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Influence of interposed ventilation pressure upon artificial cardiac output during cardiopulmonary resuscitation in dogs

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ABSTRACT

This study was conducted to determine the effects of high pressure interposed ventilations during cardiopulmonary resuscitation (CPR). Cardiac output was measured by a modified indicator dilution technique in eight anesthetized, intubated mongrel dogs. Positive pressure ventilations (12/min, 80% O₂) were interposed after every five chest compressions (performed at 62/min) by a mechanical chest compressor (Thumper[®]). On repeated trials in the same animal, ventilation pressures from 10-50 cm of H₂O were tested in randomized sequence, while the technique of chest compression was held constant. Arterial blood gases immediately after resuscitation were monitored. Increasing ventilation pressure had surprisingly little effect on cardiac output during CPR, although blood gases were profoundly altered. For ventilation pressures of 10, 20, 30, 40, and 50 cm of H₂O, producing mean tidal volumes 23, 38, 61, 83, and 94 ml/kg; cardiac output remained nearly constant, averaging 21, 25, 23, 26, and 24 ml/min/kg. Corresponding mean post-resuscitation pH was 7.24, 7.41, 7.51, 7.56, and 7.53; PCO₂ was 41, 26, 18, 16, and 15 torr. The post-resuscitation arterial oxygen tension was greater than 100 torr at all ventilation pressures except 10 cm of H₂O. Interposed ventilations of pressure and volume more than adequate to prevent acidosis during CPR did not impair artificial cardiac output. If anything, cardiac output was slightly improved by more forceful ventilation.

INTRODUCTION

Ventilation in CPR may accomplish more than pulmonary gas exchange. Certain recent findings suggest that ventilation pressure may be put to work to augment blood flow during CPR as well as to provide necessary gas exchange. The phenomenon of cough CPR reported by Criley et al. [1] proves that generalized pressure increases within the chest are capable of causing forward flow of blood without manual chest compression. (A detailed account of cough-CPR is reported by Niemann et al. in this issue of *Critical Care Medicine*). Chandra [2], Weisfeldt [3], and associates at Johns Hopkins have investigated the hemodynamic benefits of forceful ventilation applied simultaneously with external chest compressions in such a fashion as to produce the greatest generalized intrathoracic pressure rise during CPR. Their technique of "new CPR" is developed on the theoretical premise that traditional CPR is effective not because the heart is squeezed between the sternum and the spine, but rather because a generalized increase in intrathoracic pressure squeezes blood from the lungs, through the heart, and into the periphery. Their preliminary results in both animals and man suggest that cerebral blood flow may be increased several-fold by this technique. In view of these recent developments, the authors studied the effect of increasing the ventilation pressure during standard CPR in animals.

METHODS

Animal Preparation

Eight mongrel dogs weighing 6-12 kg served as subjects. Relatively younger animals with compliant chest walls and with dorsal-ventral versus right-left thoracic diameters less than 1.6:1.0 were selected for the study. Each animal was anesthetized with pentobarbital sodium (30 mg/kg iv) and intubated with the largest possible cuffed endotracheal tube. Intravascular catheters were inserted as follows: (1) a pigtail catheter advanced into the left ventricle for injection of indicator to measure cardiac output, (2) a catheter advanced to the midthoracic aorta and attached to a motor-driven syringe for withdrawal of blood during inscription of dilution curves, and (3) a catheter to monitor arterial pressure advanced into the right brachial artery to a position just distal to the thoracic inlet. Heparin (1 mg/kg iv) was given to retard clot formation in the catheters, to permit reinfusion of blood withdrawn during inscription of dilution curves, and to diminish intravascular coagulation during periods of circulatory arrest. The animal was placed on a V-shaped board which was fixed securely to the baseplate of a specially modified Thumper[®] mechanical resuscitator (Michigan Instruments, Inc., Grand Rapids, MI). Subcutaneous electrodes for recording the electrocardiogram (lead II) were secured in place, and wire mesh electrodes for sternal-to-back defibrillation were applied to the shaved skin of these regions with electrolytic jelly. The wire mesh of the sternal electrode was molded to the chest compression pad of the Thumper[®]. With this electrode arrangement, defibrillation could be accomplished easily on the down-stroke of compression, without interrupting CPR.

Physiological Monitoring

A four-channel graphic record was inscribed using a Physiograph direct-inking recorder (Narco Bio-Systems, Houston, TX). Channels 1 and 2 displayed the ECG and arterial blood pressure. Electronically derived mean arterial pressure (MAP) could be obtained with front-panel switches on the pressure transducer driver so that the MAP could be recorded during episodes of CPR.

Intraesophageal pressure was recorded on channel 3 as a monitor of chest compression and ventilation. The amplitude of esophageal pressure pulses was used to normalize chest compression among animals of different size and to prevent traumatic over-pressurization of the chest. Pressure was detected by a 25 cm long x 1 cm diameter soft rubber tube placed in the esophagus between the thoracic inlet and the diaphragm. This tube was filled with water and connected by a 0.2 cm internal diameter line to a pressure transducer.

Channel 4 of the graphic record displayed indicator dilution curves for the measurement of cardiac output by the saline-conductivity method [4-5]. This method employs 5% NaCl solution as the indicator and a calibrated, flow-through conductivity cell as the detector. Two ml aliquots of 5% saline indicator were injected forcibly into the left ventricle and blood samples were withdrawn through the detector via the catheter placed in the thoracic aorta. This injection-sampling configuration is similar to the right ventricular injection-pulmonary artery sampling scheme employed in commercial thermodilution systems and appears to permit adequately uniform mixing of indicator in blood, even under the circumstances of CPR [5]. Dilution curves were inscribed by withdrawing blood from the thoracic aorta through the conductivity cell at a rate of 10 ml/min, a value at most 10%, and typically about 5%, of the cardiac output during resuscitation.

Experimental CPR

In a typical arrest/resuscitation sequence, ventricular fibrillation was produced by 60 Hz electrical stimulation of the left ventricular endocardium. A fine, 0.1 mm, stainless steel wire threaded through the lumen of the left ventricular catheter facilitated conduction of electric current to the heart for this purpose. Ventricular fibrillation was confirmed by the presence of chaotic fibrillation waves in the ECG and by loss of arterial blood pressure.

Immediately after confirmation of fibrillation, ventilation and chest compression were initiated using the mechanical Thumper. The 6 x 10 cm chest compression pad was centered in the midline with its caudal edge at the level of the xiphisternal junction. The Thumper was energized with 100% oxygen at 60 psi, and the effective inspired oxygen concentration (FIO₂) was approximately 80%. Ventilation pressures were selected as specified by the protocol. Ventilations were always interposed by the Thumper after every fifth chest compression for a duration of 1.0 sec. The overall compression rate was 62/min, and the compression duration was 50% of compression cycle time. The force of compression was adjusted to maintain a peak esophageal pressure during CPR of 50 ± 5 mm Hg on all trials, so that effective compression

amplitude was held constant. After a 20 sec stabilization period, high chart speed (1 cm/sec) records of pulsatile pressure changes were recorded; then electronically derived MAP was recorded at a chart speed of 1 cm/sec for 30-50 sec, during which time a dilution curve was obtained. After inscription of a final 10 sec record of pulsatile data at 1 cm/sec, a damped sine wave defibrillator shock of 20-50 joules was applied during the downstroke of the chest compression via the chest-to-back electrodes, and the animal was allowed to recover until a stable MAP was attained.

Immediately after defibrillation, arterial blood samples were obtained and analyzed immediately for pH, PCO₂, and PO₂ with the aid of an Instrumentation Laboratories Model 213 blood gas analyzer. These values were maintained as close as possible to normal by administration of sodium bicarbonate between resuscitations and by adjustment of airway dead space to counteract the tendency toward mixed metabolic acidosis and respiratory alkalosis.

Manipulation of Ventilation Pressure

On successive trials, the ventilation pressure was adjusted to 10, 20, 30, 40, or 50 cm of H₂O according to a quasi-random sequence in which high inspiratory pressures were deliberately tested after low inspiratory pressures and vice versa to minimize perturbations in the prearrest arterial blood gas values. Tidal volume for each dog at each ventilation pressure was measured with a Wright respirometer. During recovery periods between resuscitations, ventilation was adjusted to bring the arterial blood gases as close as possible to the normal values before the next trial, and the animal's spontaneous breathing of room air was encouraged. In this manner, at least two and often three repeated measurements of cardiac output, MAP, and arterial blood gases were obtained at each level of ventilation.

Postmortem Examination

At the end of the study, a careful postmortem examination was performed with special attention to possible complications of high pressure ventilation, such as pneumothorax and pulmonary rupture.

RESULTS

Increased ventilation pressure generated increased ventilation volume in an almost linear fashion (Fig. 1). A tidal volume of approximately 2 ml/kg body weight/cm of H₂O inspiratory pressure was obtained at all levels of ventilation, meaning that at 50 cm of H₂O inspiratory pressure, a tidal volume of 1 liter was produced in a typical 10-kg dog. The subjective impression of the authors was that this extreme inspiratory pressure caused over-ventilation of the animal, although no traumatic sequelae of high pressure ventilation were seen at necropsy.

FIG. 1. Mean tidal volume ($\pm$ SE) in eight dogs during standard CPR as a function of interposed ventilation pressure.

Arterial blood gas data (Fig. 2) further characterized the range of inspiratory pressures tested. At 10 cm of H₂O inspiratory pressure the animals were acidotic, compared to the initial control values obtained before any resuscitations were carried out (mean control pH = 7.34, PCO₂ = 42 torr, breathing room air). Inspiratory pressures of 20 cm of H₂O produced nearly normal post-resuscitation blood gases; whereas greater ventilation pressures caused progressively more severe hypocarbia and alkalosis. These changes in acid-base status after high pressure ventilation are remarkable in that they occurred after relatively brief durations of CPR (always less than 2 min). Some intrapulmonic shunting of venous blood past unventilated alveoli is evidenced by the relatively low PaO₂ values measured in the face of ventilation with 80% O₂.

FIG. 2. Arterial blood gas values (mean $\pm$ SE) immediately after 1 to 2-min episodes of standard CPR as a function of interposed ventilation pressure. In (c) PaO₂ exceeded the upper sensitivity limit of the blood gas apparatus for inspiratory pressures of 40 and 50 cm of H₂O and was greater than, or equal to 300 torr.

FIG. 3. Relative cardiac output at various inspiratory pressures during standard CPR in eight dogs. A CPR output of 100% represents the grand mean value for each animal over all ventilation pressures tested: each data point represents one animal.

The effect of increasing interposed ventilation pressure on cardiac output is illustrated in Figure 3. Each data point represents the mean cardiac output for a given animal at a given inspiratory pressure, expressed as a percentage of the grand mean for that animal over all inspiratory pressures. This normalization procedure corrected for the animal-to-animal variations in overall mean cardiac output, which were considerable in this study: 39, 31, 28, 26, 24, 21, 13, and 10 ml/min/kg for the eight animals. Nonetheless, in a given animal, there was little effect of inspiratory pressure upon cardiac output. In particular, increased positive pressure ventilation did not decrease cardiac output during CPR. Rather, there was a slight, but not significant, increase ($r^2 = 0.07$). Similarly, there was no significant effect of interposed ventilation pressure upon MAP during CPR ($r^2 = 0.01$).

DISCUSSION

High pressure, interposed ventilation during CPR is practicable if performed through a cuffed endotracheal tube, so that the risk of massive gastric insufflation is minimal. Higher ventilation pressures during standard CPR tend to improve the PaO_2 and reduce intrapulmonic shunting, without decreasing cardiac output. The risk associated with this benefit is a tendency toward severe respiratory alkalosis, even after brief periods of cardiac arrest and resuscitation. For this reason, a clear advantage can be seen in performing interposed ventilation during CPR at pressures sufficient to provide adequate arterial oxygenation. However, there appears to be no great hemodynamic benefit from a further increase in ventilation pressure during standard CPR.

The authors do emphasize, however, that the present findings are strictly limited to the conventional interposed ventilation mode (i.e., ventilation between, not during chest compressions). Opposite conclusions could well be possible for simultaneous ventilation and compression as recently described by Chandra et al. [2] and Weisfeldt et al. [3]. In particular, high tidal volumes and over-ventilation with CO_2 depletion may be much less of a problem because simultaneous chest compression would tend to oppose lung inflation. Further, the high pressure spikes generated by chest compression simultaneous with ventilation would also tend to collapse the esophagus and mitigate against gastric insufflations, even in the absence of a cuffed endotracheal tube. For these reasons, the authors would encourage further investigations of the simultaneous ventilation mode, although the advantages of high pressure interposed ventilation appear to be minimal.

REFERENCES

1. Criley JM, Blaufuss AH, Kissel GL: Cough-induced cardiac compression: self administered form of cardiopulmonary resuscitation. *JAMA* 236:1246, 1976
2. Chandra N, Rudikoff M, Weisfeldt ML: Simultaneous chest compression and ventilation at high airway pressure during cardiopulmonary resuscitation (CPR) in man. *Circulation* 58: 203, 1978
3. Weisfeldt ML, Rudikoff MT, Cohen J. et al: Movement of blood through manipulation of intrathoracic pressure (abstr). *Med Instrumentation* 14:59, 1980
4. Geddes LA, Peary E, Steinberg R: Cardiac output using an electrically calibrated flow-through conductivity cell. *J Appl Physiol* 37:972, 1974
5. Geddes LA, Babbs CF: A new technique for the measurement of cardiac output during cardiopulmonary resuscitation. *Crit Care Med* 8:132. 1980