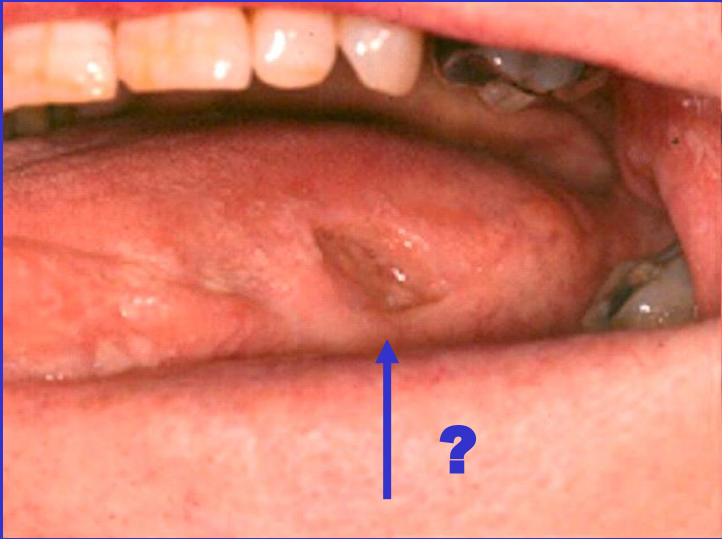


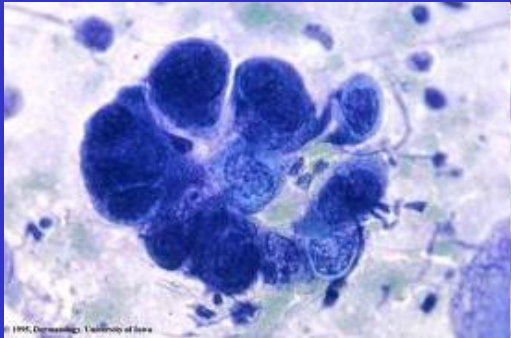
**ORAL PATHOLOGICAL SAMPLING  
AND  
PROCESSING**

**ATTILA ZALATNAI**

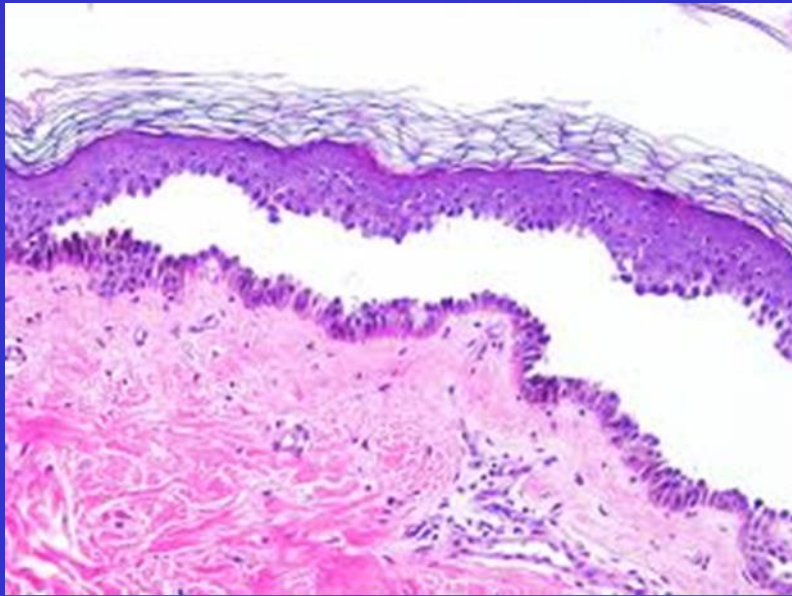
Cytology



Tzank-cells

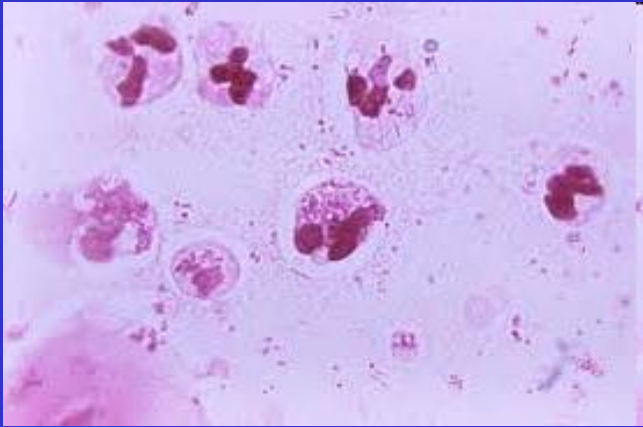
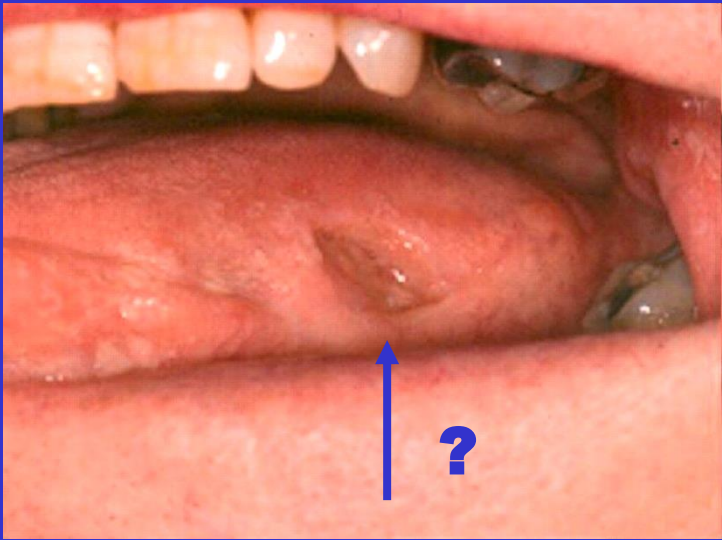


pemphigus

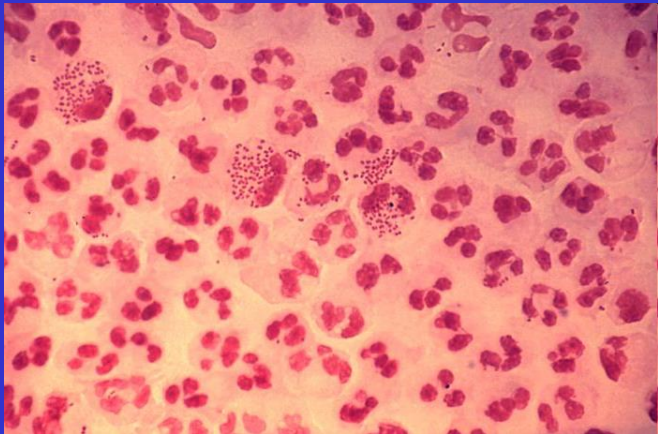




# Cytology



leukocytes, bacteria

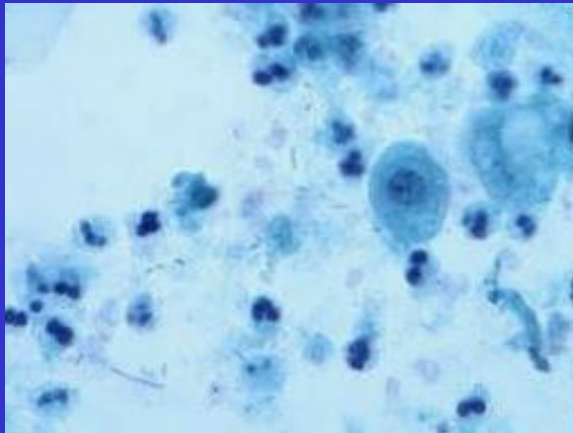
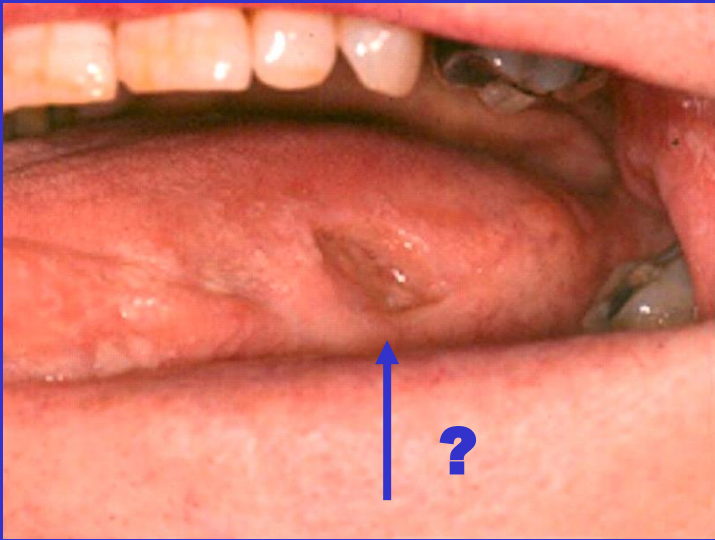


Gram -neg. gonorrhea

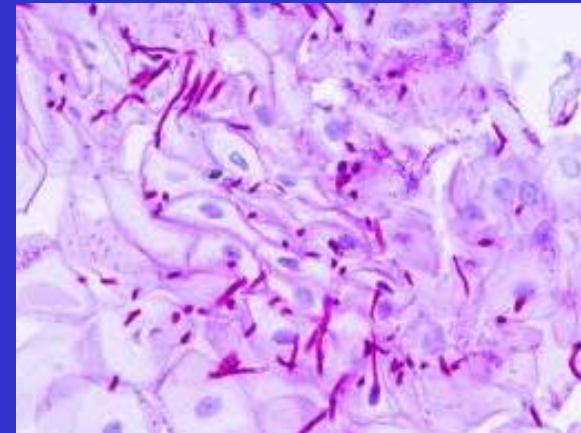
No pathological processing is needed...



# Cytology

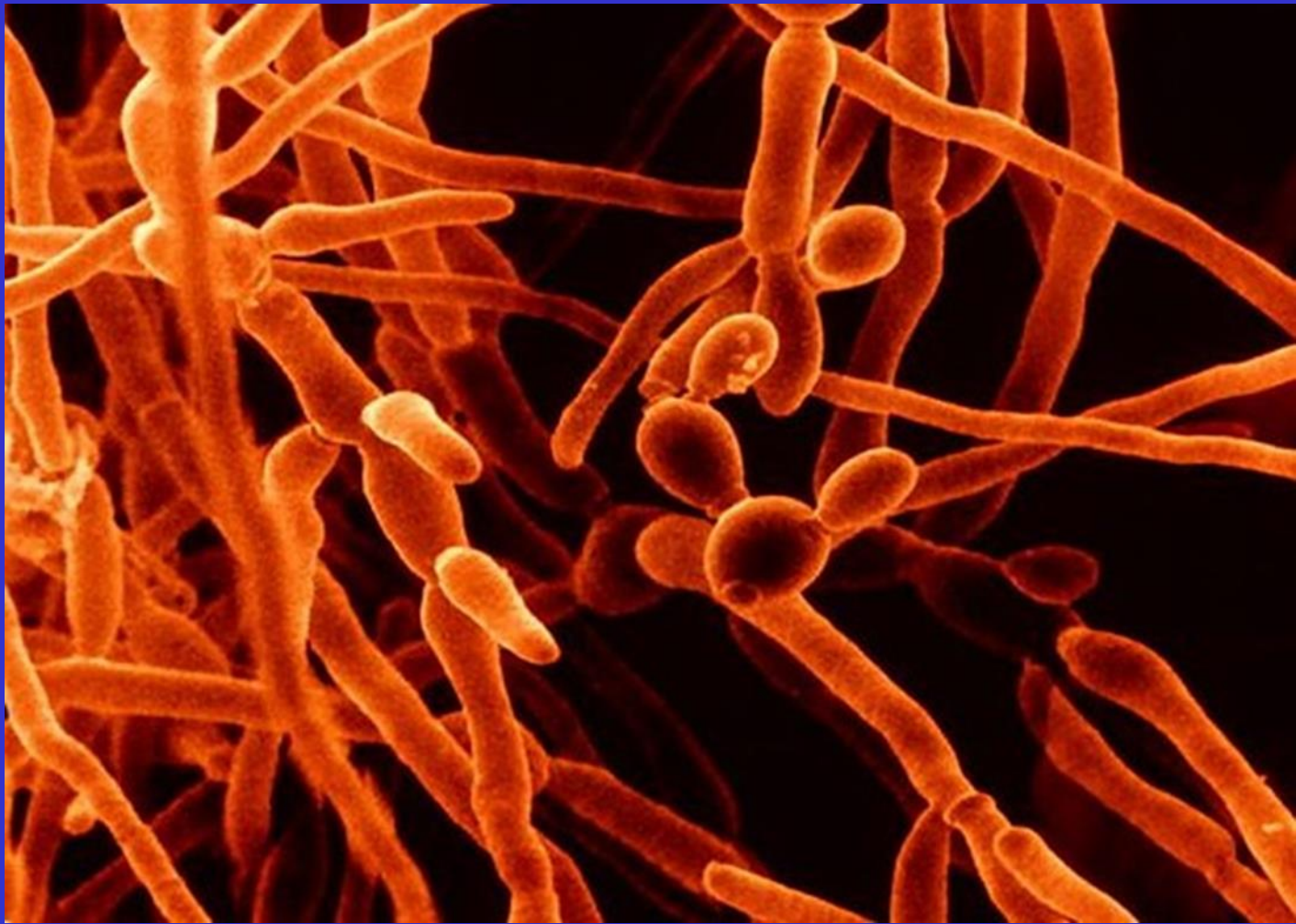


inflammatory cells, fungus-like elements

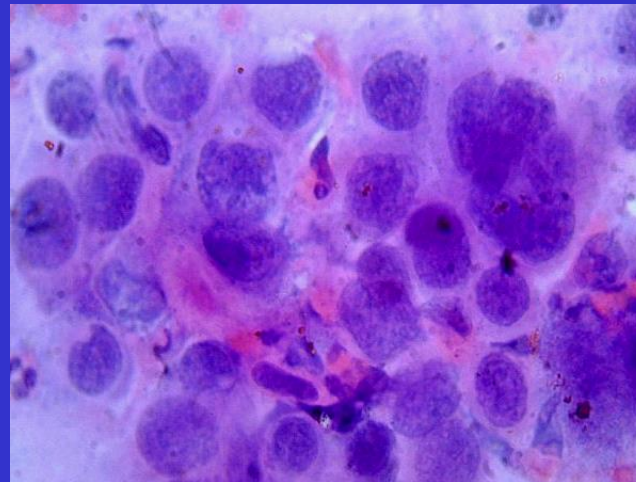
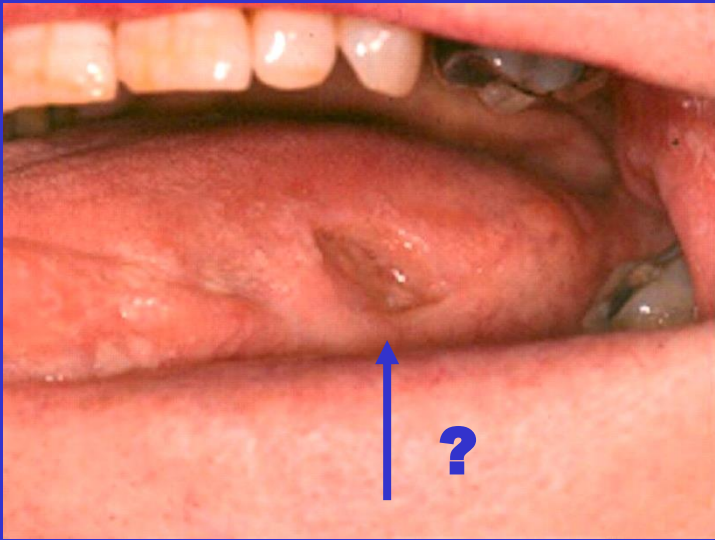


PAS - Candida infection





## Cytology

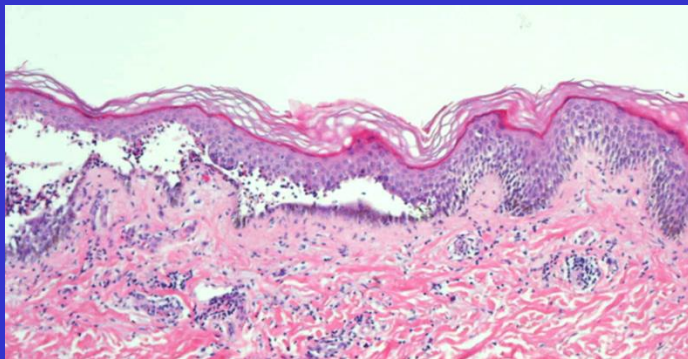


Polymorphous cells - squamous cell cancer

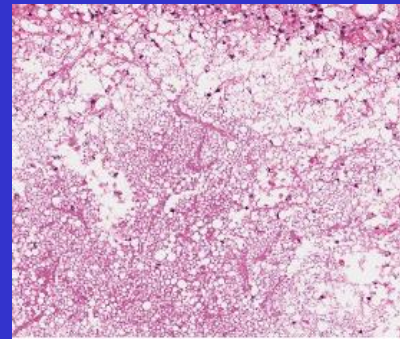




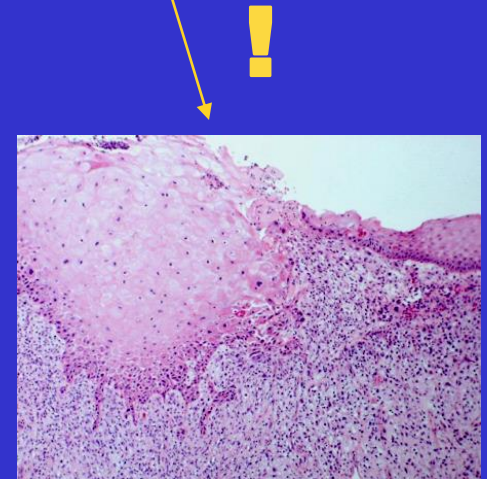
# Histology



Suprabasal dyshesion (pemphigus)

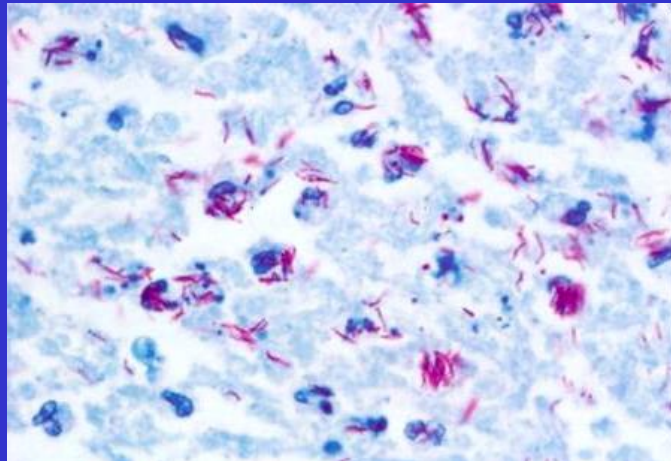
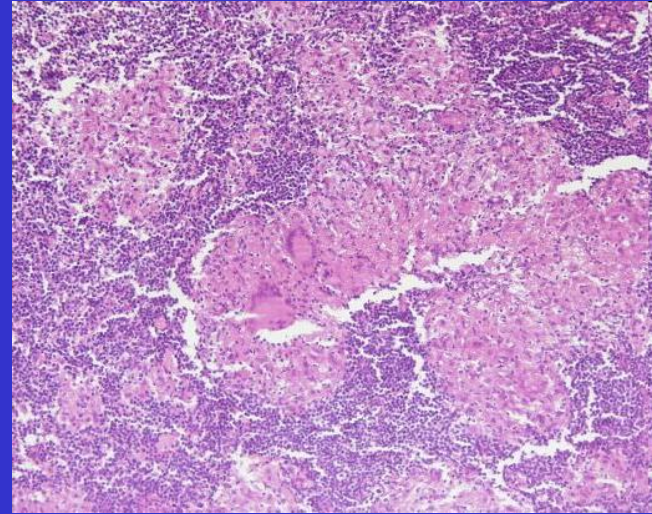


Ø





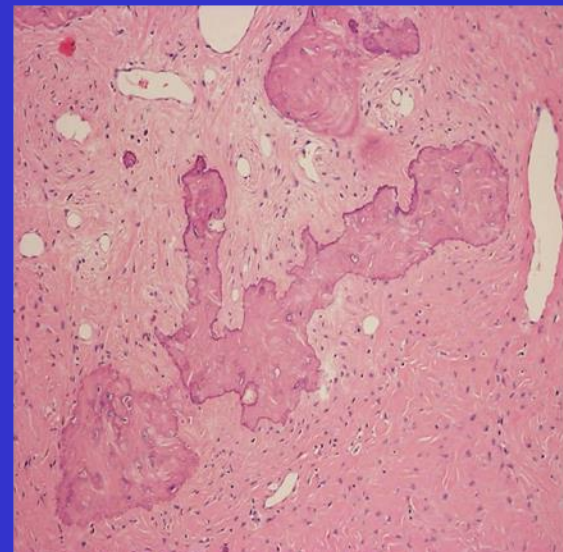
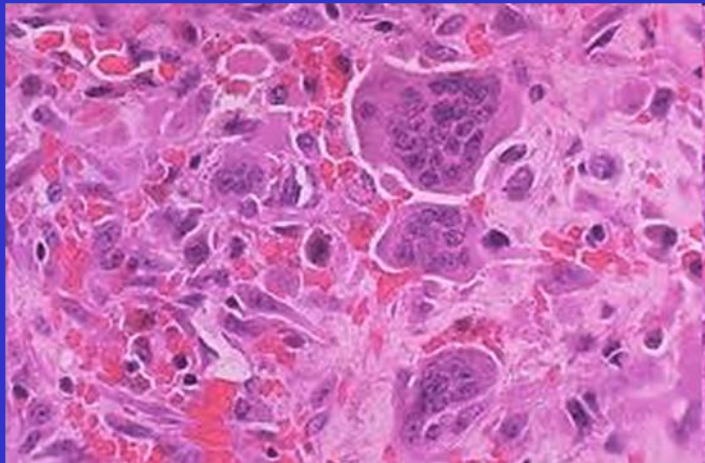
## Histology



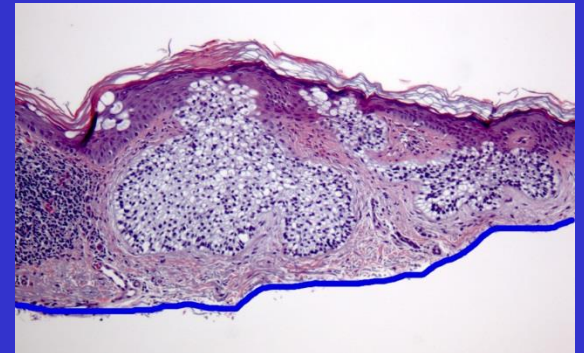
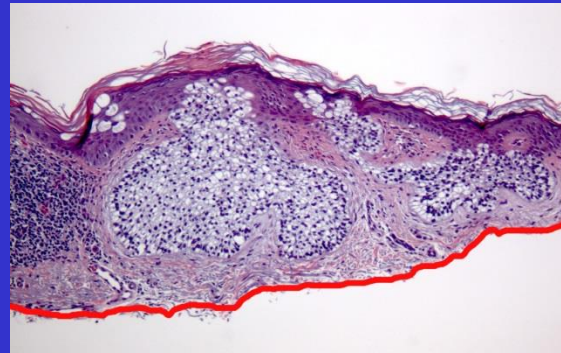
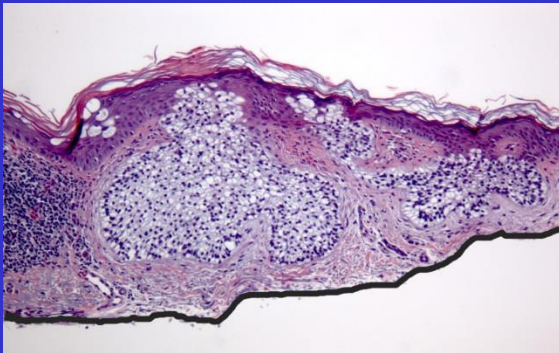
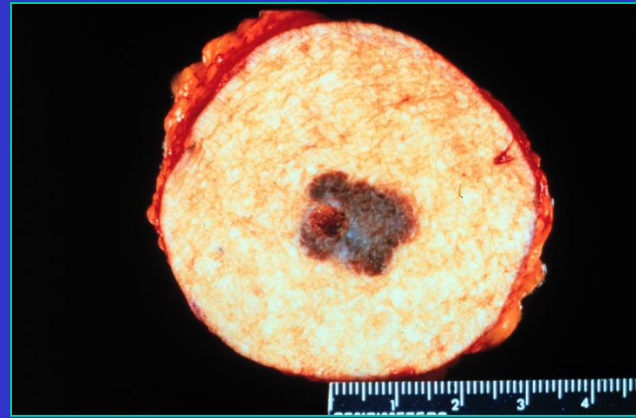
(Ziehl-Neelsen)



## Bony lesions

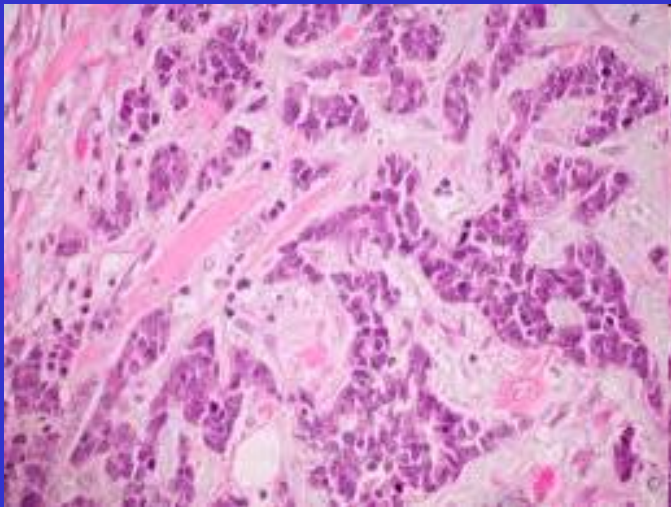
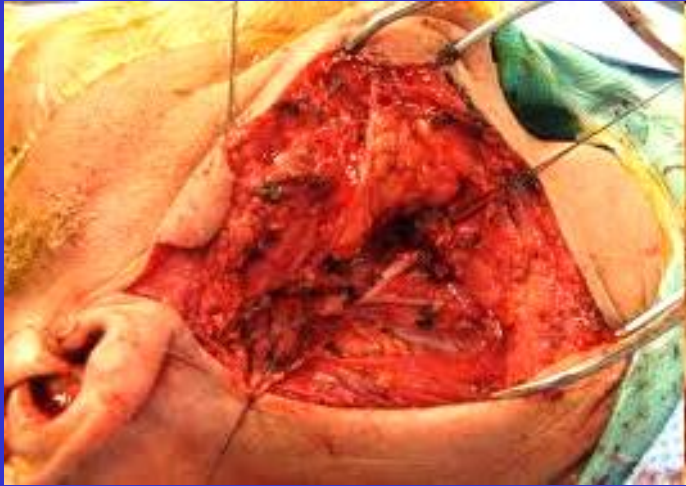






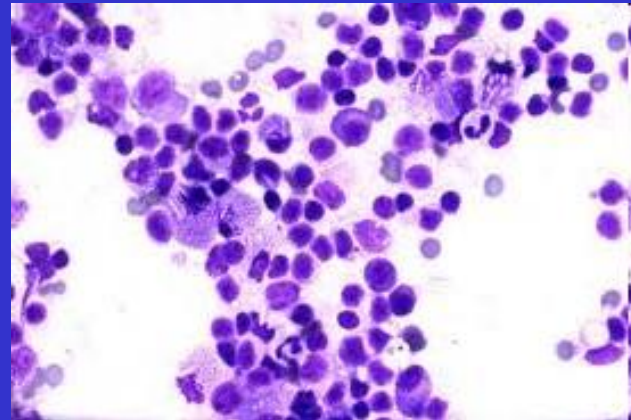
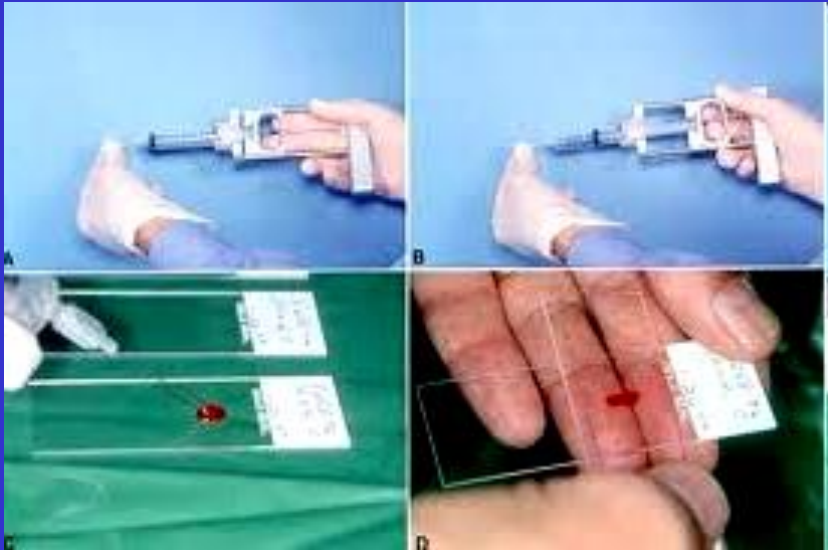


# Intraoperative frozen sections





# Fine needle aspiration cytology (ABC, FNAC, FNAB)



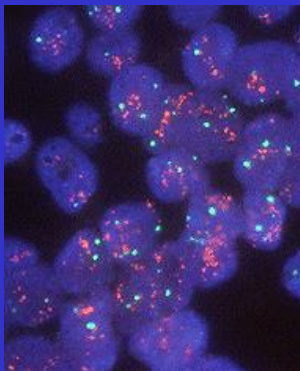
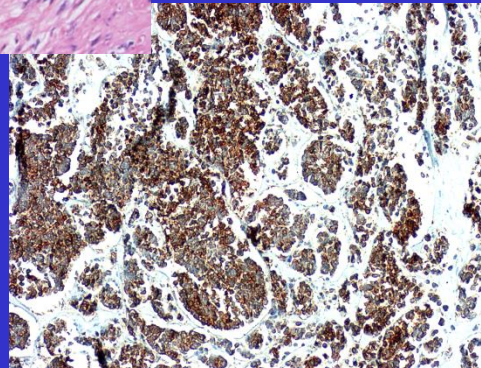
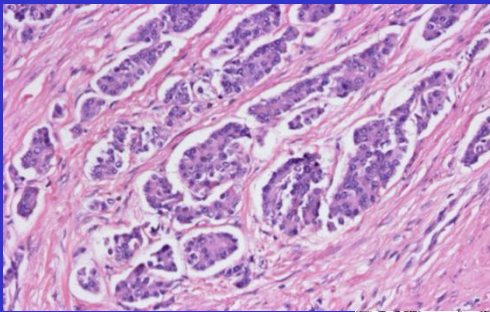
# Sending materials to pathology

## Formalin (4 – 8 %)

Routine histology

Molecular pathology

Immunohistochemistry

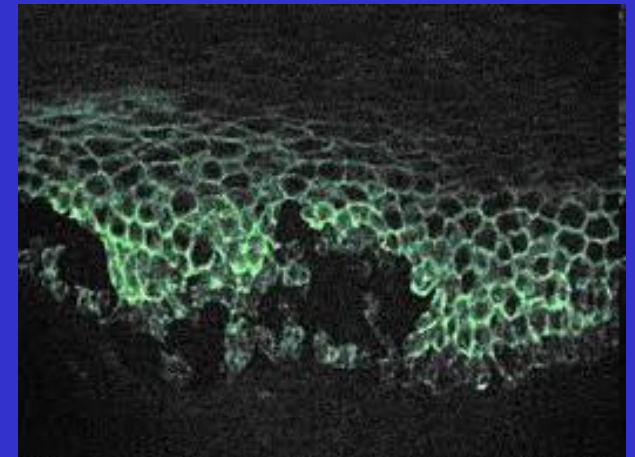


## Saline

Frozen sections

Immunofluorescent m.

(Electron microscopy)



# **OROFACIAL MALFORMATIONS**

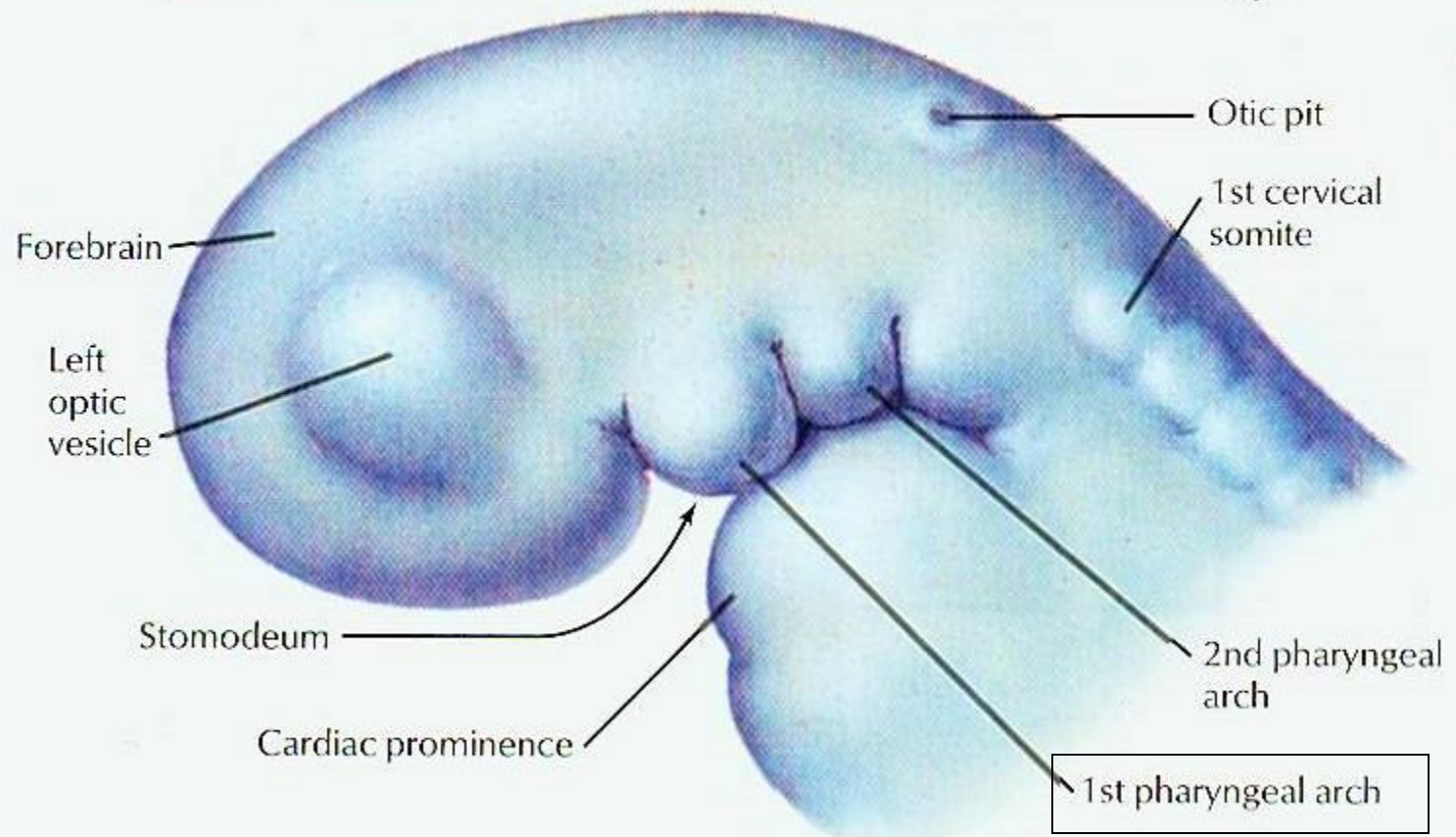
**ATTILA ZALATNAI**



Embryo at 3 to 4 weeks

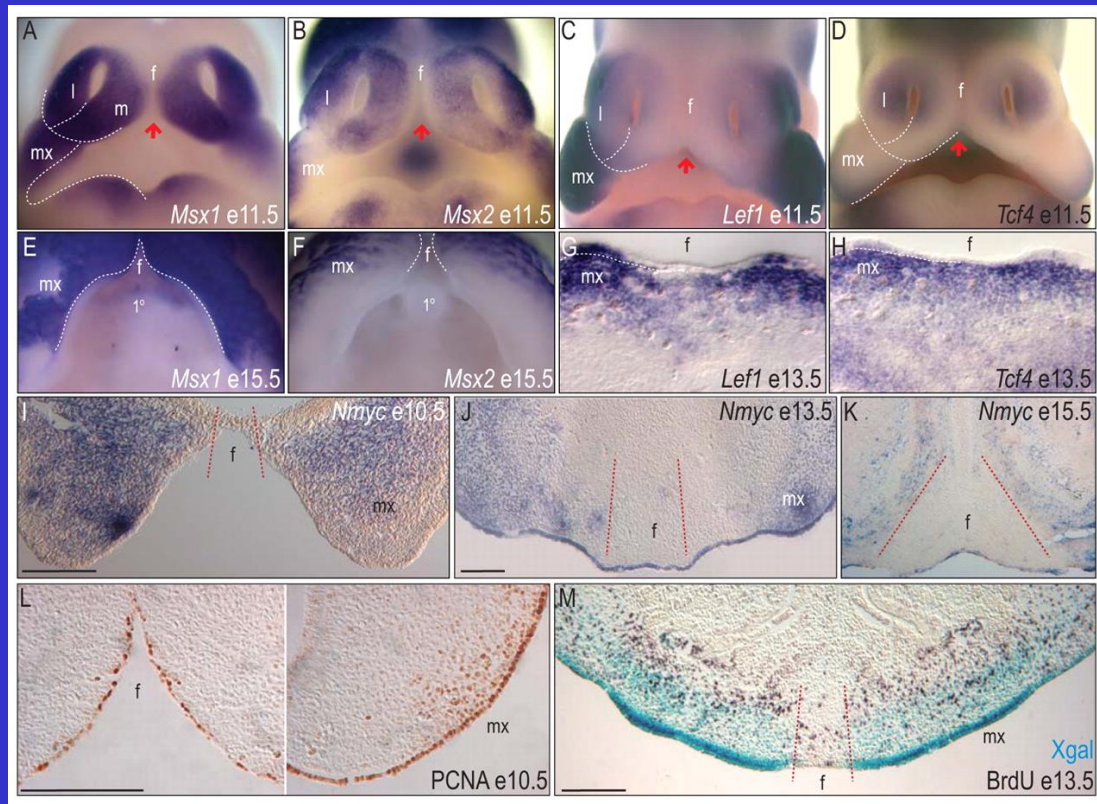
Lateral view

1.0 mm



derivates of the 1st branchial arch are:  
maxilla, mandible, zygomatic, palate,  
muscles of mastication

WNT-signaling pathway has a key importance



WNT family

Regulate cell-to-cell  
interactions during  
embryogenesis

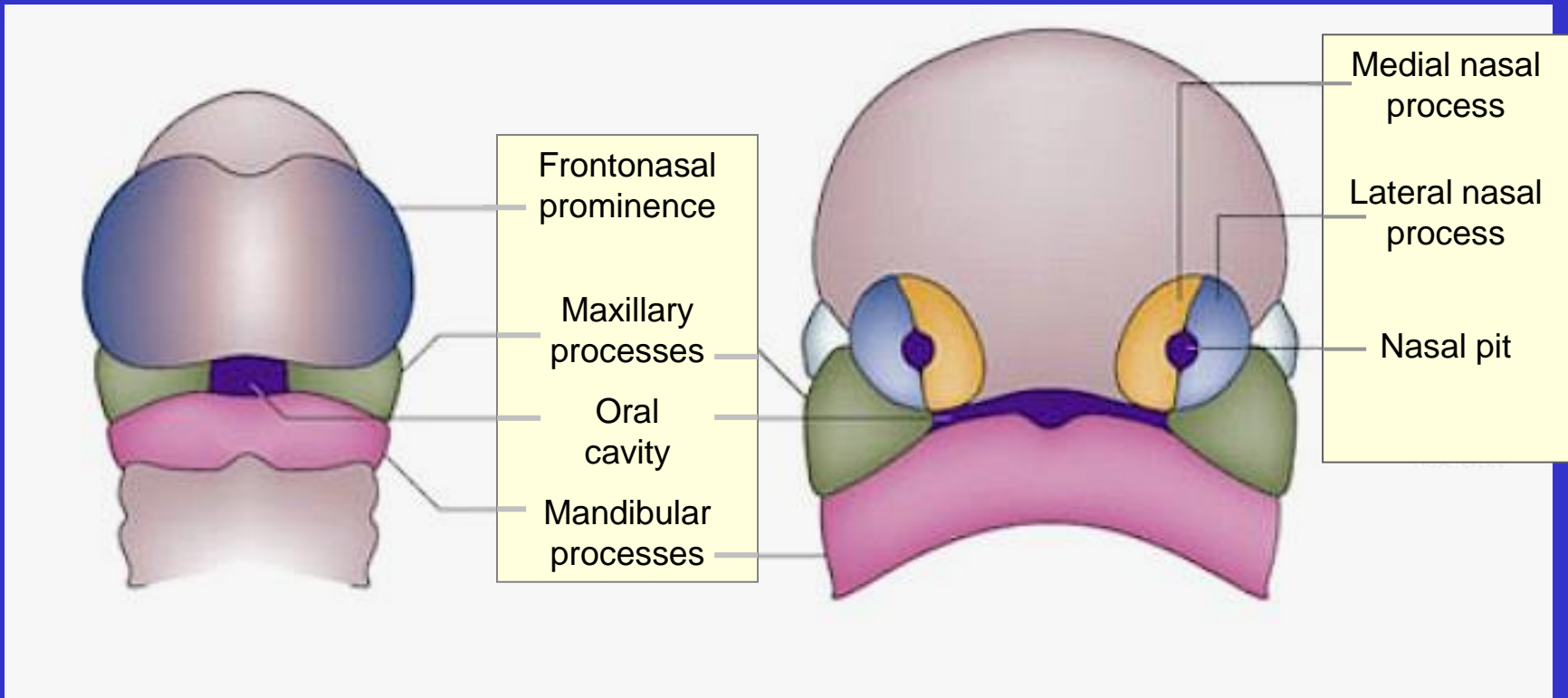
Mutations:

lead to malformations

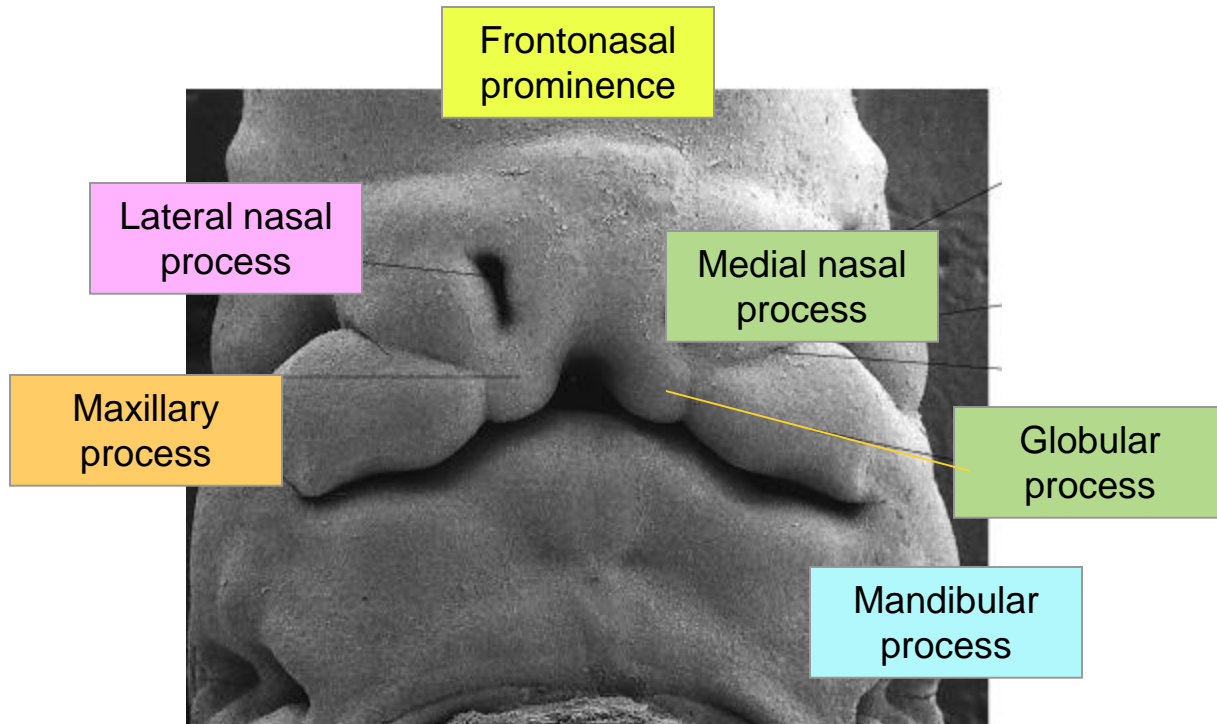
week 4



week 5







# Week 6

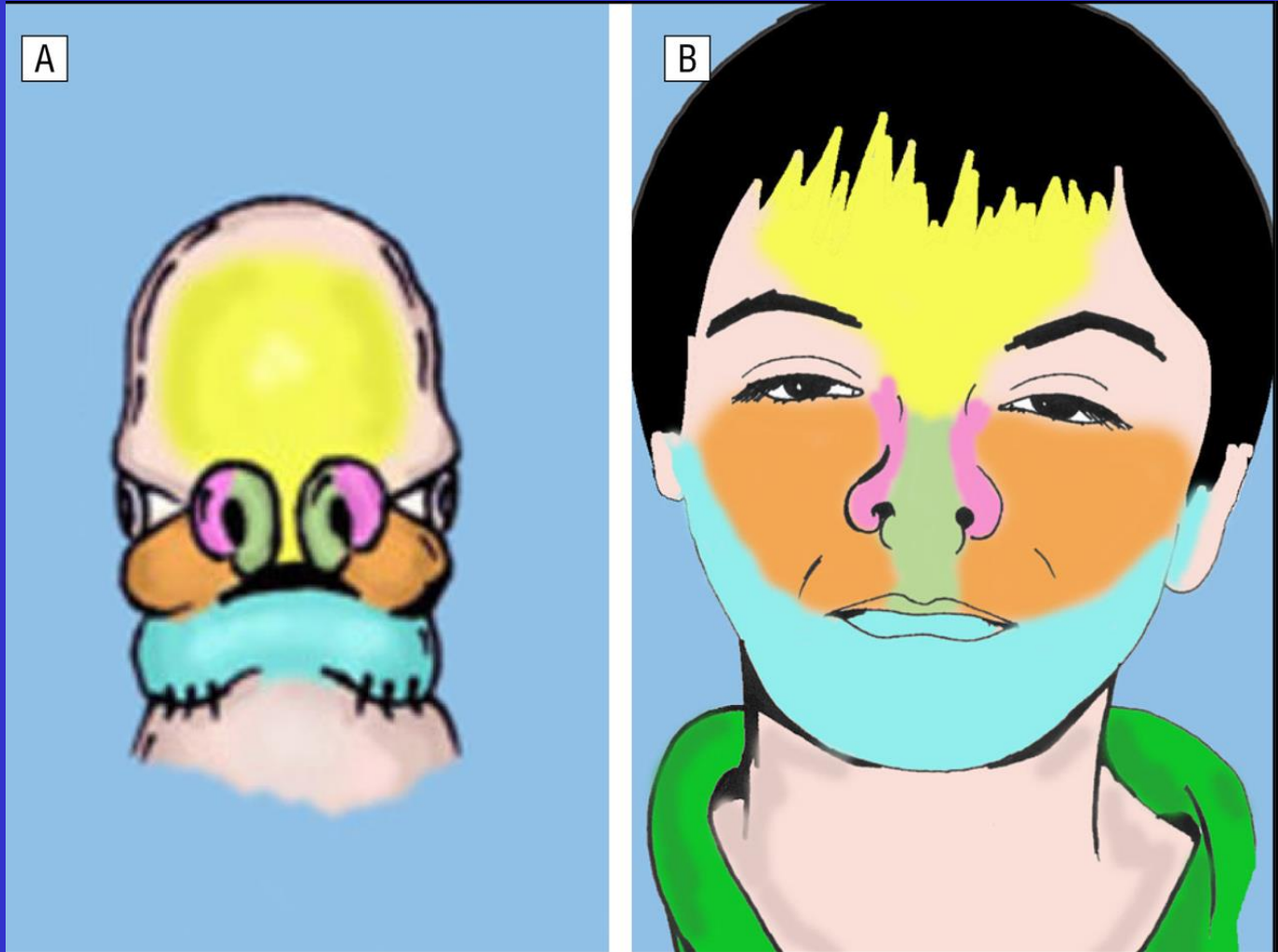
Frontonasal prominence

Lateral nasal process

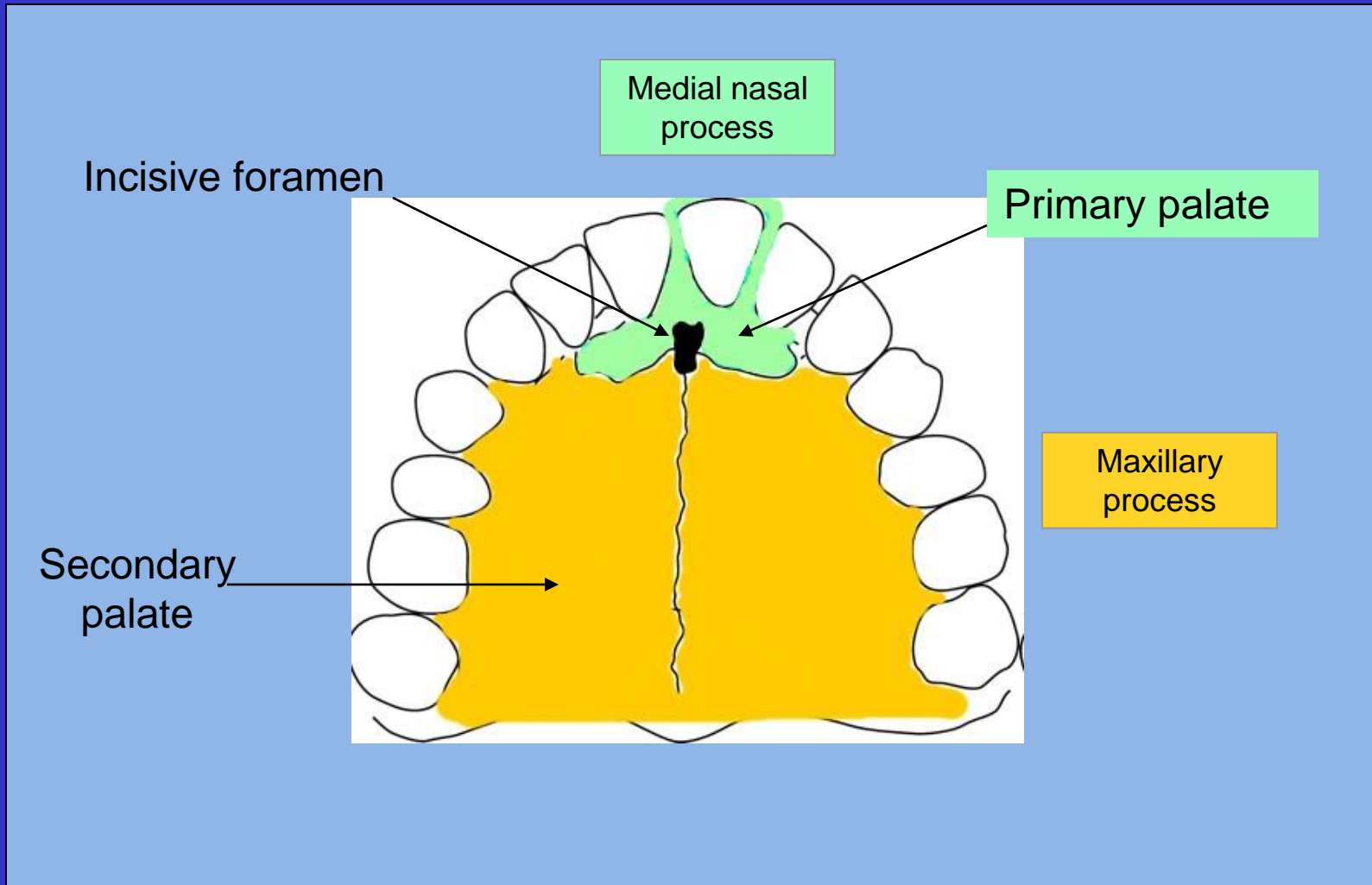
Medial nasal process

Maxillary process

Mandibular process

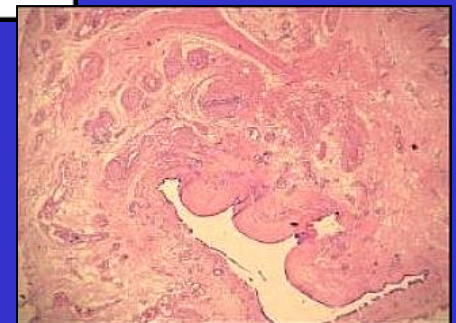
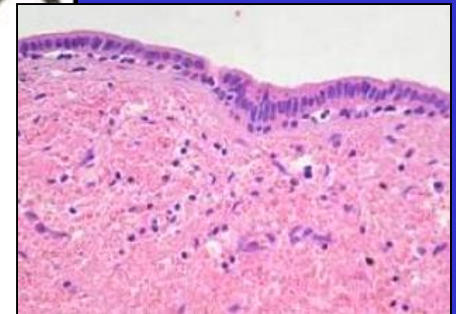
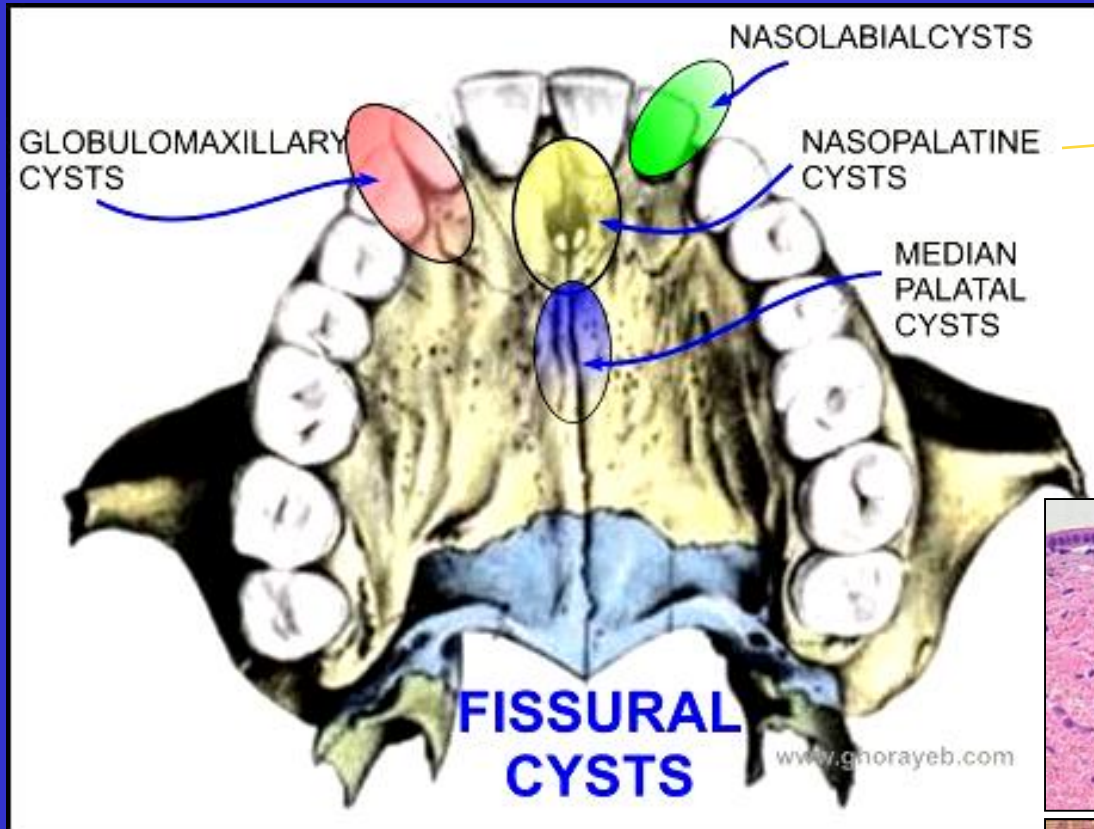


# Development of palate





# Palate malformations – fissural (non-odontogenic) cysts –



## Facial clefts (defective fusion)

Frequency at birth (Hungary) : 2 ‰ (180-200 cases per year)

Multifactorial origin:

(mutations in the genes governing migration, noxas affecting during pregnancy:

smoking, viral infections, drugs, chemicals, overuse of vitamin A, X-linked forms)

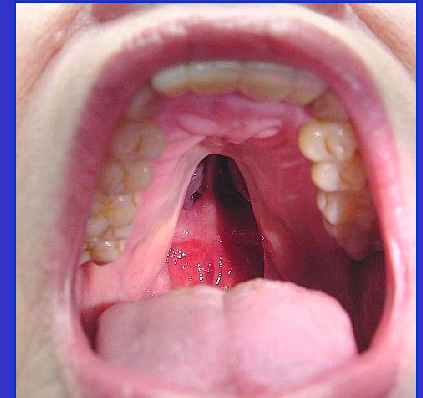
As parts of various syndromes (appr. 150)

- a. **Cleft lip (labium leporinum) ± cleft palate (faux lupina)**
- b. **Isolated cleft palate**
- c. **Gnathoschisis (split alveolar process)**
- d. **Cheilognathopalatoschisis**

# Cleft lips

Unilateral or bilateral

Complete or incomplete



Cleft palate



a. Cleft lip (labium leporinum) ±  
cleft palate (faux lupina)

teratogenic effects: 7-9. gestation weeks

b. Isolated cleft palate

teratogenic effects: 10-14. weeks

+ different genetic background

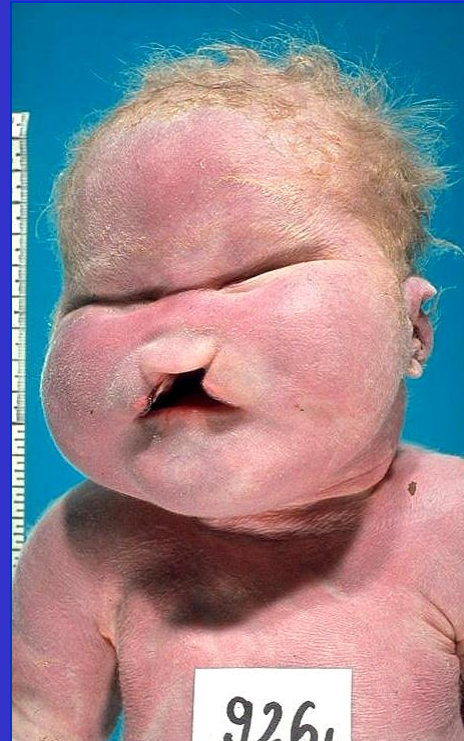


**BEFORE**



**AFTER**

# Cheilognathopalatoschisis







179/2006

# Disorders of the development of teeth I.

Abnormalities in the differentiation of the dental lamina or the tooth germs

1. Abnormalities of morphodifferentiation (number, size, shape)
2. Abnormalities of histodifferentiation (structural alterations in the teeth)
3. Both stages of differentiation are abnormal

1. Hypodontia, anodontia (1 or more teeth are missing)

most common in females

permanent teeth are typically involved

mutations in the regulatory genes

Hyperdontia (supernumerary teeth)

more frequent in females and in the maxilla

permanent teeth are involved (in deciduous teeth exceptional)

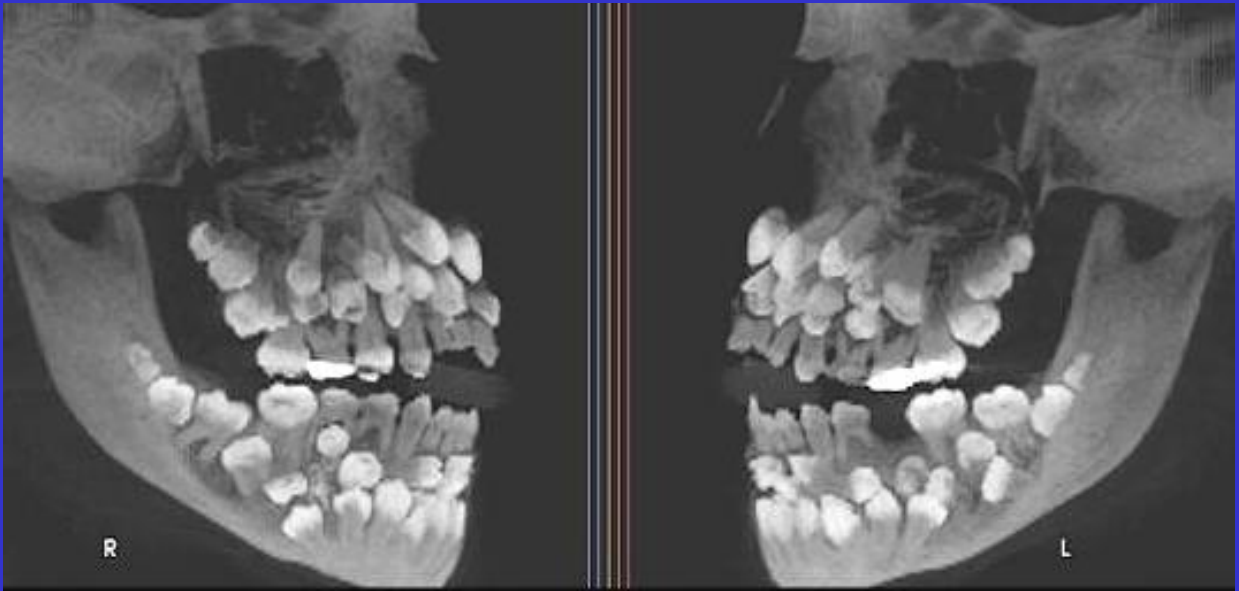
may be associated with other abnormalities (e.g. cleft palate)

Macrodontia. Microdontia

microdontia can be associated with Down-syndrome, congenital heart malformations,



**mesiodens**





## Disorders of the development of teeth II.

Disturbances in tooth form may involve the crown, the root or both

Double teeth

2. Enamel hypoplasia (defective matrix production by the ameloblasts)



Amelogenesis imperfecta (AD, X-linked - mutation in amelogenin gene  
Dentitionogenesis imperfecta

- associated with osteogenesis imperfecta (Type I)
- autosomal dominant (Type II, only the teeth are affected)

Dentinal dysplasia (rootless teeth)

Hypercementosis (idiopathic or resulted from known causes – periapical inflammation, mechanical stimulation, Paget's disease)

## Eruption disturbances

Rare

- a.) Associated with other bone lesions
  - cretinism
  - rickets
- b.) Local factors
  - e.g. gingival fibromatosis