Fast or Slow Rescue Ventilations: A Predictive Model of Gastric Inflation

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BACKGROUND: Rescue ventilations are given during respiratory and cardiac arrest. Tidal volume must assure oxygen delivery; however, excessive pressure applied to an unprotected airway can cause gastric inflation, regurgitation, and pulmonary aspiration. The optimal technique provides mouth pressure and breath duration that minimize gastric inflation. It remains unclear if breath delivery should be fast or slow, and how inflation time affects the division of gas flow between the lungs and esophagus. METHODS: A physiological model was used to predict and compare rates of gastric inflation and to determine ideal ventilation duration. Gas flow equations were based on standard pulmonary physiology. Gastric inflation was assumed to occur whenever mouth pressure exceeded lower esophageal sphincter pressure. Mouth pressure profiles that approximated mouth-to-mouth ventilation and bag-valve-mask ventilation were investigated. Target tidal volumes were set to 0.6 and 1.0 L. Compliance and airway resistance were varied. RESULTS: Rapid breaths shorter than 1 s required high mouth pressures, up to 25 cm H2O to achieve the target lung volume, which thus promotes gastric inflation. Slow breaths longer than 1 s permitted lower mouth pressures but increased time over which airway pressure exceeded lower esophageal sphincter pressure. The gastric volume increased with breath durations that exceeded 1 s for both mouth pressure profiles. Breath duration of ~1.0 s caused the least gastric inflation in most scenarios. Very low esophageal sphincter pressure favored a shift toward 0.5 s. High resistance and low compliance each increased gastric inflation and altered ideal breath times. CONCLUSIONS: The model illustrated a general theory of optimal rescue ventilation. Breath duration with an unprotected airway should be 1 s to minimize gastric inflation. Short pressure-driven and long duration-driven gastric inflation regimens provide a unifying explanation for results in past studies. Key words: basic life support; gastric inflation; rescue breathing; tidal volume; unprotected airway; ventilation.

Introduction

Positive-pressure ventilation is an essential component of resuscitation from respiratory and cardiac arrest. Health care providers and lay rescuers may be called on to administer rescue breaths by mouth-to-mouth ventilation with exhaled air or by manual ventilation with air or oxygen with a bag-valve-mask.1,2 Tidal volumes must be large enough to assure oxygen delivery; however, applying high airway pressures that exceed the lower esophageal sphincter pressure can force ventilating gas into the stomach.3,4 Gastric inflation increases the risk of regurgitation and aspiration of stomach contents,5 particularly in patients who received concurrent chest compressions for cardiopulmonary resuscitation,6 victims of drowning who swallowed water,7 and cases in which lower esophageal sphincter pressure dropped severely due to prolonged hypoxia.8 An endotracheal tube should be inserted to protect the upper airway in these situations, particularly if reduced respiratory system compliance due to lung injury necessitates higher airway pressures; however, placement delays ventilation in victims who are already severely hypoxic, and responders may not be trained or equipped for intubation.9 Gastric inflation could be minimized by performing compression-only cardiopulmonary resuscitation; however,
Rescue Ventilations and Gastric Inflation

The adequacy of passively induced tidal volumes ($V_T$) is not assured with this technique, and victims of primary respiratory arrest may be severely hypoxic and need active ventilation.17

Historically, mouth-to-mouth ventilation was unpopular and rarely used until Elam et al.,12 Safar and Elam,13 and Safar et al.14 conducted studies on human volunteers in the 1950s, which demonstrated superior gas exchange compared with manual chest-pressure arm-lift methods. Emphasis at that time was on delivering as large a $V_T$ as possible, which sometimes exceeded 1.5 L, with little concern about the risk of gastric inflation.14 It was recognized from experience with anesthetics that regurgitation could cause aspiration, and this remains a concern during resuscitation.6,15,16 More recent studies confirmed that a smaller $V_T$ reduces gastric inflation when the airway is unprotected.17-20

Basic life support guidelines, therefore, have been revised to recommend lower $V_T$ so as to limit peak airway pressures and reduce the risk of gastric inflation.15 $V_T < 500$ mL caused unacceptably low arterial oxygen levels in patients who were anesthetized and arrest victims who received cardiopulmonary resuscitation, and resulted in intolerable hypoxia and hypercarbia in awake volunteers.17 Present recommendations are for $V_T$ of 0.5–0.6 L to be given over 1 s.1,2 Rescuers who performed simulated mouth-to-mouth ventilations on a mannequin often delivered $V_T$ of < 0.4 L, whereas those who performed bag-valve-mask ventilations tended to hyperventilate patients during actual arrest situations.24 Tighter control of ventilation delivery to an optimum level might improve survival outcomes.

Breaths should be delivered quickly or slowly, however, is an issue that is frequently raised and continues to be debated. The relationship among breath duration, mouth pressure needed to overcome compliance, and mechanics of gastric inflation is complex. Optimal inflation time has not been clearly established. The ideal technique to minimize gastric inflation must depend on victim variables of lung compliance, airway resistance, and lower esophageal sphincter pressure as well as rescuer variables of the mouth pressure profile and the time over which mouth pressure exceeds lower esophageal sphincter pressure during the inflation cycle. Lower esophageal sphincter pressure is typically ~20 cm H$_2$O or greater, and drops to 5 cm H$_2$O during progression to severe hypoxia. Parameters that define the ventilation pressure profile of each breath are maximum $V_T$, which determines necessary peak airway pressure, and inflation time.28,29 Studies that compared ventilation strategies on manikins and subjects who were anesthetized contributed to resuscitation guidelines, but there is no encompassing quantitative theory that defines optimal ventilation in terms of these parameters.

This study used a physiological model to investigate how gastric inflation varies with breath duration over the range of 0.5–2.5 s and declines in lower esophageal sphincter pressure. Airway and esophageal flows ($Q_{aw}$) were calculated for typical inflation profiles, $V_T$, and lower esophageal sphincter pressures. The mouth pressure profiles chosen for investigation were those that approximated mouth-to-mouth and bag-valve-mask ventilation. Although experimental studies in the literature provide a sampling of gastric inflation responses under various conditions, this model provides a more general and comprehensive view of this relationship, which allows clarification of the effects of fast and slow breaths.

**Methods**

Shown in Figure 1, the model configuration is composed of mouth, airway, esophagus, and lungs. Alveolar pressure ($P_A$) equals lung volume ($V_L$) divided by respiratory system compliance ($C_{RS}$), which includes the chest wall and lungs acting as a coupled unit. $V_L$ change is the integral of airway flow ($Q_{aw}$) over time calculated by adding volume increments over small time steps. Airway resistance ($R_{aw}$) equals the pressure difference between mouth ($P_M$) and $P_A$ divided by $Q_{aw}$. $R_{aw}$ is assumed to be constant.

Gas flows into the stomach when airway pressure rises above lower esophageal sphincter pressure, which was set be 20, 15, 10, or 5 cm H$_2$O to account for the decline...
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Fig. 1. The model configuration is based on the applied mouth pressure profile, \( P_m(t) \), of duration \( T_i \). Flow drives lung volume (\( V_L \)), which expands according to compliance of the respiratory system, \( C_{RS} \). Alveolar pressure, \( P_A \), lags \( P_m \) according to airway resistance, \( R_{aw} \). Gas flows into the esophagus if \( P_m \) exceeds sphincter pressure threshold lower esophageal sphincter pressure (LESP). Gastric volume, \( V_G \), is proportional to the area under \( P_m(t) \) that lies above LESP. \( P_m \) = mouth pressure; \( T_i \) = inflation times; \( P_A \) = alveolar pressure; LESP = lower esophageal sphincter pressure; \( V_L \) = air flow into lungs; \( R_{aw} \) = airway resistance; \( Q_{ES} \) = esophageal flow; \( R_{ES} \) = esophageal resistance; \( PIP \) = peak inspiratory pressure; \( V_l \) = lung volume; \( V_T \) = tidal volume; \( C_{RS} \) = compliance of the respiratory system; \( V_G \) = volume of gas entering the stomach.

measured during human hypoxic arrest.\(^8\) Esophageal resistance, \( R_{ES} \), therefore, is considered infinite until \( P_m(t) \) exceeds lower esophageal sphincter pressure, above which resistance is a fixed constant. Gastric volume (\( V_G \)) is the integral of \( Q_{ES} \) over time and increases whenever \( P_m \) exceeds lower esophageal sphincter pressure. The focus is on \( V_G \) per breath, rather than a regurgitation threshold, so the gastric pressure was set to zero, with no back pressure. \( Q_{ES} \) is proportional to \( P_m \) minus lower esophageal sphincter pressure only when \( P_m \) is higher than the lower esophageal sphincter pressure. Ventilation must achieve a specified target \( V_L \), \( V_T \), and, hence, \( P_A \) must reach a corresponding peak pressure of at least \( P_T = V_T/C_{RS} \). Model input was the \( P_m(t) \) specified by its shape, peak inspiratory pressure, PIP, and durations of inspiration, \( T_i \).

Two mouth pressure profiles were investigated. Mouth-to-mouth ventilation was approximated by an exponential rise toward plateau pressure, which thus represented diminishing additional inflation effort by the rescuer as PIP is reached, followed by an abrupt drop when the mouth is removed. Bag-valve-mask inflation was approximated by the positive half of a sine wave with \( T_i \) reaching a PIP. This shape conforms to the smoother cyclical effort with a slower deflation phase seen in an experimental study.\(^{31}\) The present model does not calculate oxygen or carbon dioxide exchange, so it does not derive the ideal \( V_T \) from metabolic gas calculations. \( V_T \), therefore, was assigned to be 0.6 or 1.0 L for each breath because these volumes span a clinically practical range, and lower volumes are inadequate for oxygenation.\(^{17,21}\) Gastric inflation may be expressed as a functional relationship \( V_G = f(V_T, T_i, \text{lower esophageal sphincter pressure, } C_{RS}, R_{aw}) \). The effect of breath duration \( T_i \) on gastric inflation and, therefore, how quickly each breath should be given was investigated.

Equations were evaluated in a spreadsheet. Constants that represented compliance, resistance, inflation duration, and pressure waveform were set for each run. \( V_L \) and \( V_G \) changes were integrated by multiplying respective flows by time step \( \Delta t = 0.01 \) s and by adding this to the previous volume. Respiratory parameters were chosen to represent an average supine adult victim with \( C_{RS} = 0.08 \) L per cm H\(_2\)O and \( R_{aw} = 4 \) L/s per cm H\(_2\)O. \( R_{ES} \) is not known, so it was arbitrarily set to 10 L/s per cm H\(_2\)O. \( V_G \), therefore, must be interpreted on a comparative, rather than absolute, volume scale.

Results

Shown in Figure 2 is the simulation of mouth-to-mouth ventilation to a \( V_T \) of 0.6 L over 1 s. The PIP to reach this volume is 11.0 cm H\(_2\)O. \( P_m \) drops to zero when the rescuer’s mouth is removed after ventilation. \( P_A \) lags behind \( P_m \) according to the system time constant, which is the product of compliance \( C_{RS} \) and airway resistance \( R_{aw} \). \( Q_{ES} \) occurs whenever \( P_m \) exceeds lower esophageal sphincter pressure, which depends on the shape of the pressure profile. \( V_L \) and \( V_G \) are shown at the bottom. The total volume delivered in a breath to lungs and stomach is \( V_T + V_G \).

The sinusoidal profile approximates bag-valve-mask ventilation. The \( Q_{aw} \) rate and \( V_L \) responses are also close to being sinusoidal. Gastric inflation for the bag-valve-mask profile is slightly higher than that of the mouth-to-mouth profile (0.21 vs 0.19 L per breath) because the peak mouth pressure is higher (11.0 cm H\(_2\)O vs 8.7 cm H\(_2\)O). Although the bag-valve-mask pressure profile shape spends slightly less time higher than the lower esophageal sphincter pressure, the mean pressure gradient from mouth to stomach is higher. \( V_G \) is again a function of the shape of the mouth pressure profile and the time over which \( P_m \) exceeds the lower esophageal sphincter pressure.

The left panels in Figure 3 show the peak mouth pressures required to reach target \( V_T \) of 0.6 and 1.0 L for each of the profiles. The shortest inflations required the highest mouth pressures. Inflation times of \(< 0.5 \) s would be undesirable because higher pressures would be necessary to reach the target. Higher pressures may exceed lower esophageal sphincter pressure and also tend to cause air leakage at the mouth because a perfect seal is hard to maintain with rescuer lips or a face mask.

The other 4 panels in Figure 3 compare gastric inflation volumes as functions of breath duration \( T_i \) for each profile.
A lower esophageal sphincter pressure of 15 cm H2O results in minimal gastric inflation seen at short TI and larger VT. This is a consequence of the higher mouth pressures necessary at short inflation times. The VG response to the lower esophageal sphincter pressure of 10 cm H2O is relatively flat over the entire range of TI, seen most clearly at the higher VT. This is due to the high mouth “pressure effect” at short TI transitioning into a “duration effect” at long TI in which there is more time over which PM exceeds the lower esophageal sphincter pressure. Ideal inflation time lies within a zone between these extremes, as illustrated in Figure 4. At the lowest esophageal sphincter pressure, of 5 cm H2O, longer inflation times result in more gastric inflation because the time integral of PM minus the lower esophageal sphincter pressure becomes greater as TI increases.

The response of VG increased linearly with the VT above a threshold VT/L and was greater at a lower esophageal sphincter pressure, as shown in Figure 5. Respiratory system compliance decreases with lung injury, so inflation pressures would have to increase. Because peak alveolar pressure, PA, equals VT/CRS, gastric inflation would also increase. Responses to fast inflations of 0.5 s were truncated above VT/1.2 L due to the high mouth pressure needed to drive this volume over a short time, reaching the imposed cutoff of 25 cm H2O.

Shown in Figure 6 are the effects on VG of decreasing compliance to 0.06 L/cm H2O and of increasing airway resistance to 10 L/s per cm H2O. Each alteration in -creased gastric inflation due to higher airway pressures needed to achieve VT. Breath duration that produces minimum VG depends on lower esophageal sphincter pressure. Inflations of 1.0 s seem reasonable for higher resistance, whereas lower compliance favors a shift toward 0.5 s.

Fig. 2. Breaths are shown for mouth pressures, PM, alveolar pressure, PA, and lower esophageal sphincter pressure, LESP (A and B). Airway flow into lungs is V and esophageal flow is QES (C and D). Lung volume (VL) reaches the target tidal volume (VT) of 0.60 L (E and F). The volume of gas (VG) entering the stomach in liters is labeled for each breath. Two consecutive breaths are shown for each profile to illustrate the cumulative increase in gastric volume. A, C, and E show mouth-to-mouth ventilation, while B, D, and F show bag-valve-mask ventilation. PM = mouth pressure; PA = alveolar pressure; LESP = lower esophageal sphincter pressure; V = air flow into lungs; QES = esophageal flow; VL = lung volume; VT = tidal volume; VG = volume of gas entering the stomach.

and VT of 0.6 and 1.0 L. A lower esophageal sphincter pressure of 15 cm H2O results in minimal gastric inflation seen at short T1 and larger VT. This is a consequence of the higher mouth pressures necessary at short inflation times. The VG response to the lower esophageal sphincter pressure of 10 cm H2O is relatively flat over the entire range of T1, seen most clearly at the higher VT. This is due to the high mouth “pressure effect” at short T1 transitioning into a “duration effect” at long T1 in which there is more time over which PM exceeds the lower esophageal sphincter pressure. Ideal inflation time lies within a zone between these extremes, as illustrated in Figure 4. At the lowest esophageal sphincter pressure, of 5 cm H2O, longer inflation times result in more gastric inflation because the time integral of PM minus the lower esophageal sphincter pressure becomes greater as T1 increases.

The response of VG increased linearly with the VT above a threshold VT = lower esophageal sphincter pressure × C_{RS} and was greater at a lower esophageal sphincter pressure, as shown in Figure 5. Respiratory system compliance decreases with lung injury, so inflation pressures would have to increase. Because peak alveolar pressure, PA, equals VT/CRS, gastric inflation would also increase. Responses to fast inflations of 0.5 s were truncated above VT = 1.2 L due to the high mouth pressure needed to drive this volume over a short time, reaching the imposed cutoff of 25 cm H2O.

Shown in Figure 6 are the effects on VG of decreasing compliance to 0.06 L/cm H2O and of increasing airway resistance to 10 L/s per cm H2O. Each alteration in -creased gastric inflation due to higher airway pressures needed to achieve VT. Breath duration that produces minimum VG depends on lower esophageal sphincter pressure. Inflations of 1.0 s seem reasonable for higher resistance, whereas lower compliance favors a shift toward 0.5 s.
**Rescue Ventilations and Gastric Inflation**

**Discussion**

Our main finding was that the ideal $T_I$ is ~1.0 s; this was consistent for both inflation waveforms. Very low esophageal sphincter pressure and low compliance favored a shift toward 0.5 s. Gastric inflation increased with $V_T$ and $T_I$ whenever $P_M$ exceeded lower esophageal sphincter pressure. Very short inflations seem undesirable because PIPs are needed to drive Q$_{es}$ to $V_T$, although short durations resulted in only modest gastric inflation. Long inflation times would be especially undesirable if $P_M$ exceeds lower esophageal sphincter pressure because more time is available at higher pressure to push larger volumes of gas into the stomach.

If mouth PIP is lower than lower esophageal sphincter pressure, then the inflation profile does not matter because there is no esophageal gas flow. If PIP exceeds lower esophageal sphincter pressure, then gastric inflation can occur, and one should try to minimize the time that PIP is above the lower esophageal sphincter pressure by avoiding long slow breaths. Lower lung compliance may be encountered after lung injury or prolonged resuscitation, which necessitates higher mouth pressure. Lower esophageal sphincter pressure is not generally known during resuscitation and likely depends on the degree of hypoxia, $V_{L1}$, diaphragm mechanics, abdominal pressure, and gastric distention. It is typically ~20 cm H$_2$O or greater under normal conditions, and was seen to drop during prolonged apnea, to ~5 cm H$_2$O in animal and human studies.

Without rescuer knowledge of lower esophageal sphincter pressure, the best strategy would simply be to keep both $P_M$ and $T_I$ as low as possible; however, if reaching a target $V_T$ is paramount for oxygenation, then these are conflicting objectives, and some gastric inflation may be unavoidable. In theory, gastric inflation can occur only if $V_T$ is > the lower esophageal sphincter pressure $\times$ $C_{RS}$.

The study by von Goedecke et al used a bench mannikin to investigate the role of inspiratory time on gastric inflation. The researchers observed Q$_{es}$ during 1-s inflations at a lower esophageal sphincter pressure of 15 cm H$_2$O but none with 2-s inflations, which thus exhibited an inverse relationship between gastric inflation and breath duration at relatively high esophageal sphincter pressure values close to PIP. However, at a much lower esophageal sphincter pressure, of 5 cm H$_2$O, they found the opposite trend, with higher gastric inflation volumes due to longer breaths, which were even higher during 2-s breaths. In other words, there was a reversal in scope of the $V_G$ ($T_I$) function from negative to positive as the lower esophageal sphincter pressure was decreased.

This seemingly paradoxical finding of a reversal in slope is explained by the model. There is a small “pressure-driven” effect on $V_G$ at shorter $T_I$ and a larger “duration-driven” effect on $V_G$ at longer $T_I$.

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**Fig. 3.** Mouth peak inspiratory pressure, PIP, to reach tidal volumes of 0.6 and 1.0 L must increase for short inflation times $T_I$ (A and D). Gastric volume, $V_G$, increases or decreases with, $T_I$, in seconds, depending on lower esophageal sphincter pressure, LESP, in cm H$_2$O. Low LESP results in greater stomach inflation. The 2 arrows (C) indicate the minimum gastric inflation for each LESP. A, B, and C show mouth-to-mouth-ventilation, volume, $V_G$, increases or decreases with, $T_I$, in seconds, depending on lower esophageal sphincter pressure, LESP, in cm H$_2$O. Low LESP results in greater stomach inflation. The 2 arrows (C) indicate the minimum gastric inflation for each LESP. A, B, and C show mouth-to-mouth-ventilation, volume, $V_G$, increases or decreases with, $T_I$, in seconds, depending on lower esophageal sphincter pressure, LESP, in cm H$_2$O. Low LESP results in greater stomach inflation. The 2 arrows (C) indicate the minimum gastric inflation for each LESP.
Rescue Ventilations and Gastric Inflation

Driven increase in \( V_G \) at a longer \( T_I \) and low esophageal sphincter pressure. This is shown in Figure 3 and is simplified in Figure 4. The study by von Goedecke et al.\(^{30}\) had higher peak pressures during the 1-s inflations, so it is not an exact comparison between their results and our model. They had decreased \( V_T \) at a low esophageal sphincter pressure, of 5 cm H\(_2\)O, due to delivering lower peak pressures. This could have been due to larger gastric inflation, denying some air to the lungs, given that the sum of \( V_T \) and \( V_G \) in their study is consistently \( \sim 800 \) mL.

Melker and Banner\(^\text{37}\) investigated breath duration by using a test lung in 1985. Standards at that time recommended mouth-to-mouth inflations of 0.5 s. They found that gastric inflation decreased as breath duration increased from 0.5 to 1.5 s. These results are consistent with the negative slope of \( V_G \) as a function of \( T_I \) within the pressure-driven regimen of the model. Lower esophageal sphincter pressures in that study were relatively high, at 15 and 20 cm H\(_2\)O. We surmised that, if lower levels of lower esophageal sphincter pressure of 5–10 cm H\(_2\)O had been investigated, then gastric inflation would have increased with \( T_I \) due to the duration-driven effect.

The pressure-driven effect seen at short \( T_I \) produced less gastric inflation than the duration-driven effect seen at a longer \( T_I \). The former occurred at intermediate lower esophageal sphincter pressure of \( \sim 10–15 \) cm H\(_2\)O, whereas the latter effect occurred at the lowest esophageal sphincter pressure, of 5 cm H\(_2\)O. Because lower esophageal sphincter pressure is not known during a resuscitation, this creates a dilemma in choosing the optimal \( T_I \) because minimum \( V_G \) occurs at approximately \( T_I = 1.0 \) s for lower esophageal sphincter pressure = 10 cm H\(_2\)O, yet it drops to \( T_I = 0.5 \) s for lower esophageal sphincter pressure = 5 cm H\(_2\)O. These 2 points are marked with arrows in the top right panel of Figure 3. It probably does little harm to lower \( T_I \) to 0.5 s in this case because the \( V_G \) response is fairly flat between 0.5 and 1.0 s, for which pressure and duration effects are comparable.

In terms of gas exchange, Wenzel et al.\(^{18}\) found oxygenation to be inadequate if \( T_I \) was < 0.4 s, which defines a lower bound on practical breath duration. Therefore, relatively high mouth pressures must be applied if attempting to reach target \( V_T \) by using very short inflations, but high pressures promote air leakage of the mouth seal, and \( V_T \) might still be inadequate.

Passive expiration between breaths requires \( \sim 1 \) s for normal lungs and would not affect these results unless the expiratory time is very short relative to the inspiratory time constant, which is the product of \( R_{aw} \) and \( C_{RS} \), and is < 0.6 s in most healthy people.\(^{38}\) Lung deflation is usually completed in < 2.0 s; therefore, unless the lungs are severely injured, there should be adequate time for full expiration between breaths. The stacking effect of an initial rapid breath sequence increases \( V_T \) in a stepwise manner and would increase the mean airway pressure. More importantly, breath-stacking would increase the proportion of time that mouth pressure exceeds lower esophageal sphincter pressure and promotes gastric inflation. Guidelines based on 12 breaths/min for sustained ventilation should allow ample time for full expiration.

The model is based on standard physiology with some simplifications. It disregards atelectasis, alveolar recruitment, and nonlinear airway resistance. These effects are likely minimal at low flows and \( V_T \) typical of rescue ventilations. Fixed airway resistance cannot account for complexities of upper-airway anatomy or changes in neck position. The esophagus was assumed to behave as a rigid conduit with a pressure-sensitive threshold sphincter, which ignores elastic wall properties.\(^{39}\) Data on the rise of gastric pressure during resuscitation are unavailable, so the gastric pressure was assumed to be zero to represent the worst-case scenario of maximum \( Q_{ES} \) in the absence of back
pressure. The model essentially mimics responses of a bench-top mannikin constructed with a test lung and esophageal threshold valve. A mathematical model cannot entirely replace mannikin or human studies because the shape of mouth pressure profiles depends on rescuer techniques and the mechanics of bag-valve-mask devices, which cannot be predicted from theory alone. Mouth pressure data must be acquired experimentally, but it can be incorporated into quantitative models to obtain a more complete functional relationship between variables.

Conclusions

This model predicts gastric inflation during positive-pressure ventilation with an unprotected airway and illustrates the general dependence on VT and breath duration. It brings seemingly conflicting results from bench studies in the literature into a coherent picture. Breaths of < 1 s require increased mouth pressures to reach the target volume. Breaths longer than 1 s spend more time above the lower esophageal sphincter pressure, which thus increases gastric inflation. Ventilation time of 1 s meets the target VT and minimizes gastric inflation under most assumed conditions. Short pressure–driven and long duration–driven gastric inflation regimens provide a unifying explanation of results in past mannikin studies.

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RESCUE VENTILATIONS AND GASTRIC INFLATION


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