

OBSTRUCTIVE SLEEP APNEA

Thesis

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By

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INTRODUCTION

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Obstructive sleep apnea is a bizarre disorder described relatively recently. During sleep the upper airway becomes obstructed, sometimes for well over a minute, despite vigorous attempts by the respiratory muscle to draw in a breath. The obstruction is probably due to passive collapse of the pharyngeal walls.

The muscular activity holding open the pharynx is reduced during sleep and may be unable to overcome the subatmospheric pressure generated in the airway during inspiration. Whatever the cause or the consequences are, hypoxaemia sometimes incredibly severe, and hypercapnia increase the drive to the respiratory muscles including the pharynx, which is finally pulled open with loud explosive snoring and snorting. At this point the sufferer may wake, partially or completely - a feature that may be important in terminating the apnea. The blood gas tension returns to normal after a few noisy breathes. Next the obstruction gradually returns with even louder snoring until the apnea recurs with complete silence again. [Guilleminault 1980].

This cycling of oxygen and carbon dioxide concentrations with recurrent loud snoring and arousal usually persists through the night, grossly disturbing both cardioprespiratory physiology and night sleep. Thus excessive daytime sleepiness is a prime symptom. The worst disturbances usually occur during rapid - eye - movement sleep, a particularly vulnerable period.

Many conditions may contribute to the pharyngeal collapse at night. Simple obesity with deposition of fat in the pharynx cause critical narrowing, as may other conditions such as acromegaly, myxoedema, enlarged tonsils, Scheie syndrome (Mucopolysaccharidosis), and superior vena caval obstruction. In some people no apparent abnormality can be found, and they are assumed to have inherently poor pharyngeal dilator activity during sleep. In addition, partial nasal blockage or adenoidal enlargement can lead to even lower pressures in the pharynx during inspiration and provoke collapse when it would not occur, otherwise, Combinations of all these factors may tip the balance to produce obstructive sleep Apnea, as may excessive tiredness, sedatives, and alcohol. Loud snoring without recurrent apnea and symptoms is probably a "forme fruste" that may produce development of full syndrome. The incidence of the syndrome is not known. The Stanford Sleep Institute has seen 300 causes although it is not mentioned over how many years or when.

“ Sir William Osler 1938 ” recognized the connection between obesity and hypersomnolence and commented that Joe, the fat boy in Dicken's Pickwick papers, had this problem. Osler would have approved of the fact that sleep apnoea syndrome can be diagnosed entirely on history observation. The symptoms and signs were first fully described in the obese subjects by Burwell et. al in 1956. Who labelled it a Pickwickian syndrome, through they did not appreciate the cause. “
 Eleven years later Gastaut et. al 1966 recognized that sleep apnoea was the primary cause. ”

The patient rarely reports disturbed sleep but complains of overwhelming and often crippling daytime sleepiness. The consequences of the lack of sleep include apparent intellectual deterioration, personality changes, lack of concentration, car accidents, and marital problems. Other symptoms noted less often include impotence, nocturnal enuresis, morning headaches, and abnormal motor activity during sleep to the point of sleep walking. Because of some similarities narcolepsy is often the initial diagnosis.

In the later stages of this condition other complications may appear, such as daytime hypercapnia and hypoxaemia, polycythaemia, corpulmonale, and hypertension. The usual modes of death are intractable heart failure or sudden death at night. [John R. Studling 1982].

ANATOMY

ANATOMY OF THE NOSE

External nose is pyramidal in shape. Its shape is maintained by the skeletal framework.

Anterior nares. Situated in the base of the nose and open downwards. They are separated by the columella.

Bony Constituents. Support the upper part of the external nose.

They are:

- 1 . Nasal processes of the frontal bones.
- 2 . Nasal bones.
- 3 . Ascending processes of the maxillae.

Cartilaginous Constituents. Support the lower part of the external nose. They are:

1. Upper Nasal Cartilages
- 2 . Lower Nasal cartilages.
- 3 . Quadrilateral Cartilages of the Nasal Septum, between the nasal cartilages of the two sides.
- 4 . Alar Cartilages, in the alae nasi.

The cartilages are connected with each other and with the bones by a continuous perichondrium & periosteum.

S K I N . Thin over the upper part of the nose, thicker over the lower cartilagenous part, where it contains large sebaceous glands. Vestibule is here included in the external nose because it is lined with skin and contains sebaceous glands and hairs (Vibrissae).

It is limited above and behind by a curved ridge, the Limen nasi.

Nasal Fossae . The right and left nasal fossae (cavities) are separated by the nasal septum. The nasal fossa, as here described, includes only that part which is lined with mucous membrane.

Each fossa communicates with :

1. The Paranasal Sinuses, through their ostia.
2. The Nasopharynx , through the posterior choanae.

Each posterior choana is bounded by: body of sphenoid and ala of vomer, above. Posterior margin at horizontal part of palatine bone, below.

Medial pterygoid plate at sphenoid bone, laterally. Posterior free margin of vomer, medially. Branches of the sphenopalatine artery pass medially above the posterior choana from the sphenopalatine foramen to the septum.

Each nasal fossa is bounded by :

Floor is formed by :

1. Palatine Processes of maxillae, in the anterior three-quarters.
2. Horizontal part of palatine Bones, in the posterior one - quarter.

Roof is very narrow and formed by :

1. Nasal Process of Frontal Bone, anteriorly. This slopes downwards and forwards from the highest part of the fossa.
2. Cribriform plate of ethmoid, through which fibres of the olfactory nerve pass.

3. Body of sphenoid bone, posteriorly. This slopes downwards and backwards.

Medial Wall. This is the nasal septum. The nasal septum lies in or near the midline. Crests and spurs are common.

The three main constituents are:

1. Perpendicular plate of Ethmoid, above and behind.
2. Vomer, below and behind.
3. Quadrilateral Cartilage, in the angle between 1 and 2.

These articulate with other bones which contribute in a minor way to the formation of the septum.

They are:

- a. Anterior nasal spines of maxillae.
- b. Nasal crests of maxillary and palatine bones.
- c. Rostrum and crest of sphenoid bone.
- d. Nasal spine of frontal bone,
- e. Crests of nasal bones.

Lateral Wall is formed mainly by:

1. Medial wall of Maxilla.
2. Lateral mass of ethmoid and lacrimal bone.

Other contributions are derived from:

- a. Ascending processes of maxilla, anteriorly.
- b. Perpendicular part of palatine bone and, behind it, medial pterygoid process of sphenoid, posteriorly.

The main features of the lateral wall are:

Three turbinates- superior, middle and inferior. The inferior

turbinate is the largest.

Three meatuses- named after the turbinates. Each meatus lies below and lateral to the corresponding turbinate.

Spheno - Ethmoidal recess lies above the superior turbinate and receives the ostium of the sphenoidal sinus.

Superior Meatus contains the ostia of the posterior ethmoidal cells.

Middle Meatus is the most complex and by far the most important.

The ostia of maxillary, anterior ethmoidal, and frontal sinus open into it.

The atrium is a forward continuation of the middle meatus.

The Agger nasi is a curved ridge lying above the atrium. It passes downwards and forwards from the free anterior border at the middle turbinate and may contain "agger cells".

The bulla ethmoidalis is a smooth, rounded mass formed by the anterior ethmoidal cells. The ostia of these cells open to the bulla or above it. The Hiatus semilunaris lies below and in front of the bulla and leads forwards into the infundibulum. It is bounded below by the uncinate process of the ethmoid.

Inferior meatus receives the nasal opening of the nasolacrimal duct. [John C. Ballantyne et.. al 1978].

blood supply of nose and paranasal sinuses.

Arterial supply. The nasal fossae and paranasal sinuses are supplied by branches of the external and internal carotid arteries.

1. Derivatives of External Carotid Artery.

a) Sphenopalatine artery: Via the (internal) maxillary artery supplies the turbinates and meatuses of the nose and most of the septum.

b) Greater palatine artery: A branch of the maxillary artery. contributes branches to the lateral nasal wall and (via the incisive canal) to the anterior part of the septum.

c) Superior labial artery: A branch of the facial artery. It sends branches to the tip of the septum and the alae nasi. Its anastomosis with a branch of the sphenopalatine artery and the great palatine artery (The artery of epistaxis) forms Kiesselbach's plexus in little area.

d) Infra-orbital and superior dental arteries: branches of the (internal) maxillary artery. They supply the maxillary antrum.

e) Pharyngeal branch of (internal) maxillary artery: Supplies the sphenoidal sinus.

2. Branches of Internal Carotid Artery.

Anterior and posterior ethmoidal arteries: branches of the ophthalmic artery. They supply the roof of the nose, anterior parts of the septum and lateral wall of the nose, and the ethmoidal and frontal sinuses. Bleeding from these vessels is seen above the level of the middle turbinate.

Venous Drainage. The veins form a cavernous plexus beneath the mucous membrane.

They open into:

1. Sphenopalatine vein and anterior facial vein, from the plexus.
 2. Ophthalmic veins, from the ethmoidal veins
 3. Veins on the orbital surface of the frontal lobe of the brain, through foramina in the cribriform plate.
 4. Superior sagittal sinus, through the foramen caecum (when the latter is present) . [John C. Ballantyne et...al 1978].
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