ANALYSIS OF NON-INVASIVE VENTILATION EFFECTS ON GASTRIC INFLATION USING A NON-LINEAR MULTIPLE-CYCLE MODEL

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Abstract: A mathematical nonlinear model of the esophagus was developed to analyze effects of non-invasive ventilation parameters on the severity of gastric inflation. The model is based on the nonlinear physical characteristics of biological tissue. Model predictions are congruent with the European Resuscitation Council's (ERC) recommendation in its 2000 guidelines for adult basic and advanced life support of a decrease in the tidal volume and extension of inspiratory time, in an effort to minimize gastric inflation. The model also establishes a strong correlation between the relief time between consequent ventilations and the occurrence of gastric inflation.

Introduction

Gastric inflation (GI) is a potential risk of non-invasive ventilation (NIV) during cardiopulmonary resuscitation (CPR), especially when the airway is unprotected. GI compresses the lungs, thus decreasing their compliance and forcing higher airway ventilation pressure. The latter is also associated with increased risk of GI, thus creating a vicious cycle. Serious complications of GI are regurgitation of gastric contents and pulmonary aspiration, leading to higher mortality rates within 24 - 96 hours after successful resuscitation.

The distribution of ventilation volume between lungs and stomach in the unprotected airway depends on patient variables such as lower esophageal sphincter (LES) pressure, airway resistance and respiratory system compliance, and the technique applied while performing basic or advanced airway support, such as head position, inflation flow rate, inspiratory time (TI) and tidal volume [1,2]. Airway pressure depends on inspiratory time, airway resistance and tidal volume. Large tidal volumes, high airway resistance and short TI's lead to higher airway pressure and increased probability of air entering the stomach. During cardiac arrest, LES relaxes and requires a smaller pressure gradient to open, thus making GI more likely also at relatively low airway pressures.

In an effort to minimize gastric inflation, the European Resuscitation Council (ERC), in its 2000 guidelines for Adult Basic Life Support [3], recommended a decrease in the tidal volume during non-invasive ventilation. According to the revised guidelines, it is recommended that for adult resuscitation each rescue breath (without supplemental oxygen) should deliver a volume of 700-1000 ml slowly (over about 2 seconds), or a volume of 400-600 ml over 1-2 seconds when supplementary oxygen is available. If the airway is unprotected (e.g., when using a bag-valve-facemask) the ERC states that even smaller tidal volumes can be used, as they provide adequate oxygenation with a reduced risk of GI.

During adult cardiopulmonary resuscitation, two main ventilation techniques are used: (a) Combined ventilations and chest compressions, in 2:15 ratio, at approximately 15-seconds sets. Each set consists of 2 ventilations followed by 15 chest compressions. This technique is used during adult cardiopulmonary resuscitation when signs of spontaneous circulation are not detected; (b) Single ventilation at 4-5 seconds cycles (1:5). This is the common strategy of ventilation during basic cardiopulmonary resuscitation (rescue breathing) when signs of circulation were detected or after the restoration of spontaneous circulation (ROSC).

To study the effects of different NIV parameters on gastric inflation, we developed a mathematical model of the primary components that are involved in the procedure. The multi-element nonlinear model includes 25 serially connected esophageal subsections.

Materials and Methods

A 25 cm-long esophagus was modelled as a network of 25 time-invariant, non-linear serially connected segments, where each segment represents 1 cm of the esophageal length and characterized by two elements (Figure 1, left panel). Rx represents the longitudinal resistance to airflow. Ceso is esophageal wall compliance and is described by a polynomial based on physiological nonlinear pressure-volume relation (Figure 2). Paw is the pressure at the airway opening.

At the distal end of the esophagus, the LES (Lower Esophageal Sphincter) resistance to longitudinal flow (R_{LES}) was modelled as a volume-dependant resistor following equation 1:

$$R_{LES}(t) = \frac{8\eta L}{\pi r_{LES}^4(t)} = \frac{K}{(V_{LES}(t))^2} \qquad (1)$$

where L is the length of the LES, r is the LES radius, and η is the viscosity of air. K is a resistance coefficient equals to $8\eta\pi L^3$. $V_{LES}(t)$ is the volume of air in the LES.

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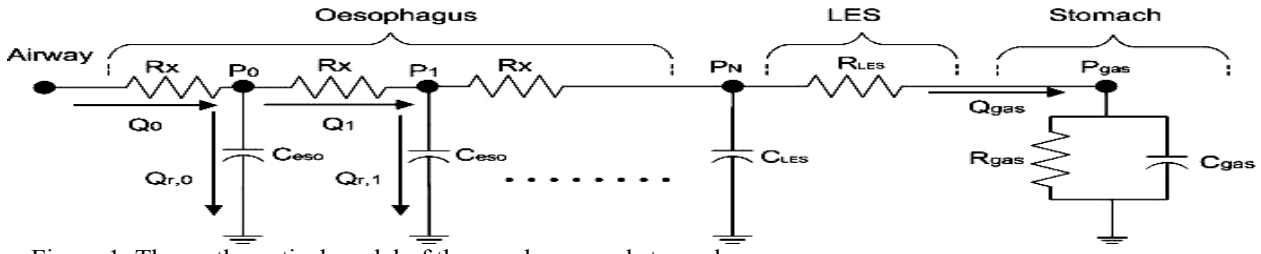

Figure 1: The mathematical model of the esophagus and stomach.

The stomach (Figure 1, right panel) was modelled as a terminating compartment in the form of a parallel resistor (Rgas) and large capacitor (Cgas). Pgas is the gastric pressure.

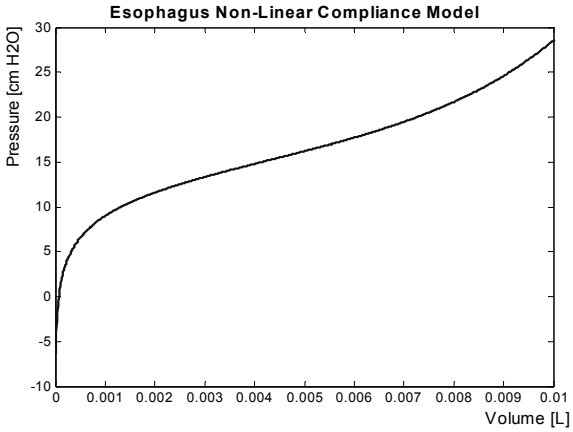

Figure 2: Esophageal subsection pressure to volume relation.

Longitudinal airflow along the esophagus can be calculated according to the pressure gradient between successive segments according to equations 2-4:

$$Q_n(t) = \frac{1}{R_x}(P_n(t) - P_{n+1}(t)) \quad (2)$$

$$P_n(t) = f\{V_n(t)\} \quad (3)$$

$$Q_{r,n}(t) = Q_n(t) - Q_{n+1}(t) \quad (4)$$

where Pn(t) and Qn(t) are the longitudinal pressure and flow in the nth segment at time t, respectively. Qr,n(t) is the radial flow of the nth segment – the flow required to inflate or deflate a segment.

Gastric air volume was calculated as the integral of flow into that section. Inertance of air inside the esophagus was neglected. To achieve accurate results, time interval (ΔT) was set to 1 msec.

Results

We used our model to simulate intra-esophageal pressures during adult basic cardiopulmonary resuscitation according to the latest guidelines of the American Heart Association and European Resuscitation Council. The effects of the main techniques of ventilation on GI were quantified. Airway pressure was set to 25 cm H2O during ventilation. With the 2:15 technique expiration relief time-intervals (RT) with atmospheric pressure between consequent ventilations were set to 1 seconds. With the 1:5 ventilations/seconds technique the time between subsequent ventilations was set to 4 second.

The effect of ventilation pressures on the time delay until gastric inflow begins is quantified in Figure 3. The interval between initiation of ventilation and first gastric incoming flow is presented as function of the pressure at the airway opening. Note that the curve defines an upper bound for TI. We defined this interval as the 'safe inspiratory time' (SIT).

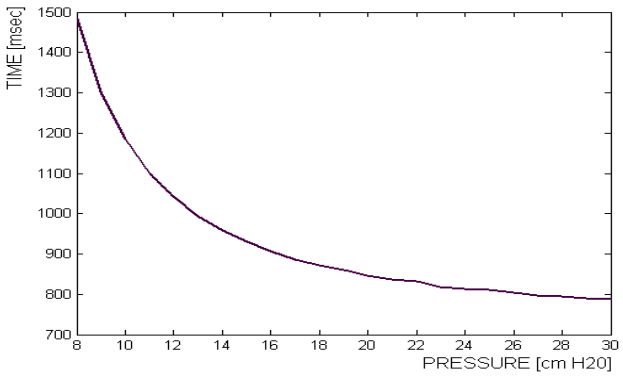

Figure 3: The relationship between the pressure at the airway opening and the safe inspiratory time (SIT).

The model was tested with varying RT's between consequent ventilations. As shown in Figure 4, under a threshold ventilation pressure of 12 cm H2O, RT and SIT are linearly dependant (r=0.98). At this range long RT allows increased SIT (a≈0.56). Linear correlation between SIT and RT abruptly changes when airway pressure exceeds 12 cm H2O, indicating that air enters the stomach during the first ventilation.

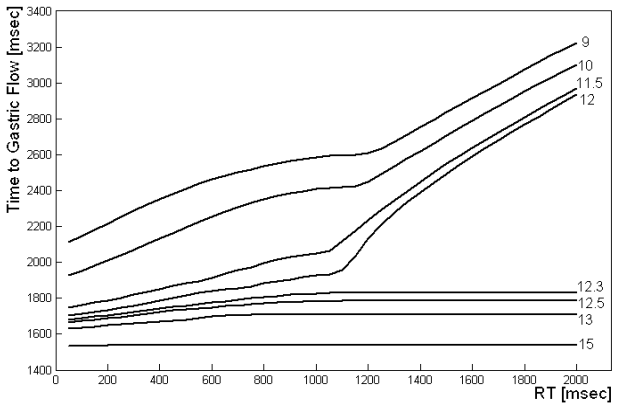

Figure 4: SIT as function of RT at different ventilation pressures.

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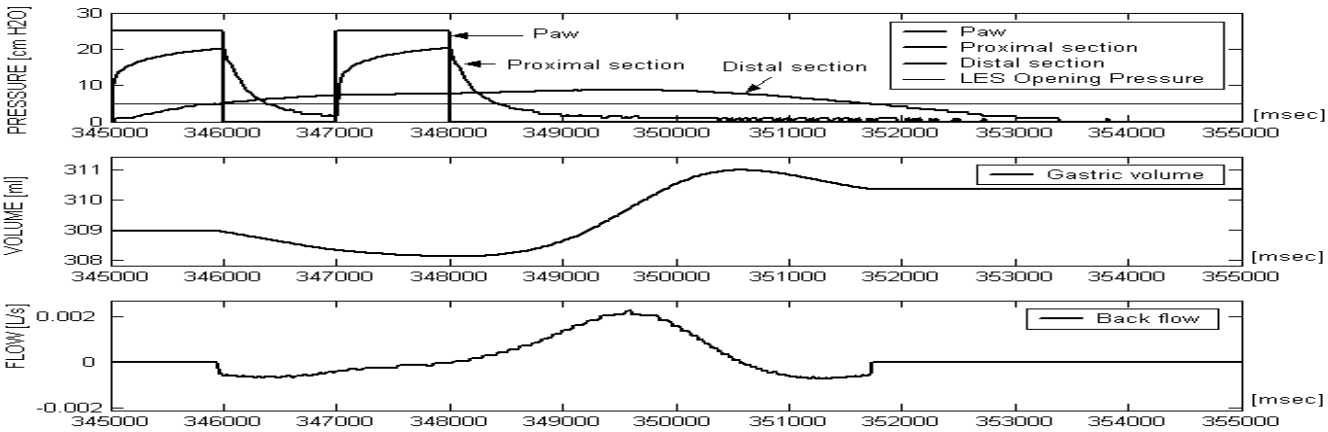

Figure 5: The 2:15 technique after prolonged ventilation. The interval when esophageal distal section pressure is above LES opening pressure is approximately 5.5 seconds, thus allowing greater amount of air to enter the stomach, even at relatively low airway pressures.

Esophageal pressure, gastric added volume and gastric flow with the 2:15 ventilations/ seconds technique are presented in Figure 5. When the stomach fills with enough air, its pressure equals the LES pressure. Under these conditions backflow from the stomach occurs. After prolonged ventilation, mean pressures at the distal section and the amplitude of backflow are higher with the 2:15 technique compared to the 1:5 technique.

Discussion

To evaluate the effect of different NIV parameters on the development of gastric inflation, a computer-simulation model of the esophagus was developed and presented. The model defines the duration of the relief time between consequent ventilations as a significant parameter affecting the development of gastric inflation. Using this model, different NIV ventilation techniques can be simulated to determine optimal modes of ventilation that will avoid massive gastric inflation resulting in life-threatening pulmonary complications.

Although numerous studies in the field provide extensive clinical results that depict a clear understanding of the parameters influencing GI, most researchers focus solely on a clinical observation and do not provide a solid model capable of explaining and predicting the occurrence of GI in a given scenario. Even studies that used mechanical models of the respiratory system and esophagus rarely take esophageal elasticity into account [4].

We were able to satisfactorily reproduce results observed in previously detailed experimental trials. Melker [5,6], who used a mechanical model of the airway and upper gastrointestinal tract, suggested that gastric inflation could be avoided if longer inspiratory time is used, resulting in lower peak inspiratory pressure and peak inspiratory flow rate. Weiler [7], similar to Melker, concluded that airway pressures higher than 20 cm H2O should be avoided and longer inspiratory time of four times the respiratory time constant should be used. The increase in the occurrence of gastric inflation due to high inspiratory pressures (Figure 3), as predicted by the model, is similar to experimental results reported by both Melker and Weiler. Model predictions are also congruent with the results reported by Wenzel [8] regarding the relation between gastric inflation and the decrease of LES opening pressure.

Our model establishes a linear dependence between the relief time between consequence ventilations (RT) and the occurrence of gastric inflation. According to the results of Figure 4, when ventilating with airway pressures lower than the threshold pressure, linear relation exists between RT and SIT. With the 1:5 technique each ventilation cycle can be considered to be independent since air deflates before the next cycle starts, resulting in time-invariant initial conditions along the esophagus at the beginning of each ventilation cycle. In the event where two (or more) ventilations are performed continuously (as may happen during cardiopulmonary resuscitation, with the 2:15 technique) when the subsequent ventilation starts the esophagus may already contain some air. In this case, intra-esophageal pressure increases faster and SIT shortens, thereby allowing greater amount of air to enter the stomach, even at relatively low airway pressures. This can also explain how the phenomenon of gastric inflation can be associated with hyperventilation, since it shortens RT and SIT, and raises esophageal mean pressure, therefore allowing more air to enter the stomach. This conclusion is supported by results of a multiple-cycle analysis presented in Figure 5.

The phenomenon of patient regurgitation during cardiopulmonary resuscitation is not yet completely understood. Under normal conditions, vomiting is a reflex controlled by medullary centers (i.e., the vomiting center). However, during critical care conditions these centers may be dysfunctional; therefore, physical stimuli may have greater effect. Our model may provide such a physical explanation. Under the 1:5 ventilation/seconds technique, air is accumulated in the stomach in a relatively constant and small rate. When the stomach fills with enough air, its pressure equals the

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LES pressure. Under these conditions backflow from the stomach will occur, possibly resulting in regurgitation. This kind of a simple model can serve as useful tool for simulating the likelihood of patient regurgitation during cardiopulmonary resuscitation.

Some limitations of the model design should be noted. The model was designed to be simple and useful and parameters' values have been carefully determined. However, the model cannot mimic precisely the mechanics of an in vivo human esophagus during cardiopulmonary resuscitation. To ensure usefulness of the model predictions, only the major factors determining the development of gastric inflation were included. Due to their secondary effect, lung compliance and respiratory time constant were not considered.

Conclusions

Our model predictions are congruent with the European Resuscitation Council's (ERC) recommendation in its 2000 guidelines for adult basic and advanced life support of a decrease in the tidal volume and extension of inspiratory time, in an effort to minimize gastric inflation. Our model also establishes an important relationship between the relief time between consequent ventilations and the occurrence of gastric inflation. Using this model, different NIV ventilation techniques can be evaluated to determine optimal modes of ventilation that will avoid massive gastric inflation resulting in life-threatening pulmonary complications. The study also points out the advantages of a simple non-linear multi-cycle model for simulating complex mechanical phenomena such as gastric inflation.

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