common testicular tumor age 3 or less; often pure; good prognosis at this age (80%+ are stage I)
- Adults: usually part of a mixed tumor, has prognosis of embryonal carcinoma
- 95%+ of patients with tumors containing yolk sac elements have elevated serum AFP although some children have physiologic elevations of AFP without yolk sac tumors

YOLK SAC TUMOR
ENDODERMAL SINUS TUMOR



YOLK SAC TUMOR
ENDODERMAL SINUS TUMOR



SCHILLER -DUVAL BODY

EMBRYONAL CARCINOMA

- **Age at presentation: 30's**
- **Pure in 2% of the cases; most commonly part of mixed germ cell tumor**
- **More than 60% have metastases at presentation**
- **Multiple morphologic patterns**

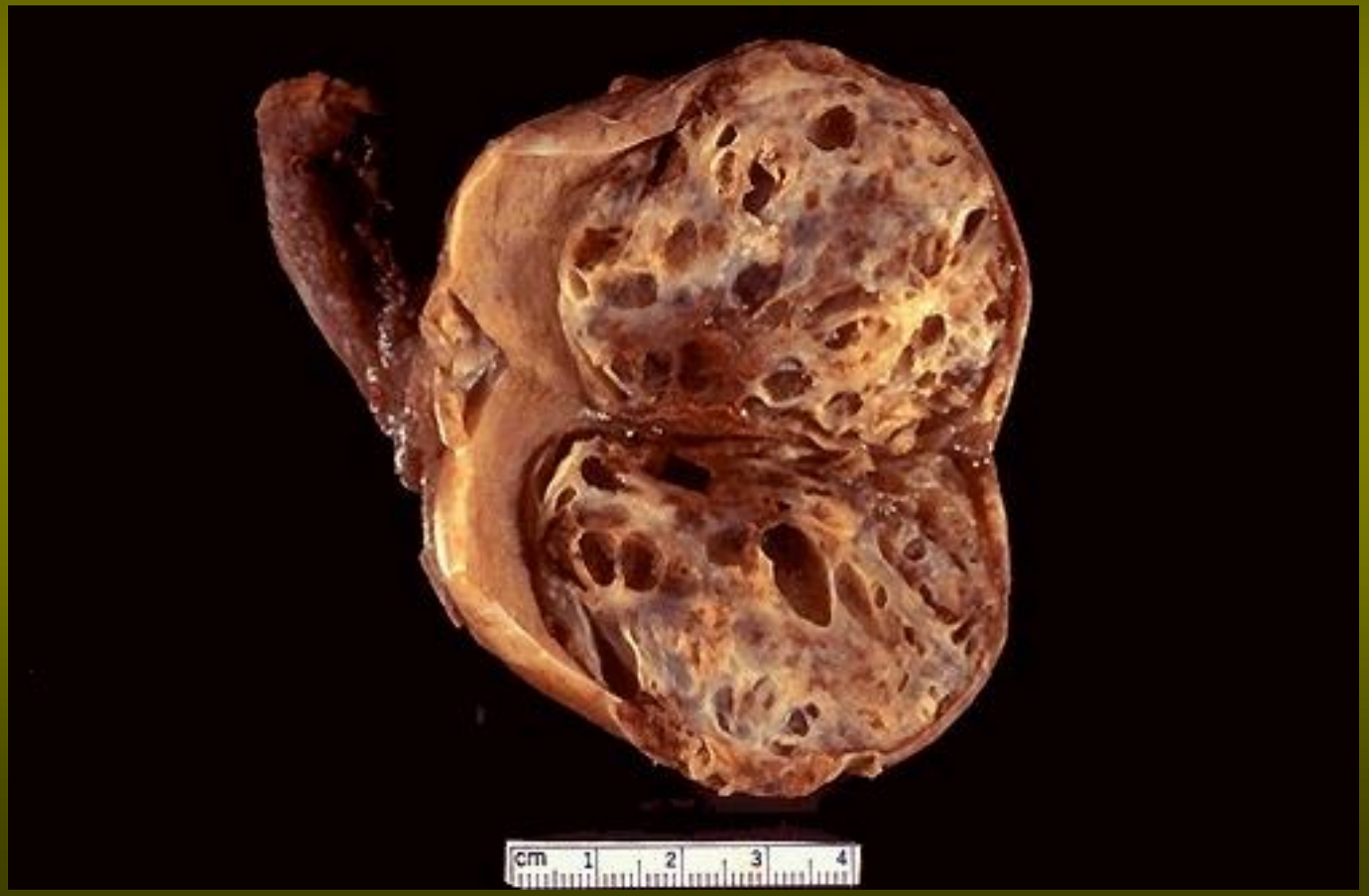
EMBRYONAL CARCINOMA

- **Peak age is 30 years, 10 years younger than seminoma**
- **Rare in prepubertal children**
- **Common as a component of mixed germ cell tumors (GCT) (40% of all mixed GCTs)**
- **Rare in pure form, 2% of all GCTs**

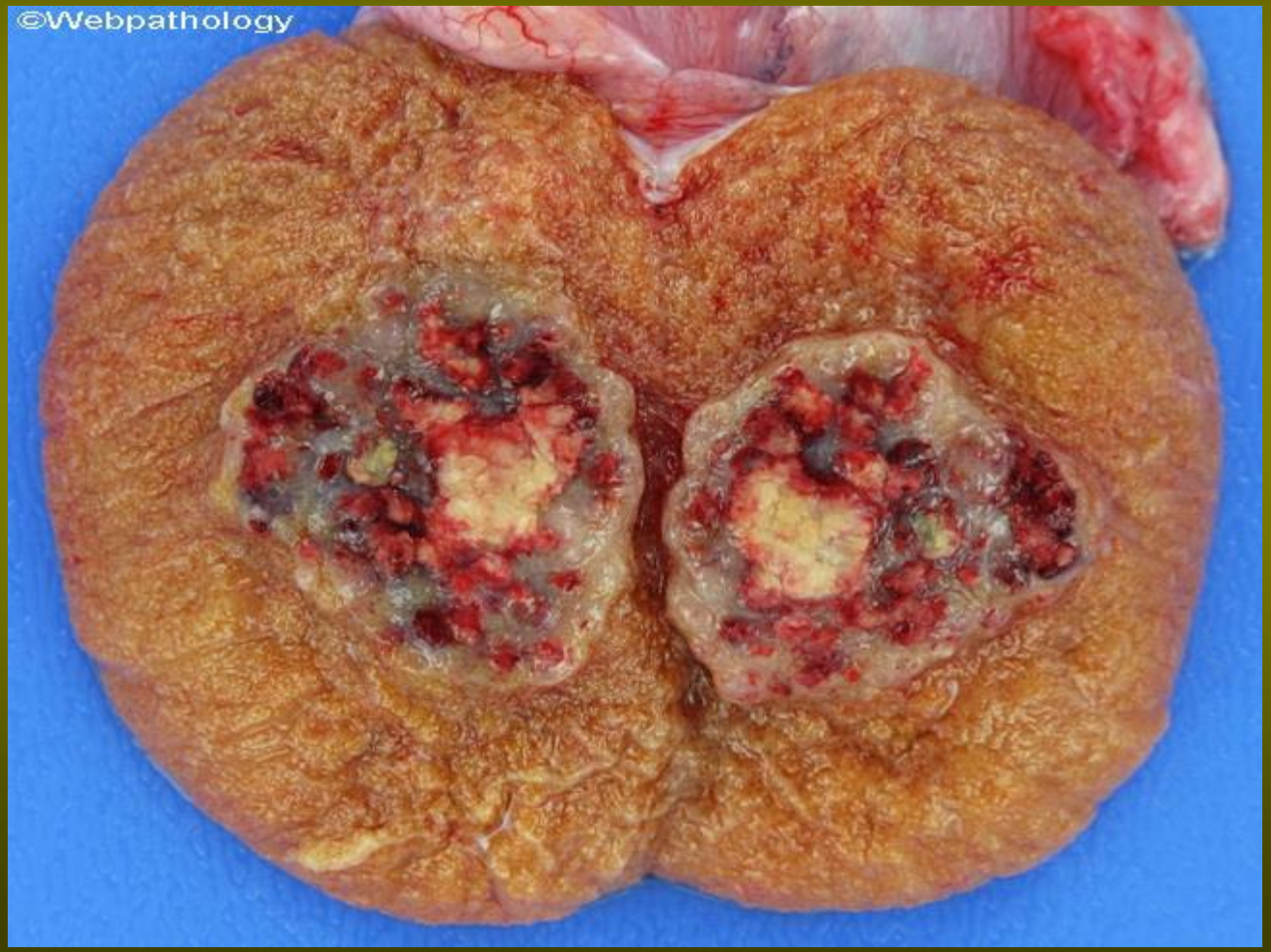
EMBRYONAL CARCINOMA

- **Painless swelling of the testis**
- **Occasionally testicular pain**
- **Gynecomastia**
- **Symptoms related to metastasis**
- **Metastases are common at presentation (60%)**
- **95% cure rate after multimodal therapy including radical orchiectomy, retroperitoneal lymphadenectomy and chemotherapy**

EMBRYONAL CARCINOMA



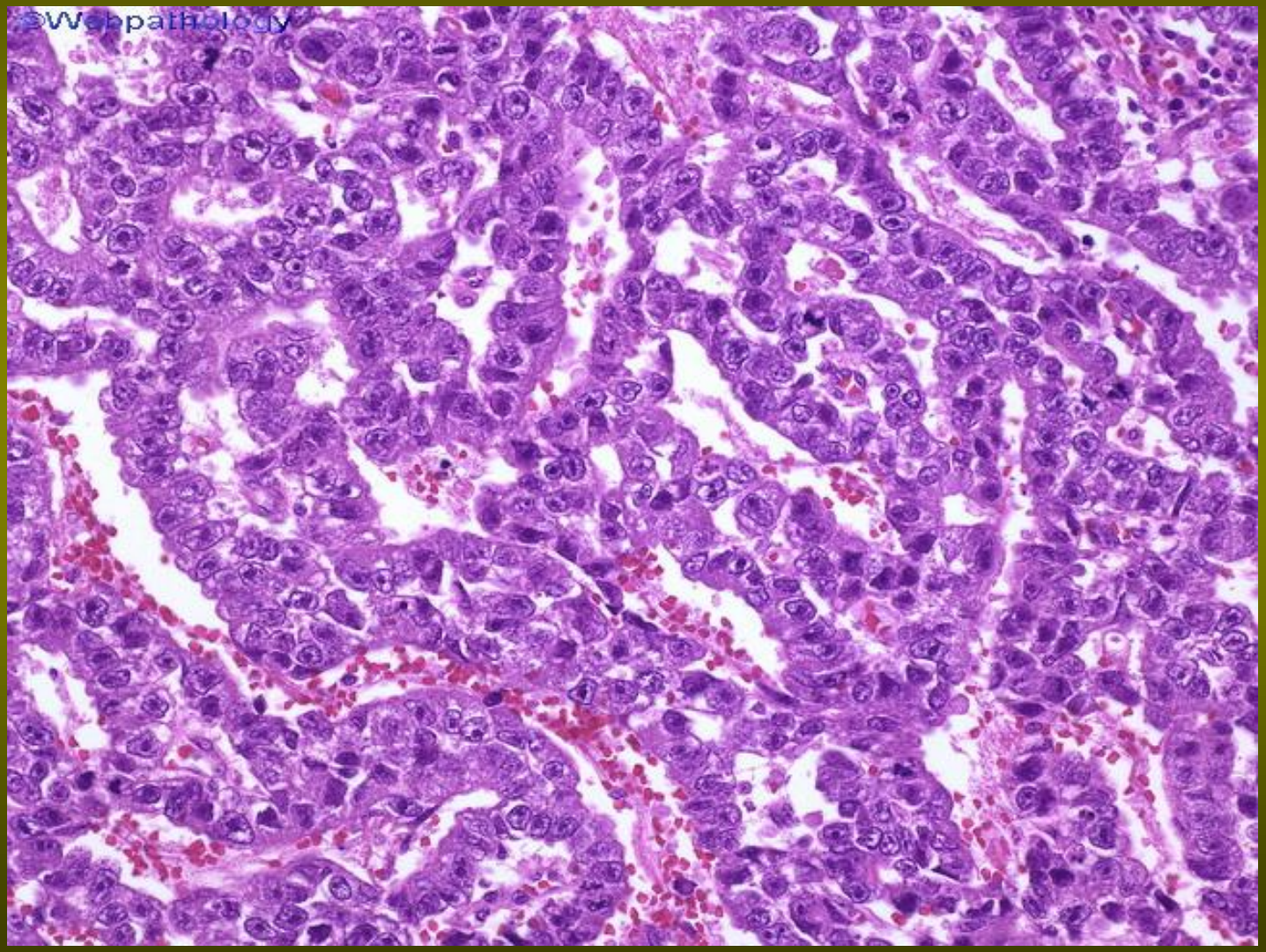
EMBRYONAL CARCINOMA



EMBRYONAL CARCINOMA

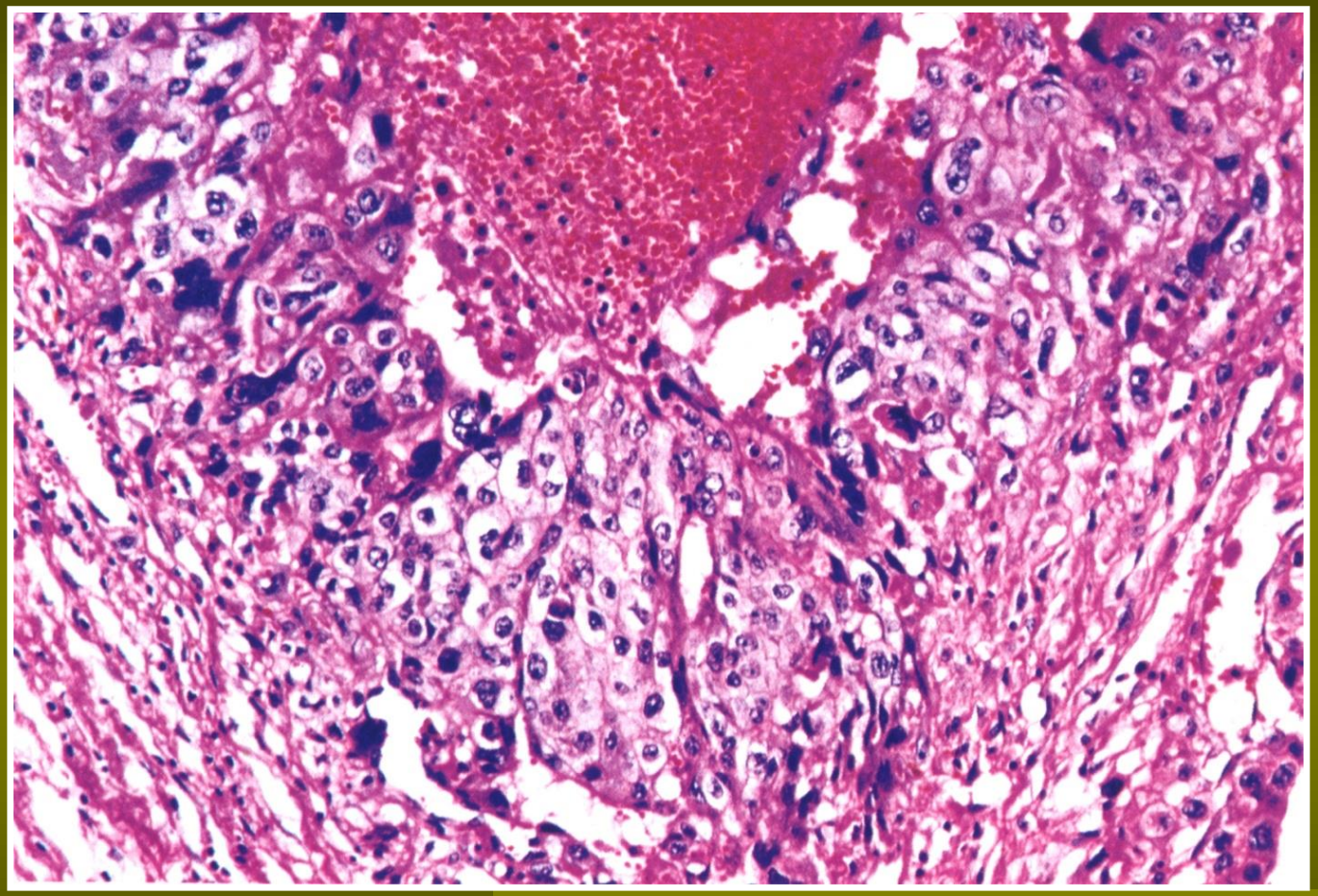
- Solid, pseudoglandular, alveolar, tubular or papillary patterns; Primitive epithelial type cells with minimal features of differentiation
- High grade features of large, epithelioid, anaplastic cells with prominent nucleoli, indistinct cell borders with nuclear overlapping, pleomorphism, frequent mitoses
- Giant cells with granular, pink, amphophilic cytoplasm
- Often mixed with other nonseminomatous germ cell tumors; No distinct fibrous septa
- Intratubular embryonal carcinoma often present adjacent to invasive lesion, often with calcifications
- Stromal component suggests presence of teratoma

EMBRYONAL CARCINOMA



PATHOLOGY OF TESTIS

TUMORS

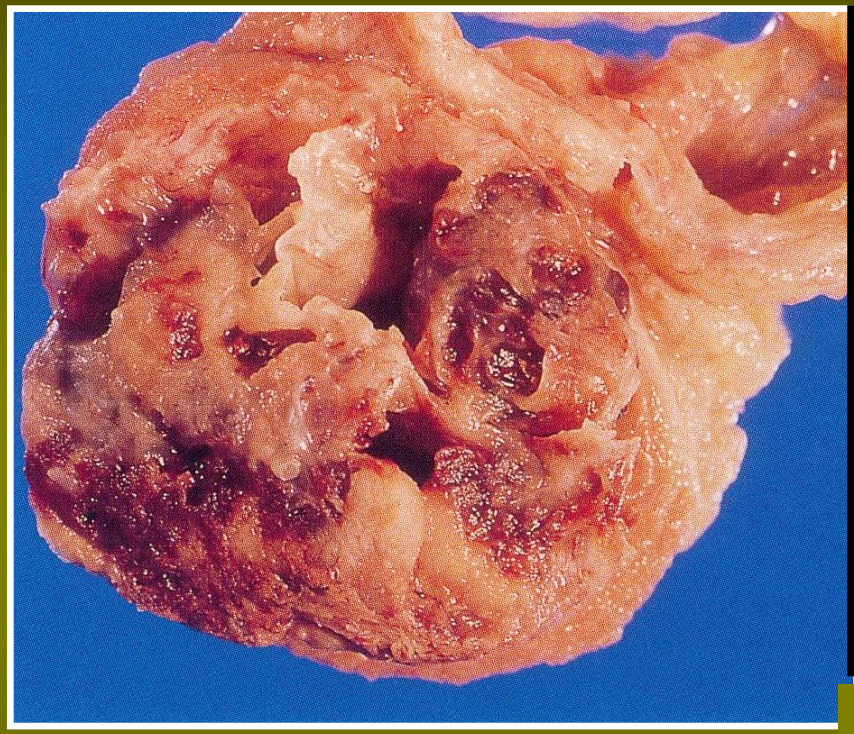


CHORIOEPITHELIOMA = CHORIOCARCINOMA

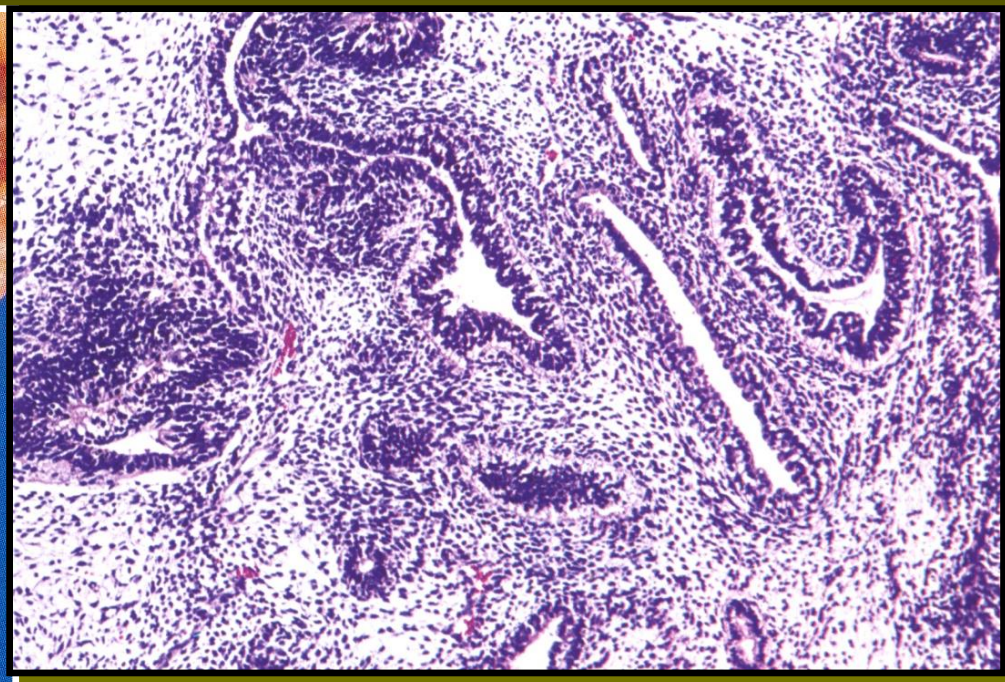
MALIGNANT TUMOR DERIVED FROM TROPHOBLAST (FETAL STRUCTURE) – PRODUCES A SIGNIFICANT AMOUNTS OF GONADOTROPIN – POSITIVE PREGNANCY TEST !!!

PATHOLOGY OF TESTIS

TUMORS



EMBRYONAL TERATOMA

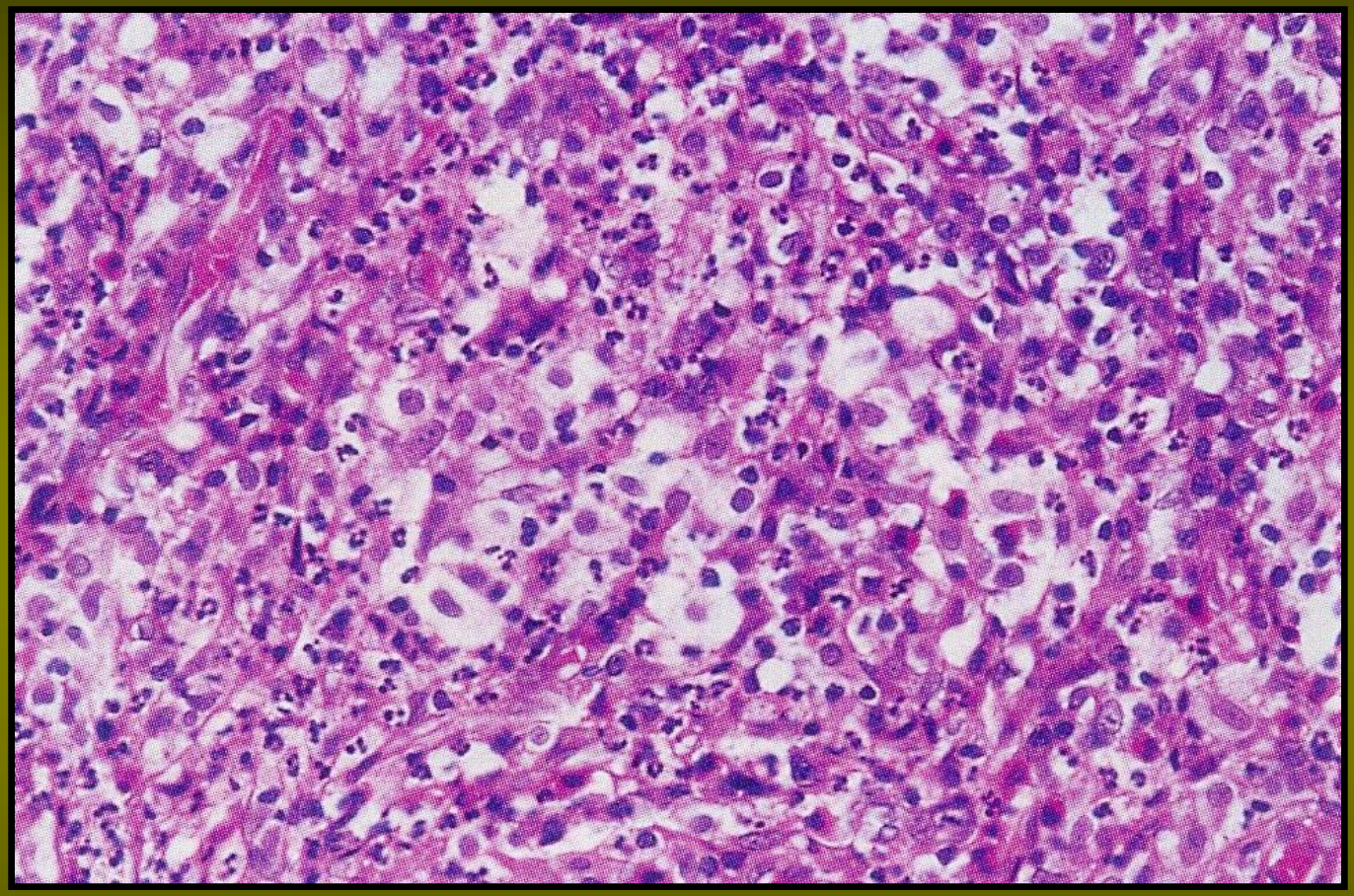


IMMATURE EPITHELIUM AND MESENCHYMA IN THE TUMOR STRUCTURE, OFTEN IN COMBINATION WITH OTHER TUMORS

REMEMBER! 95% OF TESTICULAR TUMORS ARE MALIGNANT. HISTOLOGICAL VERIFICATION OF ANY TYPE OF MASS IN TESTIS IS NECESSARY

PATHOLOGY OF PROSTATE GLAND

PROSTATITIS

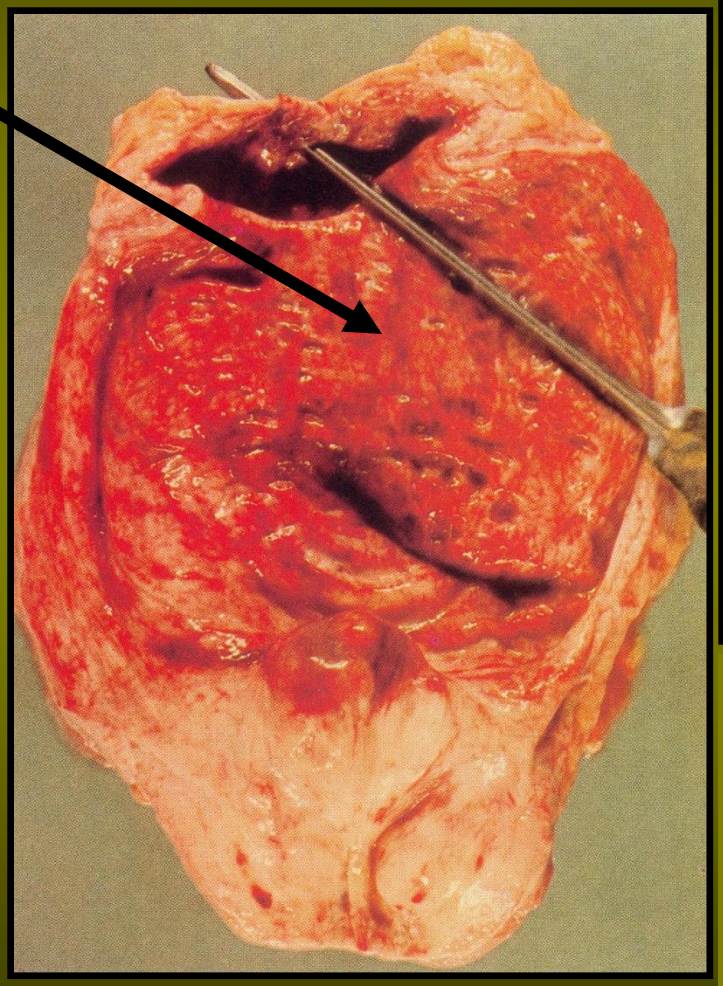


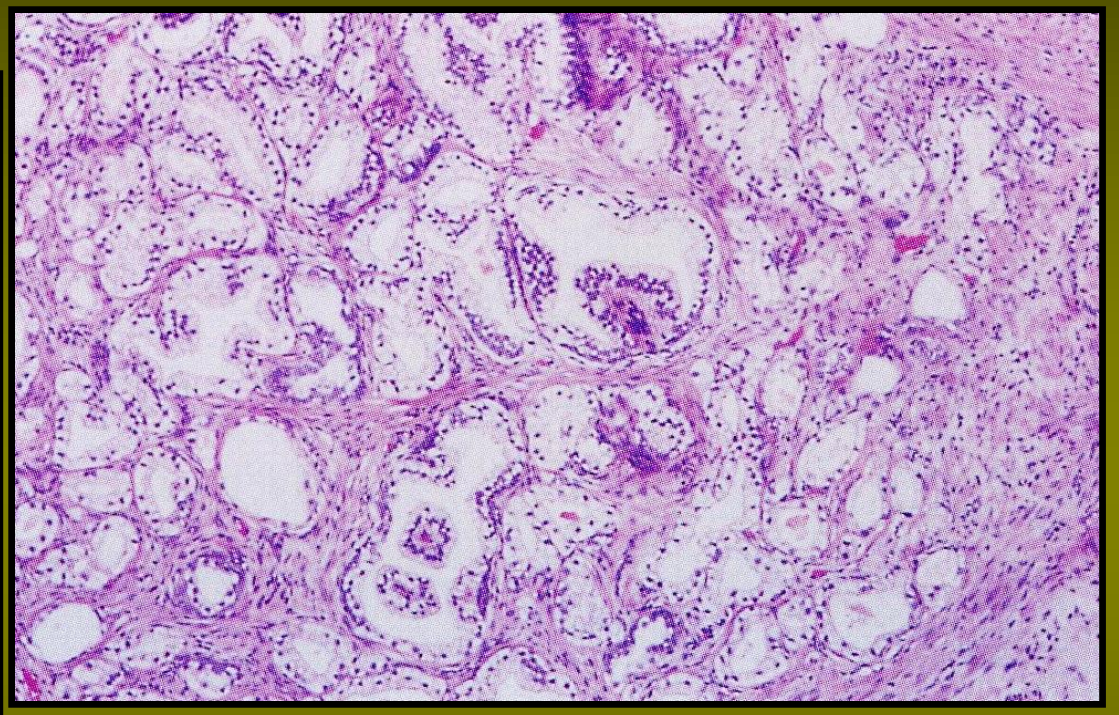
CHRONIC, EXACERBATING PROSTATITIS – *PROSTATITIS CHRONICA EXACEBRANS (*NON-SPECIFIC INFLAMMATION, PURULENT) – COMMON COMPLICATION OF CATHETERISATION

SPECIFIC INFLAMMATION – TUBERCULOSIS – COMMON IN THE CASE OF TUBERCULOSIS IN THE EPIDIDYMIS

PROSTATE GLAND PATHOLOGY

NODULAR HYPERPLASIA – „ADENOMA”

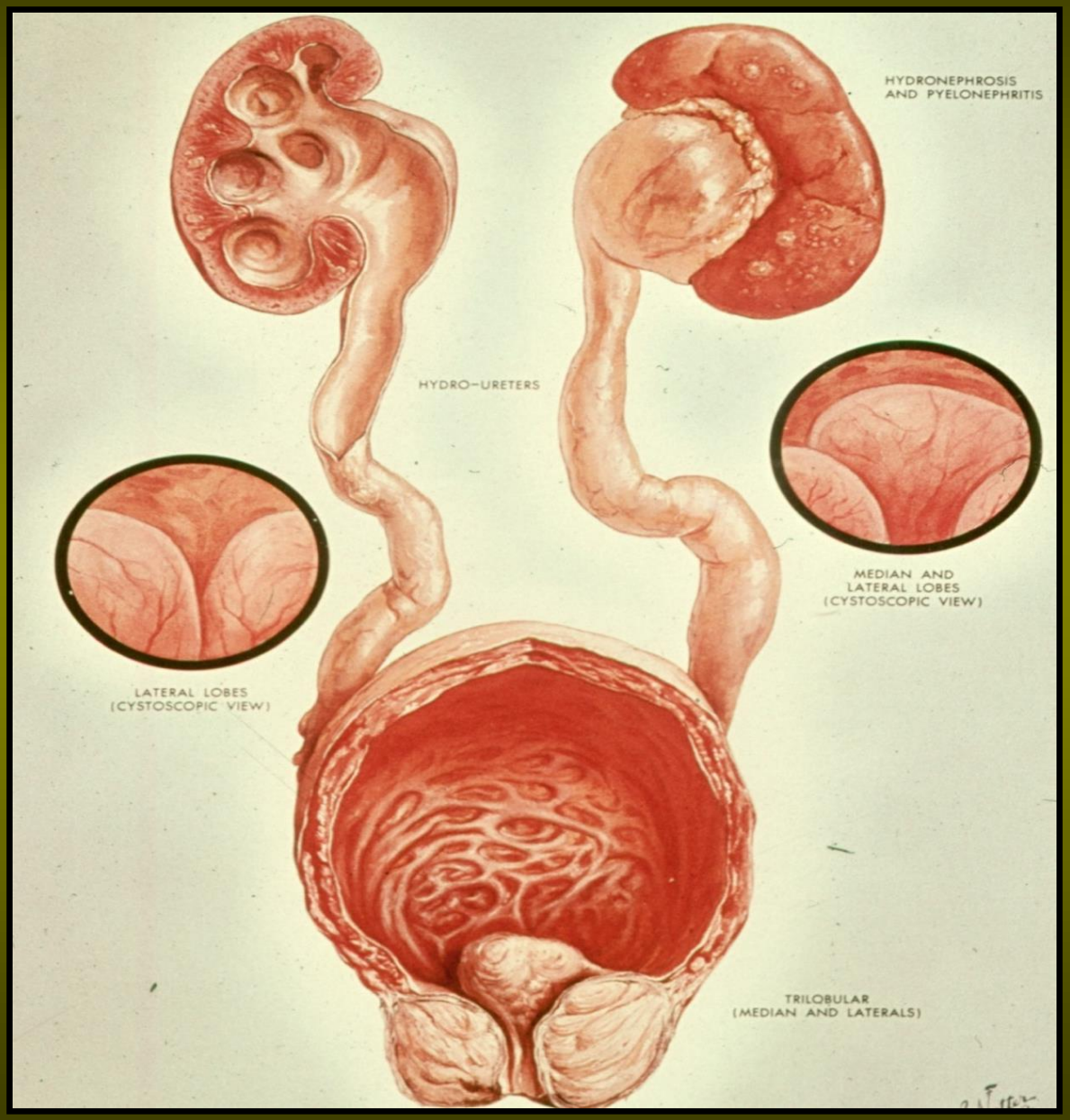




MACROSCOPY AND MICROSCOPY

UP TO 100% OF MEN OVER 70. HYPERPLASIA OF THE GLANDULAR ELEMENTS, COMMONLY OF THE STROMA AND SMOOTH MUSCLES. USUALLY LEADS TO THE CONSTRICTION OF THE PROSTATIC PART OF THE URETHRA

COMPLICATIONS OF THE PROSTATE GLAND HYPERPLASIA



NARROWING OF THE PROSTATIC PART OF THE URETHRA OR TOTAL OBSTRUCTION

HYPERPLASIA AND ECTASIA OF BLADDER

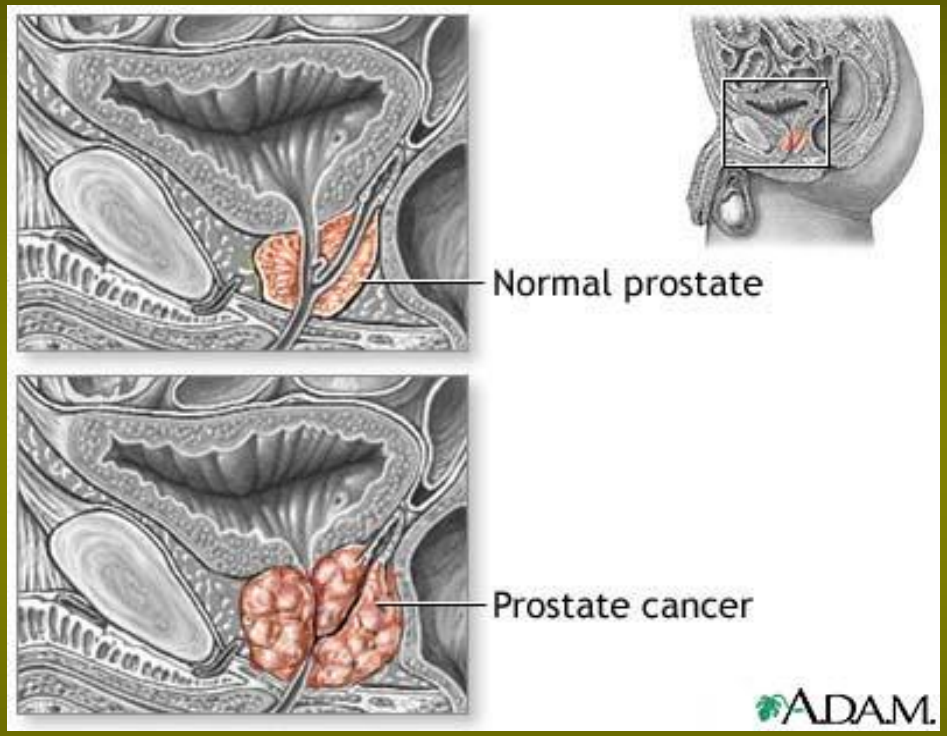
BILATERAL HYDROURETER

BILATERAL HYDRONEPHROSIS

PYELONEPHRITIS

Definitions

- It occurs when cells of the prostate mutate and begin to multiply out of control.
- These cells may spread (metastasize) from the prostate to other parts of the body, especially the bones and lymph nodes. Prostate cancer may cause pain, difficulty in urinating, erectile dysfunction and other symptoms.



Backround

- Prostate cancer develops most frequently in men over fifty. It is the most common type of cancer in men in the United States, where it is responsible for more male deaths than any other cancer.
- However, many men who develop prostate cancer never have symptoms, undergo no therapy, and eventually die of other causes. Many factors, including genetics and diet, have been implicated in the development of prostate cancer.

Symptoms

- Both early and advanced carcinoma of the prostate may be asymptomatic at the time of diagnosis.
- In symptomatic subjects symptoms may be:
  - Dysuria, difficulty in voiding, increased urinary frequency, complete urinary retention, back or hip pain, and hematuria.

Pathophysiology

- Prostate cancer is classified as an adenocarcinoma, or glandular cancer, that begins when normal semen-secreting prostate gland cells mutate into cancer cells. The region of prostate gland where the adenocarcinoma is most common is the peripheral zone. Initially, small clumps of cancer cells remain confined to otherwise normal prostate glands, a condition known as carcinoma in situ or prostatic intraepithelial neoplasia (PIN).

Causes

- A man's risk of developing prostate cancer is related to his age, genetics, race, diet, lifestyle, medications, and other factors. The primary risk factor is age.
- The average age at time of diagnosis is 70
- Genetic backround contributes to a risk of developing prostate cancer.
- Dietary amounts of certain foods, vitamins and minerals can contribute to prostate cancer risk
- Trans fats from the hydrogenation of vegetable oils
- Lower blood levels of vitamin D
- Intake of animal fat

Prevention

- Several medications and vitamins may also help prevent prostate cancer. Two dietary supplements, vitamin E and selenium, may help prevent prostate cancer when taken daily. Estrogens from fermented soybeans and other plant sources (called phytoestrogens) may also help prevent prostate cancer. The selective estrogen receptor modulator drug toremifene has shown promise in early trials. Two medications which block the conversion of testosterone to dihydrotestosterone, finasteride and dutasteride.

Prevention

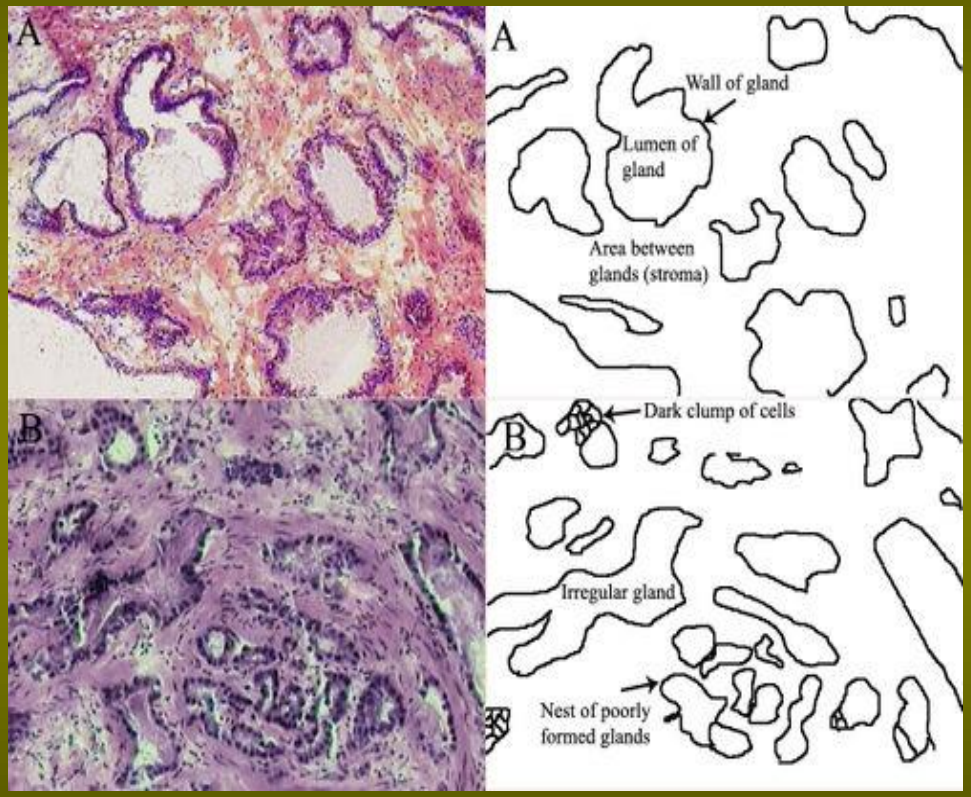
The chemical found in peppers, has been shown to cause 80% of cancerous prostate cells to undergo apoptosis in mice. For prostate cancer cells whose growth is dependent upon testosterone, Capsaicin curbed the proliferation of such cells by freezing the cells in a non-proliferate state, and cancerous prostate cells that are androgen independent "suicided" as well.

Screening

- **Prostate cancer screening is an attempt to find unsuspected cancers. Screening tests may lead to more specific follow-up tests such as a biopsy, where small pieces of the prostate are removed for closer study. Prostate cancer screening options include the digital rectal exam and the prostate specific antigen (PSA) blood test.**
- **Digital rectal examination (DRE) is a procedure where the examiner inserts a gloved, lubricated finger into the rectum to check the size, shape, and texture of the prostate.**
- **The PSA test measures the blood level of prostate-specific antigen, an enzyme produced by the prostate. Specifically, PSA is a serine protease similar to kallikrein.**

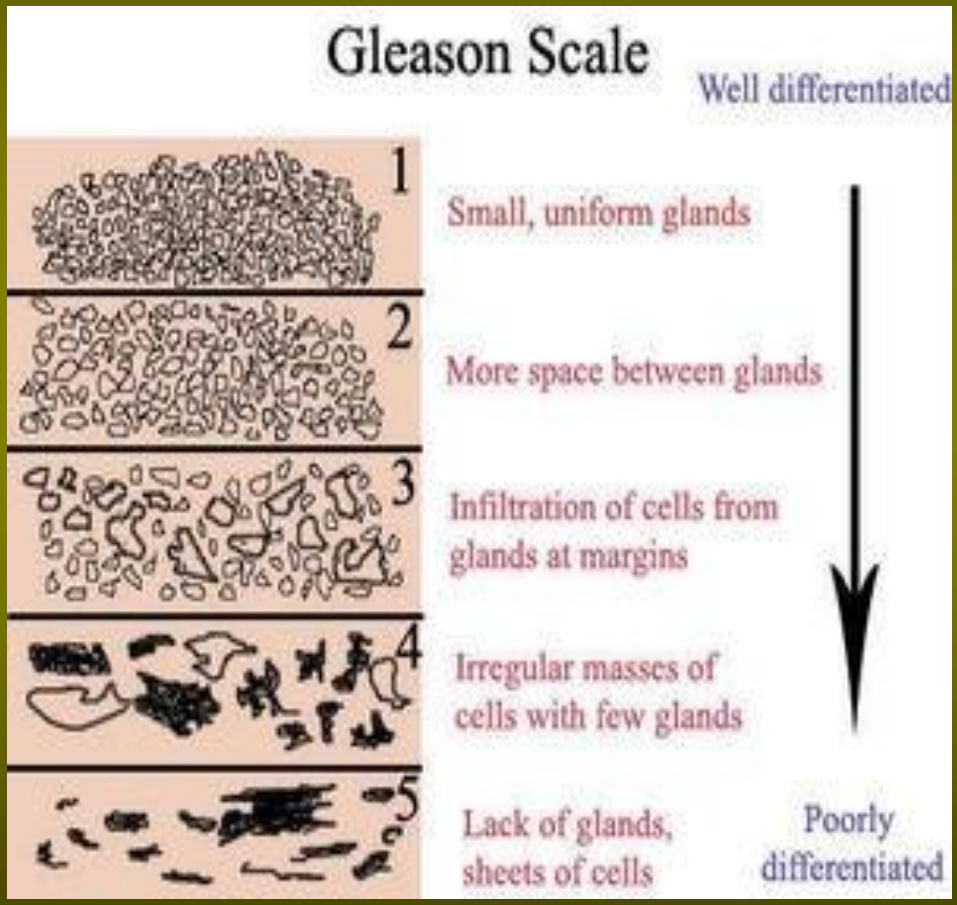
Diagnosis

- If cancer is suspected, a biopsy is offered, the removal of small pieces of the prostate for microscopic examination.



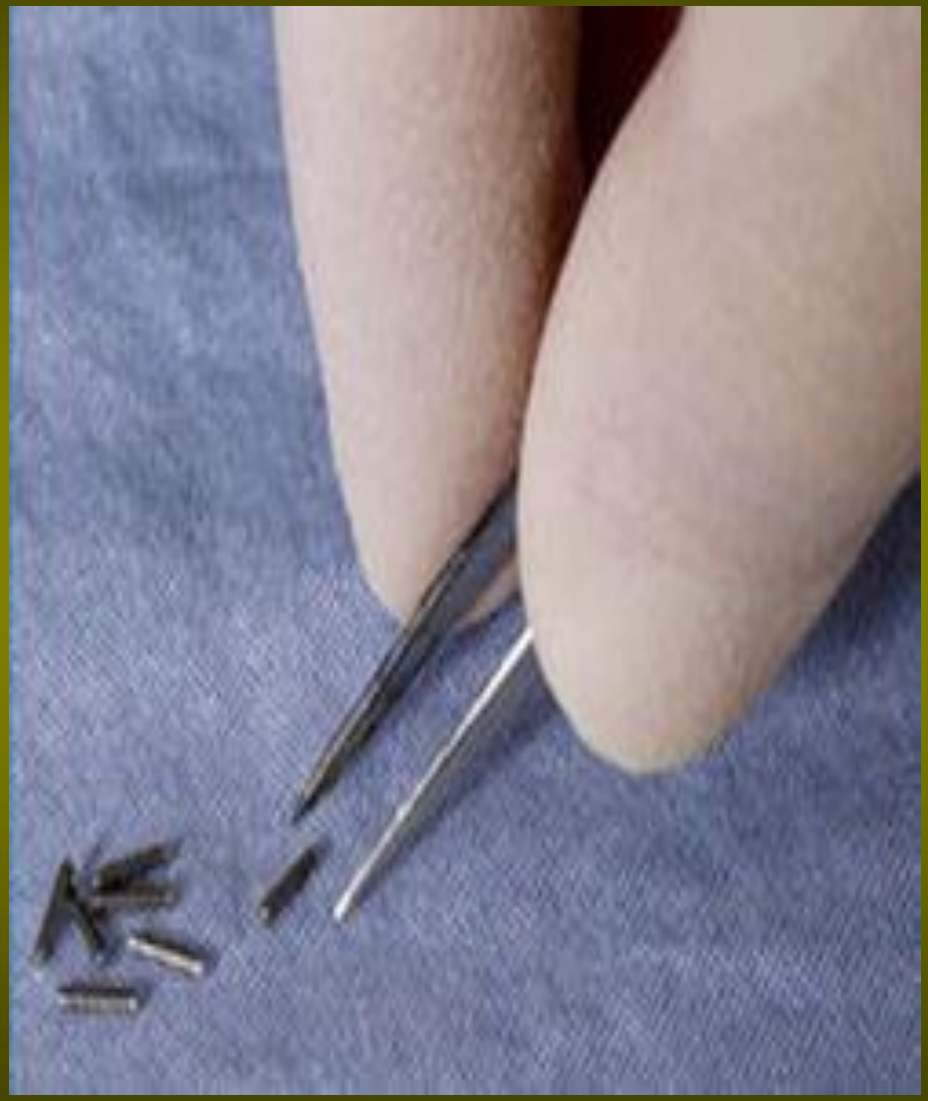
Gleason Scale

- **A Gleason score is given to prostate cancer based upon its microscopic appearance. The Gleason score is important because higher Gleason scores are associated with worse prognosis. This is because higher Gleason scores are given to cancer which is more aggressive.**

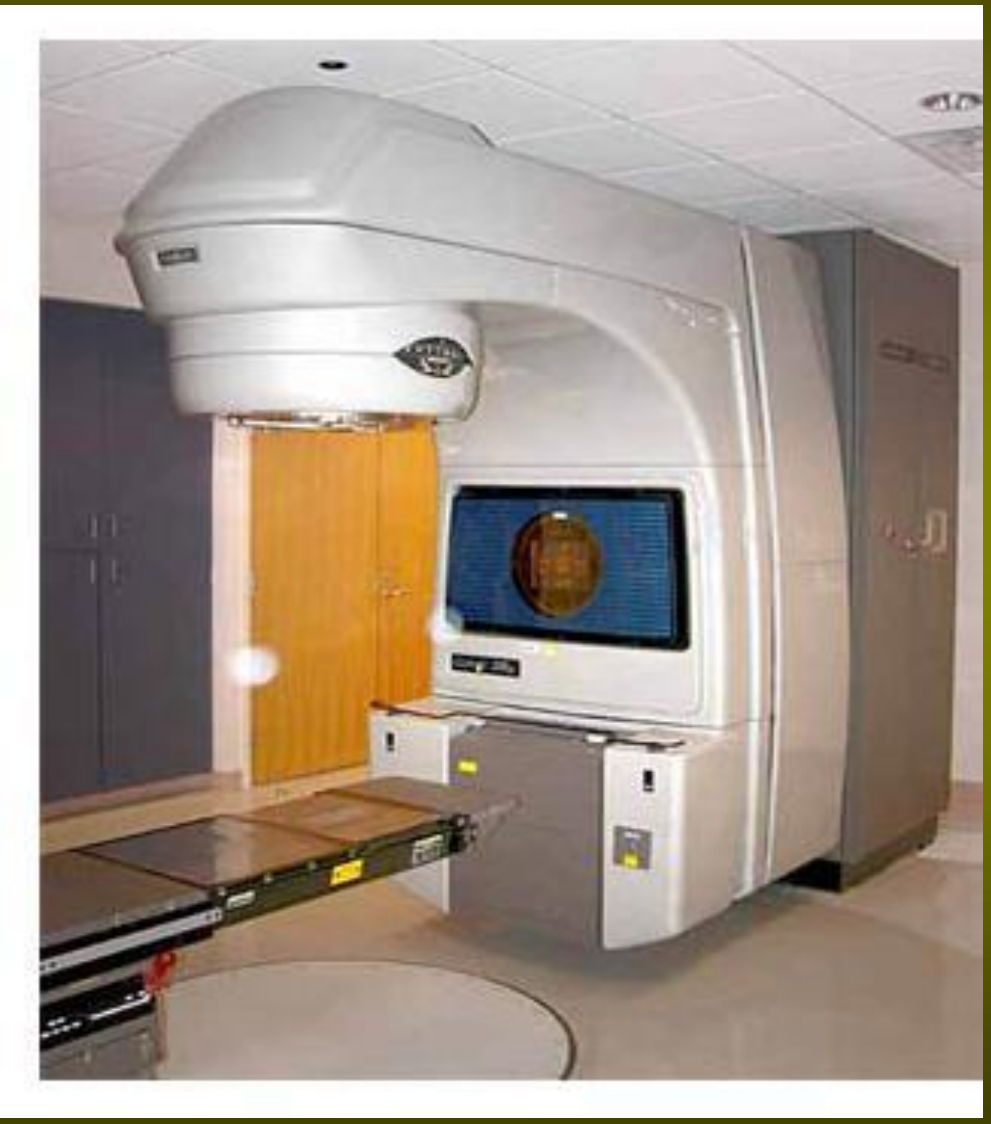


Treatment

- **Treatment for prostate cancer may involve surgery, radiation therapy, High Intensity Focused Ultrasound (HIFU), chemotherapy, cryosurgery, hormonal therapy, or some combinations.**
- **Which option is best depends on the stage of the disease, the Gleason score, and the PSA level. Other important factors are the man's age, his general health, and his feelings about potential treatments and their possible side effects. Because all treatments can have significant side effects, such as erectile dysfunction and urinary incontinence, treatment discussions often focus on balancing the goals of therapy with the risks of lifestyle alterations.**



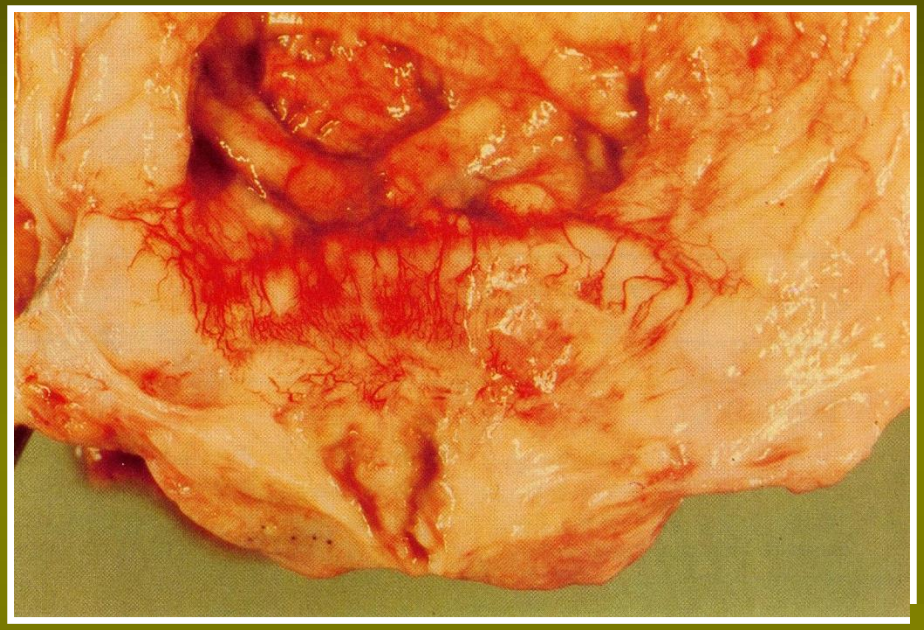
- Brachytherapy for prostate cancer is administered using "seeds," small radioactive rods implanted directly into the tumor.



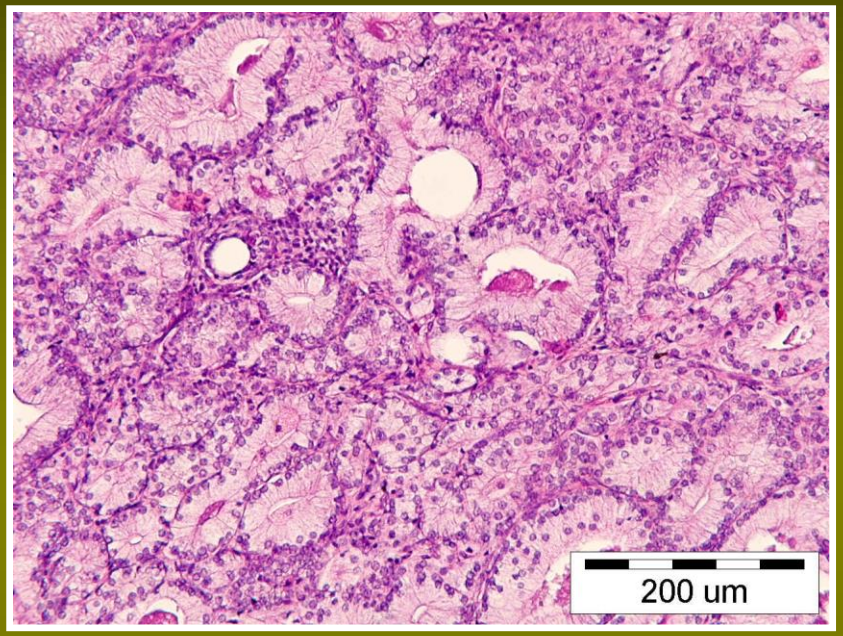
- External beam radiation therapy uses a linear accelerator to produce high-energy x-rays which are directed in a beam towards the prostate.

PROSTATE GLAND PATHOLOGY

CARCINOMA



CARCINOMA

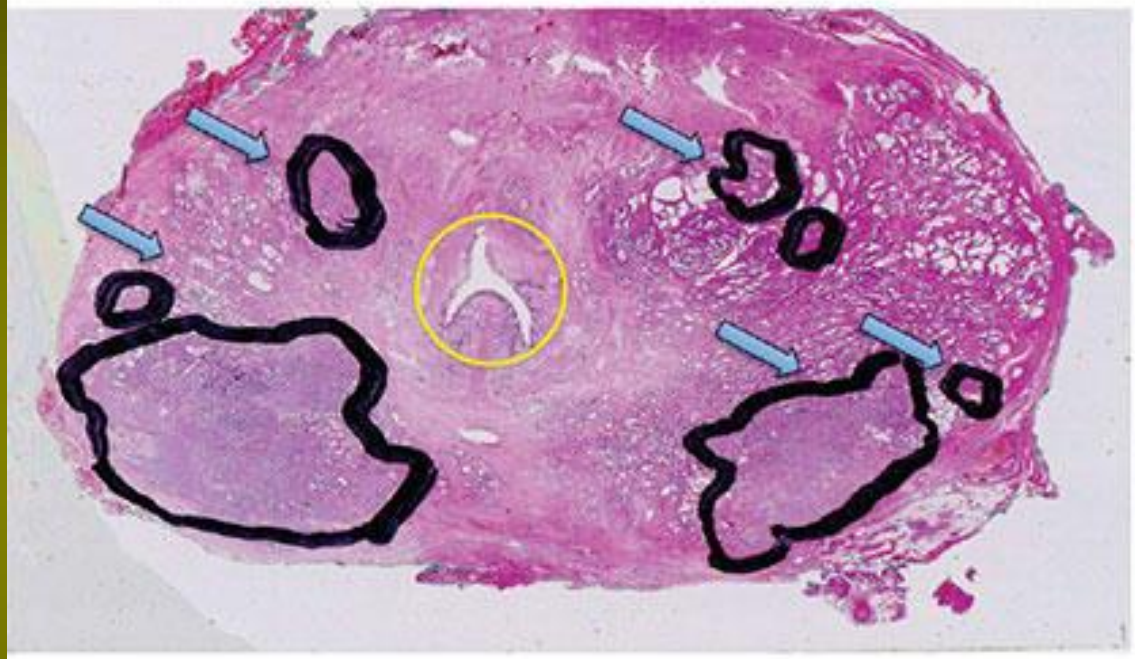


HIGHLY DIFFERENTIATED CARCINOMA OF THE PROSTATE GLAND

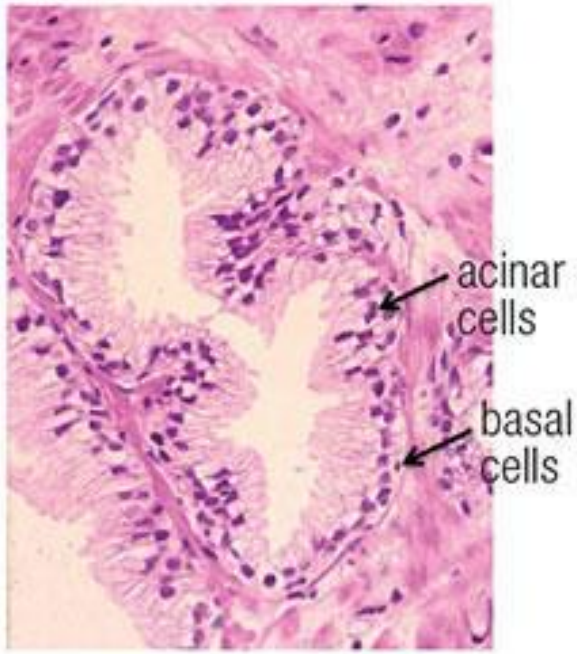
GROWS SLOWLY. EARLY METASTASES TO BONES. TWO BIOCHEMICAL MARKERS ARE INVOLVED: ACID PHOSPHATASE AND PSA (PROSTATIC SPECIFIC ANTIGEN) WHICH MAKE THE DIAGNOSIS EASIER AND ALLOW OBSERVATION OF THE PROGRESS OF DISEASE. GLEASON HISTOLOGICAL CLASSIFICATION AS A PROGNOSTIC MARKER.

Prostate cancer is often multifocal

(Whole section through the prostate gland; blue arrow: prostate cancer, yellow circle: urethra)



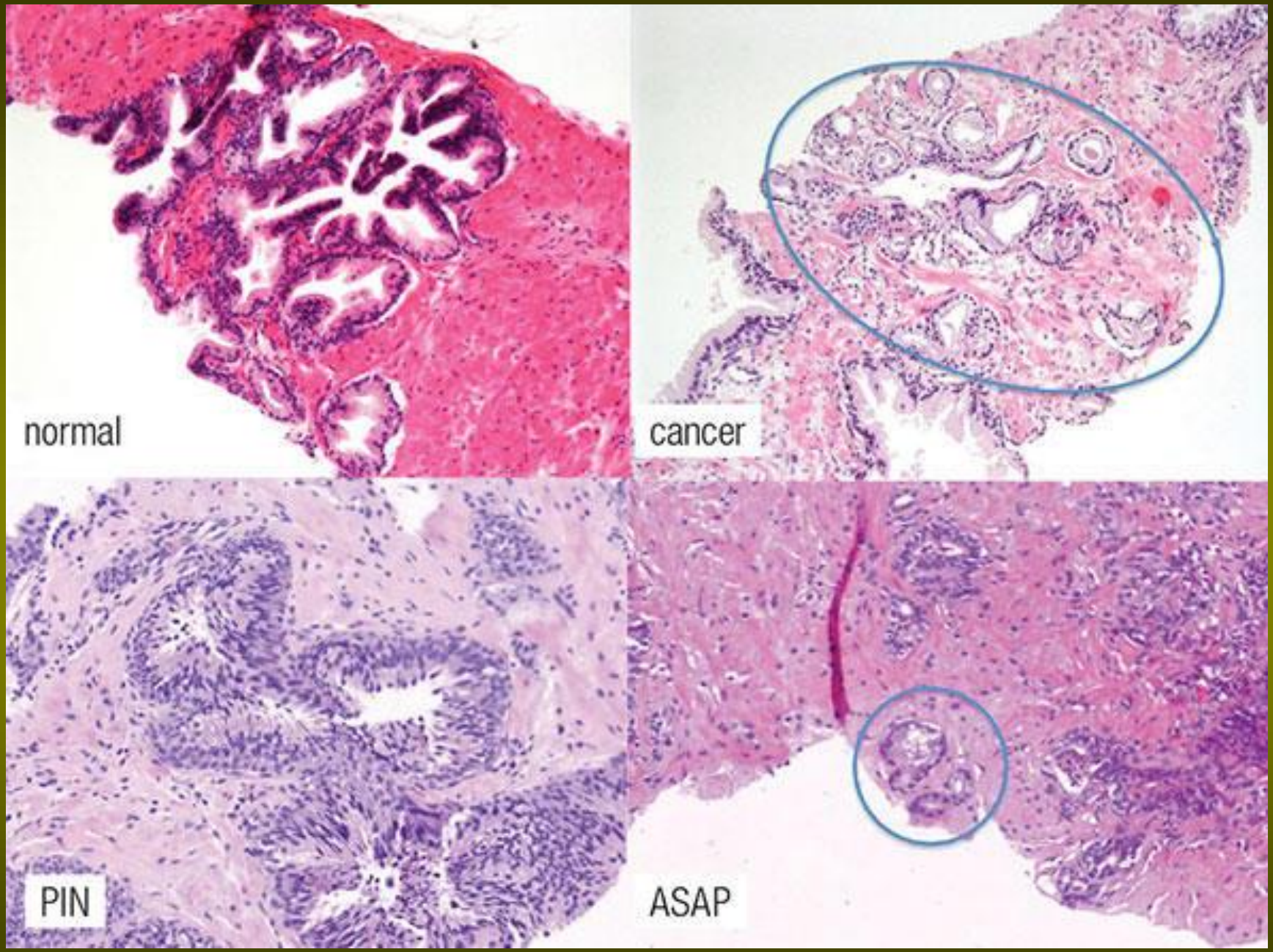
Normal prostate gland



Clinical and Pathological Classification of the Primary Tumour T

T1	Clinically unapparent tumour
T2	Tumour confined within the tumour
T3	Tumour extends through the prostatic capsule
T4	Tumour invades adjacent structures other than seminal vesicles

https://oncologypro.esmo.org/Education-Library/Essentials-for-Clinicians



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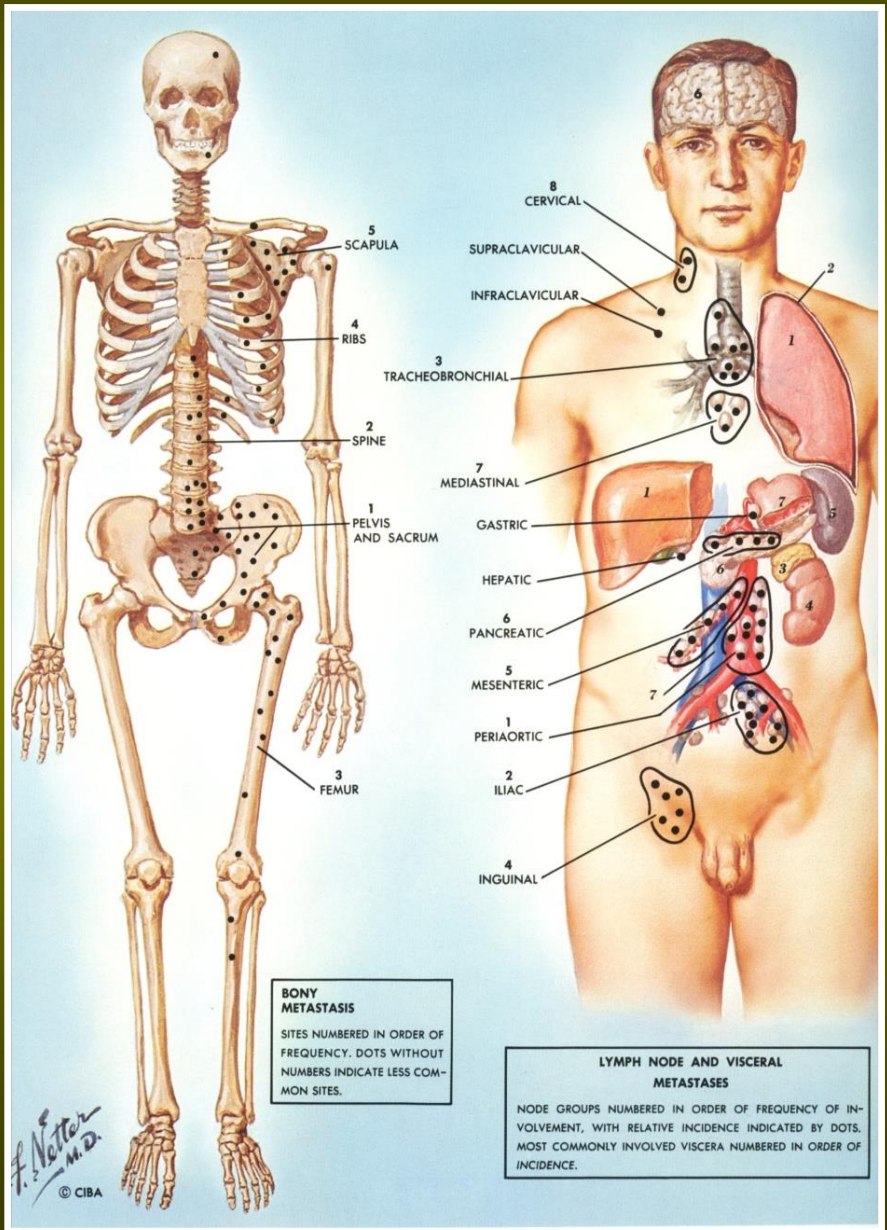
PATHOLOGY OF THE PROSTATE GLAND

CARCINOMA



LOCALISATION OF METASTASES FROM PROSTATE GLAND CANCER

- In patients who undergo treatment, the most important clinical prognostic indicators of disease outcome are stage, pre-therapy PSA level and Gleason score. In general, the higher the grade and the stage, the poorer the prognosis.
- The predictions are based on the experience of large groups of patients suffering from cancers at various stages.

Famous people diagnosed with prostate cancer

- Harry Belafonte Singer and actor
- Bob Dole World Former Republican senator from Kansas
- Robert De Niro Actor director
- Emperor Akihito of Japan
- Louis Farrakhan Civil right activist, leader of the Nation of Islam
- Rudy Giuliani Former New York City mayor
- Charlton Heston Actor
- John Kerry Vietnam veteran, Democratic senator of Massachusetts
- Nelson Mandela South African Former President, Anti-apartheid activist, Lawyer
- Francois Mitterand Former President of France
- Roger Moore Actor
- Colin Powell Retired Secretary of State,

THANK YOU