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ABSTRACT

The ability of a new modification of cardiopulmonary resuscitation (CPR) to deliver oxygen to tissues was evaluated. The method utilizes standard CPR techniques with the addition of manual abdominal compressions (≈100 mm Hg) interposed between chest compressions, and is termed interposed abdominal compression-CPR (IAC-CPR). Oxygen delivery was measured by a spirometer in a closed circuit designed to permit positive-pressure ventilation synchronized with mechanical chest compression. Ventricular fibrillation was induced electrically in 10 anesthetized dogs. In each dog, trials of IAC-CPR and standard CPR were alternated every five minutes during a 30-minute period. Arterial and central venous blood pressures, oxygen consumption, and Fick cardiac output were monitored. The addition of interposed abdominal compression significantly ($P < 0.01$) increased each of these hemodynamic indicators. Oxygen delivery increased from 4.12 ± 0.39 ml O_2/kg/min during standard CPR to 6.37 ± 0.35 ml O_2/kg/min during IAC-CPR. Arterial systolic blood pressure increased from 67 ± 5 mm Hg to 90 ± 5 mm Hg, while diastolic arterial blood pressure rose from 15 ± 2 mm Hg to 33 ± 3 mm Hg. Cardiac output increased from 19.9 ± 2.6 ml/min/kg to 37.5 ± 2.7 ml/min/kg.

Key words: adjuncts, blood flow, cardiac arrest, IAC-CPR, ventricular fibrillation

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INTRODUCTION

The immediate goal of manual cardiopulmonary resuscitation (CPR) is to deliver oxygen to peripheral tissues in a victim of cardiopulmonary arrest until definitive therapy can be administered [1, 2]. Recently we developed an apparent improvement in manual CPR which increases blood pressures and cardiac output [3]. We have termed this modification "IAC-CPR" to denote the addition of interposed abdominal compressions (IAC) to standard CPR. This method of manual abdominal counterpulsation is illustrated (Figure 1). It includes all the procedures of standard CPR, and so constitutes an evolution rather than a revolution in technique. To determine if IAC-CPR actually improves oxygen delivery and, if so, to elucidate the mechanism, we conducted the following study in which oxygen delivery during CPR was measured directly.

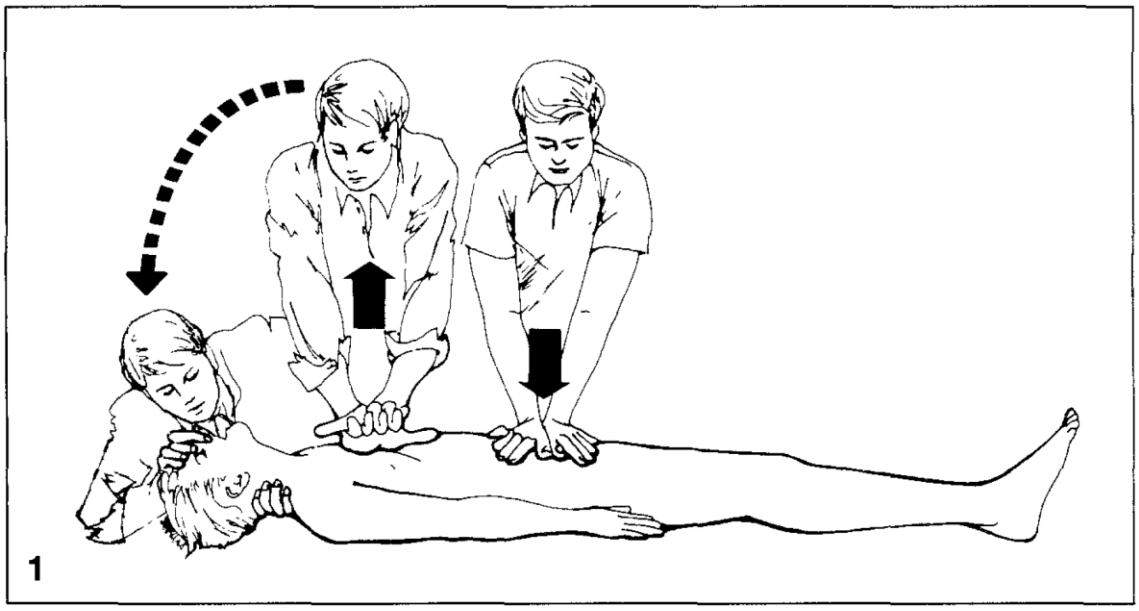

Fig. 1. ***Artist's conception of basic rescuers performing IAC-CPR. For clarity, both rescuers are shown on the same side of victim. With two rescuers, the first compresses the chest and ventilates while the second compresses the abdomen. With three rescuers, ventilation, chest compression, and abdominal compression are performed by separate individuals.***

METHODS

Oxygen Delivery During CPR

To compare oxygen delivery during IAC-CPR with that during standard CPR, we measured oxygen uptake with a spirometer during alternate five minute trials of the two techniques in dogs with electrically induced ventricular fibrillation. The positive-pressure ventilation system that enabled measurement of oxygen uptake is diagrammed (Figure 2). The system forms a closed loop between a recording spirometer and the trachea. The system is driven by the ventilation line of a mechanical chest compressor and ventilator (Thumper™, Michigan Instruments Inc., Grand Rapids, MI).

During CPR, the ventilatory pulse generated by the Thumper does not ventilate the dog directly, but instead raises the pressure in an air-tight container housing a bellows. Compression of the bellows forces 100% oxygen through a one-way valve into the lungs. Simultaneously a solenoid valve on the return line from the dog to the spirometer is actuated to prevent the positive-pressure pulse from reaching the spirometer. After ventilation, this valve opens and passive deflation of the lungs forces exhaled gas through a CO_2 absorber into the spirometer. A counterweight causes the bellows to refill, via another one-way valve, with 100% oxygen from the spirometer, completing the cycle. Changes in the position of the spirometer bell are monitored by means of a rotary potentiometer fixed to the pulley suspending the counterweighted bell. This potentiometer provides an electrical signal linearly related to the bell volume which is displayed on a graphic recorder. In each experiment, this signal was calibrated by introducing known volumes of gas into the bell with a 1000-ml syringe. The slope of the graphically recorded spirogram provides a direct measure of oxygen consumption.

Fick Cardiac Output

Given oxygen consumption data, cardiac output was measured by the Fick method as:

$$\text{Cardiac output (ml/min)} = \frac{\text{O}_2 \text{ uptake (ml O}_2\text{/min)}}{\text{arteriovenous O}_2 \text{ difference (ml O}_2\text{/ml blood)}}$$

The oxygen contents of arterial and mixed venous (right ventricular) blood samples, withdrawn from the brachial artery and the right ventricle, were determined with a Lex-O_2-Con apparatus (Lexington Instruments Corp., Waltham, MA). All gas volumes were corrected to conditions of standard temperature and pressure--dry (STPD).

Animal Model

Ten mongrel dogs were selected. The dogs weighed 10 to 14.5 kg (mean, 12.3 kg) and had dorsal-ventral chest diameters ranging from 18 to 22 cm (mean, 20 cm) at the level of the heart and chest circumferences of 47 to 56 cm (mean, 52 cm). Each dog was anesthetized with pentobarbital sodium (30 mg/kg IV). The trachea was intubated and the cuff was fully inflated to prevent leaks.

Two catheters were inserted: one was advanced into the right ventricle via a jugular vein for monitoring right-sided diastolic pressures and for withdrawal of mixed venous blood samples; the other was advanced 5 to 10 cm into the right brachial artery for monitoring arterial pressure and for withdrawal of arterial blood samples. Heparin (1 mg/kg IV) was given to retard clot formation in the catheters, to permit reinfusion of withdrawn blood, and to diminish intravascular coagulation during CPR.

The animal was placed in dorsal recumbency on a V-shaped board, and the limbs were securely tied to the board to prevent lateral motion of the chest during CPR. The Thumper mechanical resuscitator was used to compress the chest and to power the closed-circuit ventilation system. Subcutaneous electrodes for recording the electrocardiogram (lead II) were secured in place.

The pad used for abdominal compression was a standard 12-cm width blood pressure cuff folded to 12 x 15 cm and inflated with air to a thickness of approximately 3 cm. The bladder of the cuff was attached to an aneroid manometer and to a linear core pressure transducer in order to monitor pressure applied to the abdomen. IAC-CPR was performed by manual compression of the mid-abdomen with this inflated pad in such a way as to generate pressure pulses of 100 to 120 mm Hg. The fingers were spread to provide a large surface area of compression approximately equal to that of the flattened blood pressure cuff. The duty cycle of abdominal compression was complementary to that of chest compression, i.e. 50% of cycle time (0.5 sec abdominal compression duration).

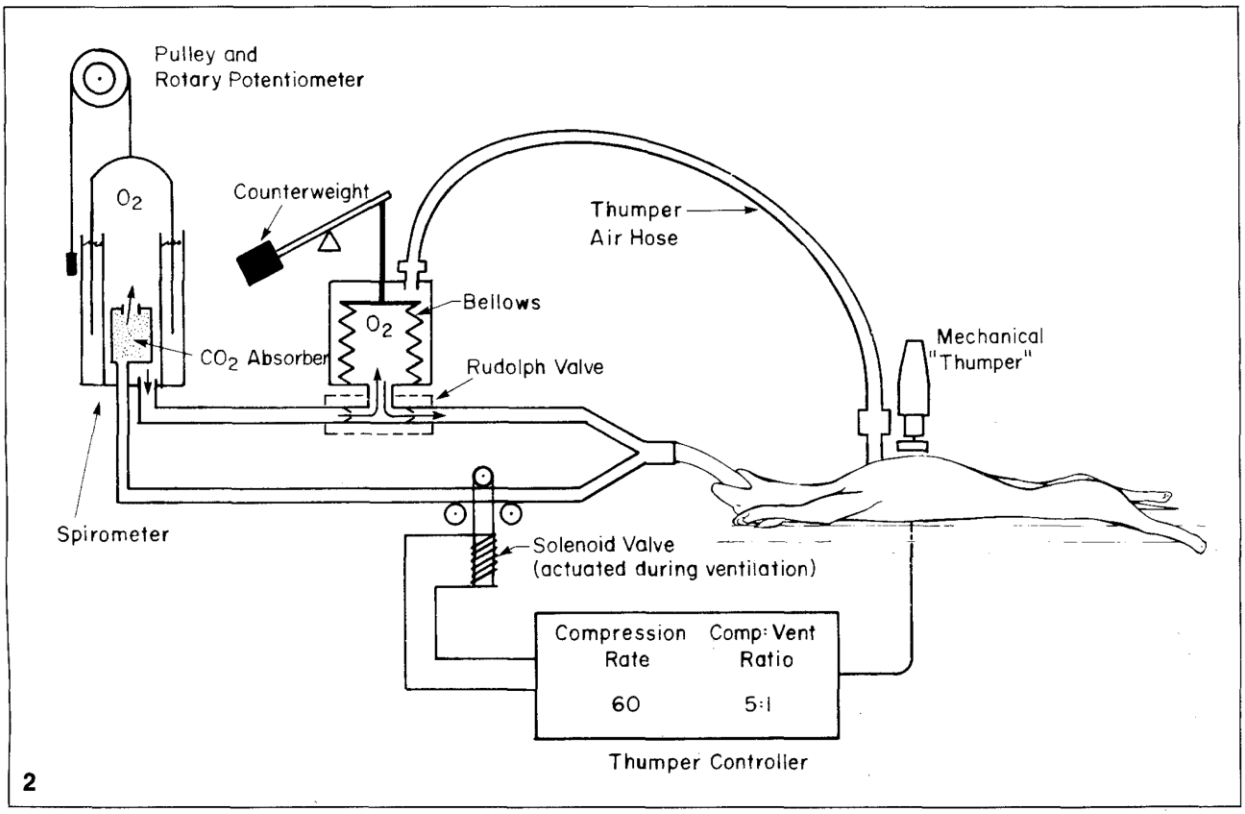

Fig. 2. ***System to measure oxygen uptake during CPR with positive-pressure ventilation. Unidirectional flow occurs in a closed circuit including a bell spirometer. The bellows provides positive-pressure ventilation. The spirometer is protected from positive-pressure ventilation by the Rudolph valve and by the solenoid valve which is synchronously actuated by the Thumper. Expired gas flows passively into the bell through a carbon dioxide absorber.***

Physiologic Monitoring

A six-channel graphic record was inscribed using a Physiograph direct-inking recorder (Narco Bio-Systems, Houston, TX). Channels 1 and 2 of the graphic record displayed calibrated changes in spirometer volume. Channel 2 had twice the gain of channel 1, and was electronically filtered to facilitate measuring slopes in the presence of oscillations induced by CPR. Channels 3 and 4 displayed arterial and right ventricular blood pressures, respectively. While the heart was beating normally, channel 5 displayed the ECG. After ventricular fibrillation was confirmed, channel 5 was used to display the abdominal compression pressure. Pressure channels were calibrated and their linearity confirmed using a mercury manometer.

Experimental CPR

First, control measurements of blood pressures, oxygen uptake, and cardiac output were obtained with the heart beating normally. As a check for leaks in the closed circuit ventilation system, oxygen uptake was measured during positive pressure ventilation at rates of 12, 20, and 30 breaths per minute. (In the presence of a leak, the apparent oxygen uptake will increase with ventilation rate during this maneuver. In a tightly closed system, oxygen uptake remains constant.) Next, a single episode of ventricular fibrillation was produced by 60 Hz electrical stimulation of the right ventricular endocardium. A fine, 0.1-mm, stainless steel wire threaded through the lumen of the right ventricular catheter carried electric current to the heart for this purpose. Immediately after electrocardiographic confirmation of fibrillation, ventilation and chest compression were initiated using the Thumper and the closed circuit for ventilation previously described. This system provided standard CPR continuously throughout the experiment.

An initial two-minute period of standard CPR was performed in all dogs to adjust the force of compression. The chest compression force, 40 to 60 lbs, was selected to produce approximately 20% to 25% sternal displacement (expressed as a percentage of dorsal-ventral chest diameter) during standard CPR, and was not changed thereafter during the experiment. During both standard CPR and IAC-CPR, the ventilation duration was 0.5 seconds, and ventilations were interposed after every fifth chest compression. Ventilation was adjusted to deliver tidal volumes of 30 ml/kg during both types of CPR. In both standard and IAC-CPR the compression rate was 60/min, and the duty cycle of chest compression was 50% of cycle time (compression duration, 0.5 seconds).

The technique of abdominal compression was added to the CPR provided by the Thumper during alternate five-minute intervals. Three five minute trials of IAC-CPR and three five-minute trials of standard CPR were performed alternately in the same animal during the one continuous episode of ventricular fibrillation. In half the dogs IAC-CPR was begun first, and in the other half standard CPR was begun first. In this sense, each animal served as its own control, and the experiment was not biased by virtue of treatment order.

Blood Velocity Measurements

At the end of the experiment, blood velocity measurements were made in the inferior vena cava, at the level of the diaphragm, with a Carolina Instruments electromagnetic flow velocity probe in four of the ten dogs. CPR was stopped entirely for 30 seconds to provide a zero flow reference level, and then the uncalibrated flow velocity signal was recorded during alternate periods of standard CPR and IAC-CPR to determine if the qualitative patterns of blood flow were different during the two techniques.

Postmortem Examination

Finally, a gross postmortem examination was performed to identify any trauma to the abdominal viscera as a result of trials of IAC-CPR.

Data Analysis

To compare effects of experimental CPR, mean oxygen uptake during the three trials of standard CPR and the three trials of IAC-CPR was calculated for each animal. Student's t statistics for paired data were computed for these mean values to test the null hypothesis that the oxygen uptake per kilogram was the same during IACCPR and standard CPR. A similar analysis was performed for measurements of Fick cardiac output, A-V O_2 difference, brachial artery blood pressure, and the diastolic pressure difference between the brachial artery and the right ventricle. A square root transformation was performed for diastolic arterial blood pressure and diastolic arteriovenous pressure difference to meet the criterion of equal variances for the t test [4].

RESULTS

Records of standard CPR (Figure 3, left) and IAC-CPR obtained less than one minute later (Figure 3, right) are shown. The slopes of the spirograms in channel 1 (top, unfiltered) and channel 2 (filtered) are clearly steeper during IAC-CPR. At the same time, both systolic and diastolic arterial blood pressure increase (channel 3), while right ventricular filling pressure during chest recoil (diastole) remains near zero (channel 4). Hence the arteriovenous diastolic pressure difference is increased by IAC-CPR.

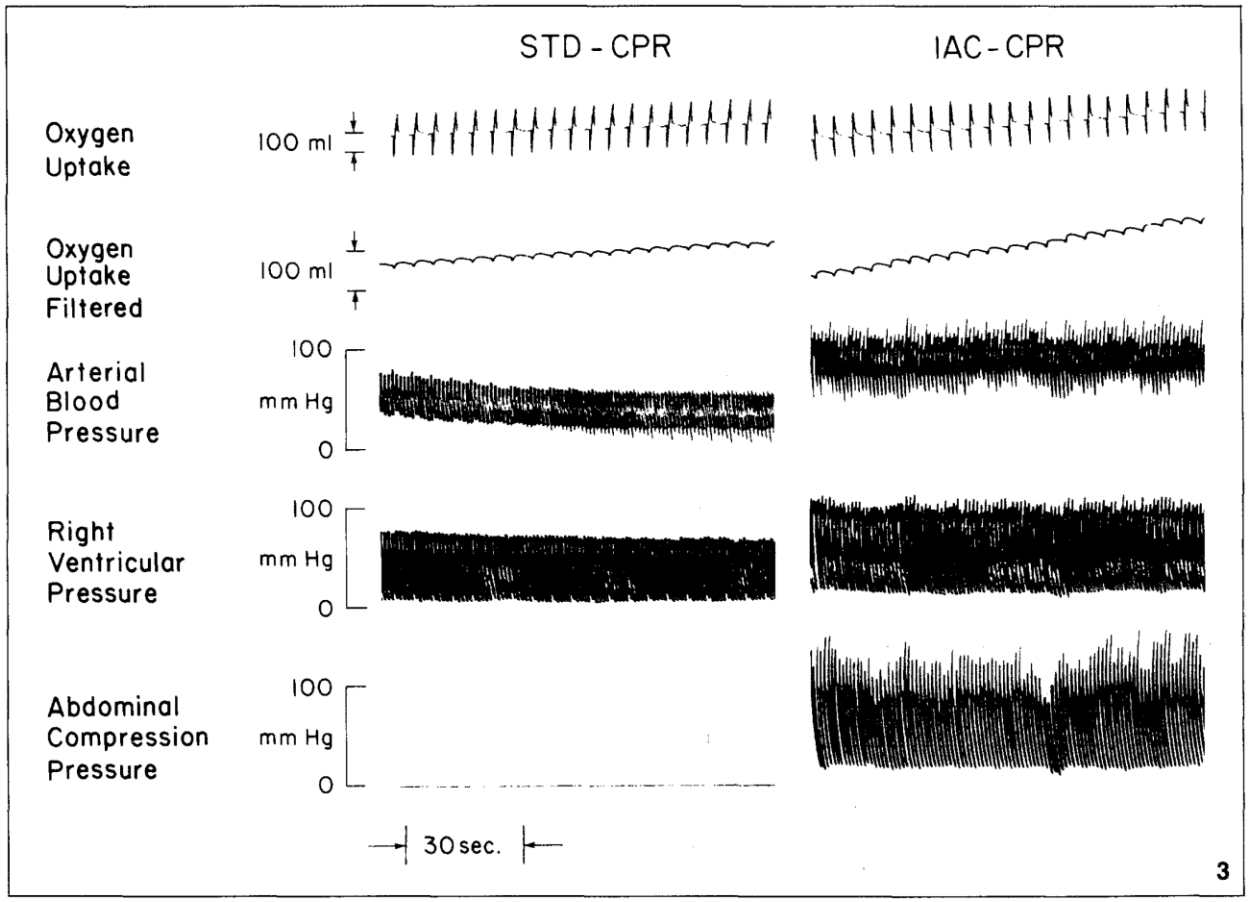

Fig. 3. ***Graphic records of oxygen uptake and blood pressures obtained during a typical experiment. The recording of IAC-CPR (right) was obtained less than 1 min after that of standard CPR (left).***

Oxygen Delivery

The mean value of oxygen consumption for three trials in each of 10 dogs for both standard and IAC-CPR is shown (Figure 4). Oxygen consumption was significantly greater ($P < 0.01$) during IAC-CPR than during standard CPR. Overall oxygen consumption was about 1.8 times greater during IAC-CPR (Table). Oxygen extraction (the arteriovenous O_2 difference) was not significantly different between IAC and standard CPR; mean values were 17.7 ± 0.7 and 18.6 ± 0.8 ml O_2/100 ml blood, respectively.

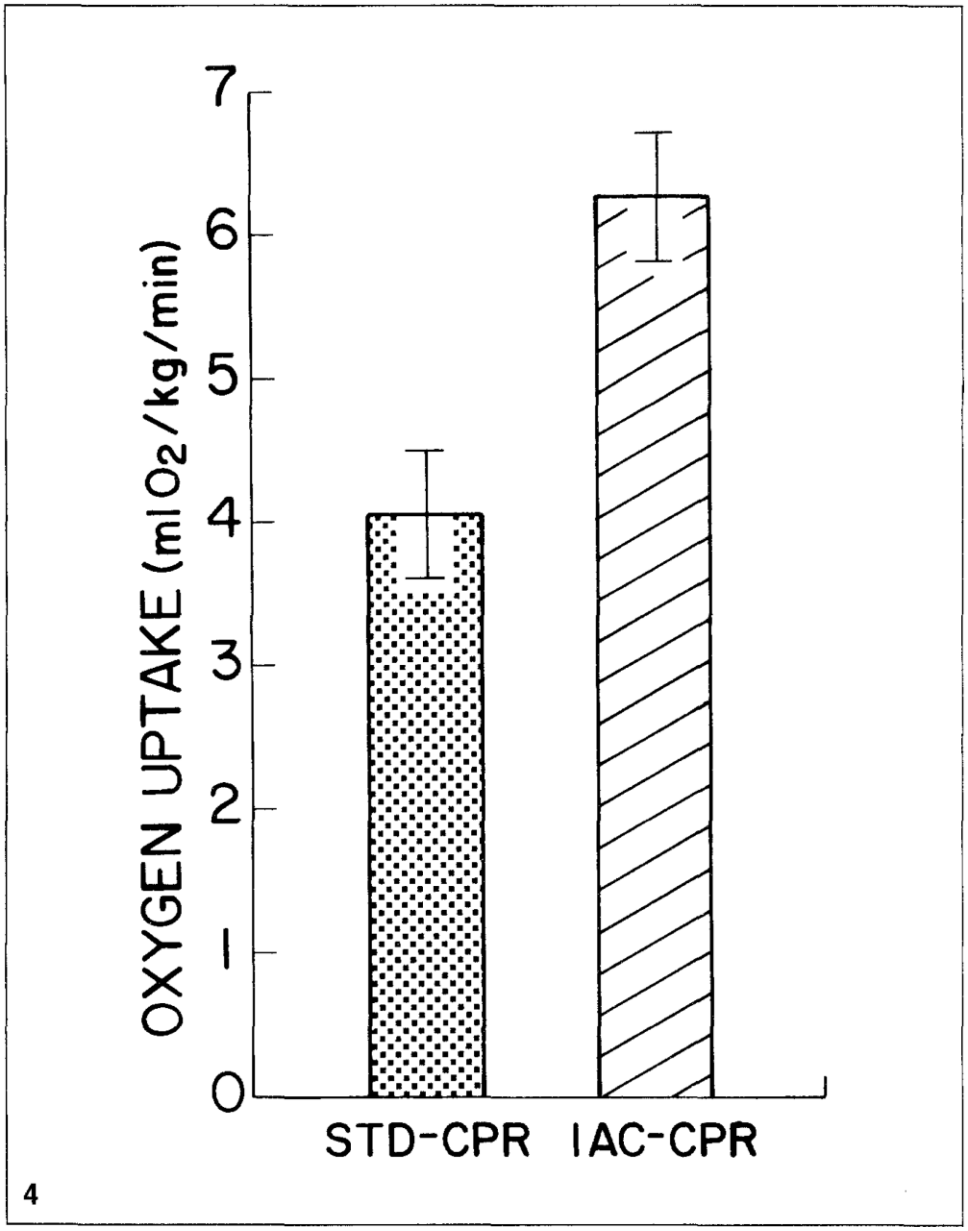

Fig. 4. ***Mean values (±SE) of oxygen uptake during standard CPR (STDCPR) and CPR with interposed abdominal compressions (IAC-CPR) in 10 dogs.***

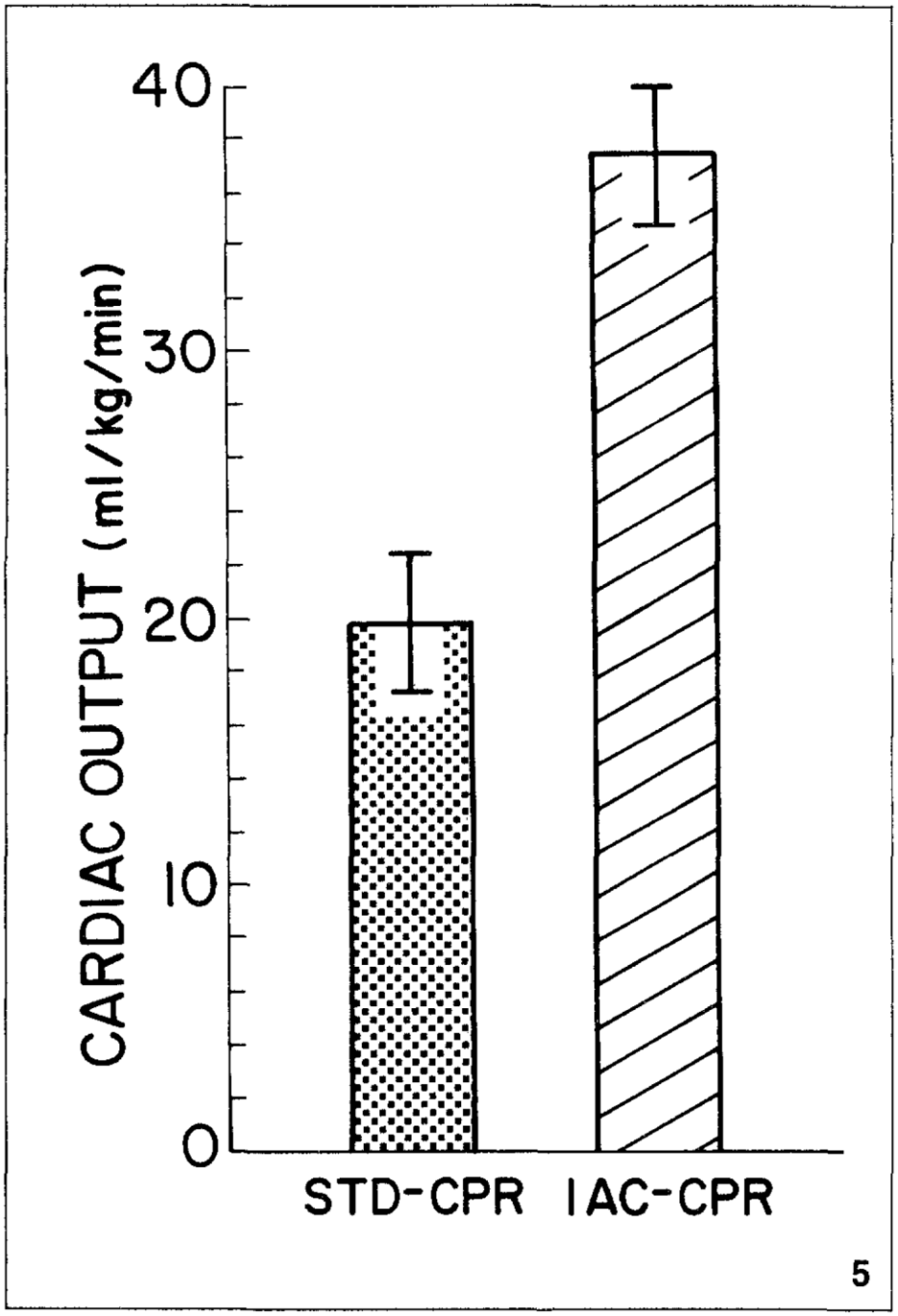

Fig. 5. ***Mean values (±SE) of cardiac output determined by the direct Fick method during standard CPR (STD-CPR) and CPR with interposed abdominal compressions (IAC-CPR) m 10 dogs.***

Cardiac Output

Cardiac output, determined by the direct Fick method, was significantly greater ($P < 0.01$) during IAC-CPR than during standard CPR (Figure 5). IAC-CPR improved cardiac output an average of 2.3 times (Table).

Blood Pressures

Systolic arterial blood pressure (Figure 6) and diastolic arterial blood pressure (Figure 7) were both significantly higher ($P < 0.01$) during IAC-CPR than during standard CPR. The diastolic arteriovenous blood pressure difference (diastolic arterial pressure --diastolic right ventricular pressure) was also significantly higher ($P < 0.01$) during IAC-CPR compared to standard CPR (Figure 8). Systolic arterial pressure during IAC-CPR was about 1.4 times that measured during standard CPR, and diastolic arterial pressure was about 2.6 times greater. The diastolic arteriovenous pressure difference was about 2.4 times greater during IAC-CPR (Table).

TABLE. *Ratios of values obtained during IAC-CPR to those obtained during standard CPR in 10 dogs*

	Systolic Arterial Pressure	Diastolic Arterial Pressure	Diastolic A-V Pressure Difference	Oxygen Uptake	Cardiac Output
	1.36	4.40	4.83	2.32	2.66
	1.83	3.48	4.70	2.94	4.09
	1.32	2.34	2.10	1.26	4.43
	1.42	2.36	2.23	1.24	1.41
	1.00	1.59	1.24	0.85	0.91
	1.91	3.61	2.95	2.42	2.15
	1.31	1.40	0.61	1.24	1.37
	1.34	3.60	2.48	2.30	2.60
	1.25	1.59	1.13	1.61	1.86
	1.27	1.71	1.49	1.38	1.50
Mean	1.40	2.61	2.38	1.76	2.30
± SEM	0.09	0.34	0.46	0.22	0.37

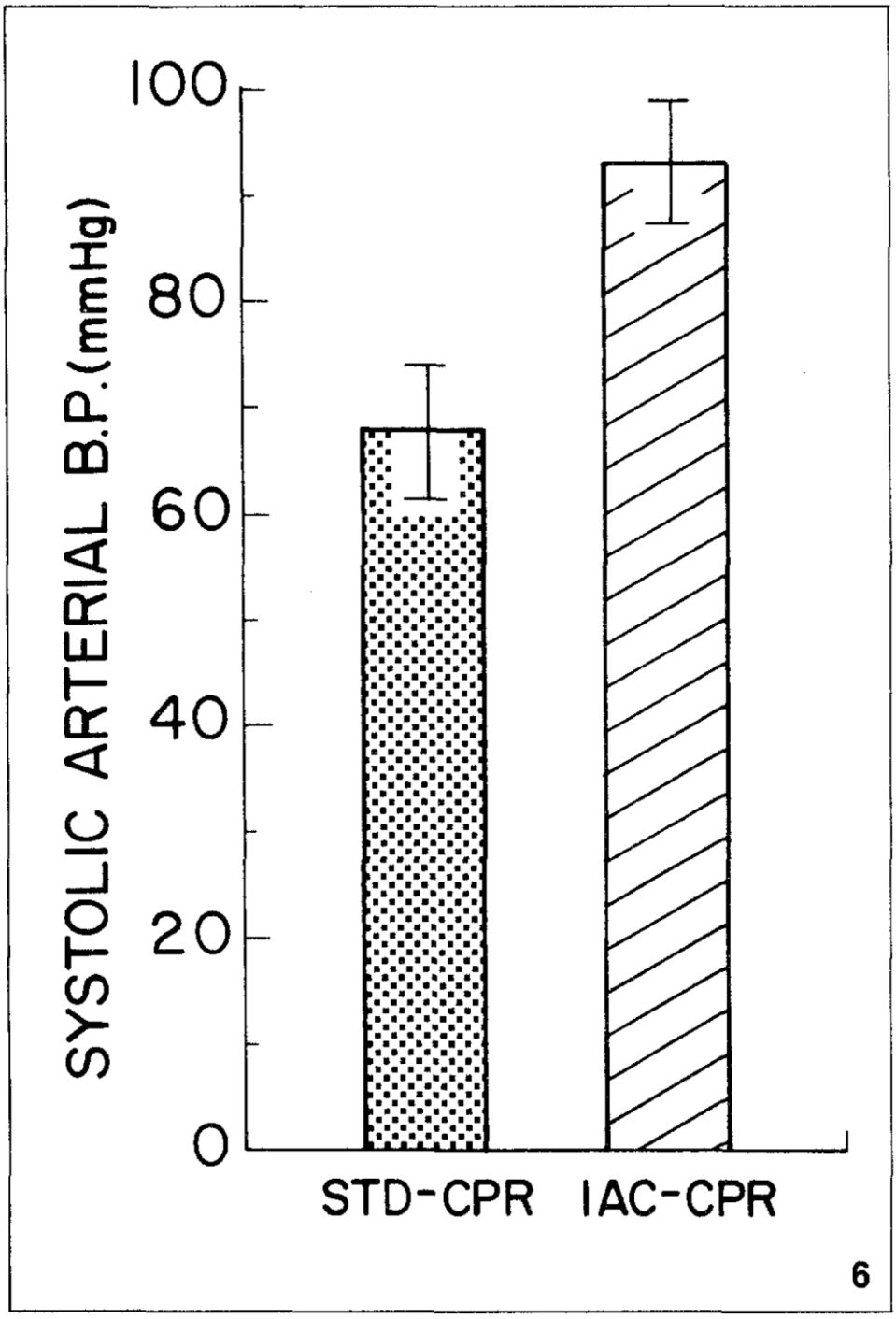

Fig. 6. ***Mean values (±SE) of systolic arterial blood pressure during standard CPR (STD-CPR) and CPR with interposed abdominal compressions (IAC-CPR) in 10 dogs.***

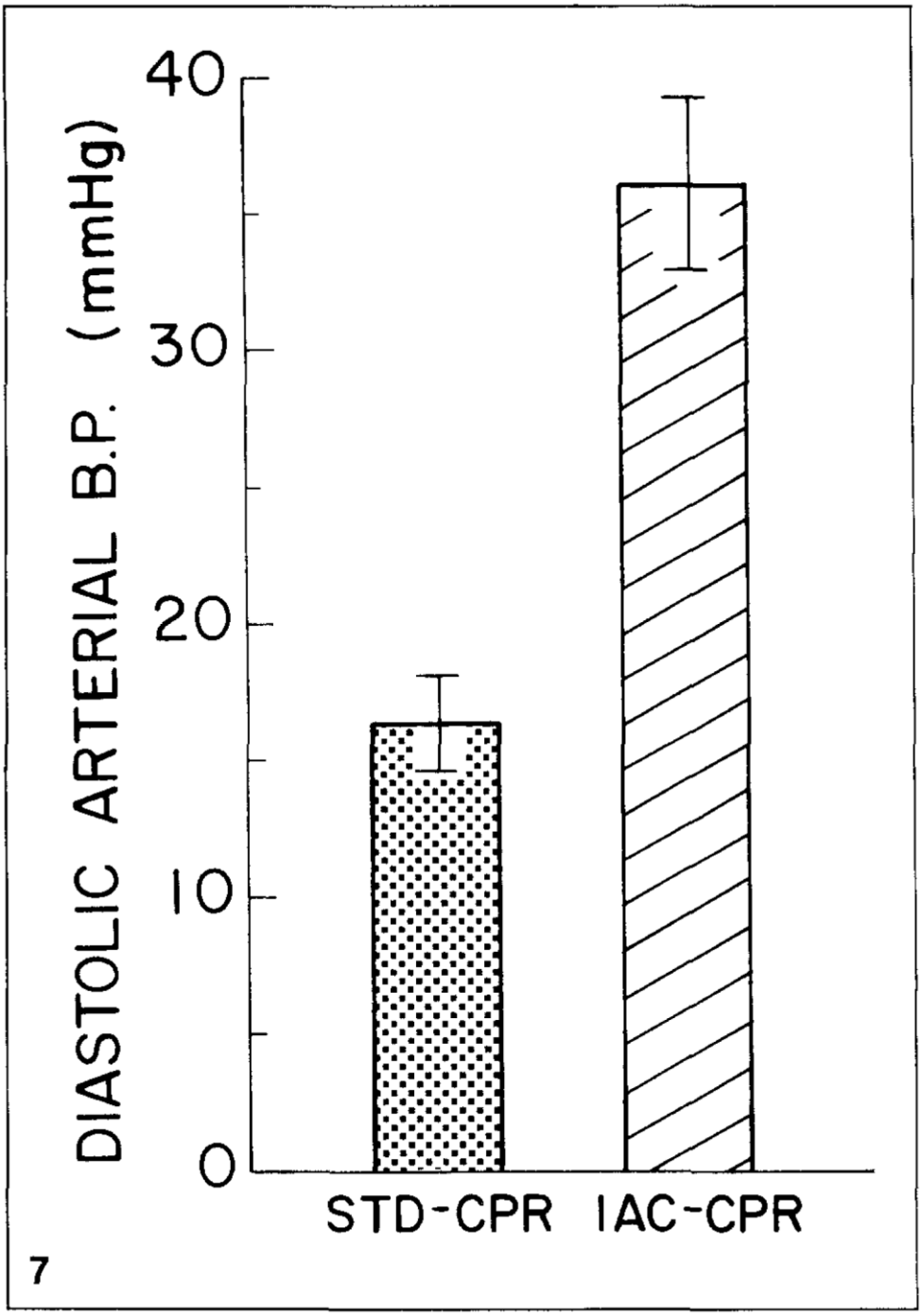

Fig. 7. ***Mean values (±SE) of diastolic arterial blood pressure during standard CPR (STD-CPR) and CPR with interposed abdominal compressions (IAC-CPR) in 10 dogs.***

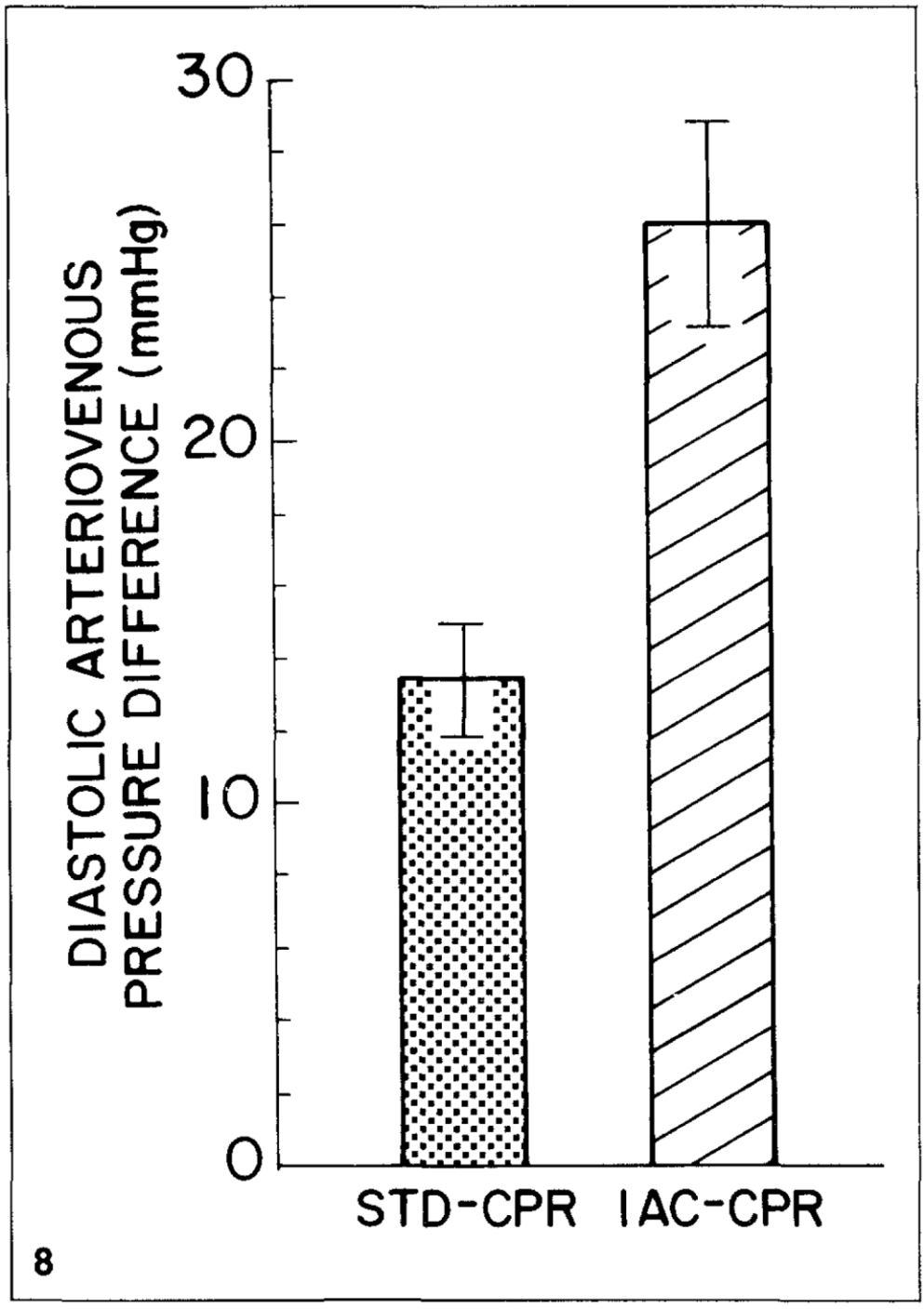

Fig. 8. ***Mean values (± SE) of diastolic arteriovenous blood pressure difference during standard CPR (STD-CPR) and CPR with interposed abdominal compressions (IAC-CPR) in 10 dogs.***

Flow Velocity

A record of relative blood velocity (channel 1, top) measured in the inferior vena cava at the level of the diaphragm during standard CPR and IAC-CPR is shown (Figure 9). The addition of abdominal counterpulsation (indicated by the pulsations in abdominal compression pressure in channel 3, bottom) causes a distinct increase in antegrade flow (channel 1). Both systolic and diastolic brachial artery pressure (channel 2) increase after the onset of IAC-CPR, and a new peak in the arterial pressure occurs coincident with abdominal compression.

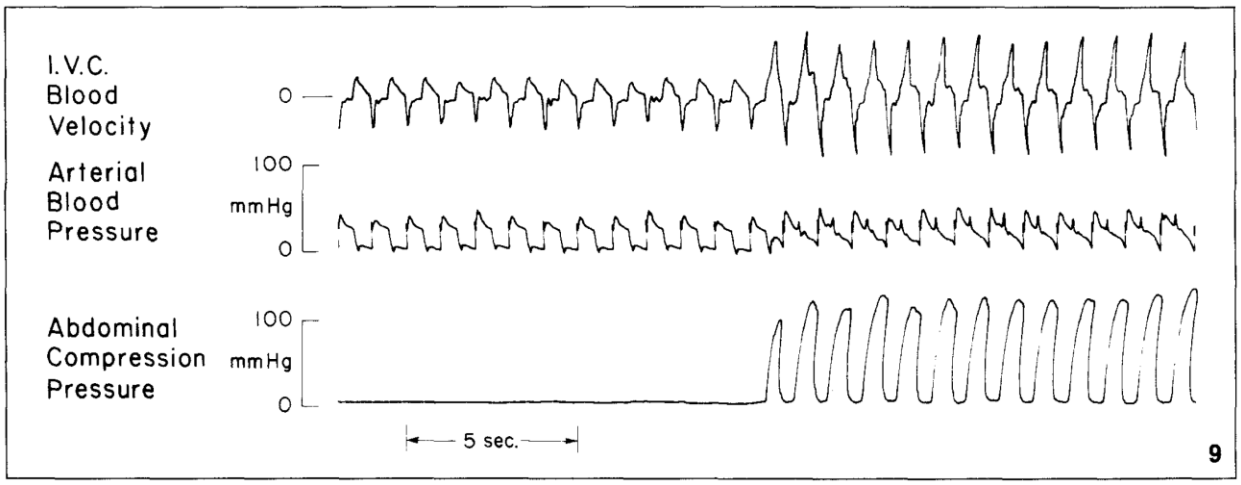

Fig. 9. ***Graphic recording of uncalibrated blood flow velocity in the inferior vena cava and arterial blood pressure during standard CPR (left) and after the onset of IAC-CPR (right).***

Postmortem Findings

No significant gross trauma to intra-abdominal organs was seen at postmortem examination. A small amount of serosanguinous fluid was observed in the abdomen in two of the ten animals, and intramesenteric hemorrhages were observed in one animal. These findings are not considered unusual in heparin-treated animals. Liver laceration never occurred.

DISCUSSION

Oxygen delivery during experimental CPR was measured noninvasively by the closed-circuit system described. In the absence of leaks, changes in spirometer volume are caused by only two major factors: 1) changes in oxygen absorption, and 2) changes in resting expiratory level. The latter may occur during CPR with progressive breakdown of the chest wall due to closed chest massage. However, alternation of experimental and standard CPR techniques in this protocol excludes this mechanism as an explanation for the observed results. Evidently the addition of

interposed abdominal compression to standard CPR significantly improved oxygen delivery, as well as blood pressure and blood flow in the small dog model of cardiopulmonary arrest.

The improvement in oxygen delivery is due to increased cardiac output, for the A-V O_2 difference is not changed. One mechanism appears to be improved pump priming, as indicated by the higher right ventricular pressures and by the augmented antegrade flow velocity in the inferior vena cava (Figure 9). Similar effects of phasic abdominal compression have also been observed while the heart is beating normally by Abel and Waldhausen [5]. It is likely that abdominal compression, like atrial contraction in the normally beating heart, augments blood flow into the main pumping chamber, which during CPR may include the thorax as a whole, the cardiac ventricles, or both [6]. Moreover, diastolic abdominal pressurization must, to some degree, alter the distribution of blood flow, probably favoring the brain and the heart as compared to kidneys, intestines, and lower extremities.

The improvement in thoracic aortic pressure during the diastolic phase (release of chest compression) and diastolic arteriovenous pressure difference by IAC-CPR is significant in that it is likely to enhance coronary perfusion. Coronary flow during standard CPR is reduced at least in proportion to cardiac output [7] (and perhaps even more [8, 9]), but is essential for return of cardiac function and survival [10]. IAC-CPR may offer an especially effective means of increasing coronary flow, both by improving total flow and by favorably altering the distribution of aortic run-off during chest recoil. Harris and associates [11] found that continuous manual compression of the abdomen increased carotid flow by a factor of 2/3, a degree of flow augmentation similar to that in our study. However, they did not recommend manual compression of the upper abdomen during CPR because lacerations of the liver were noted in two of six dogs. In 1971 Redding demonstrated improved carotid artery flow and survival in experimental CPR with continuous abdominal compression by a blood pressure cuff secured around the mid-abdomen [12], while observing no greater incidence of liver damage during CPR with continuous abdominal binding than in similarly resuscitated animals without abdominal binding. Recently Bircher, Safar, and Stewart reported a study of experimental CPR in dogs in which a pressure suit was continuously inflated around the legs and abdomen [13]. They found "no major lacerations of the liver" in 12 dogs receiving this treatment, which did increase arterial pressure and carotid flow at least transiently. Finally, Rosborough and coworkers have reported that synchronous abdominal compression and lung inflation can produce effective artificial cough-CPR in dogs [14] with no evidence of visceral trauma. We believe that the small but significant incidence of liver laceration with continuous abdominal binding is due to entrapment of the liver by the rib cage as the chest is compressed.

During interposed (as opposed to continuous) abdominal compression, however, the liver is allowed to recede at the time the chest is compressed, so that entrapment and laceration of the liver are probably less likely. We have observed such back-and-forth motion of the liver and diaphragm fluoroscopically during IAC-CPR in two dogs, using techniques we have previously described [15]. Although it is certainly possible that excessively rough or vigorous abdominal compression could traumatize the liver or spleen, we believe that central abdominal compression over a large area with 100 to 120 mm Hg pressure is both safe and adequate to augment perfusion.

Abdominal counterpressure during CPR did not induce regurgitation in our study, but it is fitting to speculate on the possibility of provoking regurgitation and aspiration by IAC-CPR. In our animals an endotracheal tube was securely in place, and gastric insufflation did not occur. Gastric insufflation is a common sequela of mouth-to-mouth ventilation in man [16], and abdominal pressure may induce vomiting after the stomach is distended with air [17]. However, if the IAC technique were used consistently from the beginning of resuscitation, gastric insufflation might be prevented entirely by the abdominal counterpressure. In our study abdominal pressure was applied and maintained throughout ventilation, in exact counterpoint to the rhythm of chest compression. It is likely this technique would prevent passage of air into the stomach during mouth-to-mouth rescue breathing in man. The most probable situation in which interposed abdominal compressions might induce regurgitation would be if the technique were added after a period of conventional CPR -- as might occur after others come to the aid of a lone rescuer. Because there are no good animal models for mouth-to-mouth ventilation, this issue will have to be settled by clinical experience.

SUMMARY

The addition of interposed abdominal compression to standard CPR appears to be a simple, safe, and effective means of improving perfusion and oxygen delivery during initial resuscitative efforts, at least in dogs. The technique may be applicable to field CPR by basic rescuers and emergency medical personnel. It requires no extra mechanical equipment and, if proven effective in human trials, could be easily incorporated into existing training programs for lay rescuers and hospital personnel.

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