Sleep Apnoea

Detailed History of Presenting Illness (HPI)

- **!! SNORING !!**
- **Daytime Sleepiness**
- **Confusion**
- **Short-term Memory Dysfunction**
- **Headache in the Morning**

ALSO USUALLY

- **Obesity**
- **Ankle Swelling**
- **Extertional Dyspnoea**
- **Peripheral Cyanosis**
- **a sore throat and nearly always a dry throat**.
  This is because the vibration of the soft palate for several hours leads to trauma of the tissues with inflammation and often oedema of the palate and uvula

List of Differential Diagnoses (DDx)

- Sleep apnea,
- Hypoxia
- Drug intoxication (eg. benzo)
- Thyroid dysfunction (hypothyroidism)

List Pertinent Findings on History (Hx)

- **Disruption to work** due to marked daytime sleepiness
- increasing ankle oedema,
- weight gain
- worsening breathlessness on exertion.
- sleeping poorly waking 2-3 times per night with a dry mouth and/or sore throat.

OFTEN, THIS IS A TRUCKER'S DISEASE

- **a snorer since adolescence** (extremely loud with **snorting and periods of choking and gasping**).
- **periods of silence or 'no breathing'.**

Good questions:

- How long has snoring been present for?
- Is snoring a worsening problem?
- If snoring occurs every night?
- Are there any factors that make it worse?
- Does it occur in all positions or only when lying on the back?
- What part of the throat does the noise appear to come from (high up behing the nose palatal snoring or low down behind the tonque base of tongue snoring)?
- Does the subject ever stop breathing altogether (apnoea), and if so, what actually happens?
- When was apnoea first noticed?
- How long does it last for?
- What is the first breath like at the end of the silence, and does the patient stir in their sleep at that moment?
- Is your nose easy to breath through?
- Do you get to sleep easily at night?
- Do you get up in the night to pass urine?
- Are you a restless sleeper?
- Do you have vivid dreams, nightmares, or awaken with difficulty breathing?
- Is your throat sore or dry in the mornings, and do you have a headache on awakening?
- Are you refreshed from sleep and ready to start the day when you wake up?
- When is the best and worst time of day for you to concentrate without sleepiness?
- Are you as motivated now to do jobs at home at the weekend as you were 5-10 years ago, or is there more difficulty to get tasks started?
- What is your current weight (compared with young adult life)?
- What is your current collar size (compared with young adult life)?
- Are you taking any current medicines at home?
- Any past medical history, allergies?
- Do you smoke?How much alcohol do you drink?

Personal history :
- **Smokers** / **drinkers most affected** (drinkers especially)

Family history:
HISTORY OF HEAVINESS, SNORING and HYPERTENSION.

List pertinent findings on Examination (Ex)

!! OBESITY ! most common; + FAT NECK

VITALS:
- Expecting a **high blood pressure**
- Expecting an **increased respiratory rate**

Respiratory Exam:
- **Plethoric facies with thick neck**.
- **"Crowded" upper airway with reddened oedematous palate and uvula.**
- Percussion note decreased bi-basally with reduced breath sounds in the bases. No added sounds

Tests and Investigations

FBC
- Expecting maybe **high or normal haemoglobin,** (adaptive reaction) otherwise unremarkable

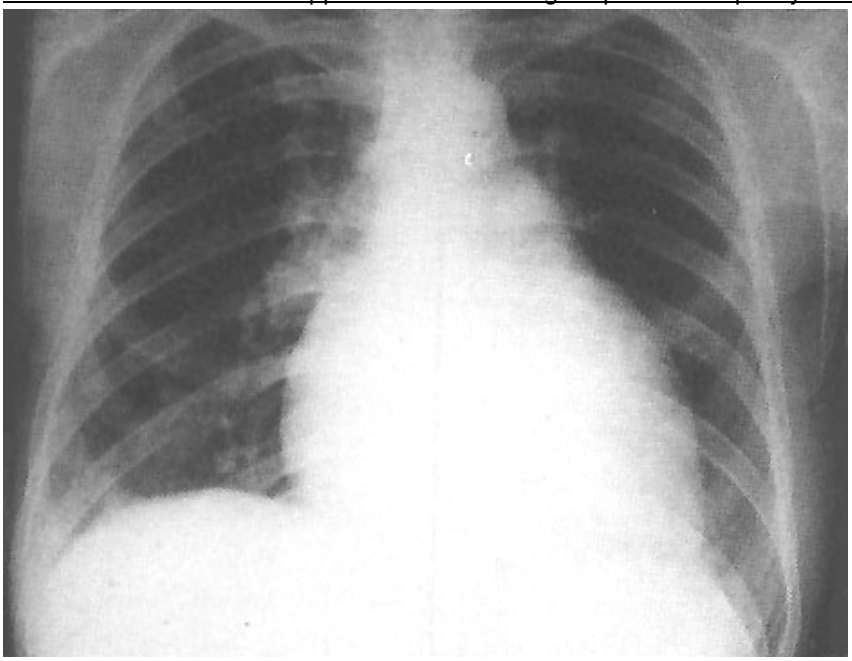
Plasma Biochemistry:
- Expect **unremarkable biochemistry**, with possibly elevated CO_2

However: In the event of right heart failure, there will be PORTAL HYPERTENSION
THEREFORE: Bilirubin and Liver Enzymes will be Elevated ← !!

Arterial Blood Gases
- Expect **HYPERCAPNEA and HYPOXEMIA with associated acidosis**
- **Low pH** (7.34-7.45),
- **High PCO_2** (35-45) mmHg,
- **Low PO_2** (85-95) mmHg,
- **Low O_2 saturation** (95+%),

ECG :
- Expect **some evidence of right heart failure or Enlargement**

Chest Xray :
The heart is enlarged, there is a prominence of both hilar regions, (see below)
…suggesting enlargement of the pulmonary arteries.
PULMONARY OEDEMA appears as a “bat-winged” pattern of opacity surrounding the heart (this is not it)



Respiratory Function:

Expect **increased Residual Volume and Inspiratory Capacity; everything else normal or decreased** (in absence of concurrent respiratory disease)

Thyroid Functions:

Should be normal in sleep apnoea; if abnormal, other pathology is present

Management

Initial:

- **low flow oxygen (1 L/min)** and blood gases repeated on oxygen.
- **commence on diuretic** (frusemide) (prepare for massive weight loss)
- **Restrict Fluid Intake**
- **commence DVT prophylaxis** using sub cutaneous low mol. weight heparin.
- **Antibiotic therapy of amoxycillin/clavulanic acid if there is exacerbating infection**.
- **!! CEASE ALL SEDATIVES AND ALCOHOL !!**

Long term:

- **Sleep Studies:** Polysomnography;

Record-

- Awake oxygen saturation
- Asleep oxygen saturation
- Record apnoeic events during study
- Record limb motility during study

2nd sleep study → WITH POSITIVE AIRWAY PRESSURE

- to gauge effect of treatment

Educate Patient: regarding CPAP or BiPAP device

A **supervised weight reduction program and reduction in alcohol intake** to help modify lifestyle.

physical obstructions (enlarged tonsils) can be removed surgically however long term this only reduces snoring and does not address airway collapse.

Dental splints by orthodontists can be made to pull the tongue forward to maintain airway patency.

Prognosis = Excellent

(assuming successful weight loss, alcohol reduction and effective PAP administration)

Treated there can be some reversal of the effects which will decrease mortality and morbidity

HOWEVER: **Untreated** there is a **poor prognosis** and **death can result** from **respiratory failure** and or **right heart failure.**

Disease Definition

Respiratory failure with associated hypoxemia that has resulted in pulmonary hypertension and right heart failure. The marked hypoxaemia , sleep disturbances and hypercapmoa has lead to confusion and all of this is exacerbated with an acute respiratory infection.

How is this diagnosis made ?

Arterial blood gases, respiratory function tests and sleep studies (polysomnogram)

The sleep studies laboratory monitors how a person sleeps during their time in the laboratory. There is monitoring of brain activity and sleep stages by EEG, the oxygen saturation is measured by blood oximetry, a microphone records the snoring episodes, an ECG measures the heart rate and function and the chin, abdomen and legs are monitored by electrodes to detect movement.

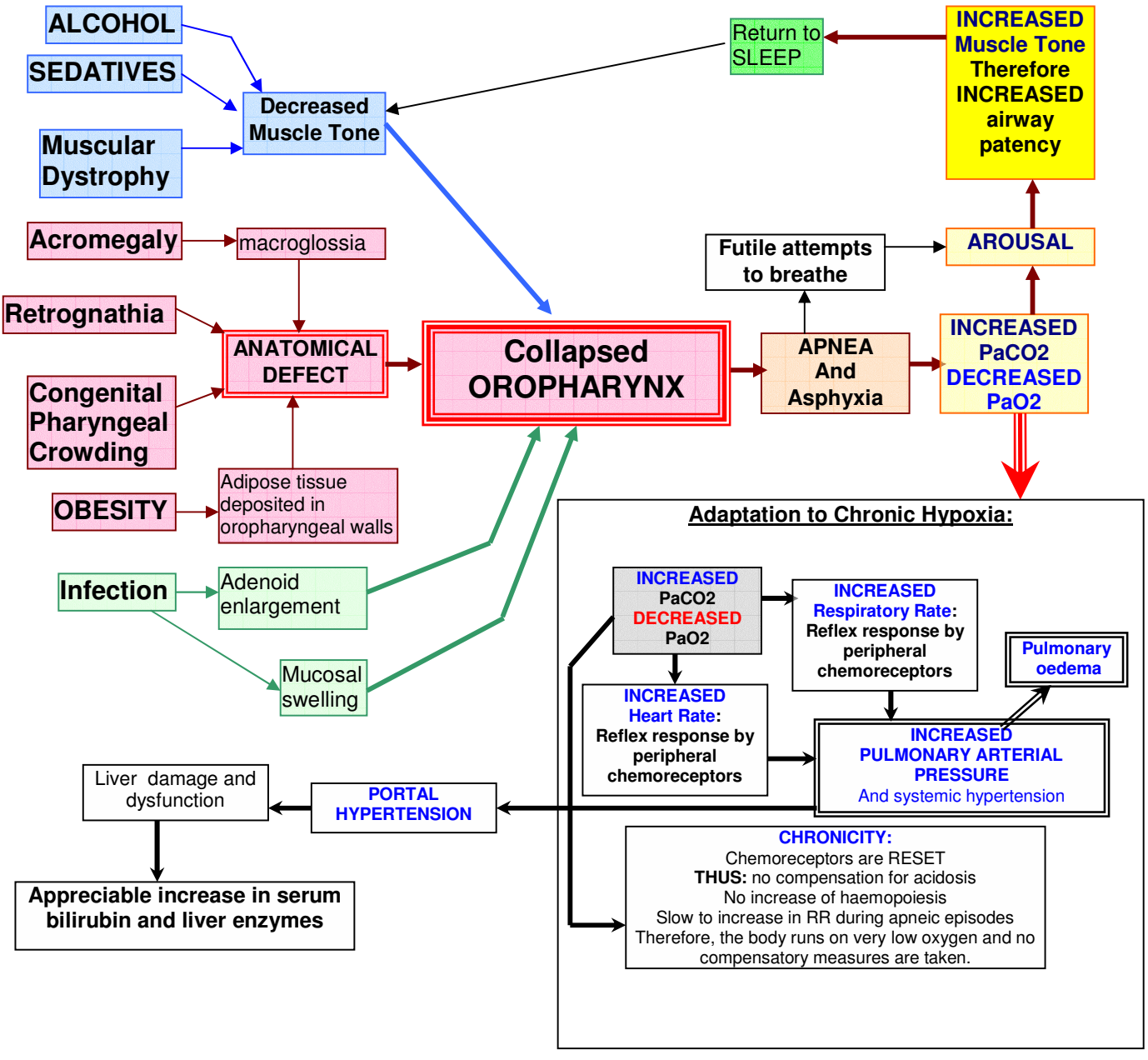
Epidemiology

- In north America 24% of men and 9% women have sleep apnoea.
- Males are 2.5 times more likely than females.
- Most commonly it occurs in middle aged men and post menopausal women

Aetiology

- Most tend to be snorers with **obesity, smoking and alcohol implicated**.
- There is an abnormally narrow or floppy airways
- there can be a large neck girth.

Overall, anatomical predisposition + exacerbating factors



Physiology:
NORMAL SLEEP

Stages:

WAKEFULNESS

- Active (alert; eyes open)
- EEG - "active"/"desynchronized" (sinusoidal; 10-30 µvolts; 16-25 Hz) REM abundant
- Relaxed (eyes closed)
- EEG- alpha activity (20-40 µvolts; 8-12 Hz), REM is scarce
- Submental EMG may be moderate/high in both

Non-REM Stage 1

- A transitional stage into sleep
- characterized by theta waves
- mixed frequency 3-7 Hz (cycles/sec)
- diminution of alpha wave activity
- slow rolling eye movements
- 1-7 minutes

Non-REM Stage II

- The most abundant stage (50% in young adults).
- EEG records sleep spindles (12-14 Hz activity lasting at least 0.5 sec) and K-complexes
- lasts about 30-60 minutes
- no eye movements
- short mundane fragmented thoughts

Non-REM Stages III & IV (slow wave sleep)

- slow wave/deep sleep
- Highest auditory arousal threshold to awaken from stages III and IV.
- characterized by Delta waves
- high amplitude (>75 µv)
- low frequency (0.5-2 Hz)
- 20-50% stage III
- >50% stage IV

REM - Rapid Eye Movements

- First REM sleep about 60 to 90 min after sleep onset
- low amplitude desynchronized and saw tooth waves on EEG
- First REM cycle usually lasts only few minutes followed by NREM (stage II, III and IV). Later REM cycles progressively increase up to one hour through the night.
- Most dreams occur while awakening from REM sleep
- Dreams are emotionally charged, complex and bizarre

In REM the ascending reticular activating system (ARAS) activity is virtually completely gone, but high cholinergic activity from the basal forebrain causes thalamic neurons to remain in tonic mode.

- increased cerebral blood flow
- Atonia - marked reduction or absence of muscle tone in weight-bearing muscles
- increased brain T°
- increased O2 consumption
- penile/clitoral tumesence
- autonomic dysregulation (T°/HR/RR/BP)

In adults, **sleep of 8-8.4 hours is considered fully restorative**. In some cultures, total sleep often is divided into an overnight sleep period of 6-7 hours and a midafternoon nap of 1-2 hours.
Stage I is considered a transition between wake and sleep. It occurs upon falling asleep and during brief arousal periods within sleep and usually accounts for 5-10% of total sleep time.
Stage II occurs throughout the sleep period and represents 40-50% of total sleep time.
Stages III and IV delta sleep occur mostly in the first third of the night. They are distinguished from each other only by the percentage of delta activity and represent up to 20% of total sleep time.
REM represents 20-25% of total sleep time.

BREATHING DURING NORMAL SLEEP

Physiological changes occur within the respiratory system during normal sleep. These include:

- an alteration of the sensitivity of the central respiratory controller to changes in arterial levels of oxygen and carbon dioxide (i.e. chemoreceptor feedback)
- changes in its sensitivity to feedback from chest wall and respiratory muscle receptors.
- a fall in the neural output from the respiratory center (i.e. a fall in "respiratory drive").
- the fall in muscle tone that occurs in sleep means that the resistance of the upper airway (against which the respiratory muscles must work) increases.
- During rapid eye movement (REM) sleep, the only active muscle of respiration is the diaphragm - thus if its action is compromised, hyperventilation may be seen in REM sleep.

SNORING

Snoring is a loud noise usually generated during the inspiratory phase of respiration during sleep **by rapid vibration of the soft palate and pharyngeal walls**. It is indicative of inspiratory airflow limitation in the oropharynx i.e. that the airway is partially obstructed.

In general, the louder the snoring the greater the obstruction.

Snoring is very common, affecting as many as 30% of an adult male population.

Snoring is less common in females but its prevalence increases to the male level after the menopause.

Heavy, habitual snoring (all night, every night) is a risk factor for the development of cardiovascular disease. It is a provoking cause of systemic hypertension and is usually the forerunner of obstructive apnoea.

- **Moderate weight gain and nasal obstruction** typically precipitates snoring in many subjects
- **Alcohol consumption** is another important provoking factor and invariably precipitates obstructive apnoea in heavy habitual snorers if taken before sleep.

APNOEAS:

a. Severe: in these patients **sleeping and breathing are mutually exclusive!** The cycle of apnoea, then awakening with 4-5 breaths, occurs for the entire sleep period, with between 200 and 400 apnoeic episodes per night. During each apnoea there is a marked fall in oxygen level in the arterial blood, typically to below 80% saturation for most of the night (remember = PaO2 of less than 47mmHg). Although the character of sleep in these patients is abnormal - interrupted and variable - they do have REM-like sleep; it is in this state that the most severe desaturation occurs. These patients often present with cardiac failure, awake respiratory failure or neurologic symptoms (see below), as well as the more typical symptoms of loud snoring, witness apnoeas and excessive daytime somnolence.

b. Moderate: in this group there is repetitive apnoea throughout the night and less severe desaturation. These patients often go on for years without developing other obvious complaints apart from the major symptom of daytime somnolence.

c. Mild: in this group, apnoea tends to occur in epochs throughout the night with periods of loud snoring without apnoea. The runs of apnoea may occur for 10-15 minutes, typically at sleep onset or in REM sleep and are more likely to be dependent on sleep posture (e.g. occur only in the supine position). These patients often have only mild sleepiness but defective short-term memory function appears to be an important consequence.

Cause of upper airway obstruction

The oropharynx is a muscular tube which depends on muscle tone for patency.

During sleep there is a loss of background muscle tone and reduced phasic inspiratory activation of the dilator muscles in normals - thus the upper airway resistance increases.

Major underlying cause of sleep apnoea is a physically narrow oropharyngeal airway - often because of fat infiltration of the surrounding tissues. When this underlying narrowing is combined with a loss of muscle tone (in sleep), complete obstruction occurs.

However, any cause of neuromuscular weakness of the upper airway (e.g. bulbar palsy) may cause sleep apnoea.

Mechanism of Symptoms

Two major events cause the symptoms of sleep apnoea:

Sleep fragmentation

Apnoeas are terminated by a reflexly induced arousal (the asphyxia, stimulates chemoreceptors, causing arousal). When this occurs repetitively all night there is major fragmentation of sleep. The subject is unable to proceed through the orderly cycles of sleep. Although the total sleep time may appear greater than normal, it is of poor quality. This sleep fragmentation causes a main symptom - excessive daytime sleepiness.

Repetitive asphyxia

Repeated episodes of unstable respiratory failure (e.g. upwards of 400 episodes of progressive asphyxia to between 80% and 50% saturation every night) is the major cause of the serious medical complications of sleep apnoea. They are:

- Right heart failure - this results from hypoxia-induced pulmonary hypertension
- Sustained hypoventilation even while awake
- Brain damage
- Left heart failure
- Cardiac arrhythmias

Behavioural science

Cognitive dysfunction can occur in lung disease and this is **mainly due to hypoxia** and or concomitant hypercapnia.

mild to moderate hypoxia will affect

- **concentration,**
- **short term memory,**
- **new learning and critical judgement**.

Severe hypoxia (even transiently) will result in

- **sustained memory deficits** due to lesions in the hippocampus and anterior thalamus critical for new memory formation.
- **The motor speed and verbal fluency is also slowed.**

Chronic hypoxia results in

- impaired perceptual motor integration,
- motor speed and
- abstracting ability.

Specific pattern of cognitive dysfunction leading to

- impaired verbal tasks,
- impaired verbal memory
- preservation of visual attention.

Sleep fragmentation can cause **mood changes and a worsening of self perception** of general health status. Daytime sleepiness results in a decline in short term memory and concentration.

Pathology of Hypoxia

The brain is the first organ affected in severe hypoxia

death will result in 5 minutes if the inspired PO_2 falls below 20 mmHg.

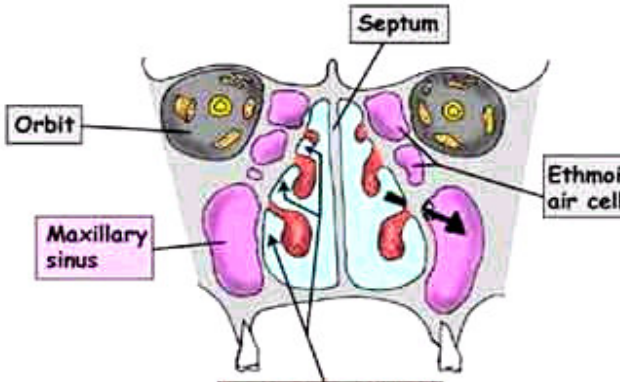
The response to hypoxia is to

- increase ventilation (by peripheral chemoreceptors),
- increased red blood cells,
- increasing the diffusing capacity of the lungs,
- increased vascularity of the tissues
- increased ability of the tissues to use oxygen despite low PO_2.
- increase of blood flow to the brain and heart at the expense of the periphery.
- increases in pulmonary arterial pressure as a result of the reflex pulmonary vasoconstriction.

An increased cardiac output increases the pulmonary blood flow and a worsening or continued hypoxia can result in cor pulmonae and right heart failure.

NASAL CAVITY BOUNDARIES & CORONAL VIEW

- Nasal cavity extends from nares to choanae (posterior
- Floor: Hard palate
- Roof: Sphenoid and ethmoid
- Medial wall: Septum
- Lateral wall: medial orbit, ethmoidal air cells, maxillary

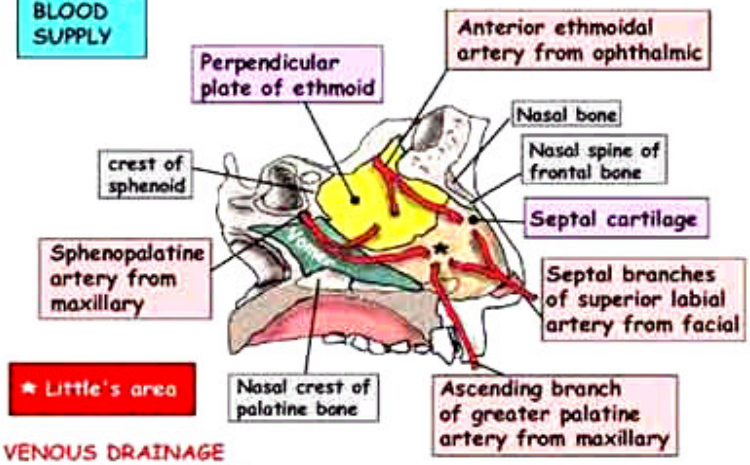


MUCOSA

Olfactory nerve
Vestibular - skin & hair
Respiratory - Pseudostratified ciliated columnar

NASAL SEPTUM

BONES & BLOOD SUPPLY



VENOUS DRAINAGE

Anterior - to face
Posterior - to pterygoid plexus. Also via ethmoidal veins to ophthalmic and inferior cerebral veins. 1% via foramen caecum to superior sagittal sinus

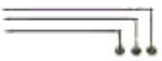
LYMPHATIC DRAINAGE

Lateral wall and septum. Posterior: to retropharyngeal and to anterior/superior deep cervical. Anterior: to submandibular

LINING

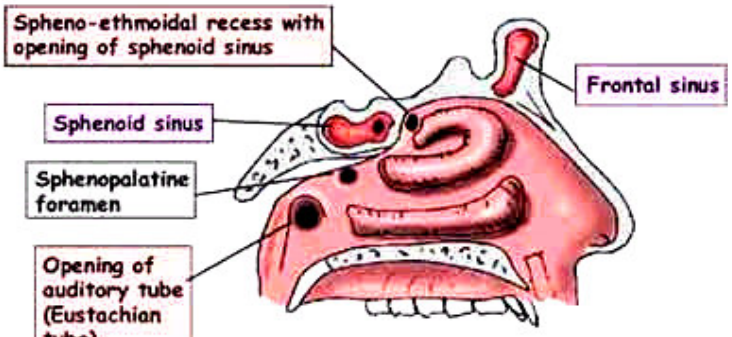
Respiratory epithelium - pseudostratified ciliated columnar with mucous cells and very vascular

Olfactory epithelium - ciliated nerve cells, yellowish, on roof & septum, under superior concha & in spheno-ethmoidal recess



LATERAL WALL OF LEFT NASAL CAV

Superior meatus
Under superior concha (cut away). Opening of posterior ethmoidal air cells



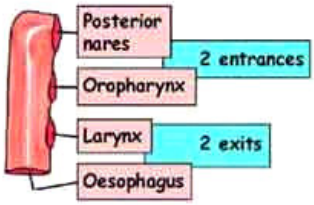
Inferior meatus
Under inferior concha (cut away). Opening of nasolacrimal duct

Middle meatus
Under middle concha (cut away). Opening of
1. Maxillary sinus
2. Middle ethmoidal sinus on bulla ethmoidalis
3. Infundibulum from frontal sinus into which anterior ethmoidal sinus usually drains (it may drain separately. The infundibulum is at the anterior end of the
4. Hiatus semilunaris

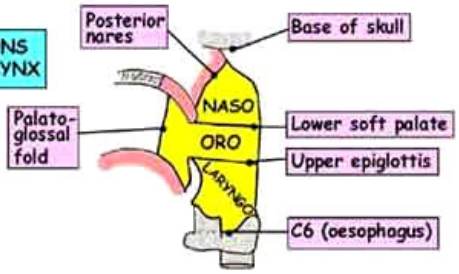
PHARYNX - DIVISIONS

- 5" (13cm) long fibromuscular tube
- Suspended from skull & anterior to prevertebral fascia
- Extends from nose to C6 (oesophagus)
- Like a mask applied to back of face
- Walls are mucous membrane, fibrous submucosa, muscle & thin buccopharyngeal fascia
- Muscles are:
  - 3 constrictors
  - Stylopharyngeus, palatopharyngeus, salpingopharyngeus
  - Note: levator palati is wholly intra-pharyngeal

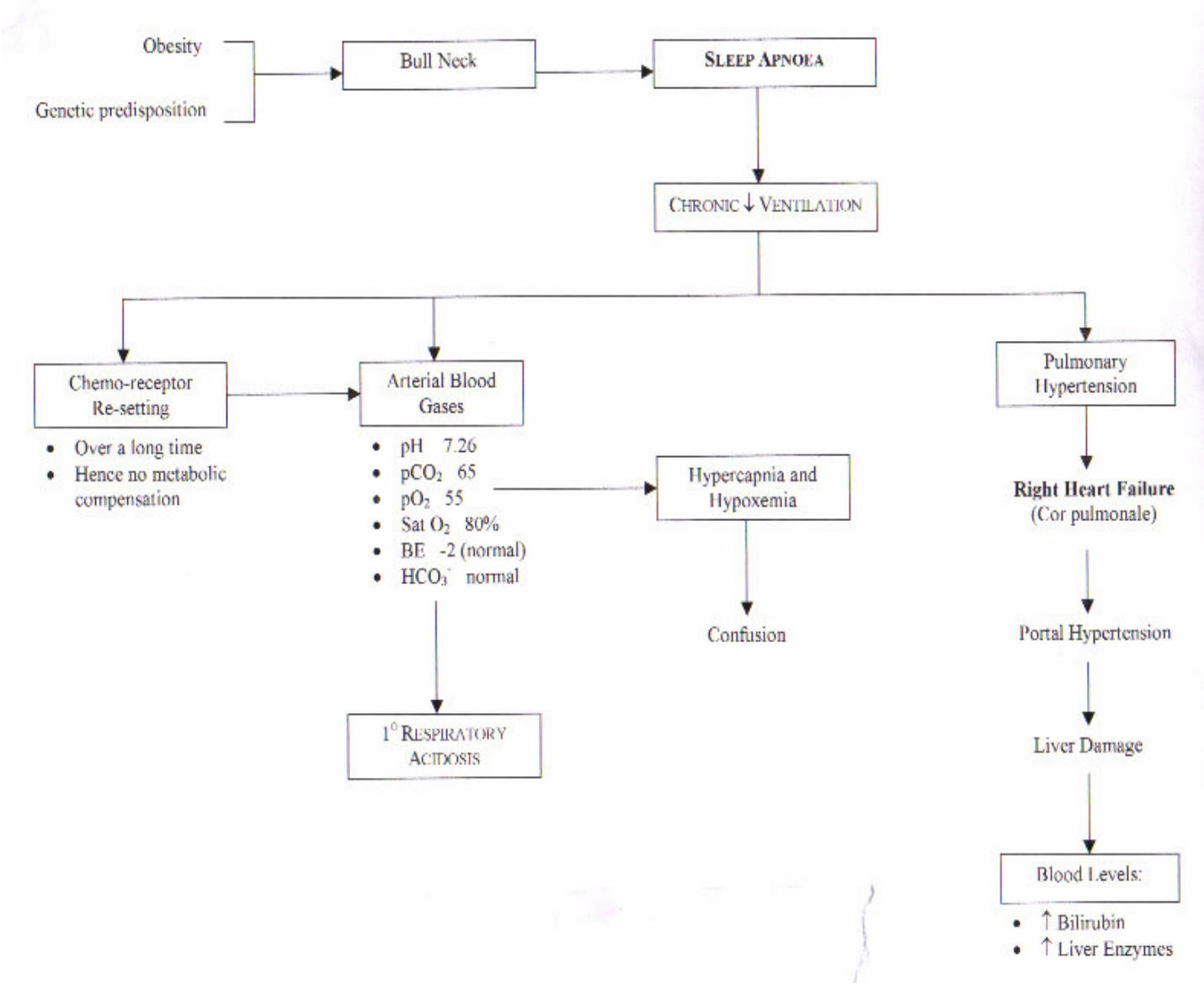
CARTOON OF PHARYNX



DIVISIONS OF PHARYNX



Mechanism:



CLASSIFICATIONS OF APNOEA:

Obstructive (OSA):

The principle event is a collapse of the pharyngeal airway during onset of REM sleep. This part of the airway relies on muscle tone to maintain its patency (no bones or cartilage). As the airway collapses the intrathoracic pressure must increase in order to overcome the barrier. This is evidenced by increasing brain and abdominal EEG activity on a polysomnogram. **The drive to breathe increases as hypoxia and hypercapnia increase and the person is roused briefly. The muscle tone and breathing thus resume.**

Central (CSA):

A less common disorder in which there is apnoea but **no increase in the drive to breathe**.

Central Apnoea results in more complete arousals than OSA.