

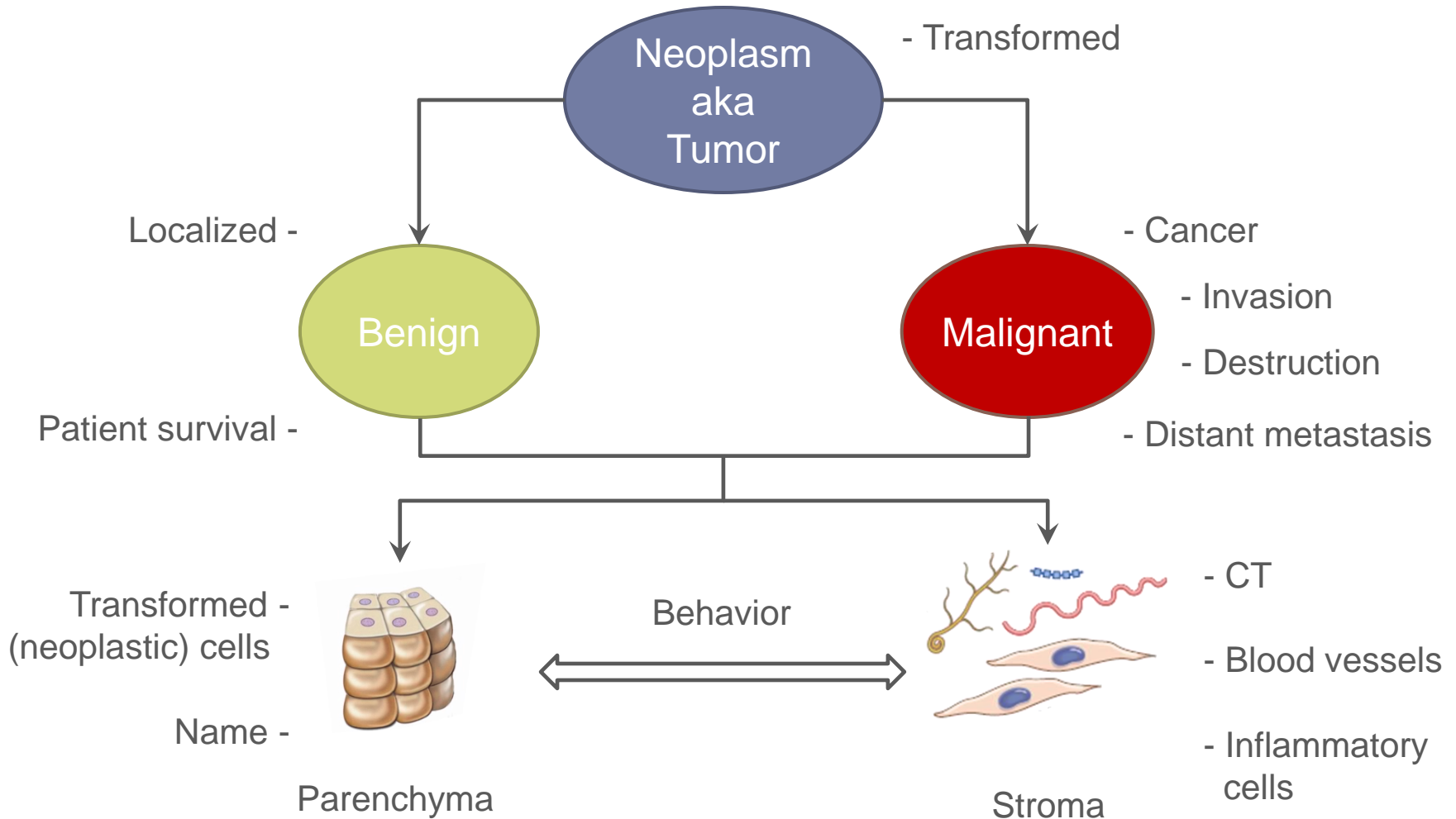


Apoptosis  
Hyperplasia  
Neoplasia  
Repair  
Atrophy  
Cytokines  
Inflammation  
PATHOLOGY  
Immunity  
Cell  
Hypertrophy  
ROS  
Proliferation  
Necrosis  
Virchow  
Metaplasia

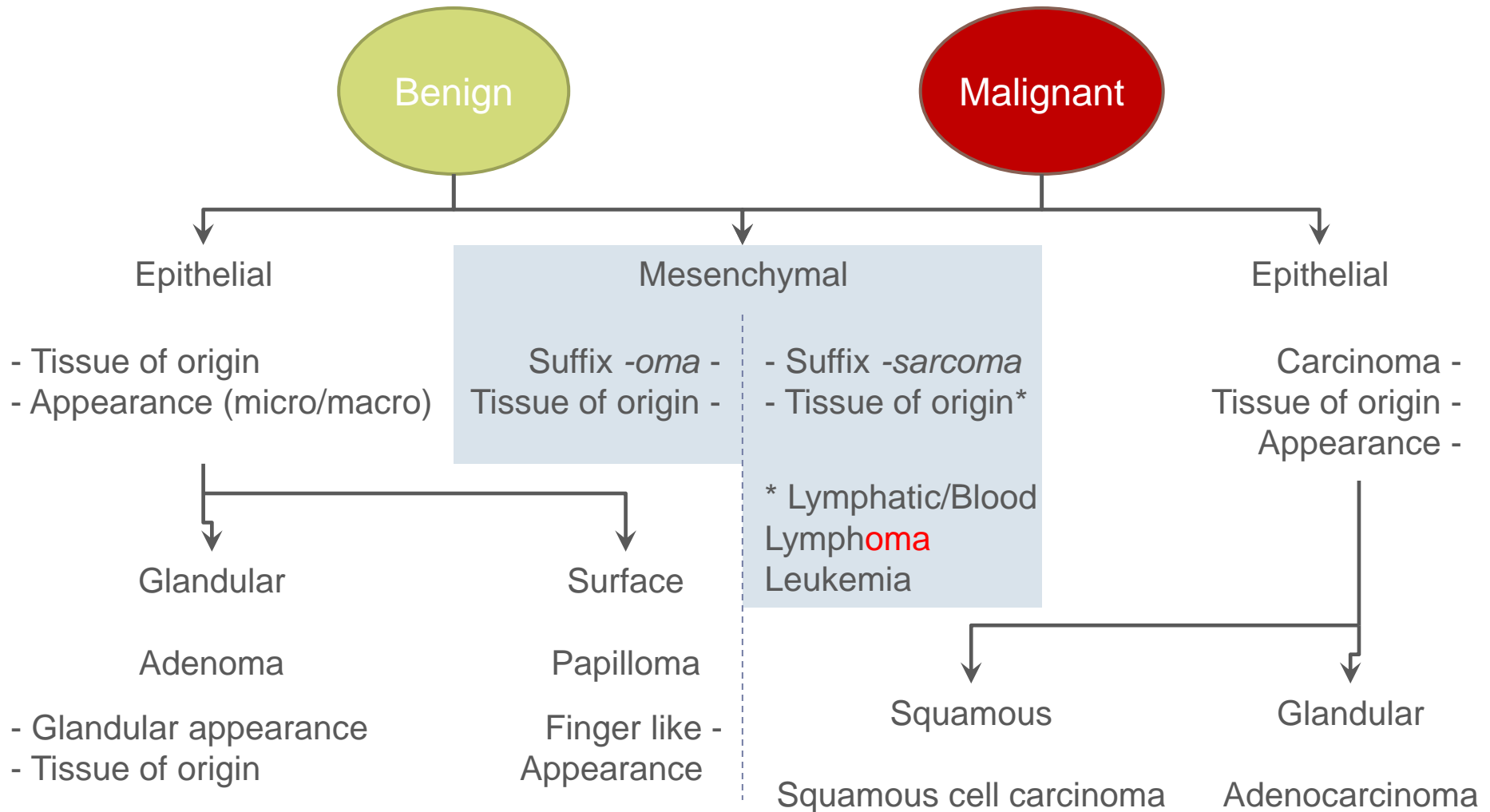
Neoplasia

Dr. Mazin Al-Salihi

# Neoplasia (new growth) & Oncology



# Nomenclature



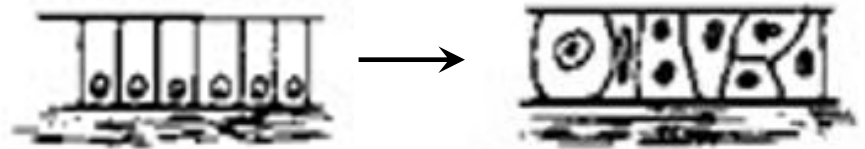


Benign or Malignant?

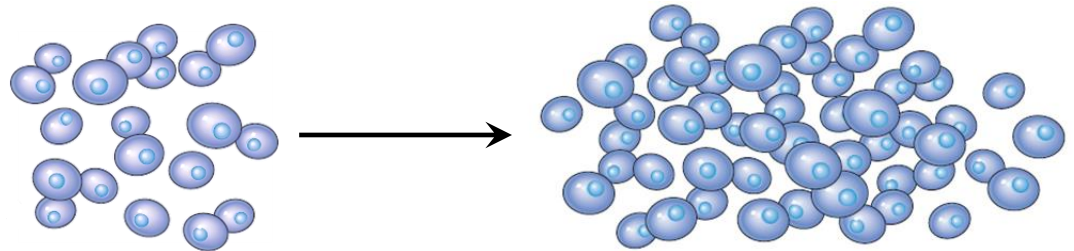
## 4 Major Criteria

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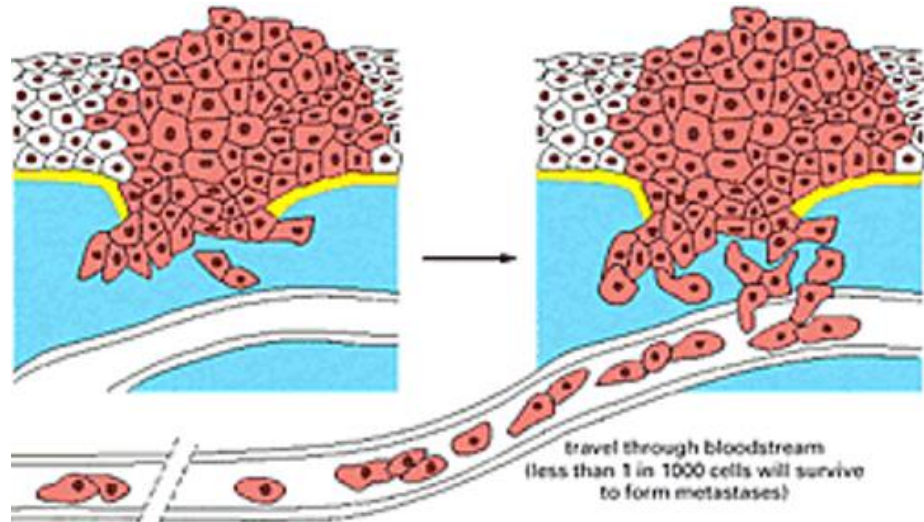
### 1. Differentiation & Anaplasia



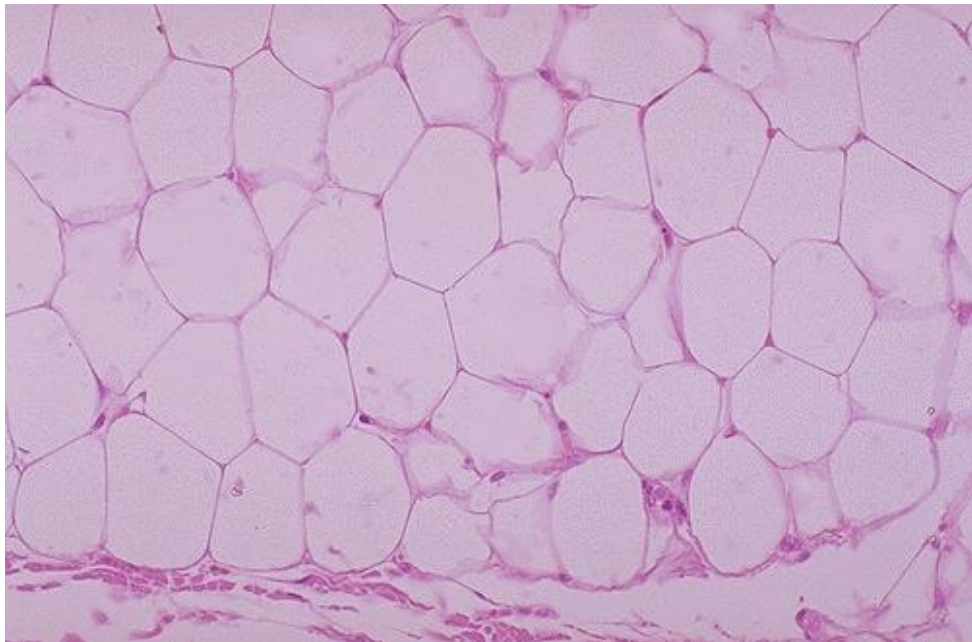
### 2. Rate of growth



### 3. Local invasion



### 4. Metastasis



## Differentiation & Anaplasia

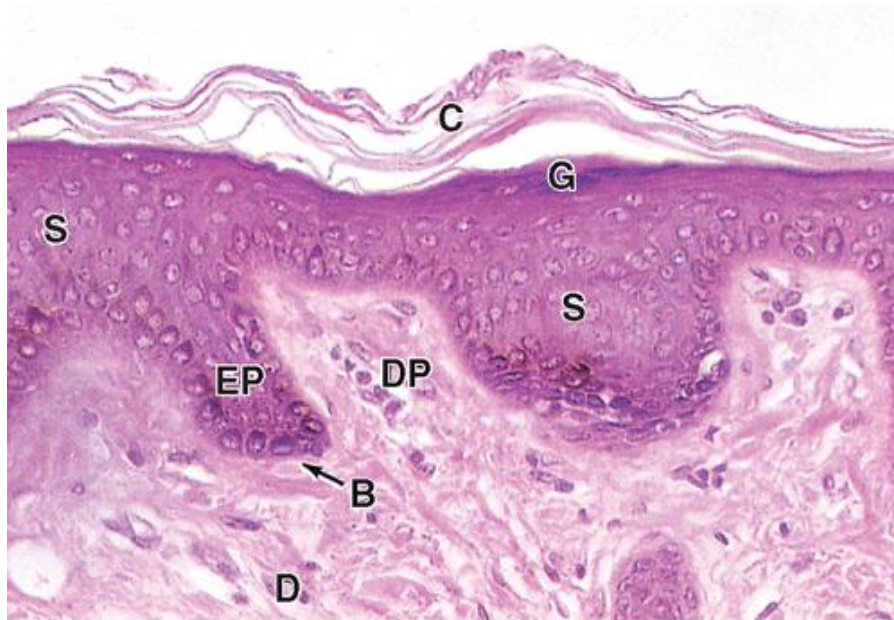
### Benign neoplasia

Well differentiated

Morphological &  
Functional differentiation

Mitotic figures  
rare/normal



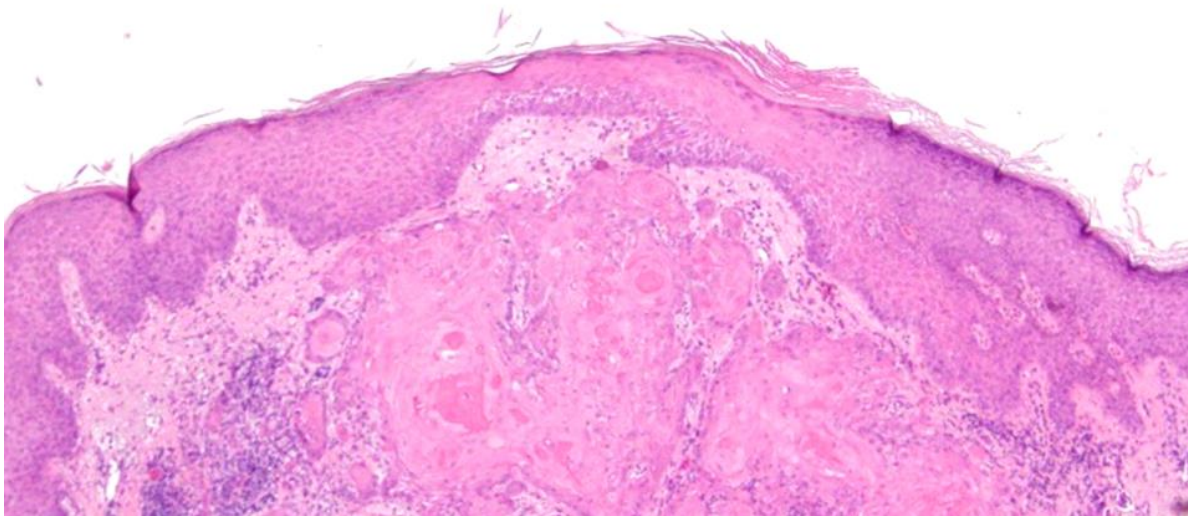


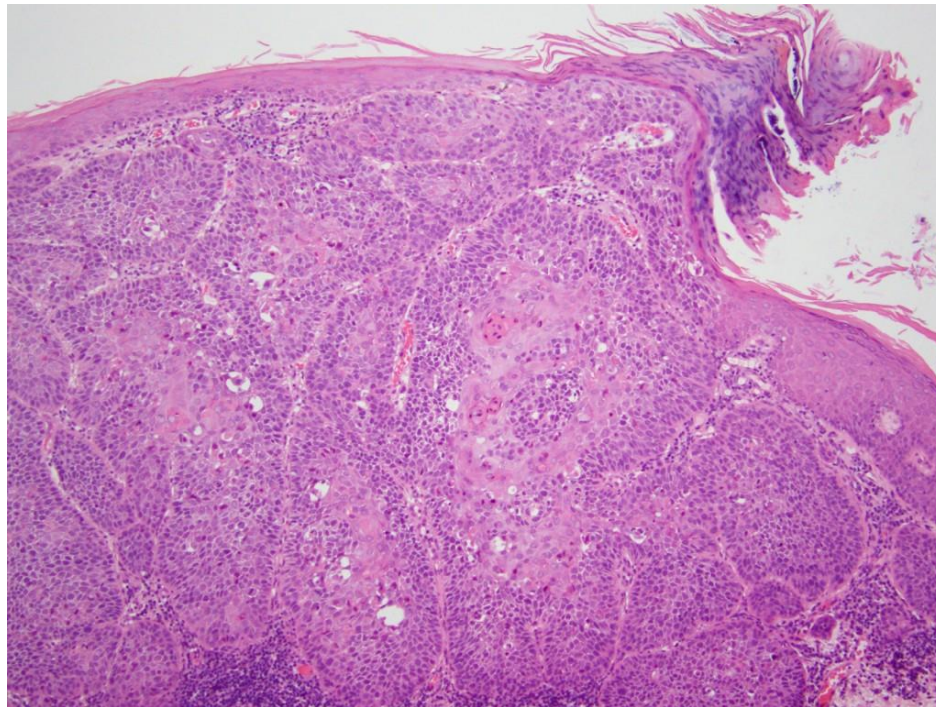
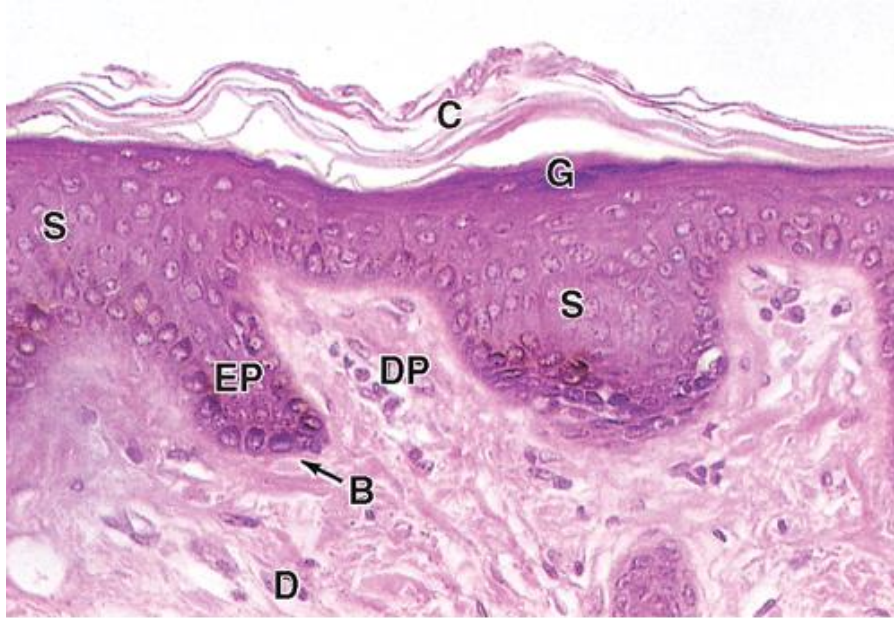
## Differentiation & Anaplasia

### Malignant neoplasia

Well-poorly differentiated  
parenchymal cells

Morphological &  
Functional differentiation





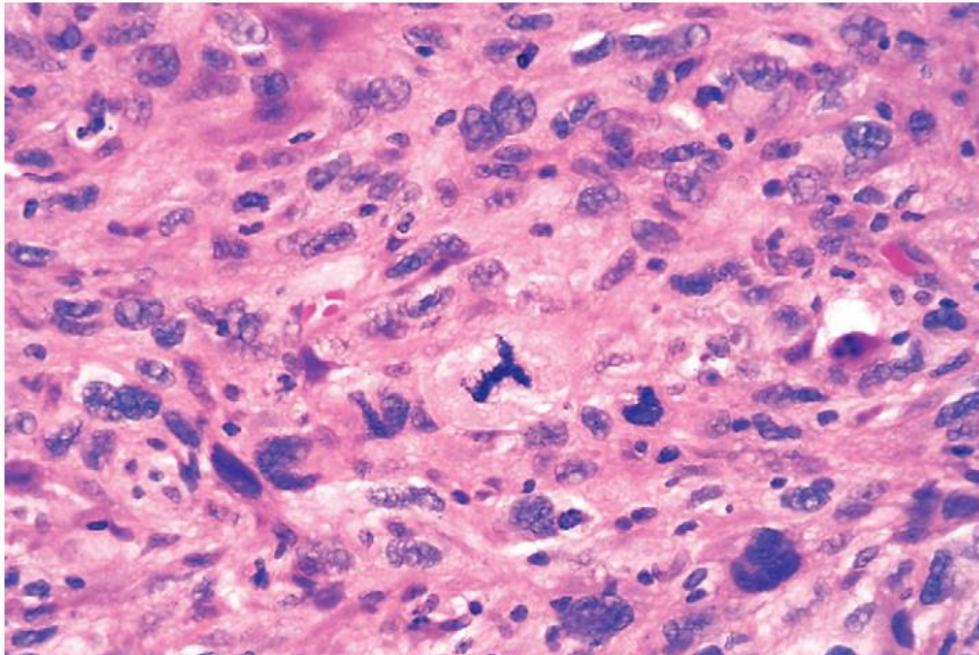
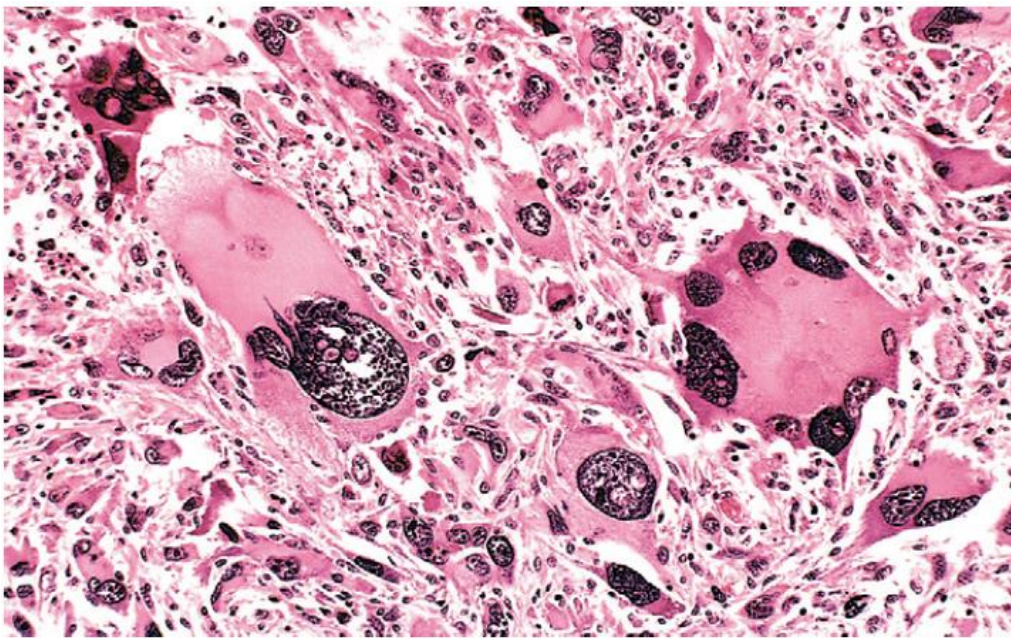
## Differentiation & Anaplasia

### 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

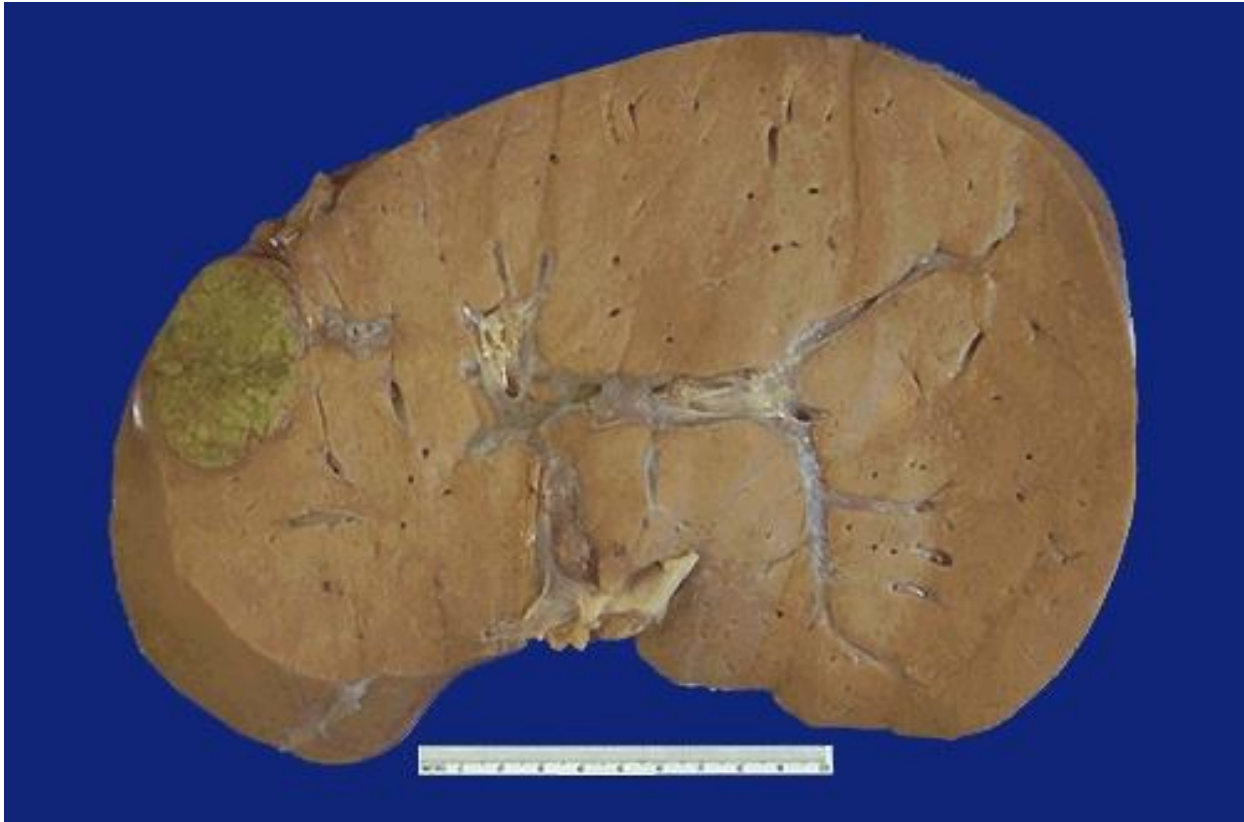
## Differentiation & Anaplasia

### Functional Significance

Well differentiated neoplasms typically retain the original tissue function

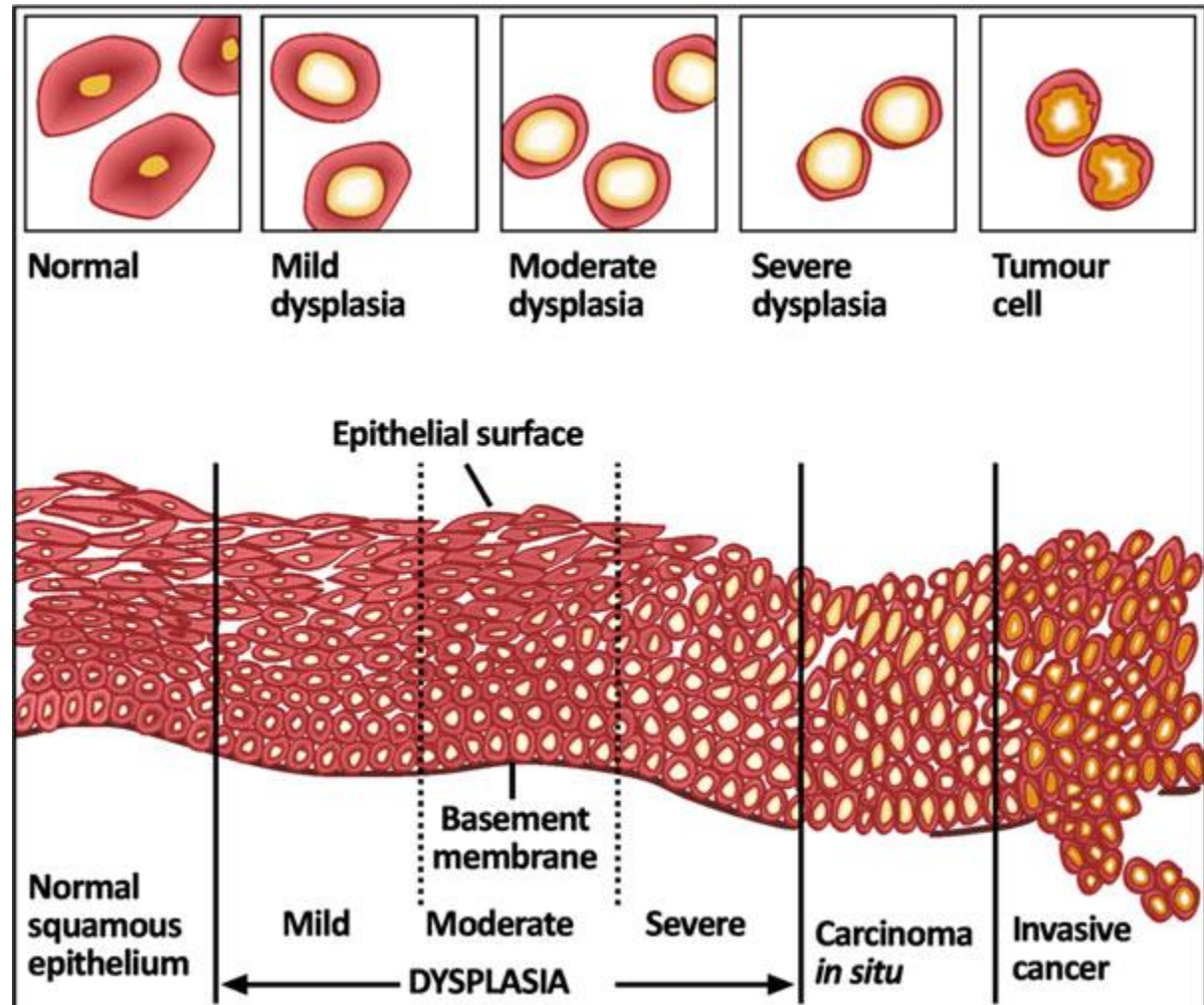
Hormonal tissue neoplasms may still produce the relevant hormones

Ectopic hormone production





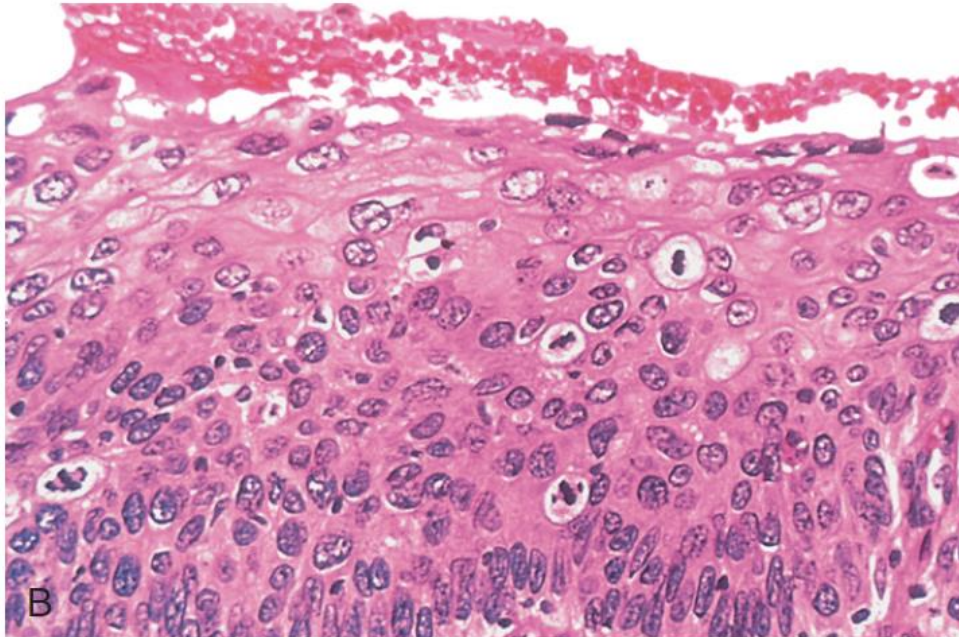
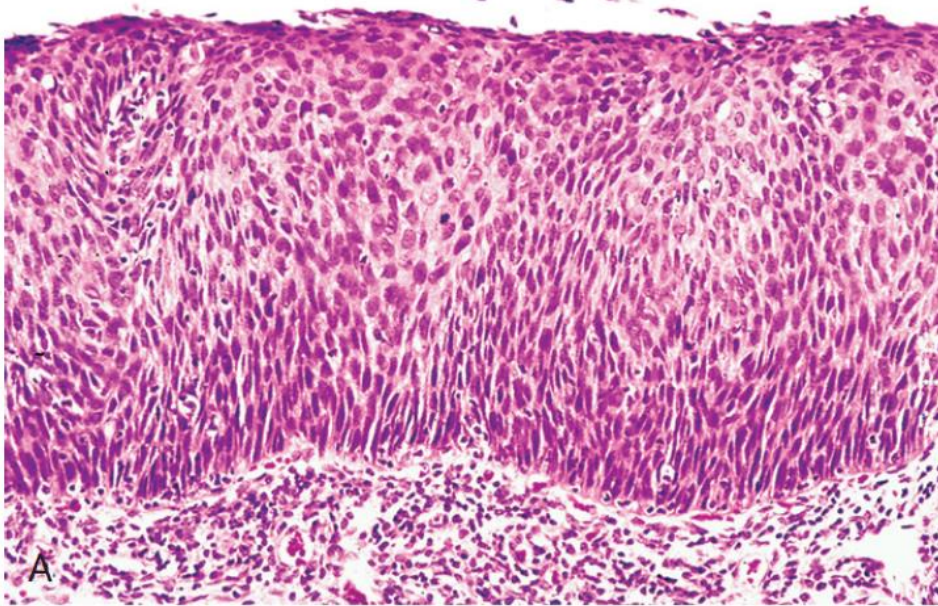
# What about Dysplasia?



Pleomorphism

Hyperchromatic  
nuclei

Mitotic figures more  
frequent & outside  
the basal layer



## Carcinoma-in-situ

Preinvasive

Whole epithelium  
thickness involved

Basement membrane  
intact

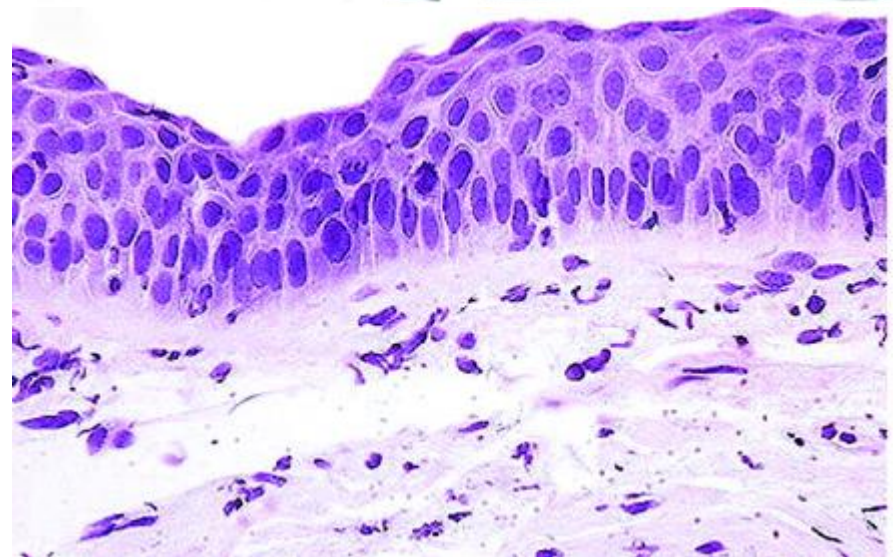
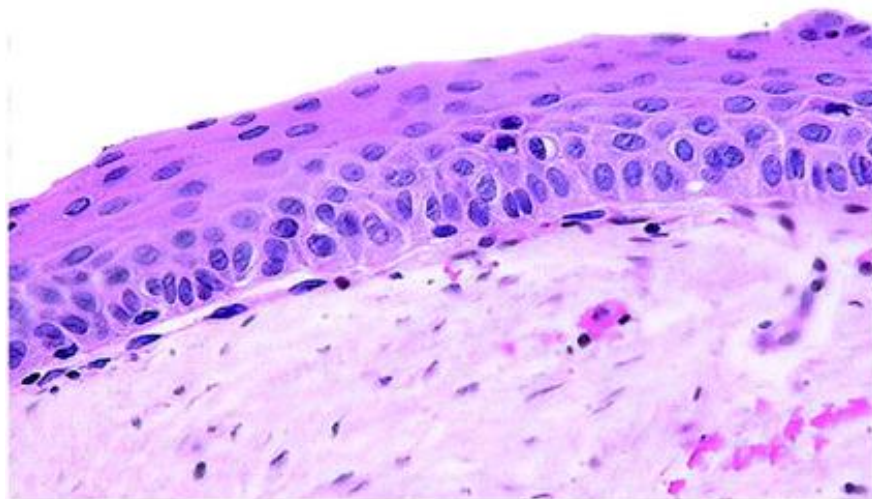
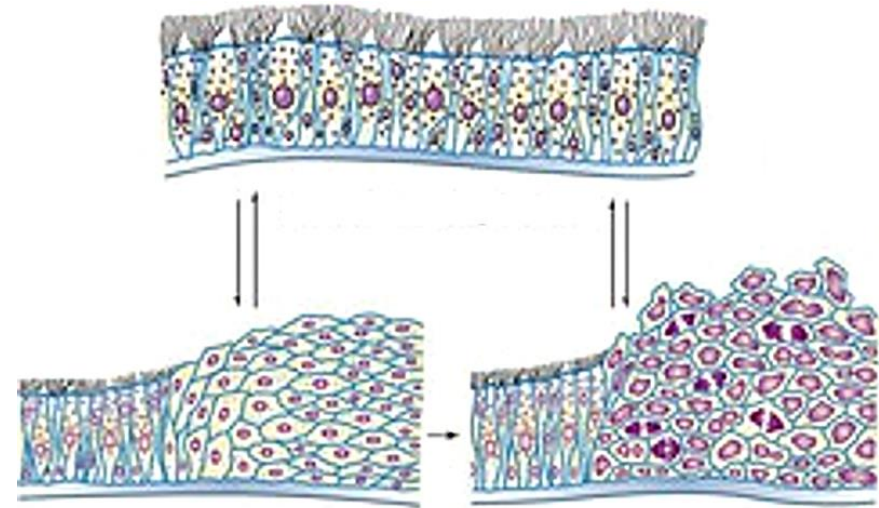
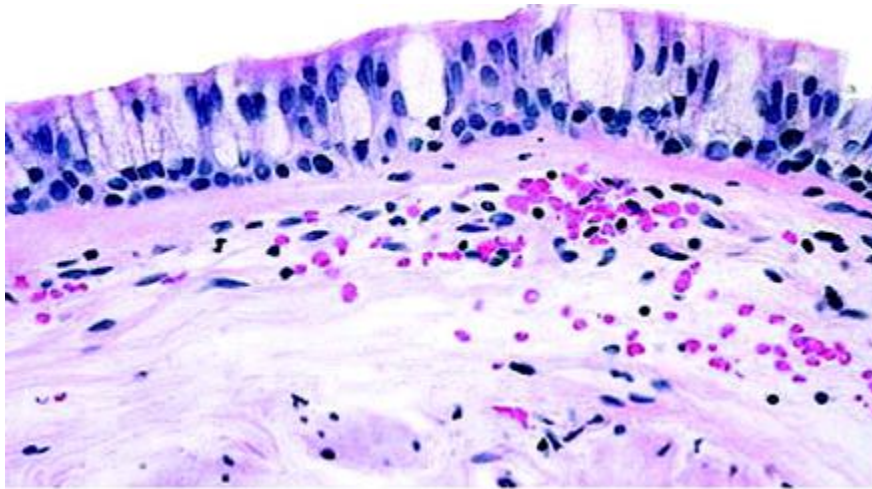
Pleomorphism

Hyperchromatic nuclei

Mitotic figures more  
frequent and outside the  
basal layer



# Metaplasia-Dysplasia-CIS







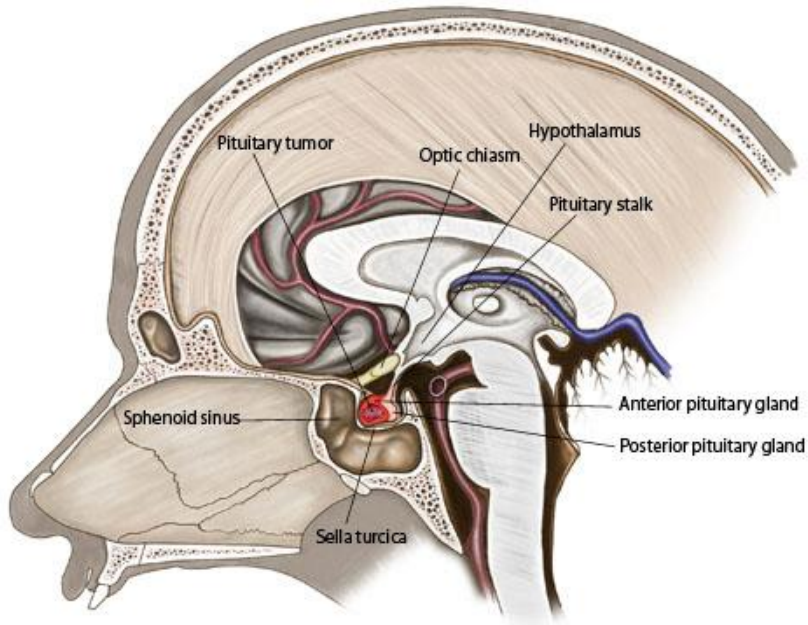
## Rate of growth

Benign/well differentiated  
= slow\*

Malignant/poorly  
differentiated = fast\*

## Factors

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

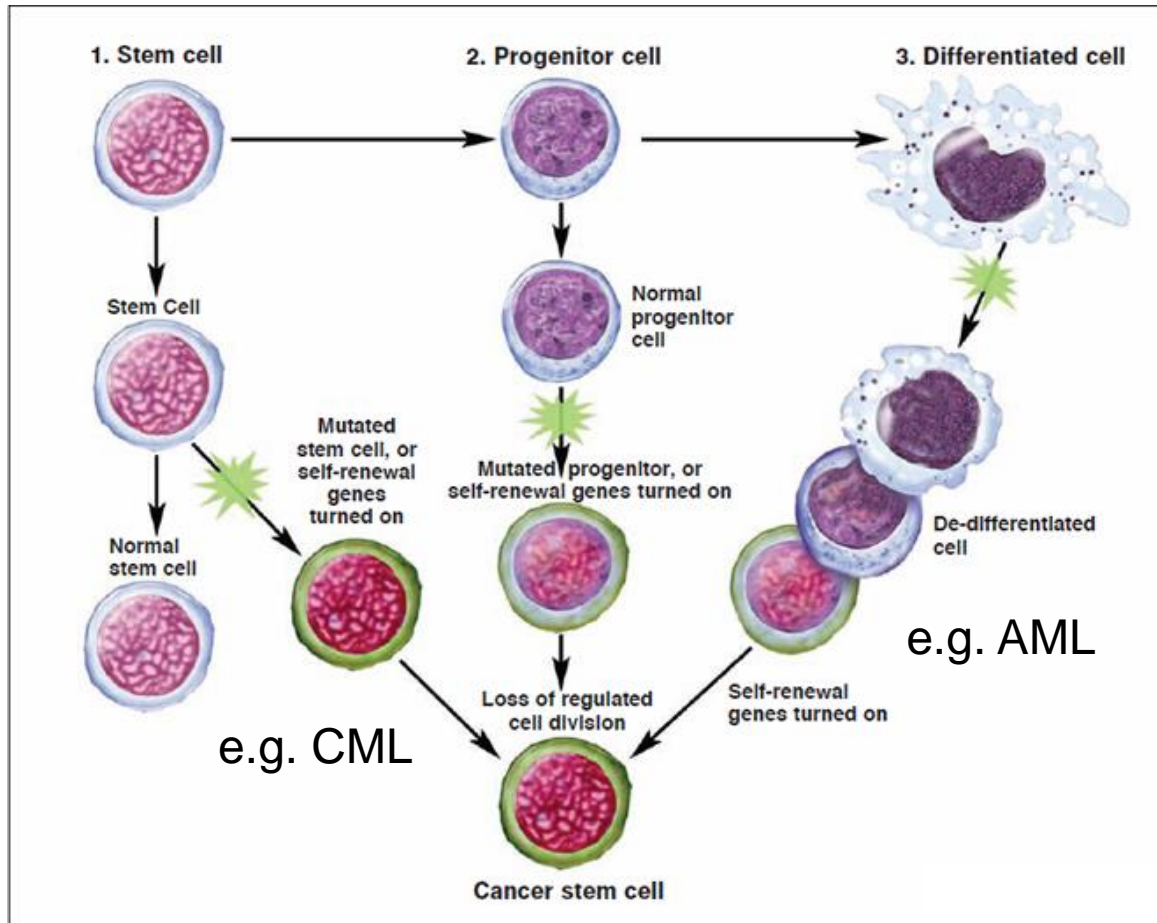


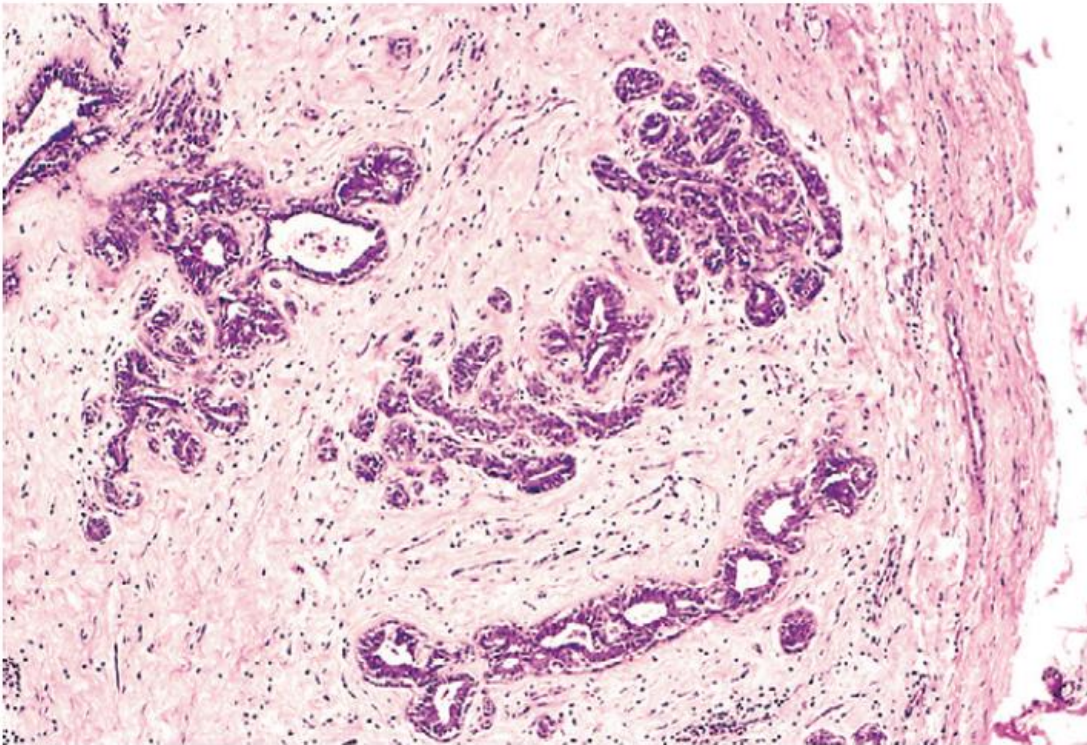
## Rate of growth

### 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





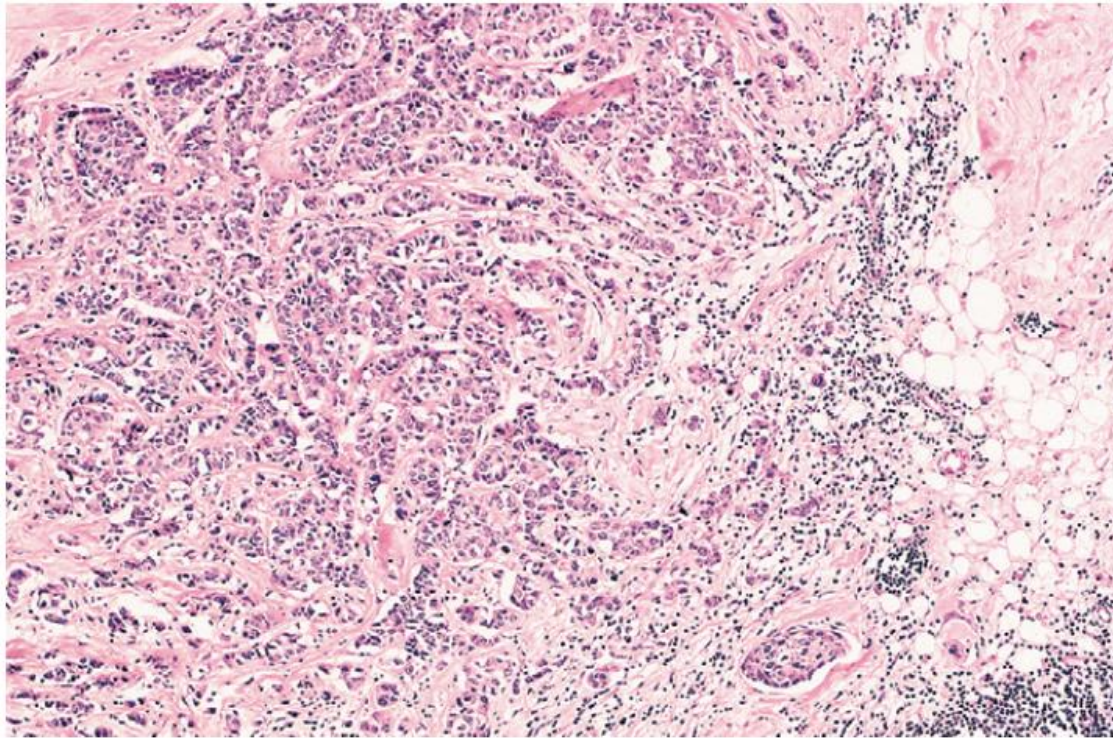
## Local Invasion

### 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 cytostasis
- 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

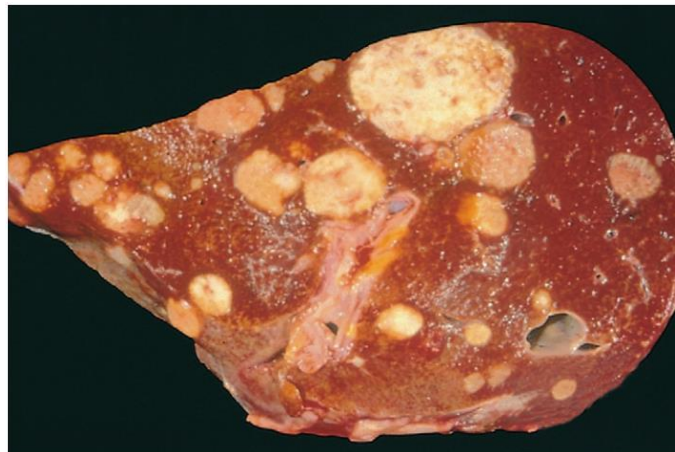
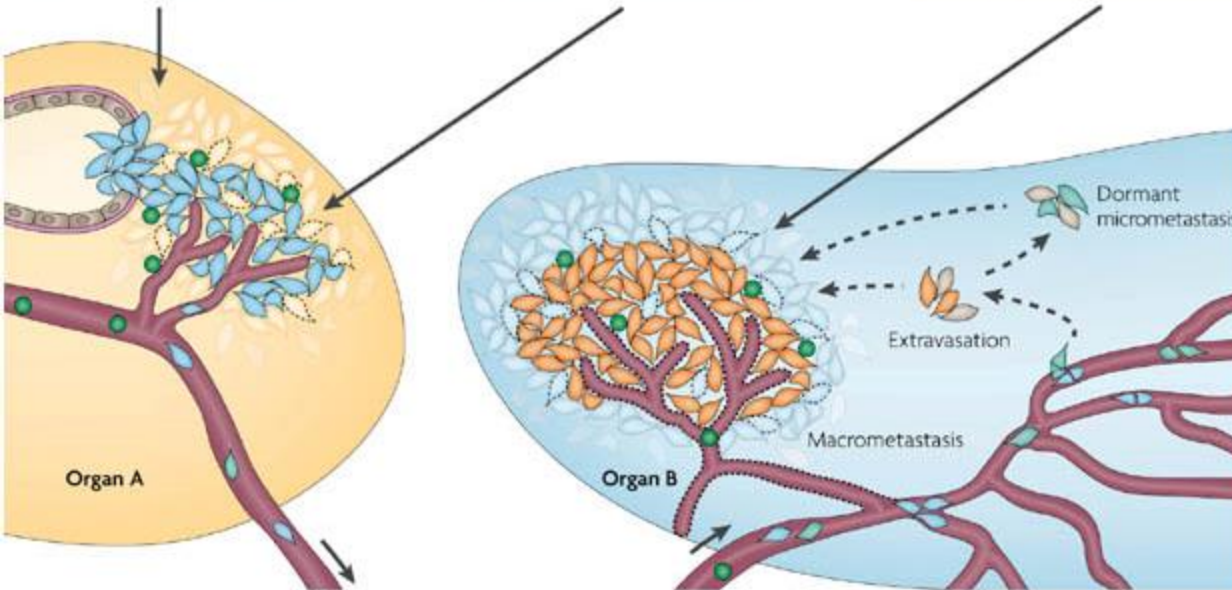
Benign neoplasia do not metastasize

Not all malignant neoplasia metastasize  
(Biology/Time)

Anaplasia & big → more likely to metastasize\*

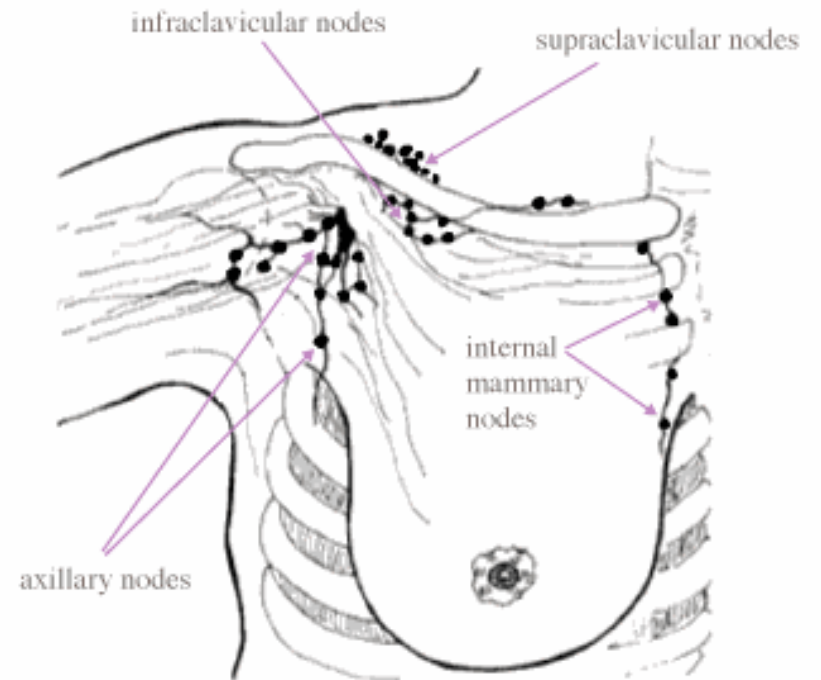
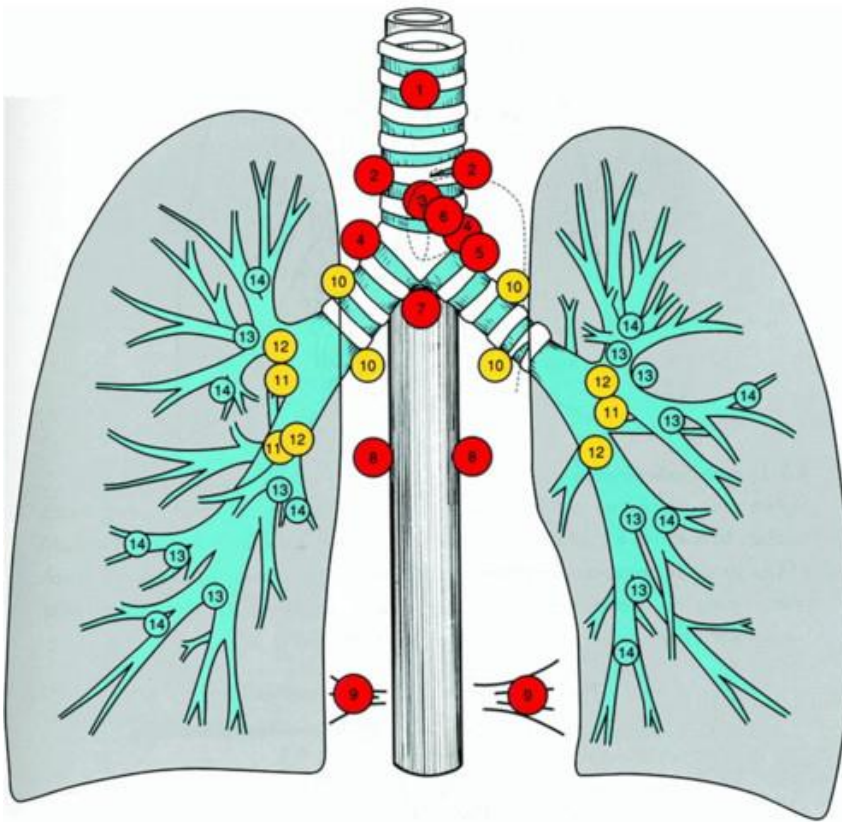
Spread by:

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



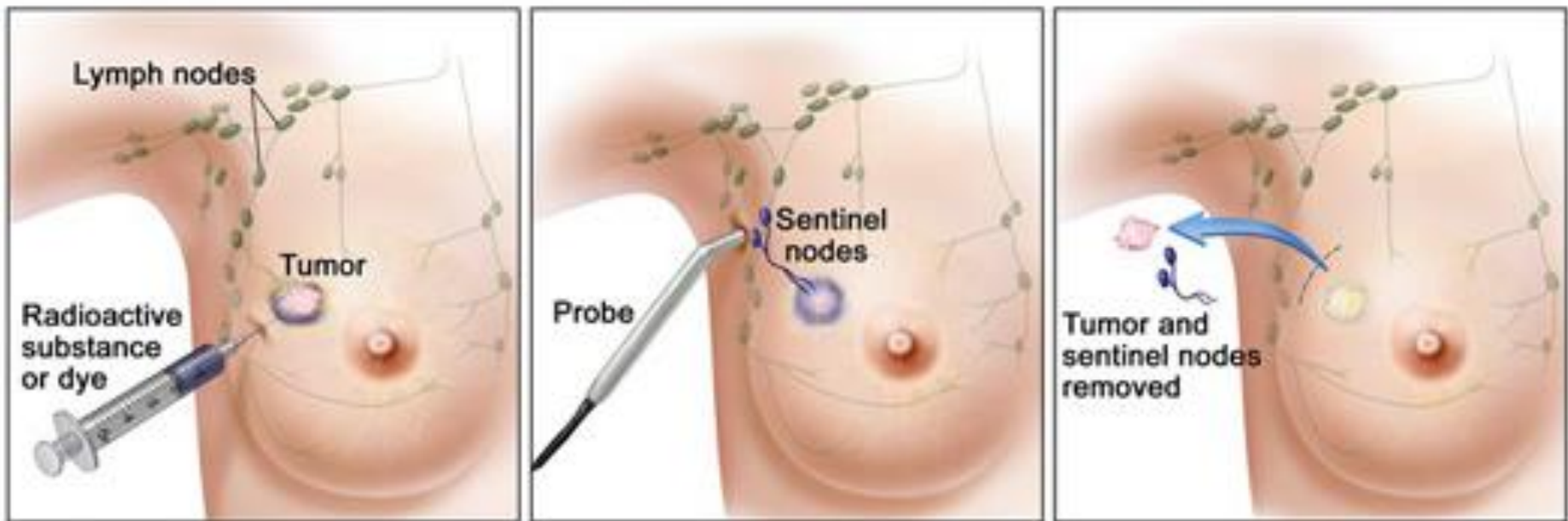


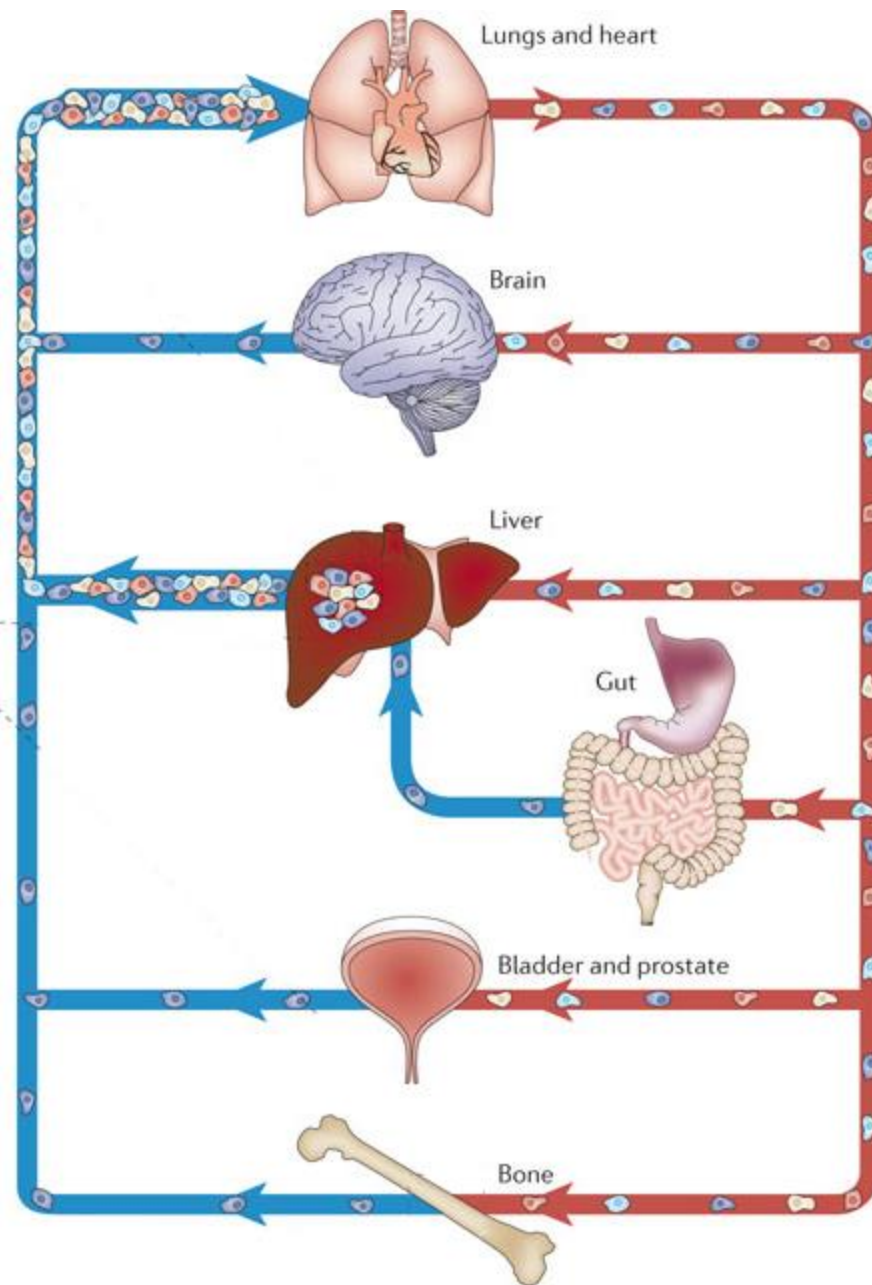
# Lymphatic drainage



# Sentinel Node Biopsy

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## Hematogenous metastasis

Vein invasion

First capillary bed encountered

Portal → liver

Caval → lung

Anatomy cannot explain all metastasis\*

# Summary of extremes

