ANATOMY & PHYSIOLOGY OF UPPER AIRWAY OBSTRUCTION

It is known that there is upper airway obstruction during sleep in patient with obstructive sleep apnoea. How it happens, one can appreciate better if anatomical and physiological consideration in upper airway is clearly understood. Technically, the upper airway includes the extra thoracic trachea, larynx, pharynx and nose. The area of great interest in the present context is the pharyngeal airway, segment bounded by cranially by the nasopharynx and caudally by the glottis chink. It is the site of airway closure or narrowing in the upper airway during sleep.

Pharynx is multipurpose passage and a common pathway for respiratory, digestive and phonation functions. With the exception of the two ends of the airway, i.e. the nares and small intrapulmonary airways, the pharynx is the only collapsible segment of the respiratory tract. Pharynx remains open at all times, except during momentary closures associated with swallowing, regurgitation, eructation and speech. It can occlude in unconscious individuals and also during sleep.

The pharyngeal patency during wakefulness is largely controlled by higher nervous system. During sleep this supervision is disrupted to some extent and defence of patent airway is compromised. If there is anatomic abnormality, server narrowing or closure of pharyngeal airway can occur during sleep resulting into obstructive sleep hypopnoea. Obstructive sleep apnoea constitutes extension of obstructive sleep hypopnoea to the limit at which upper airway resistance become infinite. Pharyngeal obstruction during sleep can be periodic and non-periodic. In non-periodic airway obstruction, patient displays a rather stable elevated respiratory resistance associated with sustained arterial oxygen desaturation and repetitive large respiratory efforts. The upper airway resistance may increase as high as 75 cm H2O/ litre/sec.

The 'BALANCE OF PRESSURES CONCEPT' as proposed by Remmers (15) and Brouillete and Thatch (16) is pertinent in understanding the mechanism of pharyngeal collapse during sleep. The size of the pharyngeal lumen depends on the

balance between outward forces developed by actively contracting pharyngeal muscles and inward forces resulting forces resulting from sub atmospheric luminal pressure during inspiration.

A more comprehensive theory considers transluminal pressure (Ptm), the difference between luminal and tissue pressure (Ptm=Pi – Pti). Ptm determines the size of the pharynx in accordance with a 'tube law' for the pharynx. When Ptm increases, the cross sectional area of passive pharynx increases according to a Ptm-area relationship referred to as tube law for the passive pharynx.

Pi: It is defined as intraluminal pressure acting on the luminal surface of the pharyngeal wall that is lateral pressure. It is different than atmospheric pressure during airflow. During inspiration, Pi decreases because of the dissipation of energy of at upstream resistance and because of acceleration of gas flowing through a narrowed airway. During application of N-CPAP, Pi increases thereby increasing Ptm.

Pti: It is defined as tissue pressure acting on the outside surface of the pharyngeal wall. Neck compression from outside, submendibular fat and large tongue can influence the Pti.

P Close: It is often referred to as closing pressure represent the maximal pressure associated with no lumen. The curvilinear nature of the tube law for pharynx means that its slope (dA/ dPtm) often referred to as compliance varies with calibre (Figure – 4 a,b,c).

There is shift in tube law when passive pharynx becomes active. When upper airway dilator muscle Genioglossus contracts, there is increase in luminal area for the same values of Pi and Pti by providing a muscle pressure (Pmus) that acts on the outside of the pharyngeal wall. Obstructive sleep apnoea patients have significantly greater genioglossus activity compared with normal subjects, while they are awake. It shifts the area pressure relationship upward and making it flatter and thus decreasing dA/dPtm. This shift in tube law compensates for anatomic factors tending to reduce the size of pharynx in these patients. It is well documented that in awake subjects, pharyngeal size increases during inspiration and decreases during expiration. This phenomenon reverses during sleep.

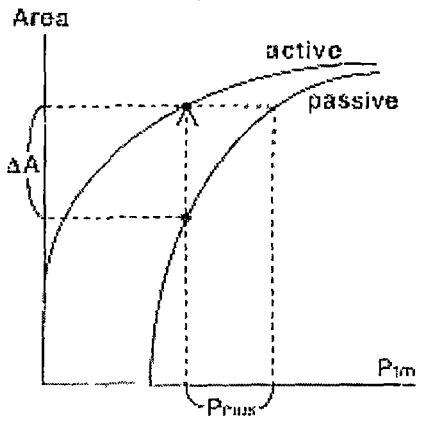


Fig4 (a):showing that contractions of pharyngeal dilators increases the cross sectional area resulting from a change in the tube law of the pharynx

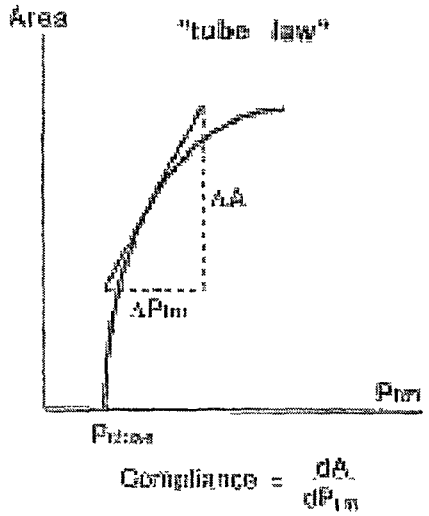
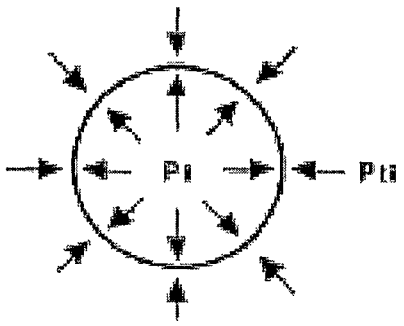


Fig 4(b): showing that increase in Ptm results in an increase cross sectional area in accordance with tube law of pharynx, slope represent compliance



$$Ptm = Pi - Pti$$

↑ : Area ↑ : Ptm ↓ : Area ↓

Fig4(C)- The concept of Transmural pressure and tube law, Ptm—transluminal pressure, P1 – Intraluminal pressure, pti- tissue pressure

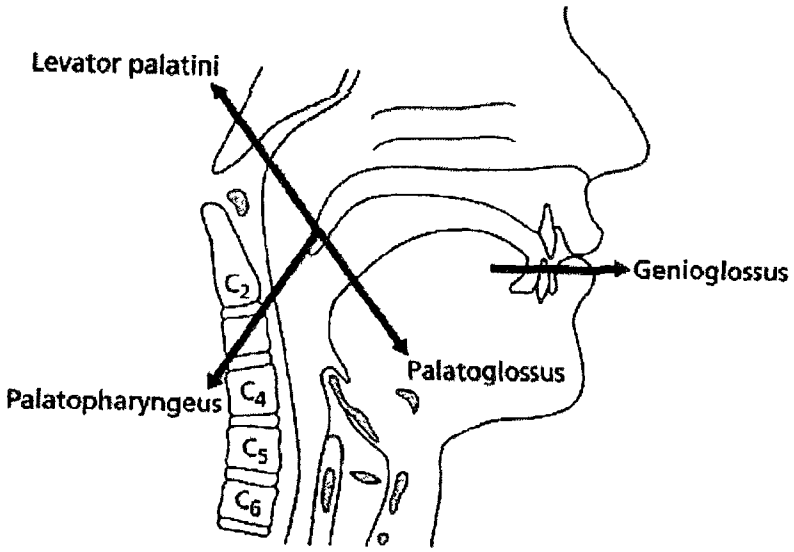


Fig 5: Diagram showing pharyngeal dilator muscles crucially placed to maintain upper airway patency

The control of upper airway size and stiffness depends on the relative contraction of host of paired muscles, which includes palatal, pterygoid, genioglossus, intrinsic pharyngeal and hyoid muscles. The medial pterygoid, tensor, palatani, genioglossus, geniohyoid and sternohyoid all tend to promote a patent pharyngeal lumen and receive phasic activation during inspiration (figure - 5).

Pharmacological agents that tend to depress the reticular activating system such as anaesthetic, hypnotics, diazepam and ethanol decreases motor output to the genioglossus muscle. Propioceptive feedback can also influence this motor output. The perhaps the most convincing evidence demonstrating the overall importance of neuromuscular factors in maintenance of airway potency is that obstructive sleep apnoea abnormally high in patients with this disorder while they are awake, the resistance rises dramatically on going to sleep.Does a neural, sleep related neuromuscular abnormality contribute to obstructive sleep apnoea? No evidence at present indicates the existence of a primary neural factor but this simply reflects our inability to assess this sort of abnormality. Nonetheless, evidence implicates the nervous system as a secondary contributor in the pathogenesis of obstructive sleep apnoea.

If a neural factor does not play a primary in initiating obstructive sleep apnoea, what does? The theory of cyclic alterations in pharyngeal P mus, caused by changes in central nervous system states of vigilance related to periodic changes in blood gases does not sufficiently explain why certain individuals have this disorder whereas others not. One hint of non-neural factors is that changes in body weight frequently produce substantial changes in severity of obstructive sleep apnoea (18). Factors determining pharyngeal narrowing during sleep are shown below in figure – 6.

There are certain mechanical factors like compliance, surface adhesive forces, pharyngeal lumen pressure and effects of geometry of the pharynx on pressure and flow are to be considered in understanding the pathophysiology of obstructive sleep apnoea. Nasopharynx (soft palate) is the commonest site of obstruction in patients with obstructive sleep apnoea during sleep. According to the extent of narrowing at P close, sites of narrowing are defined as primary sites (more than 75% reduction of the area from the control value) or secondary sites (25% to 75% reduction) shown in Figure – 7.

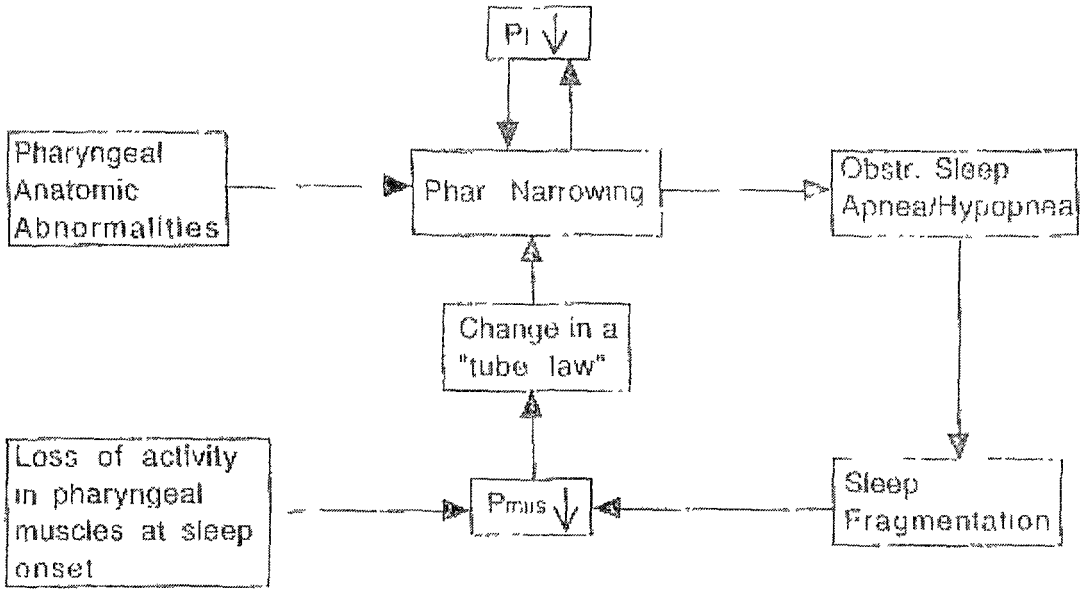
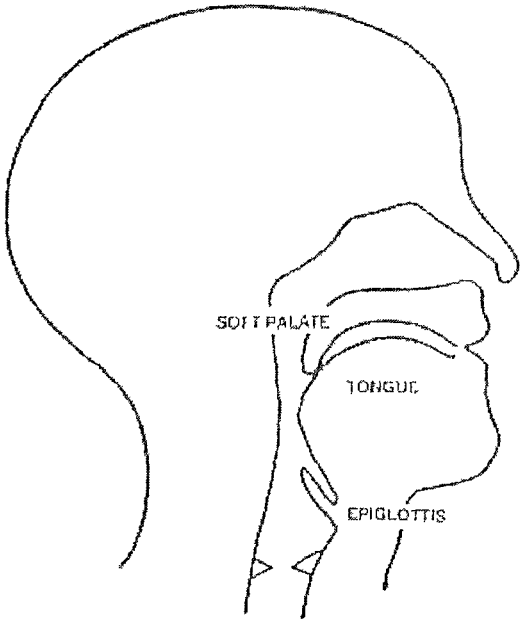


Fig- 6: Flow Diagram showing factors affecting pharyngeal lumen



	Primary	Secondary
NP	81 %	8 %
OP	38 %	25 %
HP	22 %	33 %

(64 OSA patients)

Fig- 7: Diagram showing common sites of obstruction in obstructive sleep apnoea

In spite of much understanding of sleep, still we do not know why does airway behave differently during sleep? Are there primary neural factors causing sleep related neuro muscular disorder? Role of pharyngeal dilators singularity or together is yet to be established.