

# Physiology of the Upper Airway

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Human subjects do not usually snore during wakefulness. Why do they develop snoring or obstructive apnea only when they lose consciousness? My discussion on physiology of the upper airway (UA) obstruction begins with the simple question and extends to contribution of impairment of neural compensatory mechanisms and/or UA anatomical abnormalities during unconsciousness to UA obstruction.

The audience will learn developmental changes of UA maintenance and pathophysiology of UA obstruction during sleep and anesthesia.

Followings are the topics to be presented in my talk.

- 1) Two important factors determining UA maintenance (Reference 1,2)
  - Neuromuscular control of the UA
  - Anatomical properties of the UA
- 2) Balance of forces model explaining the neural and anatomical interaction (Reference 1,2)
- 3) Our unique method to separate neuromuscular factor from anatomical factor (Reference 3)
  - Total muscle paralysis under general anesthesia
- 4) Developmental changes of UA collapsibility
  - Anatomical properties (Reference 3,4,5)
    - Neonates: most collapsible
    - Infants: collapsibility decreases within a year
    - Infancy to childhood: further decreasing collapsibility
    - Childhood to adulthood: increasing collapsibility
  - Neuromuscular mechanisms (Reference 6,7,8)
  - Operating in infants and children (more important in infants)  
(Reference 9)
  - Not operating in adults  
(Reference 10)

- 5) Two major hypotheses for pathogenesis of obstructive sleep apnea (OSA)
  - Anatomic hypothesis: Abnormal UA structures (proved))
  - Neural hypothesis: Impaired neural regulation of the UA muscle activation (not tested)
- 6) Conclusive evidence for the anatomic hypothesis
  - Closing pressures of the passive pharynx in paralyzed apneics were significantly greater than those in age and BMI-matched normals. (Both in adults and children) (Reference 3,4)
- 7) A mechanical model for understanding physiology of UA obstruction
  - Balance between the amount of soft tissue inside the mandibular enclosure (meat) and the size of the mandibular enclosure (container) determines the passive UA size. (Reference 11)
- 8) Mechanical factors influencing the collapsibility of the passive pharynx
  - Mandibular position (forward movement) (Reference 12)
  - Neck position (flexion, extension, and rotation)
  - Mouth opening
  - Body position (supine, sitting, lateral, and prone) (Reference 13,14)
  - Lung volume

### **Implications for pediatric anesthesia**

- 1) UA managements during anesthesia for neonates:
  - Preservation of neuromuscular compensatory mechanisms is crucial in neonates. Therefore, consider awake tracheal intubation in neonates, and extubate the tube only when residual anesthetic effects are eliminated.
- 2) UA managements during anesthesia for children
  - The UA rarely closes completely without neuromuscular compensation in children without sleep-disordered breathing, and therefore these children do require but little airway support. Increased diaphragm muscle contraction, particularly during slow induction of anesthesia, would result in increase in UA collapsing forces and therefore UA obstruction even in these children.

- 3) Mechanical support for patent airway during induction of anesthesia and after tracheal extubation  
Increase mandibular enclosure size by advancing the mandible or extending the neck.  
Sniffing position is preferable for airway maintenance. Consider lateral or sitting position after extubation if possible.

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