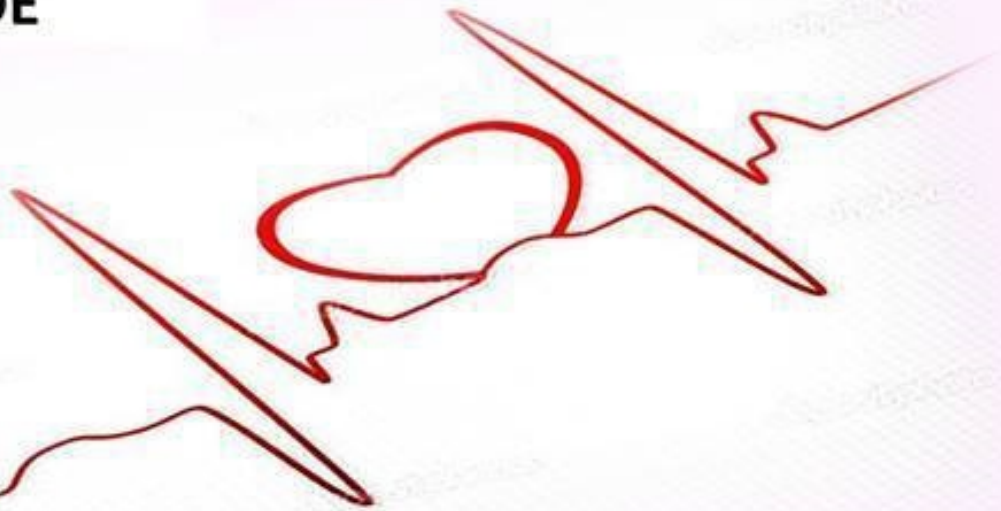


SHEET



SLIDE



Slide : 14- Neoplasia



Doctor: Dr. Mazen



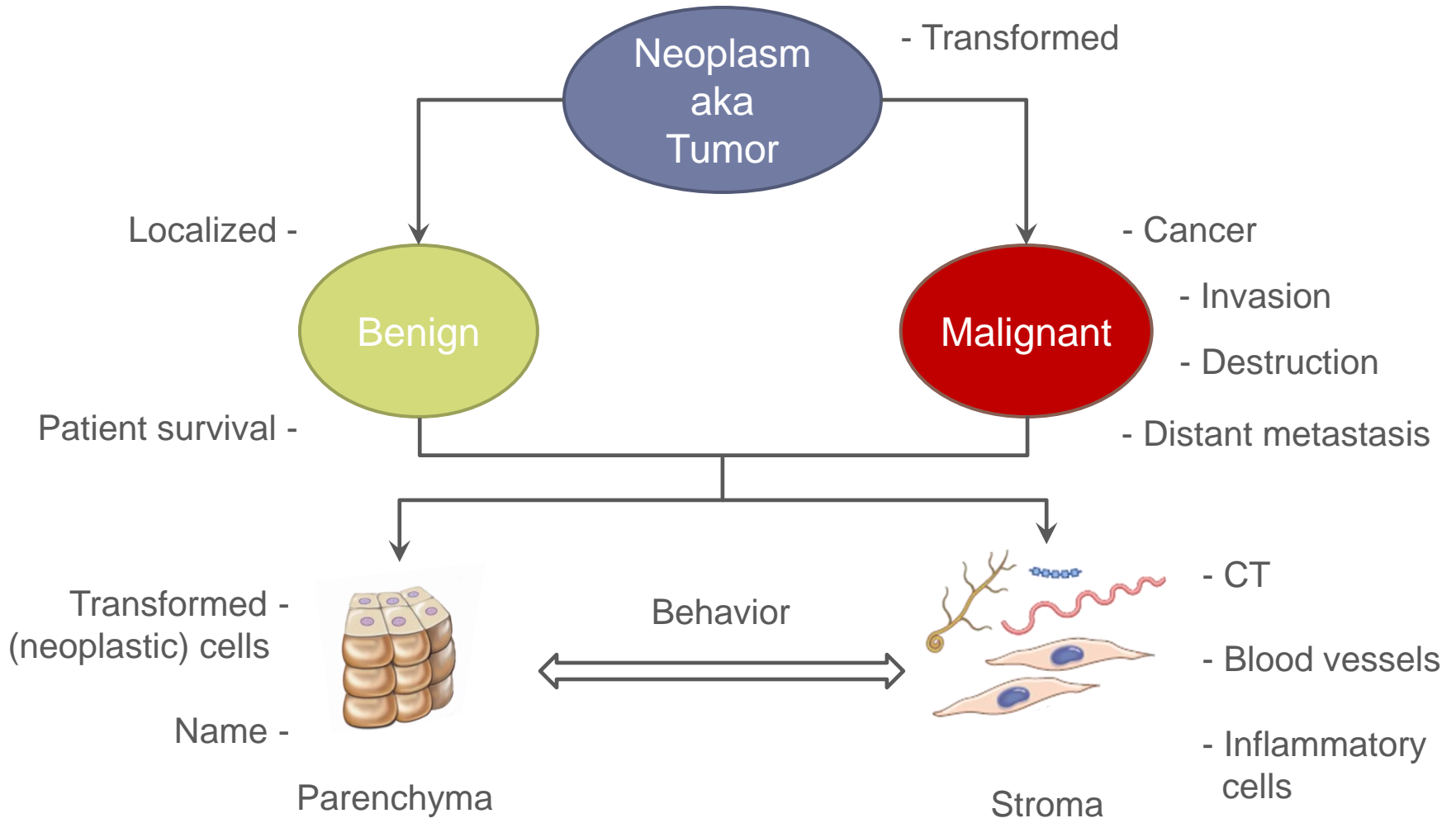


Apoptosis
Hyperplasia
Neoplasia
Repair
Atrophy
Cytokines
Inflammation
PATHOLOGY
Immunity
Necrosis
Cell
Hypertrophy
Virchow
Metaplasia
ROS
Proliferation

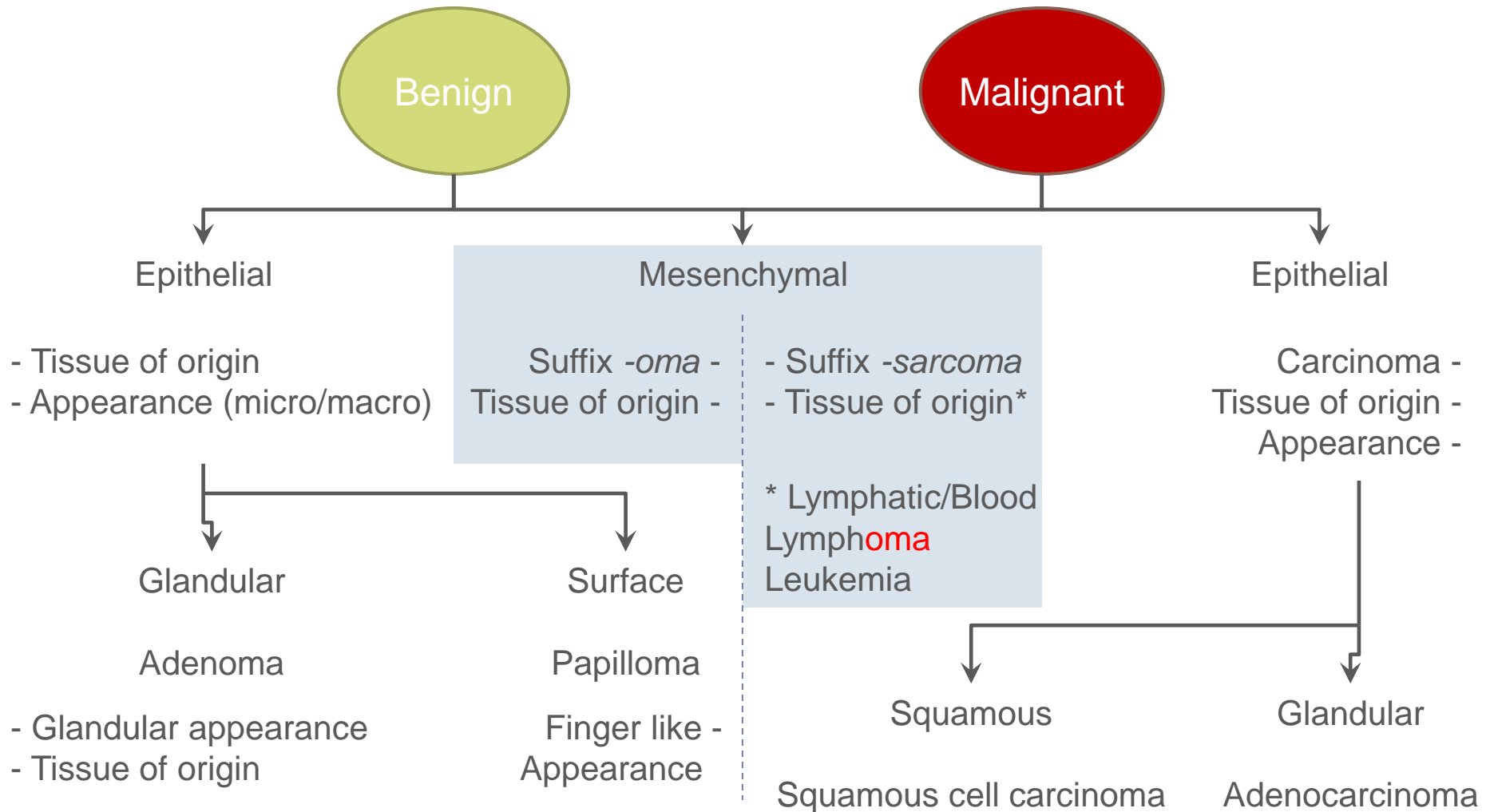
Neoplasia

Dr. Mazin Al-Salihi

Neoplasia (new growth) & Oncology



Nomenclature

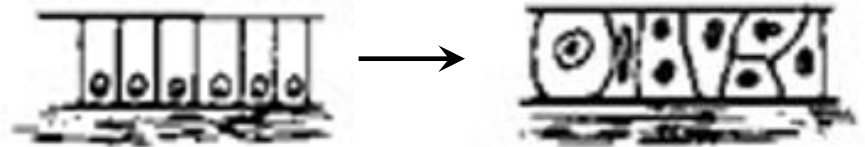




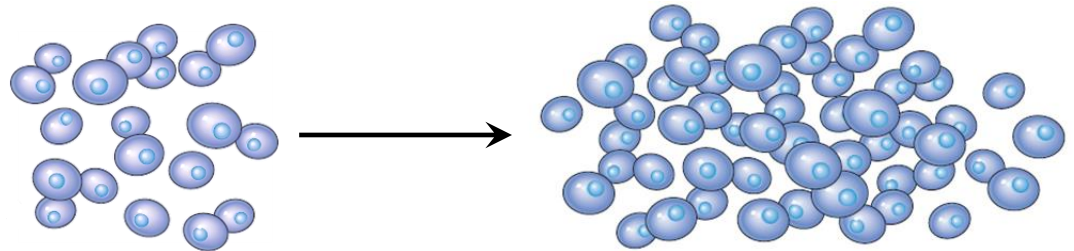
Benign or Malignant?

4 Major Criteria

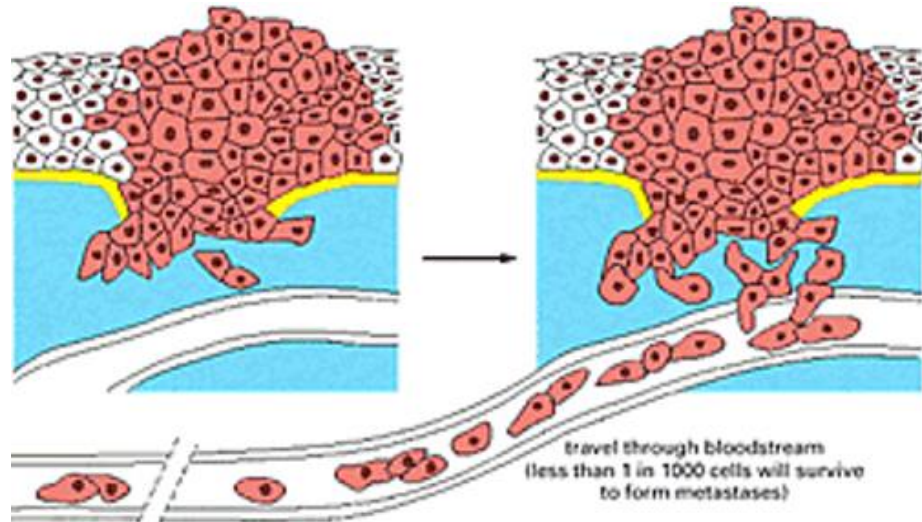
1. Differentiation & Anaplasia



2. Rate of growth

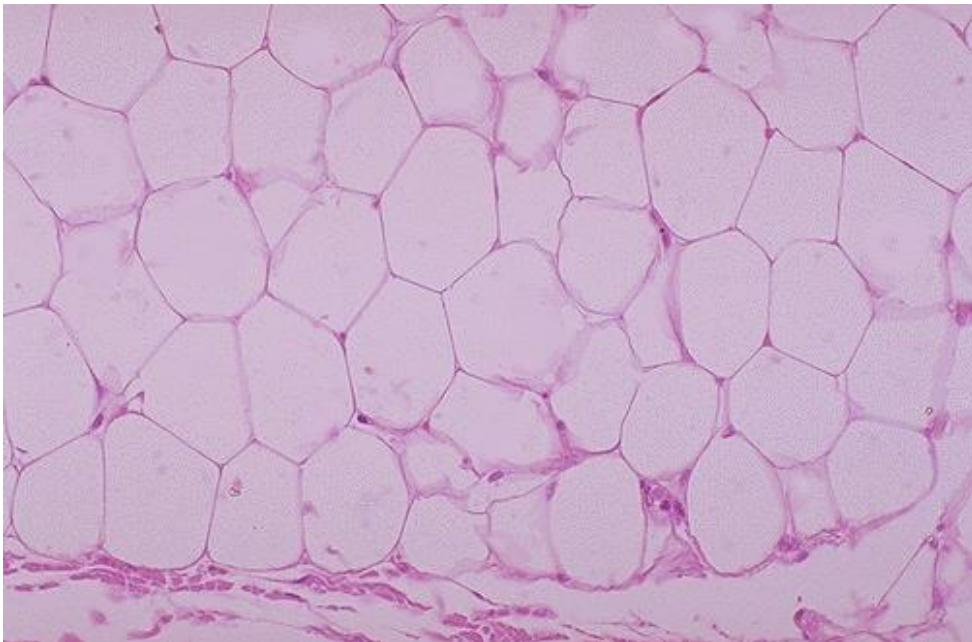


3. Local invasion



4. Metastasis





Differentiation & Anaplasia

Benign neoplasia

Well differentiated

Morphological & Functional differentiation

Mitotic figures rare/normal

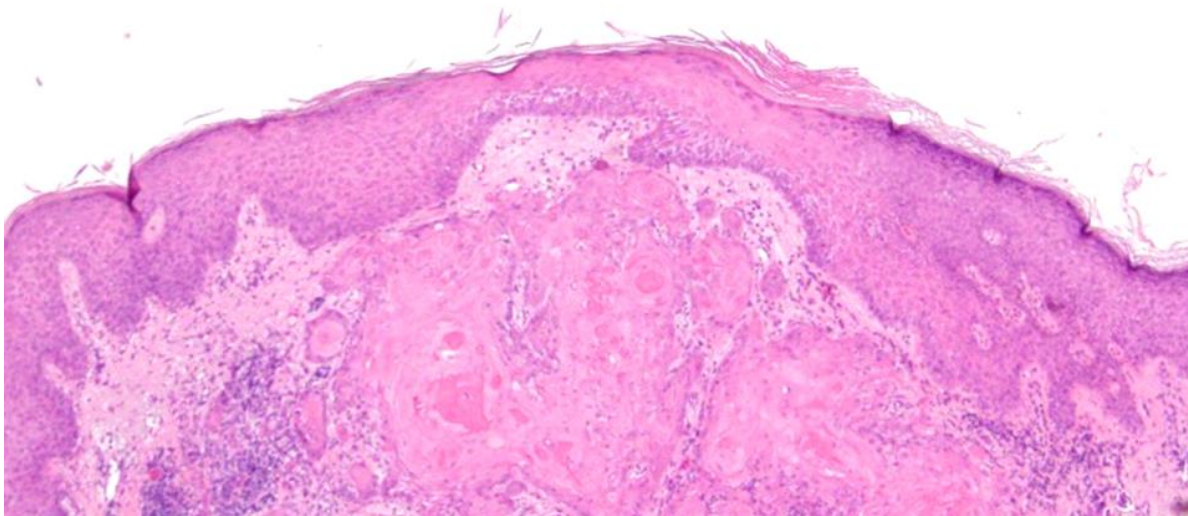


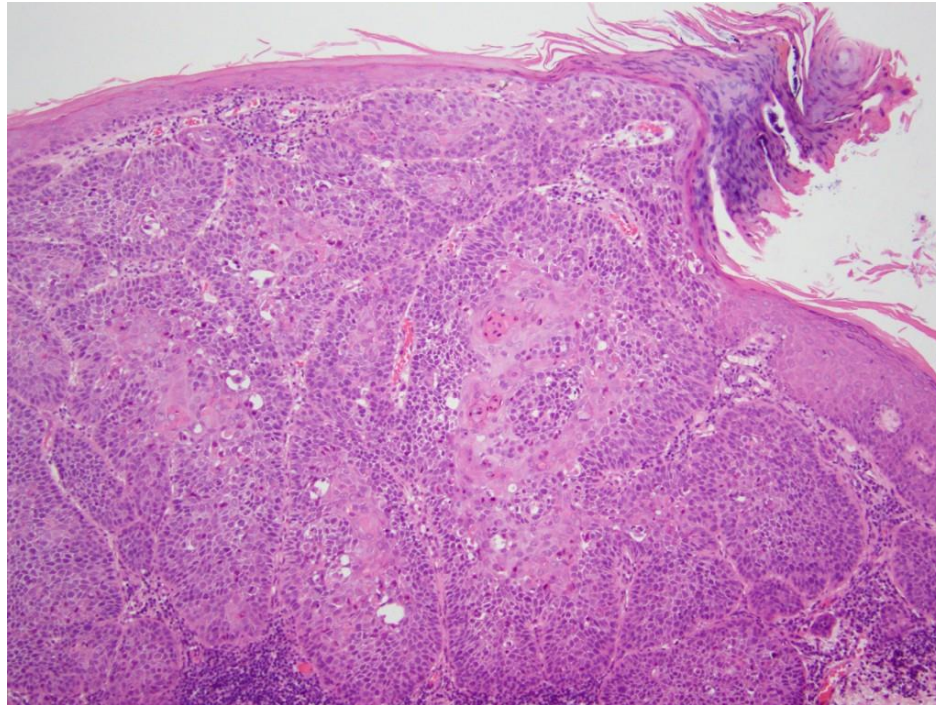
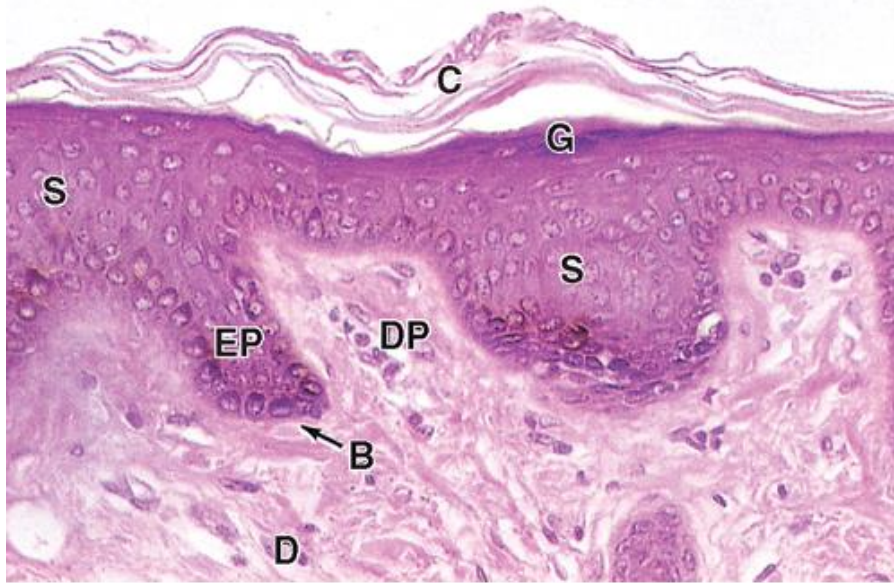
Differentiation & Anaplasia

Malignant neoplasia

Well-poorly differentiated parenchymal cells

Morphological & Functional differentiation



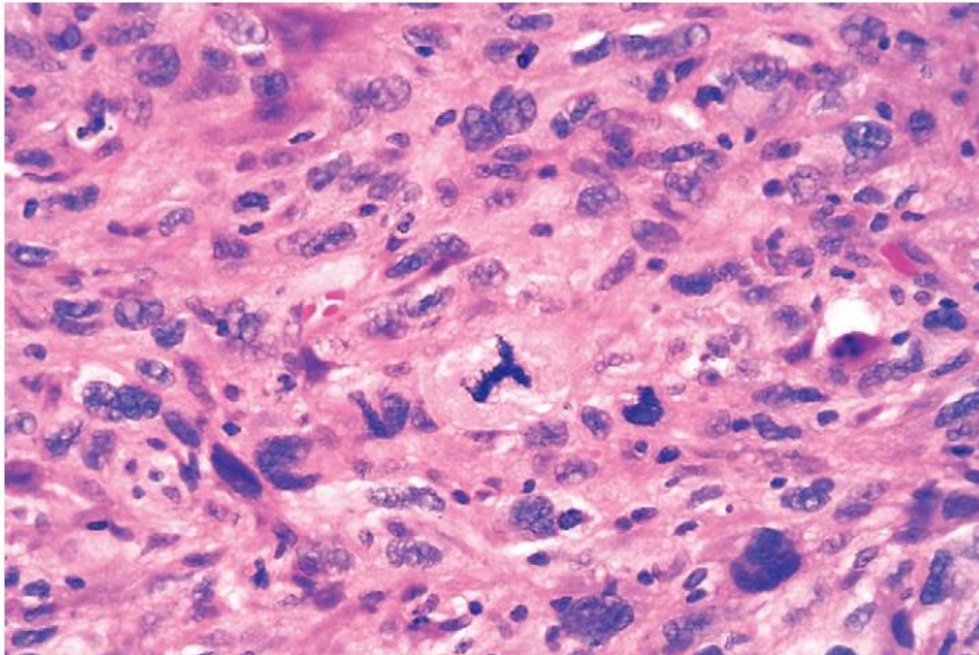
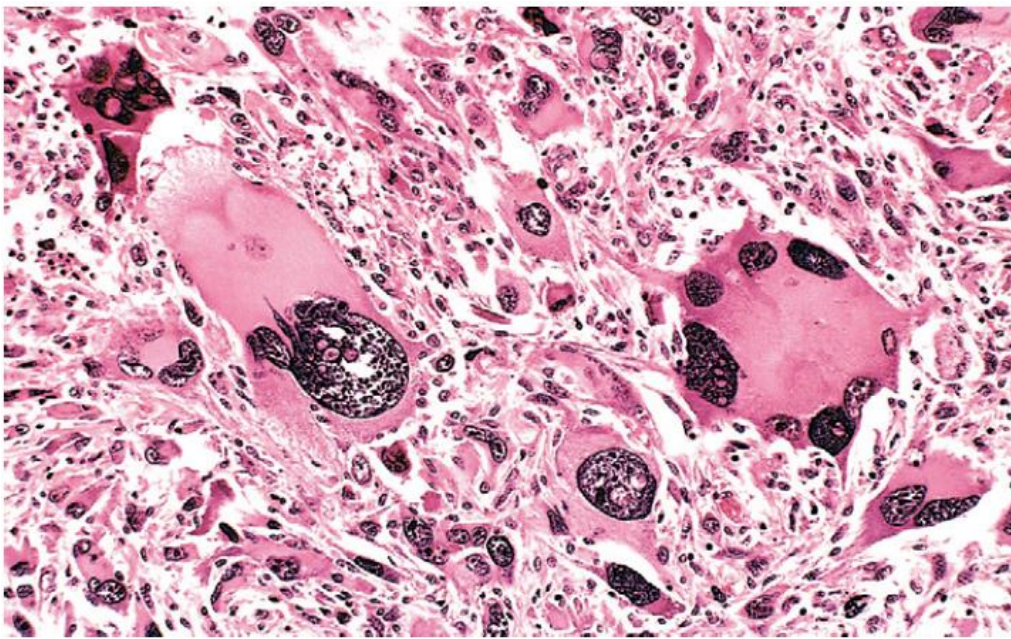


Differentiation & Anaplasia

Malignant neoplasia

Well-poorly differentiated parenchymal cells

Morphological & Functional differentiation



Differentiation & Anaplasia

Malignant neoplasia

Poorly differentiated = anaplastic

- Stem cells
- De-differentiation

- Pleomorphism
- Loss of polarity
- Giant cells
- Hyperchromatic Nuc.
- Large Nucleus
- Abnormal shape nuc.
- Multiple Nuclei
- Mitotic figures frequent/abnormal

Differentiation & Anaplasia

Functional Significance

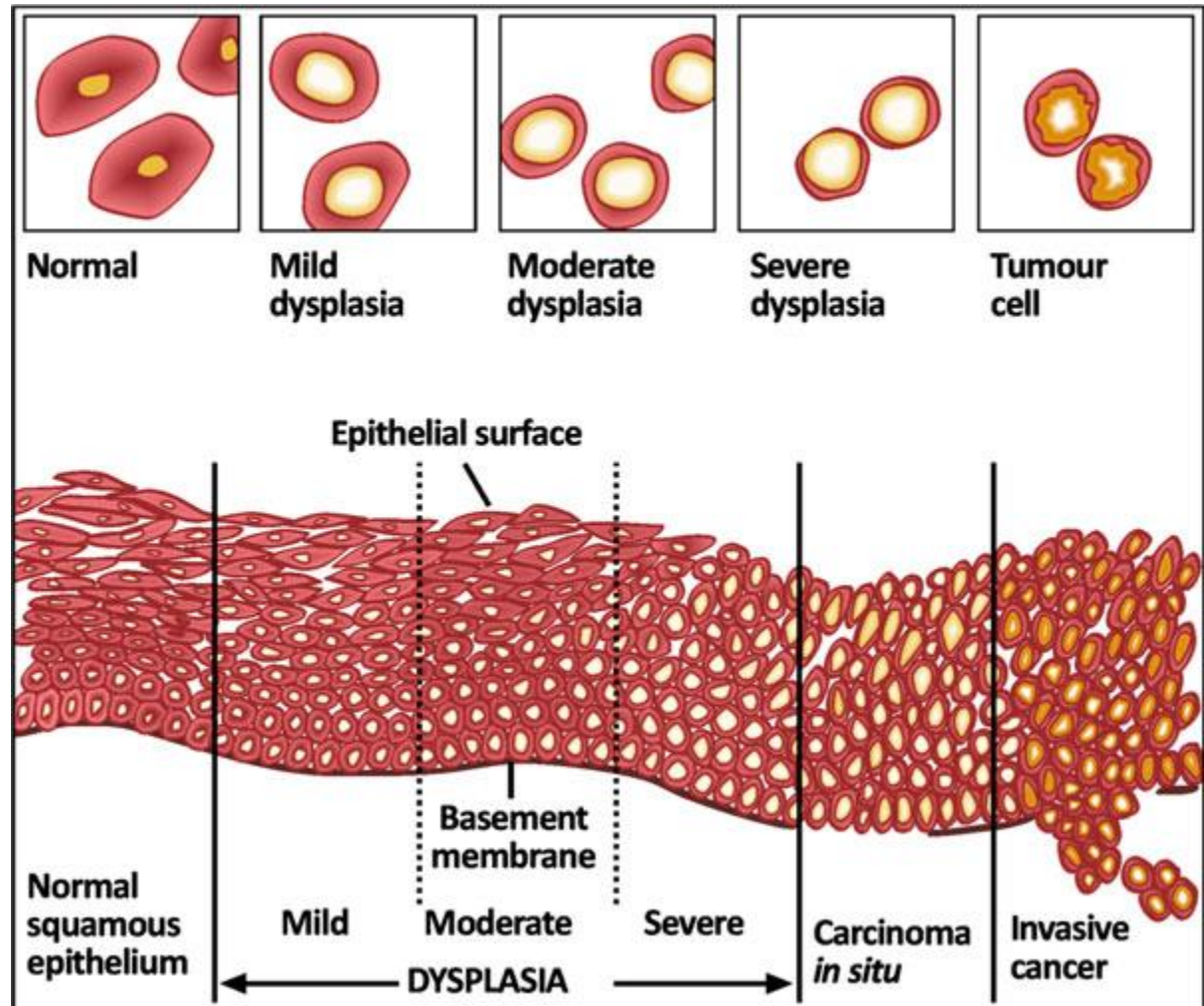
Well differentiated neoplasms typically retain the original tissue function

Hormonal tissue neoplasms may still produce the relevant hormones

Ectopic hormone production



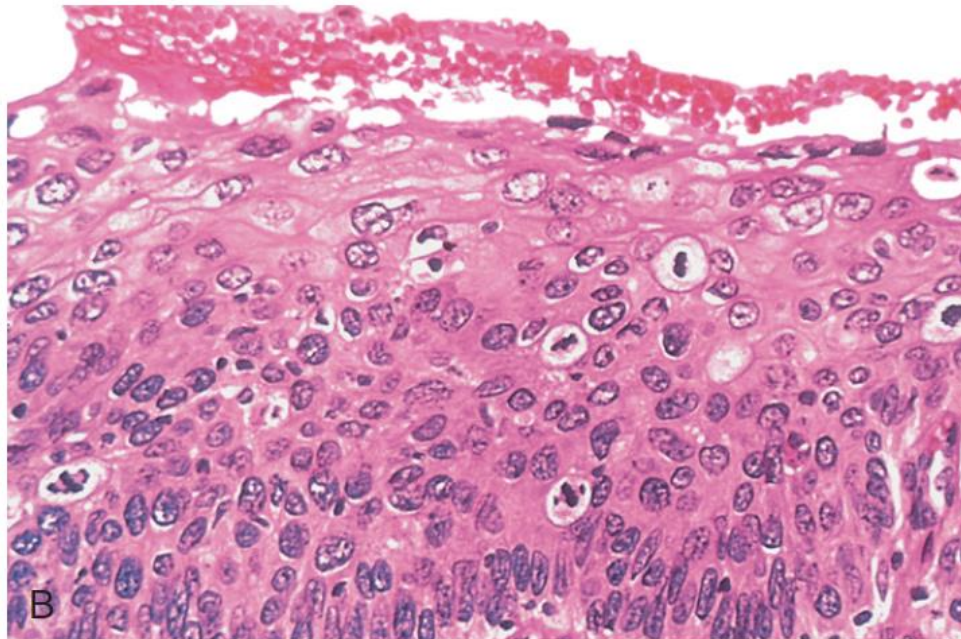
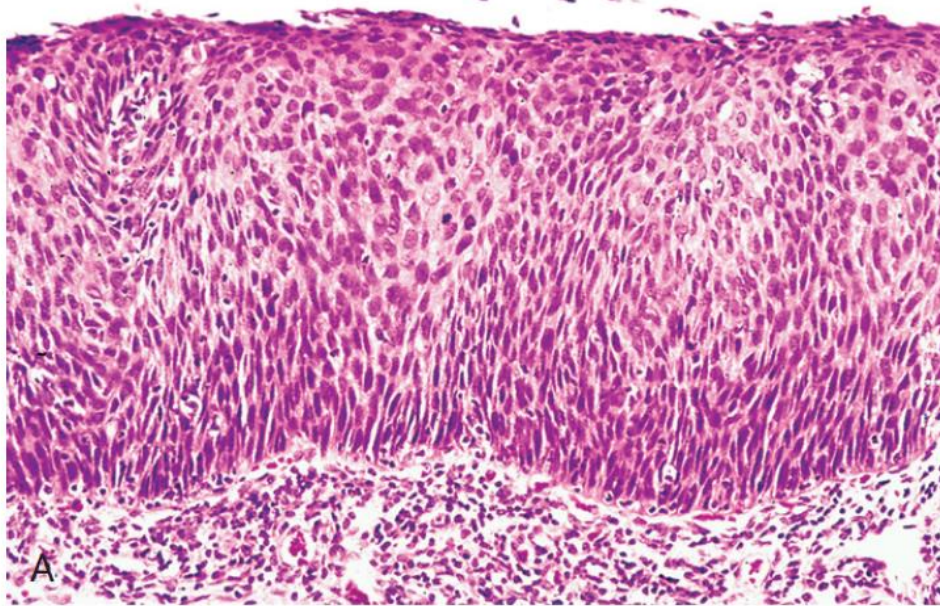
What about Dysplasia?



Pleomorphism

Hyperchromatic nuclei

Mitotic figures more frequent & outside the basal layer



Carcinoma-in-situ

Preinvasive

Whole epithelium
thickness involved

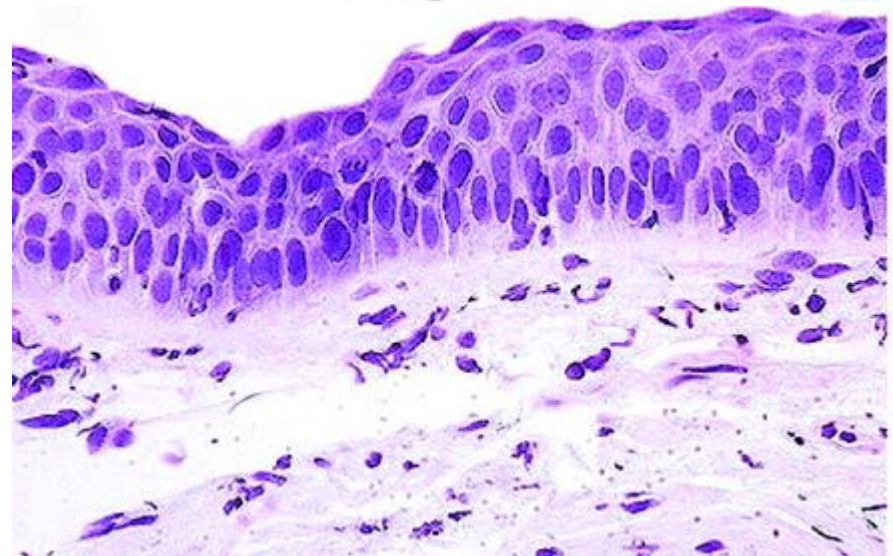
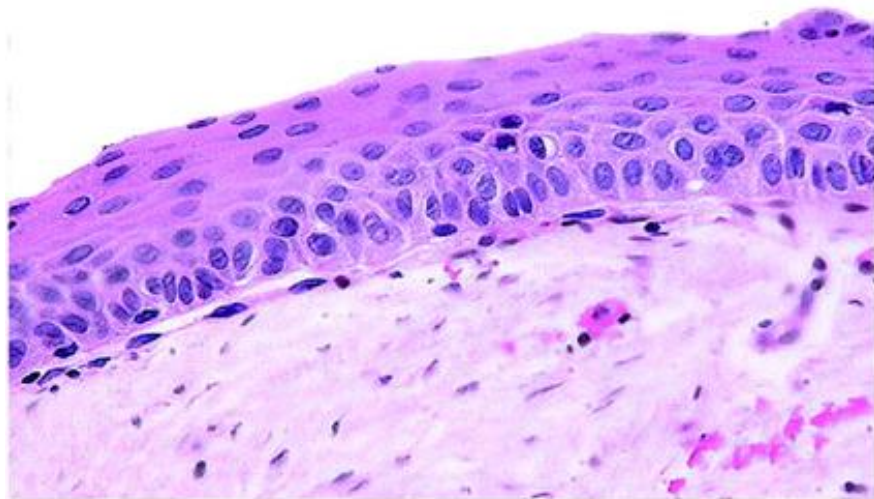
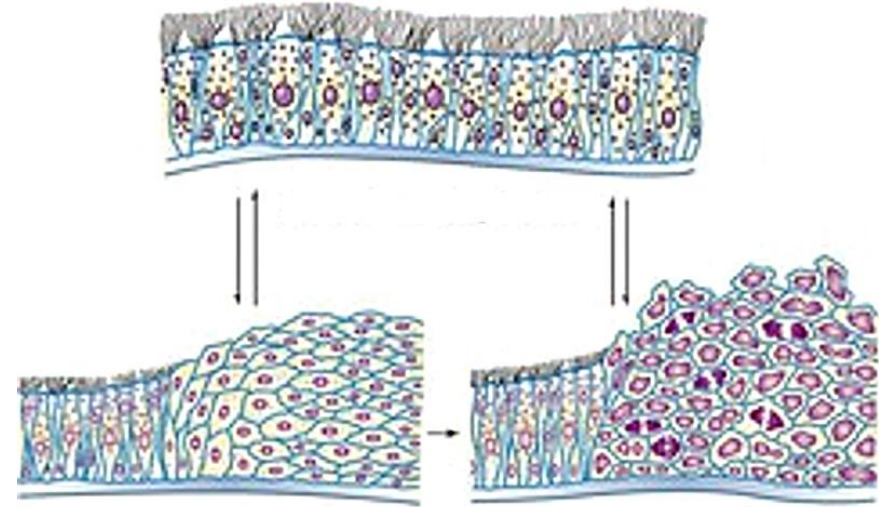
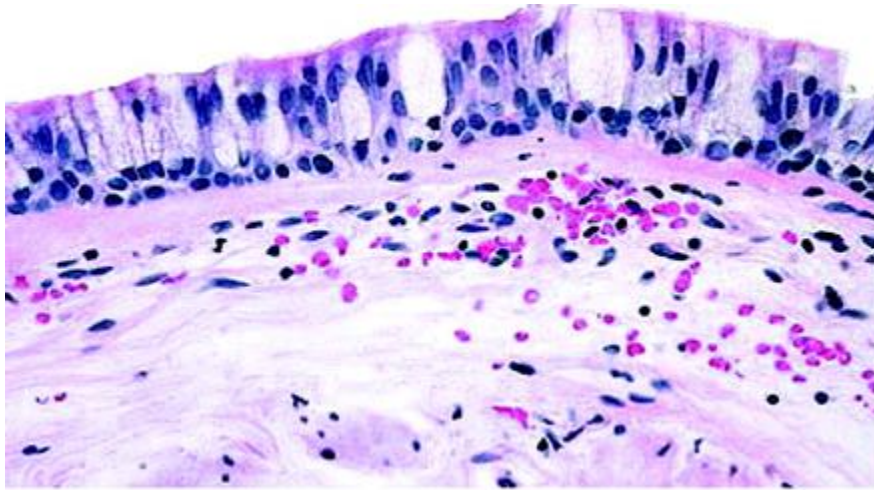
Basement membrane
intact

Pleomorphism

Hyperchromatic nuclei

Mitotic figures more
frequent and outside the
basal layer

Metaplasia-Dysplasia-CIS





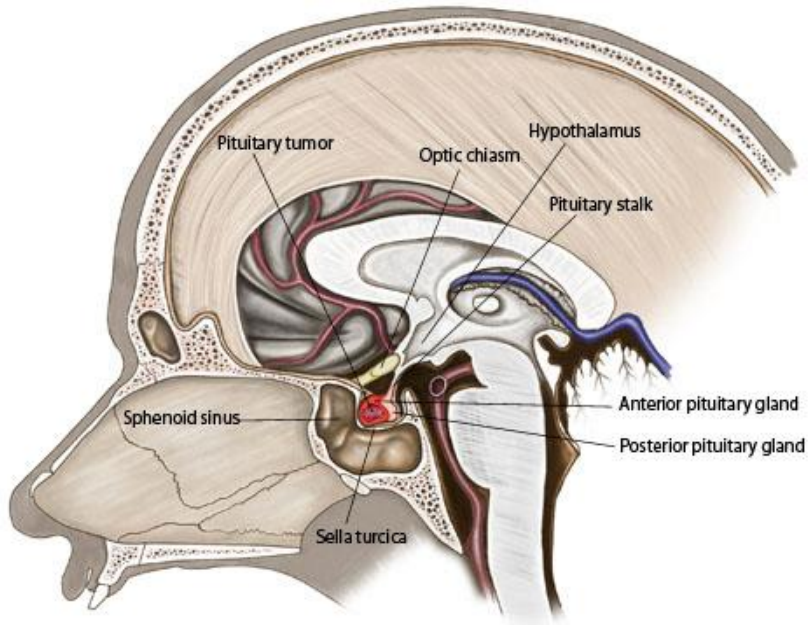
Rate of growth

Benign/well differentiated
= slow*

Malignant/poorly
differentiated = fast*

Factors

- Blood supply
- Hormone/GF effect
- Anatomical limitations
- SMT/subclone
- Cancer stem cell hypothesis

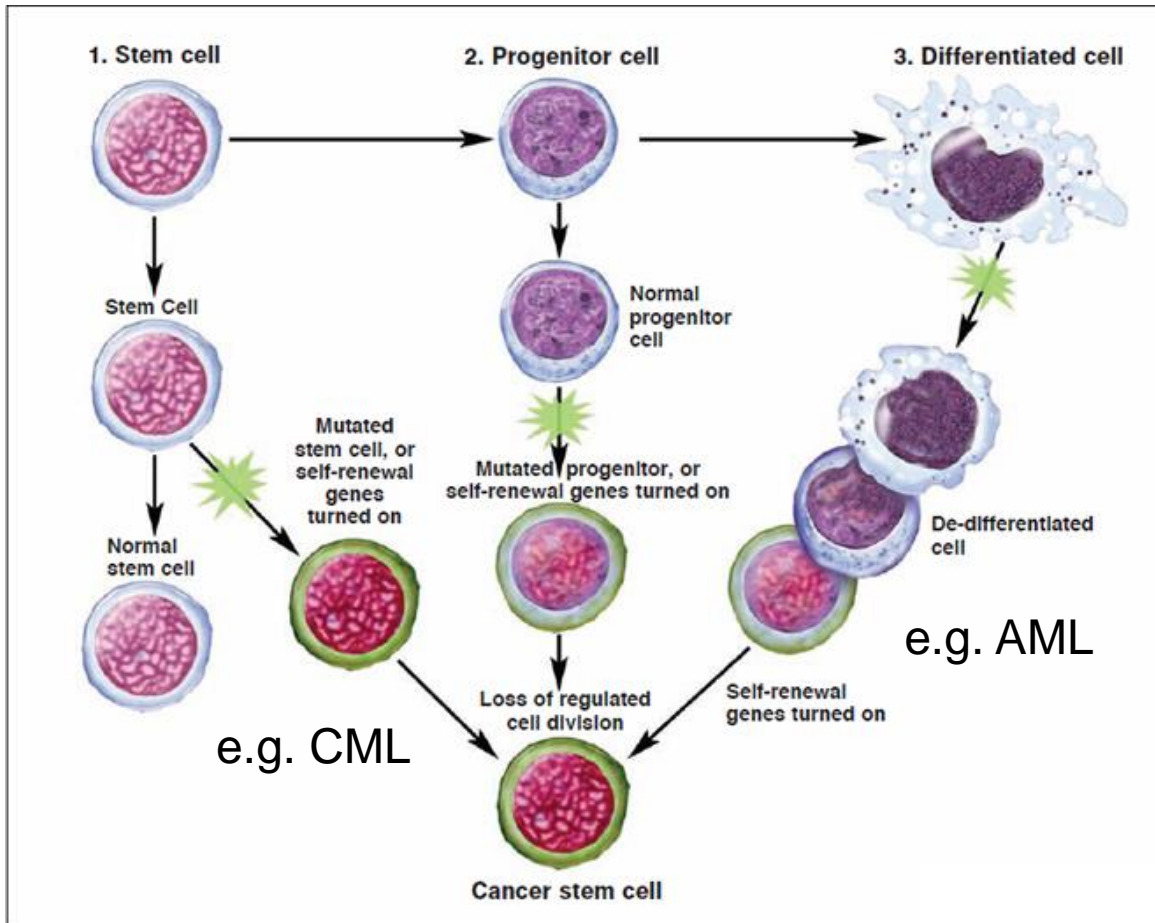


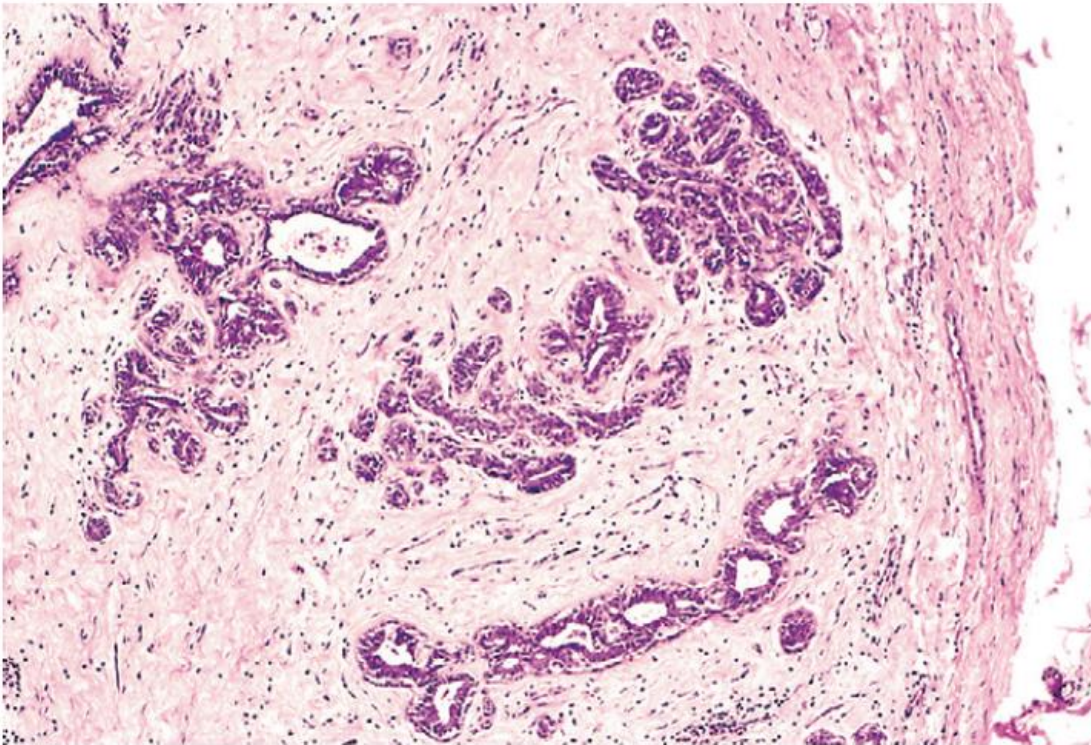
Rate of growth

Cancer stem cell hypothesis

- Renewal capacity
- MDR-1
- Leukemia stem cells
- Solid tumor stem cells?

Without killing the cancer stem cells a cancer can recur





Local Invasion

Benign neoplasia

Most (not all) benign neoplasia have a fibrous capsule clearly demarcating their edge

They do not infiltrate, invade, or metastasize



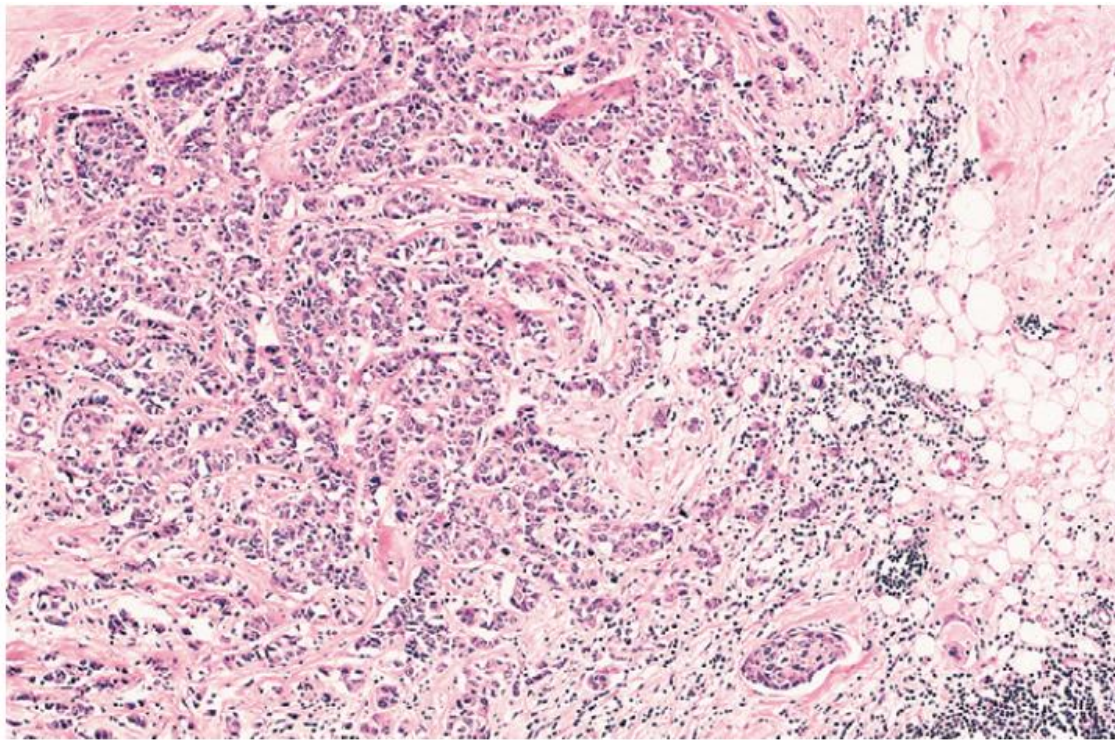
Local Invasion

Malignant neoplasia

Do not develop well-defined capsules*

Do infiltrate & invade

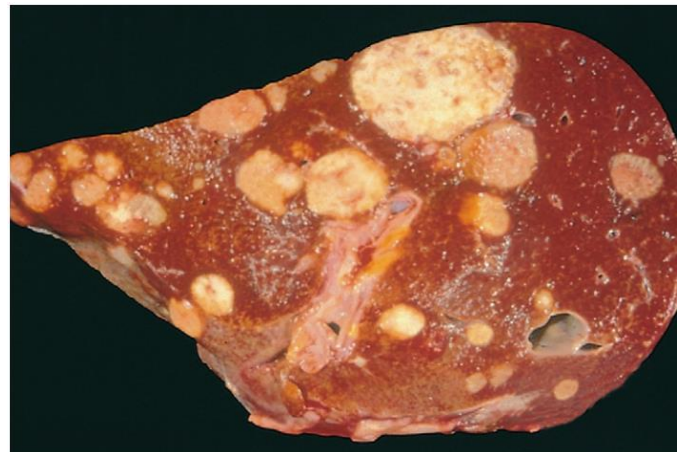
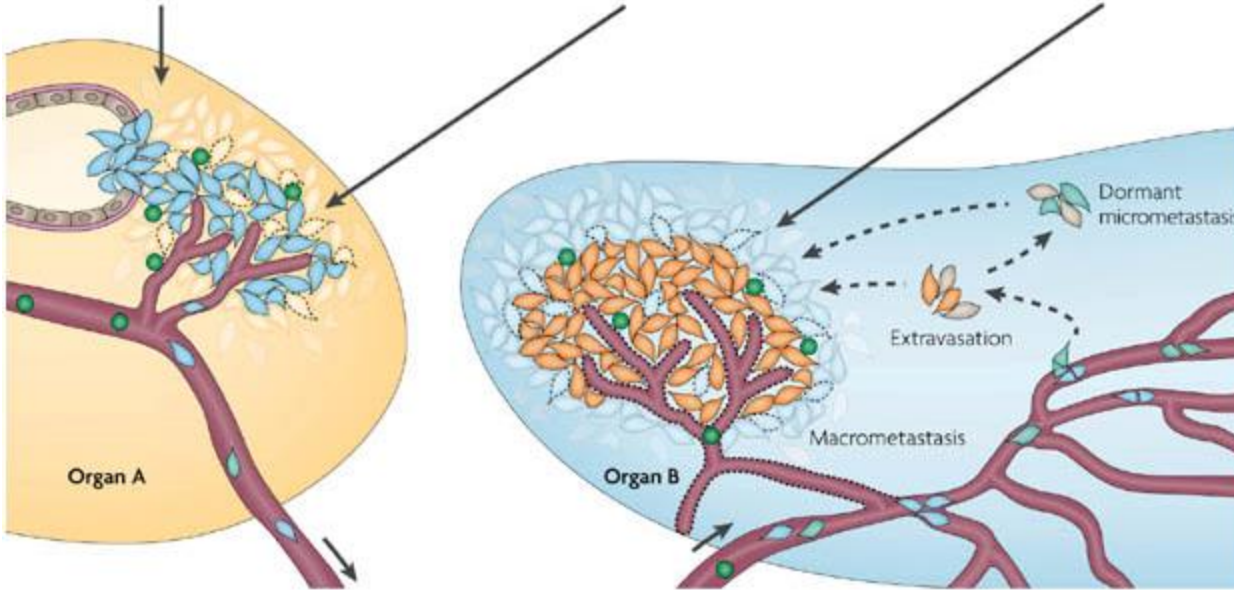
Clean margins required for local resection



- Proliferative autonomy
- Genomic instability
- Self-renewal
- Evasion of death
- Evasion of cytotaxis
- Evasion of immunity
- Resistance to hypoxia

- Detachment
- Motility
- Invasion
- Angiogenesis
- Intravasation

- Survival in circulation
- Embolism
- Capillary adhesion
- Extravasation
- Adaption to new environment
- Emergence from dormancy
- Organ-specific colonization



Metastasis

Benign neoplasia do not metastasize

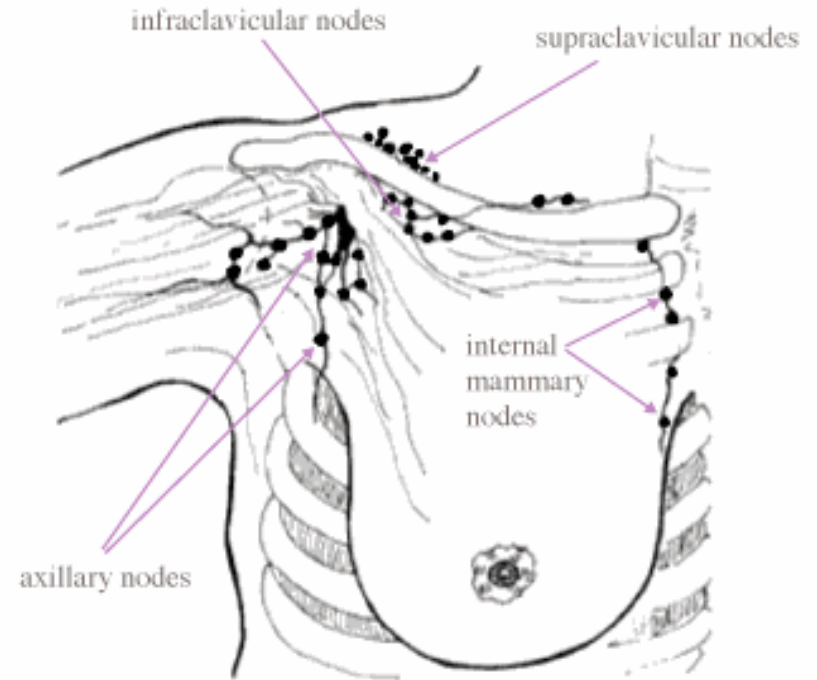
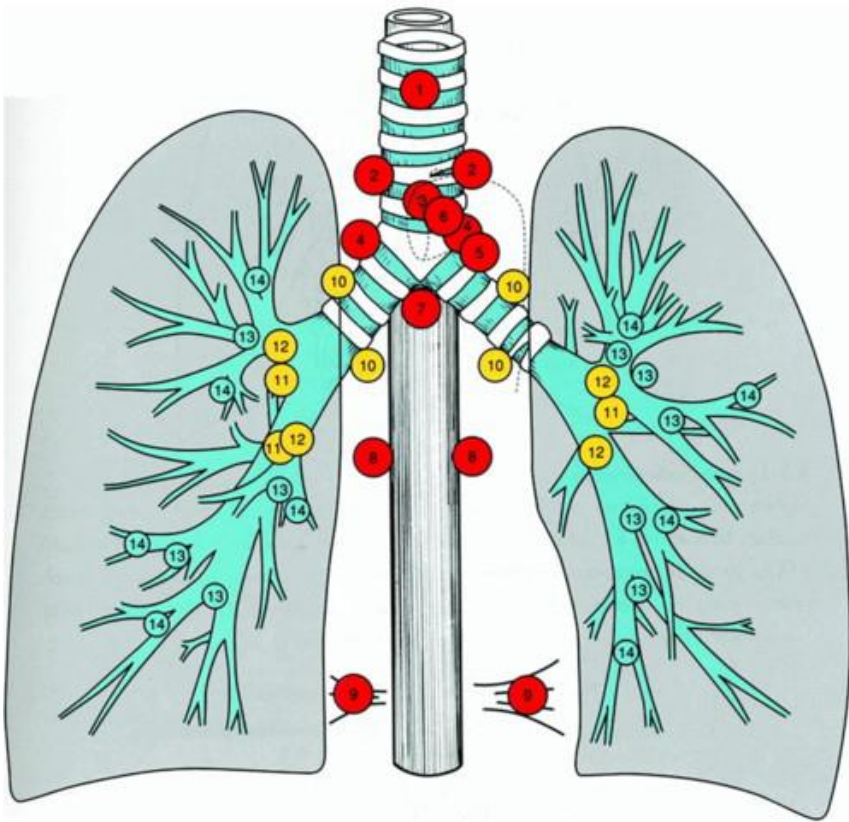
Not all malignant neoplasia metastasize (Biology/Time)

Anaplasia & big → more likely to metastasize*

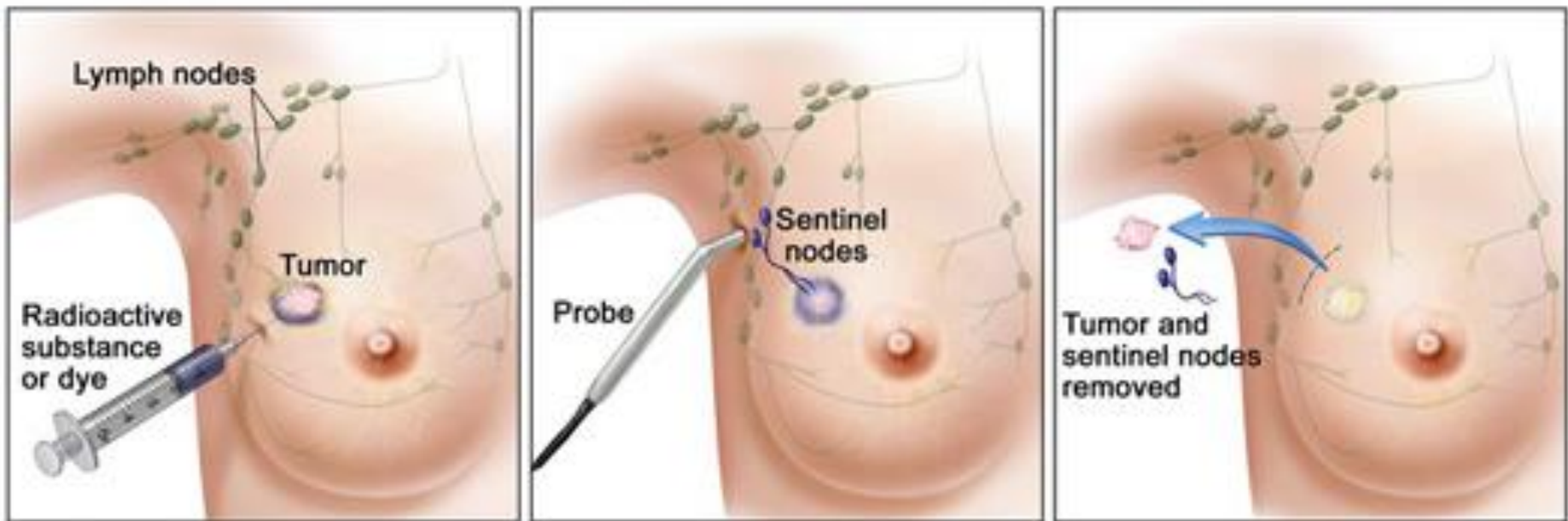
Spread by:

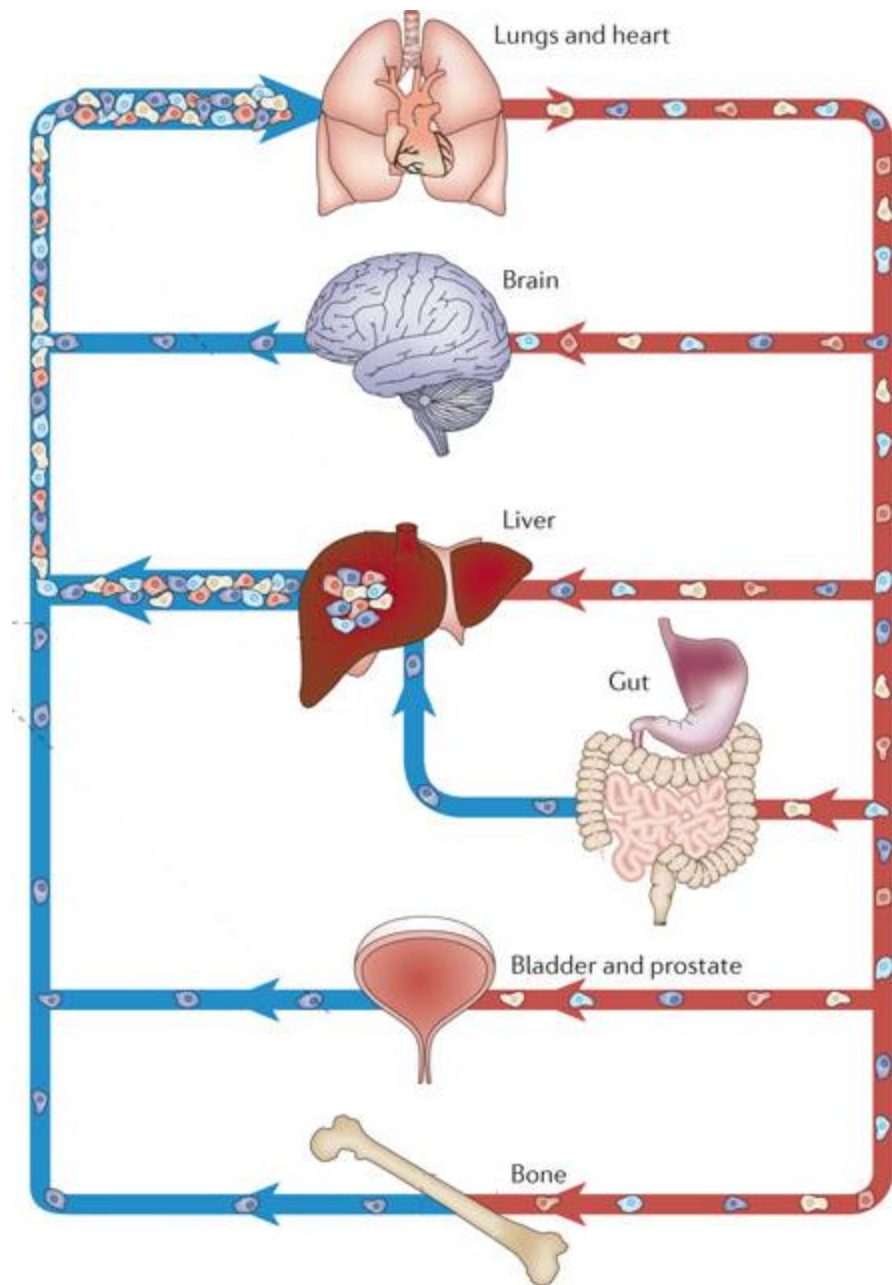
1. Seeding (body cavity)
2. Lymphatic (carcinoma)
3. Hematogenous (sarcoma)

Lymphatic drainage



Sentinel Node Biopsy





Hematogenous metastasis

Vein invasion

First capillary bed encountered

Portal → liver

Caval → lung

Anatomy cannot explain all metastasis*

Summary of extremes

