Modeling The Apneic Upper Airway

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Sleep Apnea

Obstructive Sleep Apnea Syndrome (OSAS) is a medical condition characterized by disruptions in sleep due to the partial or complete collapse of the upper airway. OSAS is quantified by the apnea-hypopnea index (AHI), which is the number of apneic events per hour. It affects 1 in 4 men and 1 in 9 women and has short term symptoms including fatigue, cognitive dysfunction and weight gain, and long term systems including hypertension, stroke and heart failure.

Respiratory System Background

The body's respiratory system utilizes a natural control system to protect the body from blood oxygen deprivation (hypoxia) or elevated blood carbon dioxide levels (hypercapnia). Neurochemical signals respond to carbon dioxide concentration in the blood, evoking arousal when they are above a threshold. Apneic events cause brain arousal to reverse oxygen deprivation. Apneic events are destructive since they prevent fully restorative sleep.

Objective

The aim of this project is to augment an existing system model [1] of respiratory control and sleep apnea to be more physiologically accurate. The upper airway is modeled as a collapsible tube. The pressure, area and velocity profile areobtained from fluid mechanics fundamentals and a tube law [2].

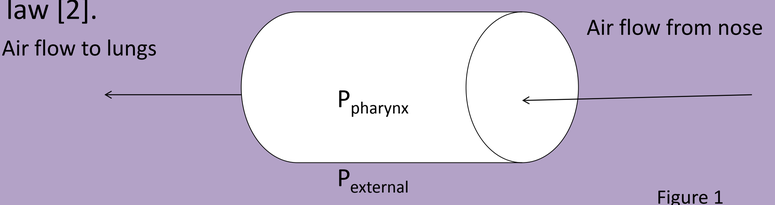

Figure 1

When $P_{transmural}$ ($P_{transmural}$= $P_{external}$ –$P_{pharynx}$) is negative, the airway is subject to collapse. The goal is to implement fluid mechanics and a tube law to model this behavior.

Existing Systems Model

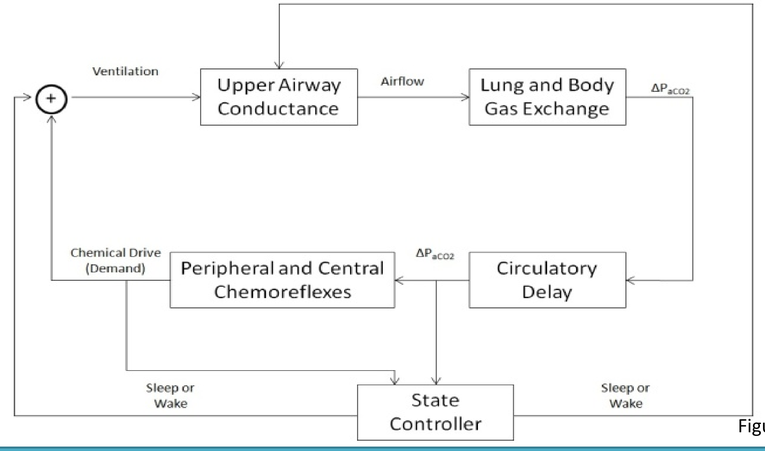

Figure 2

Motivation

The basis of the sleep apnea model is a systems respiratory model, which uses negative feedback control (Fig 2) to model respiration during sleep. Blood gas concentration and sleep/wake state are the controllers of the ventilation system. The system is outlined visually in Fig. 3.

• The model simulates the "upper airway conductance" with a numerical parameter that gradually reduces airflow without any physiological cause, thereby reducing blood oxygen concentration and stimulates an arousal from sleep. In actuality, arousal is triggered by a decrease in blood oxygen concentration, which is caused by collapse of the upper airway.

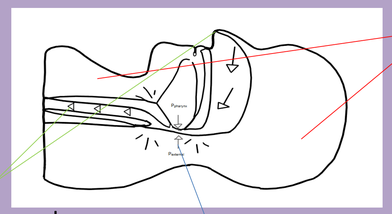

Figure 3

1. Chemoreceptors in carotid body and brain sense P_{CO2}

2. To meet demand, lungs and nose increase pressure to increase ventilation

3. Low muscle tone causes upper airway to be vulnerable to collapse

Figure 2: Schematic of Breathing Mechanisms

Neural
Muscular
Respiratory

Tube Law & Fluid Mechanics

Fluid Mechanics and Tube Law:

$$S = \frac{u}{c} \qquad p = \alpha^{n1} - \alpha^{n2}$$

$$c^2 = \frac{A}{\rho}\frac{\partial P}{\partial A} \qquad -\frac{1}{\rho}\frac{\partial p}{\partial x} = u\frac{\partial u}{\partial x}$$

A_0 = resting area — p = transmural pressure

A = deformed area — u = air speed

c = wave speed — ρ = air density

$$\alpha = \frac{A}{A_0}$$

Equations for deriving properties of upper airway flow. Resistance to flow, or flow rate, will be integrated into systems model.

$$\frac{\partial \alpha}{\partial x} = \frac{\alpha}{1-S^2}\left[\frac{S^2}{A_0}\frac{dA_0}{dx} - \frac{P}{\rho c^2}\frac{dK_p}{dx}\right]$$

$$\frac{\partial S^2}{\partial x} = \frac{S^2}{1-S^2}\left[\frac{[-2+(2-M)S^2]}{A_0}\frac{dA_0}{dx} + \left(\frac{MP}{\rho c^2} - \frac{1-S^2}{K_p}\right)\frac{dK_p}{dx}\right]$$

$$M = 3 + \alpha\left[\frac{d^2P}{d\alpha^2}\Big/\frac{dP}{d\alpha}\right]$$

Future Work

The goal is to integrate a tube law describing the behavior of the upper airway into a respiration systems model (Fig. 2). To achieve expected results, the tube law can be implemented with an added friction factor. In addition, other numerical methods of integration of partial differential equations can be explored. Also, the model should be validated and synthesized with actual patient data.

References

[1] Khoo, Michael C. K. *Physiological Control Systems: Analysis, Simulation, and Estimation*. New York: IEEE, 2000. Print.
[2] Ku, "One-Dimensional Steady Inviscid Flow Through a Stenotic Collapsible Tube", Journal of Biomechanical Engineering, 1990.