Communication and Swallowing Disorders

Tamer Mesallam MD, PhD,
Associate Professor of Phoniatrics
Communication and Swallowing Disorders Unit (CSDU)
ORL/HNS Department
College of Medicine
King Saud University

Objectives

- Understand physiology of communication.

- Recall classifications of communication and swallowing disorders.

- Differentiate between different causes of communication and swallowing disorders.

- Understanding the assessment and management of these disorders.

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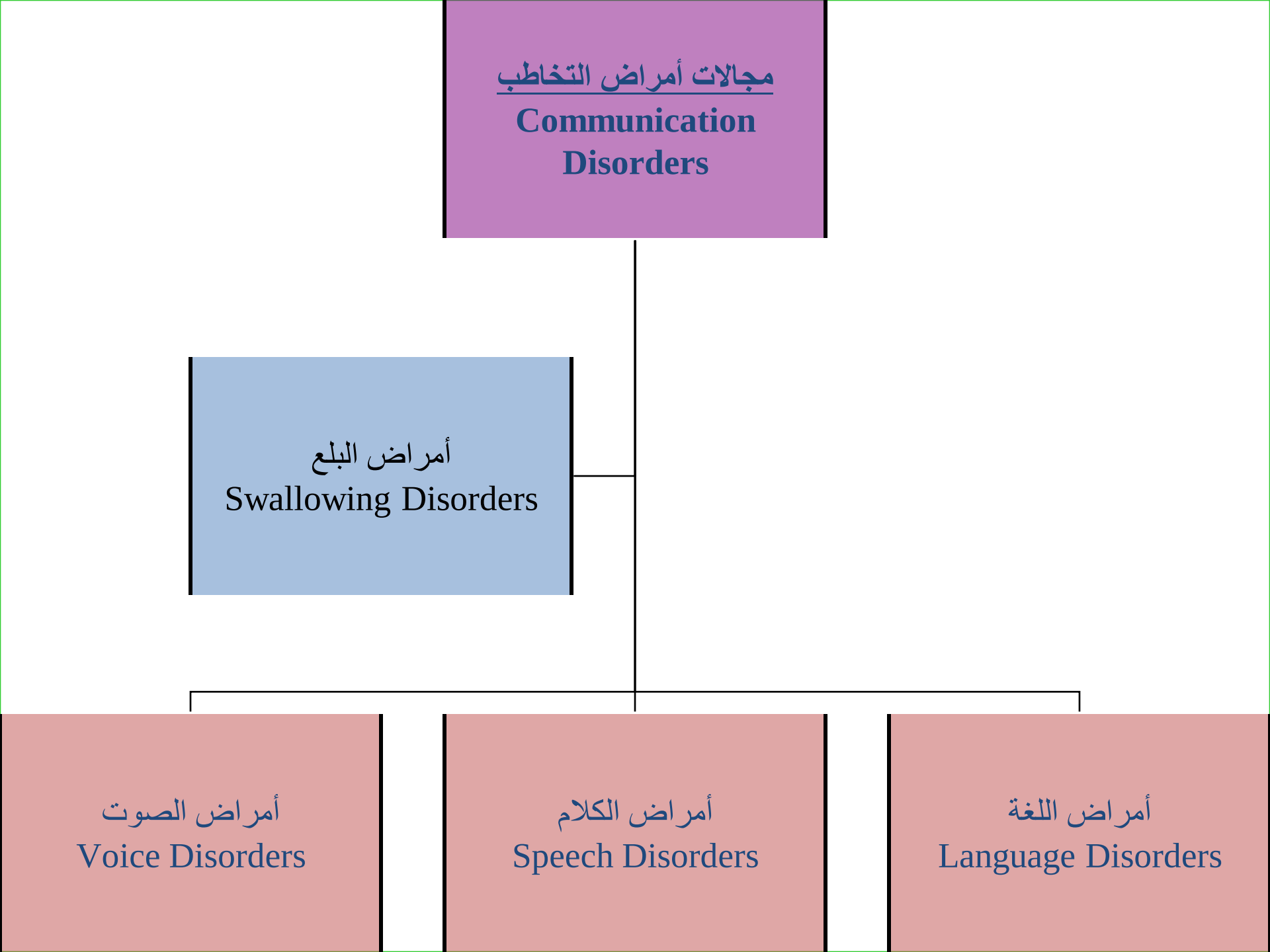
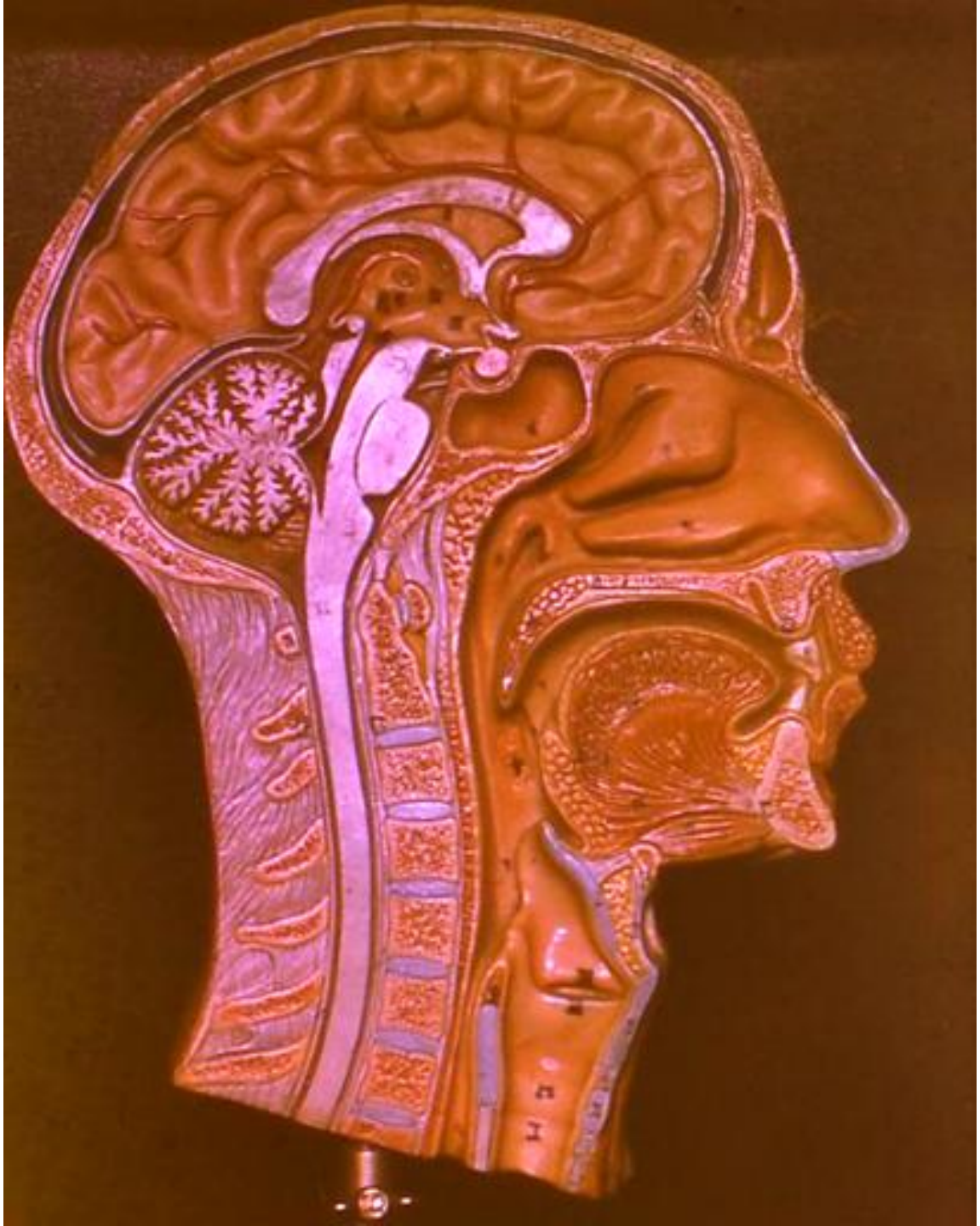

مجالات أمراض التخاطب
Communication Disorders
أمراض البلع
Swallowing Disorders
أمراض الصوت
Voice Disorders
أمراض الكلام
Speech Disorders
أمراض اللغة
Language Disorders

Symbolization

Articulation

Phonation

Respiration



LANGUAGE

SPEECH

VOICE

Language

A symbolic arbitrary system relating sounds to meaning.

Speech

A neuro-muscular process whereby language is uttered. It includes the coordination of respiration, phonation, articulation, resonation and prosody.

Voice

The result of vibration of the true vocal folds using the expired air.

Swallowing

The process of successful passage of food and drinks from the mouth through pharynx and esophagus into the stomach.

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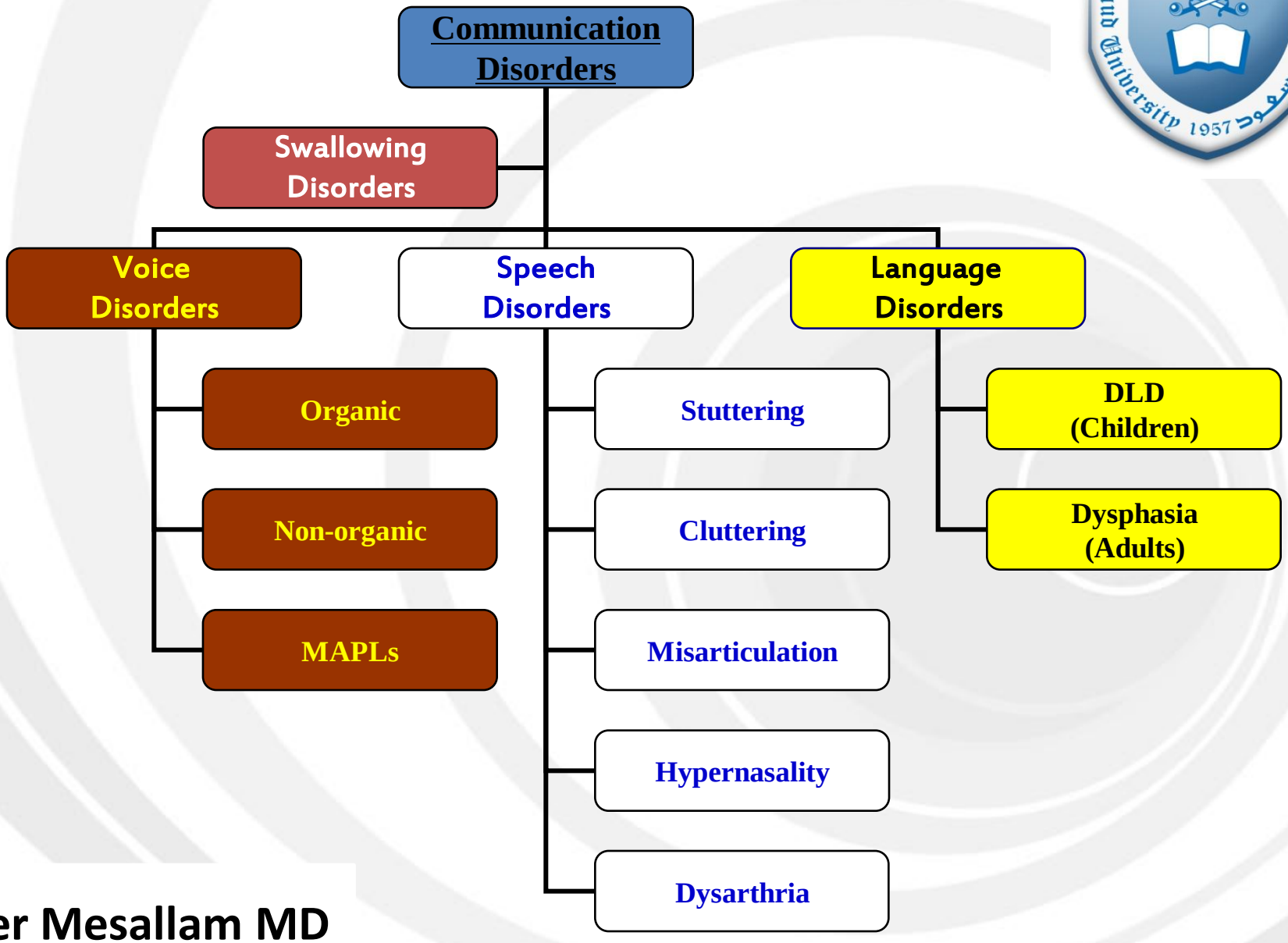

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Language Disorders

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I. Language Disorders:

[1] Delayed Language Development (DLD)

[2] Dysphasia

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[1] Delayed Language Development (DLD)

<u>Definition of DLD:</u>

Delay or failure to acquire language matched with age.

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Stages of normal language development

- 2-4 months; Babbling
- 6 months; Vocal play
- 9 mo-1 year; 1st word
- 1-1/2 years; 20 words
- 2 years; 200 words, 2 word sentence
- 3 years; 2000 words, 3 word sentence
- 4 years; 4 word sentence
- 5-7 years; Full maturation of all language modalities.

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Pre-requisites of normal language development

- Intact brain functions (conceptual, motoric and cognitive abilities).
- Intact sensory channels;
  - Auditory
  - Visual
  - Tactile
  - Kinesthetic
- Intact psyche.
- Stimulating environment.

Etiology of delayed language development

- Brain damage

  - Diffuse subcortical lesion (M.R.).
  - Localized brain damage with motor handicap (BDMH).
  - Minimal brain damage (ADHD).

- Sensory deprivation.
  - Hearing impairment
    - Conductive
    - Sensorineural
    - Mixed
    - Central auditory processing disorder
  - Visual impairment

Etiology of delayed language development (Cont.)

- Psychiatric illness
  - Autism.
  - Autism Spectrum Disorder (ASD).
- Environmental deprivation
- Idiopathic (Specific Language Impairment).

Assessment of language development

I. History taking.
II. Physical examination.
III. Investigations:
    Psychometry (IQ).
    Audiometry.
    Brian Imaging
    EEG
    Ophthalmological consultation

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Management of DLD:

- Early detection.
- Providing the suitable aid
  - Hearing (HA or CI).
  - Visual Aid.
  - Physiotherapy
- Family counseling.
- Direct language therapy (Individual- group).
- Medications (Autism and ADHD).

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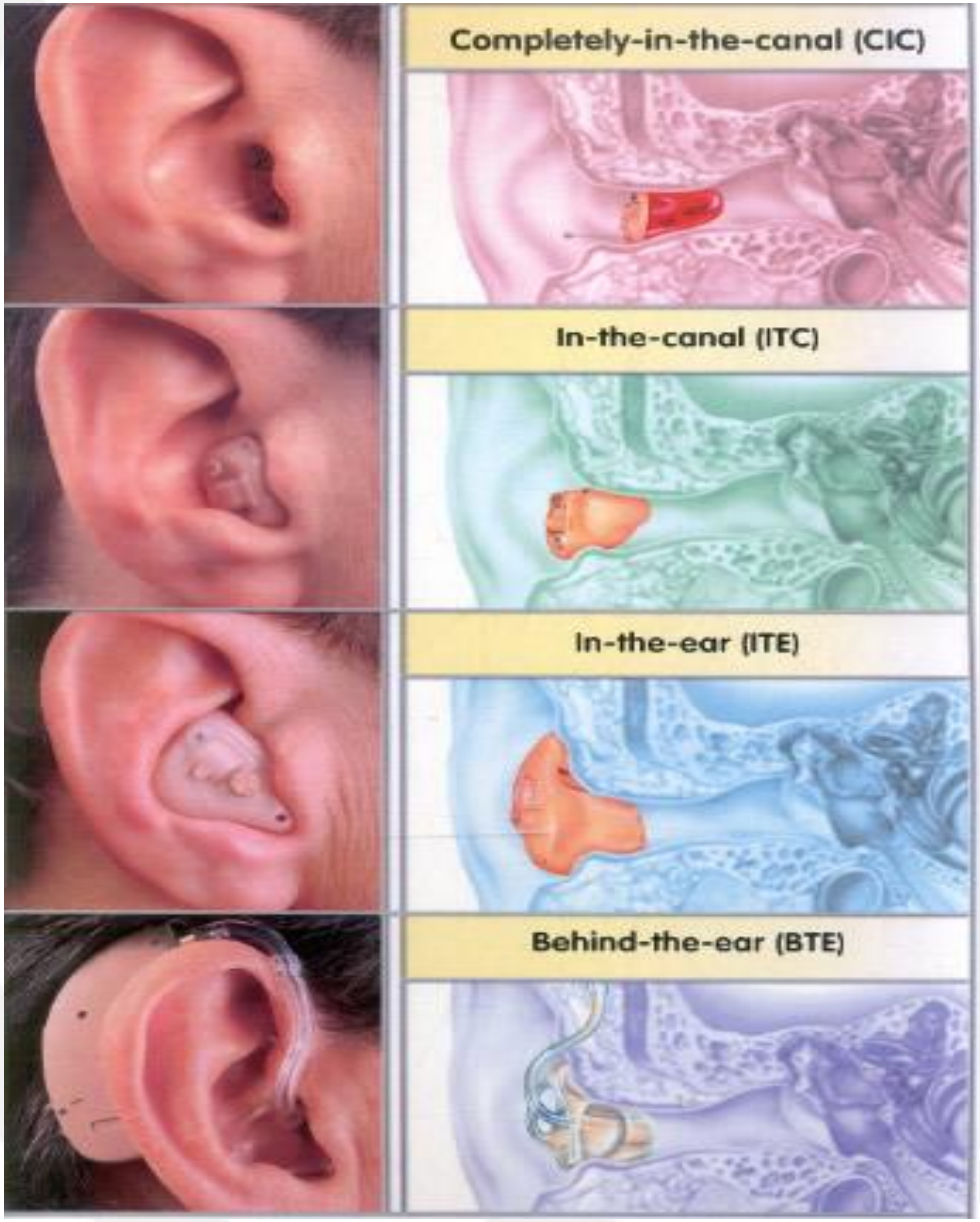

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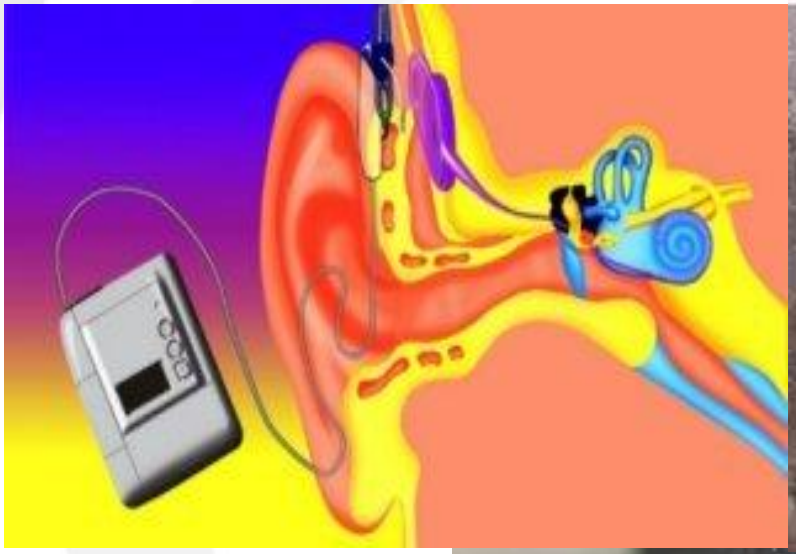

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I. Language disorders:

[2] Dysphasia:

Definition:

Language deterioration after its full development due to brain insult: infarction, hemorrhage, atrophy, etc

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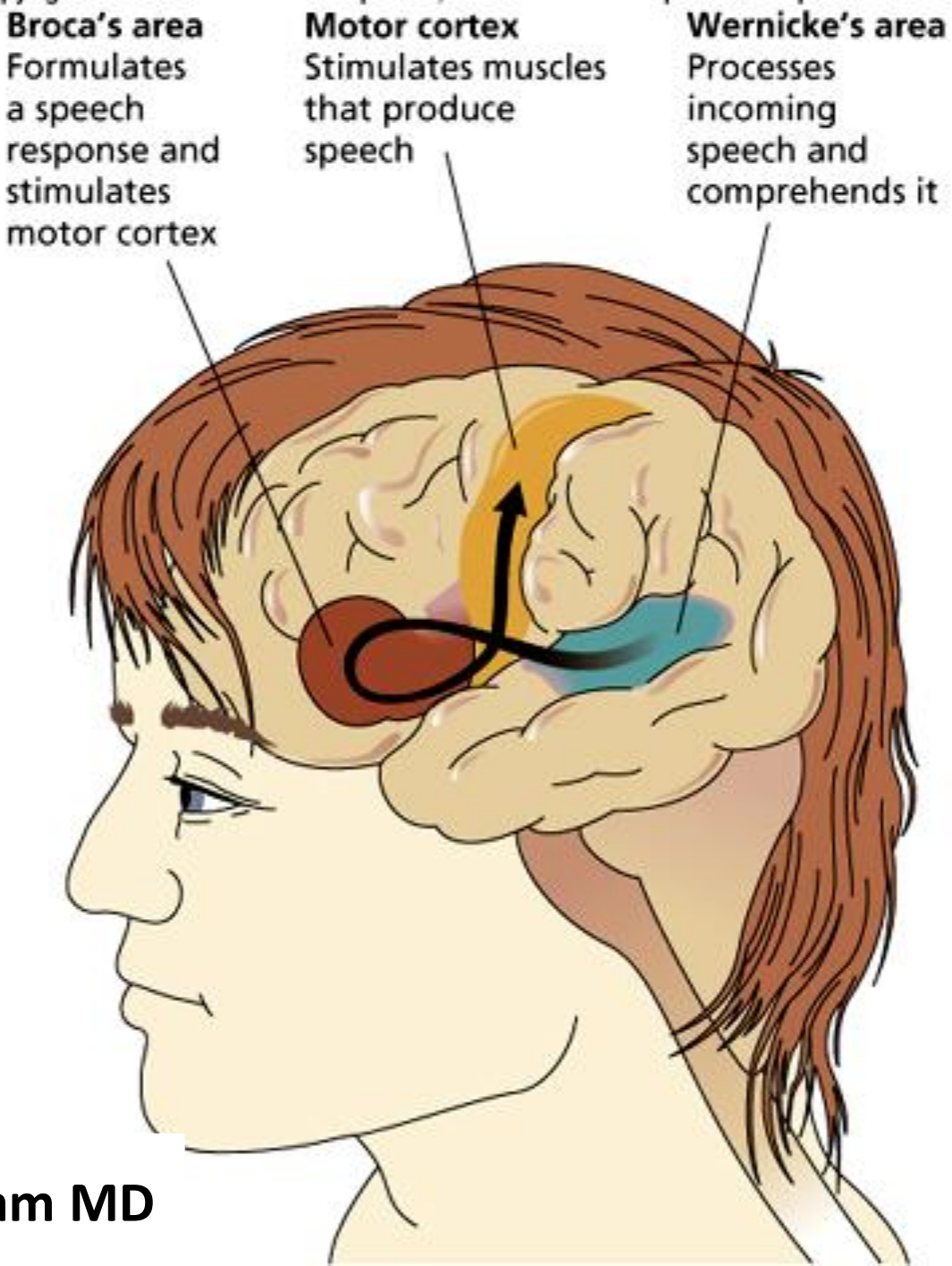

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Etiology;

- ❑CVA
- ❑Neoplastic
- ❑Traumatic
- ❑Inflammatory
- ❑Degenerative
- ❑Metabolic
- ❑Poisoning

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Types of dysphasia:

1. Expressive.
2. Receptive.
3. Mixed predominantly expressive.
4. Mixed predominantly receptive.
5. Global.

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Assessment of Dysphasia

I. History taking.
II. Physical examination: … , neurological exam.
III. Investigations:
- CT / MRI brain.
- Dysphasia test.
- Psychometry (IQ).
- Audiometry.

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Management of Dysphasia

- Management of the cause.
- Physical rehabilitation (Physiotherapy).
- Family counseling.
- Language therapy.
- Alternative and augmentative communication.

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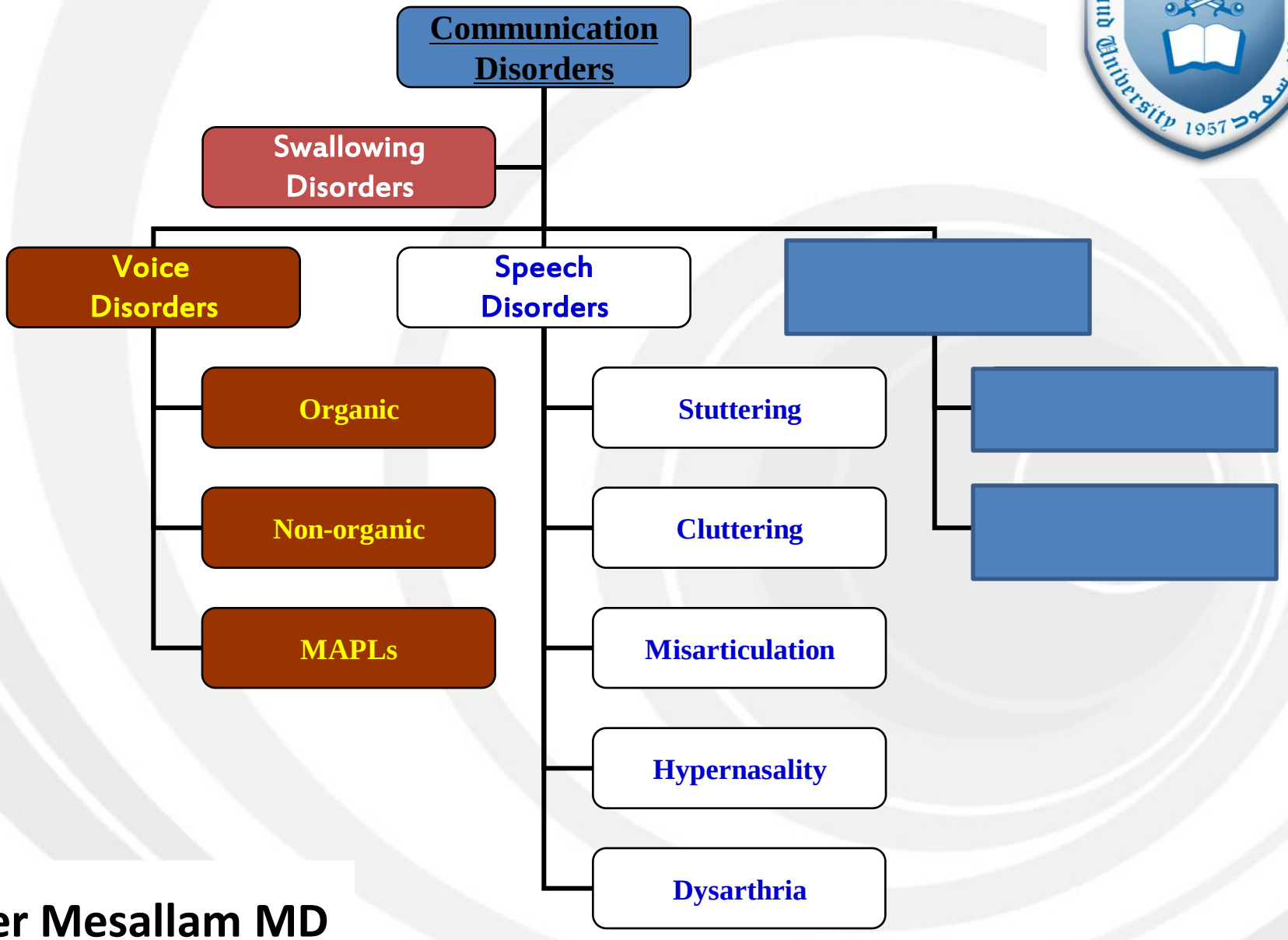

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Speech Disorders

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II. Speech disorders:

1. Dyslalia (Misarticulation)
2. Stuttering
3. Dysarthria
4. Hypernasality

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II. Speech disorders:

1. Dyslalia (Misarticulation):

Definition:

Faulty articulation of one or more of speech sounds not appropriate for age.

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Types of dyslalia:

A) Sigmatism (/s/ defect):-
- Interdental sigmatism.
- Lateral sigmatism.
- Pharyngeal sigmatism.

B) Back-to-front dyslalia:-

/k/	/t/
/g/	/d/

C) Rotacism (/r/ defect).

D) Voiced-to-nonvoiced dyslalia:-

/g/	/k/
/d/	/t/
/z/	/s/ etc…

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Assessment of dyslalia:

I. History taking.
II. Physical examination: … , tongue, …
III. Investigations:
- Audio recording.
- Articulation test.
- Psychometry (IQ).
- Audiometry.

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Management of dyslalia:

- Treatment of the cause:
  - . Tongue tie.
  - . Dental anomalies.
  - . Hearing aids
- Speech therapy.

II. Speech disorders:

2. Stuttering:

Definition:

The intraphonemic disruptions resulting in sound and syllable repetitions, sound prolongations, and blocks.

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Normal dysfluency:

- 3-6 years.
- Only repetitions.
- No associated muscular activity.
- Not aware.

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Incidence of stuttering: 1%.

Onset:

- Earliest = 18 months.
- Latest = 13 years.

Epidemiology:

- more in families with history of stuttering.
- can occur in mentally retarded.
- very rare in the hearing impaired.

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Gender ratio:

4 : 1 (male : female)

Theories of Stuttering:

The exact cause is unknown.

- Organic theory.
- Neurosis theory.
- Learning theory.

Assessment of stuttering:

I. History taking.
II. Physical examination: APA, VPA, …
III. Investigations:
- Audio and video recording.
- Stuttering severity (eg SSI).
- Articulation test.
- Psychometry (IQ).

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Auditory Perceptual Analysis (APA):

A. Core behaviors:

- Intraphonemic disruption.
- Repetitions.
- Prolongations.
- Blocks.

B. Secondary reactions:

- Muscular activity and struggle.
- Interjection.
- Word substitutions and circumlocution.

C. Concomitant reactions:

- Fear.
- Breathing (antagonism, interruption, prolongation, cessation, …).
- Eye contact.
- Skin pallor/flushing.

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Management of stuttering:

- Family and patient counseling.

- Speech therapy:
  - a. Indirect therapy: if not aware.
  - b. Direct therapy: if aware.

II. Speech disorders:

3. Dysarthria:

Definition:

Any combination of disorders of respiration, phonation, articulation, resonance, and prosody, that may result from a neuromuscular disorder.

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Types of dysarthria:

1. *Flaccid dysarthria:*
   - Lesion: lower motor neuron level.
   - Communication:
     * breathy phonation.
     * hypernasality.

2. *Spastic dysarthria:*
   - Lesion: upper motor neuron level.
   - Communication:
     * strained strangled phonation.
     * labored breathing.

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Types of dysarthria (cont.):

3. *Ataxic dysarthria:*

- Lesion: cerebellum level.
- Communication:
  * increased equal stresses.
  * irregular articulatory breakdown.

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Types of dysarthria (cont.):

4. *Dyskinetic dysarthria:*

- Lesion: basal ganglia level.

A. Hypokinetic type (Parkinsonism):

* breathy phonation.
* rapid rate.
* short rushes of speech with final decay.

B. Hyperkinetic type:

i. Quick hyperkinetic (Chorea):

* variable rate and loudness.

ii. Slow hyperkinetic (Athetosis):

* slow rate.

Types of dysarthria (cont.):

5. *Mixed dysarthria:*

- may the most common.
- Examples:
  * Motor neuron disease ….Flaccid + Spastic.
  * Multiple sclerosis …….. Ataxic + Spastic.
  * Wilson's disease ……. Ataxic + Spastic + Hypokinetic.

Assessment of dysarthria:

I. History taking.
II. Physical examination: … , mouth, palate, … , neurological exam, …
III. Investigations:
- Audio recording.
- Fiberoptic nasopharyngolaryngoscopy.
- CT/MRI brain
- Dysphasia test.
- Psychometry (IQ).
- Articulation test.
- Audiometry.
- Nasometry.
- MDVP.
- Aerodynamics (Aerophone II).

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Management of dysarthria:

Individualized:

- ❑ Management of the cause.
- ❑ Patient counseling.
- ❑ Communicative therapy:
  - * Articulation.
  - * Phonation.
  - * Resonance.
  - * Respiration.
  - * Prosody.
- ❑ Alternative and augmentative communication.

II. Speech disorders:

4. Hypernasality:

Definition:

Faulty contamination of the speech signal by the addition of nasal noise. It results from velopharyngeal dysfunction (VPD).

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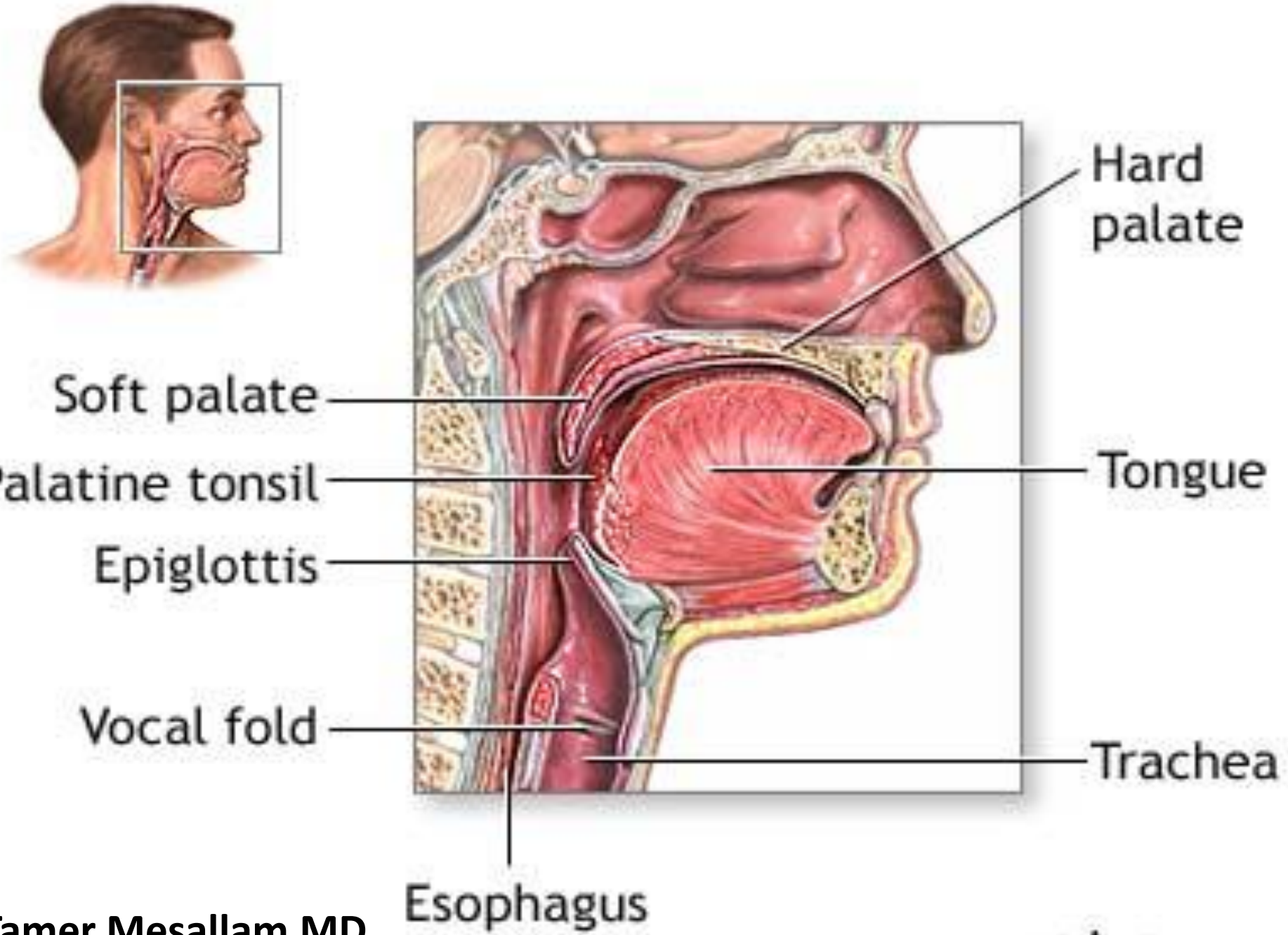

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A.D.A.M.

Velum: At rest and during speech

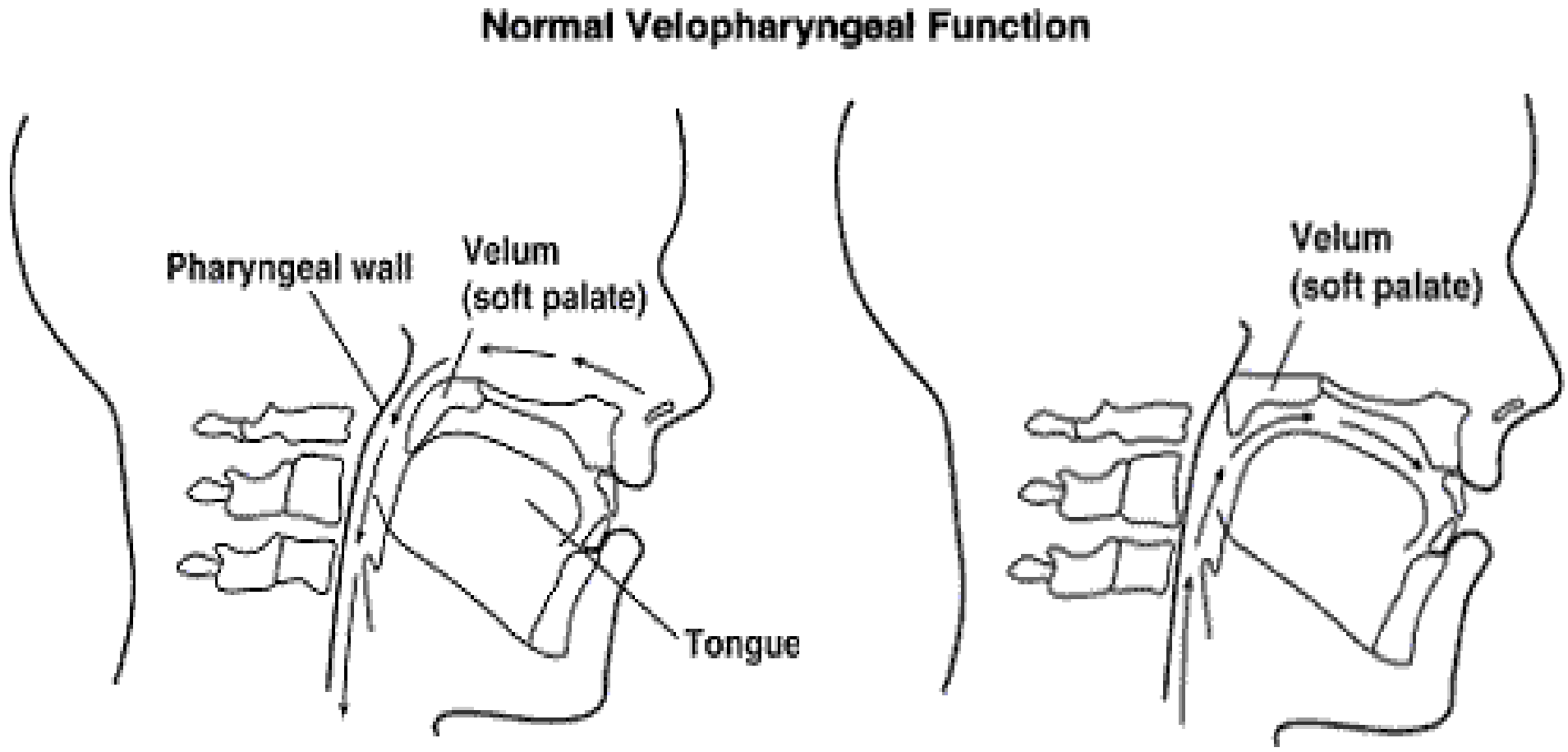

Fig. 1 Velum at rest.

Fig. 2 Velum during speech.

Causes of hypernasality:

I. Organic:

1.Structural: (VP Insufficiency)

a) Congenital:

- Overt cleft palate.
- Submucous cleft palate.
- Non-cleft causes:
  - . Congenital short palate.
  - . Congenital deep pharynx.

b) Acquired:

- Adenotonsillectomy.
- Palatal trauma.
- Tumors of the palate & pharynx.

2. Neurogenic: (VP Incompetence)

- Palatal upper motor neuron lesion.
- Palatal lower motor neuron lesion.

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Causes of hypernasality (cont.):

II. Non-organic (Functional) VP Mis-learning:

- Faulty speech habits.
- Mental retardation.
- Hearing impairment.
- Post-tonsillectomy pain.

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Effects of VPD:

- Feeding problems: nasal regurgitation.
- Psychosocial problems.
- Communicative problems:
    . Speech: hypernasality.
    . Language: DLD.
    . Voice: hyper or hypofunction.

Assessment of hypernasality (VPD)

- ❑Parent interview
- ❑Perceptual

- Simple tests:
  - . Gutzman's (a/i) test.
  - . Czermak's (cold mirror) test.
- Resonance
- Articulation
- Nasal air emission
- Voice

- ❑Intra-oral evaluation
- ❑Instrumental: - Nasopharyngoscopy
  - Nasometry

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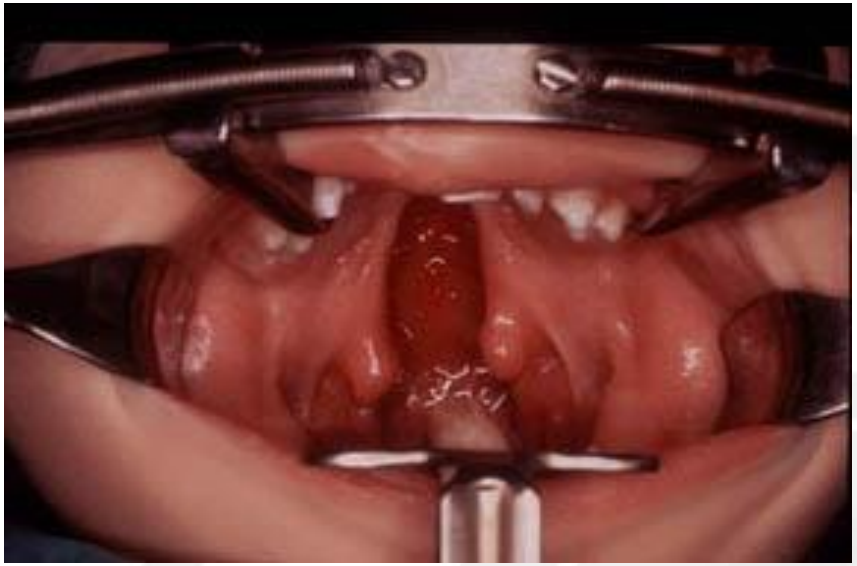

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Submucus Cleft

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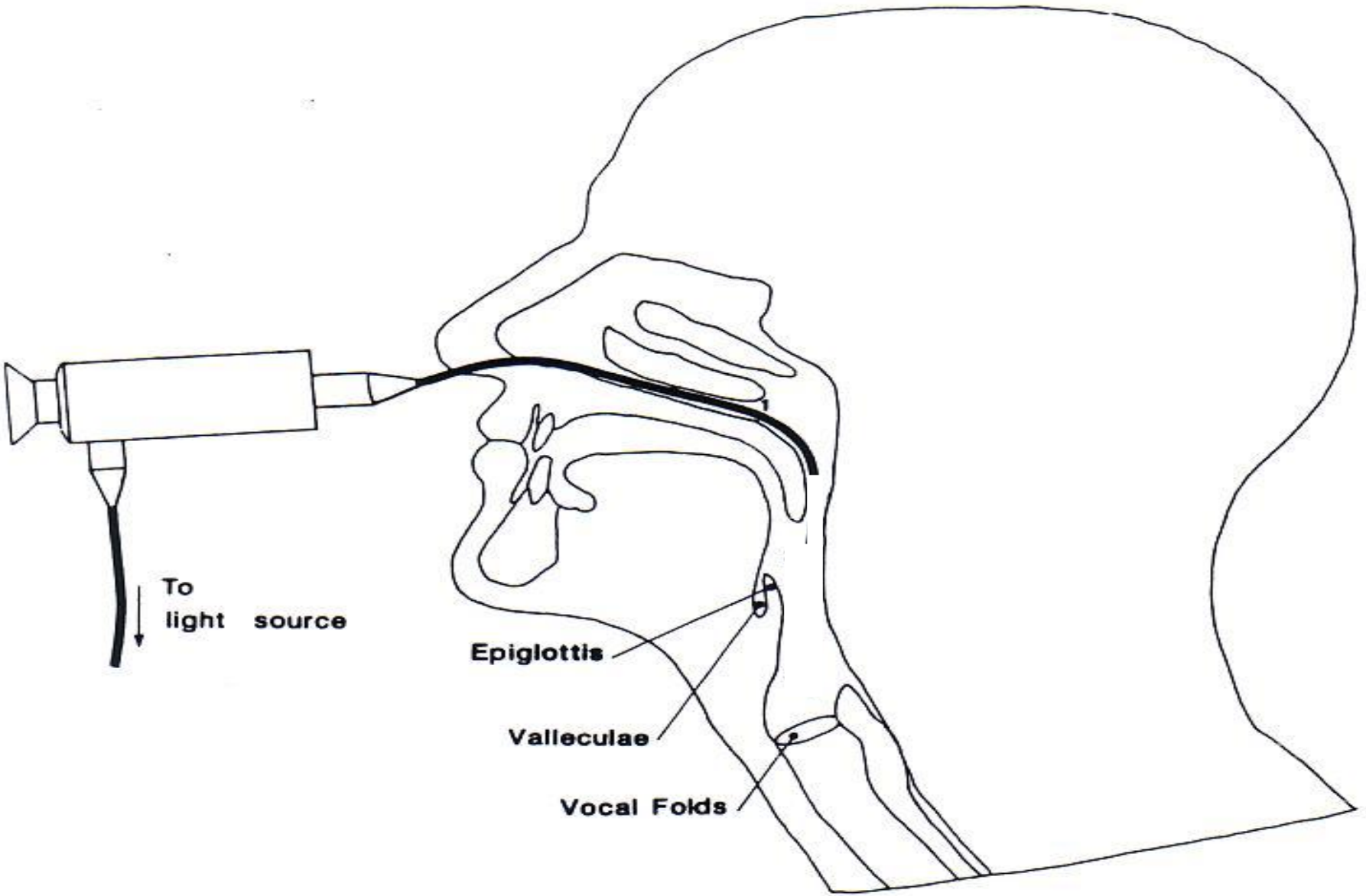
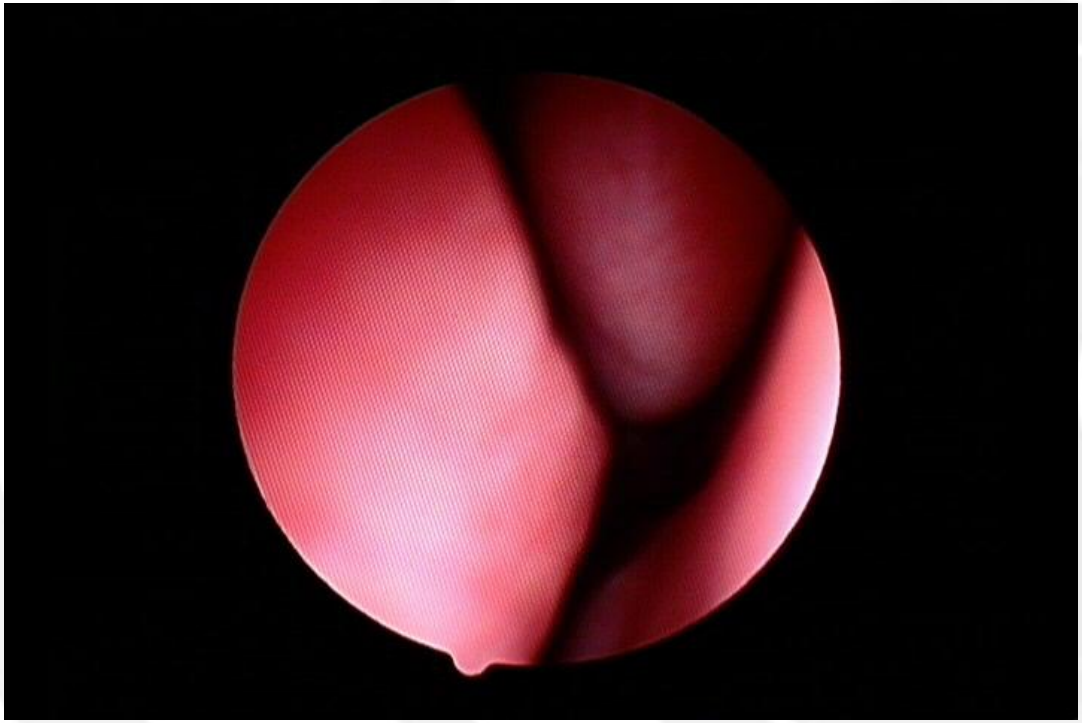
Flexible nasopharyngoscopy



Normal closure



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VPD

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Nasometry

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Management of VPD

- Multidisciplinary team
- **Family counseling**
- Management of feeding problem
- Management of otological and audiological problems
- Surgical intervention
- Orthodontic intervention
- Phoniatric intervention (language, speech, voice)

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Treatment Decision

- Velopharyngeal insufficiency
  - surgery (speech therapy post-op)

- Velopharyngeal incompetence
  - surgery (speech therapy post-op)
  - prosthetic devices
  - speech therapy

- Velopharyngeal mislearning
  - speech therapy

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Surgery

- Pharyngeal flap.
- Sphincter-platoplasty
- Post-pharyngeal wall augmentation.

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Pharyngeal flap

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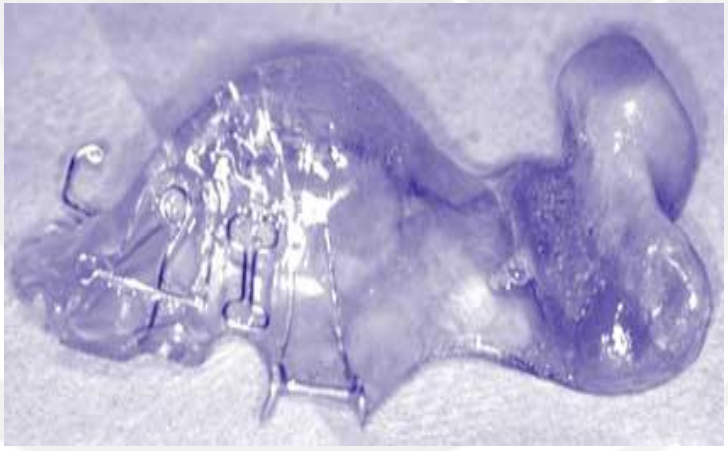
Prosthetic Devices

- Palatal lift: to raise the velum when there is poor velar movement (i.e. dysartheria)

- Platal obturator: to occlude an open cleft or fistula



- speech bulb: to occlude nasopharynx

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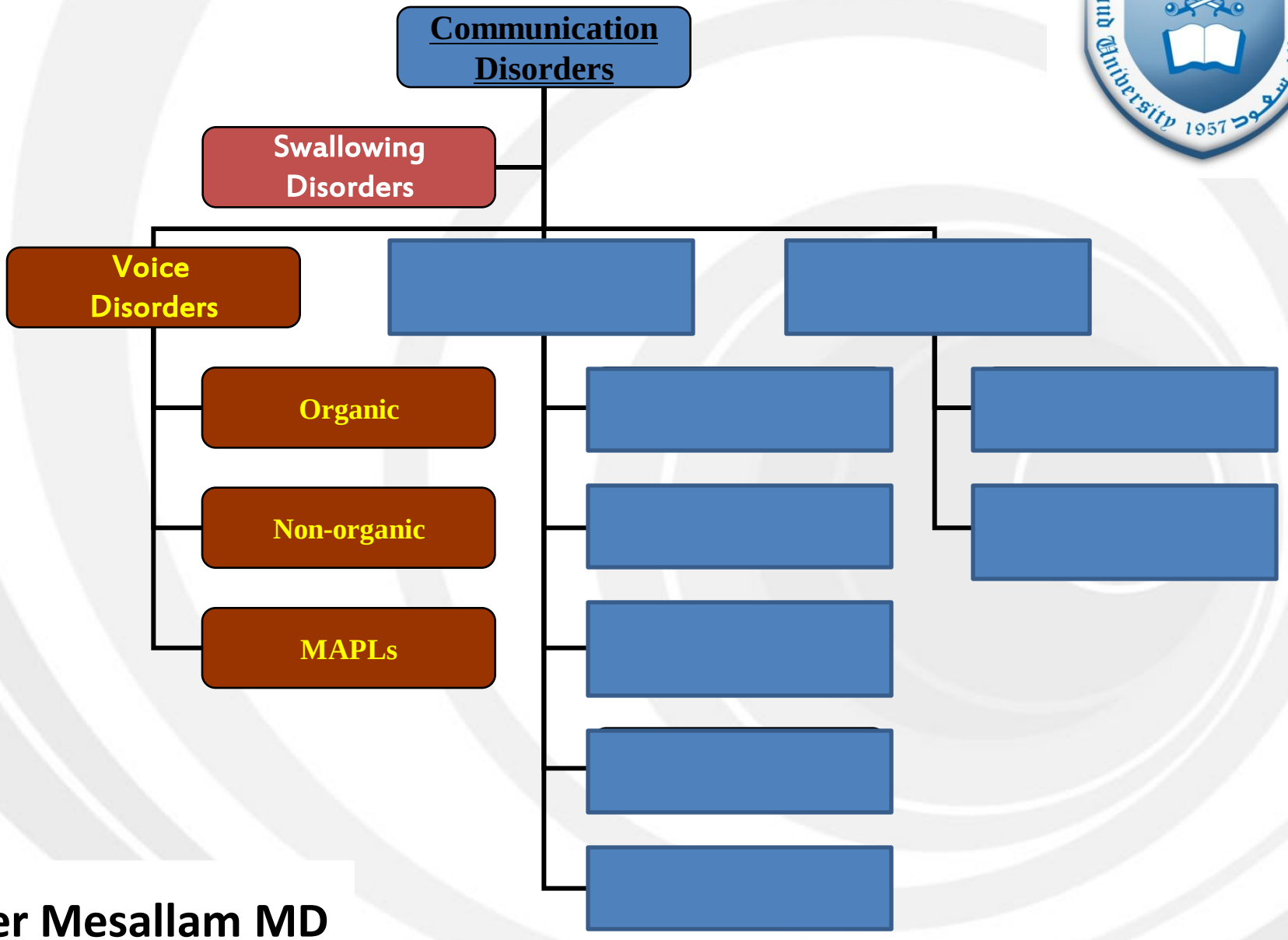

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Voice Disorders

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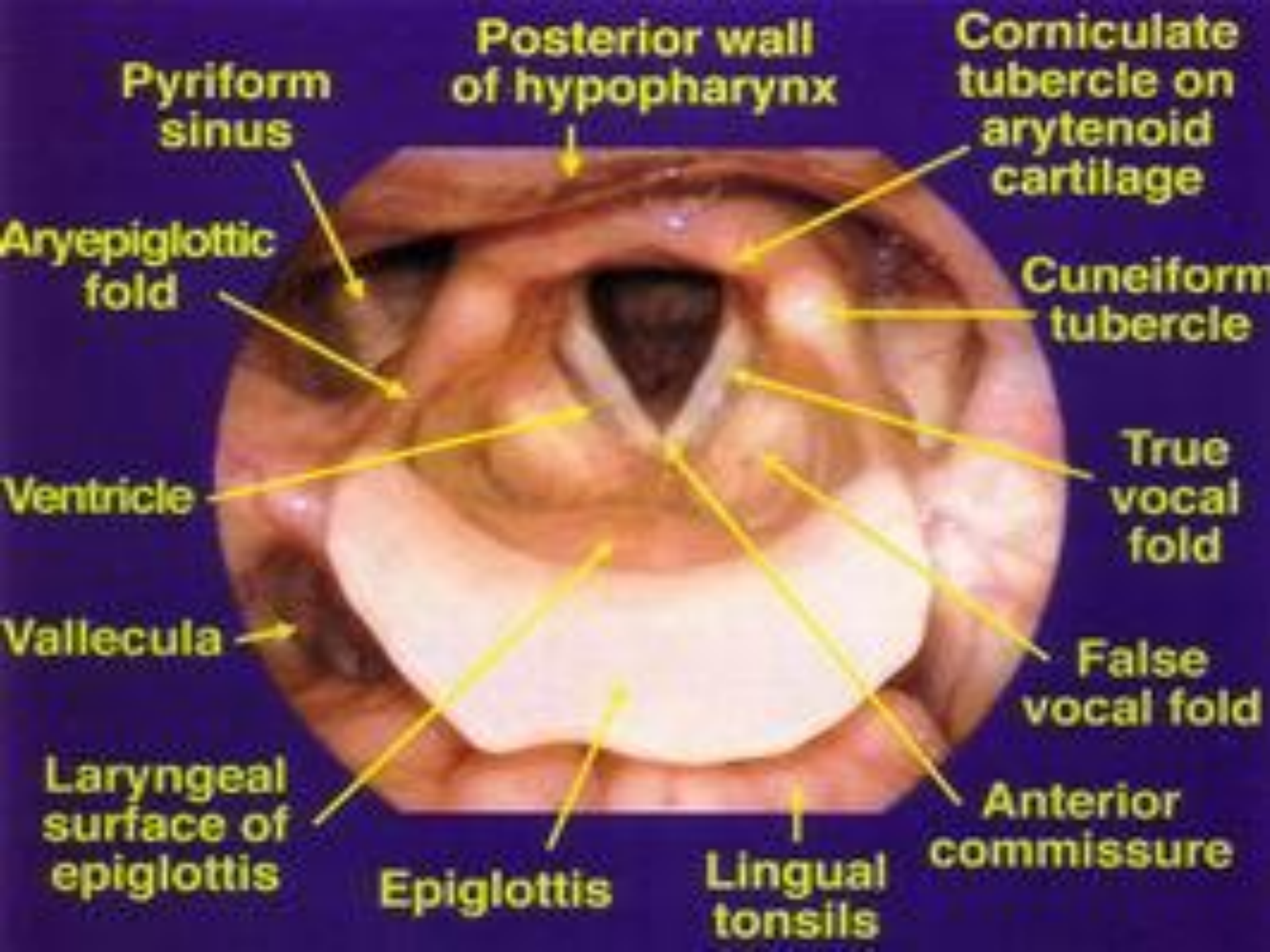
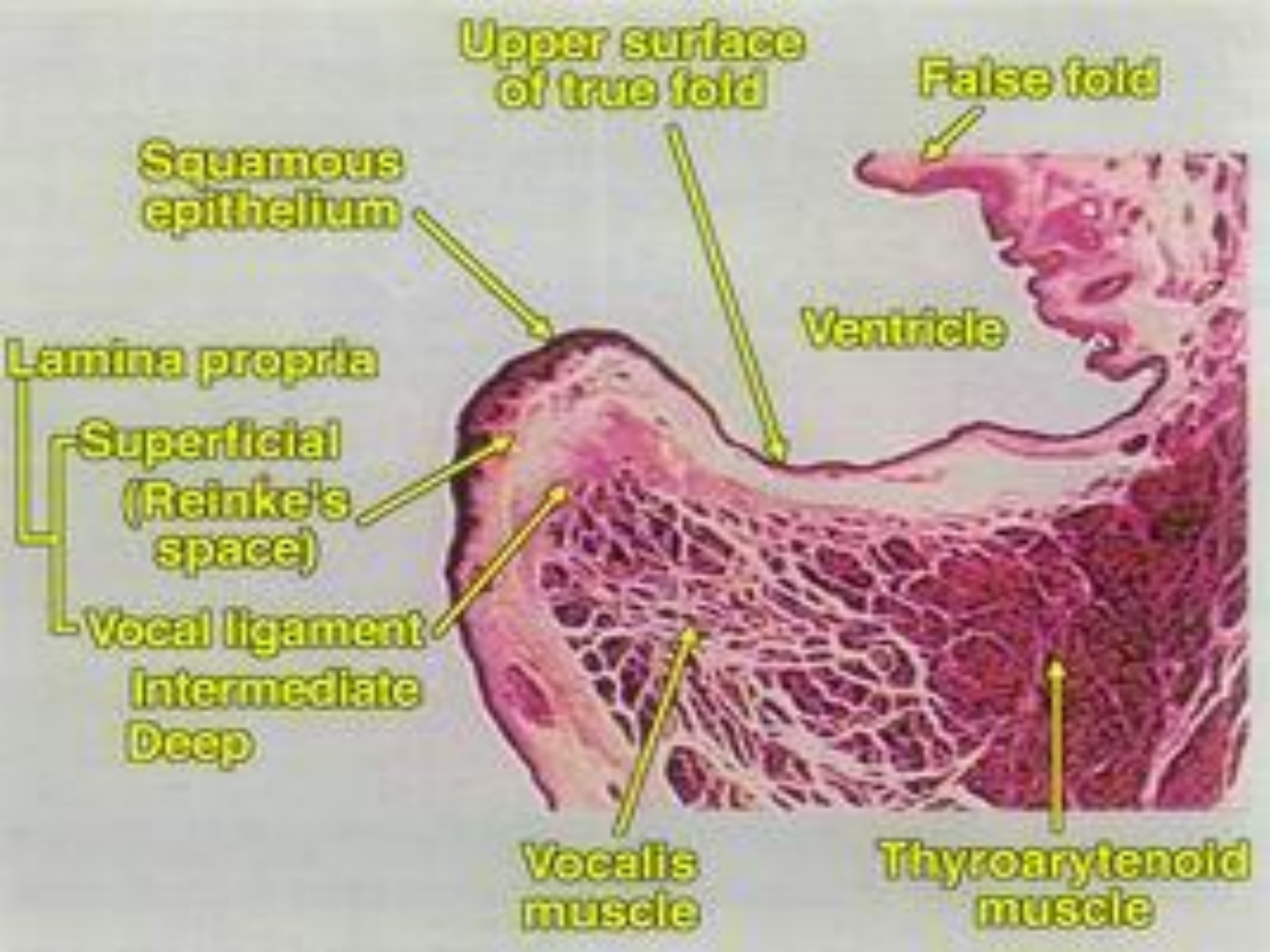

Posterior wall of hypopharynx
Pyriform sinus
Corniculate tubercle on arytenoid cartilage
Aryepiglottic fold
Cuneiform tubercle
Ventricle
True vocal fold
Vallecula
False vocal fold
Laryngeal surface of epiglottis
Epiglottis
Lingual tonsils
Anterior commissure


Upper surface of true fold
False fold
Squamous epithelium
Ventricle
Lamina propria
Superficial (Reinke's space)
Vocal ligament
Intermediate
Deep
Vocalis muscle
Thyroarytenoid muscle

Usually the presenting symptoms in voice disorders are:

- Dysphonia: Any change of the patient's voice from his habitual one.
- Aphonia: Loss of the patient's voice (functional or organic).
- Phonasthenia: a subjective complaint of dryness, tightness, globus feeling and voice fatigue, while the patient's voice and larynx is normal.
- Dysodia: Change of the singing voice while the speaking voice is normal.

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Definition of dysphonia:

- “Difficulty in phonation”.
- “Change of voice from his /her habitual”.
- “Hoarseness” = roughness & harshness of voice.

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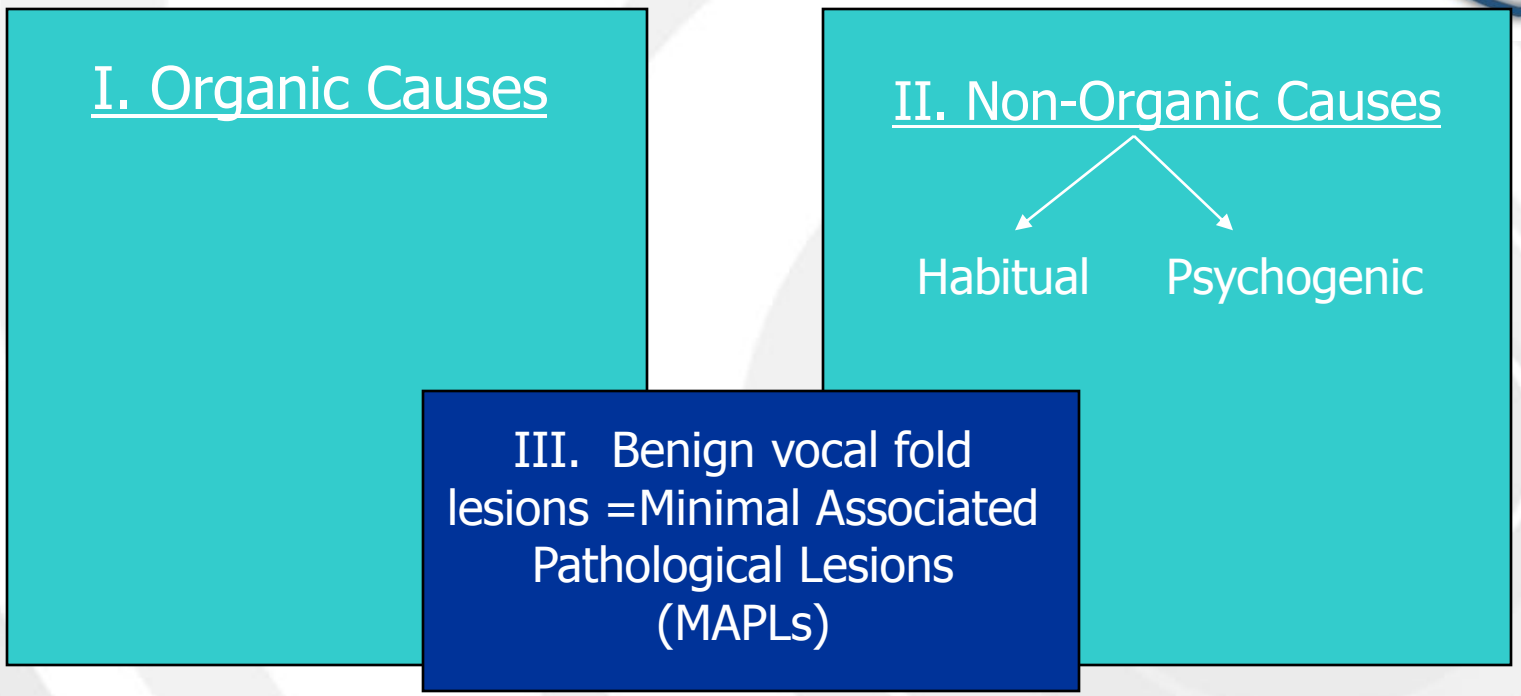
Etiological classification of dysphonia:



IV. [illegible] Neur[illegible] [illegible]iatric Ailments

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III. Voice disorders:

A) Organic voice disorders:

- Congenital.
- Inflammatory.
- Traumatic.
- Neurological.
- Neoplastic.
- Hormonal.
- Status post-laryngectomy.

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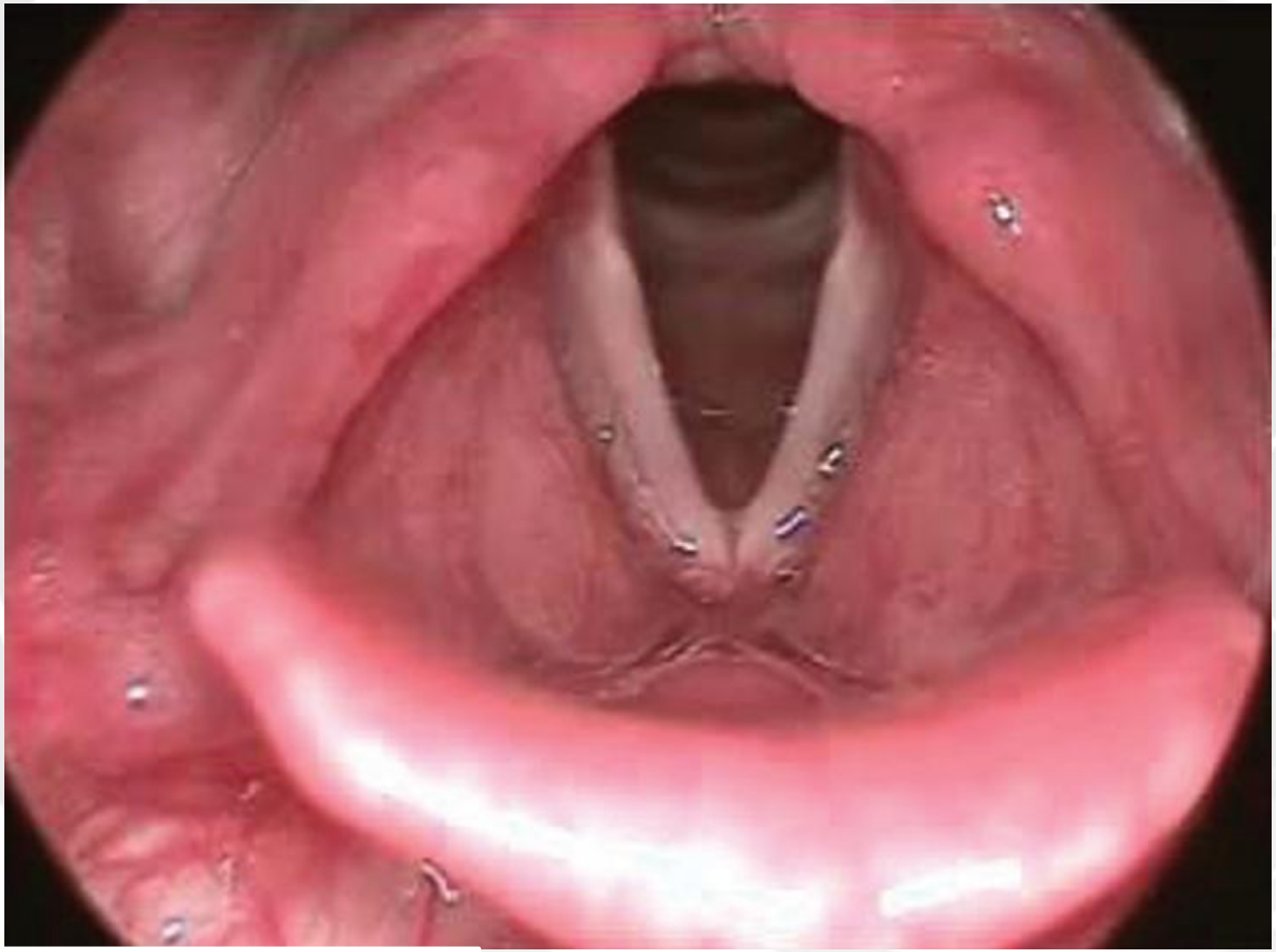
Normal



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Laryngomalacia (congenital)

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Congenital vocal folds web (Congenital)

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Laryngeal cleft (Congenital)

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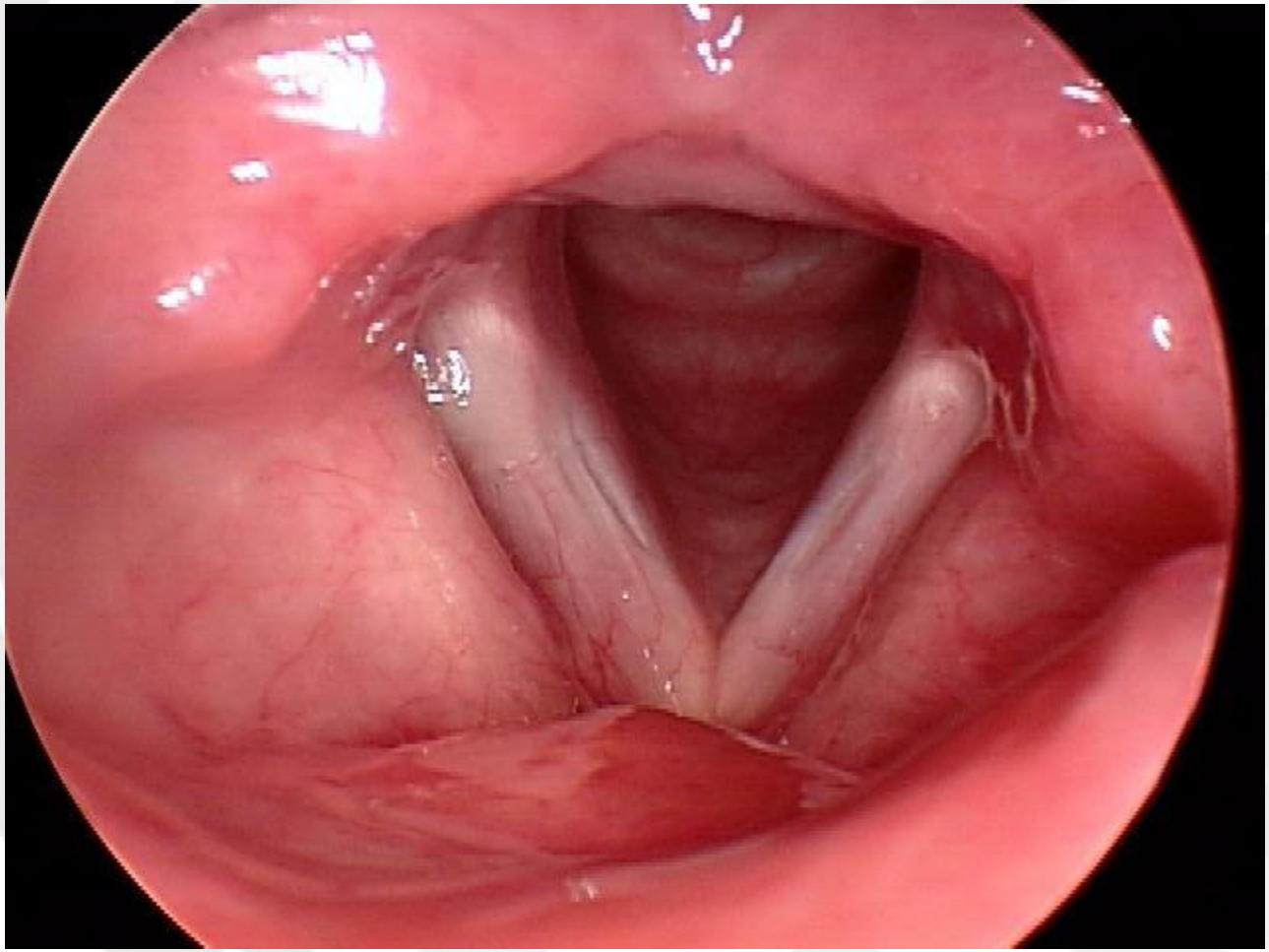
Sulcus Vocalis (Congenital)



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Fungal infection (Inflammatory)

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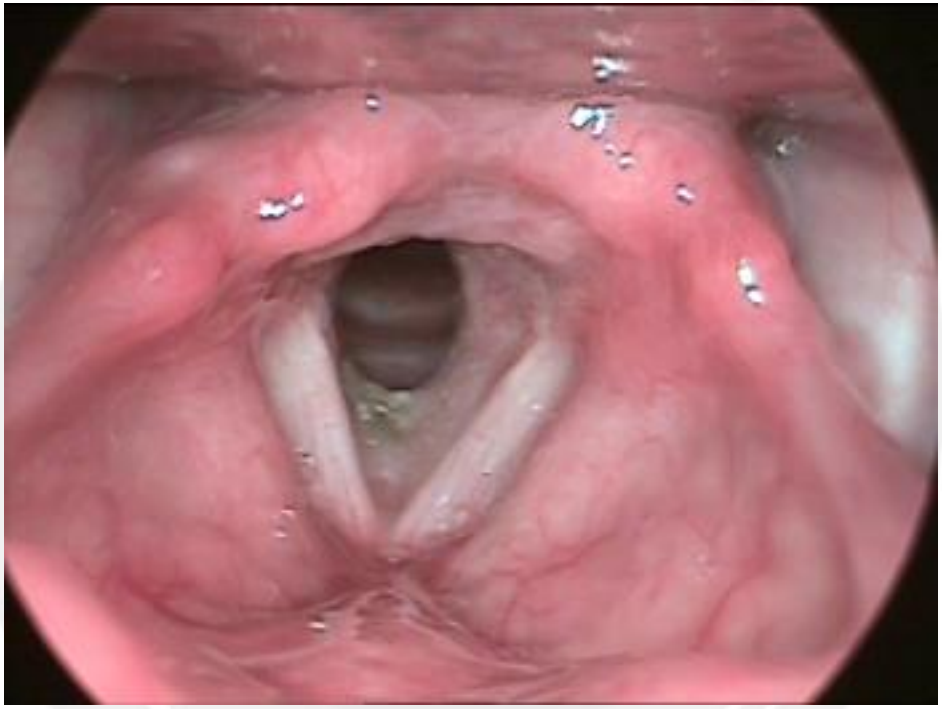
Laryngoscleroma (Inflammatory)



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Laryngeal carcinoma (Neoplastic)

Respiration

Phonation

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Cancer (Neoplastic)

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Left vocal fold paralysis (Neurological)

Respiration

Phonation

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F.B.

Respiration

Phonation

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III. Voice disorders:

B) Non-organic voice disorders:

i. Habitual:

1. Hyperfunctional childhood dysphonia.
2. Incomplete mutation.
3. Phonasthenia (Voice fatigue).
4. Hyperfunctional dysphonia.
5. Hypofunctional dysphonia.
6. Ventricular dysphonia.

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Hyperfunctional dysphonia

Respiration

Phonation

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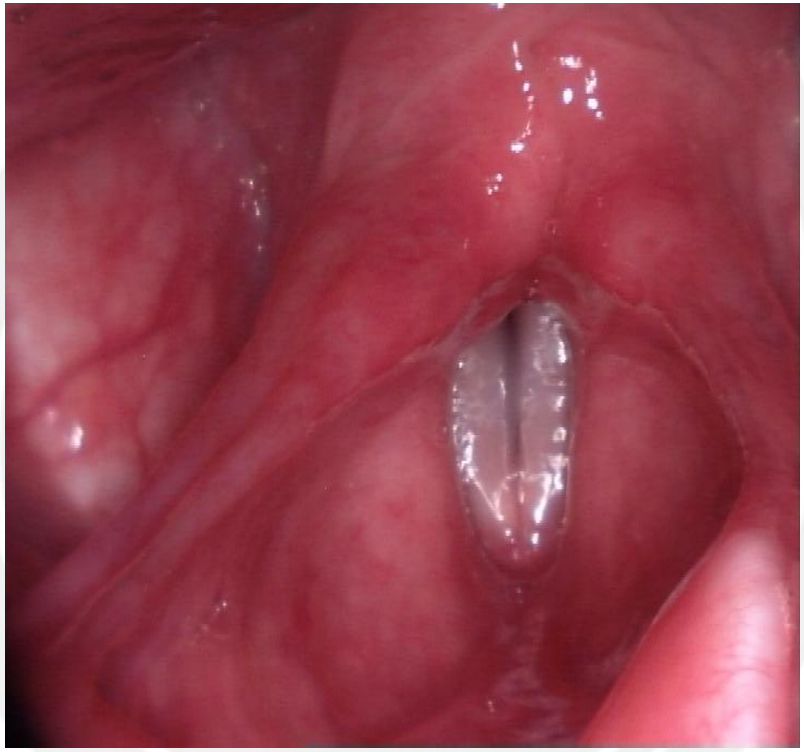
Phonasthenia

Respiration



Phonation

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B) Non-organic voice disorders (cont.):

ii. Psychogenic:

1- Psychogenic dysphonia.

2- Psychogenic aphonia.

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III. Voice disorders:

C) Benign vocal folds lesions :

1. Vocal fold nodules.
2. Vocal fold polyps.
3. Vocal fold cysts.
4. Reinke's edema.
5. Contact granuloma.

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Vocal Fold Nodules: Adult Type

Respiration

Phonation

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Vocal Fold Nodules: Juvenile Type

Respiration

Phonation

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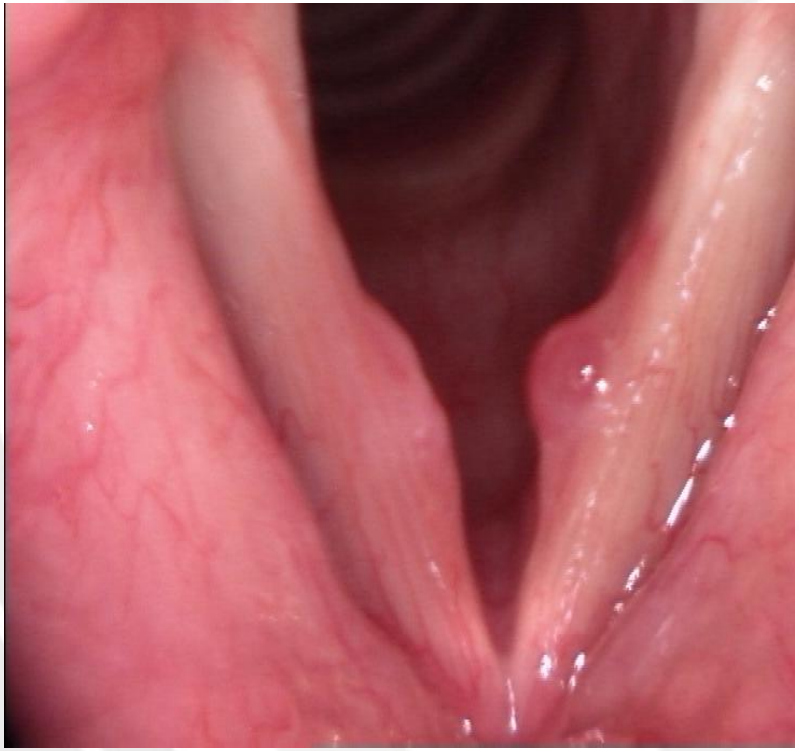
Left Vocal Fold Polyp with a Reaction



Respiration

Phonation

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Left Vocal Fold Polyp

Respiration

Phonation

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Right Vocal Fold Polyp

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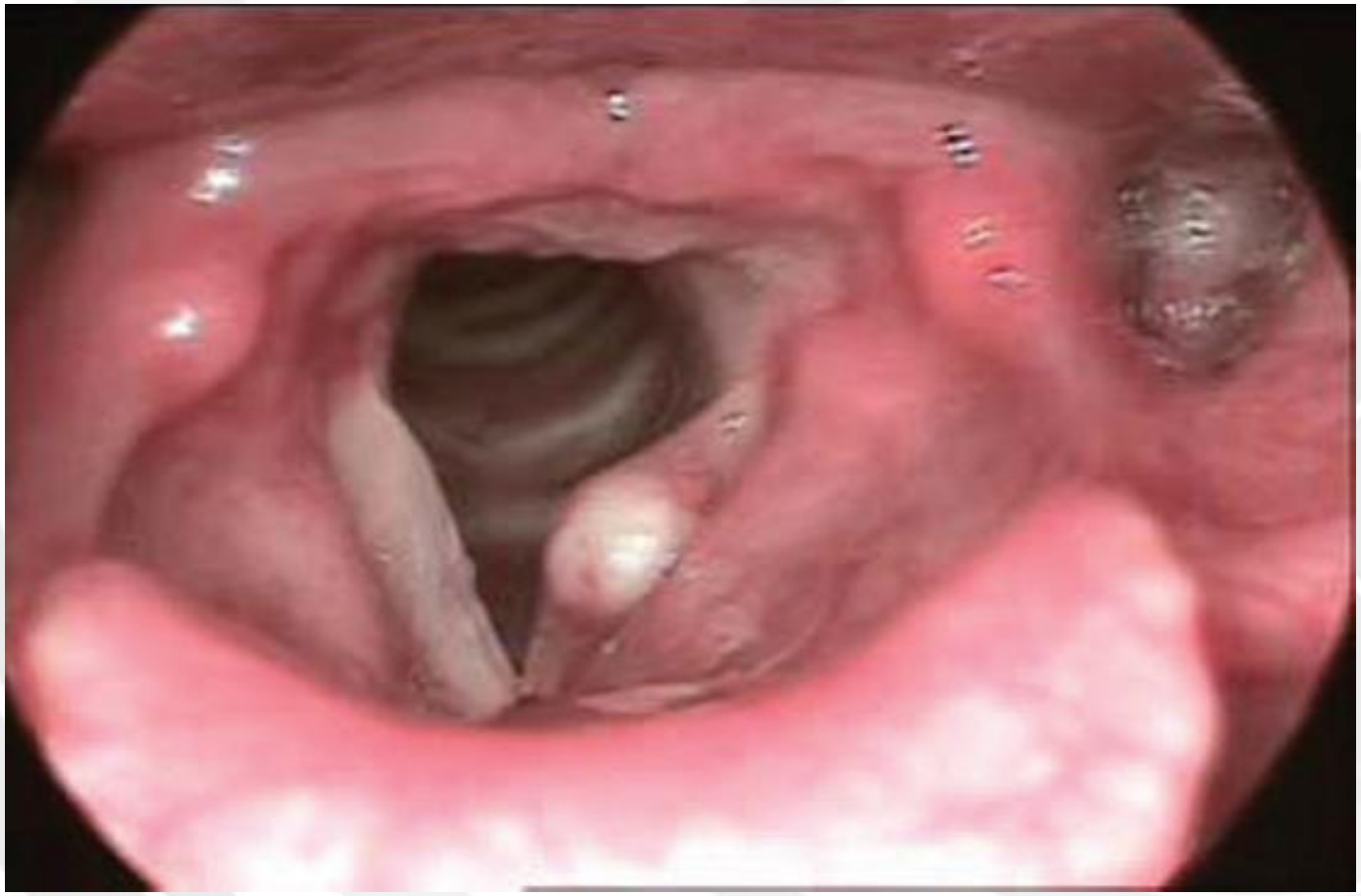
Left Vocal Fold cyst



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Bilateral Reinke's edema

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Bilateral Reinke's edema

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Right-sided Intubaion Granuloma

Respiration

Phonation

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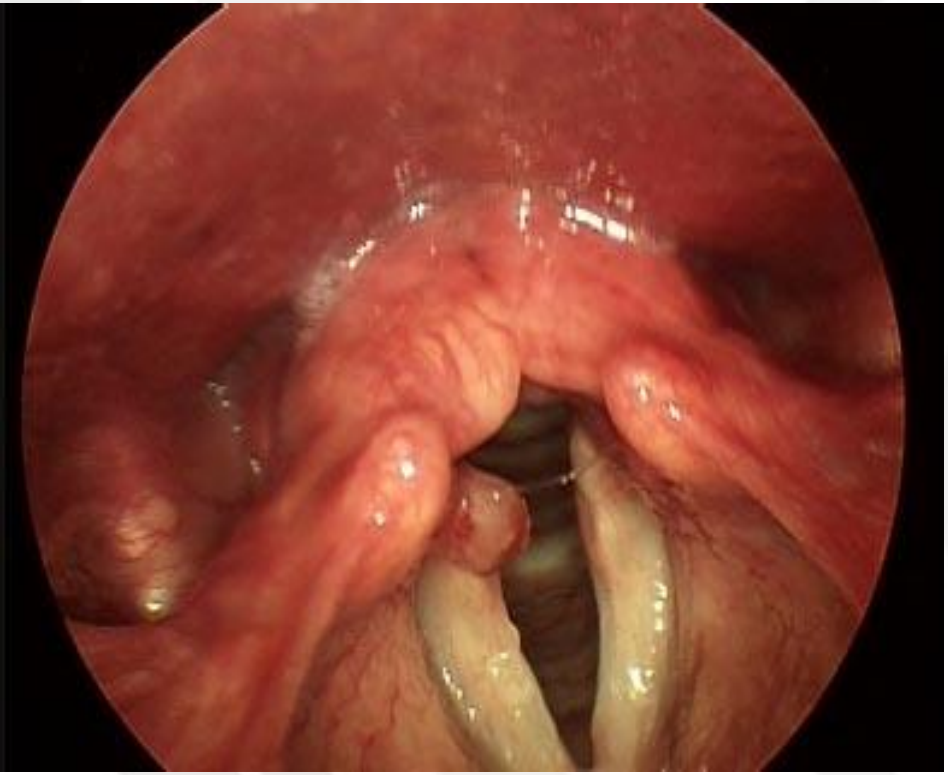
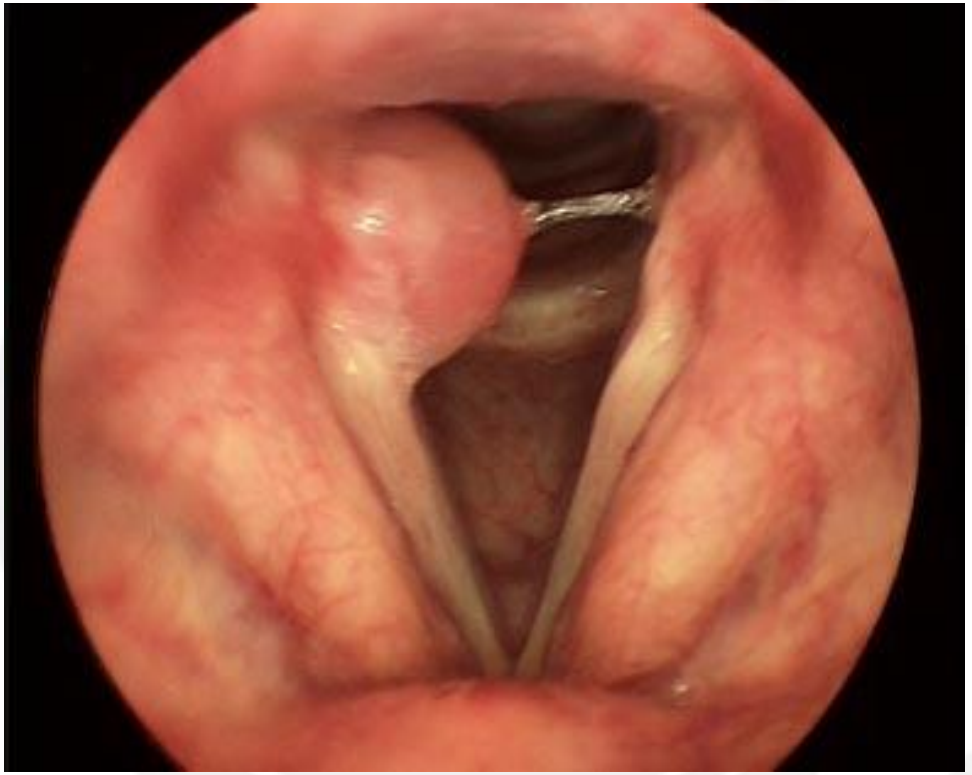
Right-sided Contact Granuloma





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Assessment of dysphonia:

I. History taking.
II. Physical examination: APA , … , neck , …
III. Investigations:
- Audio recording.
- Digital laryngostroboscopy.
- Digital laryngokymography.
- Digital High speed imaging
- Acoustic analysis (MDVP).
- Aerodynamic analysis (Aerophone II).
- GERD (LPR) work-up.
- CT neck.

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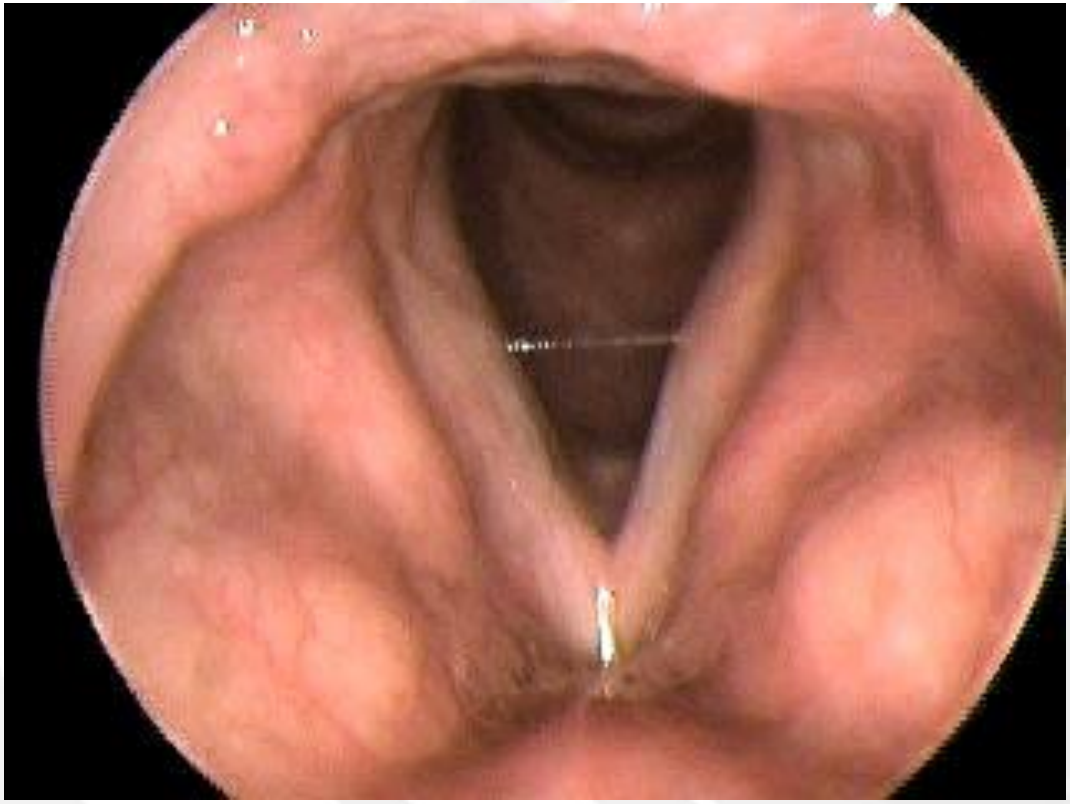
Stroboscopy



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KAUH-Strobe Examination Report

Name: Al-Bulaihi, Haila, M **Patient ID:** 00465849 RKH
Exam Original Date: 3/13/2004 9:46:18 AM

Selected Stills (Image Compression - 15:1)

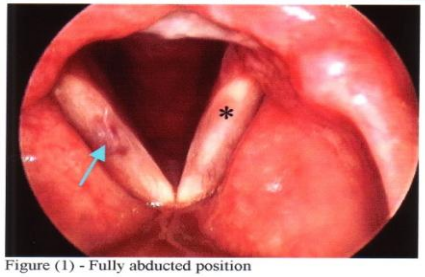

Figure (1) - Fully abducted position

Figure (2) - Fully adducted position

Thank you for referring this patient.

Telescopic videolaryngostroboscopy done, and showed:

I. Continuous light examination:

- Left vocal fold paralysis (asterisk).
- Paralytic phonatory glottal gap of about 2-3 mm at maximum width posteriorly (Figure 2).
- A patch of submucous hematoma at the middle third of membranous part of the right vocal fold (arrow).
- Mild ventricular hypertrophy.

II. Stroboscopic light examination:

- Decreased amplitude and mucosal waves on the left vocal fold.
- Asymmetry in amplitude and mucosal waves between both vocal folds.
- Aperiodecity in amplitude and glottal cycle time at the left vocal fold.
- Phase is predominantly open.

Diagnosis:

Left vocal fold paralysis with glottal gap of about 2-3 mm at maximum width posteriorly.

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High speed laryngeal imaging

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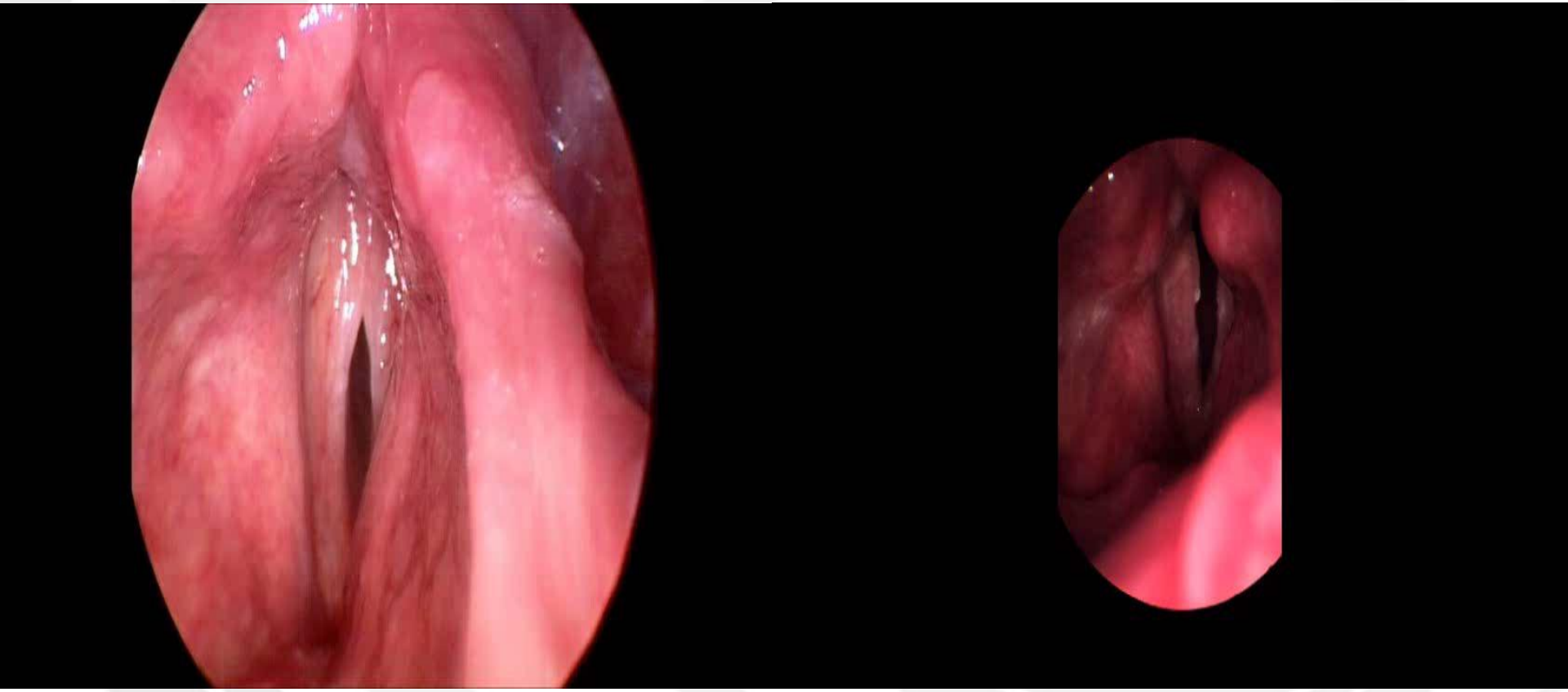
Strobe

High Speed



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Pharyngeal pH Monitoring

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Computerized speech lab. (CSL)

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Phonatory Aerodynamic System (PAS)

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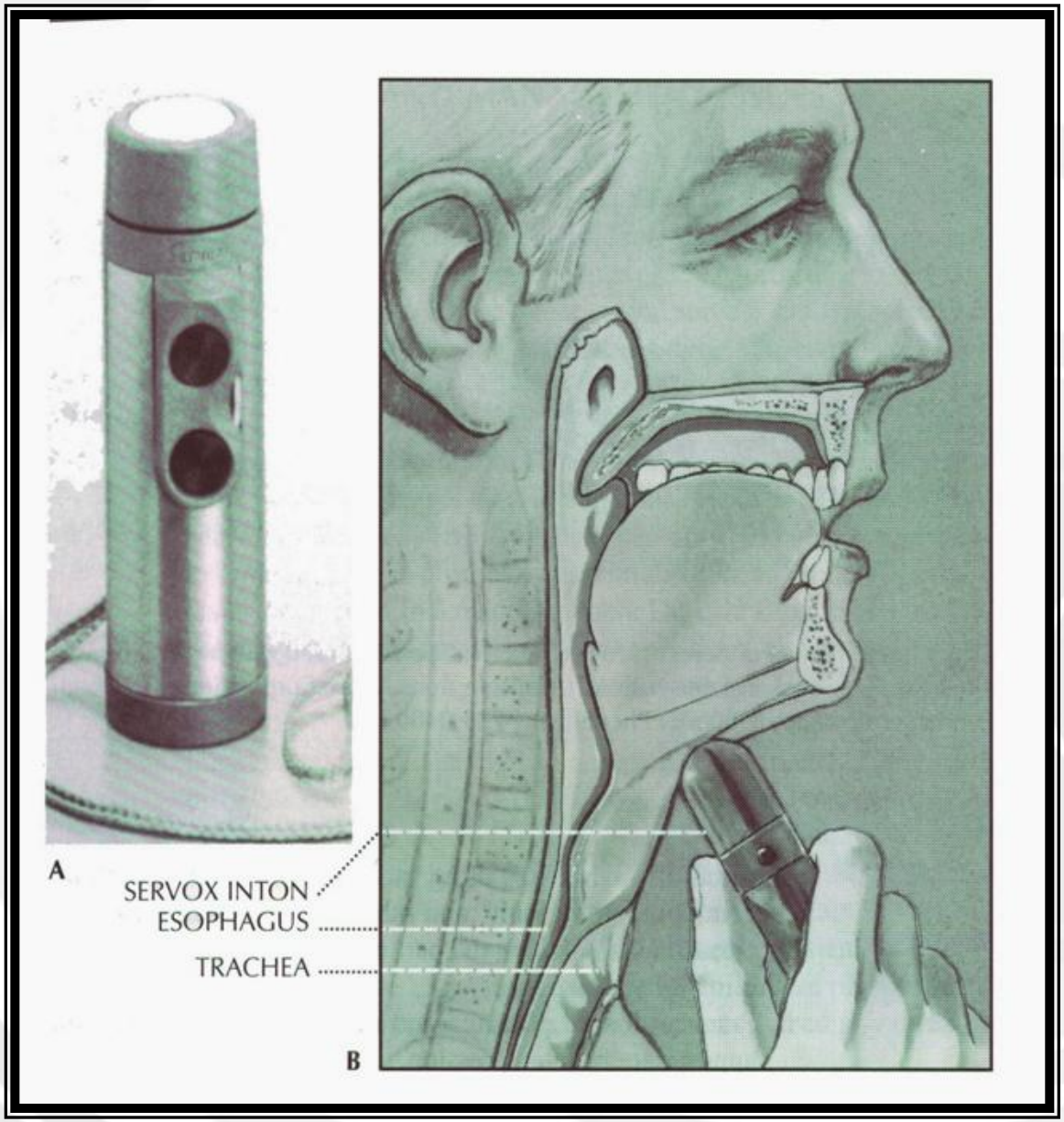
Management of voice disorders:

- Pharmacological agents.
- Surgical procedures (Phonosurgery).
- Technical aid devices.
- Voice therapy.



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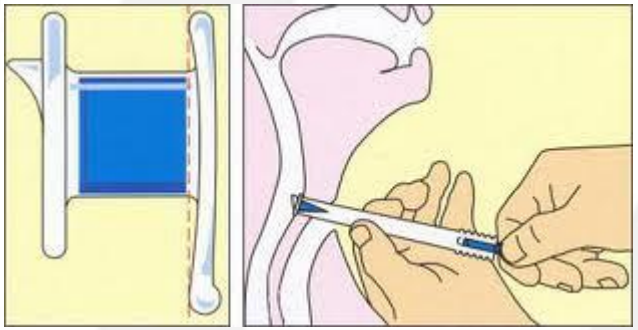
Tracheo-esophageal puncture



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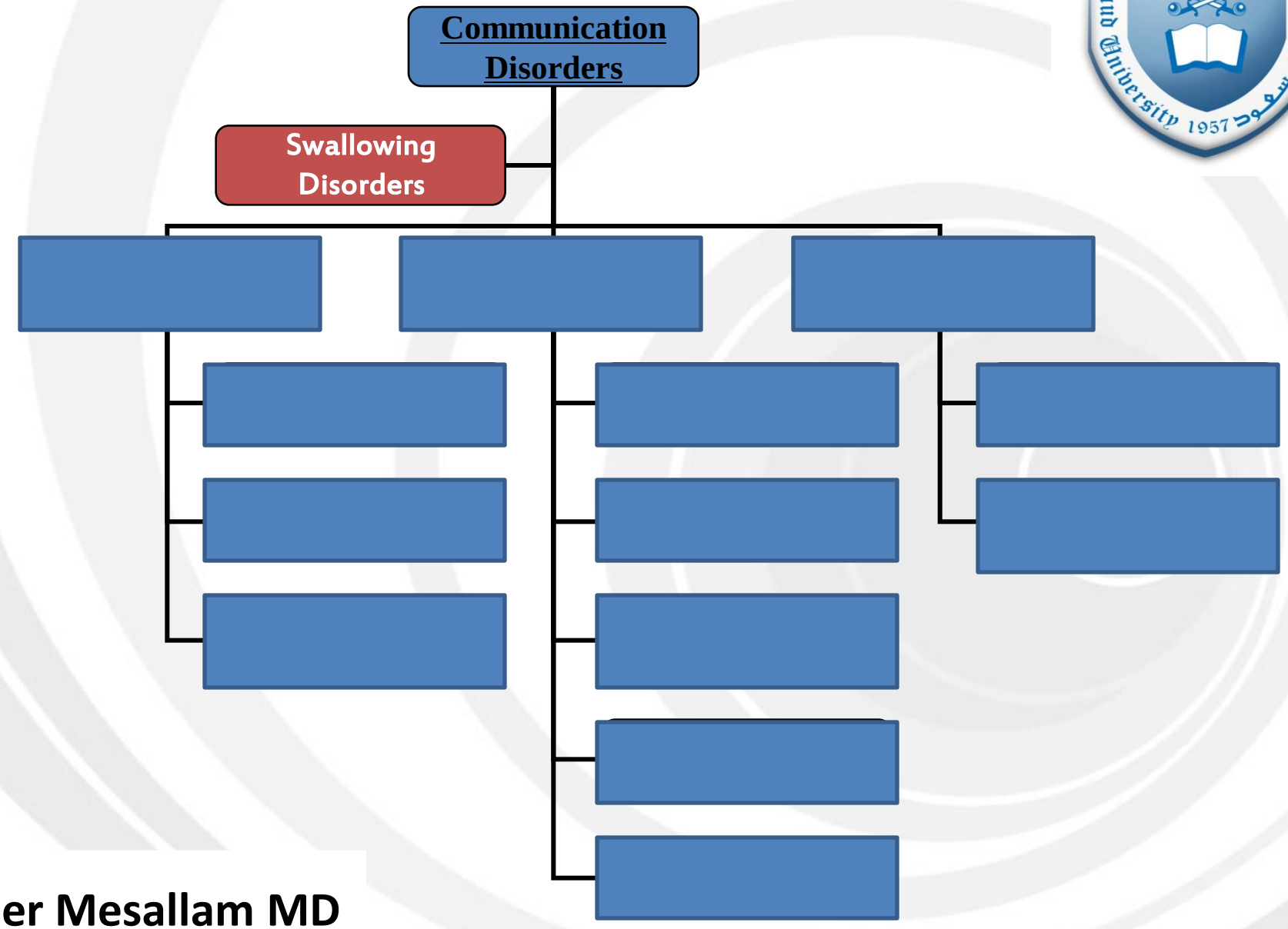

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Swallowing Disorders

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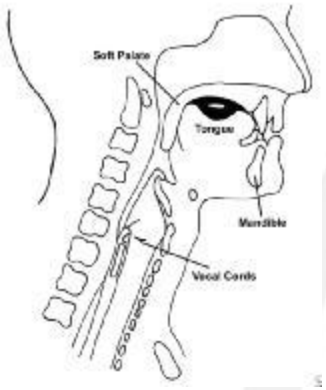
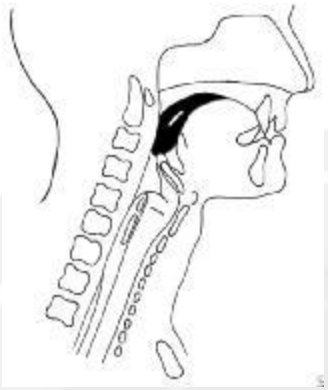
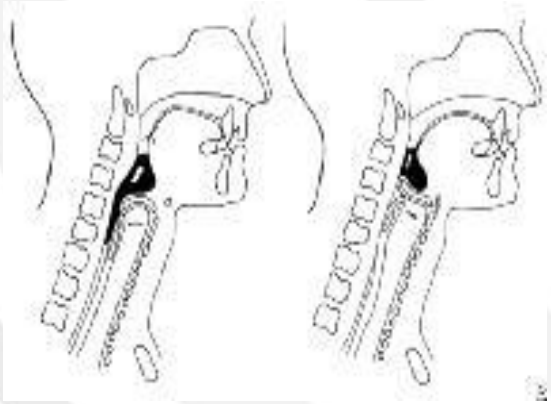
Phases of normal swallowing:



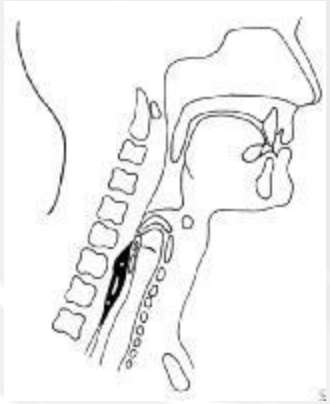
1. Oral preparatory phase



2. Oral propulsive phase



3. Pharyngeal phase



4. Esophageal phase

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Definition of dysphagia:

- "Difficulty in moving food from the mouth to the stomach".
- "Odynophagia" = painful swallowing due to a disorder of the esophagus.

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Consequences of dysphagia:

- Dehydration.
- Weight loss.
- Aspiration pneumonia.
- Airway obstruction.
- Loss of joy of eating.

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Causes of dysphagia:

Dysphagia

Oropharyngeal

- **Structural**
  - **Head & Neck Surgery**
- **Neuromuscular**
  - **CVA**

Esophageal

- **Mechanical** [Solids]
  - **Tumors**
- **Neuromuscular** (Esophageal Dismotility) [Solids & Liquids]
  - **Achalasia**

Assessment of dysphagia:

I. History taking.

II. Physical examination:

- General examination.
- Language and Speech assessment.
- Vocal tract examination.
- Neck examination.
- Trail feeding (Bed-side assessment).

III. Investigations:

- FEES.
- VFES (MBS).
- GERD (LPR) work-up.

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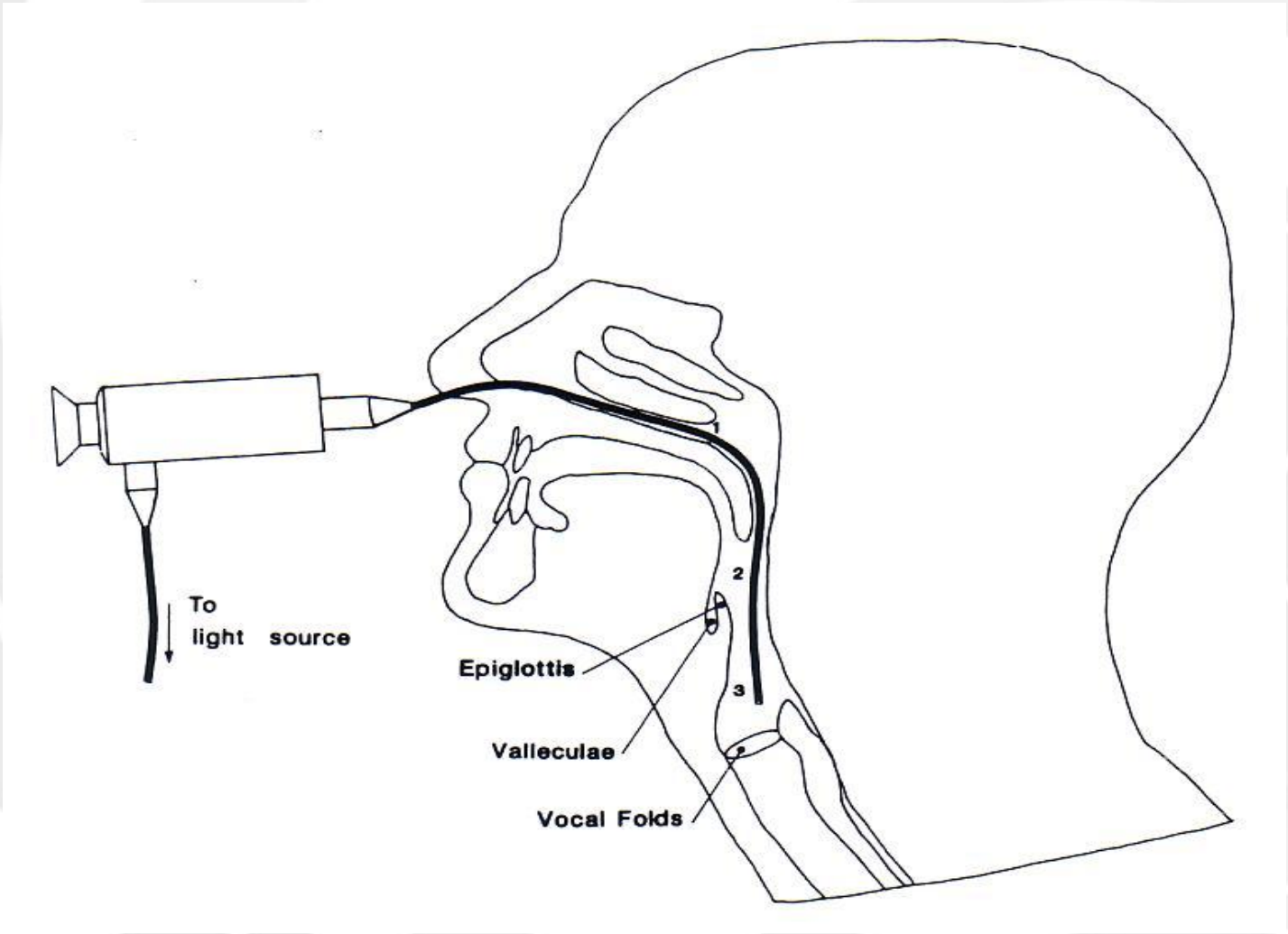

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FEES

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FEES protocol of evaluation (Langmore, 2003):

I. Anatomic and physiologic assessment.

II. Assessment of food and liquid swallowing.

III. Assessment of theraputic interventions.

Normal FEES

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Residue

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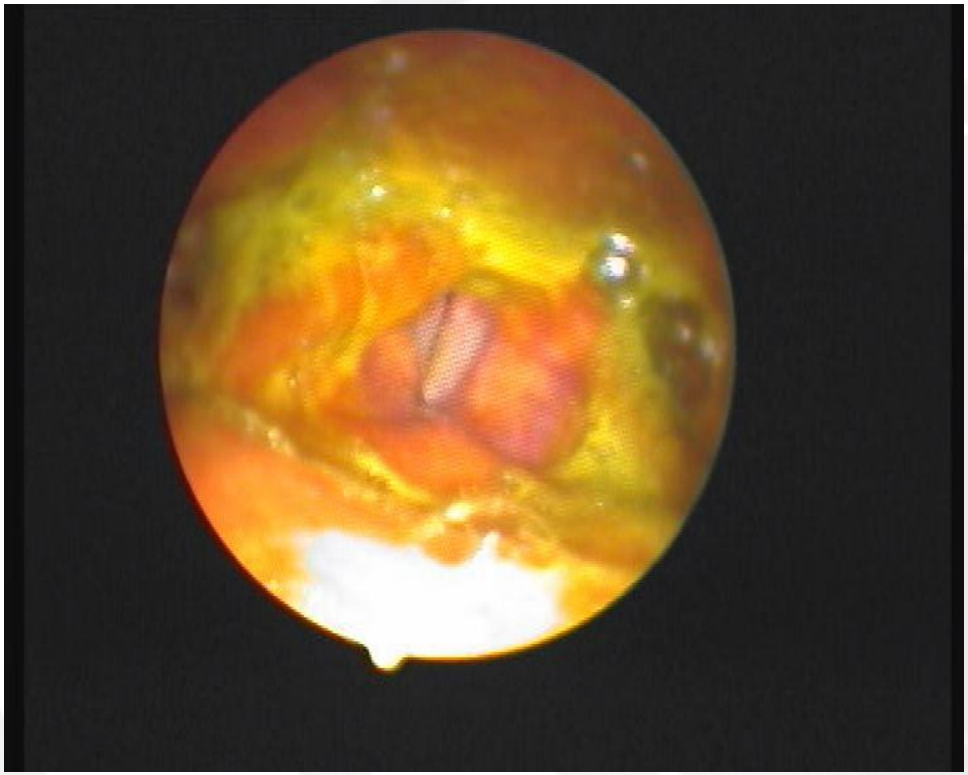

Residue

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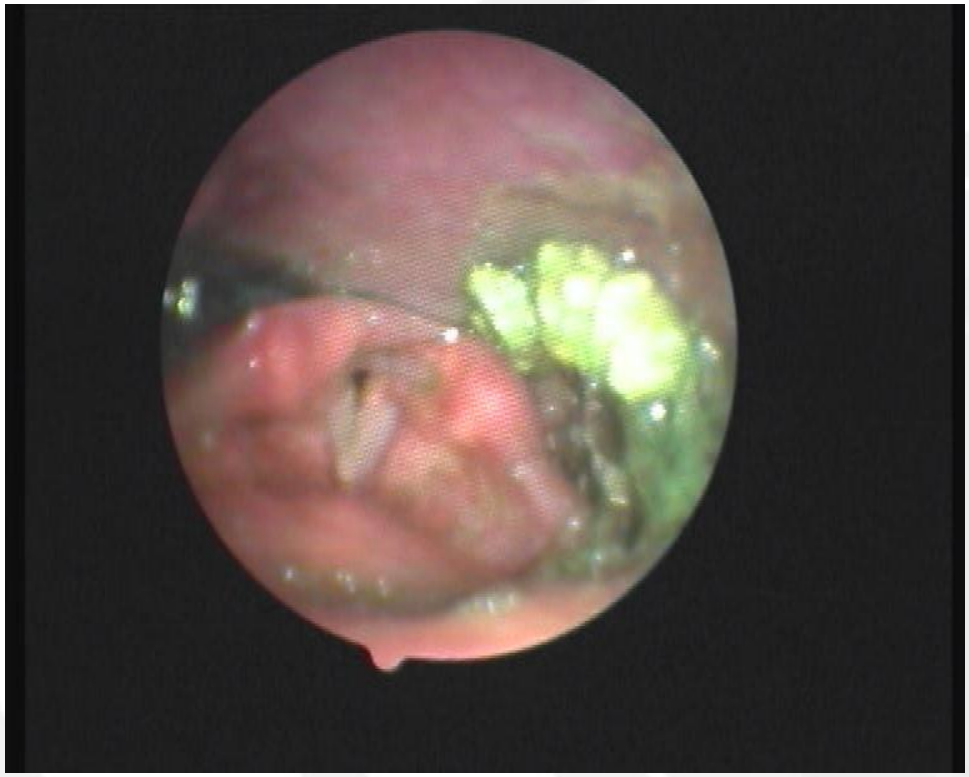

Residue

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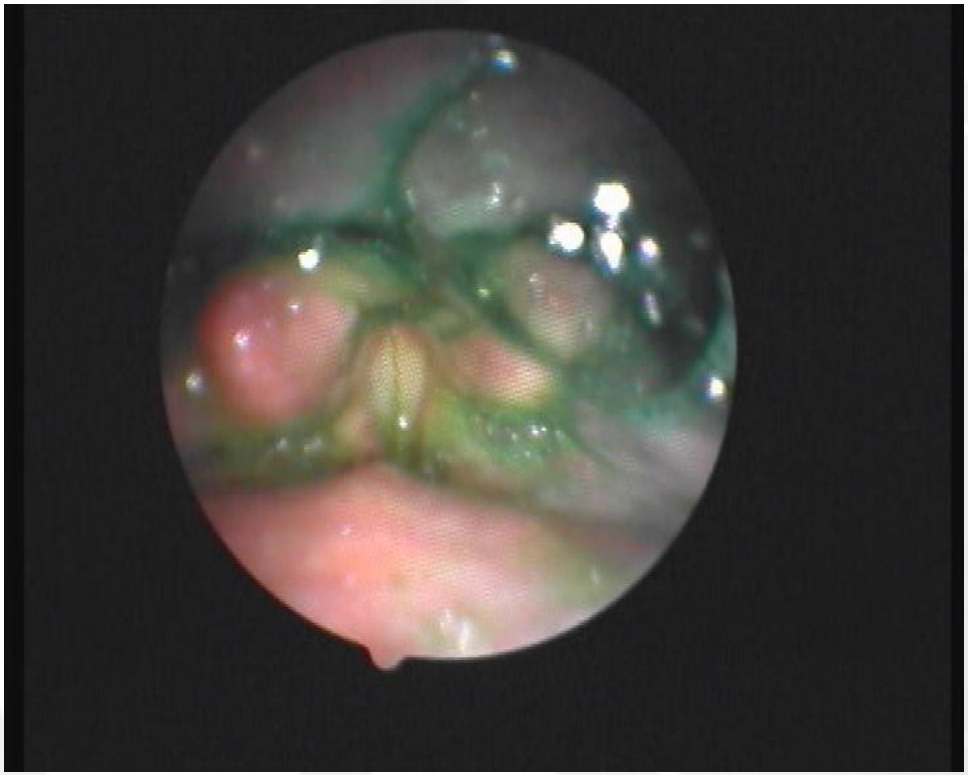

Penetration

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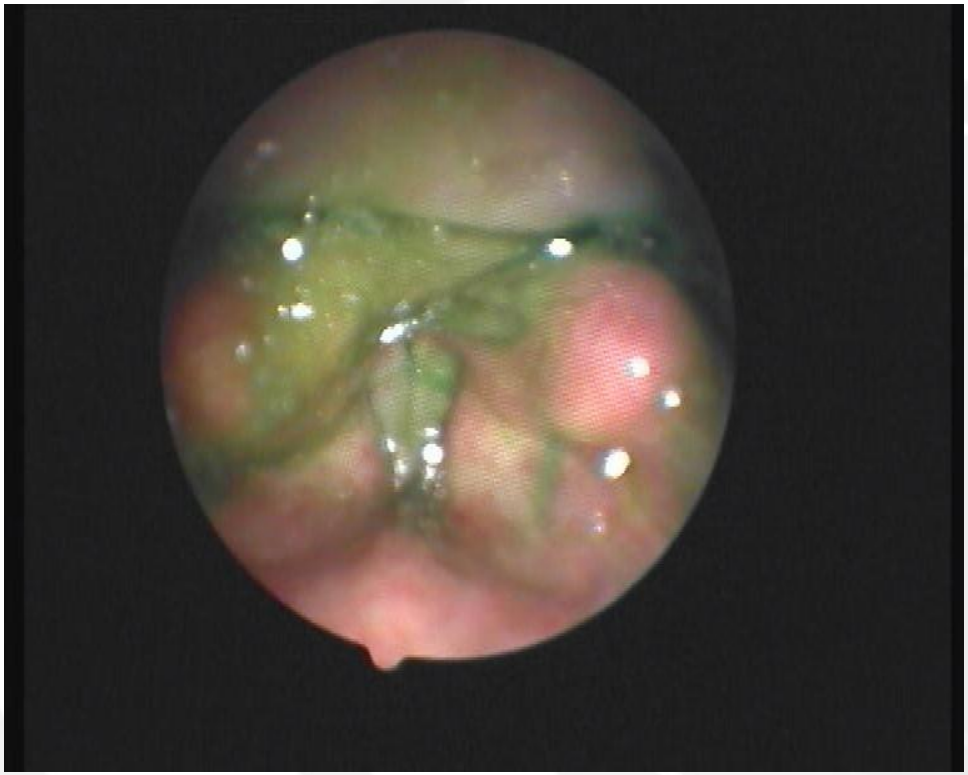

Penetration

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Aspiration

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Normal (MBS)

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Aspiration (MBS)

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Management of dysphagia:

- Swallowing therapy:
  - Diet modification.
  - Postural techniques.
  - Swallowing maneuvers.
  - Sensory enhancement techniques.
  - Motor exercises.
- Surgical treatment, eg medialization laryngoplasty.
- Medical (Drug) treatment, eg anti-parkinsonism drugs.
- Intraoral prosthesis.
- Alternative routes of feeding, eg NG tube feeding.

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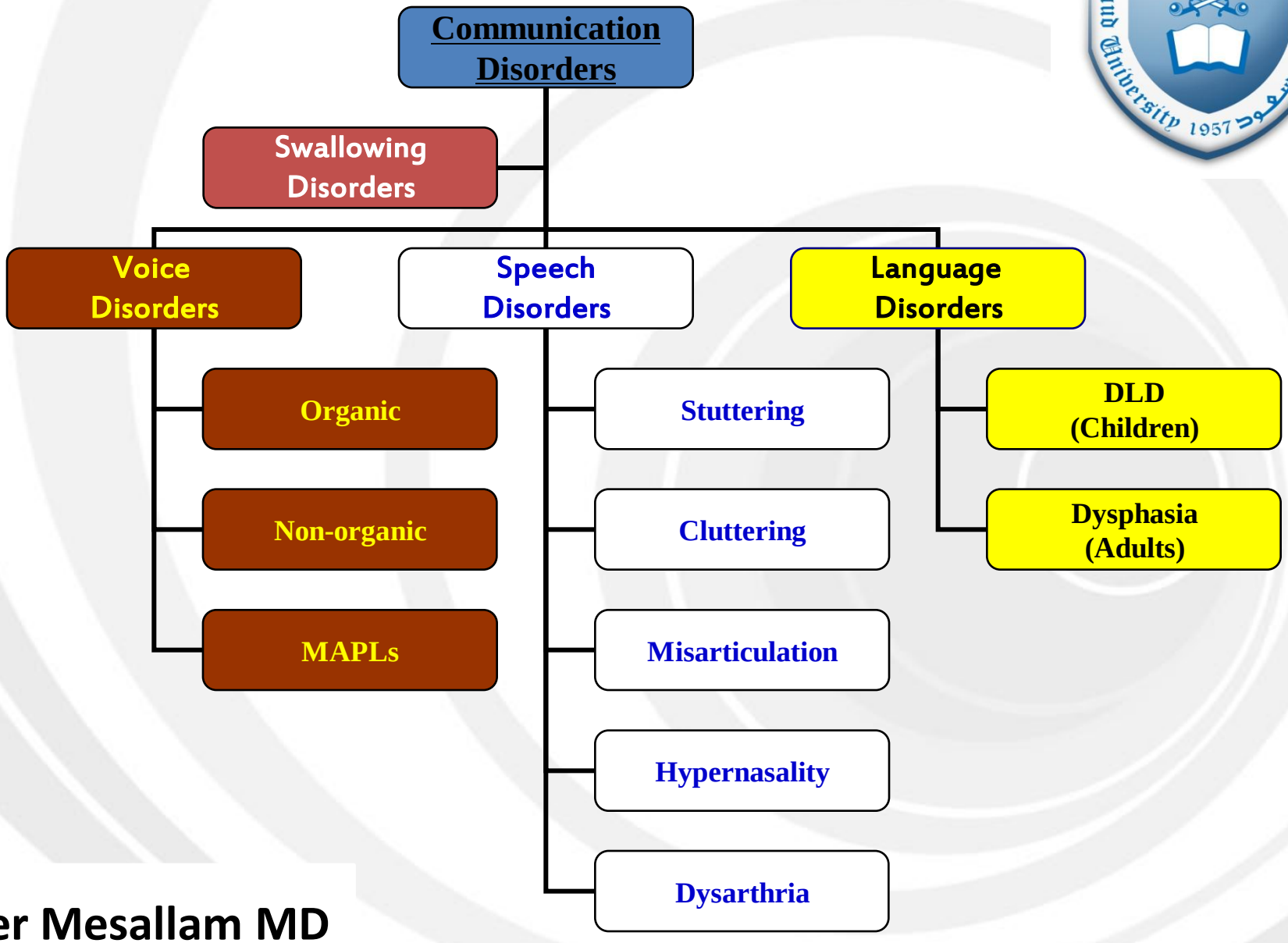

Tamer Mesallam MD

❑ Office Hours

- Sunday 1- 3 pm

- Wednesday 1-3 pm

Building 5, 2nd floor, CSDU

Tamer Mesallam MD

Thank You

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