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 H_2O 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.* [Respir Care 2018;63(5):502–509. © 2018 Daedalus Enterprises]

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,

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adequacy of passively induced tidal volumes (V_T) is not assured with this technique,[10] and victims of primary respiratory arrest may be severely hypoxic and need active ventilation.[2,11]

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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,[5] 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[21] and arrest victims who received cardiopulmonary resuscitation,[22] 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 mannikin often delivered V_T of $<$ 0.4 L,[23] 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.

Whether 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_2O or greater[4,25,26] and drops to 5 cm H_2O during progression to severe hypoxia.[8,27] Parameters that define the ventilation pressure profile of each breath are maximum V_T, which determines necessary peak airway pressure,[28] and inflation time.[29,30] Studies that compared ventilation strategies on mannikins and subjects who were anesthetized contributed to resuscitation guidelines,[15,18] but there is no encompassing quantitative theory that defines optimal ventilation in terms of these parameters.

QUICK LOOK

Current knowledge

Rescue ventilations are commonly delivered to victims with an unprotected airway during respiratory and cardiac arrest. Mouth pressure that exceeds lower esophageal sphincter pressure can cause harmful gastric inflation. Breaths delivered quickly or slowly have been shown to increase gastric inflation in various studies. There has been no attempt to explain this dichotomy or to develop a unified theory that establishes optimal breath duration.

What this paper contributes to our knowledge

Gastric inflation was estimated by using equations of pulmonary physiology based on the area under the mouth pressure curve that lies above the esophageal sphincter pressure. Faster breaths require high mouth pressures to reach target V_T, and slower breaths increase the time available for gastric inflation. These results can be explained by pressure-driven and duration-driven mechanisms. The ideal breath duration for minimum gastric inflation was found to be ~1 s for the mouth pressure profiles investigated.

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_{ES}) 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_2O to account for the decline

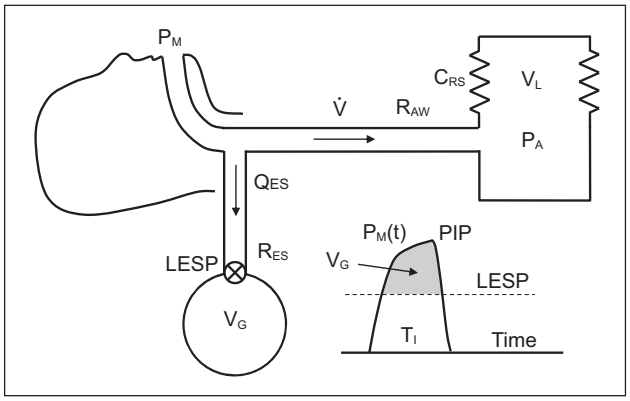

Fig. 1. The model configuration is based on the applied mouth pressure profile, $P_M(t)$, of duration T_I. Flow drives lung volume (V_L), which expands according to compliance of the respiratory system, C_{RS}. Alveolar pressure, P_A, lags P_M according to airway resistance, R_{aw}. Gas flows into the 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 that lies above LESP. P_M = mouth pressure; T_I = inflation times; P_A = alveolar pressure; LESP = lower esophageal sphincter pressure; $\dot{V}$ = 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 $V_G = f(V_T, T_I$, 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_2O and $R_{aw} = 4$ L/s per cm H_2O.[32-34] R_{ES} is not known, so it was arbitrarily set to 10 L/s per cm H_2O. 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_2O. 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_{aw} is proportional to P_M minus P_A. 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_2O vs 8.7 cm H_2O). 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

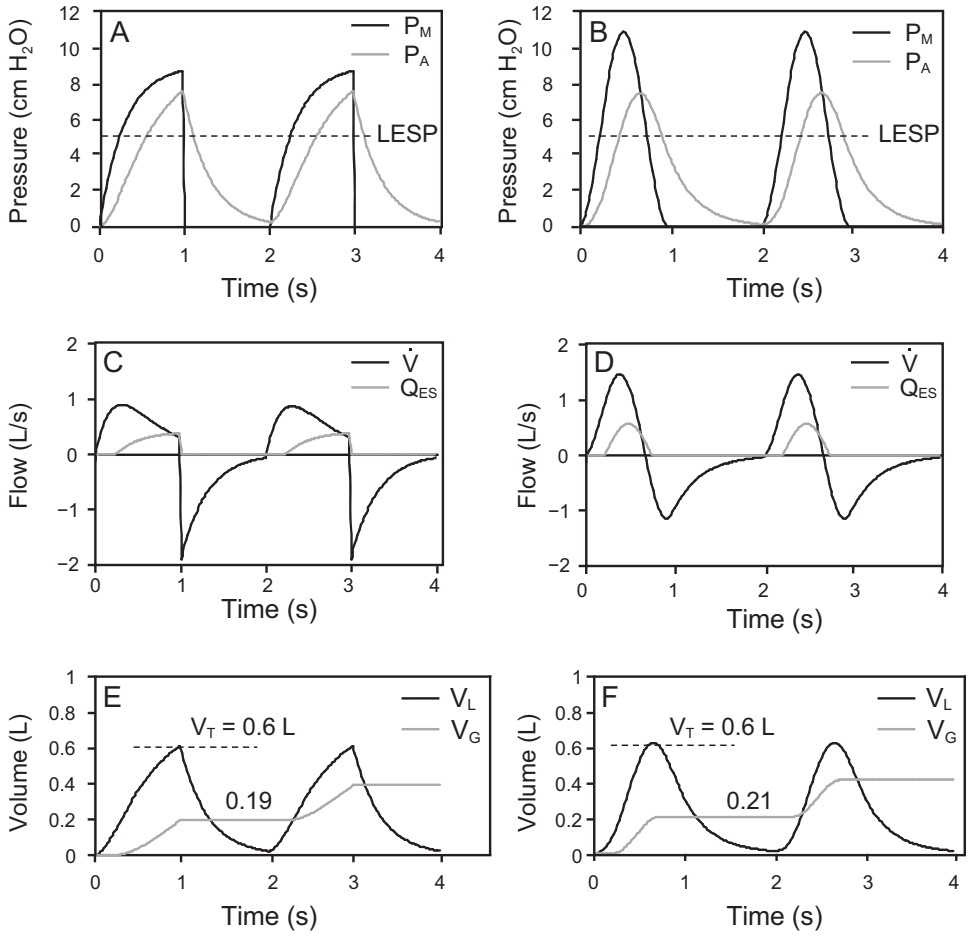

Fig. 2. Breaths are shown for mouth pressures, P_M, alveolar pressure, P_A, and lower esophageal sphincter pressure, LESP (A and B). Airway flow into lungs is $\dot{V}$ and esophageal flow is Q_{ES} (C and D). Lung volume (V_L) reaches the target tidal volume (V_T) of 0.60 L (E and F). The volume of gas (V_G) 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. P_M = mouth pressure; P_A = alveolar pressure; LESP = lower esophageal sphincter pressure; $\dot{V}$ = air flow into lungs; Q_{ES} = esophageal flow; V_L = lung volume; V_T = tidal volume; V_G = volume of gas entering the stomach.

and V_T of 0.6 and 1.0 L. A lower esophageal sphincter pressure of 15 cm H_2O results in minimal gastric inflation seen at short T_I and larger V_T. This is a consequence of the higher mouth pressures necessary at short inflation times. The V_G response to the lower esophageal sphincter pressure of 10 cm H_2O is relatively flat over the entire range of T_I, seen most clearly at the higher V_T. This is due to the high mouth "pressure effect" at short T_I transitioning into a "duration effect" at long T_I in which there is more time over which P_M 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 H_2O, longer inflation times result in more gastric inflation because the time integral of P_M minus the lower esophageal sphincter pressure becomes greater as T_I increases.

The response of V_G increased linearly with the V_T above a threshold V_T = 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,[35] so inflation pressures would have to increase. Because peak alveolar pressure, P_T, equals V_T/C_{RS}, gastric inflation would also increase. Responses to fast inflations of 0.5 s were truncated above V_T = 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 H_2O.

Shown in Figure 6 are the effects on V_G of decreasing compliance to 0.06 L/cm H_2O and of increasing airway resistance to 10 L/s per cm H_2O. Each alteration increased gastric inflation due to higher airway pressures needed to achieve V_T. Breath duration that produces minimum V_G 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.

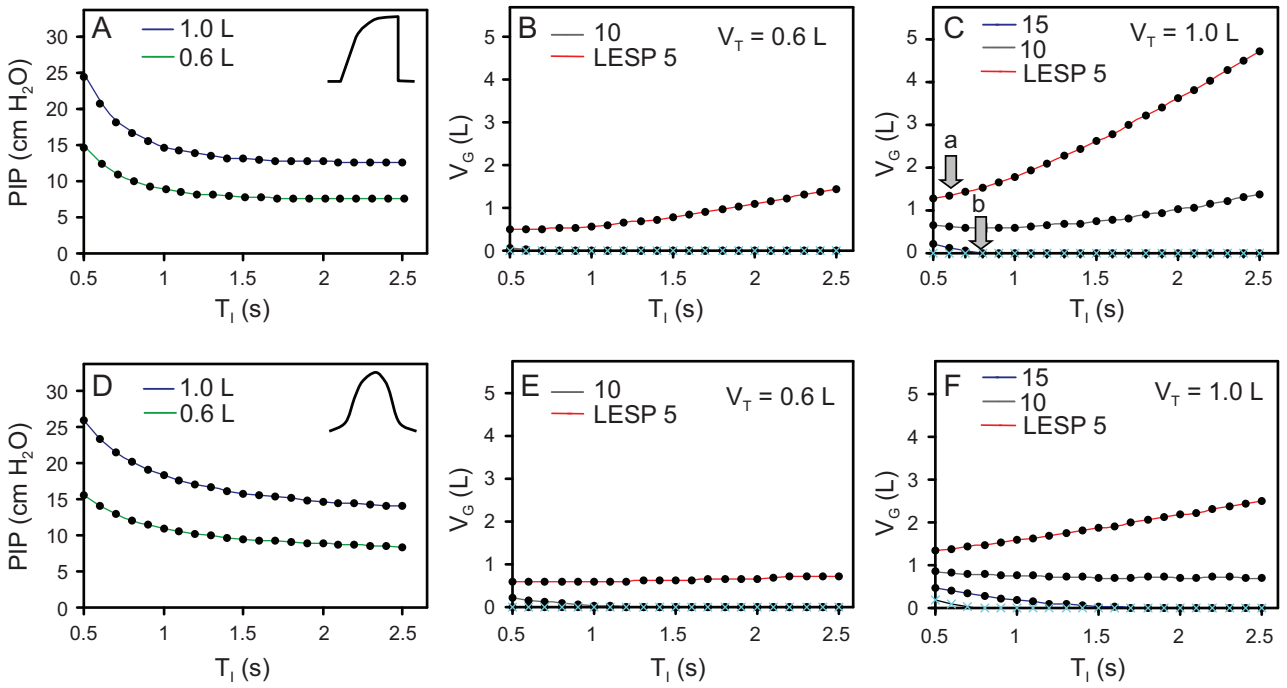

Fig. 3. Mouth peak inspiratory pressure, PIP, to reach tidal volumes of 0.6 and 1.0 L must increase for short inflation times T_I (A and D). Gastric volume, V_G, increases or decreases with, T_I, in seconds, depending on lower esophageal sphincter pressure, LESP, in cm H_2O. Low LESP results in greater stomach inflation. The 2 arrows (C) indicate the minimum gastric inflation for each LESP. A, B, and C show mouth-to-mouth-ventilation, while D, E, and F show bag-valve-mask ventilation. MTM = mouth mouth ventilation; V_T = tidal volume; V_G = volume of gas entering the stomach; LESP = lower esophageal sphincter pressure; PIP = peak inspiratory pressure.

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_{aw} 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[35,36] Lower esophageal sphincter pressure is not generally known during resuscitation and likely depends on the degree of hypoxia, V_L, diaphragm mechanics, abdominal pressure, and gastric distention. It is typically ~20 cm H_2O or greater under normal conditions,[4,25,26] and was seen to drop during prolonged apnea, to ~5 cm H_2O in animal and human studies.[8,27] 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.[6] In theory, gastric inflation can occur only if V_T is > the lower esophageal sphincter pressure × C_{RS}.

The study by von Goedecke et al[30] 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_2O 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_2O, they found the opposite trend, with higher gastric inflation volumes due to longer breaths, which were even higher during 2-s breaths.[30] In other words, there was a reversal in slope 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-

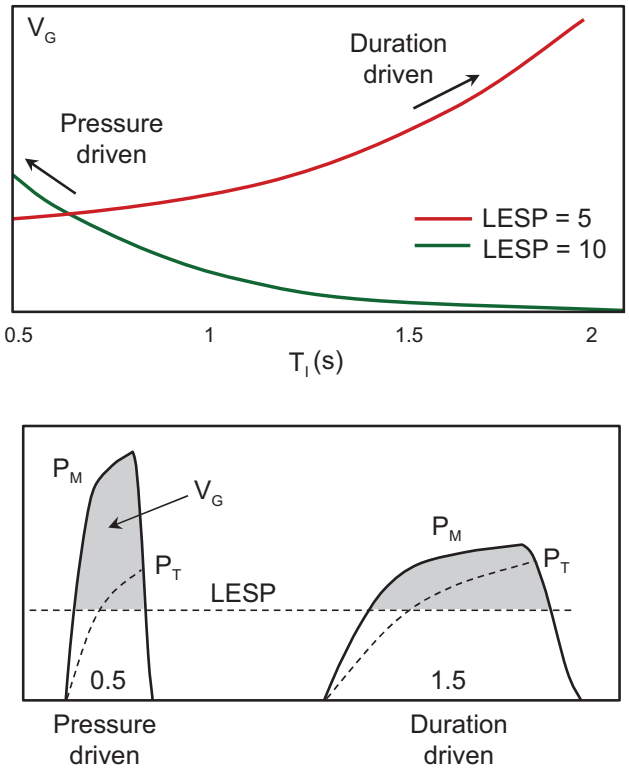

Fig. 4. Simplified diagram, illustrating the 2 effects that drive gastric inflation; each effect depends on inflation time (T_I) and lower esophageal sphincter pressure (LESP). Short inflations require high pressure, which causes the pressure-driven effect at moderate LESP. Long inflations increase the volume of gas (V_G) by a duration-driven effect when the LESP is very low. The vertical axis of the graph is for illustrative purposes and is not to scale. Gastric inflation volume is proportional to the shaded areas above the LESP in the examples shown. P_M = mouth pressure; T_I = inflation times; LESP = lower esophageal sphincter pressure; V_G = volume of gas entering the stomach.

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_2O, 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 ~800 mL.

Melker and Banner[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_2O. We surmised that, if lower levels of lower esophageal sphincter pressure of 5–10 cm H_2O 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 ~10–15 cm H_2O, whereas the latter effect occurred at the lowest esophageal sphincter pressure, of 5 cm H_2O. 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_2O, yet it drops to T_I = 0.5 s for lower esophageal sphincter pressure = 5 cm H_2O. 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 ~1 s for normal lungs and would not affect these results unless the expiratory time is very short relative to the expiratory 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_L 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

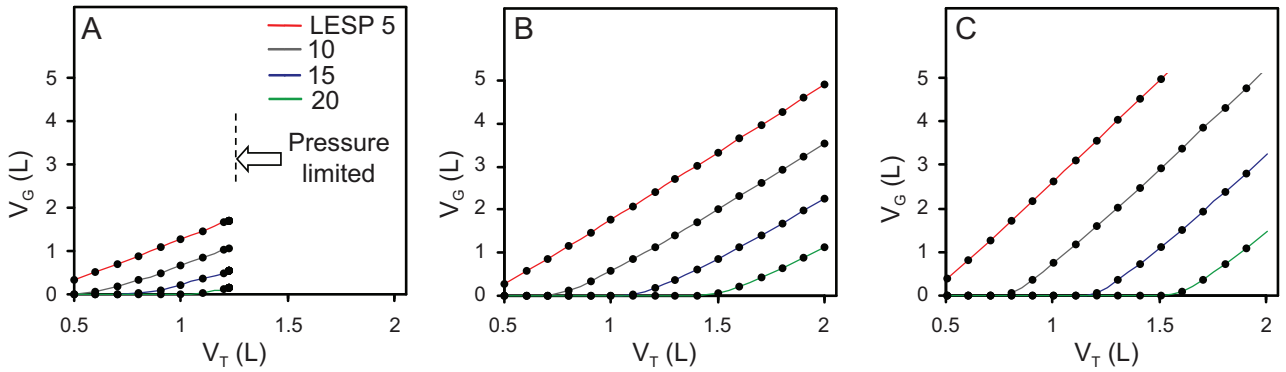

Fig. 5. Gastric volume, V_G, increases with tidal volume, V_T, and breath duration, T_I. The relation is approximately linear with V_T once mouth pressure exceeds lower esophageal sphincter pressure, LESP. Short inflations of T_I = 0.5 s (A) have a cutoff imposed due to mouth pressure that reaches 25 cm H_2O, which risks mouth seal leakage and exceeds normal LESP. TI = 1 s (B) and 1.5 s (C). TI = inflation time; LESP = lower esophageal sphincter pressure; V_G = volume of gas entering the stomach; V_T = tidal volume.

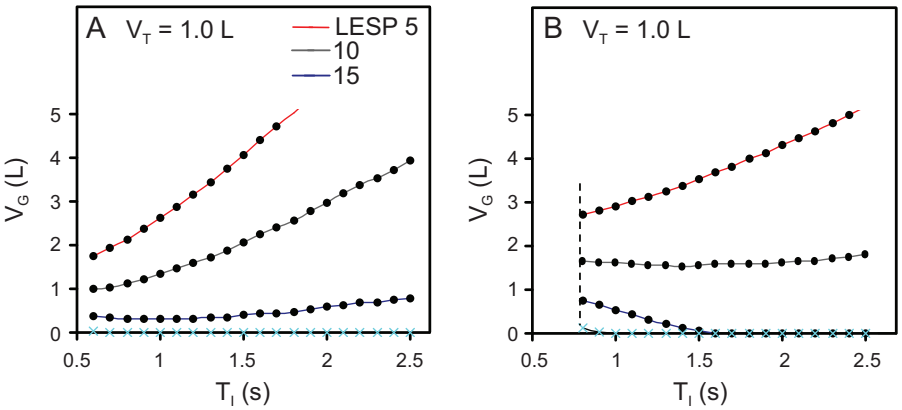

Fig. 6. Decreasing compliance to 0.06 L per cm H_2O (A) and increasing airway resistance to 10 L/s per cm H_2O (B) each increase gastric inflation volume, V_G, relative to baseline values. Tidal volume is 1.0 liters for MTM profile. Ideal TI is shorter at very low LESP. MTM = mouth to mouth ventilation; V_T = tidal volume; V_G = volume of gas entering the stomach; LESP = lower esophageal sphincter pressure; T_I = inflation times; C_{RS} = compliance of the respiratory system.

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 V_T 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 V_T 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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