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ABSTRACT

This study was undertaken to determine the effect of interposed abdominal compressions (IAC) during cardiopulmonary resuscitation (CPR) on gastric insufflation when the airway is not secured with an endotracheal tube. A canine model was used in which a common ventilation pressure was applied to separate cuffed esophageal and tracheal tubes. Gas entering the stomach was collected by a pre-placed gastrostomy tube leading to a bell spirometer. Gas entering the lungs was measured with a Wright Respirometer® in series with the endotracheal tube. During standard CPR, measurable gastric gas volume was collected in 28 of 30 trials (mean 215 ± 93 ml/ventilation). During IAC-CPR, in which abdominal pressure was maintained during ventilation after every 5th chest compression, measurable gastric gas was collected in 15 of 30 trials (mean 40 ± 11 ml/ventilation). Interposed abdominal compressions as an adjunct to standard CPR may not only be of hemodynamic benefit, but may also reduce the incidence of gastric insufflation and attendant complications.

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The addition of interposed abdominal compressions (IAC) to otherwise standard cardiopulmonary resuscitation (CPR) improves perfusion pressures, artificial cardiac output, and cerebral blood flow in animal models of cardiac arrest and resuscitation.[1-4] Early clinical studies of IAC-CPR in humans show improved arterial blood pressure during IAC-CPR and suggest improved cardiac resuscitation rates for some classes of patients.[5,6] The method appears to be safe. Only one pediatric case has been reported associating IAC-CPR with abdominal visceral injury.[7] However, all studies of IAC-CPR to date have focused on intubated subjects, and there remains the question of the likelihood of gastric insufflation and consequent esophageal regurgitation when IAC-CPR is performed without an endotracheal tube. If regurgitation were to occur much more often with IAC-CPR than with standard CPR, the addition of IAC to basic life support protocols would be much less desirable.

The possibilities are interesting. On the one hand, abdominal compression may increase the chance of regurgitation by directly compressing trapped air and gastric contents. On the other hand, we have proposed that because abdominal pressure is maintained during ventilation, IAC may actually decrease the chance of regurgitation by preventing gastric insufflation during CPR.[1] According to this concept, gastric distension would be prevented by the high intra-abdominal pressure generated as IAC is applied during positive pressure emergency ventilation.

Two clinical studies, in which IAC-CPR was compared with standard CPR with an endotracheal tube in place, have not demonstrated that regurgitation occurs during abdominal counterpulsation.[5,6] Thus, abdominal pressure alone, in the absence of gastric insufflation, does not appear to induce regurgitation. However, if gastric distension occurred in IAC-CPR to the same degree as in the standard CPR, when ventilation is not provided through an endotracheal tube, then abdominal counterpulsation might indeed increase the likelihood of regurgitation.

Accordingly, we developed an animal model in which ventilation pressure was delivered equally to both the upper esophagus at the level of the thoracic inlet and the trachea in such a way that the volumes of air entering the lungs and the stomach could be measured. Using this model, we then conducted the following investigation to determine whether abdominal compression alters the degree of gastric insufflation.

MATERIALS AND METHODS

Animal preparation. Dogs anesthetized with pentobarbitol and weighing 14 to 21 kg (mean 17 kg) served as subjects. Relatively broad-chested dogs with ventral-dorsal chest diameters averaging 19 cm and right-left chest diameters averaging 16 cm were selected for the study. The animals were deprived of food 24 hours prior to the experiment to facilitate gas collection from the stomach through tubes that might become obstructed by particulate matter. Each dog was placed in dorsal recumbency on a V-shaped board with the limbs securely tied to the board to prevent lateral motion of the chest during CPR. Subcutaneous electrodes for recording the electrocardiograph (Lead II) were placed, and the chest was shaved to permit transthoracic defibrillation. A femoral venous catheter was placed for injection of anesthetic as needed. A special jugular venous catheter, including a monopolar stimulating electrode near the tip and a lumen for pressure monitoring, was advanced into the right ventricle. The third catheter was

advanced 5-10 cm into the right brachial artery for pressure monitoring. The catheters employed for arterial and venous pressure monitoring were connected to matched Cobe-Microswitch® transducers.

After placement of vascular catheters, a midline laparotomy was performed, and a 20-French Foley catheter made of soft rubber was placed in the stomach for gas collection. This catheter was led first through a stab wound in the lateral abdominal wall, then through an incision in the antimesenteric border of the duodenum, and finally through the lumen of the pylorus and into the stomach. It was secured in position with loops of umbilical tape tied tightly around the pylorus. The duodenal incision was then closed around the catheter with a pre-placed purse string suture. The balloon of the Foley catheter was filled with water, and the system was tested to ensure that the catheter could not be withdrawn from the stomach and that gastric gas could be properly vented through the catheter. Then the abdominal incision was closed in layers, and the Foley catheter was attached to a fluid trap and, in turn, to a counterweighted recording spirometer for gas collection (Fig. 1).

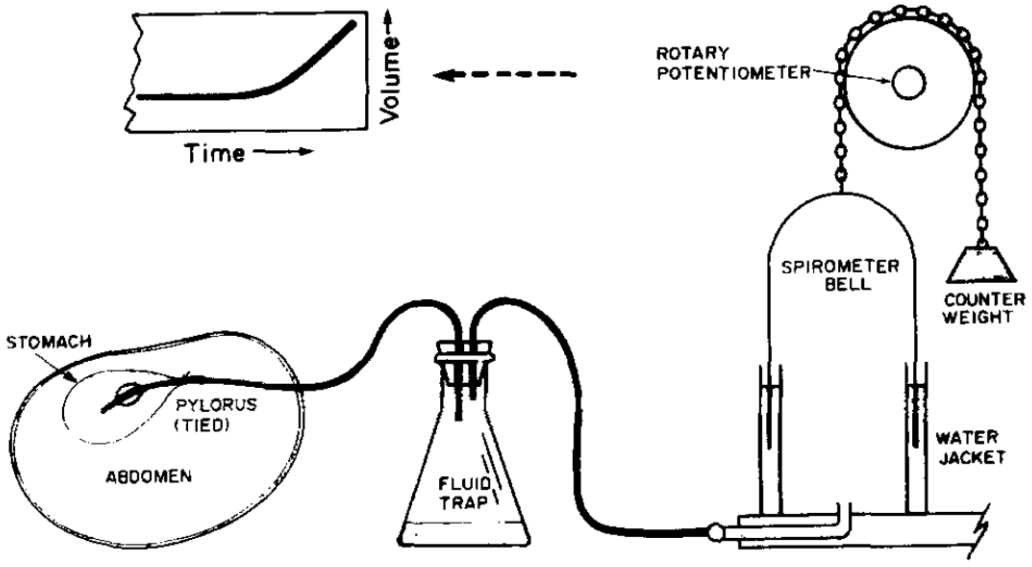

FIGURE 1. Apparatus for collection of stomach gas.

The ECG and pressure data were recorded graphically using a Physiograph® direct-inking recorder (Narco Bio-Systems, Houston, Texas). Pressure channels were calibrated and their linearity was confirmed using a mercury manometer. Pressure transducers were opened to air frequently during the experiment to confirm the zero-pressure reference level.

Unprotected airway model. To simulate mouth-to-mouth resuscitative breathing in humans, in which air is blown into the pharynx and may enter either the esophagus or the trachea, we constructed the apparatus shown in Figure 2. The ventilation hose of the Thumper® mechanical resuscitator (incorporating a constant-pressure ventilator) was connected to a "T" joint leading to two parallel pathways. The first pathway was a standard cuffed endotracheal tube; the second was a specially cuffed 1.25-cm ID polyethylene esophageal tube. The cuff for the esophageal tube was fashioned out of soft latex material from a surgical glove.

Calibration procedures for this unprotected airway model, done before the experiment, included the following. First the connections were checked for leaks by pressurization to 100 mm Hg. Next, the esophageal cannula was completely occluded, and a known tidal volume (1,000 ml) from a gas syringe was passed through the Wright Respirometer®. Then, the pinch clamp was adjusted until half (500 ml) of the tidal volume passed through the endotracheal tube and half of the tidal volume passed through the esophageal tube, with the ends of both tubes open to air. In this way, the model was adjusted so that gas delivery after insertion would depend principally on pulmonary and esophageal resistances.

The cuffed endotracheal tube was placed in the trachea and sealed in the usual way. The pharyngeo-esophageal tube was placed in the pharynx and upper esophagus with its distal end at the level of the thoracic inlet. This positioning of an esophageal tube has been shown by Ruben[2] to have negligible effect on esophageal opening pressure. Its cuff was inflated to a back pressure clearly greater than inspiratory pressure (e.g. 40 mm Hg) before each trial. The Wright Respirometer® (Harris-Lake, Inc., Cleveland, Ohio) in series with the endotracheal tube measured the cumulative tidal volume entering the lungs. This respiratory flowmeter registers cumulative gas flow that passes through it in the forward direction and is insensitive to reverse flow. The calibration of the Wright Respirometer® was checked at the beginning of the experiment by passing known minute volumes through it into the bell spirometer.

Gas entering the stomach was collected in the recording bell spirometer under low pressure. Because the pylorus was tied around the Foley catheter, gas entering the stomach through the esophagus could not pass around the catheter into the duodenum and, therefore, was collected in the spirometer. Changes in the position of the spirometer bell were monitored by means of a rotary potentiometer fixed to the pulley suspending the counterweighed bell (Fig 1). This potentiometer provided an electrical signal linearly related to bell volume, which was displayed on the graphic recorder. In each experiment, this signal was calibrated by introducing known volumes of gas into the bell with a 1000-ml syringe. In this way, gastric insufflation was quantified.

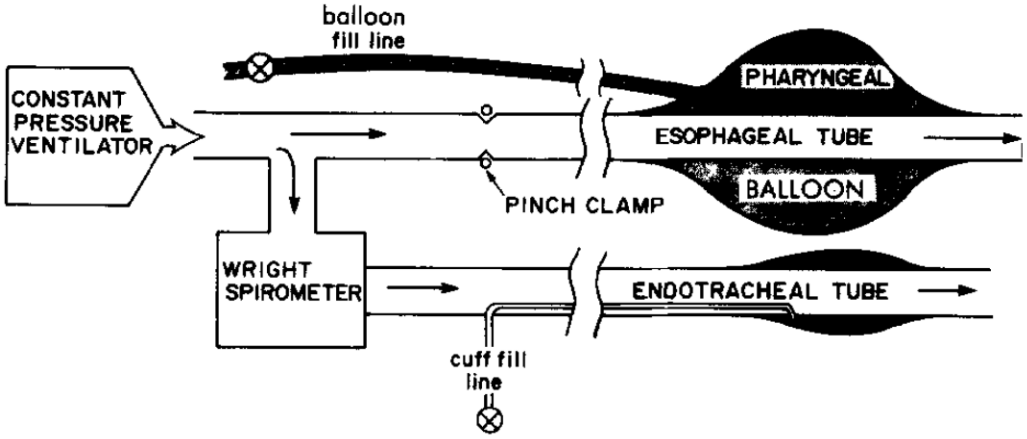

FIGURE 2. Parallel arrangement of cuffed esophageal and endotracheal tubes to create an unprotected airway model.

Abdominal compression technique. The abdomen was compressed with a standard 12-cm-wide blood pressure cuff, folded to rectangular dimensions of 12 x 15 cm, and inflated with of air to a thickness of 3 cm. The bladder of the cuff was attached via the filling hose to an aneroid manometer in order to monitor pressure applied to the abdomen. During the first phase of the experiment, in which the heart beat normally, continuous pressure of 100 mm Hg was applied by manually compressing the folded blood pressure cuff over the mid-abdomen. In the second phase of the experiment, in which standard CPR and IAC-CPR were compared, interposed abdominal compressions were applied by manual compression of the mid-abdomen during the release phase of chest compression to generate pressure pulses of l00-120 mm Hg. The duty cycle of abdominal compression during IAC-CPR was complimentary to that of chest compression (i.e., 50% of the cycle time).

Experimental design. The experiment was divided into two phases. In Phase I the heart was allowed to beat normally. Pulmonary volume and gastric volume were measured during ten positive pressure ventilations with and without continuous manual abdominal compression. The volumes of gas entering the lungs and the stomach, with and without abdominal compression, were compared at inspiratory pressures ranging from 15 to 40 cm H_2O, in steps of 5 cm H_2O. After each trial the abdomen was gently kneaded to express remaining unvented gas from the stomach into the bell spirometer. The volume collected in the spirometer, divided by ten, was the "gastric tidal volume," while the volume registered by the Wright Respirometer®, divided by ten, was the "pulmonary tidal volume."

In Phase II, we studied 12 two-minute periods of electrically induced ventricular fibrillation and CPR with ventilation pressures ranging from 15 to 40 cm H_2O, in increments of 5 cm H_2O. The order in which ventilation pressures were tested was established from a pre-determined random sequence. In each trial, ventricular fibrillation was induced by 60-Hz stimulation of the right

ventricular endocardium via the electrode catheter. After fibrillation was confirmed by the electrocardiographic and the blood pressure tracing, either standard CPR or IAC-CPR was performed according to protocol. Cardiopulmonary resuscitation was begun with the Thumper® and continued until a total of ten positive pressure breaths at the selected ventilation pressure was delivered. This device delivers chest compressions at a rate of 60/minute (0.5-second compression per cycle) with a 1.0-second pause after every fifth chest compression. During this pause, positive pressure ventilation is delivered. The force of chest compression was 50 lbs. For trials of IAC-CPR, the abdomen was manually compressed whenever chest compression was not being applied, including, in particular, the pauses for ventilation.

Ventilation was discontinued after ten breaths, the ventricles were defibrillated with a shock of 50-100 Joules, and the abdomen was kneaded to express residual gas. Volumes of gas entering the lungs (measured with the Wright Respirometer®) and gas recovered from the stomach (measured with the bell spirometer) were recorded, and ventilation was resumed though the tracheal tube only. Following a three minute recovery period, the fibrillation-CPR sequence was repeated using the alternate form of CPR at the same ventilation pressure. In half of the animals, standard CPR was tested first, while in the remaining animals, IAC-CPR was tested first at each level of inspiratory pressure. In this way, we compared gas volumes entering the lungs and stomach during trials of both standard CPR and IAC-CPR at ventilation pressures of 15, 20, 25, 30, 35, and 40 cm H_2O.

Post-resuscitation protocol. The animal was put to death by ventricular fibrillation without resuscitative measures, and a postmortem examination was performed. Special attention was given to identification of possible trauma to the abdominal viscera as a result of IAC-CPR.

Data analysis. At each ventilation pressure, pulmonary and gastric gas volumes with and without abdominal pressure were computed for the population of five dogs. Paired Student's t-tests were used to test the null hypotheses that gas volumes were equal with and without abdominal pressure at each ventilation pressure. Separate analyses were performed for static abdominal pressure applied with the heart beating normally (the basic phenomenon) and for the more complex situations of standard and IAC-CPR. A similar analysis was performed for measurements of brachial arterial blood pressure and the arteriovenous pressure difference during CPR. A P value of 0.05 was considered significant.

RESULTS

Figure 3 presents results obtained in Phase I of the experiment, with the heart beating normally. Positive pressure ventilations through the unprotected airway were compared with and without continuous manual abdominal pressure at 100 mm Hg. Pulmonary tidal volumes ranged from 37 to 73 ml/kg without abdominal compression and 21 to 52 ml/kg with abdominal compression. Gastric gas volumes ranged from 0 to 24 ml/ kg without abdominal compression and 0 to 7 ml/kg with abdominal compression. Abdominal pressure restricted both pulmonary and gastric gas volumes at all inspiratory pressures tested; however, the relative reduction in gastric gas volume was greater than that in pulmonary tidal volume.

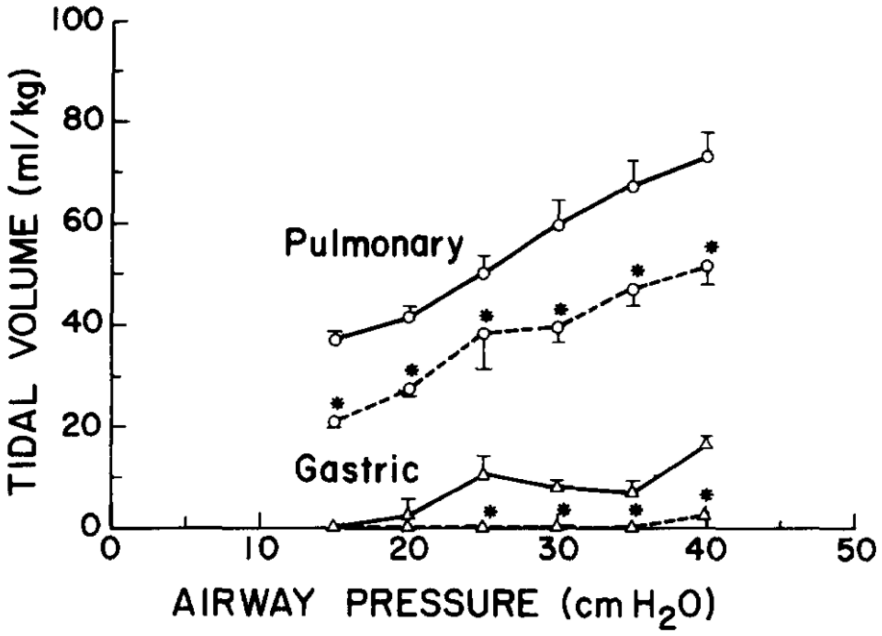

FIGURE 3. Gas volume per ventilation entering lungs and stomach with heart bearing normally. Solid lines = control; dashed lines = continuous abdominal pressure. The symbol * indicates a statistically significant effect of abdominal pressure at a particular airway pressure level (paired t-test. $P < 0.05$).

Figure 4 presents similar results of Phase II of the experiment, during experimental cardiac arrest and CPR. Positive pressure ventilations through the unprotected airway were compared both with (dashed lines) and without (solid lines) the addition interposed abdominal compressions at 100- 120 mm Hg to otherwise standard CPR provided by the Thumper® mechanical resuscitator. As in Phase I, abdominal counterpulsation, applied during the interval of positive pressure ventilation, restricted pulmonary and gastric gas volumes. The reductions were statistically significant ($P < 0.05$) at all ventilation pressures tested. There was no detectable gastric insufflation in nearly half the trials of IAC-CPR conducted over the complete range of ventilation pressures (Fig. 5). Pulmonary tidal volume, although reduced, was still more than adequate for resuscitation (at least 40 ml/kg).

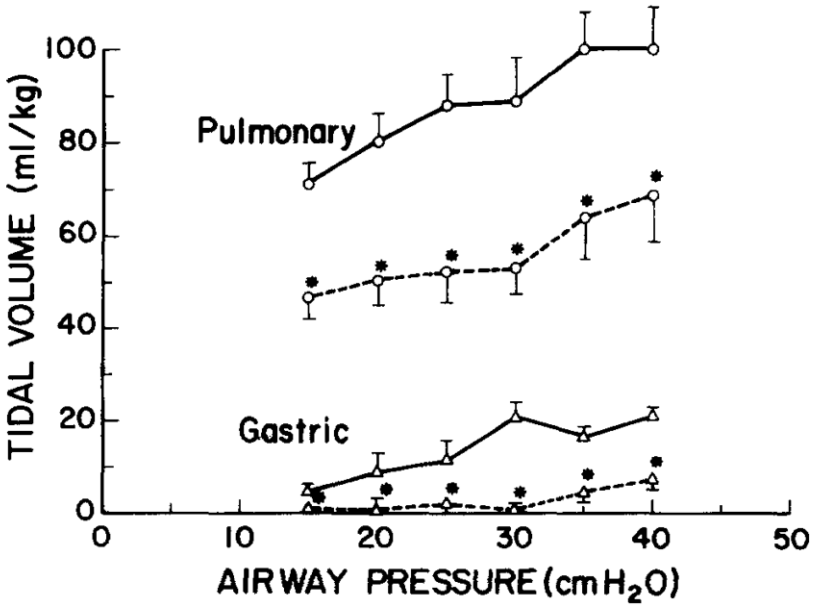

FIGURE 4. Gas volume per ventilation entering lungs and stomach during electrically induced ventricular fibrillation and CPR. Solid lines = standard CPR; dashed lines = IAC-CPR. The symbol * indicates a statistically significant effect of abdominal pressure at a particular airway pressure level (paired t-test, $P < 0.05$).

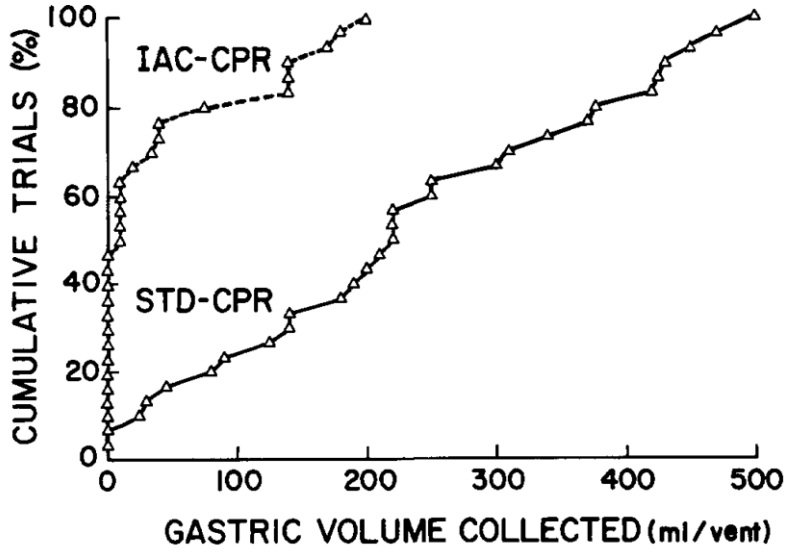

FIGURE 5. Cumulative distribution of gastric gas volumes collected during IAC-CPR versus standard CPR. Vertical axis represents percentage of all trials at all ventilation pressures. Horizontal axis represents collected gas volume. Curves indicate percent of trials in which collected gas volume was less than a chosen value on the horizontal axis. In half the trials with IAC-CPR, there was no detectable gastric insufflation.

Beneficial effects of IAC-CPR upon arterial blood pressures that were similar to, but less dramatic than, those reported in previous studies, were also seen in this study. Mean diastolic arterial pressure was 29.0 ± 1.8 mm Hg during IAC-CPR and 24.0 ± 1.8 mm Hg during standard CPR, a statistically significant difference. Mean systemic perfusion pressures (diastolic arterial minus diastolic right ventricular pressure) were 23.6 ± 1.7 mm Hg during IAC-CPR and 21.1 ± 1.6 mm Hg during standard CPR, a difference that did not reach statistical significance. Postmortem examination revealed no grossly observable injuries to abdominal organs other than those directly attributable to the surgical preparation. In particular, no lacerations of the liver were observed in any of the dogs.

DISCUSSION

Gastric insufflation and distension are major complications of CPR. Nagel and coworkers,[9] for example, report gastric dilation in 28% of 2,228 cases of prehospital CPR. Oropharyngeal or tracheal vomitus was found at autopsy in 18 percent of the cases. It is generally believed that gastric inflation is the antecedent cause of regurgitation during CPR. Gordon[10] has pointed out that gastric distension has a number of deleterious effects in CPR. The distended stomach elevates both hemidiaphragms, which may distort the position of the heart and great vessels and impair venous return. The enlarged stomach compresses intra-abdominal viscera and reduces venous return and, ultimately, cardiac output. The distended stomach may promote vagal reflexes that are deleterious to the circulation. The distended stomach is prone to regurgitation, which carries liquid gastric contents into the pharynx where they are frequently aspirated into the tracheobronchial tree.

Stephenson[11] cites clinical evidence that if gastric distension is prevented by hyperextension of the head and opening of the airway, the incidence of regurgitation is reduced. The present study suggests that gastric distension can be prevented or significantly reduced by abdominal counterpulsation during the ventilation phase, provided that IAC-CPR is started at the beginning of basic life support, before the stomach becomes distended with air. If standard CPR without IAC has already led to gastric dilatation, the stomach can be decompressed manually, as is usually taught. Then, IAC-CPR can be initiated in an effort to prevent further distension of the stomach with air.

Although IAC-CPR did reduce pulmonary tidal volumes, as well as gastric insufflation, when a constant pressure ventilator was used, the tidal volumes obtained (45-70 ml/kg) were still more than adequate. Moreover, if fixed-volume ventilation is applied, IAC-CPR actually improves oxygen uptake from the lungs (from 4.1 to 6.4 ml O_2/kg/min) because of enhanced perfusion.[2]

The higher inspiratory pressures tested in our study are not necessary for effective CPR. Indeed, high ventilation pressures during experimental CPR produce respiratory alkalemia, with arterial blood P_{CO2} values approaching 10 mm Hg when ventilation pressures in excess of 20 cm H_2O are applied.[12,13] Previously, we reported that normal arterial blood P_{CO2} tension is maintained with an inspiratory pressure of 20 cm H_2O during experimental CPR in dogs.[14] Ralston has shown

that normal blood gas levels are maintained for 20 minutes of experimental CPR, with ventilation provided by the Thumper® at 20 cm H_2O.[15] Most likely, inspiratory pressures in the range of 20 to 30 cm H_2O are satisfactory for tissue oxygenation during basic life support, and under these circumstances, IAC reduced median gastric insufflation from 200 to 0 ml per ventilation in our model. Accordingly, we believe that IAC-CPR can be performed safely with moderate ventilation pressure.

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