



**UNIVERSITÉ
DE GENÈVE**

FACULTÉ DE MÉDECINE

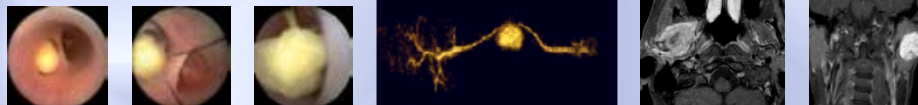


SALIVARY GLANDS

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HUG - Genève



Salivary glands - outline

Physiology

Semiology

Infections

Sialolithiasis

sialendoscopy

..... Inflammations

Tumors

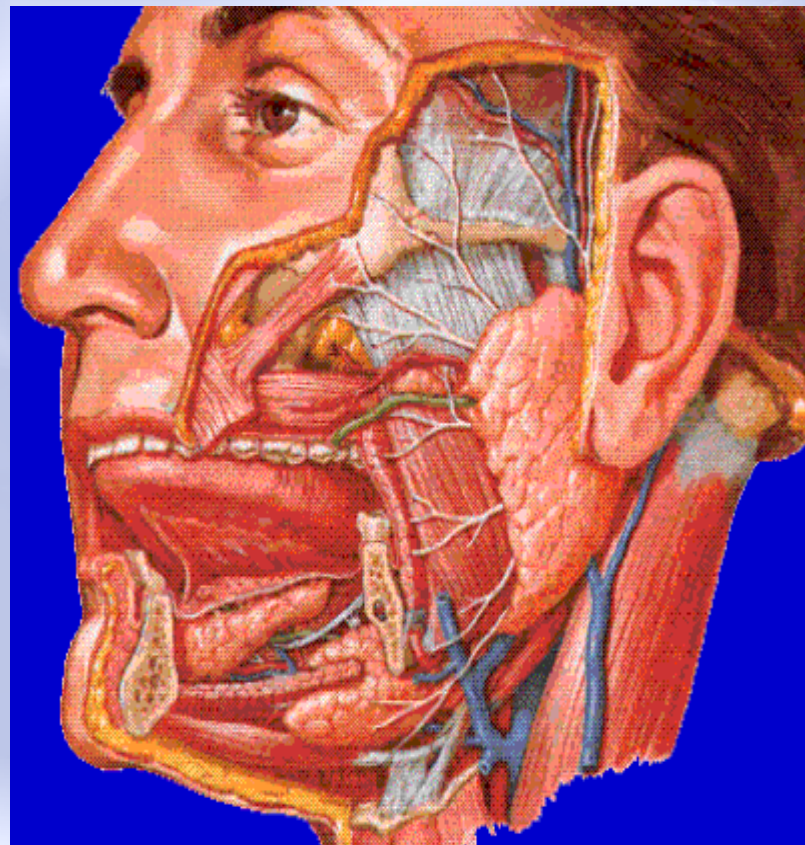
benign

pleomorphic adenoma

cancers

Surgery

parotidectomy



Salivary glands - objectives

Semiology of salivary gland diseases

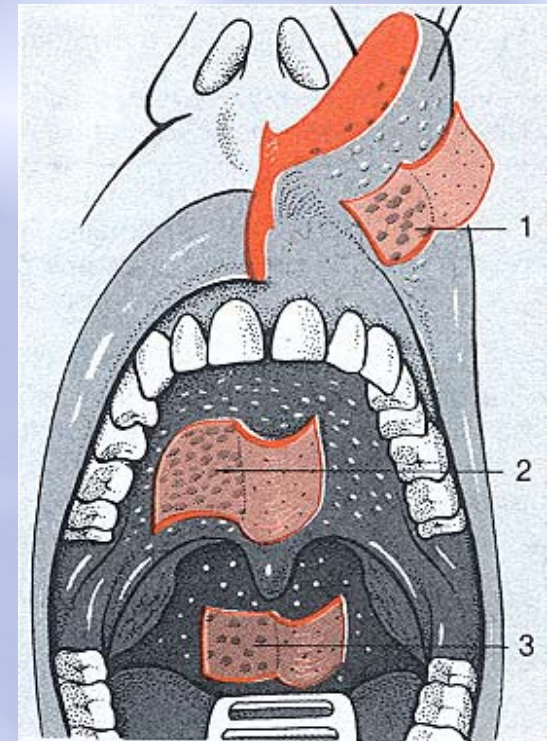
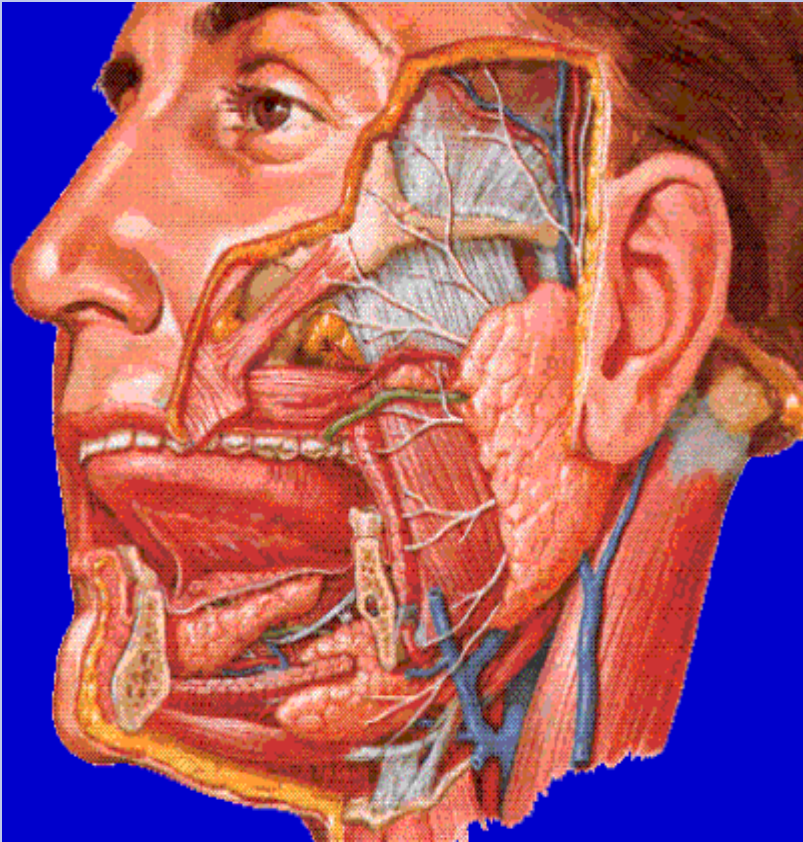
Management of salivary ductal obstruction

Salivary gland tumors

Pleomorphic adenoma

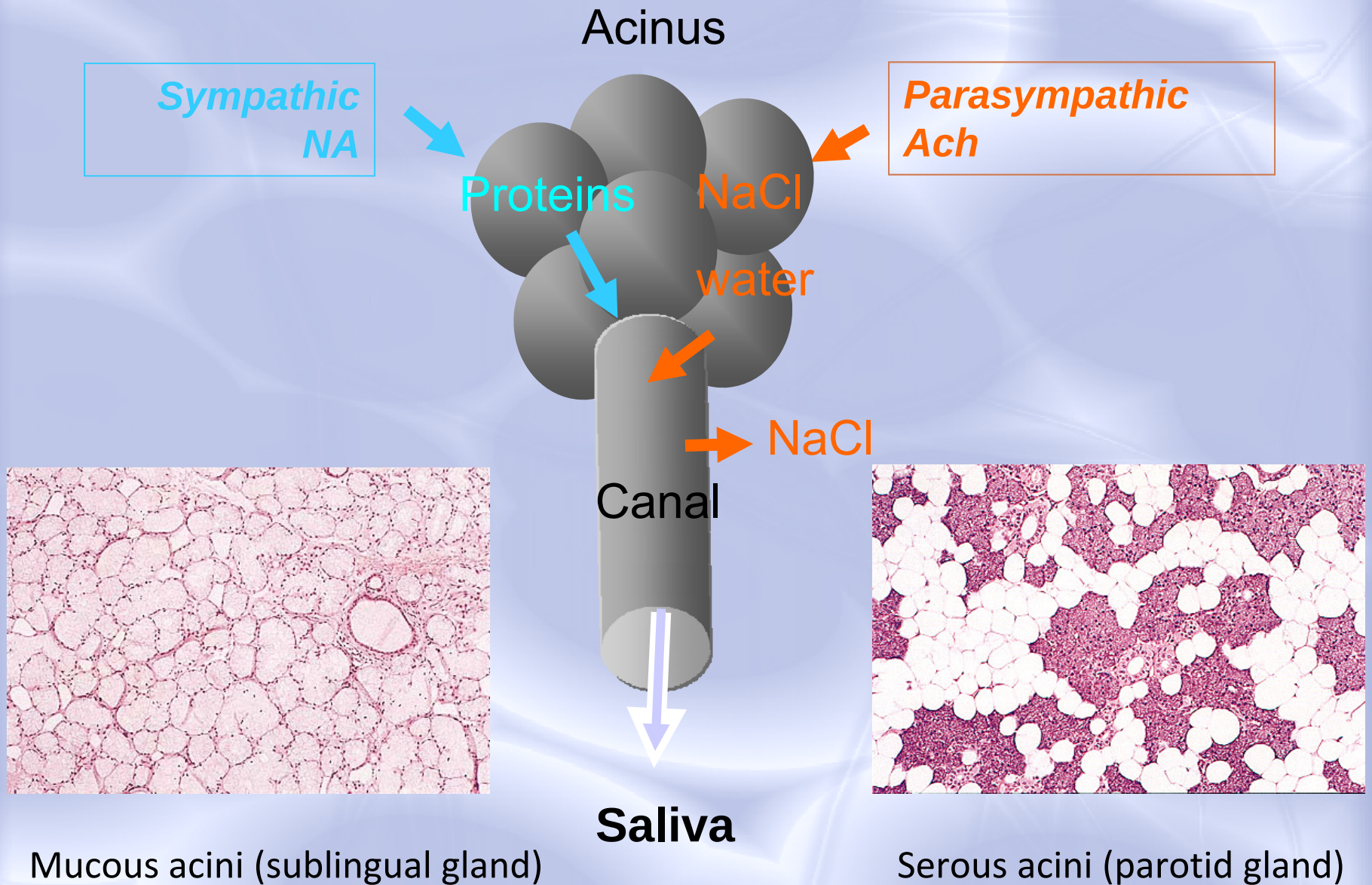
Parotidectomy

Treatment of salivary gland carcinoma



Minor glands in all upper AD tract

Same histology → same tumors

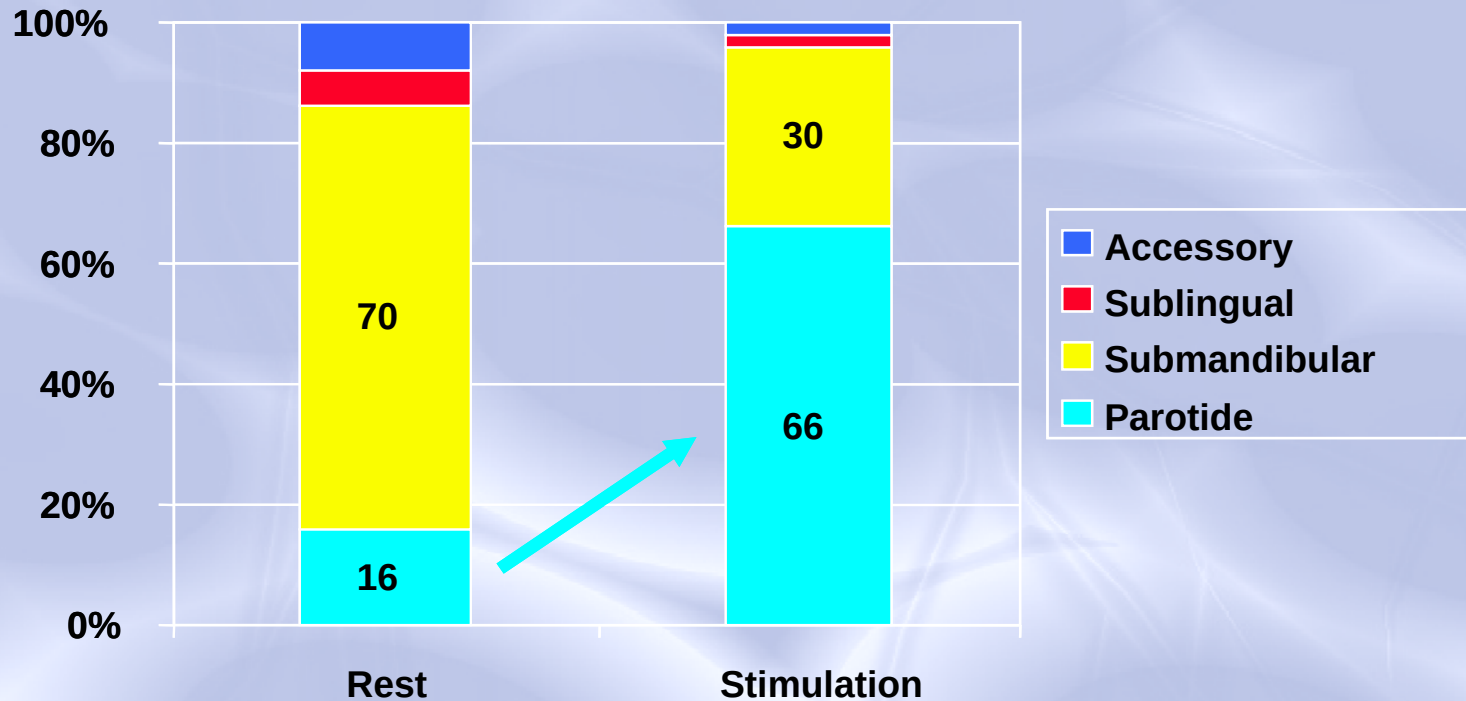


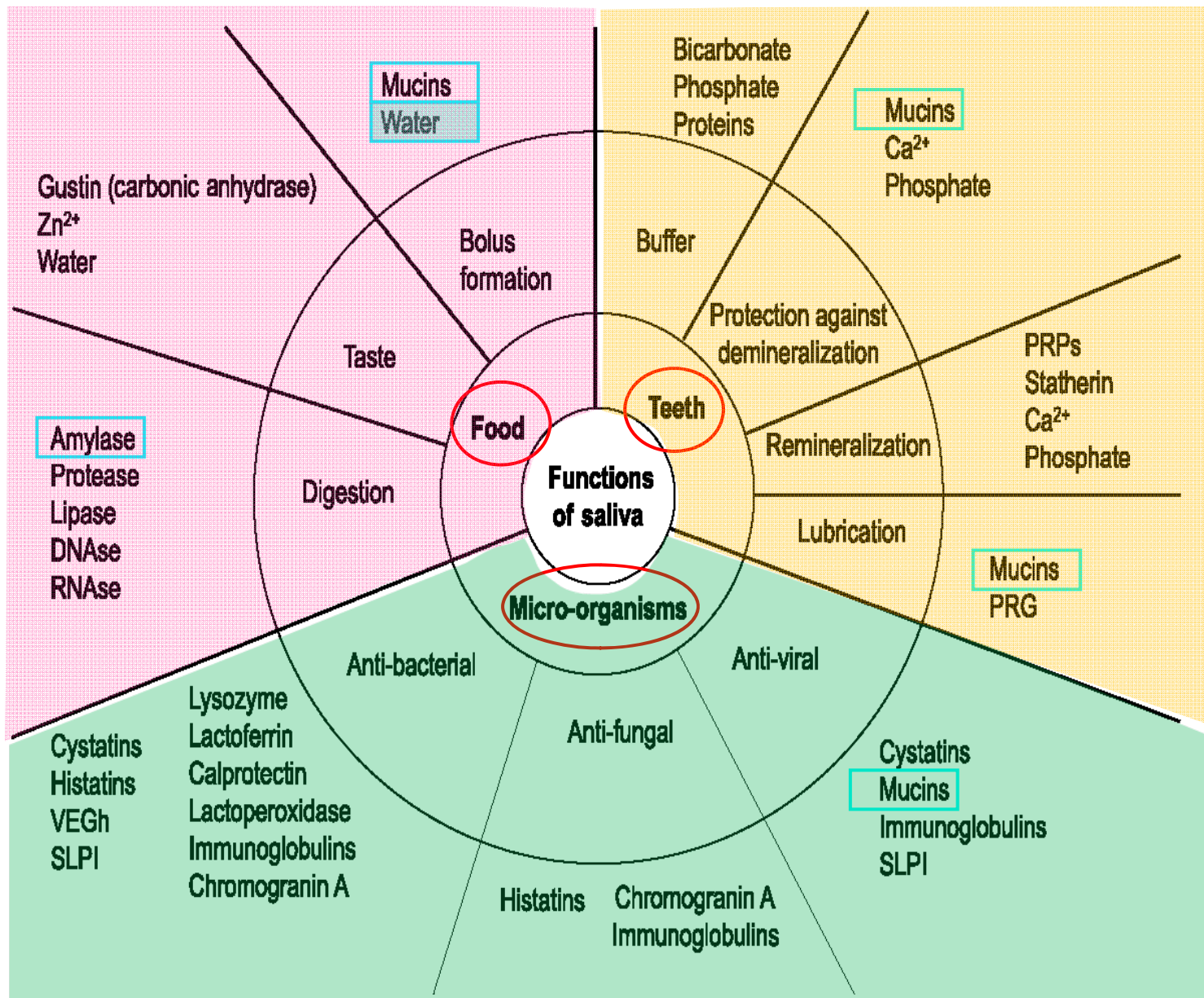
Production of saliva:

basal, continuous : 0.39 ± 0.2 ml/min (~ 600 ml/d)

upon gustatory stimulation : 4.3 ± 2.1 ml/min

composition > 99% water





Pain ↻ meals

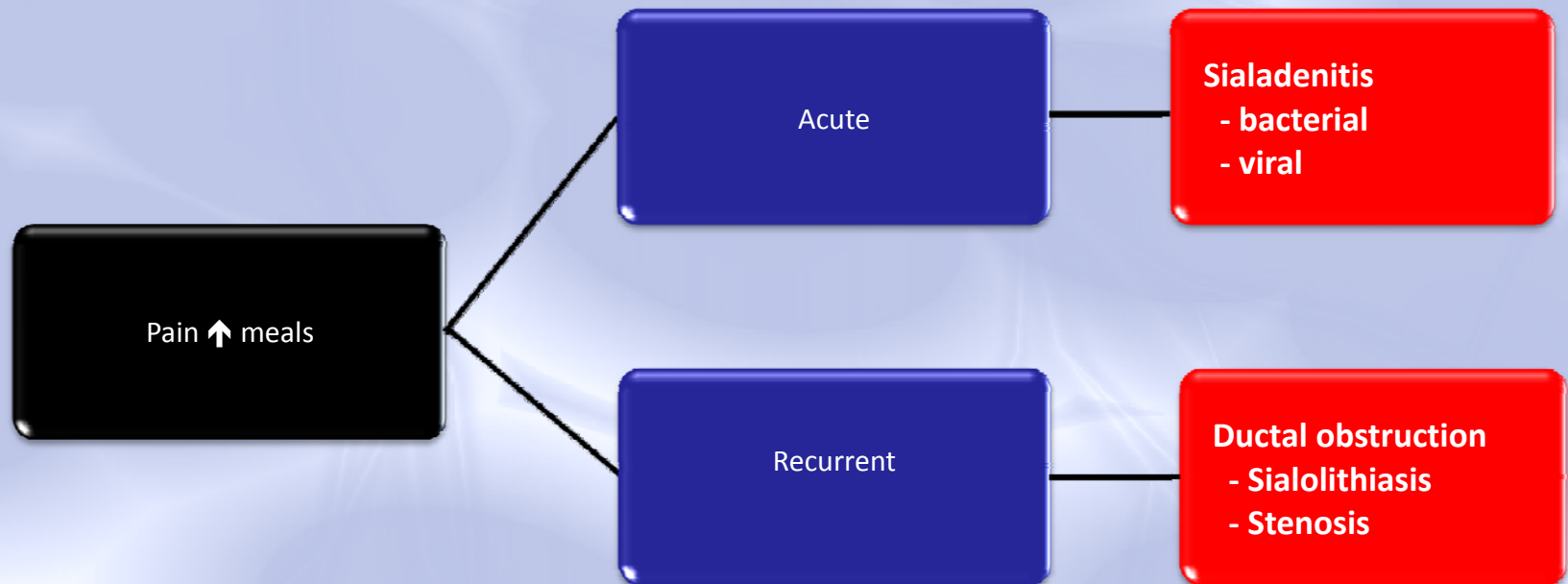
Swelling

Xerostomia

Facial paralysis

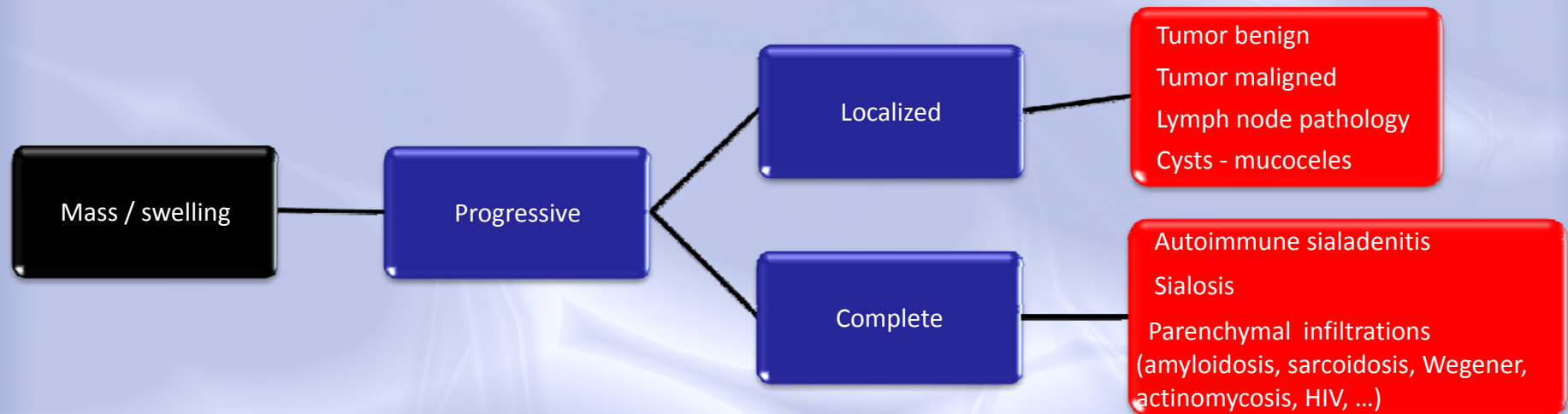
Pain ↑ meals

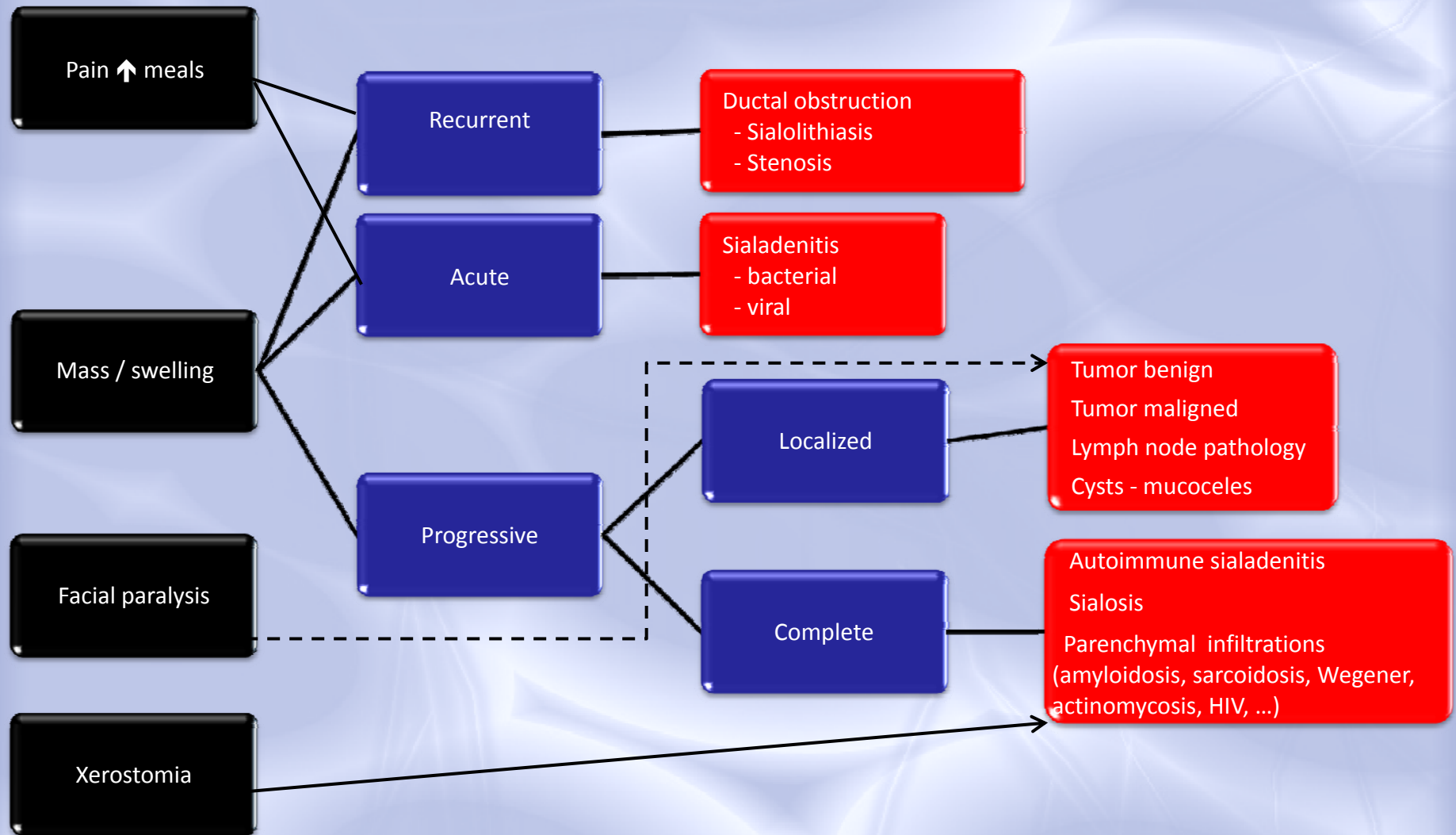
- acute: infection, sialolithiasis
- recurrent: sialolithiasis
- chronic: inflammation



Swelling

- localized: mass, tumor
- diffuse 1 gland: infection, sialolithiasis
- diffuse >1 gland: inflammation









Viral

Mumps (oreillons)

Bacterial acute

Chronic (specific)

tuberculosis

actinomycosis

CAVE : Lymph nodes present within the parotid space (absent from the submandibular gland)

→ Any form of cervical pathology could be found in the parotid

Etiology : Paramyxovirus (human parainfluenza virus types 1&3)

transmission by air (high contagion; by 15 years > 90% antibodies)

incubation 2-3 weeks

viremia: 2-3 days

Clinical presentation:

Age: 5-10 years

Prodromal signs: headaches, myalgia, anorexia

Symptoms: parotid pain increasing during meals, trismus

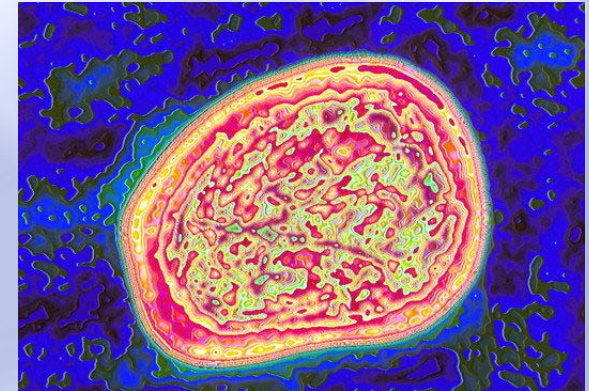
Signs:

parotid swelling

no erythema

75% bilateral parotid involvement (often sequential)

no or light fever



Diagnosis:

- clinical

- serology (IgM – ELISA)

 - Antibody "S" (NP protein): 2 weeks to 9 months

 - Antibody "V" (HN protein): few weeks to 5 years

"Complications":

- orchitis (25% post puberty)

- oophoritis (5% post puberty)

- aseptic meningitis (10%)

- temporary high frequency hearing loss (4%)

- rare permanent unilateral deafness (1/20'000)

Treatment:

- vaccination (since 1967)

- symptomatic

Physiopathology:

Deshydration

Sialolithiasis



Salivary stasis
Retrograde migration
of oral bacteria

Glands : Parotids >> Submandibular

no continuous secretion

few anti-infectious substances in parotid saliva

few muscular contractions to advance the saliva in the duct

despite more sialolithiasis in Wharton's duct

Deshydration (newborns, geriatric population, post-surgery,
xerostomia post-radiation, drugs, anorexia/boulimia, Sjögren)

Bacteriology:

Staphylococcus aureus (50-90%)

Other aerobes (rare)

Probable role of anaerobes (50%)

Aerobic and facultative bacteria

<i>Streptococcus pneumoniae</i>	3
<i>Streptococcus pyogenes</i>	2
<i>Staphylococcus aureus</i>	10 (10)*
<i>Haemophilus influenzae</i>	4 (2)
<i>Escherichia coli</i>	2 (2)
Alpha-hemolytic streptococcus	4
Subtotal	25 (14)

Anaerobic bacteria

<i>Peptostreptococcus</i> ssp.	5
<i>Peptostreptococcus magnus</i>	1
<i>Peptostreptococcus intermedius</i>	1
<i>peptostreptococcus anaerobius</i>	1
<i>Actinomyces israelii</i>	2
<i>Propionibacterium acnes</i>	4
<i>Eubacterium lentum</i>	2
<i>Fusobacterium</i> ssp.	1
<i>Fusobacterium nucleatum</i>	3
<i>Bacteroides</i> ssp.	1
<i>Bacterioides fragilis</i>	1 (1)
<i>Prevotella melaninogenica</i>	1
<i>Prevotella intermedia</i>	4 (2)
<i>Porphyromonas assacharolytica</i>	2
Subtotal	30 (3)
Total	55 (17)

*Number in parenthesis is the number of beta-lactamase-producing bacteria. (From Brook [20]. Reproduced with permission.)

Symptoms:

- acute onset (hours to few days)
- pain increased during meals
- diffuse parotid swelling (90% unilateral)

Physical exam:

- diffuse parotid swelling
- hard and painful gland
- erythema and pus of Stensen's duct orifice
- rare fluctuation (abscess)
- palpation for sialolithiasis



Diagnosis:

clinical

leucocytosis with left shift

culture of pus (duct, aspiration of abscess)

standard x-ray or ultrasound to rule out stone

NO SIALOGRAPHY

US / CT for abscess

Treatment:

rehydration

increase salivary secretion (stop medications, lemon juice, massages)

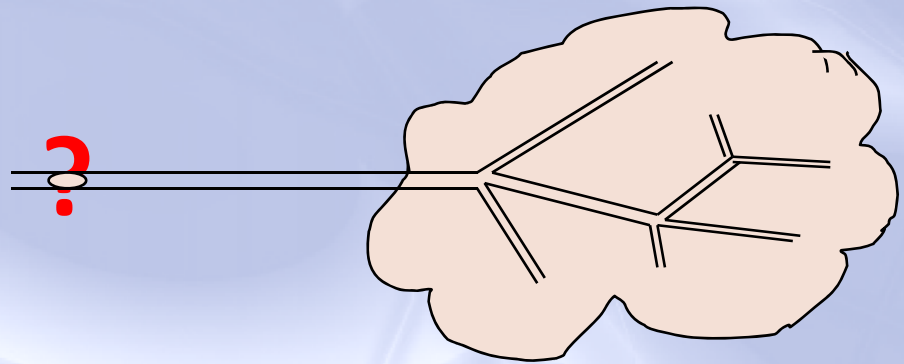
NSAID + analgesics

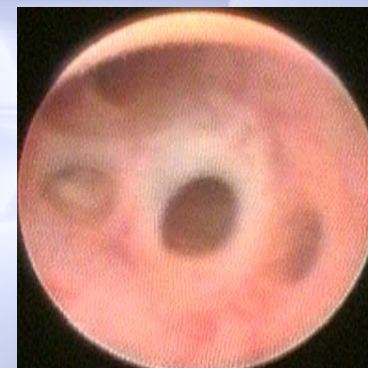
oral antibiotics: ampicilline-clavulinate, clindamycine

if no improvement after 48 h: iv antibiotics + Gram – coverage; MRSA

drainage of abscess



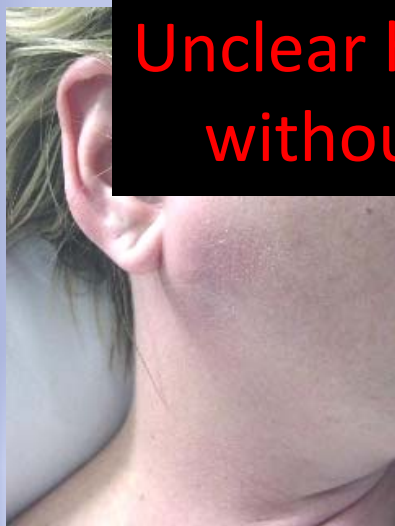




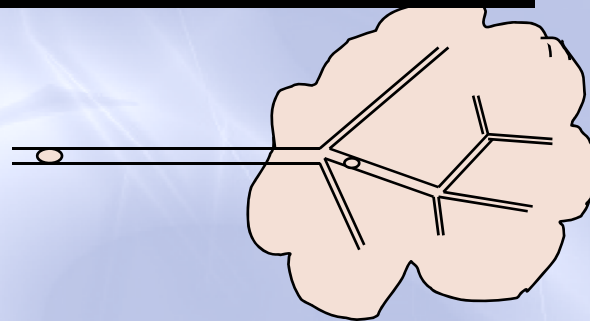
Other ductal obstructions:
- ductal stenosis

Impossible to distinguish sialolithiasis & other ductal obstructions without ductal system evaluation / sialendoscopy

Unclear how often infectious sialadenitis exists without sialolithiasis / ductal obstruction



Sialadenitis



Incidence : 200 / 1'000'000

Age : 42y

Etiology: ???

no relation with dietary calcium

no relation with kidney or gallbladder stones

Physiopathology:

nidus (bacteria, micro-calcifications, cellular debris, foreign body)

growth

hypersaturation with calcium / phosphorus

decrease of crystallization inhibitors

interaction crystal – canal cells

decrease in salivary flow

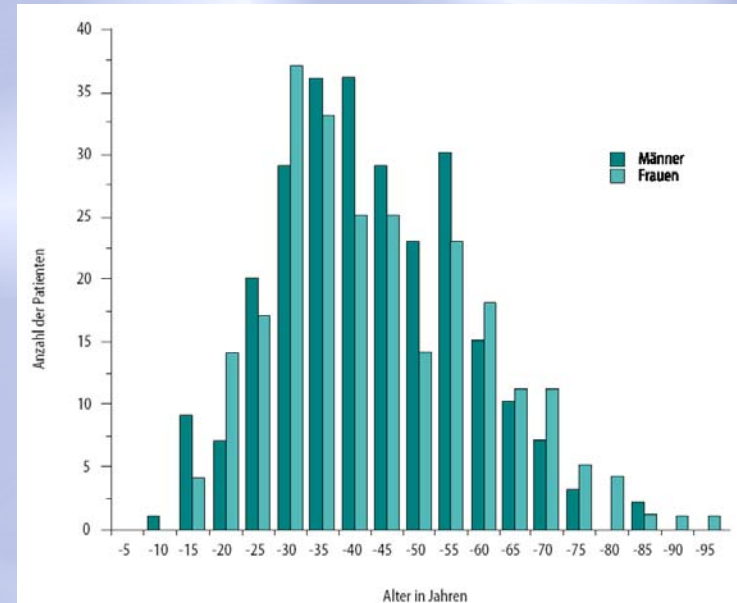
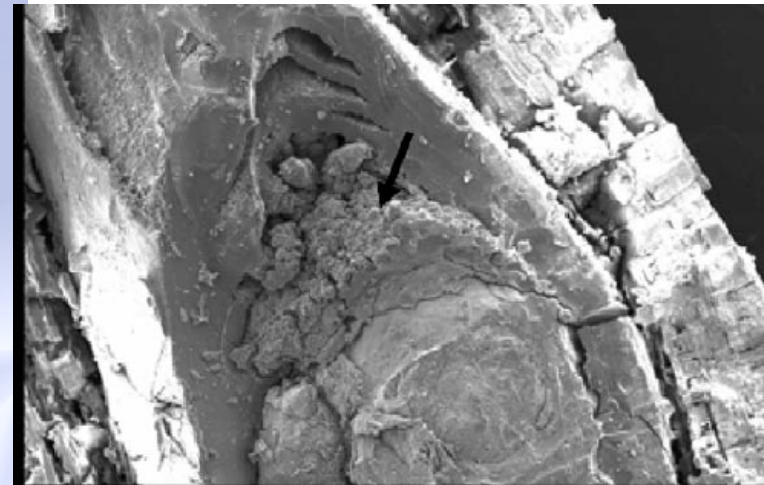


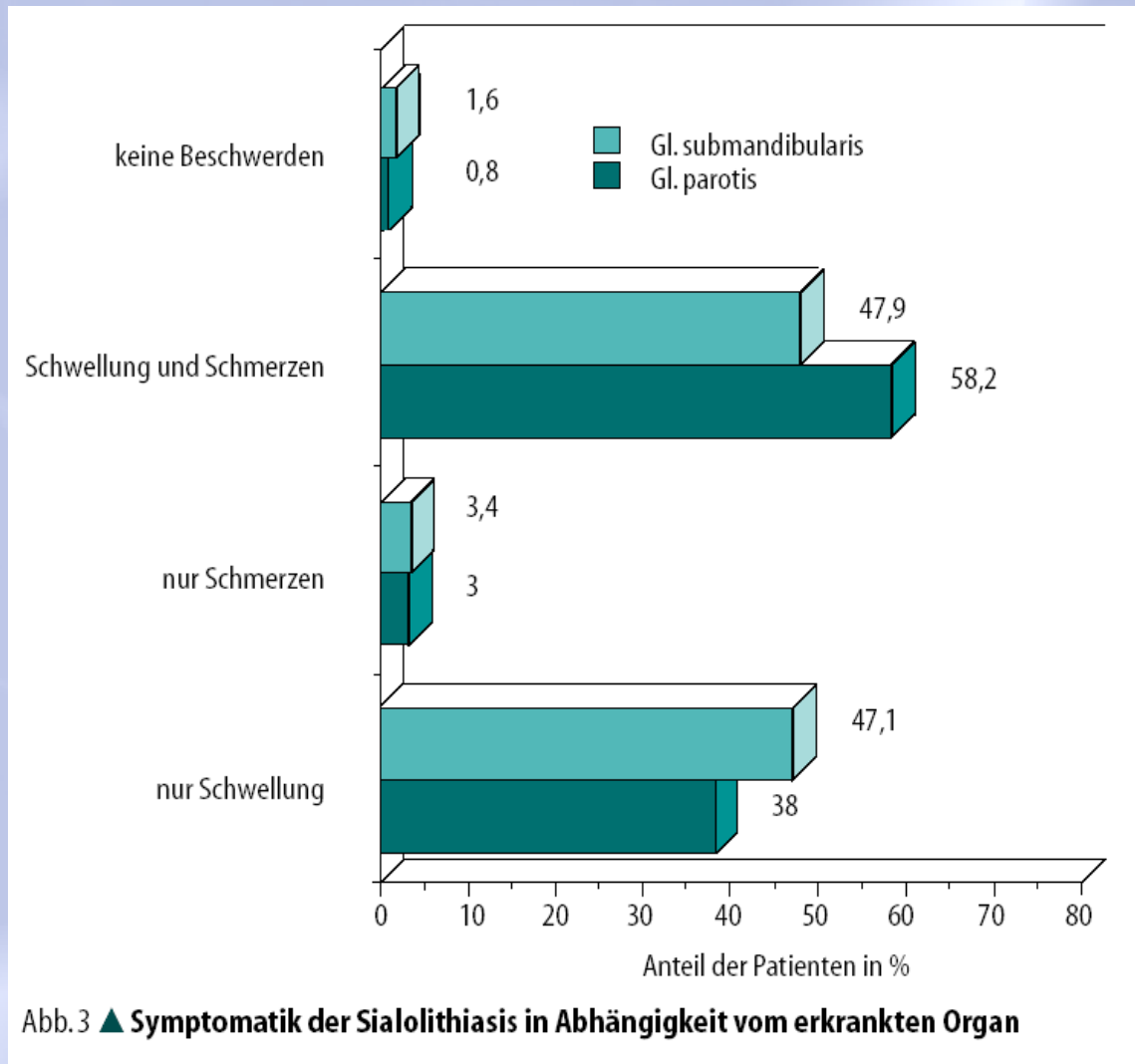
Abb. 1 ▲ Auftreten der Sialolithiasis in der Glandula submandibularis in Abhängigkeit von Geschlecht und Alter



Hydroxyapatite

2mm

Sialolithiasis present with pain and swelling



Acute phase : antibiotics, NSAID, corticosteroids

Definitive treatment – classic :

- spontaneous passage

- anterior stones: sialodochoplastie (Wharton)

- posterior stones: open surgery

Definitive treatment – 1990's:

- external lithotripsy

 - numerous sessions, pain

 - fragments left in the duct

 - limited by the size of the stone

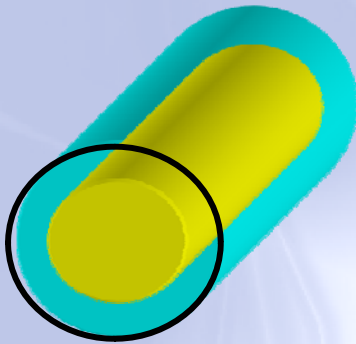
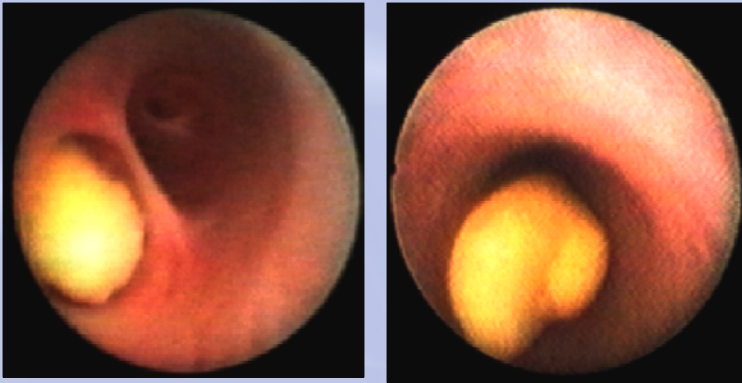
 - success: 75% (parotid) et 40% (submandibular)

- internal lithotripsy

- external lithotripsy with endoscopic retrieval

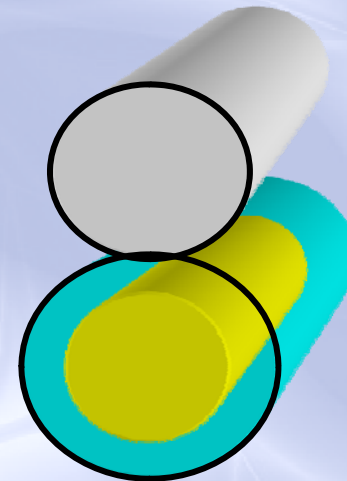
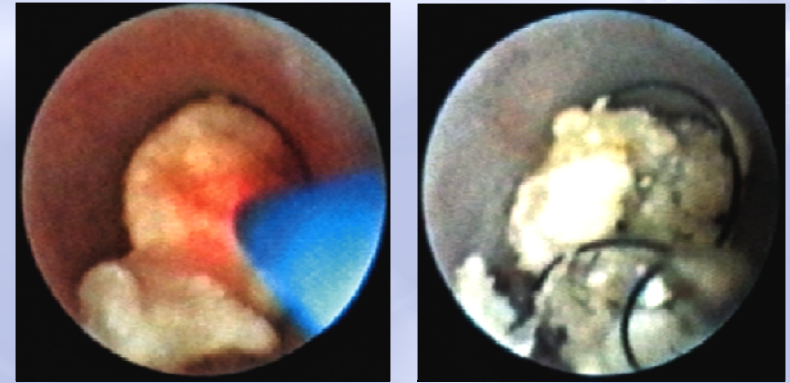
SG - Sialendoscopy

Diagnostic sialendoscopy



One channel endoscope for vision
Rinsing

Interventional sialendoscopy



Two channels: vision + work
Rinsing



SG - Diagnostic sialendoscopy



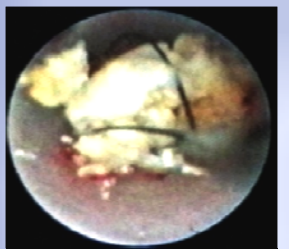
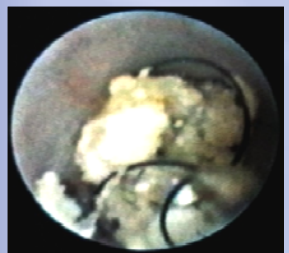
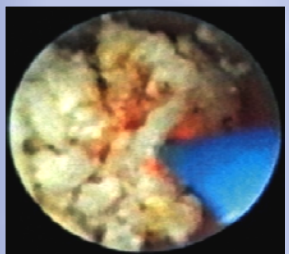
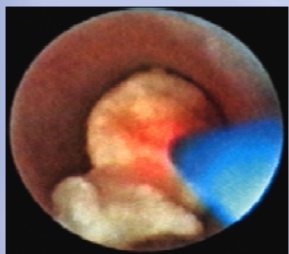
Diagnostic sialendoscopy possible in 98% of cases

Diagnosis	Sialolithiasis	58%
	Stenosis	11%
	Sialodochitis	37%
	Normal	32%
Duration	22 min (100 last cases: 11 min)	
Pain	2.2 ± 1.4 (scale 0-10)	
Limitations	Fibrosis, angulations, mouth opening and prominent teeth	
Complications	0	

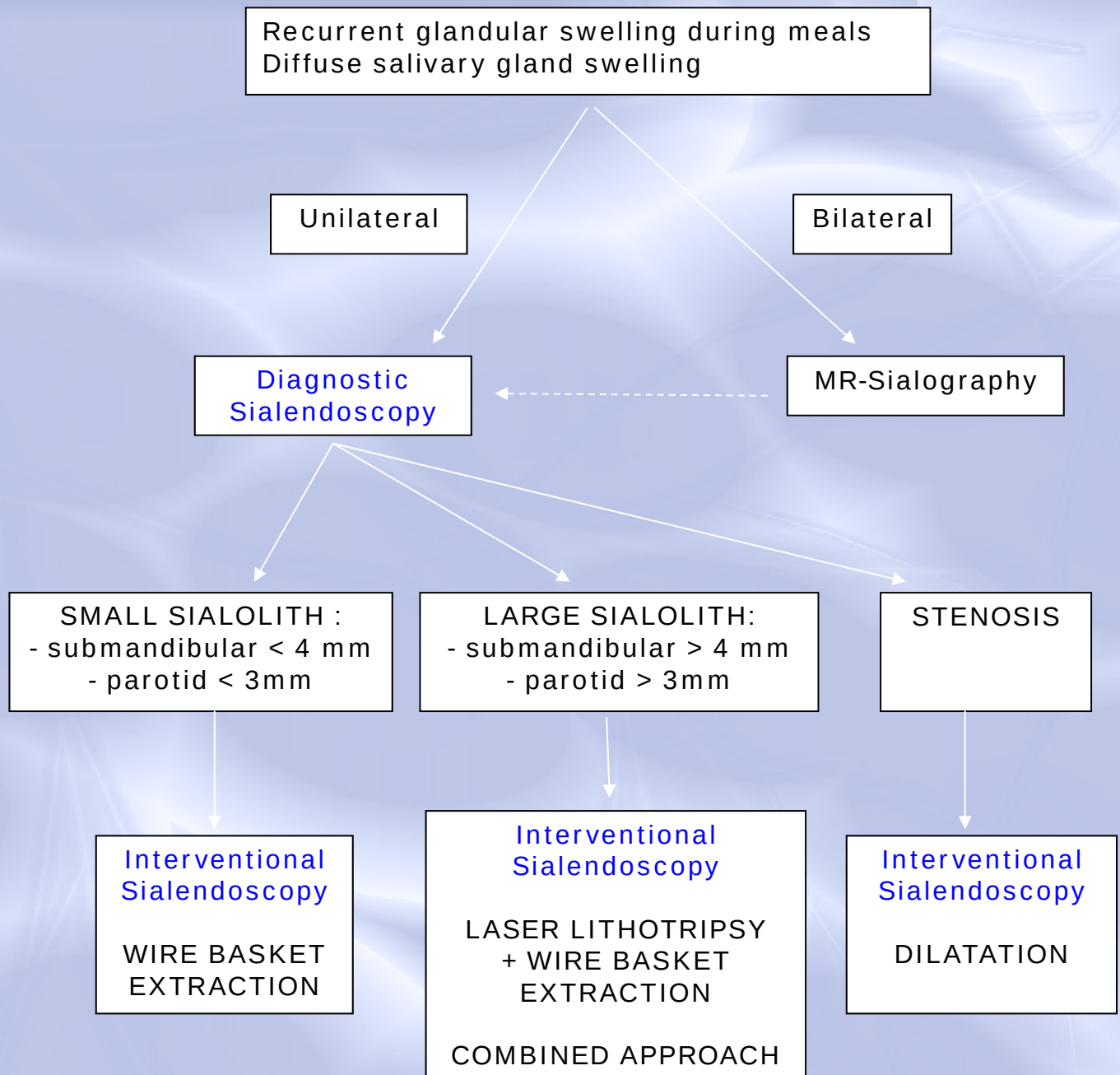




SG - Interventional sialendoscopy



Success	356 (79%)	Stones removed (1 session)	235
		Stones removed (>1 sessions)	121
		Stenosis dilated	38
Failures	94 (21%)	Incomplete fragmentation / extraction	56
		Embedded stones	11
		Recurrent stenosis	7
		Submandibular gland resection	19
		Parotidectomies	1
Complications	43 (10%)	Perforations	18
		Blocked wires	25
		Broken wire baskets	3
		Bleeding	0
		Nerve damage	0
Duration	71 ± 41 minutes		



True positive	34
True negative	36
False positive	2
False negative	10
Sensitivity	34/44 (77%) CI [63 - 87]
Specificity	36/38 (95%) CI [82 - 99]
Positive predictive value	34/36 (94%) CI [81 - 99]
Negative predictive value	36/46 (78%) CI [64 - 87]
Accuracy	70/82 (85%) CI [76 - 92]

Salivary glands - ductal pathologies

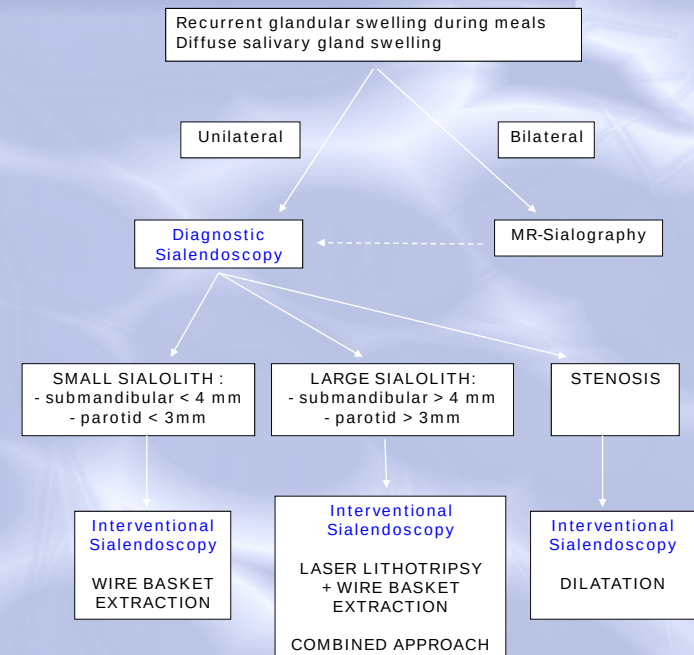
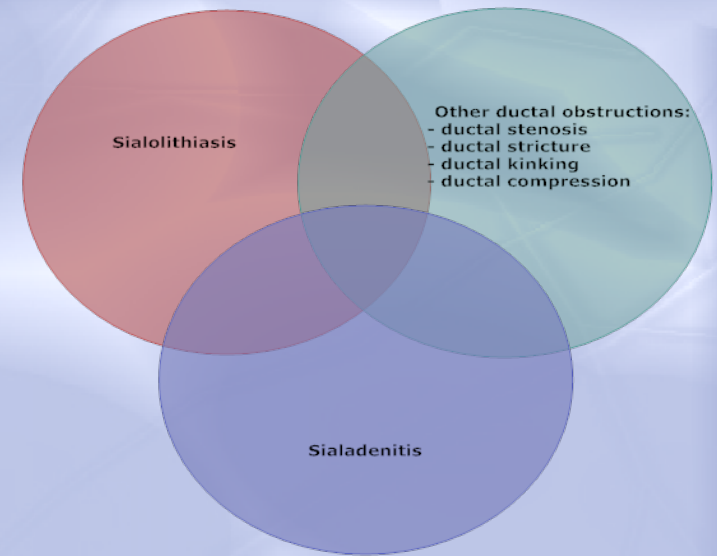
Impossible to distinguish sialolithiasis & other ductal obstructions without ductal system evaluation / sialendoscopy

Ductal pathologies & sialolithiasis present with pain and diffuse gland swelling

Diagnosis is clinical / diagnostic sialendoscopy

Management:

- Acute phase : antibiotics, NSAID, corticosteroids
- Submandibular anterior stone: sialodochotomy
- Else: sialendoscopy



Salivary glands - outline

Physiology

Semiology

Infections

Sialolithiasis

sialendoscopy

.....

Inflammations

Tumors

benign

pleomorphic adenoma

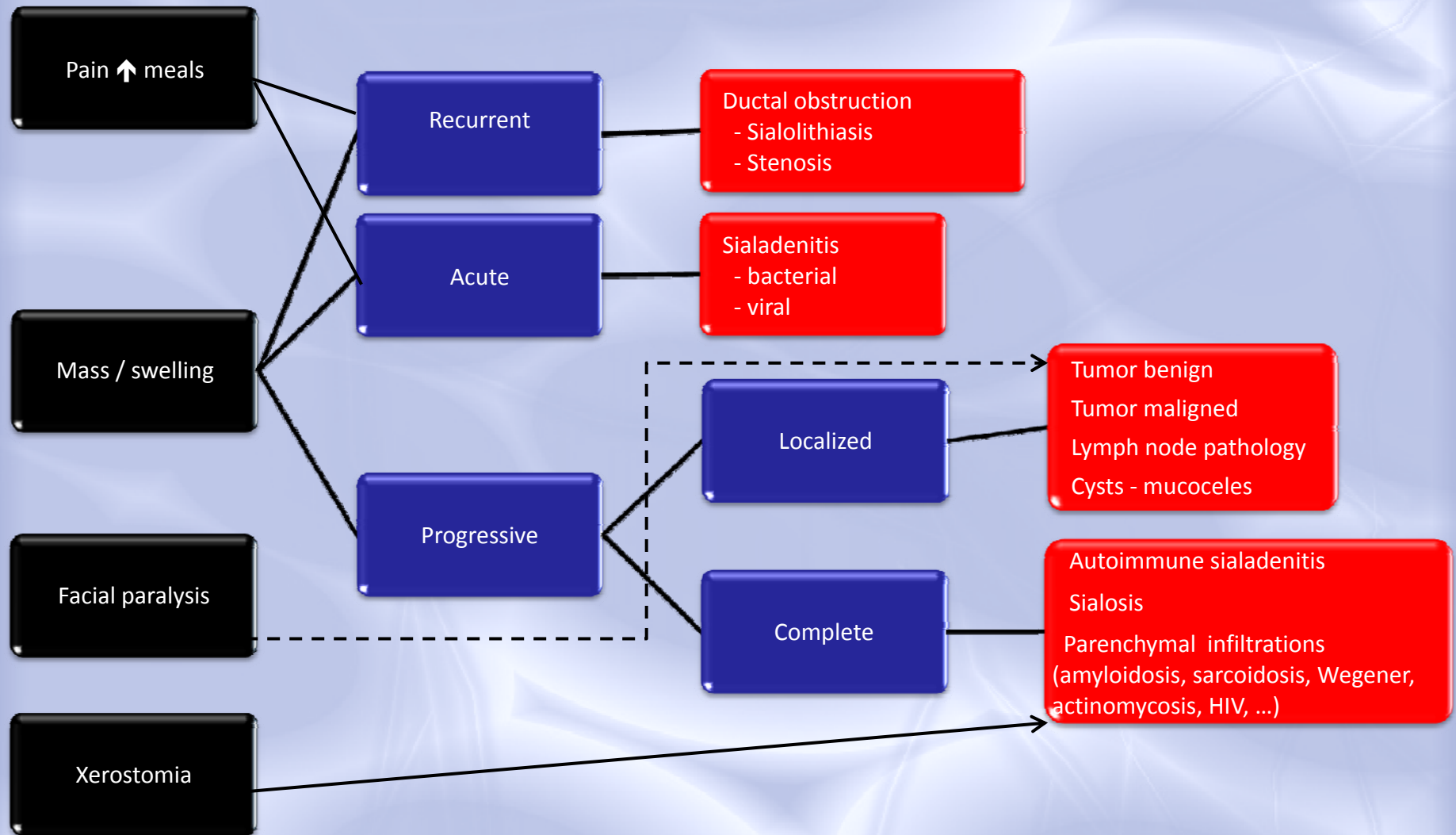
cancers

Surgery

.....

parotidectomy

Anatomy



Syndrome de Sjögren

Sarcoïdose

Syndrome Melkersson-Rosenthal

Parotides récidivantes de l'enfant

Sialadénite post-radique

Kystes

- Mucocèles

- Kyste lympho-épithélial parotidien

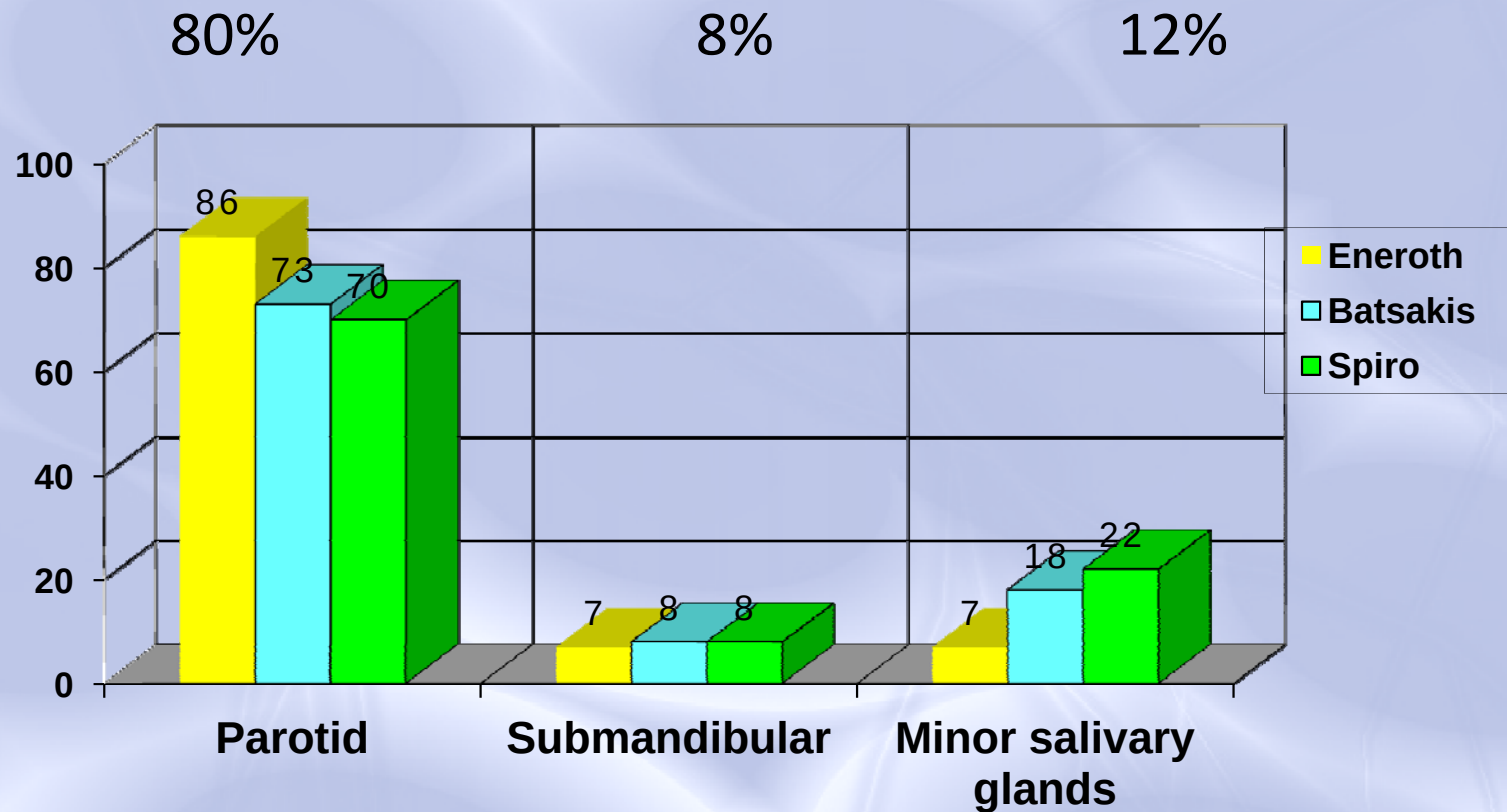
- Pseudokystes parotidiens de l'affection VIH

Sialo(adéno)se

Sialométaplasie nécrosante

Most salivary gland tumors are in the parotid

Salivary glands tumors: ~ 5/100'000



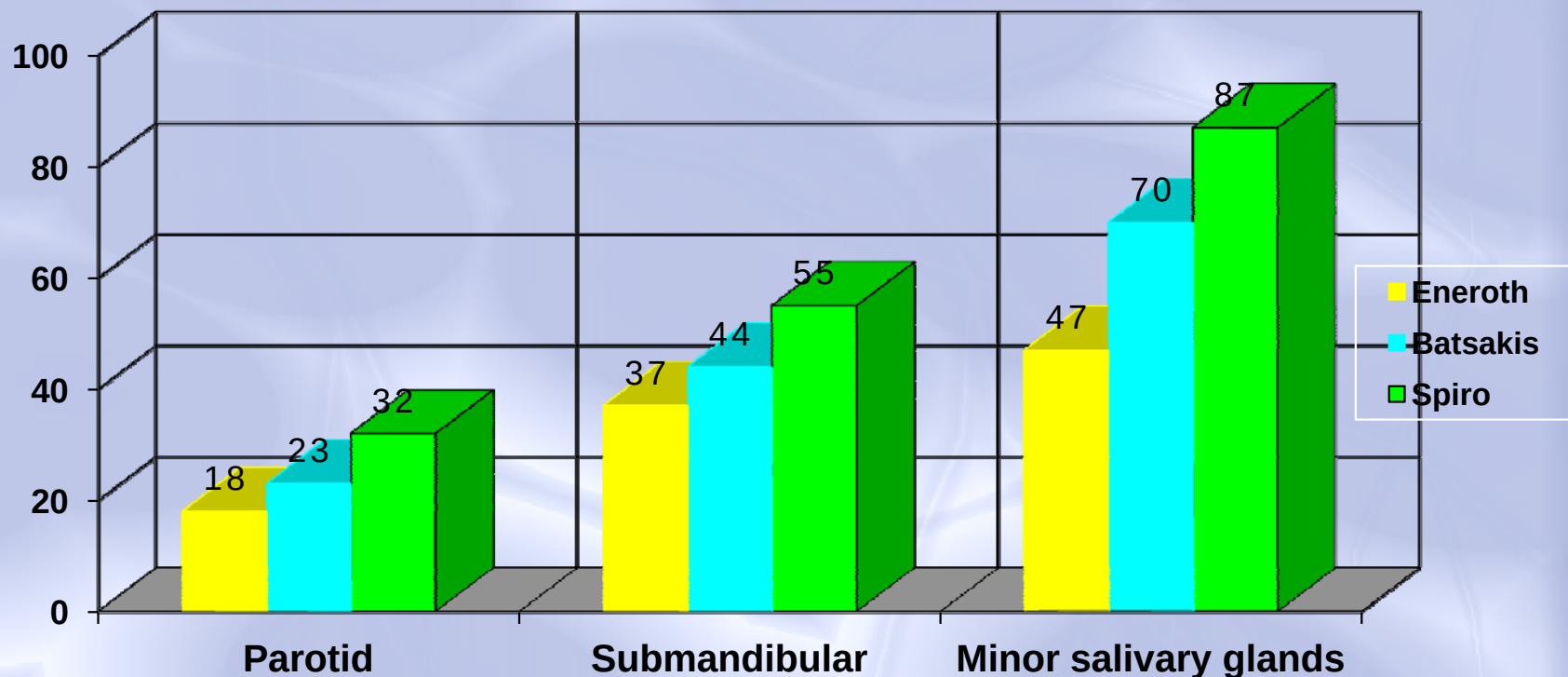
The incidence of carcinoma varies with the gland involved

Salivary glands cancers: $\sim 1/100'000$

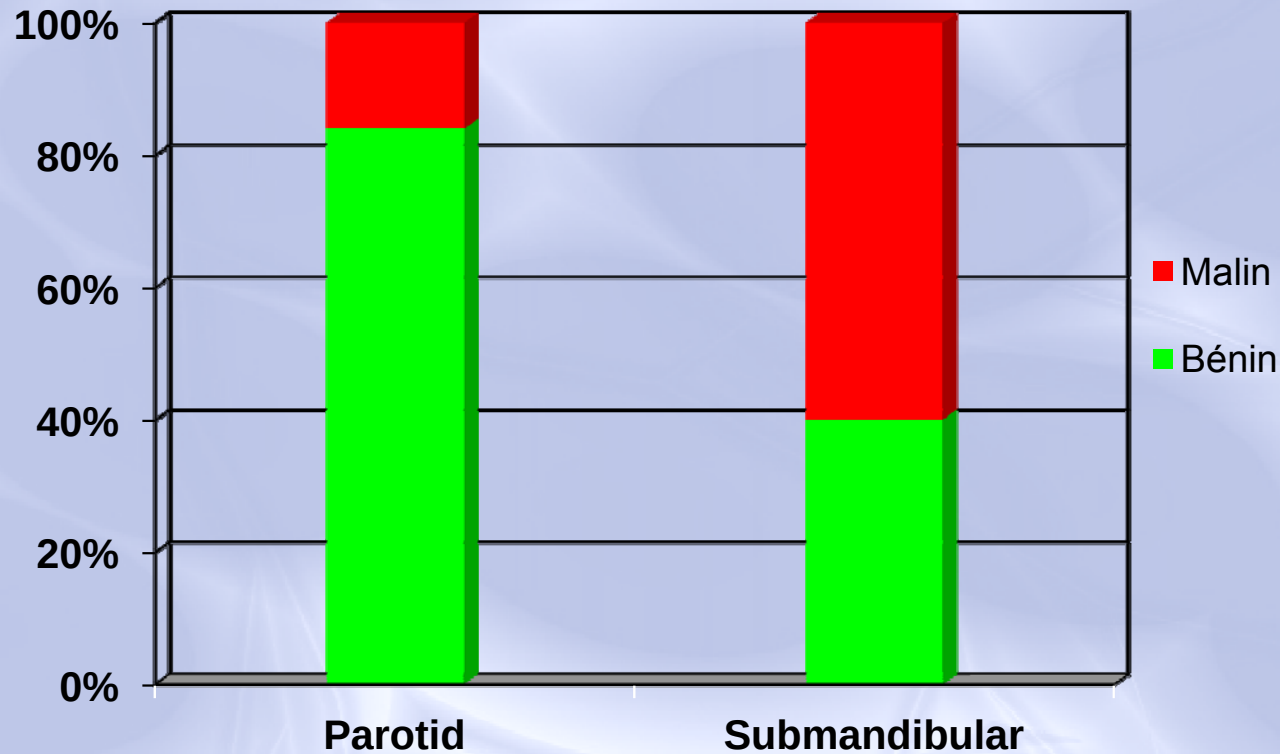
%cancer **20%**

50%

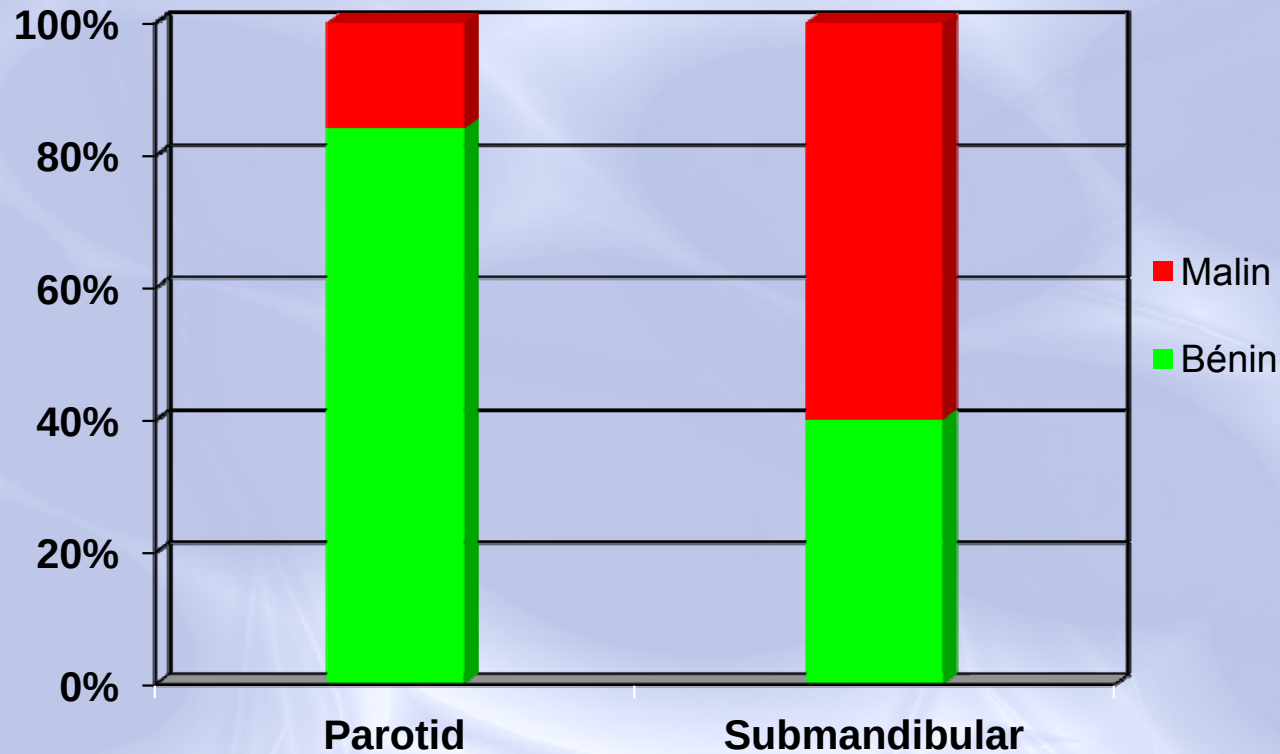
75%



Most parotid tumors are benign



Most parotid tumors are benign



SG tumors histology

Malignant epithelial tumours

Acinic cell carcinoma	8550/3
Mucoepidermoid carcinoma	8430/3
Adenoid cystic carcinoma	8200/3
Polymorphous low-grade adenocarcinoma	8525/3
Epithelial-myoepithelial carcinoma	8562/3
Clear cell carcinoma, not otherwise specified	8310/3
Basal cell adenocarcinoma	8147/3
Sebaceous carcinoma	8410/3
Sebaceous lymphadenocarcinoma	8410/3
Cystadenocarcinoma	8440/3
Low-grade cribriform cystadenocarcinoma	
Mucinous adenocarcinoma	8480/3
Oncocytic carcinoma	8290/3
Salivary duct carcinoma	8500/3
Adenocarcinoma, not otherwise specified	8140/3
Myoepithelial carcinoma	8982/3
Carcinoma ex pleomorphic adenoma	8941/3
Carcinosarcoma	8980/3
Metastasizing pleomorphic adenoma	8940/1
Squamous cell carcinoma	8070/3
Small cell carcinoma	8041/3
Large cell carcinoma	8012/3
Lymphoepithelial carcinoma	8082/3
Sialoblastoma	8974/1

Benign epithelial tumours

Pleomorphic adenoma	8940/0
Myoepithelioma	8982/0

Basal cell adenoma	8147/0
Warthin tumour	8561/0
Oncocytoma	8290/0
Canalicular adenoma	8149/0
Sebaceous adenoma	8410/0
Lymphadenoma	
Sebaceous	8410/0
Non-sebaceous	8410/0
Ductal papillomas	
Inverted ductal papilloma	8503/0
Intraductal papilloma	8503/0
Sialadenoma papilliferum	8406/0
Cystadenoma	8440/0

Soft tissue tumours

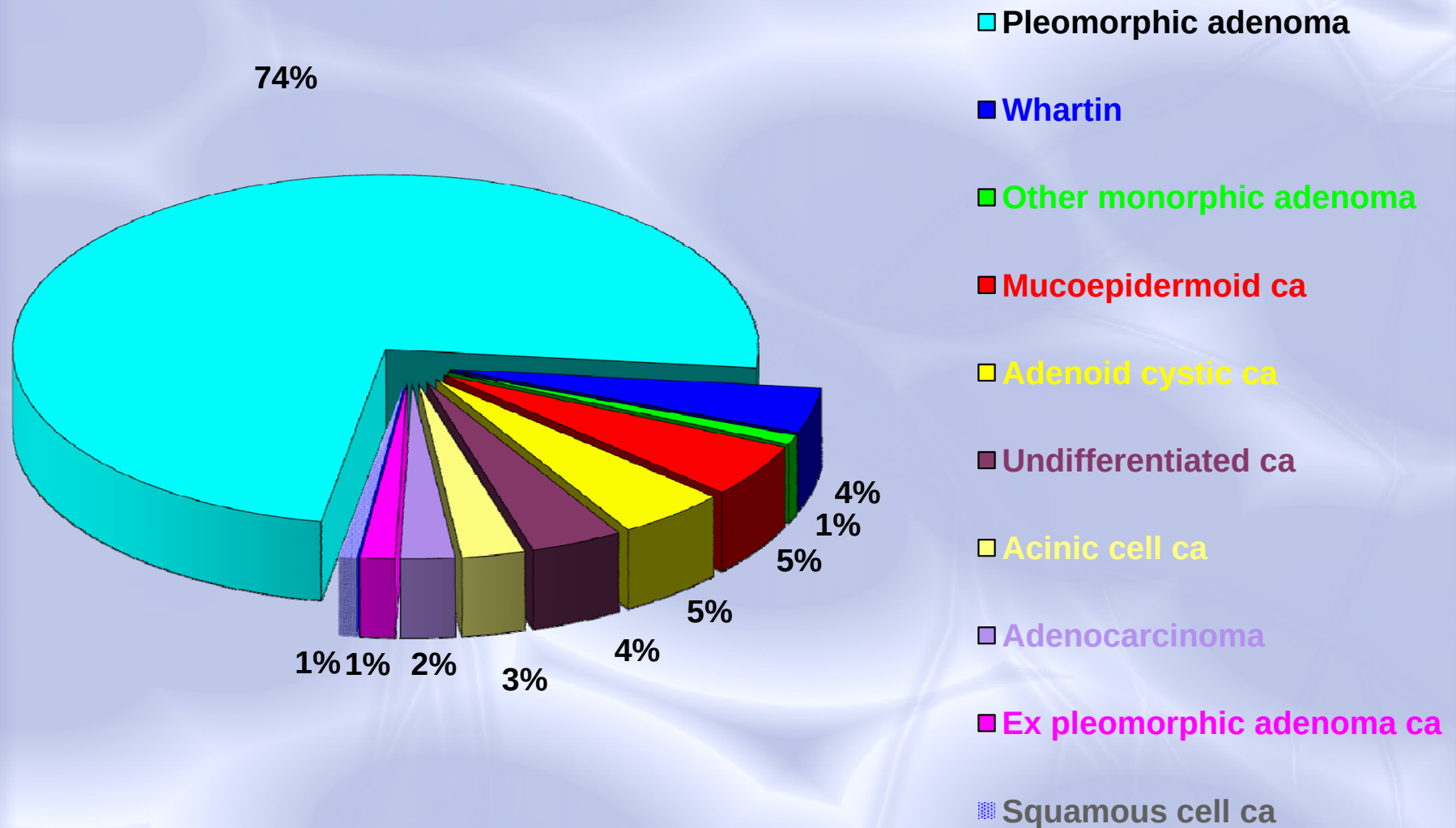
Haemangioma	9120/0
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Haematolymphoid tumours

Hodgkin lymphoma	
Diffuse large B-cell lymphoma	9680/3
Extranodal marginal zone B-cell lymphoma	9699/3

Secondary tumours

Pleomorphic adenoma is the most frequent SG tumor



The etiology of most salivary gland tumors is unknown

No relation to smoking, infections ...

Irradiation :

Japan – atomic bomb

Irradiation for benign pathologies in childhood; dental X-rays; I¹³¹

Delay : 15 year

Carcinoma (RR=11): muco-epidermoid, squamous cell carcinoma,

Benign tumors (RR=3): Whartin, pleomorphic adenoma

Pleomorphic adenoma >>> cancer

Smoking >>> Whartin tumor

Age : 55-60 years (4 to 98 years)

M/F ratio : 1 / 0.5

Semiology : isolated asymptomatic mass

Signs of malignancy:

- fixation: 2-13%

- trismus: 4%

- facial paralysis: 12-24%

- neck mass: 6-20%

No prognostic role: rapid growth, pain

Comparison of American Studies With Non-American Studies for Malignant vs Benign Lesions*

	American Studies (n = 9)	Non-American Studies (n = 55)
Area under the curve	0.94 (0.91-0.96)	0.97 (0.95-0.98)
Inconsistency, I^2 (%)	<1 (not determined)	97 (95-99)
Log rank Q, df, P	0.05, 2, .49	65.9, 2, .00
Sensitivity	0.83 (0.77-0.88)	0.79 (0.74-0.83)
Specificity	0.93 (0.87-0.93)	0.97 (0.97-0.98)
Positive likelihood ratio	8.8 (6.3-12.3)	33.7 (23.4-48.4)
Negative likelihood ratio	0.19 (0.13-0.26)	0.22 (0.18-0.27)
Disease prevalence	0.43	0.23
Positive predictive value	0.90	0.89
Negative predictive value	0.89	0.94

* Values in parentheses are 95% confidence intervals.

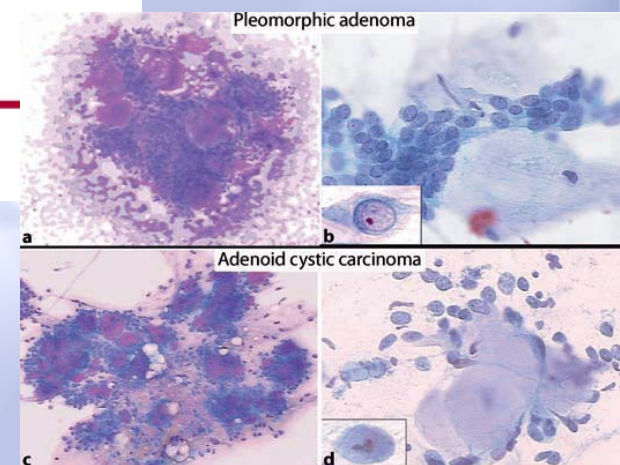


Table 2

Summary of Accuracy Estimates for Frozen Section and Comparison With FNAC

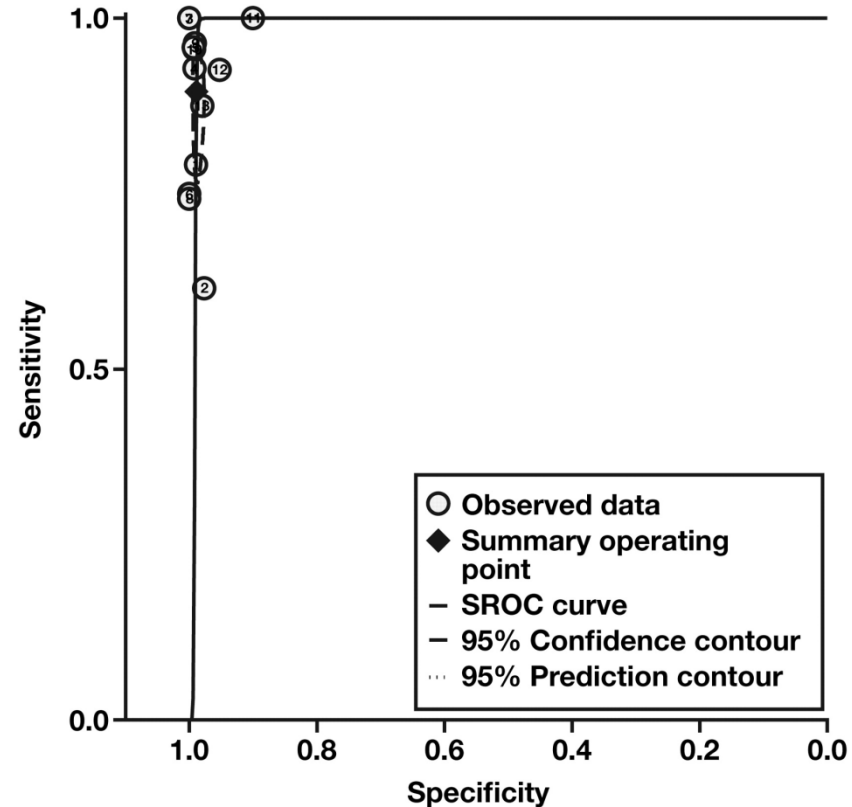
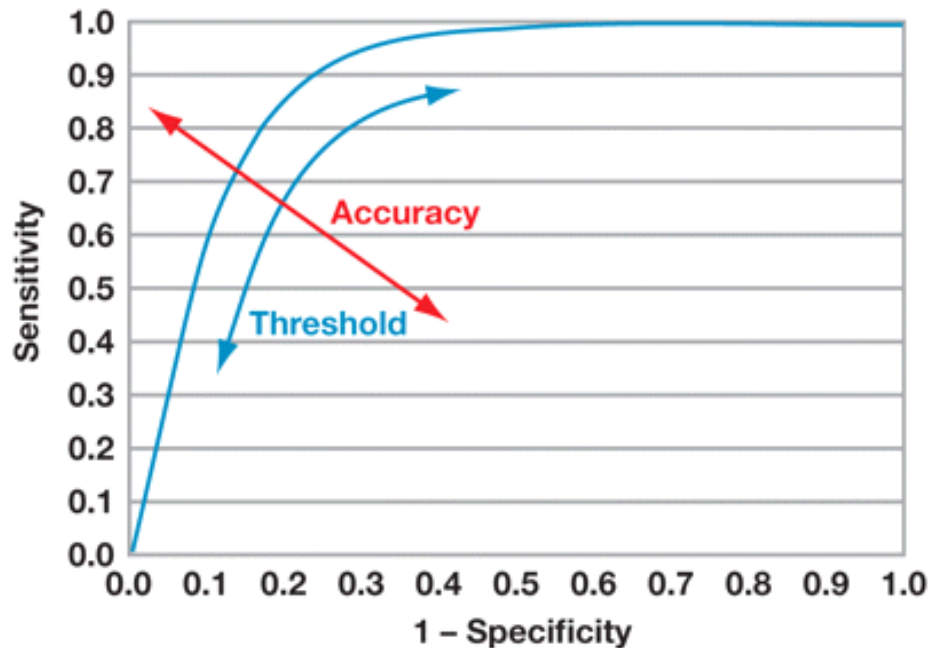
Parameter	Frozen Section		FNAC*	
	Point Estimate	95% CI	Point Estimate	95% CI
Sensitivity	0.90	0.81-0.94	0.80	0.76-0.83
Specificity	0.99	0.98-0.99	0.97	0.96-0.98
Positive likelihood ratio	80.6	47.5-137.0	28.6	20.7-42.0
Negative likelihood ratio	0.11	0.06-0.19	0.21	0.17-0.26
Area under summary receiver operating characteristic curve	0.99	0.98-1.00	0.96	0.94-0.97
Inconsistency, I ²	53	0-100	98	97-99

CI, confidence interval; FNAC, fine-needle aspiration cytology.

* Data from Schmidt et al.¹

Frozen sections

FNA



US can distinguish benign and malignant tumors (in experienced hands)

Sensitivity: 91%

Specificity: 93%

Accuracy: 93%



A comparative table of ultrasound based diagnosis of focal lesions and final histological diagnosis

	Ultrasound-based diagnosis							
	Pleomorphic adenoma	Warthin's tumours	Granulomatous lesion	Reactive nodes	Benign	Indeterminate	Non-lymphomatous malignancies	Lymphoma
Histological diagnosis								
Pleomorphic adenoma (<i>n</i> = 65)	40	10	0	0	6	6	3	0
Other adenomas (<i>n</i> = 4)	1	2	0	0	0	1	0	0
Warthin's tumours (<i>n</i> = 54)	12	31	0	0	1	7	2	1
Granulomatous lesions (<i>n</i> = 6)	0	0	2	0	0	2	2	0
Reactive lymph nodes (<i>n</i> = 6)	0	0	0	3	0	2	1	0
Non-lymphomatous malignancies (<i>n</i> = 29)	1	1	0	0	0	4	23	0
Lymphoma (21)	0	2	0	0	0	3	7	9

MRI is no better than US; CT is worse

Sensitivity: 71% → 86%

Specificity: 86% → 97%

Accuracy: 82% → 92%





NO preoperative open biopsy

FNA: false negatives for malignancy ~ 20%

Frozen sections: false negatives for malignancy ~ 10%

Role of core needle biopsy ?

Imaging:

cannot differentiate benign vs. malignant

useful if:

- 1) the presence of mass is uncertain**
- 2) the location (superficial or deep lobe) is unclear**
- 3) an extension outside the parotid gland is suspected**

EXCEPTIONS:

1. Pathology

Infection / inflammation / systemic disease

Lymphoma

Metastasis

Pediatric hemangioma

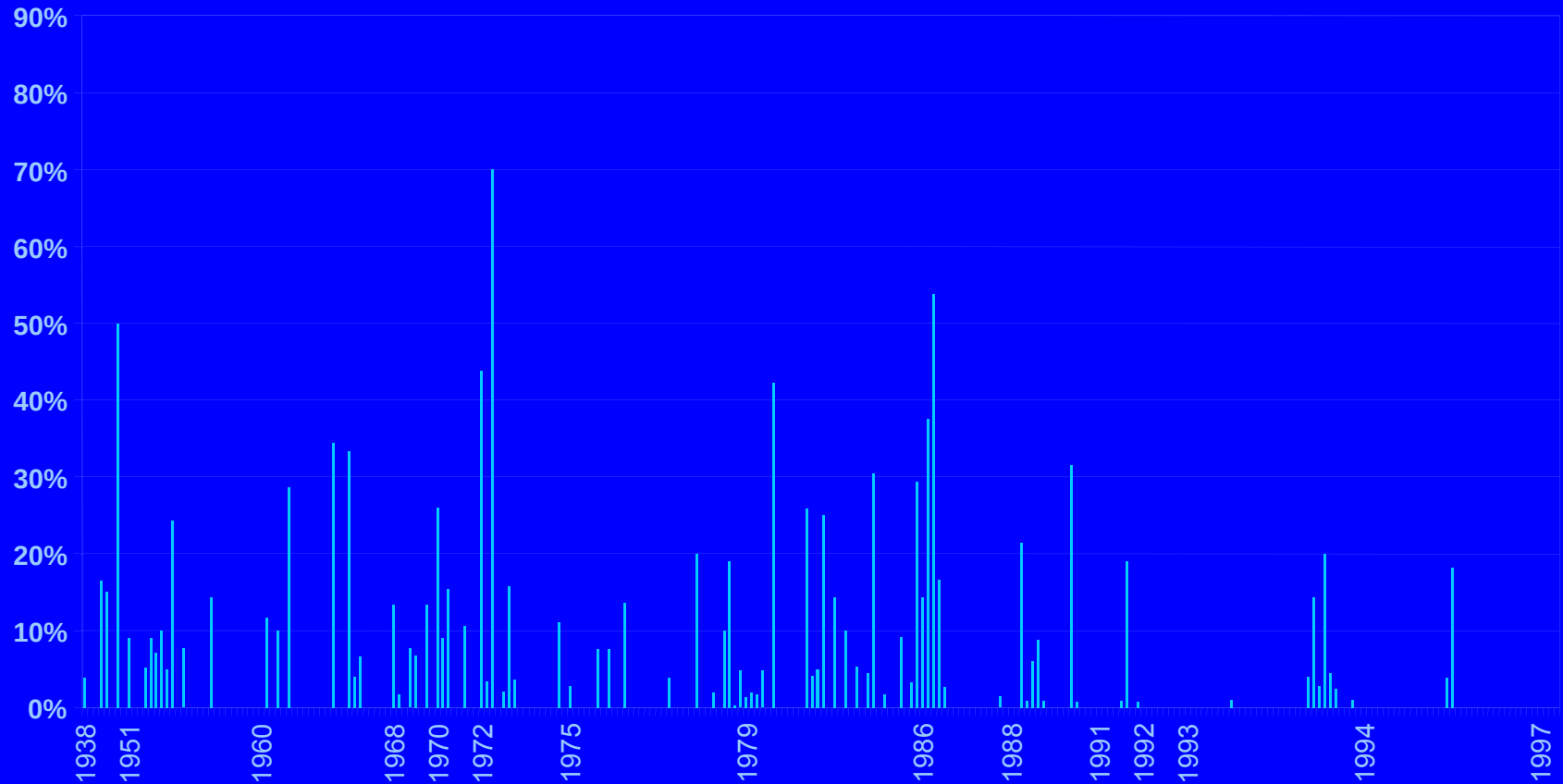
Benign: Whartin tumor in a old patient

2. Patient not fit for surgery



The recurrences of pleomorphic adenoma is highly variable

Incidence of recurrences



Multicentricity of pleomorphic adenoma

- ▶ *Redon J Chir 1945*

Capsule rupture (tumor dissemination in the wound)

- ▶ *Maran et al. Arch Otolaryngol 1984*

Incomplete resection (islands of tumor tissue left behind during surgery)

- ▶ *Maran et al. Arch Otolaryngol 1984*

Pleomorphic adenomas are not multicentric

Mc Farland 1926-1945: 5 papers

- clinical - pathological correlation of tumors

- recurrence of pleomorphic adenoma : 30%

- not operate small tumors because recurrence is more frequent

- if repeat operations, tumors more aggressive

- Malignancy ?

- Role of radiotherapy ?

Delarue (1956): multicentric tumors

- Redon : always total parotidectomy

Patey & Thackray (1958) :

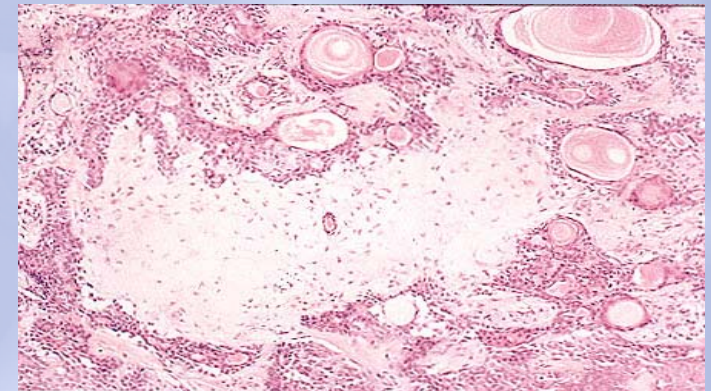
- capsule of variable thickness

- capsule often incomplete

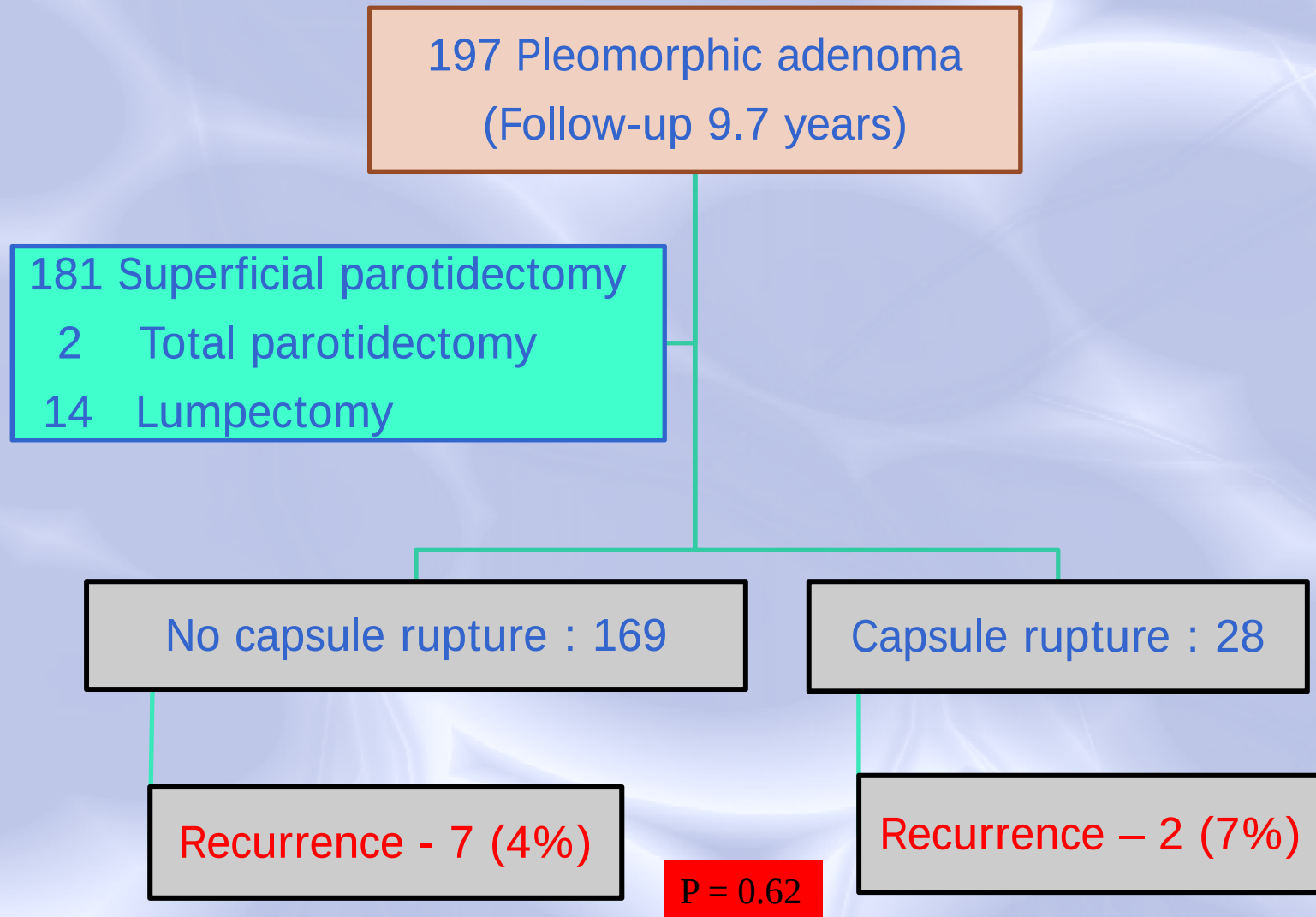
- growth by polypoid extensions

- polypoid extensions more frequent in small tumors

- no multifocal tumor



It is unclear if capsule rupture is associated with increased recurrences of pleomorphic adenoma (I think so)

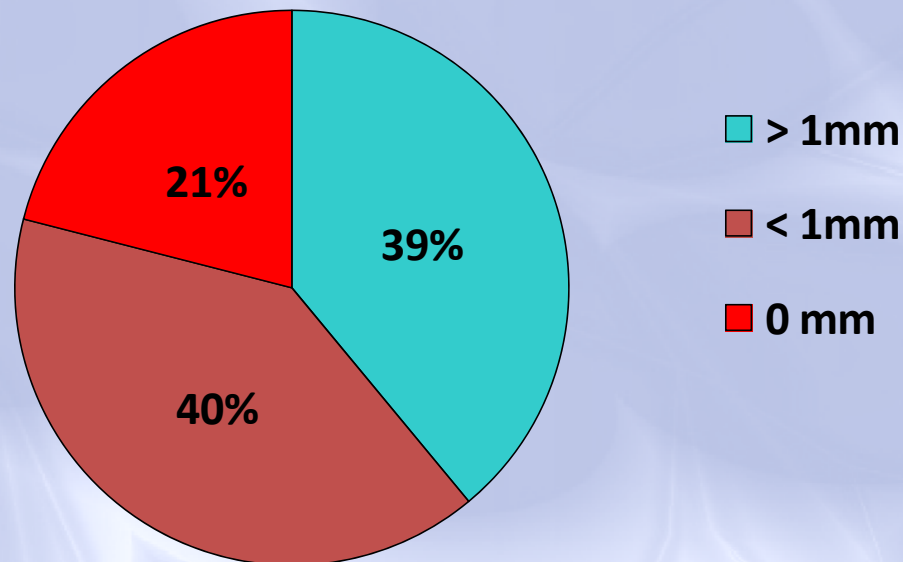


Does seeing the capsule increases the risk of recurrence?

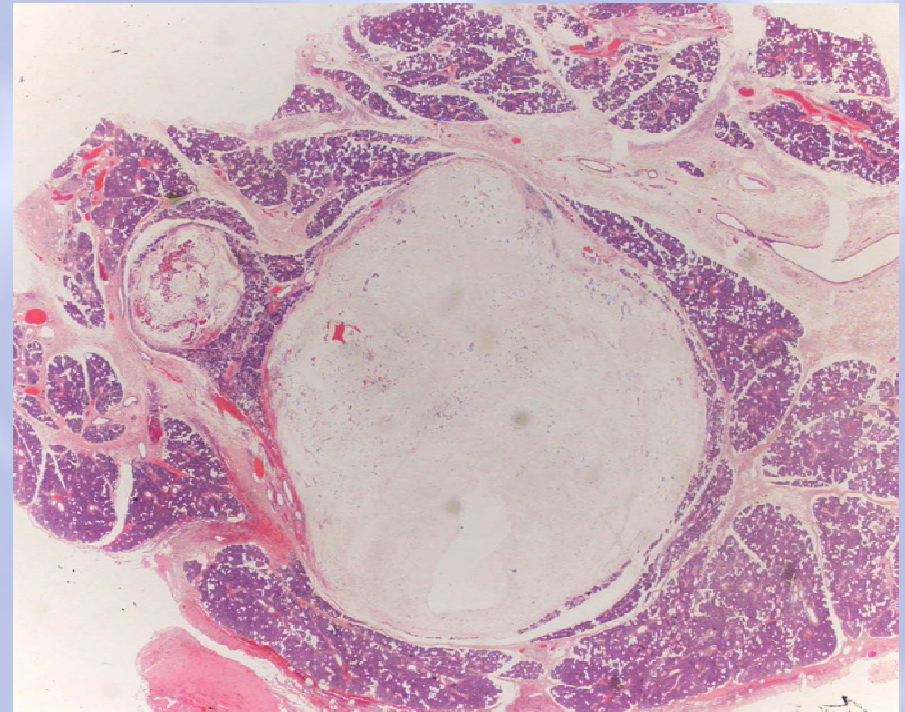
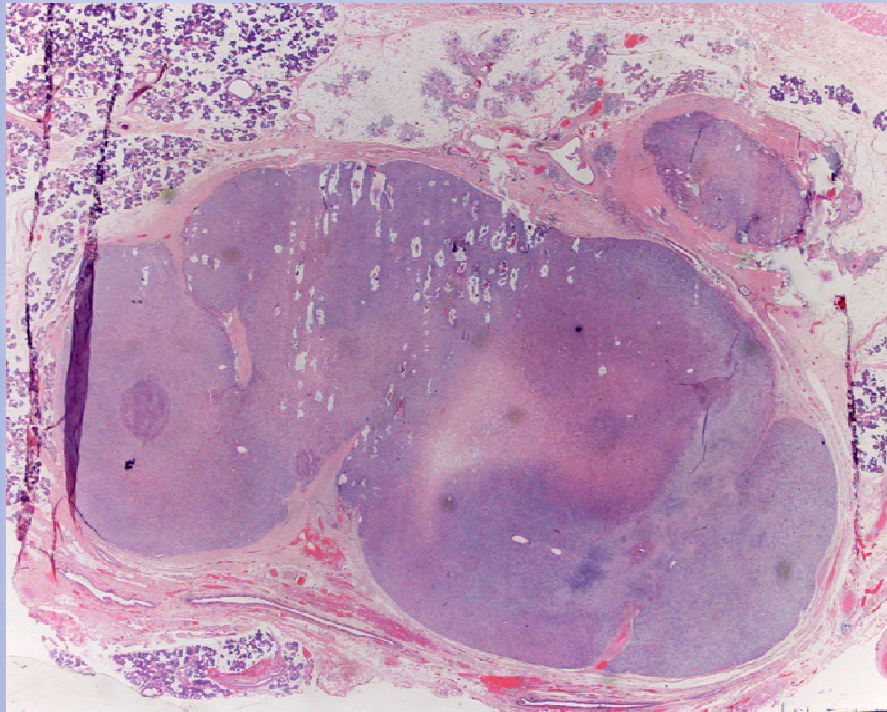
SURGERY	N	RECURRENCES
Capsule ruptured with macroscopic spillage	26	2 (8%)
Part of tumor capsule visualized	26	2 (8%)
No visualization of capsule	126	3 (2.5%)

Obtaining adequate normal tissue margin is often impossible

Danovan & Conley (1984) : distance tumor – margins of resection



Pleomorphic adenoma often exhibit pseudopodia



Author	Reference	Incidence of pseudopodia or satellite nodules
Stennert et al.	<i>Laryngoscope</i> 2001	72%
Paris et al.	<i>Ann Otolaryngol Chir Cervicofac</i> 2004	28%
Zbaeren et al.	<i>Head Neck</i> 2007	48%

The capsule of pleomorphic adenoma is often thin or absent

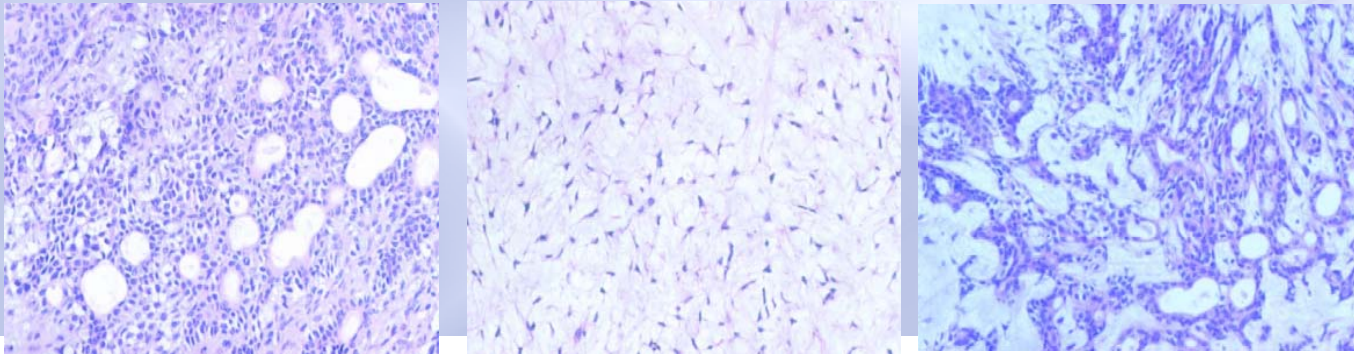


TABLE II.
Capsular Features of the Pleomorphic Adenomas.

	All	Cellular Type	Myxoid Type	Classic Type
No. of patients	100	35	51	14
Focally very thin capsules	97	34 (97%)	49 (96%)	14 (100%)
Focal absence of encapsulation	46	4 (11%)	36* (71%)	6 (43%)
Pseudopodia and satellite nodules	28	8 (23%)	17 (33%)	3 (21%)

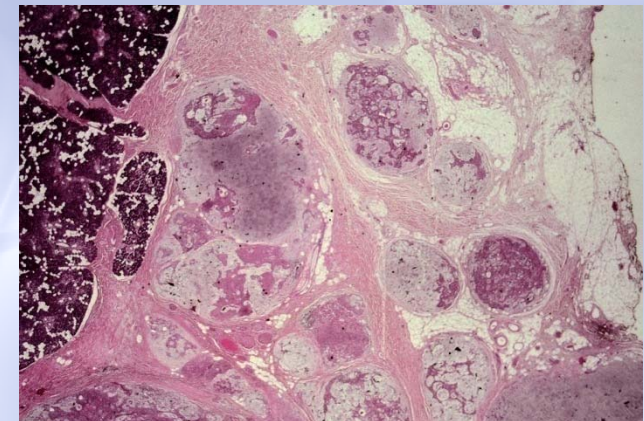
*Significantly different from other subtypes (Pearson's Chi-Square Test; $P \leq .01$).

Recurrences after pleomorphic adenoma surgery occur years after the initial surgery

		N	Mean interval
Renehan	1996	114	8 y
Leverstein	1997	29	10 y
Yugueros	1998	39	14 y
Carew	1999	31	9 y
Glas	2001	52	3 y
Maxwell	2004	35	5 y
Leonetti	2005	42	5 y
Zbären	2005	33	12 y

Recurrences after pleomorphic adenoma surgery are multicentric

		N	First recurrence		First and subsequent recurrence	
Phillips	1995	126			85	(67%)
Renahan	1996	114	52	(46%)		
Maxwell	2004	35	20	(57%)		
Stennert	2004	31			28	(90%)
Leonetti	2005	42			42	(100%)
Zbären	2005	33	24	(73%)		



Patients with recurrent pleomorphic adenoma are younger at first presentation by 15-20 years

		with recurrent pleomorphic adenoma		without recurrent pleomorphic adenoma	
		No of patients	mean age	No of patients	mean age
Maran	1984	19	27	64	51
Mc Gregor	1988	31	30	143	47
Renehan	1996	114	32	551	47
Zbären	2007	42	36	294	46

more aggressive tumors in young patients ?
less extensive surgery in young patients ?

Pleomorphic adenoma recurrences are associated with further recurrences

		No of patients	Re - recurrence
O'Dwyer	1986	32	31%
Renehan	1996	114	15%
Yugueros	1998	39	21%
Maxwell	2004	35	24%
Zbären	2007	42	19%

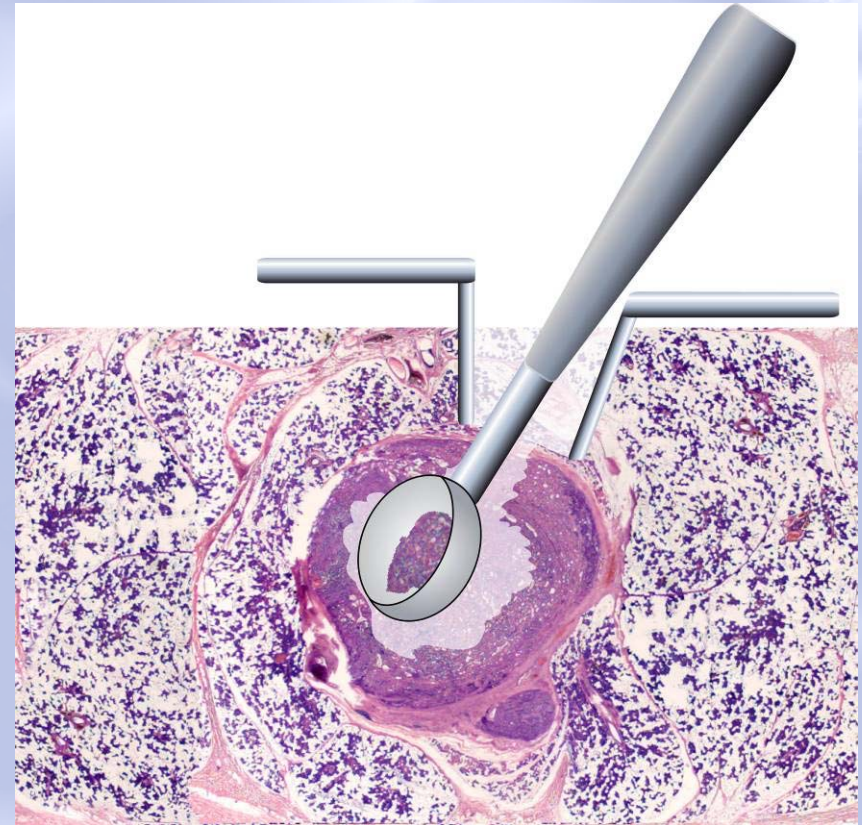
- Second recurrences more frequent ~ 15-30%

Surgery for pleomorphic adenoma recurrences are associated with more facial nerve complications

		No of patients	Facial nerve paresis
Maran	1984	19	31%
O'Dwyer	1986	32	15%
Renahan	1996	114	24%
Zbären	2005	42	19%

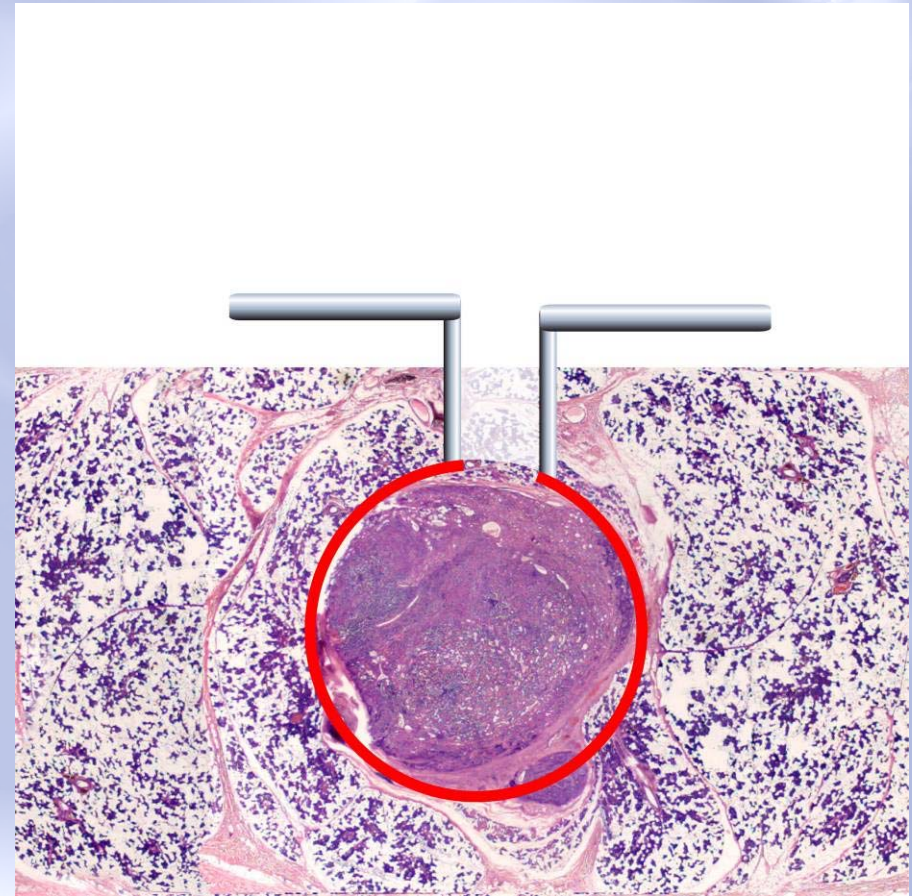
Type of parotid surgery - Enucleation intracapsular

"There are three possible planes in which a parotid tumor may be removed.
In the first place, the rather delicate capsule may be opened and the contained tumor tissue expressed. After this has been done an attempt may be made to remove the capsule."

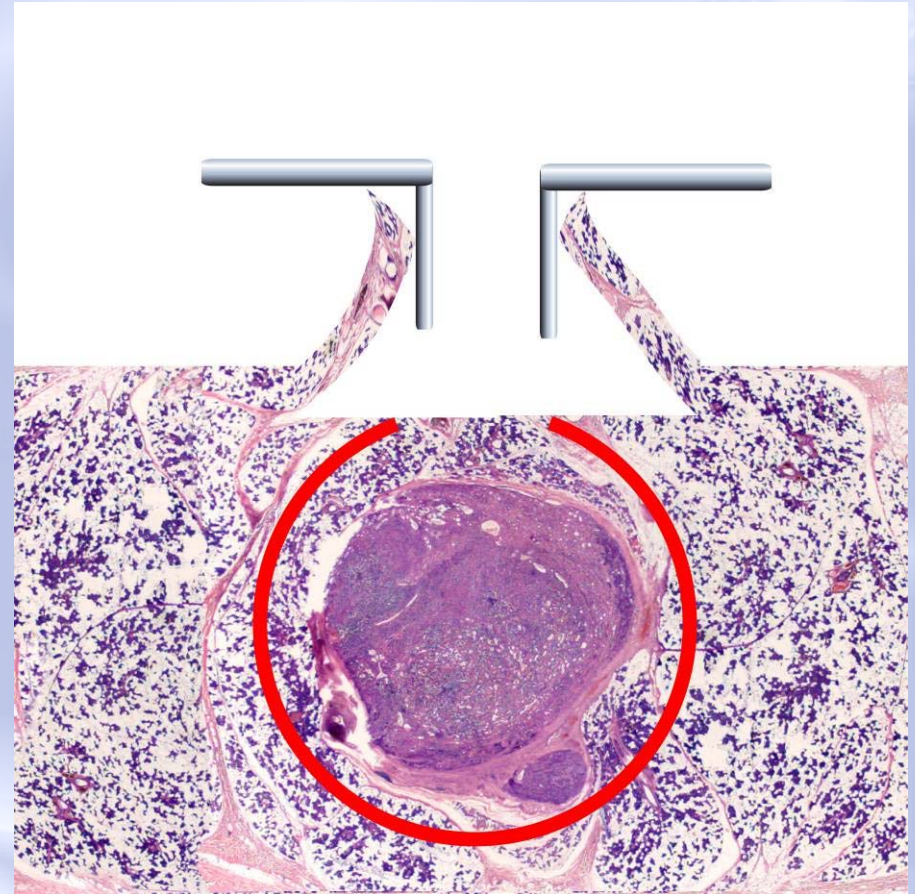


"curettage"
"shelling out"

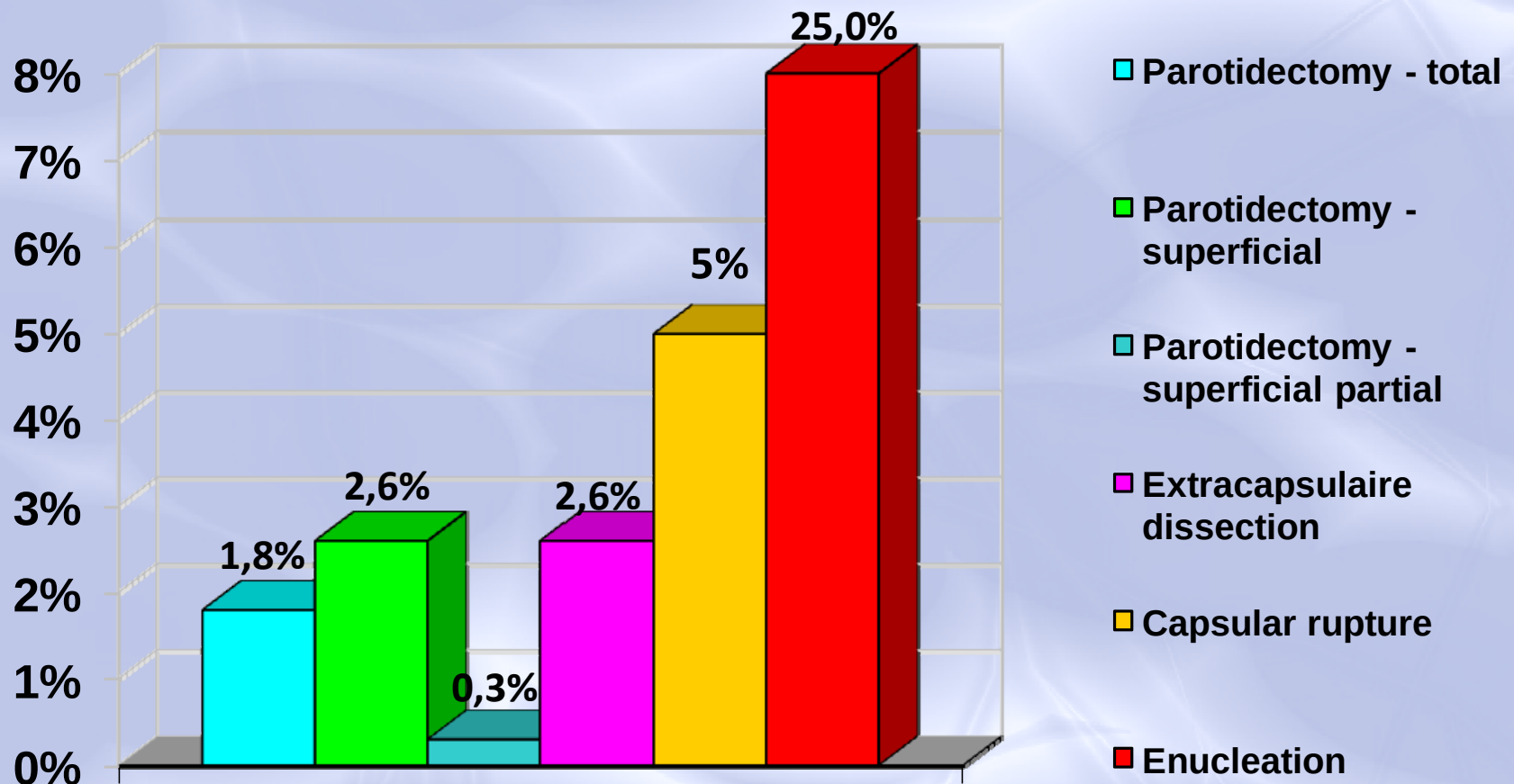
"Secondly the tumor may be enucleated in the layer of loose alveolar tissue which lies between it and the surrounding normal salivary glandular tissue..."



"Finally the tumor may be removed with a margin of surrounding salivary glandular tissue..."



Enucleation is associated with more recurrences



Why do pleomorphic adenoma recur?

- Capsule rupture (tumor dissemination in the wound) ?
- Incomplete resection (islands of tumor tissue left behind during surgery)
 - ▶ *inadequate surgery (enucleation, extracapsular dissection)*
 - ▶ *impossible because of facial nerve proximity*
 - ▶ *inadvertant ...*
- ▶ *More aggressive surgery in young patients*
- ▶ *Recurrence should be below 2%*

Treatment:

SURGERY

few recurrences

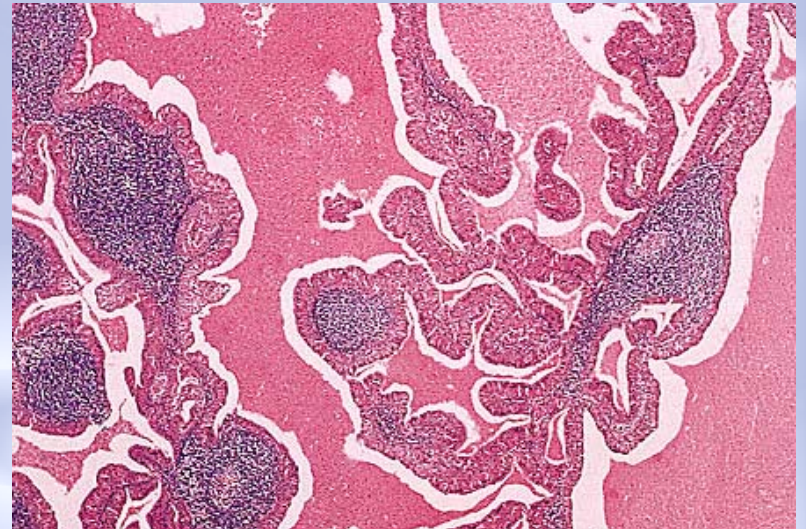
no transformation into cancer

Cystadenolymphoma (Whartin tumor)

association with smoking

FNA pretty good

10% bilateral



Difficulties with salivary gland carcinoma:

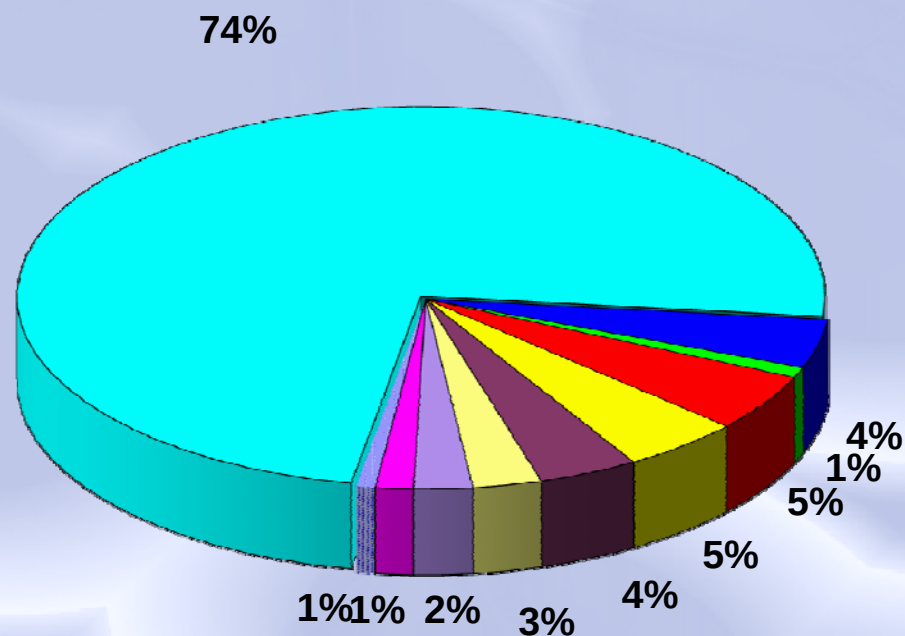
24 different histologic types

Rarity of most histologic types

knowledge based exclusively on retrospective studies → **controversies**

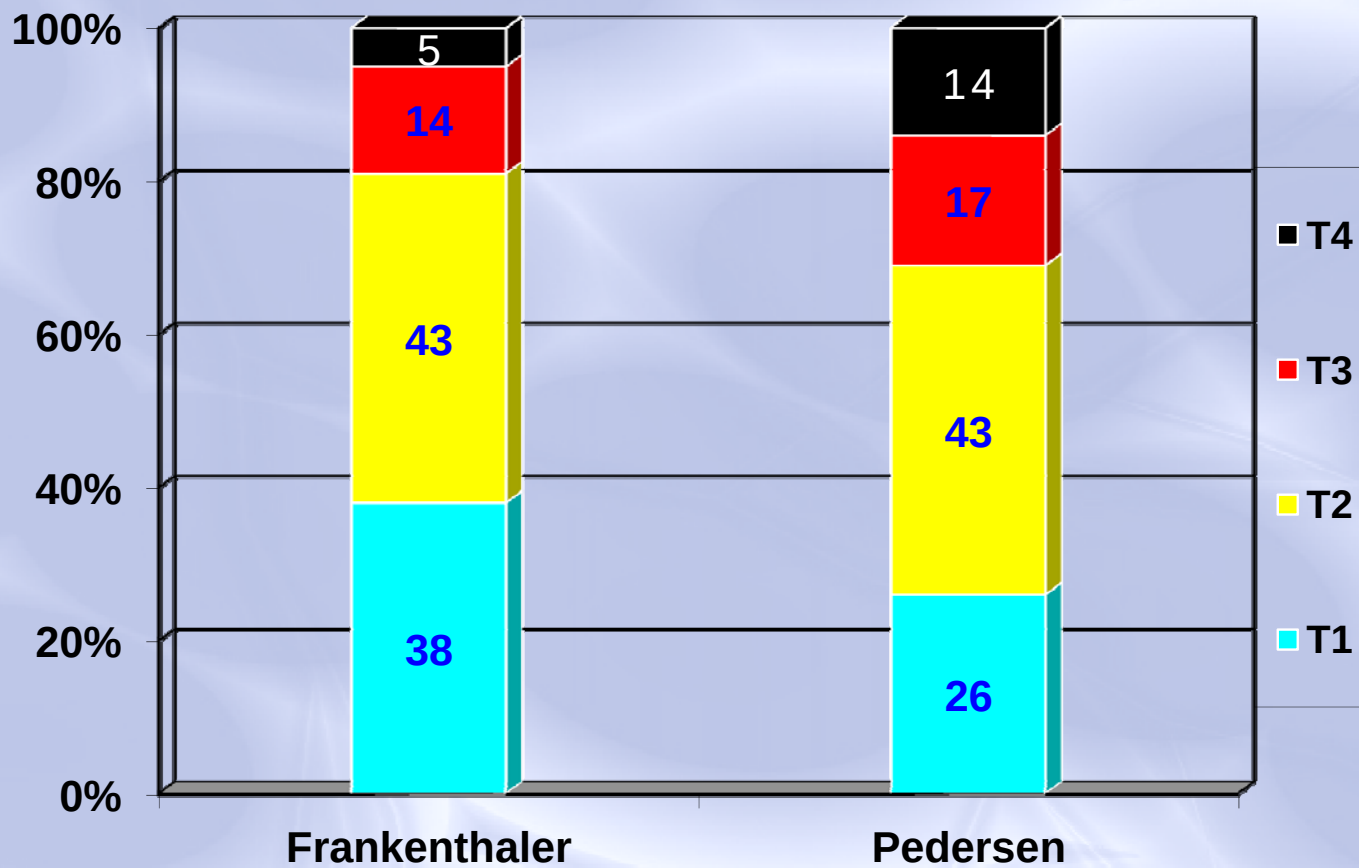
Malignant epithelial tumours

- Acinic cell carcinoma
- Mucoepidermoid carcinoma
- Adenoid cystic carcinoma
- Polymorphous low-grade adenocarcinoma
- Epithelial-myoepithelial carcinoma
- Clear cell carcinoma, not otherwise specified
- Basal cell adenocarcinoma
- Sebaceous carcinoma
- Sebaceous lymphadenocarcinoma
- Cystadenocarcinoma
- Low-grade cribriform cystadenocarcinoma
- Mucinous adenocarcinoma
- Oncocytic carcinoma
- Salivary duct carcinoma
- Adenocarcinoma, not otherwise specified
- Myoepithelial carcinoma
- Carcinoma ex pleomorphic adenoma
- Carcinosarcoma
- Metastasizing pleomorphic adenoma
- Squamous cell carcinoma
- Small cell carcinoma
- Large cell carcinoma
- Lymphoepithelial carcinoma
- Sialoblastoma



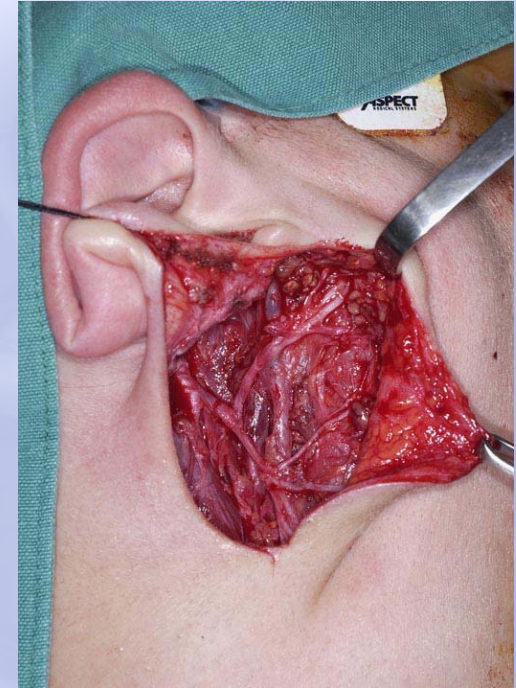
La tumeur primitive	
T1	Tumeur ≤ 2 cm dans sa plus grande dimension
T2	Tumeur > 2 cm et ≤ 4 cm dans sa plus grande dimension
T3	Tumeur > 4 cm et ≤ 6 cm dans sa plus grande dimension
T4	Tumeur ≥ 6 cm
Ces stades sont subdivisés en fonction de la présence ou non d'extension locale qui est définie comme une extension macroscopique aux nerfs, l'os, les tissus mous et la peau.	
a	Absence d'extension locale
b	Présence d'extension locale

Parotid cancer - T stage



Total parotidectomy is the standard surgery for parotid carcinoma

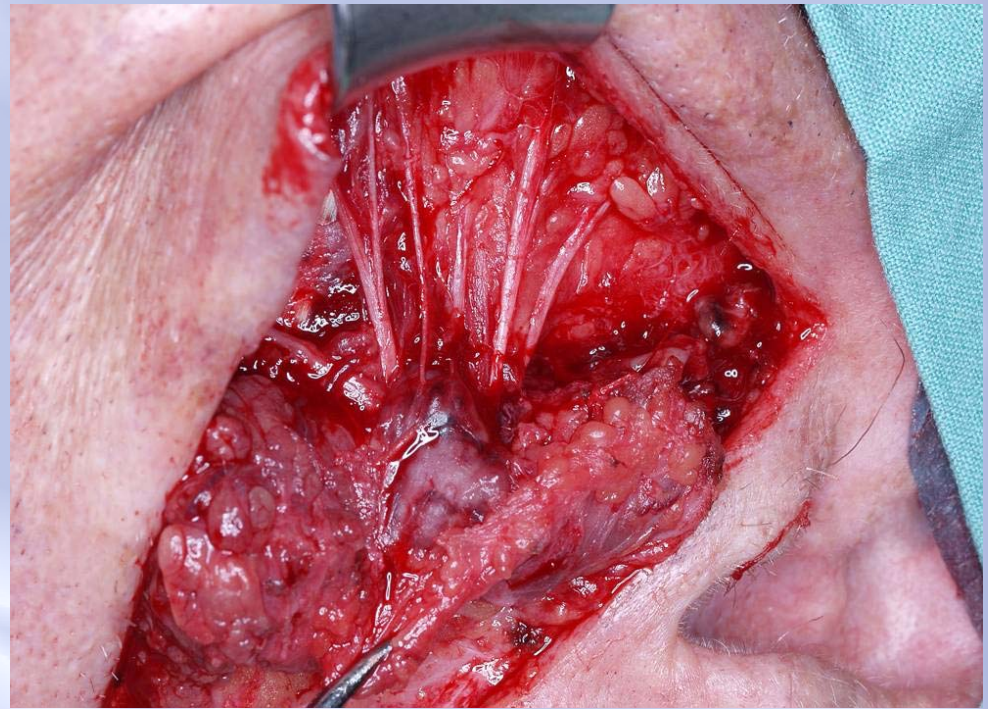
T1/T2 low grade	Superficial parotidectomy
Others	Total parotidectomy



All T	Total parotidectomy
-------	---------------------

No facial nerve sacrifice for parotid carcinoma

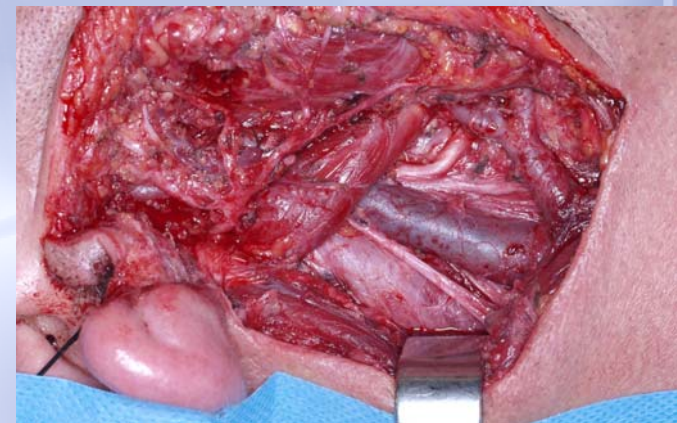
Facial nerve branches should not be sacrificed unless the tumor is adherent



Neck dissection for parotid carcinoma is controversial

N+	Modified radical neck dissection
N0 (T3/T4 ± High grade)	Selective neck dissection
N0 (T1/T2 ± Low grade)	Nothing vs. Level 2-3

BUT	5-years survival
N+	9%
N0	74%
Neck recurrence	17 %



Incidence of neck metastasis

Author	Journal	Year	n	Neck metastasis
Mc Guirt	<i>Arch Otolaryngol Head Neck Surg</i>	1989	90	28%
Witten	<i>Cancer</i>	1990	105	23%
Kelley	<i>Arch Otolaryngol Head Neck Surg</i>	1996	121	13%
Regis	<i>Arch Otolaryngol Head Neck Surg</i>	2001	145	23%
Stennert	<i>Arch Otolaryngol Head Neck Surg</i>	2003	160	13%
Zbären	<i>Am J Surg</i>	2003	98	21%

Adjuvant radiation

T3 / T4

High grade

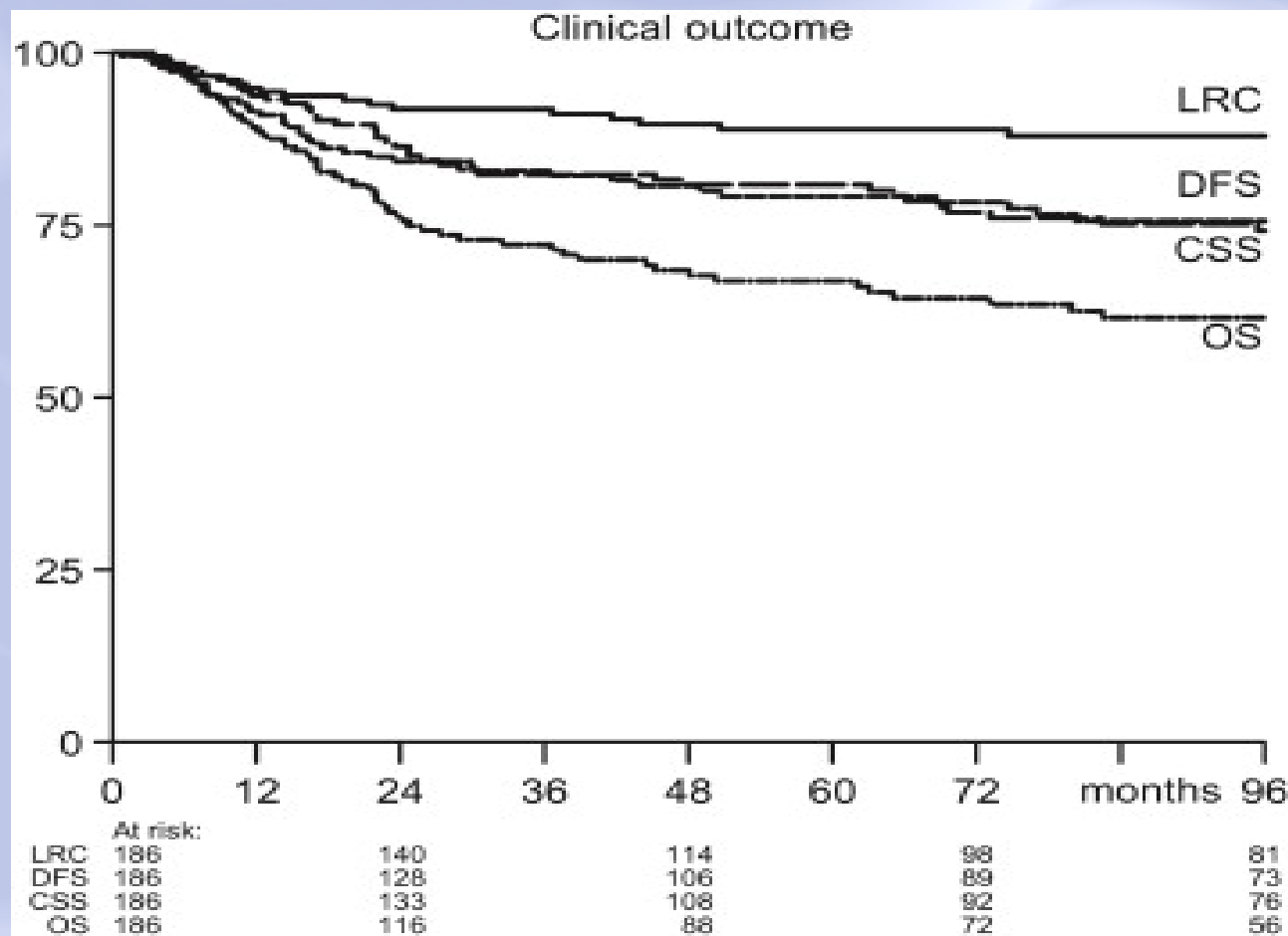
R1 - resection

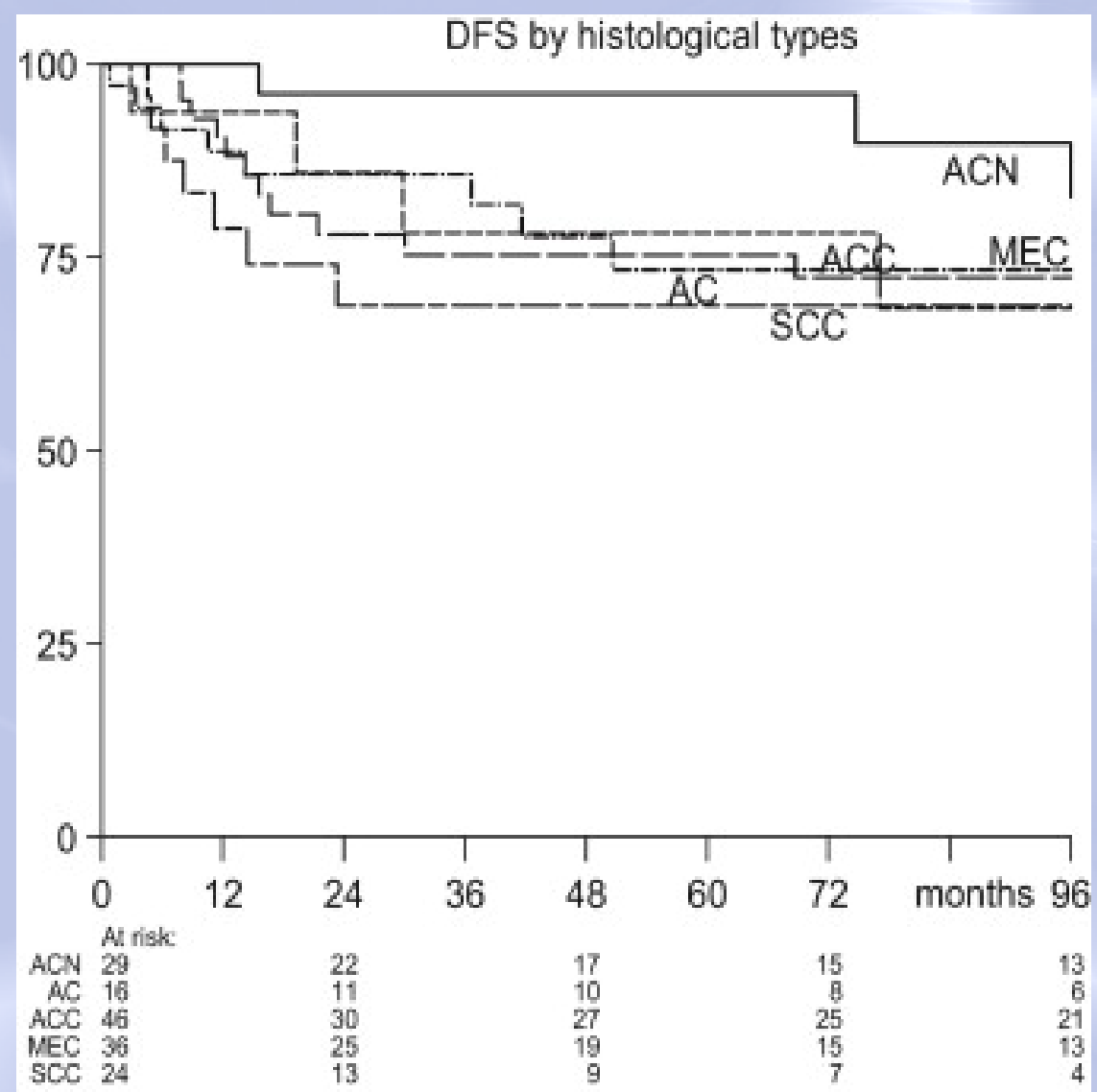
LN - metastases

Prognostic factors

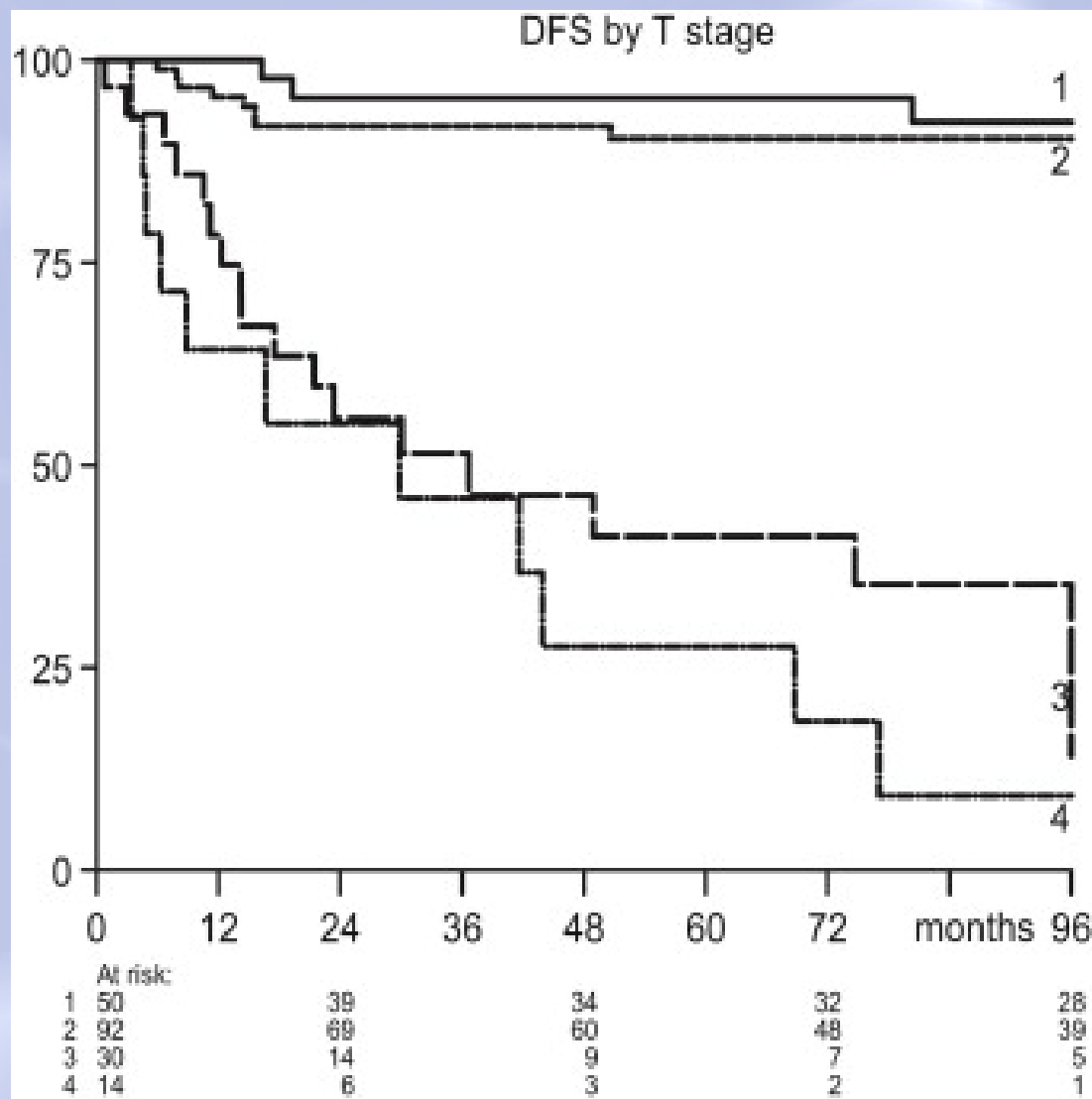
Clinical stage	I-II vs. III-IV
Grade	Low vs. high
T	T1-2 vs. T3-4
Facial nerve palsy	
Pain	
Neck metastasis	

SG Carcinoma - outcome





SG Carcinoma - outcome



1. Prevention of recurrence, which requires a complete tumor removal, ideally with a cuff of normal parotid tissue, and without tumor seeding by spillage
2. Facial nerve protection and preservation unless the nerve is directly involved by a malignant neoplasm
3. Prevention of occurrence of Frey syndrome
4. Prevention of other complications, such as salivary gland fistula, hematoma, wound infection, skin anesthesia
5. Optimal cosmetic results (incision; depression)

Parotidectomy - Positioning

General anesthesia:

- oral intubation

- tube to the contralateral side

- curare derivatives ➡ 20'

Patient positioning:

- torso & head at 20° (decrease of venous congestion)

- head gently extended

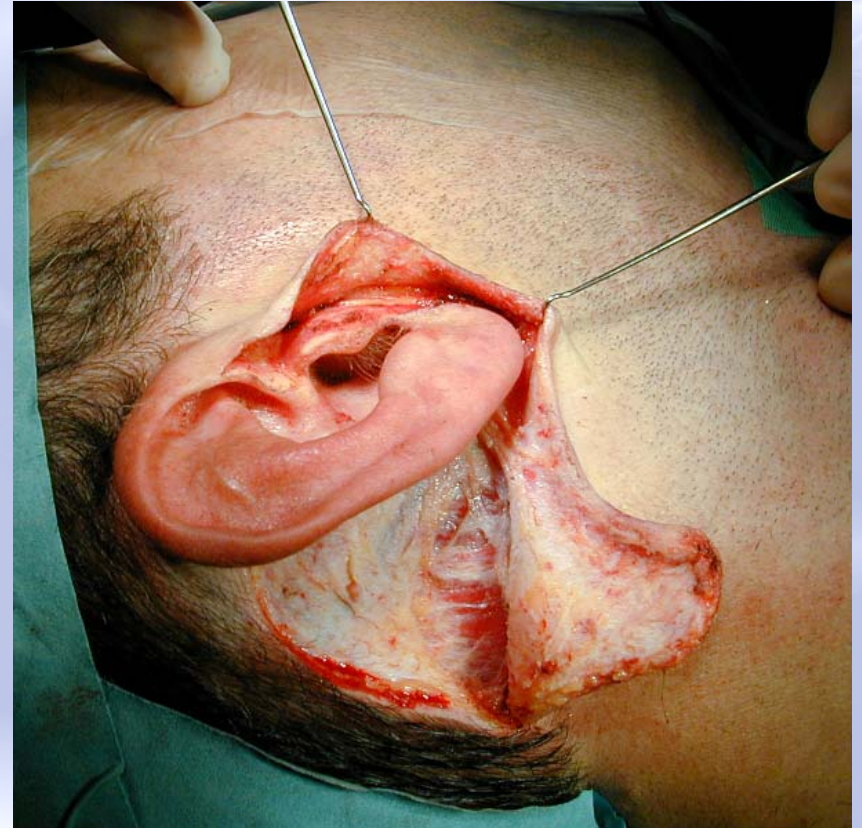
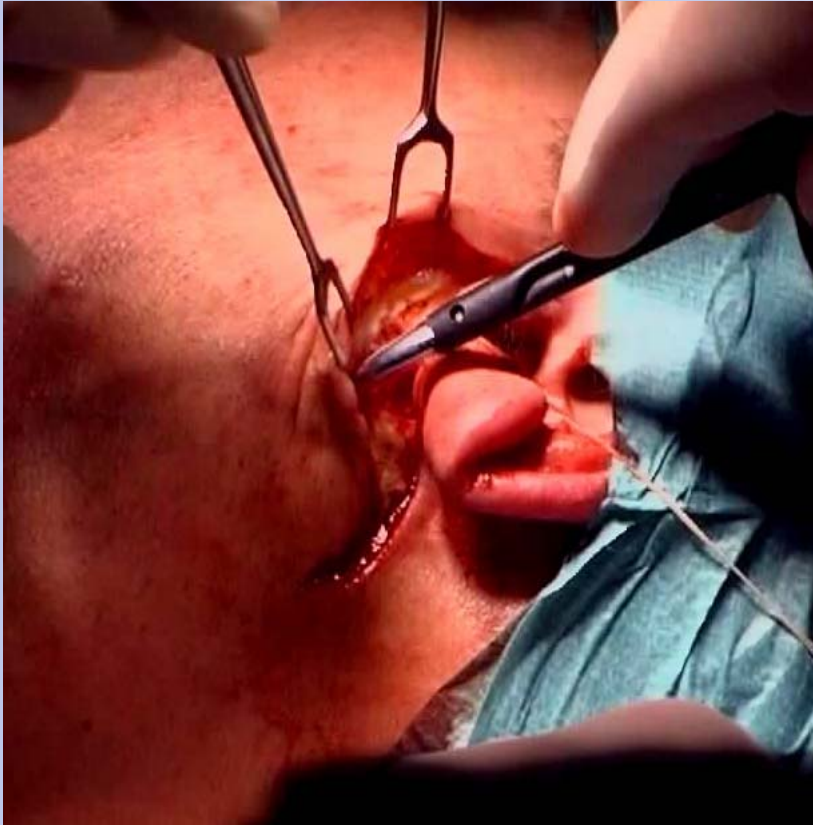
- head gently rotated to the other side

Infiltration : adrenalin 1:20 000 without lidocaine

Draping: entire facial half visible



Parotidectomy - Incision



Incision: lazy S or facelift

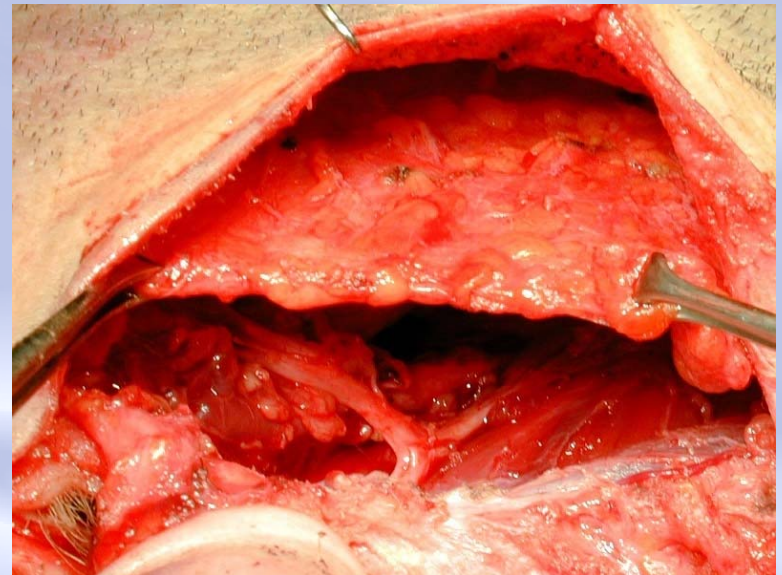
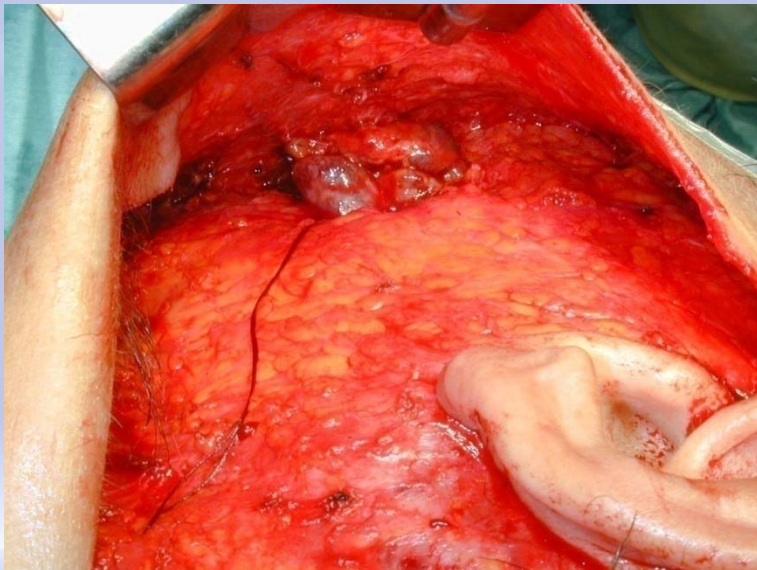
identical anterior or preauricular limb

facelift:

good for superficial lobe and inferiorly situated tumors

difficult for anterior and superior (preauricular) tumors

Anterior subcutaneous flap dissection



Parotidectomy - Greater auricular nerve

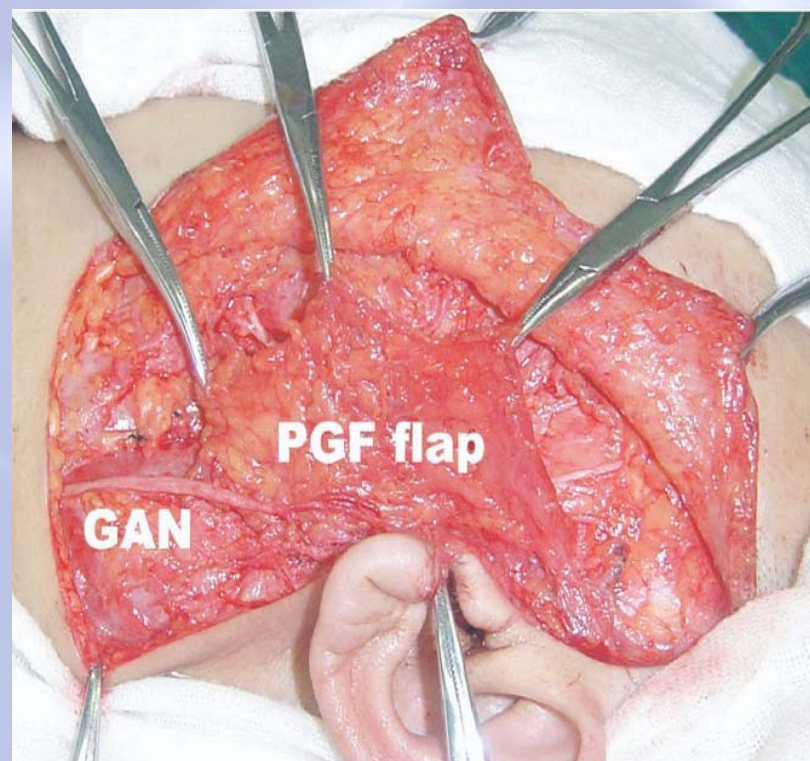
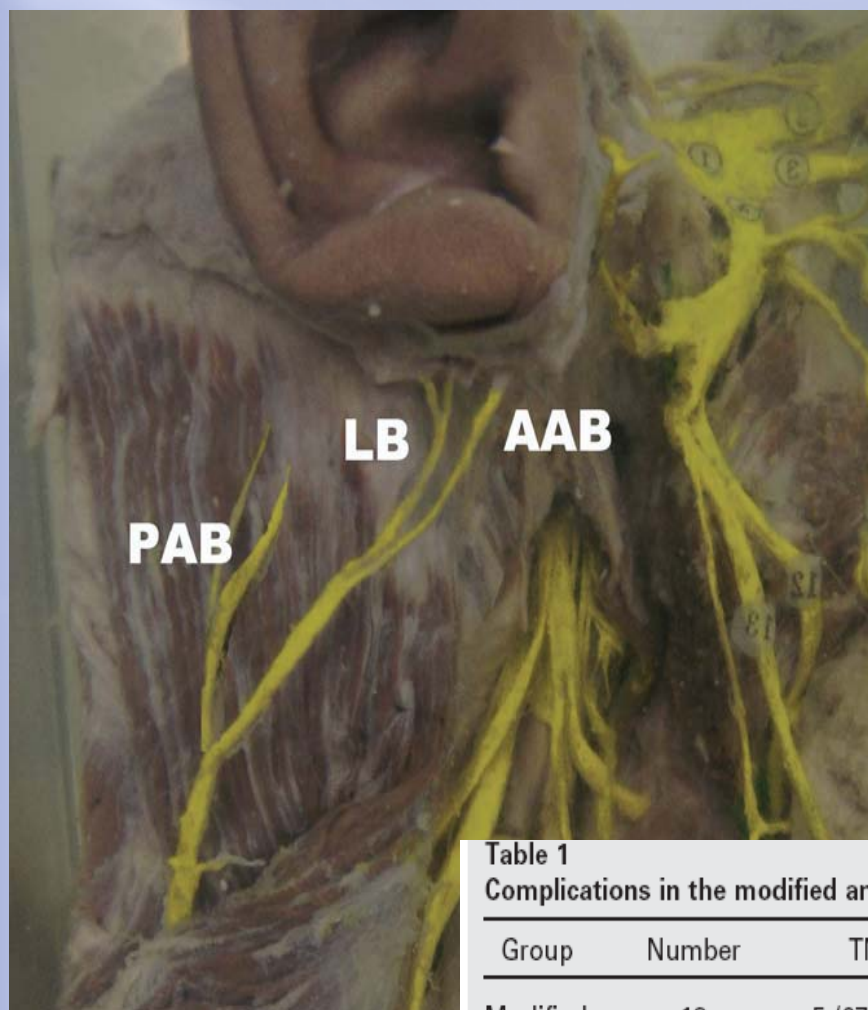


Table 1
Complications in the modified and control groups

Group	Number	TN	PN	FS	TND	PNP	SF	TR
Modified	18	5 (27.7%)*	0*	3 (16.7%)*	11 (61.1%)	0	0	0
Control	30	30 (100%)	4 (13.3%)	20 (66.7%)	17 (56.7%)	0	1 (3.3%)	0

* $P < 0.01$, TN, transient numbness; PN, permanent numbness; FS, Frey's syndrome; TND, transient facial nerve disable; PNP, permanent facial nerve disable; S, salivary fistula; TR, tumor recurrence.

Parotidectomy - Facial nerve identification

Useful landmarks for the facial nerve trunk include:

1. the "cartilaginous pointer" - trunk is said to be 1 cm deep and 1 cm inferior to the pointer

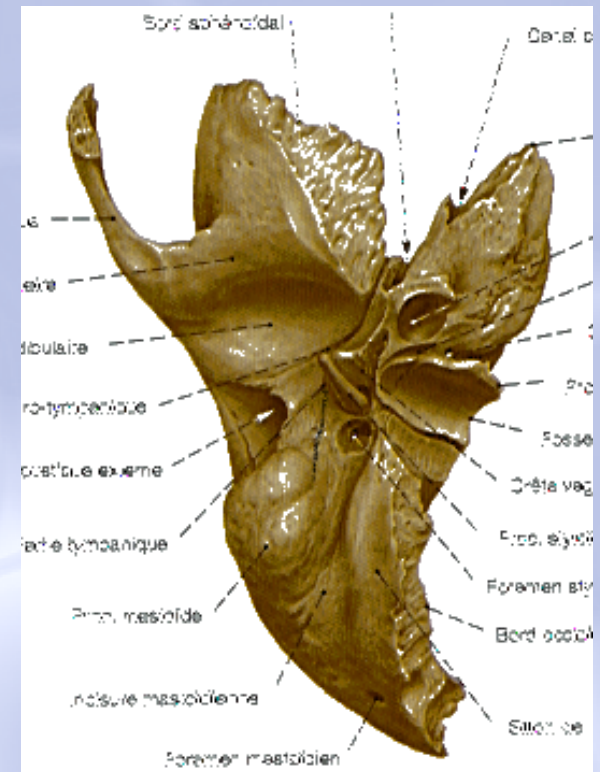
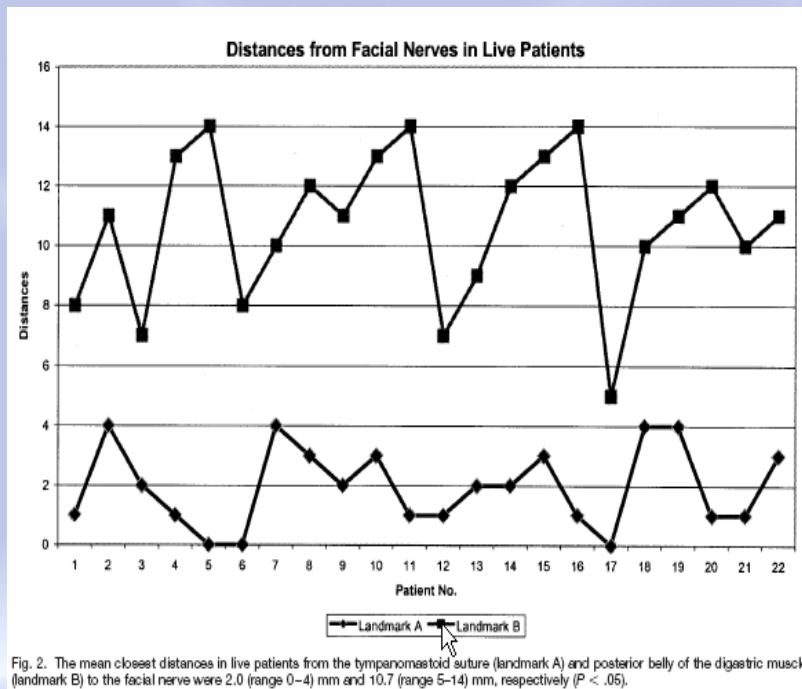
TABLE I.
Facial Nerve Measurements* during Parotidectomy.

Topography	Mean	95% Confidence Interval	Minimum	Maximum
Tragal pointer to main trunk facial nerve (n = 78)	6.37	5.84-6.89	2	14

Parotidectomy - Facial nerve identification

Useful landmarks for the facial nerve trunk include:

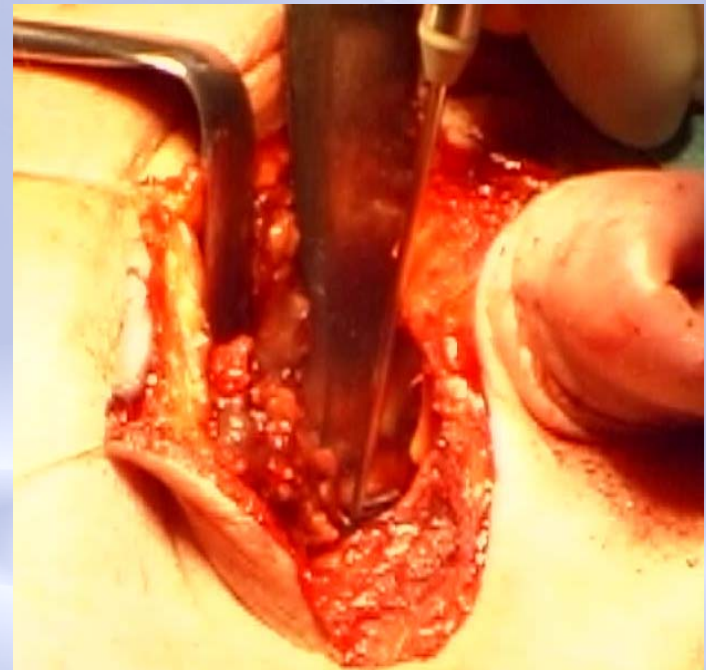
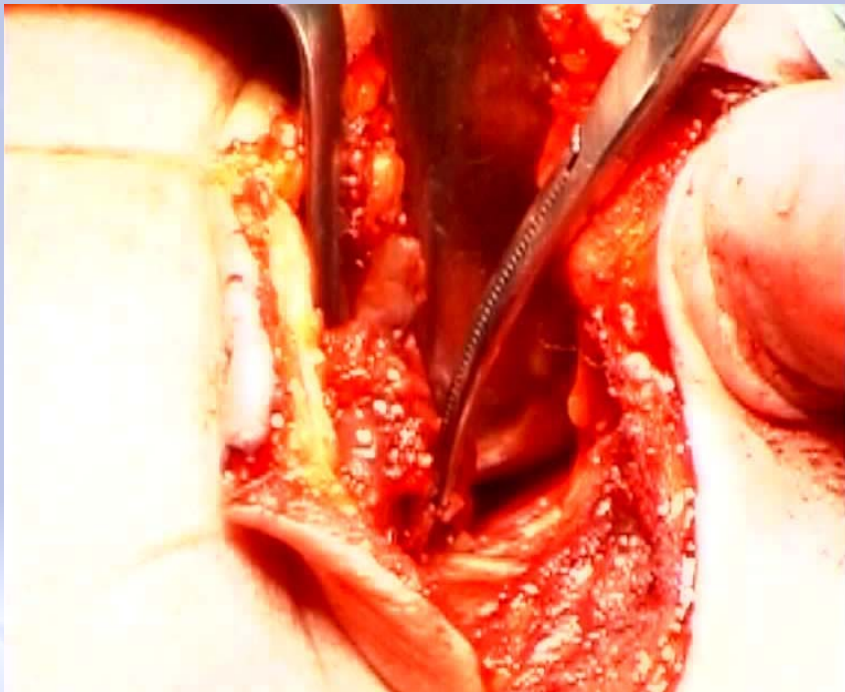
- the posterior belly of the digastric muscle and its mastoid insertion, which is slightly lateral to the stylomastoid foramen
- the tympanomastoid suture, which can be appreciated by palpation. The trunk is said to be 2 mm from the "inferomedial end of the suture"



Parotidectomy - Facial nerve identification

Useful landmarks for the facial nerve trunk include:

4. the stylomastoid artery running with its vein a few millimeters lateral to the facial nerve
5. the styloid process which is located deep to the facial nerve. It can be palpated, but its visualization before identification of the nerve usually means that the nerve has been injured



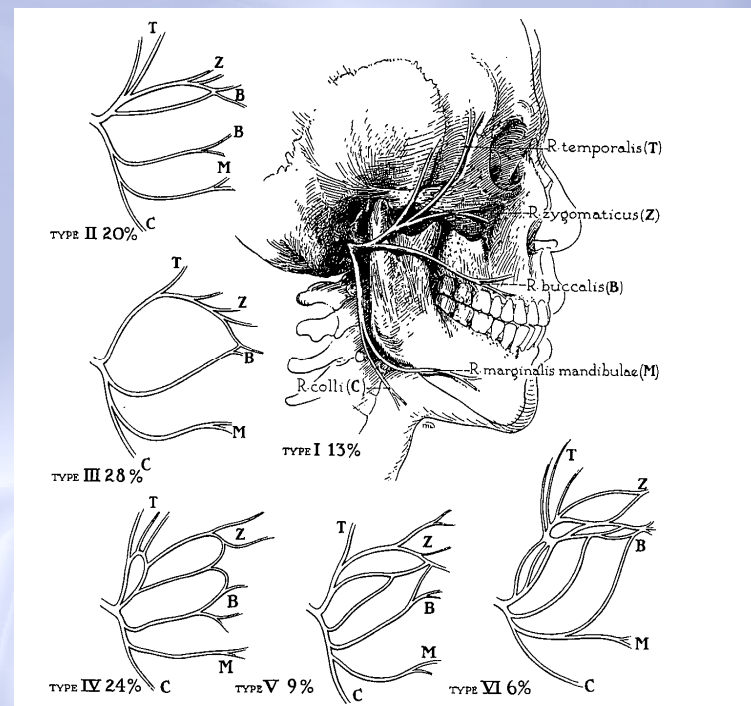
Parotidectomy - Facial nerve branches

Branches located at the extremities of the nerve distribution receive fewer anastomosis with other branches

The majority of anastomosis occur between the buccal and zygomatic divisions, forming the so-called parastenon plexus

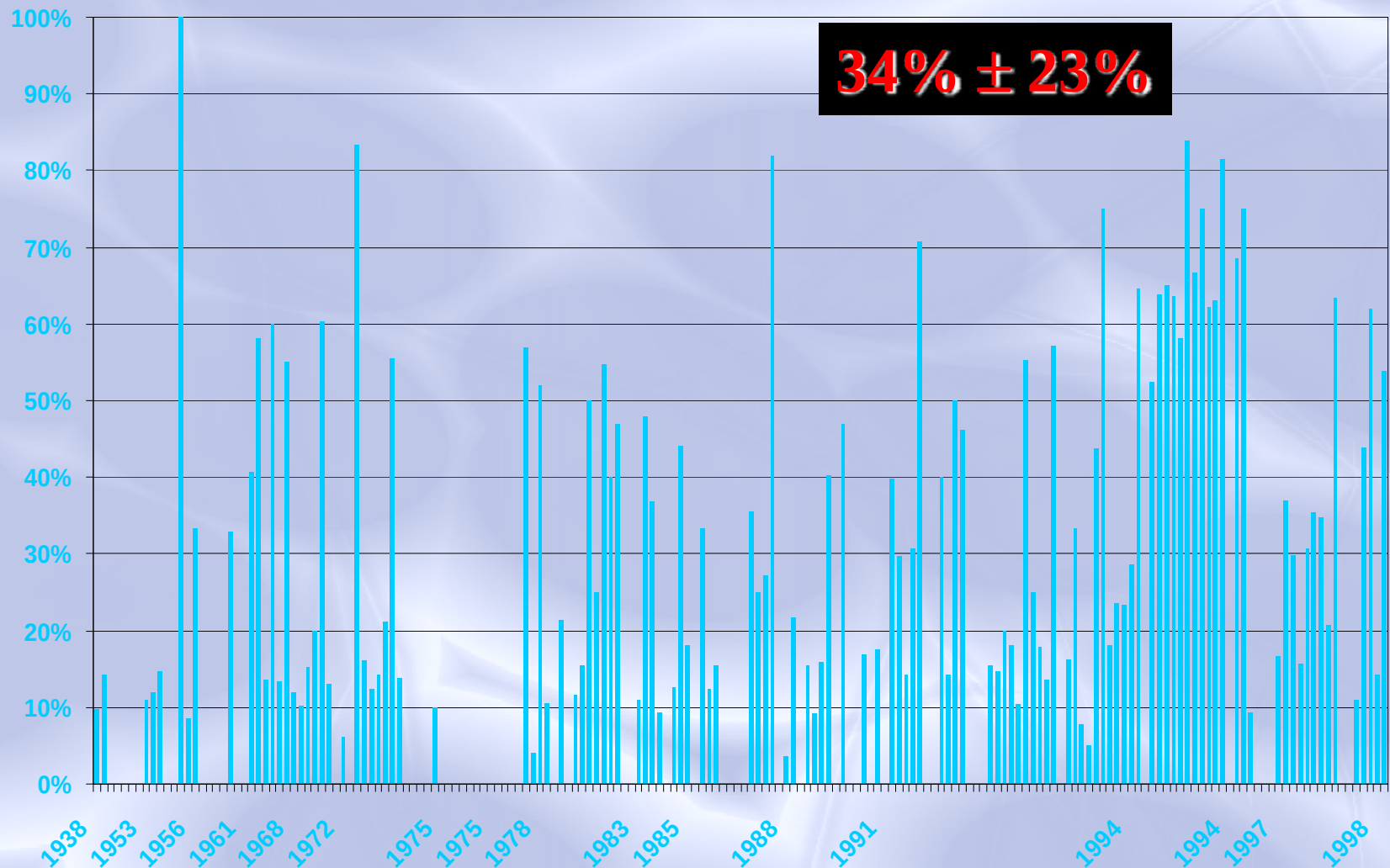
The number of anastomosis decreases in caudal branches, with the marginal mandibular branch receiving anastomosis in only 6.3%

There is no anastomosis between the cervical and other branches

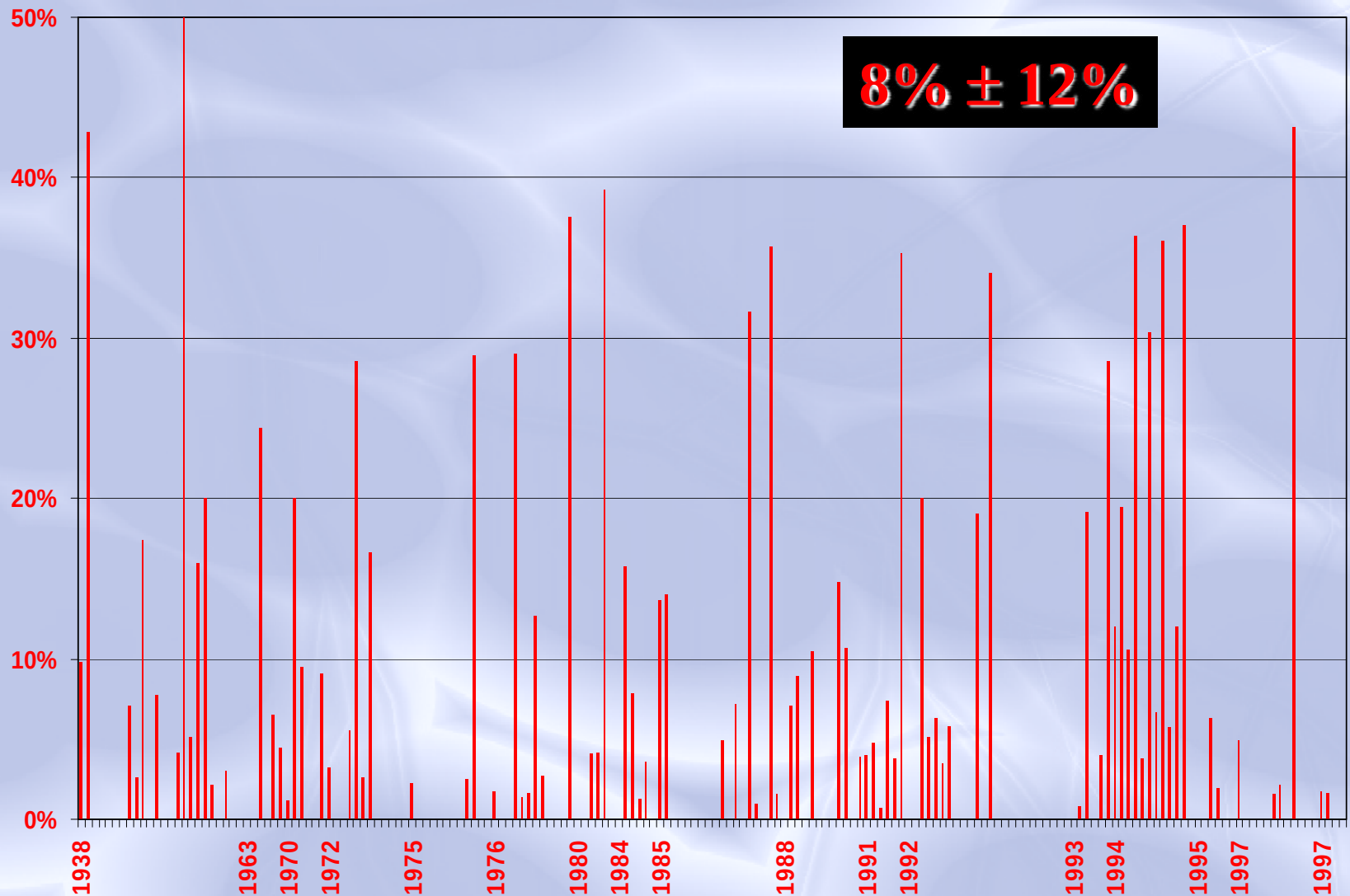




Facial paralysis - immediate or transitory

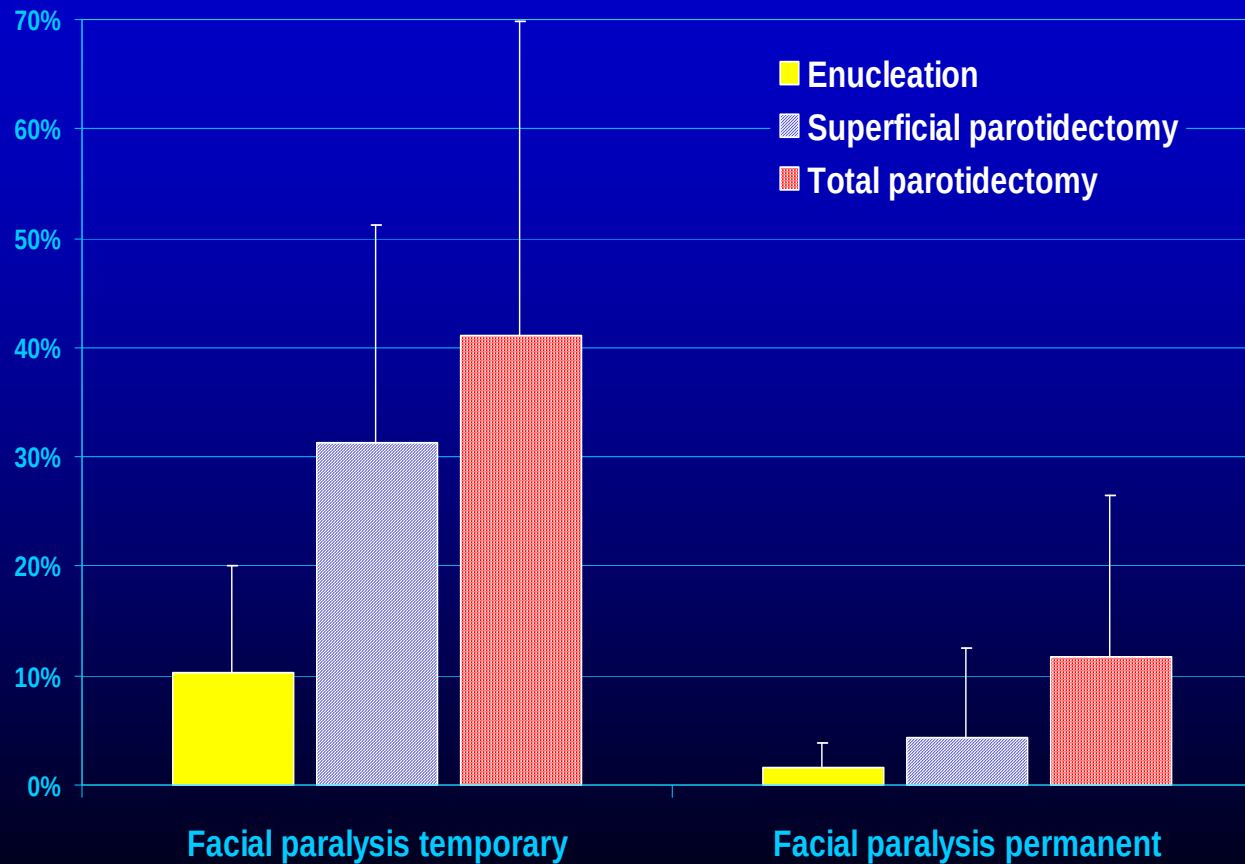


Facial paralysis - definitive

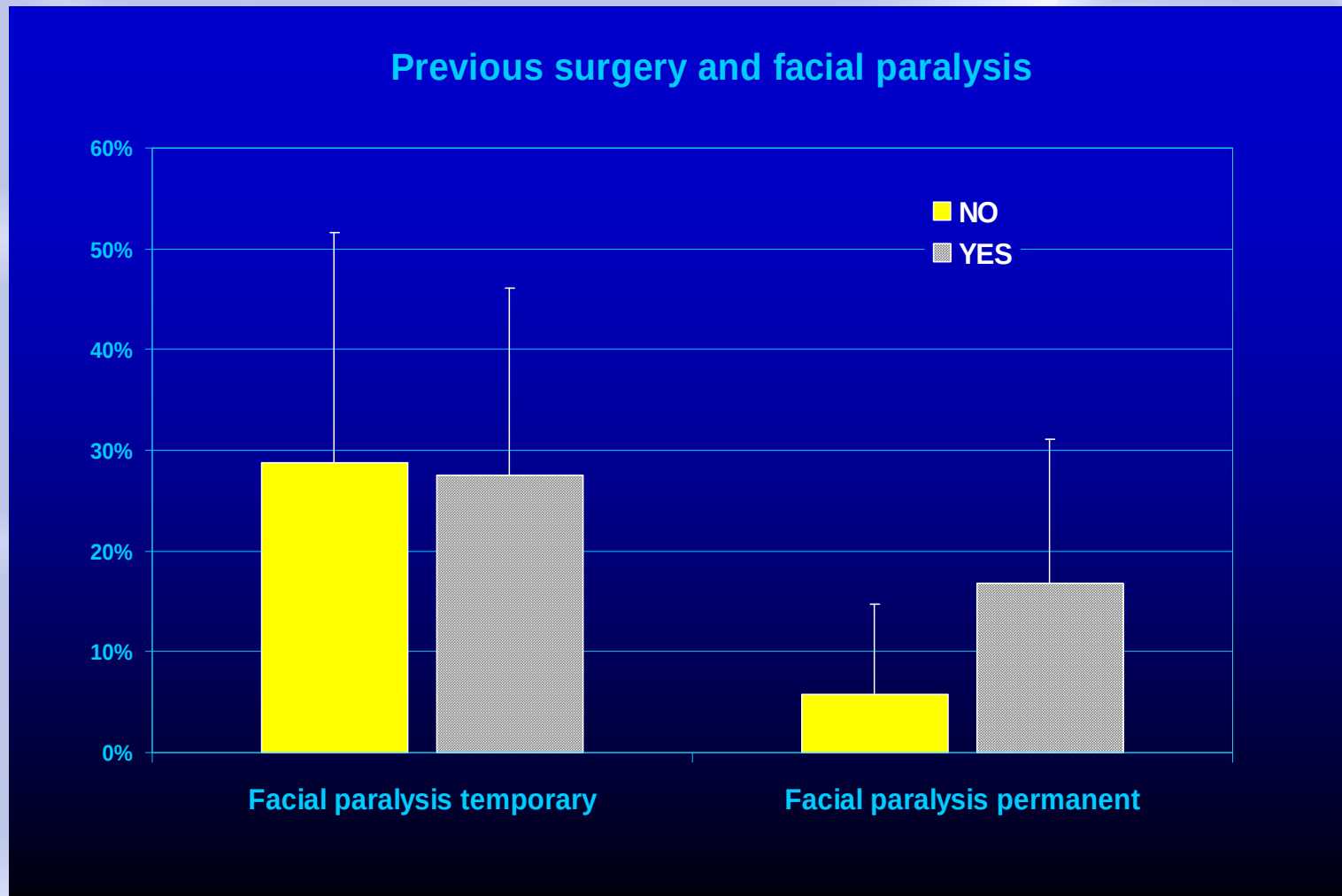


Facial paralysis increases with the extent of surgery

Type of parotidectomy and facial paralysis

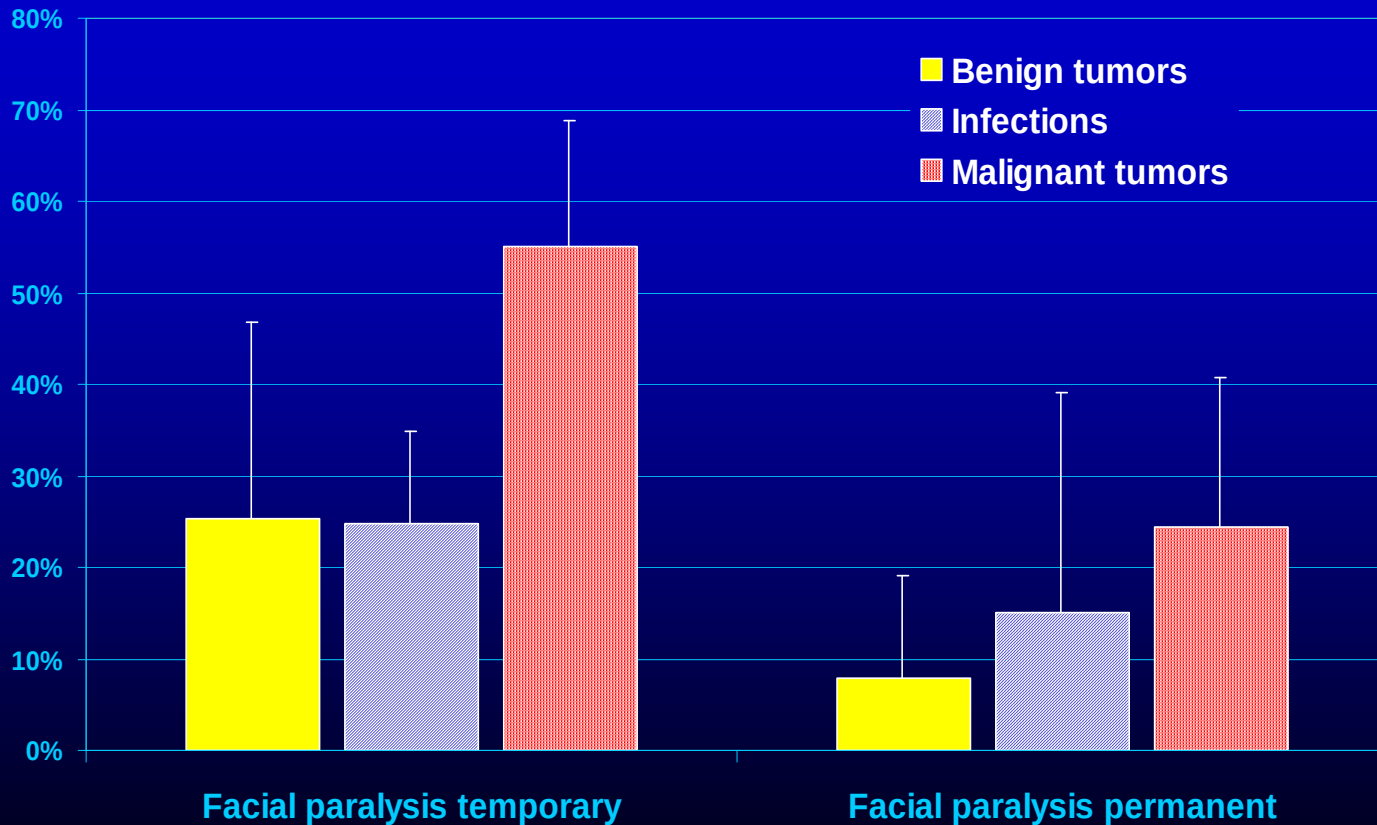


More permanent facial paralysis during re-operations



Facial paralysis is increased when surgery for infection or cancer

Histology and facial paralysis



Variable	Values	VMGi average	VMG standard deviation	Statistical test	p
Age		89.11%	18.26%	Pearson correlation	.199
Sex	M	89.49%	17.06%	Student t-test	.841
	F	88.60%	20.04%		
Type of surgery	Superficial	93.67%	9.3%	Student t-test	.007
	Total	81.85%	25.62%		
Branches sectioned	No	91.90%	13.92%	Student t-test	.001
	Yes	67.50%	31.57%		
Histology	Benign	94.49%	7.52%	One way ANOVA	< 0.001
	Cancer	77.29%	28.57%		
	Infection	66.33	30.14%		
Diameter of the lesion		89.11%	18.26%	Pearson correlation	.04
Duration of the procedure		89.11%	18.26%	Pearson correlation	<0.001

Intraoperative monitoring of facial nerve is useful

Transitory facial paresis 27% (14%) and definitive 4% (2%).

No important paresis (HB > 2) in benign tumor.

Definitive paralysis only if section of facial nerve branches.

Factors associated with increased frequency of transitory facial paresis:

- total parotidectomy

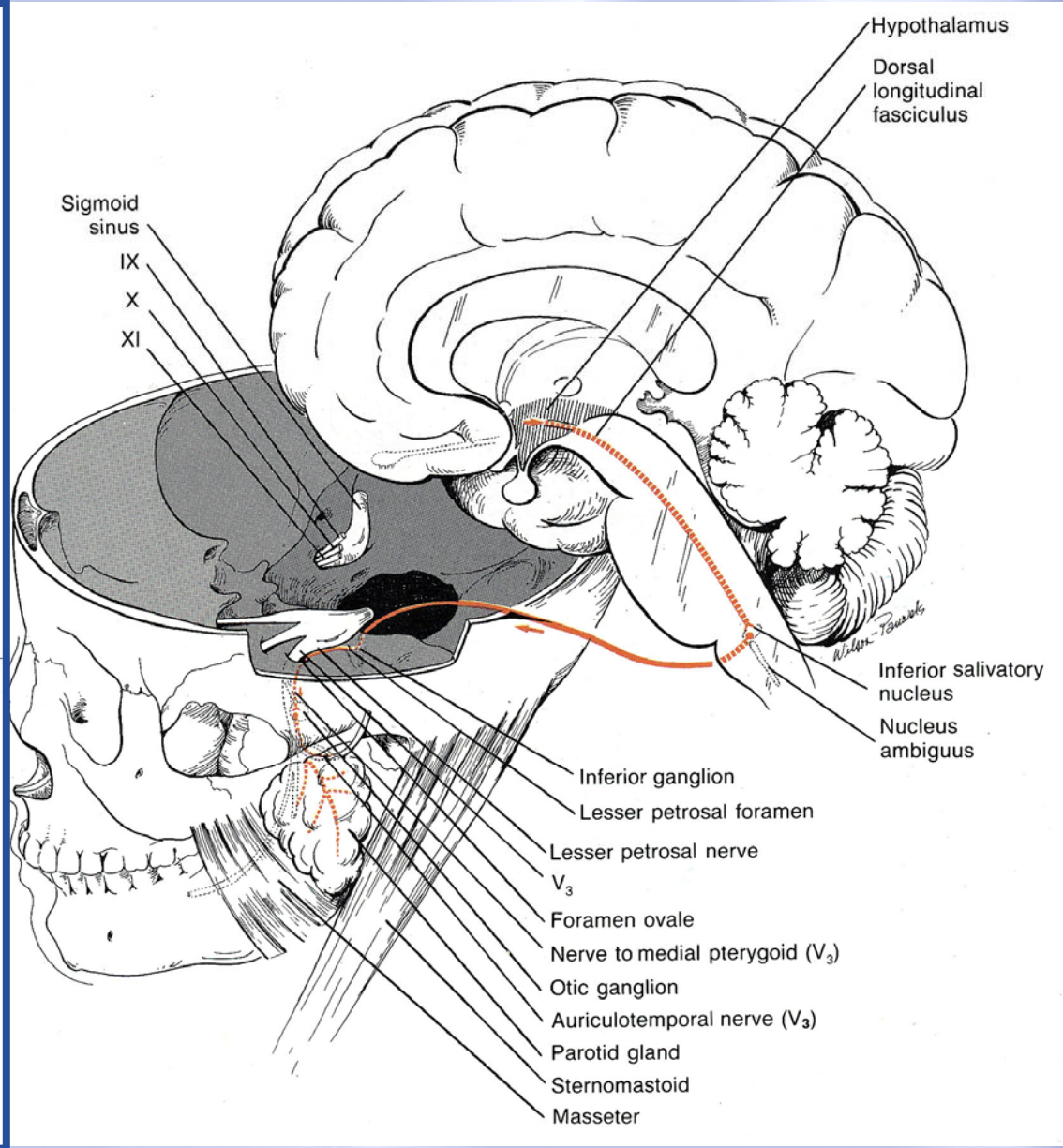
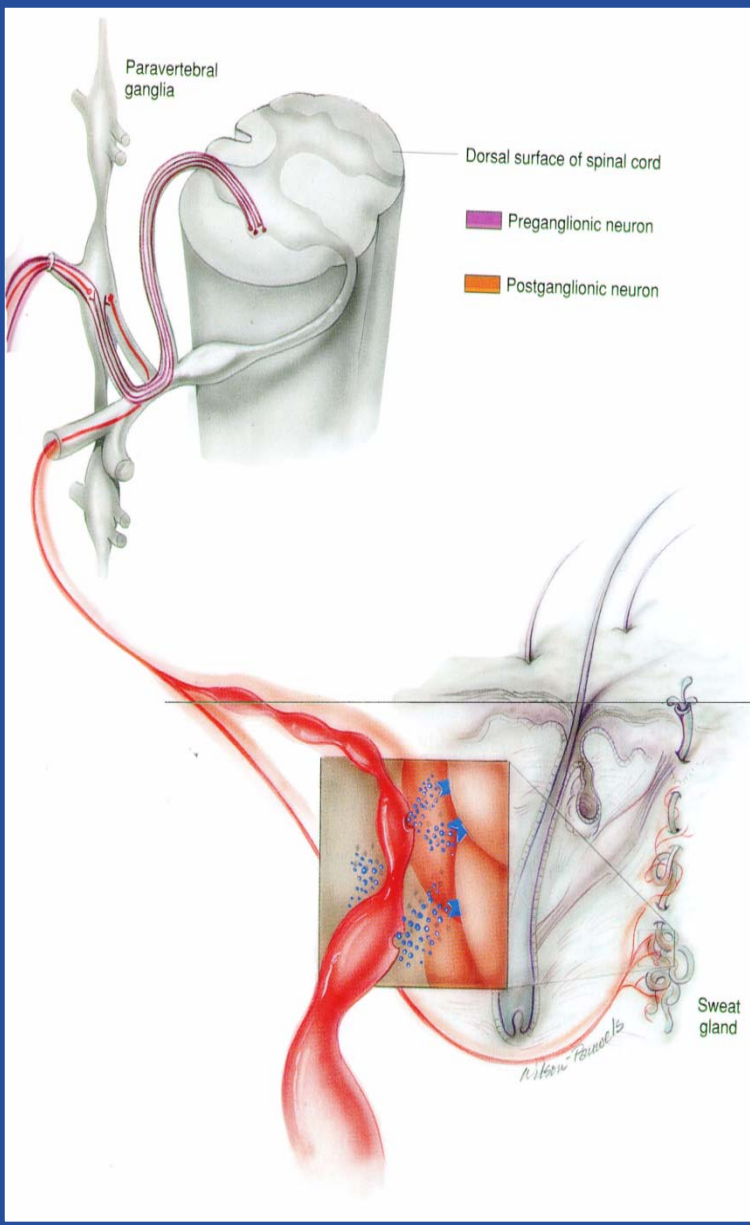
- section of facial nerve branches

- histopathology infectious or cancerous

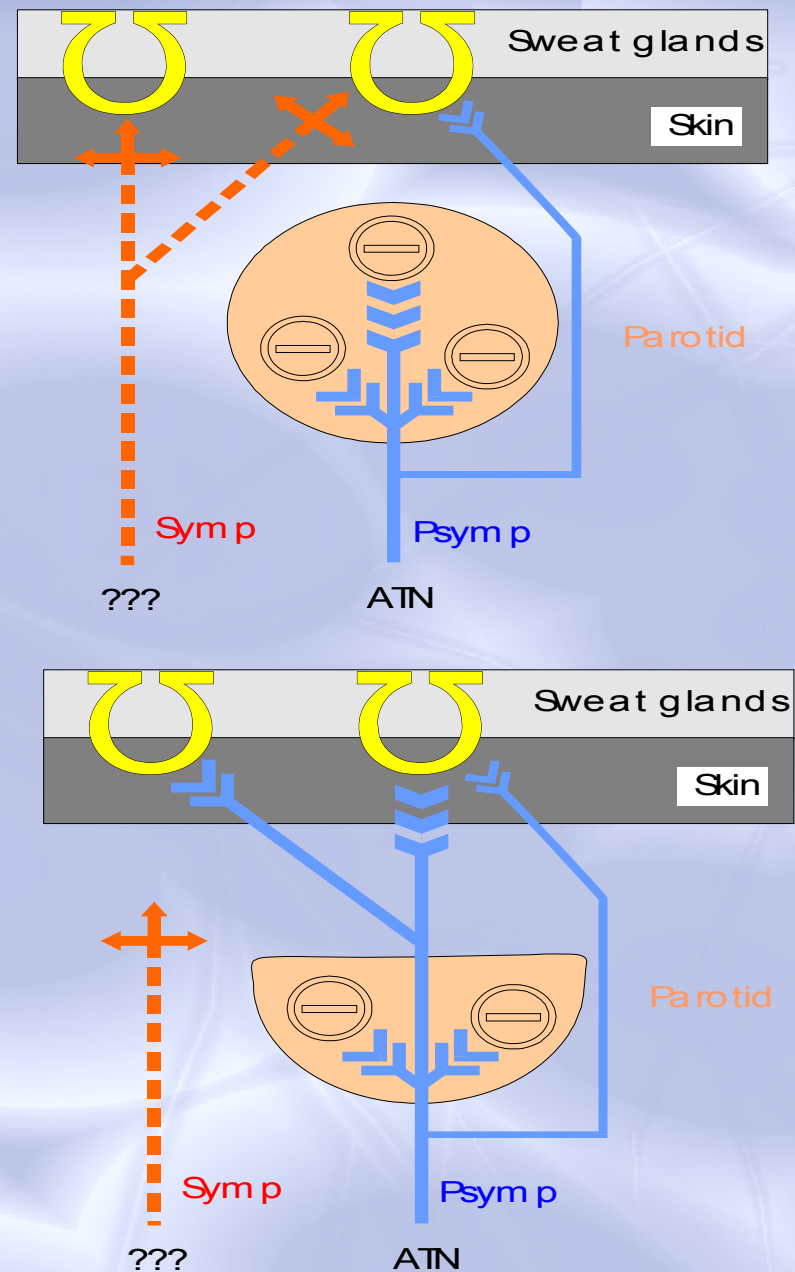
- size of lesion > 3 cm

- long duration of procedure

Facial sweating during eating
Facial flushing during eating
No relation with mastication
No spontaneous resolution



Aberrant regeneration theory



Latency of symptoms and progressive increase of the involved area

Wilson – Clin Sci, 1936
List & Peet – Arch Neurol Psych, 1938
Haxton – Brain, 1948
Laage-Hellman – Acta Otolaryngol, 1958
Linder – Laryngoscope, 1997

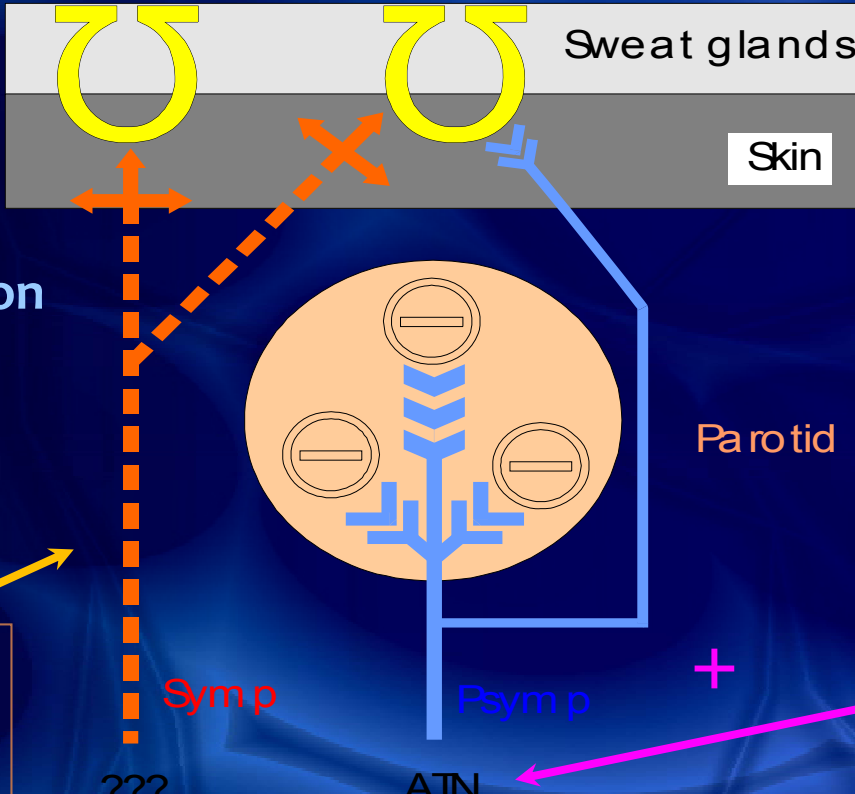
Atropine; Botox
Freedberg – J Clin Invest, 1948
Glaister – Brit Med J, 1958
Drobik – Acta Otolaryngol, 1995

Local absence of sympathetic function

List & Peet – Arch Neurol Psych, 1938
Freedberg – J Clin Invest, 1948
Haxton – Brain, 1948
Glaister – Brit Med J, 1958

Anesthesia
Freedberg – J Clin Invest, 1948
Glaister – Brit Med J, 1958
Drummond – J Auton Nerv Syst, 1995

Prevention by permanent barrier
Dulguerov – Arch Otolaryngol HNS, 1999



Local hypersensitivity to cholinergic drugs

Wilson – Clin Sci, 1936
Freedberg – J Clin Invest, 1948
Glaister – Brit Med J, 1958

Stimulation
Ross – Laryngoscope, 1970

Anesthesia
Freedberg – J Clin Invest, 1948
Glaister – Brit Med J, 1958

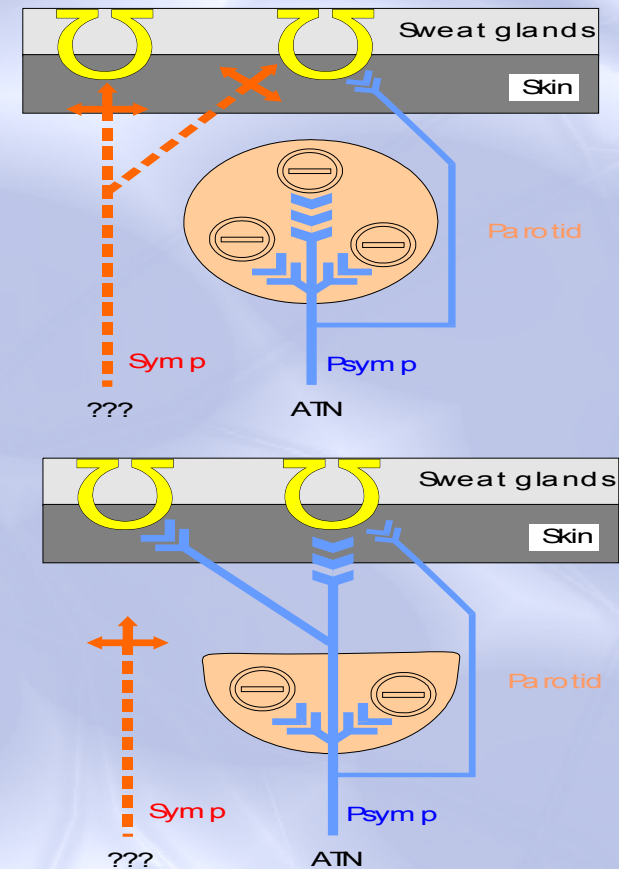
The etiology of Frey syndrome is the sympathetic denervation of sweat glands

Trauma to the parotid area

- parotidectomy
- mandibular surgery
- neck dissection
- drainage of abscess
- blunt (mandibular fractures) and penetrating trauma

Surgery or lesions of the cervical sympathetic chain

**THE KEY ELEMENT IS THE
DENERVATION OF THE SWEAT
GLANDS, THUS A LESION OF THE
SYMPATHETIC FIBERS**



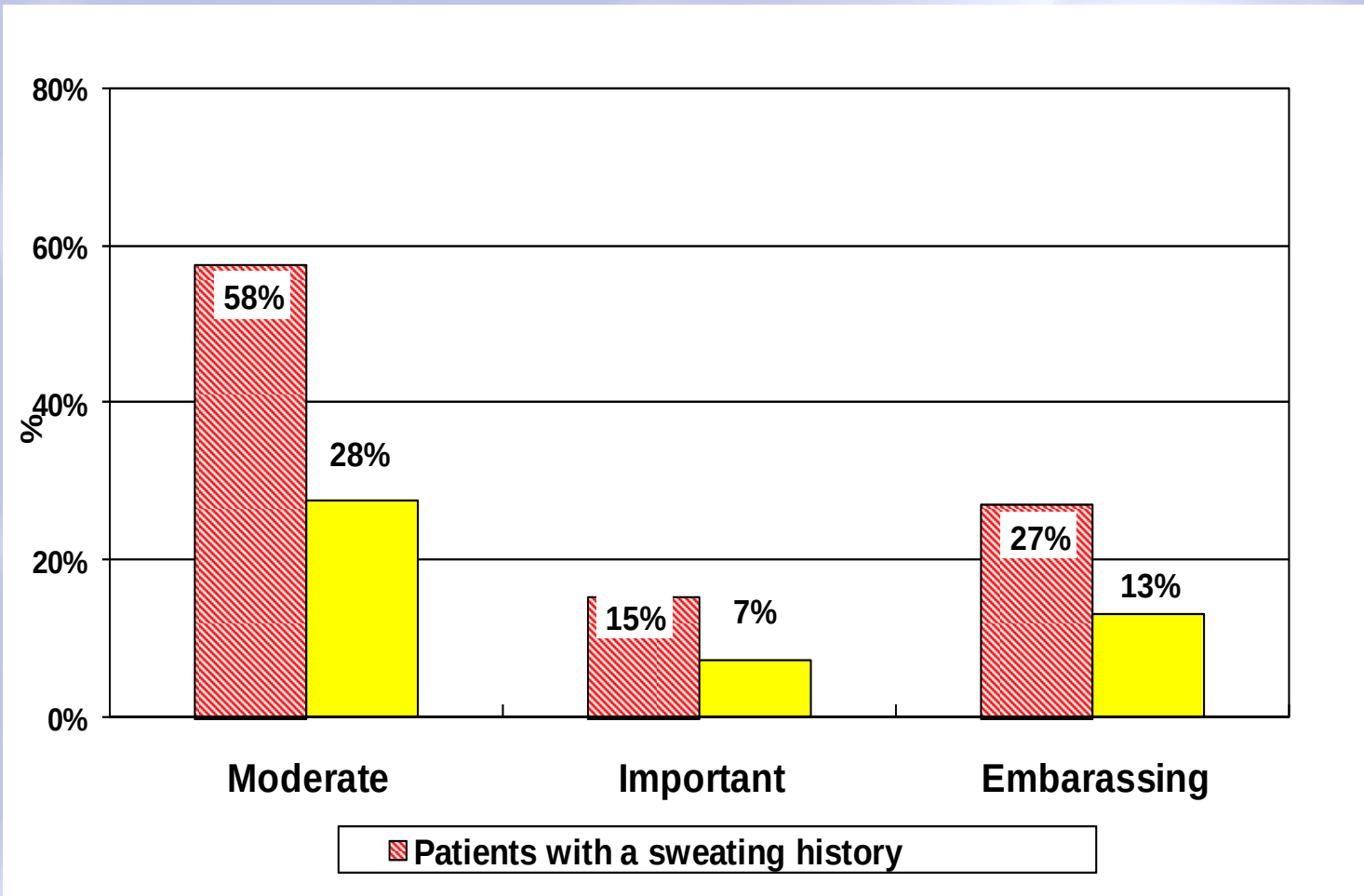
Frey syndrome is a frequent complication of parotidectomy

Author	Year	Number of patients	Incidence of clinical Frey's syndrome (%)	Incidence of objective Frey's syndrome (%)
Laage-Hellman [74]	1958	123	62	98
Kornblut et al. [69]	1974	35	43	97
Gordon and Fiddian [53]	1976	50	34	100
Farrell and Kalnins [39] ^a	1991	21	14	43
Yu and Hamilton [146]	1992	35	6	14
Allison and Rappaport [2]	1993	35	83	87
Linder et al. [88]	1997	26	43	96
Nosan et al. [104]	1991	23	44	70
Dulguerov et al. [35]	1999	24	53	76
Laskawi et al. [84]	1998	81	63	100
Cavalot [16]	2000	86	47	86

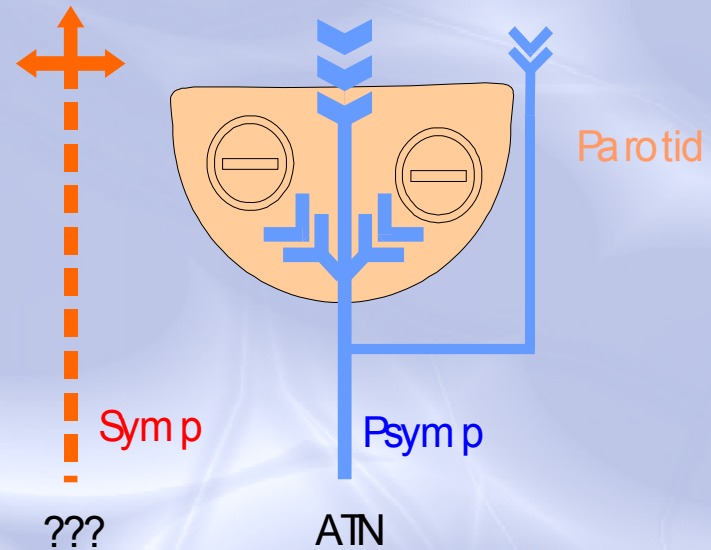
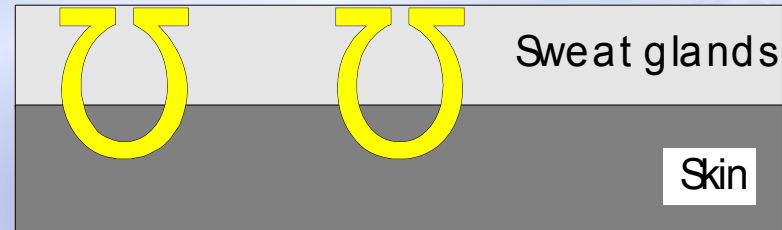
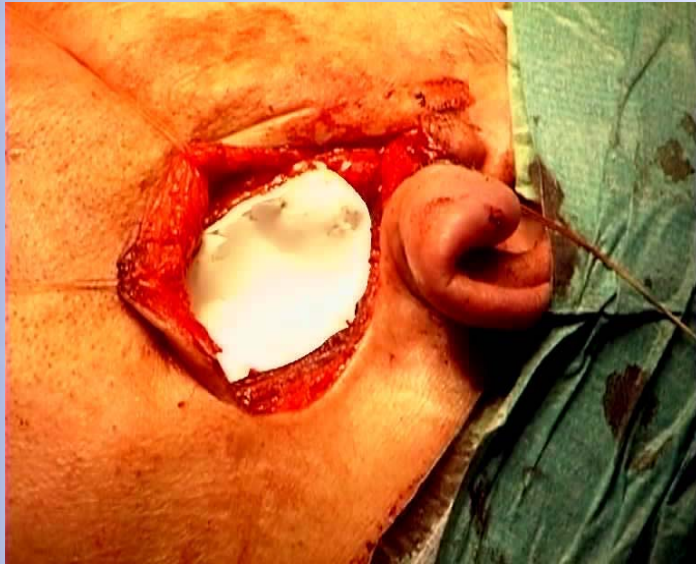
45%

90%

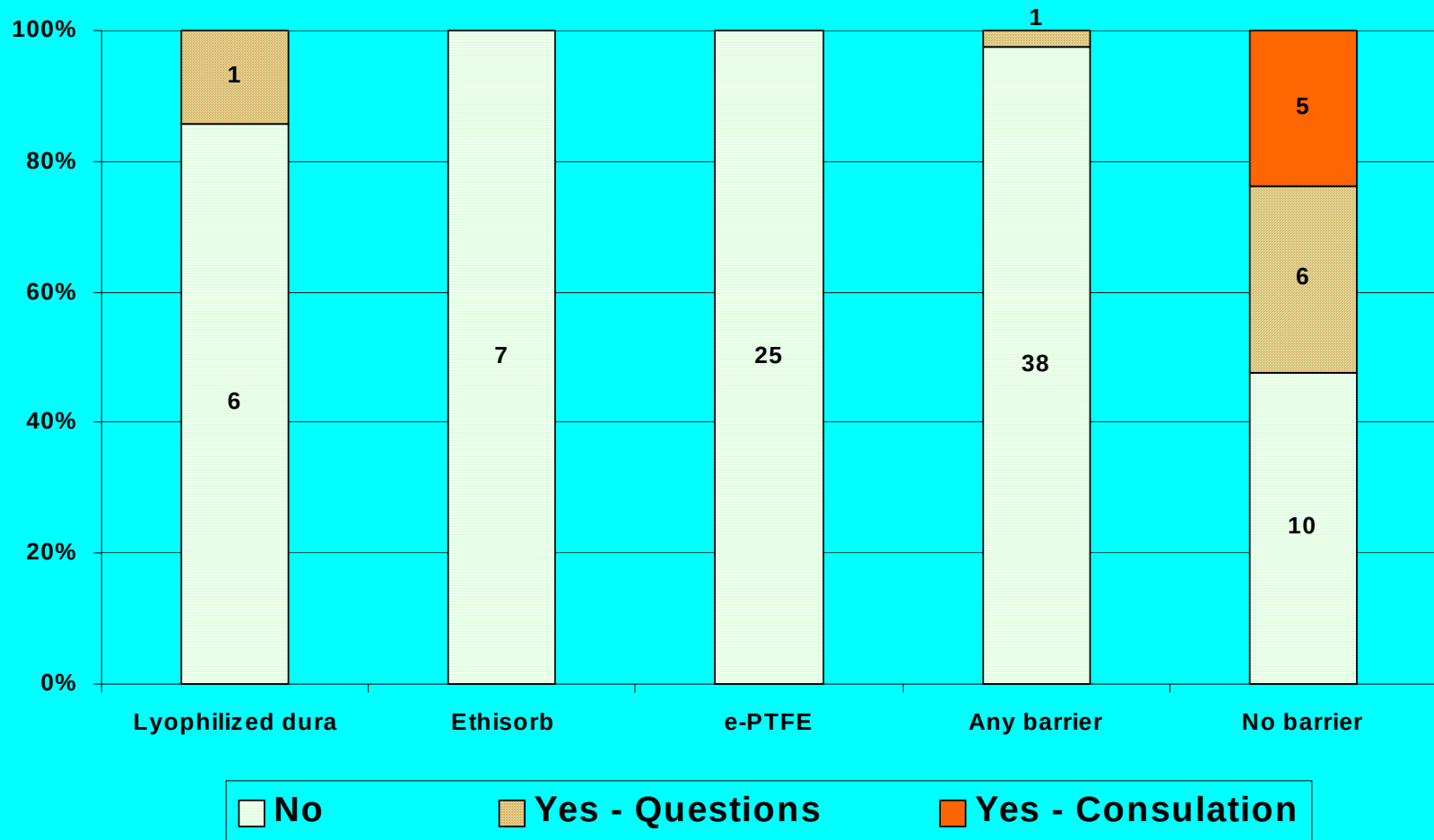
Severity of sweating is important in about 40%



Prevention of Frey syndrome

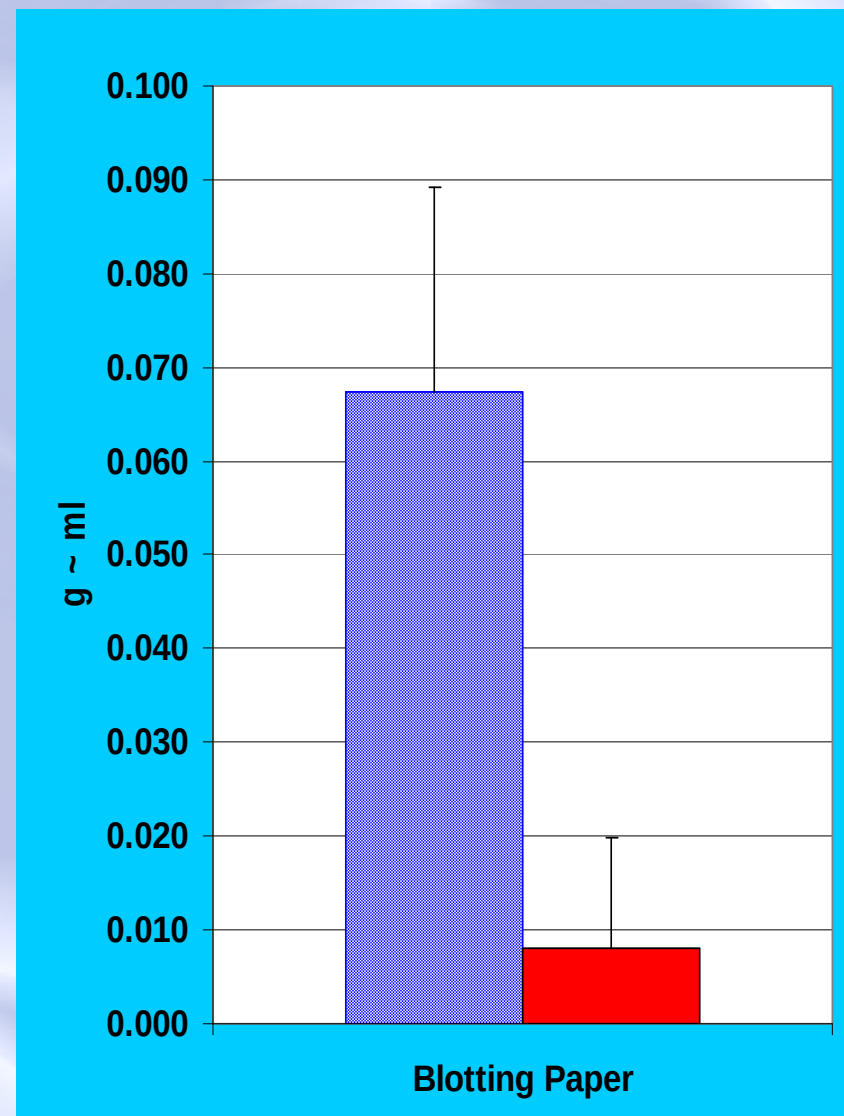
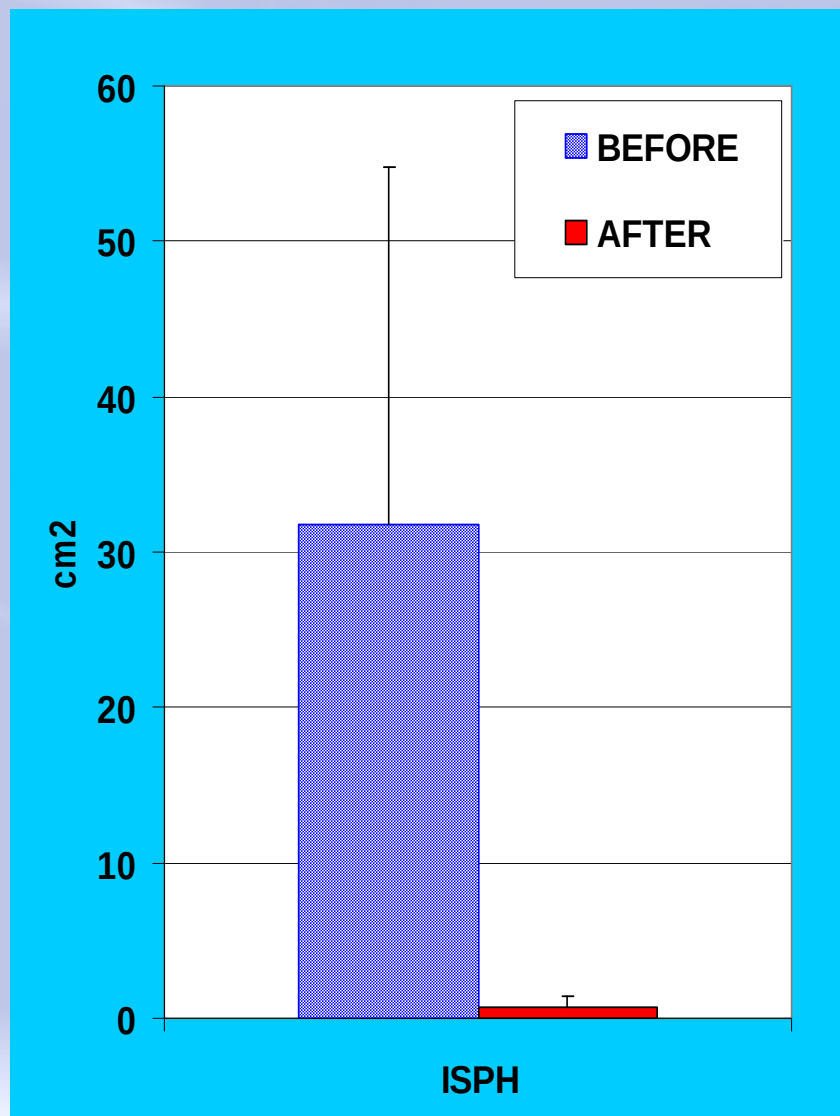


Clinical Frey syndrome



Author	Year	Number of patients	Prevention technique	Evaluation	Minimal follow-up	Incidence with prevention	Incidence without prevention	p (χ^2)
Kidd [79]	1955		Nerve section	History	?	20%	24%	0.7
Singleton [146]	1980	164	Thick flap	History	8 weeks	3%	12.5%	0.02
Kornblut et al. [81] [82]			SCM	History	1-6 years	43%	23%	0.07
				Minor	1-6 years	97%	94%	0.5
Casler [21]	1991	16 /	SCM	History	2 years	12.5%	47%	< 0.01
Kim [80]	1999	9 / 10	SCM	History	1 year	22%	50%	
Sood [151]	1999	11 / 11	SCM	Minor	1 year	18%	82%	<0.01
Gooden [59]	2001	13 / 13	SCM	Minor	1 year	31%	23%	
Kerawala [78]	2002	21 / 15	SCM	Minor	1 year	90%	73%	0.21
Filho [49]	2004	24 / 19	SCM	Minor	1 year	0%	37%	< 0.01
Casler [21]	1991	16 /	SMAS	History	2 years	0%	47%	< 0.01
Yu & Hamilton [170]	1992	20	SMAS	Minor	2.5 years*	15%	---	---
Allison & Rappaport [3]	1993	91/35	SMAS	History	1 year	1%	83%	<0.01
		79/30		Minor	1 year	2%	87%	<0.01
Belli [11]	1996	45 / 35	SMAS	History	2 years	40%	57%	0.13
Ahmed [2]	1999	24 / 23	Temporal fascia	Minor	1 year	17%	70%	<0.01
Bonanno	2000	160	SMAS	Minor	1 year	0%	---	---
Taylor [156]	2000	15 / 13	SMAS	Minor	0.8 years	73%	54%	0.14
Dulguerov [43]	1999	/ 24	GoreTex / Lyophilized dura	ISPH	1 year	10%	76%	
Govindaraj [62]	2001	15 / 15	Alloderm	Minor	0.5 years	0%	40%	<0.01
Sinha [147]	2003	10 / 10	Alloderm	Minor	1 year	20%	80%	<0.05

Frey syndrome is treatable by Botox intradermal injections



The incidence of Frey syndrome after parotidectomy is 40% for the clinical and 90% for the objective evaluation.

A subcutaneous barrier placed as prevention decreases this incidence to 2% for the clinical and 10% for the objective evaluation.

Botulinum toxin type A is an efficient, well tolerated, uncomplicated treatment of Frey syndrome