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Regional Blood Flow during Cardiopulmonary Resuscitation with Abdominal Counterpulsation in Dogs

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ABSTRACT

The addition of abdominal counterpulsation to standard cardiopulmonary resuscitation (IAC-CPR) during ventricular fibrillation has been shown to improve cardiac output, oxygen uptake, and central arterial blood pressure in dogs. The present study was performed to determine the effect of IAC-CPR on regional blood flow. Regional blood flow was measured with radioactively labeled microspheres during sinus rhythm and during alternate periods of IAC-CPR and standard CPR (STD-CPR) in nine dogs anesthetized with pentobarbital. Blood pressures and oxygen uptake were measured continuously. As in previous studies, diastolic arterial pressure was higher (30.8%) during IAC-CPR than during STD-CPR, as were cardiac output (24.5%) and oxygen uptake (37.5%). Whole brain and myocardial blood flow increased 12.0% and 22.7%, respectively, during IAC-CPR. Blood flow to abdominal organs was not changed appreciably in response to abdominal compression, and postmortem examination revealed no gross trauma to the abdominal viscera. The IAC-CPR technique is simple and is easily added to present basic life support procedures. In light of the improvements observed in myocardial and cerebral blood flow, IAC-CPR could significantly improve the outcome of CPR attempts.

Key words: abdomen, brain, heart, hemodynamics, IAC-CPR, perfusion

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INTRODUCTION

Recent studies in our laboratories have shown that the addition of manual abdominal counterpulsation to standard cardiopulmonary resuscitation (CPR) during ventricular fibrillation in dogs significantly improves cardiac output,[1] oxygen uptake,[2] and central arterial blood pressures. Coletti and coworkers[3] have found that manual abdominal counterpulsation, synchronized to the electrocardiogram, also improves blood pressures and coronary artery flow in a canine model of cardiogenic shock.

We have proposed two mechanisms responsible for the effectiveness of abdominal counterpulsation.[1] The first is an apparent increase in venous return, as demonstrated by enhanced antegrade flow velocity in the inferior vena cava during the period of chest release and abdominal compression.[2] The second is compression of the abdominal aorta sufficient to produce retrograde aortic flow and increased central diastolic arterial blood pressure during chest recoil. Such diastolic retrograde flow in the aorta may provide increased blood flow and oxygen delivery to the heart and brain. At the same time, the increase in total flow (cardiac output) during abdominal counterpulsation is associated with increased oxygen consumption. To investigate the differences in regional blood flow between standard CPR (STD-CPR) and CPR with abdominal counterpulsation (IAC-CPR), the following study was performed, radioactive tracer microspheres being used to measure regional blood flow.

MATERIALS AND METHODS

Animal Preparation

Nine dogs weighing 10-21 kg (mean 15.6 kg) were used for this study. The dorsal-ventral chest dimensions, at the level of the heart, ranged from 18 to 28 cm (mean 23 .2 cm), and the chest circumferences ranged from 49 to 63 cm (mean 55.8 cm). Each dog was anesthetized with pentobarbital sodium (30 mg/kg given intravenously) and intubated with a tightly fitting, cuffed endotracheal tube. The dogs were placed in dorsal recumbency on a V-shaped board, and their limbs were secured to prevent lateral motion of the chest during CPR. A pigtail catheter with multiple side-ports near the tip was placed in the left ventricle via the left femoral artery for injection of tracer microspheres. A second catheter was placed in the thoracic aorta just above the level of the diaphragm via the right femoral artery. This catheter was used for withdrawal of reference blood samples for calibrating regional blood flow measurements. A third catheter was inserted 6-8 cm into the left brachial artery for monitoring arterial blood pressure. A final catheter was placed in the thoracic superior vena cava via the left femoral vein for monitoring central venous blood pressure.

Physiologic Monitoring

A six-channel Physiograph direct-inking recorder (Narco BioSystems, Houston, Texas) was used to inscribe a graphic record of the experiment. Oxygen consumption was measured with the method and apparatus we have described previously.[2] Positive-pressure ventilation of the animal was performed by a specially designed closed-circuit system, which incorporated an oxygen-filled spirometer and a CO_2 absorber. The volume of the oxygen in the spirometer was

graphically recorded on channel 1, and the rate of decrease in volume was taken as oxygen consumption. The output of channel 1 was also displayed on a rectilinear strip chart recorder with increased gain and electronic filtering to facilitate measuring the rate of oxygen uptake. Channels 2 and 3 displayed arterial and venous blood pressures, respectively, measured with solid state transducers (Cobe Laboratories, Inc., Lakewood, Colorado). Channel 4 displayed the electrocardiogram (ECG) during sinus rhythm. After electrocardiographic confirmation of ventricular fibrillation, channel 4 was used to record abdominal compression pressure generated in a folded blood pressure cuff placed over the abdomen. All pressure channels were calibrated and their linearity confirmed by use of a mercury manometer. Channel 5 recorded time marks.

Tissue Blood Flow Measured with Tracer Microspheres

Tracer microspheres (New England Nuclear, Boston, Massachusetts) with mean diameters of 15 ± 0.3 μm, bearing three different radioactive labels (^{153}Gd, ^{113}Sn, or ^{46}Sc), were used to measure regional tissue blood flow. Blood flow was measured during sinus rhythm, standard CPR (STD-CPR), and CPR with abdominal counterpulsation (IAC-CPR) by use of a different type of microsphere for each condition according to techniques described by Heymann et al.[4] and adapted by our group for the low-flow conditions of CPR.[5, 6] Three injections of each type of microsphere (containing approximately 5 x 10^5 microspheres per injection) were given under each condition, for a total of about 1.5 x 10^6 of each type of microsphere. Each microsphere injection into the left ventricle was followed by a 10 ml, 0.9% saline flush to clear the catheter. Such repeated injections effectively pooled the blood flow data when the radioactivity of tissue samples was measured, yielding an average flow for the three trials.

The "reference organ" method was used to calibrate blood flow measurements, as follows: An arterial blood sample was collected in a 20 ml syringe by a calibrated withdrawal pump at a rate of 7.3 ml/minute, beginning 10 seconds before a microsphere injection and continuing for a total of approximately 150 seconds. The volume of blood withdrawn was replaced with an equal volume of the same animal's blood, which had been reserved and anticoagulated with heparin. This reserved blood, 150 ml, had been withdrawn 15 minutes after a slow intravenous infusion of 150 ml of 6% dextran 75 in 5% dextrose at the beginning of the experiment. In this way, the animal's hematocrit and blood volume were not cumulatively altered during the experiment by removal of blood samples. Maintenance of nearly constant blood volume during the experiment is important in view of the recent finding by Ditchey and coworkers[7] that coronary artery flow during CPR can be altered greatly by fluid loading.

At the conclusion of the experiment, the following tissue samples were collected: the entire brain, subdivided into left and right cerebral cortices, cerebellum, and medulla; the whole heart, divided as left and right ventricle and atria; both kidneys; both adrenal glands; spleen; and pancreas. Samples were also taken from the duodenal, jejunal, and ileal regions of the small intestine. The radioactivity, in counts per minute (CPM), due to each radionuclide in each tissue sample and in the reference blood samples, was determined by a Beckman 8000 gamma spectrophotometer. Since three reference blood samples were collected for each type of microsphere, the activity in these samples was summed to yield the total reference organ activity in CPM. Average tissue blood flow was then calculated according to this proportion:

$$\frac{\text{tissue blood flow (ml/min)}}{\text{tissue activity (CPM)}} = \frac{\text{reference organ blood flow (7.3 ml/min)}}{\text{total reference organ activity (CPM)}} \quad (1)$$

The blood flow values so obtained were divided by the weight of each tissue, so that blood flow was expressed as ml/min/g of tissue. In a similar manner, total blood flow (cardiac output) at the time of each microsphere injection was calculated from the following proportion:

$$\frac{\text{total blood flow (ml/min)}}{\text{injected activity (CPM)}} = \frac{\text{reference organ blood flow (7.3 ml/min)}}{\text{reference organ activity (CPM)}} \quad (2)$$

Cardiac output calculated in this manner was divided by the body weight of the dog and expressed as ml/min/kg.

Validation of the Microsphere Technique

The microsphere technique for measuring blood flow has several potential sources of error. Under the extremely low flow conditions of CPR, it is important to be aware of, and to minimize, these errors. Possible sources of error include the following: (1) incomplete collection of the reference blood sample, resulting in an overestimate of both cardiac output and regional blood flow; (2) incomplete ejection of the injected microsphere bolus from the left ventricle, resulting in an overestimate of cardiac output, since equation 2 is based on the assumption that all the injected microspheres are distributed during the period of reference sample collection (in this case, determination of tissue blood flow will not be affected provided collection of the reference sample is complete); (3) nonhomogeneous mixing of microspheres with blood, invalidating the proportions assumed in equations 1 and 2 and leading to possible over- or under-estimation of blood flows to different tissues.

To evaluate each of these possible sources of error under the conditions of this experiment, the following procedures were performed. First, during withdrawal of the reference blood samples, the electrical conductivity of the withdrawn blood was continuously monitored by a conductivity cell placed in-line between the right femoral arterial catheter and the withdrawal syringe. Because the conductivities of the suspending agent and the 0.9% saline flush are different from that of whole blood, the passage of these agents through the conductivity cell produces a dilution curve. Blood samples were collected at a constant withdrawal rate until the conductivity returned to baseline after the microsphere injection and the saline flush. Passage of these indicators through the conductivity cell was taken as evidence that microspheres had also passed completely through the cell into the collection syringe.

To determine whether a significant number of microspheres had remained in the left ventricle or aorta after injection, a blood sample was collected by the withdrawal pump during the period of defibrillation countershock and resumption of cardiac activity. If one makes the conservative assumption that total blood flow during this period averaged 1 L/min, then an estimate of the total amount of activity remaining in the left ventricle can be made by rearranging equation 2 for calculating total blood flow. The estimated radioactivity due to microspheres remaining in the left ventricle or aorta never exceeded 1.0% of the total activity injected in any of the nine animals.

Finally, regional blood flows to the right and left cerebral cortices and the right and left kidneys were compared. In all 18 trials of CPR (both STD-CPR and IAC-CPR), right and left cerebral cortical blood flows were not significantly different (Student's paired t- test; $P > 0.05$). In only one of the 18 trials were right and left kidney blood flows significantly different. This occurred only during STD-CPR and may be accounted for by the presence of two catheters in the abdominal aorta that may tend to obstruct either renal artery. Since unequal right and left kidney flows were observed only during STD-CPR, it seems likely that abdominal compression actually tended to prevent the catheters from continually obstructing a renal artery. Hence, no systematic evidence for poor mixing of microspheres with blood was identified.

Experimental Design

After the catheters were inserted, the dogs were attached to the closed-circuit ventilation system for measuring oxygen consumption, and the system was tested for gas leaks, which was easily done by increasing the rate of positive pressure ventilation. In the presence of leaks, this test will cause the apparent oxygen uptake to increase, as volume will be lost more rapidly from the system. This apparent increase in oxygen uptake will not occur in the absence of gas leaks in a physiologically stable preparation. Subsequently, oxygen consumption was monitored continuously throughout the experiment.

During the period of sinus rhythm, three injections of ^{153}Gd-labeled microspheres were given at 5 minute intervals, and corresponding reference organ blood samples were withdrawn for control measurement of tissue blood flow and cardiac output. The subsequent sequence of events is represented schematically in Figure 1.

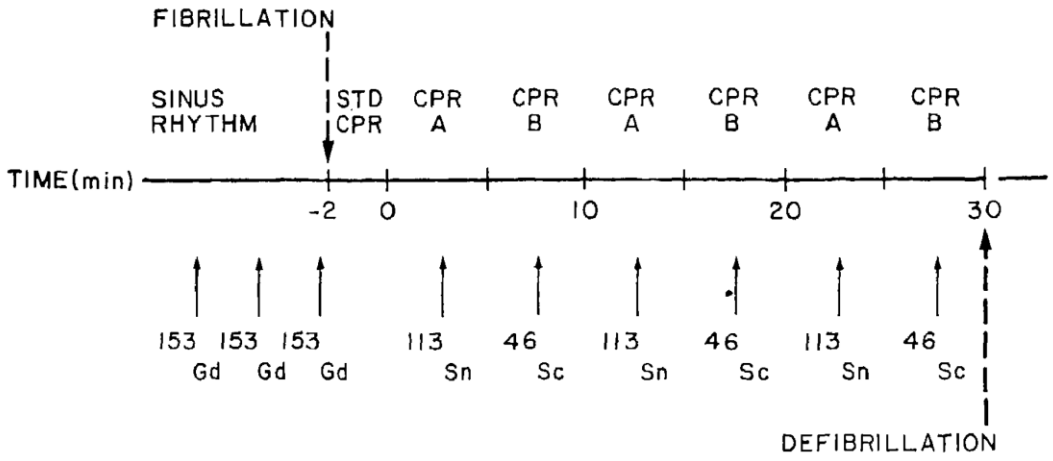

FIGURE 1. Experimental design. Regional blood flow was measured during sinus rhythm and during alternate 5 minute trials of standard CPR and CPR with abdominal counterpulsation. CPR-A and CPR-B indicate that the type of CPR performed was alternated throughout the experiment. Whether STD-CPR or IAC-CPR was performed first was randomly determined.

Ventricular fibrillation was produced by stimulation of the left ventricle with a train of approximately 5 volt, 50 Hz electrical pulses applied via a 0.1 mm stainless steel wire present in the lumen of the left ventricular catheter. Ventricular fibrillation was confirmed electrocardiographically and by the loss of arterial blood pressure. A Thumper® mechanical resuscitator was used to compress the chest and to power the closed-circuit ventilation system. Standard CPR was performed initially in every dog for 2 minutes, during which time the force of chest compression and the ventilation tidal volume were set. The force of chest compression was set to produce a depression of 20% to 25% of the dorsal-ventral chest dimension. The duty cycle of chest compression was 50%. Positive pressure ventilations were interposed after every fifth chest compression. Tidal volume was adjusted to be approximately equal to that observed during the sinus rhythm control period (20-30 ml/kg body weight). These values were not changed for the duration of the experiment.

Abdominal counterpulsation was performed during CPR by manual compression of a standard blood pressure cuff, 12 cm wide, which was folded to 12 x 15 cm, inflated to a thickness of approximately 3 cm, and placed over the mid-abdomen. The cuff was attached to a linear core pressure transducer to monitor pressure applied to the abdomen. The peak pressure used for abdominal compression, measured in the blood pressure cuff, was 100-120 mm Hg. Abdominal compression was performed with a duty cycle complementary to that of chest compression (i.e., 50% of cycle time and 0.5 seconds duration).

For the next 30 minutes, periods of STD-CPR and IAC-CPR were alternated every 5 minutes (Fig. 1). In Figure 1, the terms CPR-A and CPR-B indicate that the two types of CPR were alternated throughout the experiment. The term CPR-A refers to the type of CPR performed first in a given experiment; either STD-CPR or IAC-CPR could be begun first. Whether STD-CPR or IAC-CPR was begun first was determined randomly to prevent biasing the results with an effect of treatment order.

Oxygen uptake and blood pressures were recorded continuously. An injection of ^{113}Sn-labeled micro spheres was given, and a corresponding reference blood sample was withdrawn during each trial of CPR-A. Similarly, an injection of ^{46}Sc-labeled microspheres was given, and a reference blood sample was withdrawn during each CPR-B trial. After the last CPR-B trial, defibrillation countershock was applied. In all experiments cardioversion was achieved. At the end of the experiment the dogs were euthanized, and tissue samples were taken as previously described. A thorough postmortem examination was also performed to identify gross trauma caused by the resuscitation efforts. Special attention was given to identification of possible damage to the abdominal viscera as a result of abdominal compression.

RESULTS

Analysis of Data

Two-tailed paired t-tests were performed on tissue blood flow, oxygen uptake, cardiac output, and blood pressure data. The null hypotheses that population means were equal during STD-CPR and IAC-CPR for each of these data sets were tested, using the $P < 0.05$ level of significance for all tests.

Regional Blood Flow

The addition of abdominal counterpulsation to standard CPR significantly increased cardiac output (24.5%). As observed in previous studies, myocardial blood flow changed nearly in proportion to the change in cardiac output, increasing an average of 22.7% during abdominal counterpulsation. However, because of the variation between animals, this increase was not significant at the $P < 0.05$ level. Whole brain blood flow was increased 12.0% by abdominal counterpulsation, an increase that was significant at $P < 0.05$ (Table 1). The effect of abdominal counterpulsation on blood flow to abdominal viscera was varied, and no significant changes were observed (Table 1).

TABLE 1. Tissue Blood Flow during Sinus Rhythm, Standard Cardiopulmonary Resuscitation (STD-CPR), and Cardiopulmonary Resuscitation with Abdominal Counterpulsation (IAC-CPR)

	Tissue Blood Flow			
Tissue	Sinus Rhythm (ml/min/g)	STD-CPR (ml/min/g)	AC-CPR (ml/min/g)	Difference between AC-CPR and STD-CPR (± SD) (ml/min/g)
Myocardium	1.44	0.22	0.27	0.05 ± 0.08
(range)	(0.84–2.50)	(0.10–0.40)	(0.12–0.46)	
Brain	2.11	0.25	0.28*	0.03 ± 0.03
(range)	(0.57–3.30)	(0.20–0.34)	(0.19–0.34)	
Kidney	5.04	0.69	0.72	0.03 ± 0.20
(range)	(3.76–6.89)	(0.30–1.23)	(0.26–1.11)	
Adrenal glands	4.36	0.56	0.57	0.02 ± 0.10
(range)	(2.36–5.30)	(0.14–1.28)	(0.17–1.20)	
Spleen	2.86	0.10	0.13	0.03 ± 0.05
(range)	(0.59–4.28)	(0.01–0.18)	(0.01–0.31)	
Pancreas	0.30	0.05	0.05	0.004 ± 0.007
(range)	(0.16–0.63)	(0.03–0.07)	(0.04–0.07)	
Small intestine	1.38	0.11	0.12	0.01 ± 0.02
(range)	(0.74–3.13)	(0.05–0.15)	(0.05–0.18)	
Cardiac output (ml/min/kg)	254.3	22.9	28.5*	5.6 ± 4.1
(range)	(128.9–344.2)	(16.8–38.8)	(18.8–40.3)	

* Paired *t*-test significant at $P \leq 0.05$.

Blood Pressures and Oxygen Uptake

All blood pressures (Table 2) were increased significantly by the addition of abdominal counterpulsation to standard CPR. Perhaps most important, in relation to myocardial blood flow, the diastolic arterial pressure increased an average of 30.8% during IAC-CPR. Figure 2 shows the relationship between myocardial blood flow and diastolic arterial blood pressure. In all but one case, an increase in diastolic arterial blood pressure corresponded to an increase in myocardial blood flow.

TABLE 2. Blood Pressures and Oxygen Uptake During Standard Cardiopulmonary Resuscitation (STD-CPR) and Cardiopulmonary Resuscitation with Abdominal Counterpressure (IAC-CPR)

Blood Pressure (mm Hg)	STD-CPR	AC-CPR	Difference ± SD
Systolic arterial	65	75	10 ± 5*
(range)	(37–107)	(50–114)	
Diastolic arterial	26	34	8 ± 6*
(range)	(16–35)	(22–51)	
Mean arterial	45	54	9 ± 6*
(range)	(27–70)	(35–77)	
Diastolic venous	7	12	4 ± 3*
(range)	(3–10)	(4–14)	
Oxygen uptake (ml/min/kg)	3.2	4.4	1.2 ± 0.4*
(range)	(1.7–4.3)	(2.8–6.1)	

* Paired *t*-test significant at $P \leq 0.05$.

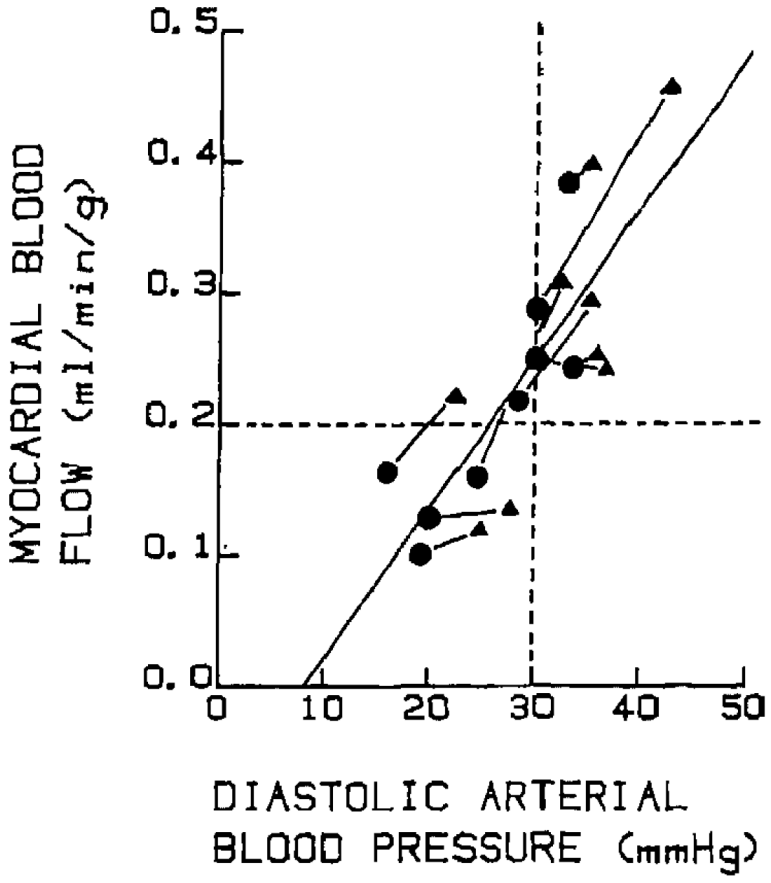

FIGURE 2. Average myocardial blood flow versus central arterial diastolic blood pressure. Circles represent standard CPR; triangles represent CPR augmented by abdominal counterpulsation. Solid lines connect the values obtained for trials of STD-CPR and IAC-CPR in individual animals. Horizontal dashed line indicates the critical level of myocardial blood flow described by Ralston.[11] Vertical dashed line indicates critical level of diastolic blood pressure described by Redding.[9] Linear regression analysis yielded the following: slope = 0.01, Y intercept = -0.09 ml/min/g; correlation coefficient = 0.80.

Abdominal counterpulsation also significantly increased oxygen uptake during CPR (Table 2). Total body oxygen uptake averaged 37.5% higher during IAC-CPR versus STD-CPR. Since blood flow to the heart and brain were increased by abdominal counterpulsation, it seems likely that oxygen delivery to these vital organs was also increased.

Postmortem Examination

Trauma normally associated with chest compression was observed in all nine dogs. This damage consisted primarily of rib fractures. The method used to produce abdominal compression caused no gross trauma to the abdominal viscera. Specifically, liver laceration was never observed.

DISCUSSION

In recent studies,[1, 2] we showed that the addition of abdominal counterpulsation to standard CPR not only improved cardiac output and systemic blood pressures but also increased whole-body oxygen uptake. The improvement observed in central arterial diastolic blood pressure led us to believe that myocardial blood flow, which has been closely correlated with resuscitability and short-term survival[8] should be increased by abdominal counterpulsation. The addition of abdominal counterpulsation to standard CPR increased arterial diastolic pressure an average of 30.8%, which was, in turn, apparently responsible for an increase of 22.7% in myocardial blood flow. Figure 2 shows the close correlation between myocardial blood flow and central arterial diastolic pressure.

Previous studies in our laboratories have shown that return of circulation after prolonged (20 minute) episodes of ventricular fibrillation and CPR in dogs is highly dependent upon the myocardial flow generated by CPR. In particular, Ralston and coworkers[8] have shown that when myocardial flow is greater than 0.2 ml/min/g, circulation is restored in 100% of dogs (18 of 18); while if myocardial flow is less than 0.2 ml/min/ g, circulation is restored in only 33% of dogs (four of 12).

In the present study, five of nine dogs had myocardial flow above 0.2 ml/min/g during STD-CPR; while seven of nine had myocardial flow above this critical level during IAC-CPR (Figure 2). Similarly, the classic work of Redding and Cozine[9] in the dog model has shown excellent chance of return of circulation if diastolic arterial pressure is greater than a critical value of 30 mm Hg during CPR. In the present study, four of nine dogs had diastolic arterial pressures at or above this level when given standard CPR, while six of nine dogs had diastolic arterial pressures above this level with abdominal counterpulsation (Fig. 2).

The addition of abdominal counterpulsation to standard CPR appears to favorably influence hemodynamic variables, which have been shown to be critical to recovery of circulation. Because both types of CPR were performed in each animal, one can only infer that the observed improvement in myocardial perfusion and diastolic arterial pressure with IAC-CPR would improve the ability to resuscitate these animals. This points to the need for outcome studies in which only STD-CPR or IAC-CPR is performed in a given animal.

Because there exist apparent critical threshold values of myocardial perfusion and diastolic arterial blood pressure required for successful initial resuscitation, the relatively modest improvement of hemodynamics reported in this study could have a significant effect upon survival. At the same time, the risk that abdominal counterpulsation will produce serious

abdominal trauma seems slight. No evidence of abdominal visceral trauma attributable to abdominal counterpulsation has been found in over 30 dogs subjected to this procedure.

The results of this study indicate that the addition of abdominal counterpulsation to standard CPR improves blood flow to the brain and myocardium, while having little effect on flow to abdominal viscera. The fact that whole-body oxygen consumption also increased during IAC-CPR suggests that oxygen delivery o the heart and brain was also increased. Since even small increases in cerebral and myocardial perfusion can produce significant improvement in resuscitability and survival after CPR, such a simple technique, which can easily be added to present procedures for basic life support, could have significant impact on the outcome of resuscitation attempts.

While it is true these results were obtained in a canine model of CPR, the basic physical and physiologic mechanisms should also hold for CPR used in human beings. The increased diastolic arterial pressure (apparently caused by effectively cross-clamping the abdominal aorta) and the increased cardiac output (probably due to compression of the abdominal vena cava causing increased venous return) should also occur in the human being. Indeed, Berryman and Phillips[10] have reported an average increase of 47% in mean arterial pressure in six non-resuscitable human subjects in whom IAC-CPR was performed after STD-CPR had failed. If results similar to those reported in this study and in the Berryman human study can be reproduced in a population of resuscitable patients, then IAC-CPR might be accepted as a significant improvement over basic CPR.

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