LABORATORY INVESTIGATION

balloon catheterization;
cardiac arrest;
CPR

Selective Aortic Arch Perfusion During Cardiac Arrest: A New Resuscitation Technique

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The technique described in this manuscript is the subject of a patent application filed in the United States Patent and Trademark Office.

Study objectives: To demonstrate the technique of selective aortic arch perfusion during cardiac arrest and to observe the hemodynamic effects of volume infusion and aortic epinephrine administration.

Design: Sequential series, nonrandomized, noncontrolled.

Type of participants: Fourteen mongrel dogs weighing 21 to 36 kg.

Interventions: Animals had midaortic arch pressure, right atrial pressure, and descending aortic arch balloon occlusion catheters placed. After ten minutes of ventricular fibrillation, balloon inflation and aortic arch infusions were initiated as follows: group 1 (six), 30 mL/kg/min of 0.9% NaCl for two minutes; group 2 (four), 30 mL/kg/min of oxygenated lactated Ringer's with 2 mg/L epinephrine for two minutes, followed by CPR; and group 3 (four), 20 mL/kg/min of oxygenated perfluorochemicals with 4 mg/L epinephrine for one minute, then CPR.

Measurements and main results: Midaortic arch pressure, right atrial pressure, and coronary perfusion pressure each rose significantly in all groups. Midaortic arch pressure and coronary perfusion pressure increases were greater in groups 2 and 3 than in group 1. In groups 1 and 2, right atrial pressure increases at end-selective aortic arch perfusion were excessive as midaortic arch pressure and right atrial pressure increased linearly and similarly after 20 to 30 seconds. In groups 2 and 3, CPR-diastolic midaortic arch pressure and coronary perfusion pressure after selective aortic arch perfusion were good and similar to midaortic arch pressure and coronary perfusion pressure at end-selective aortic arch perfusion.

Conclusion: Selective aortic arch perfusion is technically feasible, but excessive right atrial pressure increases limit maximal infusion rates and volumes. Selective aortic arch perfusion infusates with epinephrine produce greater midaortic arch pressure and coronary perfusion pressure during infusion than infusate without epinephrine. Controlled studies are needed to determine if selective aortic arch perfusion improves resuscitation outcome.

[Manning JE, Murphy CA Jr, Hertz CM, Perretta SG, Mueller RA, Norfleet EA: Selective aortic arch perfusion during cardiac arrest: A new resuscitation technique. *Ann Emerg Med* September 1992;21:1058-1065.]

INTRODUCTION

During closed-chest CPR, blood flow is only a small fraction of normal cardiac output.[1,2] This appears to be especially true if there is a delay of more than a very few minutes before initiating CPR.[3] Epinephrine is given primarily to enhance CPR blood flow by increasing peripheral arterial resistance, which leads to greater aortic pressure and coronary perfusion pressure during the relaxation phase of CPR (CPR-diastole).[4,5] However, delivery of epinephrine to its arterial effector sites is dependent on the artificial circulation of CPR. Thus, in the low-flow state of CPR, not only is vital organ perfusion usually poor, but delivery of beneficial pharmacologic agents to their sites of action is markedly delayed.

Resuscitation drugs have been most commonly administered through a peripheral IV route. However, because of the long drug circulation times observed during CPR, other routes have been sought to provide faster and more effective therapeutic effects. Central venous drug administration provides more rapid delivery of drugs to the arterial system than peripheral venous administration but requires the placement of a central line.[6,7] The intratracheal route is attractive because it does not require any vascular access and delivers drugs at the level of the pulmonary vasculature. However, absorption can be variable, and there is no clear evidence that this route is more effective than the IV routes.[8,9] The intracardiac route, although advocated in the past, has been abandoned.[10] Intra-arterial epinephrine administration was used in laboratory studies by Crile and Dolley[11] in 1906 but appears to have been largely forgotten since then and has never been recommended for clinical use in human cardiac arrest.

Invasive artificial perfusion techniques, including open-chest cardiac massage, direct mechanical ventricular assistance, and cardiopulmonary bypass, clearly provide better vital organ perfusion than closed-chest CPR, but these techniques would be difficult to perform in the prehospital setting.[12-14] The hypothesis that a less invasive technique providing vital organ flow and rapid delivery of pharmacologic agents might improve survival and be adaptable to the prehospital care setting led to the development of a new technique, termed selective aortic arch perfusion.

The purpose of this technique is to isolate the cerebral and coronary circulations for selective perfusion with an oxygenated resuscitation solution containing various agents, such as catecholamines, metabolic substrates, and agents preventing reperfusion injury. Theoretically, selective aortic arch perfusion for a brief time period (eg, one to two minutes) might reverse myocardial ischemia and acidosis, enhance myocardial electrical activity and contractility, restore arterial vasomotor tone, and thereby promote return of spontaneous circulation. Simultaneous perfusion of the brain might improve post-resuscitation neurologic outcome. The placement of an intra-aortic catheter also allows for the delivery of drugs directly into the arterial system and measurement of aortic pressure to assess therapeutic response. Because the infusion technique has never before been studied, the optimal volume, infusion rate, and composition of the solution to be infused into the aortic arch are unknown.

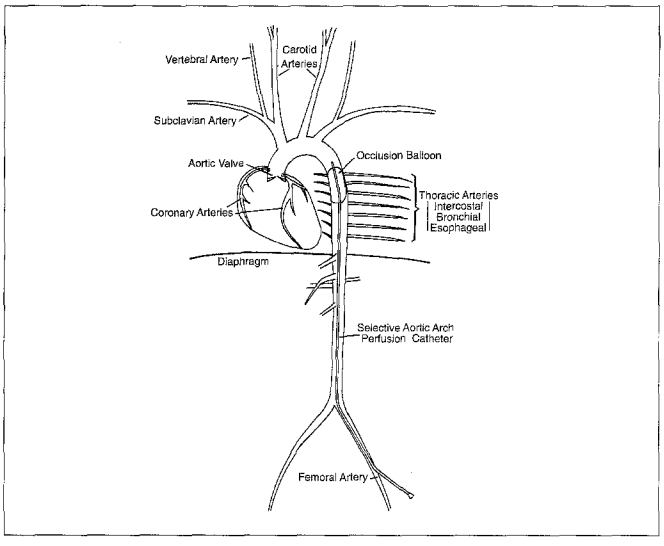
The purpose of this study was to demonstrate selective aortic arch perfusion during canine cardiac arrest using a balloon catheter positioned at the descending aortic arch. Different infusions were used to gain preliminary insight into optimal infusion parameters and resuscitation solution composition.

MATERIALS AND METHODS

Technique Selective aortic arch perfusion involves the insertion of a large-lumen balloon occlusion catheter through a femoral artery into the high thoracic aorta at the end of the descending aortic arch (Figure 1). Inflation of the balloon prevents flow into the distal aorta. A solution then is infused mechanically into the isolated aortic arch, thus providing heart and brain perfusion for a brief time.

Catheter Balloon-tipped catheters 65 to 70 cm in length (55- to 60-cm working length) with 50-mL balloons withstanding 300 mm Hg were created in our laboratory. Flexible plastic oxygen extension tubing (5-mm outer diameter, 3-mm internal diameter) with periodic expansions (10-mm outer diameter) for connector attachment was used for the catheter shell. A segment was cut from the middle of one of the expansions (proximal end) with a 45° bevel cut at the opposite (distal) end. Polyethylene tubing (1-mm outer diameter) was inserted through a hole at the proximal end of the catheter, passed distally in the lumen of the catheter shell, and then

Figure 1.
Positioning of the selective aortic arch persusion balloon occlusion catheter at the end of the descending aortic arch through a femoral artery. Placement of the balloon at this level restricts flow to aortic arch vessels including coronary arteries.



passed back through the catheter wall 2 cm proximal to the distal end. Cement glue and silicone rubber glue were used to seal the two holes in the catheter shell. The internal cross-sectional area was 6.0 to 6.5 mm^2 (equivalent to a 2.7- to 2.8-mm inner diameter catheter).

The catheter balloons were made by dipping a 3-mm diameter glass stirring rod in liquid latex four times at 15-minute intervals to allow drying. The dried latex was cut into 3-cm balloons and rolled onto the distal catheter shells, and the ends were secured with 5-0 prolene suture wrapped multiple times. The catheter shell holes and balloon ends were tested under water to ensure air-tight seals. The balloons were pressurized to 300 mm Hg to ensure adequate strength.

Animal Preparation Mongrel dogs weighing 21 to 36 kg were used. The experimental protocols were approved by the Institutional Animal Care and Use Committee at the University of North Carolina-Chapel Hill School of Medicine. Induction with sodium thiopental 25 to 30 mg/kg was followed by intubation and inhalational ethrane at 1.0 to 1.5 minimum alveolar concentration through a volume ventilator with an FIO_2 of 0.21. Minute ventilation was adjusted to yield an end-tidal CO_2 of 38 to 42 mm Hg, and ECG lead II was monitored.

Right external jugular vein and bilateral femoral vessel cutdowns were performed. Micromanometer-tipped catheters (Millar Instruments, Houston, Texas) were advanced from one femoral artery and vein to the midaortic arch (the area of transition from ascending to descending aortic arch) and right atrium, respectively. These pressure catheters are more resistant to artifact than fluid-filled catheters, and their hard shell prevented any alteration in pressures from the inflation of the selective aortic arch perfusion catheter balloon. The distal tip of the selective aortic arch perfusion catheter was advanced from the other femoral artery to the distal end of the descending aortic arch (below the aortic pressure catheter). A pacing-port Swan-Ganz catheter (American Edwards Laboratories, Houston, Texas) was advanced from the right external jugular vein into a pulmonary artery. A pacing wire was inserted through the pacing-port into the right ventricle. All catheters were positioned using fluoroscopy.

The ECG and pressures were recorded on a multichannel recorder (Model 7 Polygraph, Grass Instruments, Quincy, Massachusetts). In group 1 only, an electromagnetic flow probe (Carolina Medical Electronics, King, North Carolina) was placed on the left common carotid artery.

Preinfusion Phase Baseline measurements of heart rate, aortic arch pressure, right atrial pressure, and left common carotid flow were taken prior to cardiac arrest. Ventricular fibrillation was induced using the pacing wire

Table.
Hemodynamic data including preinfusion pressures, pressures during the infusion (infusion aortic pressure, infusion right atrial pressure, infusion coronary perfusion pressure), postinfusion pressures without CPR in group 1, and CPR-diastolic pressures after infusion (diastolic aortic pressure, diastolic right atrial pressure, diastolic coronary perfusion pressure) for groups 2 and 3

	Preinfusion Phase	Infusion Phase (seconds)						Postinfusion Phase	CPR	
	(0 Seconds)	10	20	30	60	90	120	(60 Seconds)		(60 Seconds)
Group 1										
Infusion midaortic arch pressure	5±4	24±11	26±13*	28±11	35±8*	44±8*	51±9*	23±2		—
Infusion right atrial pressure	3±4	7±5*	12±5*	16±5*	26±7*	36±9*	44±10*	21±5		—
Infusion coronary perfusion pressure	3±3	16±13*	14±14	12±11	9±8	8±7	7±5	2±3		—
Group 2										
Infusion midaortic arch pressure	7±3	28±5	47±7*	60±7*	76±5*	94±7*	110±9*	—	Diastolic midaortic arch pressure	62±4
Infusion right atrial pressure	3±5	7±6*	11±6*	16±7*	32±10*	50±14*	64±15*	—	Diastolic right atrial pressure	26±6
Infusion coronary perfusion pressure	4±7	21±10*	36±12*	44±10*	44±9*	44±8*	46±7*	—	Diastolic coronary perfusion pressure	36±5
Group 3										
Infusion midaortic arch pressure	8±2	27±9*	47±20*	62±22*	80±22*	—	—	—	Diastolic midaortic arch pressure	70±24
Infusion right atrial pressure	5±4	7±4*	11±5*	13±4*	22±5*	—	—	—	Diastolic right atrial pressure	14±5
Infusion coronary perfusion pressure	4±3	20±10*	36±20*	49±22*	58±19*	—	—	—	Diastolic coronary perfusion pressure	56±20

All data in mm Hg (mean ± SD).
* $P < .05$, infusion versus before infusion.

and an AC current generator. After ten minutes of ventricular fibrillation without any resuscitative efforts, the selective aortic arch perfusion catheter balloon was inflated with 40 mL of air (250 to 300 mm Hg) to occlude the aorta and isolate the aortic arch for perfusion.

Infusion Phase At the ten-minute mark of ventricular fibrillation, with the selective aortic arch perfusion catheter balloon inflated, the experimental solution was infused into the aortic arch using a low-pressure perfusion pump (Sarns, Inc, Ann Arbor, Michigan). The Sarns pump is a roller-wheel volume pump that generated pressures in the range of 40 to 150 mm Hg in our experiments, depending on the combined resistance created by the catheter and the aortic pressure generated during the infusion. No other resuscitative efforts (ie, no chest compressions or ventilations) were used during the infusion period to evaluate the hemodynamic effects of the infusion alone.

Three groups of animals were studied sequentially, with each group receiving a different infusion. The changes made in the second and third groups were based on observations made in the previous group. This was done in an effort to determine optimal infusion characteristics. In group 1, six animals (21 to 36 kg) received an infusion of approximately 30 mL/kg/min of 0.9% NaCl (23 to 25 C) over two minutes (1,500 mL for a 25-kg dog]. In group 2, four animals (24 to 26 kg) received approximately 30 mL/kg/min of oxygenated lactated Ringer's (23 to 25 C) containing epinephrine 2 mg/L over two minutes (1,500 mL and 3 mg [0.06 mg/kg/min] of epinephrine for a 25-kg dog). In group 3, four animals (26 to 29 kg) received approximately 20 mL/kg/min of an oxygenated 20% perfluorochemical emulsion (Oxypherol®, Alpha Theapeutic Corporation, Los Angeles, California) containing epinephrine 4 mg/L (23 to 25 C) over one minute (500 mL and 2 mg [0.08 mg/kg/min] of epinephrine for a 25-kg dog).

Postinfusion Phase No resuscitative efforts were used after the infusion in group 1. The midaortic arch pressure and right atrial pressure were simply observed to see how rapidly they would fall. In the first two animals in group 1, 50 mL of IV contrast was injected into the aortic arch during fluoroscopy to confirm balloon occlusion of the aorta. In groups 2 and 3 only, sequential defibrillation attempts at 200 J, 300 J, and 360 J were made over approximately one minute. If unsuccessful, CPR was performed manually by the same individual in each experiment at a compression rate of 120 per minute with a 5:1 compression:ventilation ratio. Ventilation was performed manually using a bag-valve apparatus at an FIO_2 of 1.00. CPR was continued for one minute before pausing to evaluate the ECG pattern.

Further resuscitative efforts followed American Heart Association guidelines for advanced cardiac life support and were continued only until ventricular fibrillation was converted successfully to an organized rhythm.[10] The time required to successfully defibrillate the ECG pattern to an organized ECG rhythm regardless of midaortic arch pressure was recorded. Hemodynamic monitoring was continued to measure right atrial pressure and to determine if systolic midaortic arch pressure reached 60 mm Hg. Vasopressors were not used to increase midaortic arch pressure after ECG rhythm conversion.

Measurements Midaortic arch pressure and right atrial pressure were recorded continuously during each infusion. Because the pressures generated during the infusion were not pulsatile, systolic and diastolic phases were not present. Therefore infusion midaortic arch pressure and infusion right atrial pressure represent mean pressures at the time points studied. Infusion midaortic arch pressure, infusion right atrial pressure, and infusion coronary perfusion pressure (infusion coronary perfusion pressure = infusion midaortic arch pressure – infusion right atrial pressure) were measured at ten-second intervals during

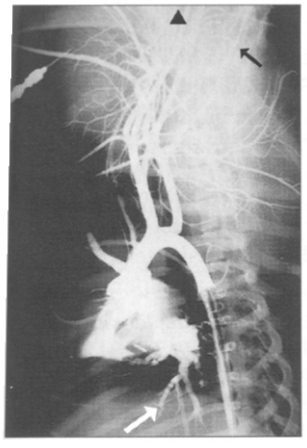
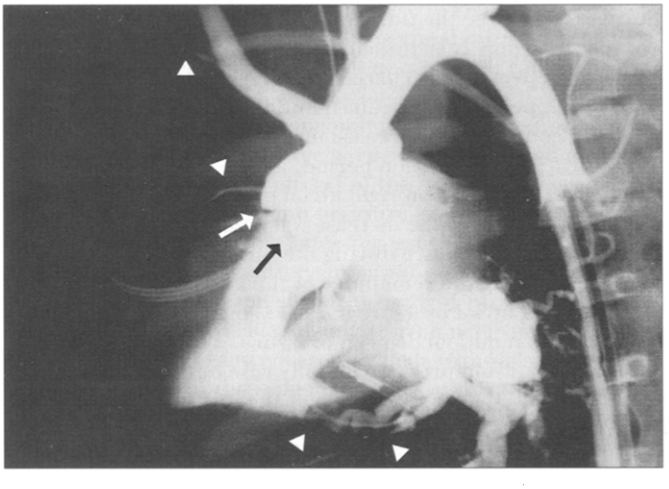


Figure 2.
Radiograph obtained during a subsequent experiment at the end of a two-minute 30-mL/kg/min normal saline infusion as was used in group 1 experiments. The 50-mL contrast injection demonstrates aortic occlusion at the descending aortic arch, flow to the arch vessels, and some retrograde flow into left ventricle, left atrium, and pulmonary veins (white arrow). Internal carotid arteries (arrowhead) and vertebral afteries (black arrow) supplying the brain are well visualized.

Figure 3.
Close-up of the contrast radiograph (Figure 2), which shows two of the aortic valve leaflets (arrows), the coronary vessels (arrowheads), and the regurgitation into the left ventricle and atrium



infusion in all three groups. In group 1, which did not receive CPR, midaortic arch pressure and right atrial pressure were measured one minute after completion of the infusion. During CPR in groups 2 and 3, pulsatile waveforms were generated. CPR-systolic (midaortic arch pressure and right atrial pressure) pressures were measured as the average of the peak pressure of five consecutive compressions. CPR-diastolic midaortic arch pressure and right atrial pressure pressures were measured as the average of the mean diastolic phase pressure of five consecutive compressions. CPR-diastolic coronary perfusion pressure was calculated (diastolic coronary perfusion pressure = diastolic midaortic arch pressure − diastolic right atrial pressure). In group 1, digital left common carotid flow was recorded before arrest and at ten-second intervals during infusion.

Statistical Analysis Infusion pressures (infusion midaortic arch pressure, infusion right atrial pressure, infusion coronary perfusion pressure) at ten, 20, 30, 60, 90, and 120 seconds of infusion were compared with preinfusion, using repeated-measures analysis of variance and paired *t*-test for each group individually. $P < .05$ was considered statistically significant.

RESULTS

Prearrest hemodynamic parameters were similar for all groups. For group 1, 2, and 3, respectively (mean + SD), midaortic arch pressure (mm Hg) was $100 \pm 20/73 \pm 16$, $106 \pm 8/86 \pm 10$, and $117 \pm 9/97 \pm 9$; right atrial pressure (mm Hg) was -2 ± 3, -2 ± 2, and -1 ± 1; and heart rate was 114 ± 13, 123 ± 6, 132 ± 10. The hemodynamic data listed (Table) demonstrate the infusion midaortic arch pressure, infusion right atrial pressure, and infusion coronary perfusion pressure just before and at various time points during the selective aortic arch perfusion infusions. Calculated infusion rates were 30 ± 5 mL/kg/min for group 1, 32 ± 2 mL/kg/min for group 2, and 19 ± 1 mL/kg/min for group 3.

Hemodynamics During Infusion A repeated-measures analysis of variance showed significant trends in the pressures over the time course of the infusion in all groups. In group 1, significant increases in infusion midaortic arch pressure occurred by ten seconds of selective aortic arch perfusion ($P < .05$), and infusion midaortic arch pressure continued to increase linearly throughout the rest of the two-minute infusion period. There was also a significant increase in infusion right atrial pressure by ten seconds, but the magnitude of the increase was smaller than for infusion midaortic arch pressure during the initial ten to 20 seconds. However, during the remainder of the infusion, the rate of infusion right atrial pressure increase slightly exceeded that of the infusion midaortic arch pressure. Thus, infusion coronary perfusion pressure initially rose but then consistently decreased over the course of the infusion. Despite the substantial flow rate used, large infusion coronary perfusion pressure gradients were not generated. Left common carotid flow readings were highly variable but consistently indicated continuous (not pulsatile) forward flow throughout the selective aortic arch perfusion infusion. Individual readings varied from 10% to 67% of prearrest values, with the averages for the group over the course of the infusion in the 38% to 54% range. There was minimal leak past the balloon in the group 1 animals with contrast injection into the aortic arch.

The contrast image (Figures 2 and 3) is from a separate experiment using the same infusion as in group 1, but it demonstrates the successful aortic occlusion that was seen in the group 1 animals during fluoroscopy. The aortic arch vessels, including internal carotid and vertebral arteries and the coronary arteries, are well visualized. The aortic valve is partially competent as demonstrated by the easily identifiable valve leaflets, but with a roughly 750-mL/min infusion rate as used in group 1, there is retrograde flow into the left ventricle, left atrium, and pulmonary venous system. After completion of the selective aortic arch perfusion infusion in group 1, midaortic arch pressure and right atrial pressure rapidly equalized and steadily declined over the next few minutes (Table).

In group 2, the addition of epinephrine resulted in a marked increase in infusion midaortic arch pressure and infusion coronary perfusion pressure. The infusion midaortic arch pressure rose rapidly during the initial 30 seconds of selective aortic arch perfusion and then showed a linear increase throughout the remaining 90 seconds of the infusion. The infusion right atrial pressure showed a slower rise initially as in group 1, but after 30 seconds, infusion right atrial pressure increased linearly and at a rate similar to infusion midaortic arch pressure. Thus, infusion coronary perfusion pressure rose quickly during the initial 30 seconds of selective aortic arch perfusion, but then plateaued while infusion right atrial pressure reached levels much higher than the normal physiologic range.

The selective aortic arch perfusion infusion used in group 3 was modified to achieve increased infusion midaortic arch pressure yet limit excessive infusion right atrial pressure rise, as seen in group 2. This also was our preliminary use of a blood substitute with high oxygen-carrying capacity. The infusion resulted in infusion midaortic arch pressure and infusion coronary perfusion pressure increases that were similar to those achieved during the initial 60 seconds of selective aortic arch perfusion in group 2. Despite the smaller total infusion volume and slower infusion rate, infusion right atrial pressure increases were still substantial after 60 seconds.
CPR hemodynamics after selective aortic arch perfusion.
The Table shows the CPR-diastolic pressures generated after 60 seconds of CPR in groups 2 and 3. The infusion coronary perfusion pressure generated during selective aortic arch perfusion was largely maintained as distolic coronary perfusion pressure with subsequent CPR. The systolic midaortic arch pressure and systolic right atrial pressure values at 60

seconds of CPR were, respectively, 100 ± 3 and 82 ± 11 for group 2 and 116 ± 25 and 98 ± 14 for group 3. Pulmonary edema was noted in both group 2 and 3 animals during CPR.

Conversion to an Organized ECG Rhythm All animals in groups 2 and 3 were defibrillated successfully into organized ECG rhythms, but only after CPR. The average time from the end of infusion to successful rhythm conversion was 5.55 ± 0.74 minutes for group 2 and 4.94 ± 1.38 minutes for group 3. All four group 2 animals and three of four group 3 attained systolic midaortic arch pressure of more than 60 mm Hg without vasopressors. The fourth group 3 animal developed third-degree atrioventricular block and bradycardia, which was not treated. Right atrial pressure immediately after restoration of spontaneous circulation was 7 ± 2 in group 2 and 7 ± 5 mm Hg in group 3. In all animals attaining restoration of spontaneous circulation with systolic midaortic arch pressure of more than 60 mm Hg, the pulmonary congestion seen during CPR appeared to largely dissipate.

DISCUSSION

This study demonstrated the technical feasibility of selective aortic arch perfusion. A balloon catheter placed in the thoracic aorta at the level of the descending aortic arch was used successfully to occlude the aorta and infuse solutions selectively into the aortic arch. The balloon was positioned at this level to limit perfusion solely to the arch vessels and the coronary arteries. Contrast injection during selective aortic arch perfusion demonstrated that this was largely successful with good filling of the internal carotid, vertebral, and coronary arteries. Although the aortic valve was partially competent as evidenced by the visualization of the leaflets, regurgitant flow into the left heart chambers and pulmonary veins was observed. However, it appeared that most of the flow went to the arch vessels as intended.

The consistent increases in infusion midaortic arch pressure seen in all animals of each group during selective aortic arch perfusion demonstrate the ability to pressurize the aortic arch. The observed increases in infusion right atrial pressure during infusion indicate flow from the aortic arch to the venous system and right heart. This right atrial pressure rise probably represents both antegrade flow through the systemic circulation and retrograde flow through the pulmonary vasculature with the relative importance of each being unclear. The brief delay of a few seconds before infusion right atrial pressure rise was thought to be due to the time required for flow to reach the right atrium and the large capacitance characteristic of the venous system.

There appear to be limitations on the total infusion volume, infusion rate, and total infusion time that can be used with this technique. The 30-mL/kg/min infusion used in groups 1 and 2 was determined as a rough estimate of the normal blood flow to the heart (about 5% of cardiac output) and brain (about 15% of cardiac output). We have observed that 25- to 30-kg dogs typically have a cardiac output in the range of 3.5 to 4.0 L/min, and 20% of this is 700 to 800 mL/min or about 30 mL/kg/min. Although not limited solely to the heart and brain, the 30-ml/kg/min infusion was chosen with the expectation of providing flows to these organs that were close to physiologic range. The decision to use a two-minute infusion was arbitrary.

The hemodynamic response in group 1 revealed that despite substantial flow rates the infusion midaortic arch pressure did not reach normal physiologic range over two minutes and the arterial-to-venous gradient (infusion coronary perfusion pressure) was small due to the increase in infusion right atrial pressure. The addition of epinephrine to group 2 resulted in infusion midaortic arch pressure increases well into the normal physiologic range and substantially greater infusion coronary perfusion pressure, but the infusion right atrial pressure increases were still high. The consistent observation of pulmonary edema during subsequent CPR in group 2 appeared to corroborate this concern. Thus, the 30 mL/kg/min (about 750 mL/min) infusion rate and 60 mL/kg total infusion volume (about 1,500 mL) were thought to be too aggressive. Furthermore, it appeared that good infusion midaortic arch pressure and infusion coronary perfusion pressure were attained by about one minute into the two-minute infusion in group 2.

Based on these observations, the infusion rate in group 3 was decreased to 20 mL/kg/min (about 500 mL/min), and the infusion time was limited to one minute. This resulted in good infusion midaortic arch pressure and lower end-infusion infusion right atrial pressure compared with group 2. Pulmonary congestion was still observed during subsequent CPR, although it was subjectively less prominent. It is unclear whether this congestion is primarily due to right heart overload from antegrade flow or the regurgitant flow into the pulmonary vasculature through the left heart. Epinephrine-induced venoconstriction also may have contributed to the right atrial pressure increase and development of pulmonary edema. Optimal infusion rates may prove to be lower, such as 10 to 15 mL/kg/min, if they can be shown to limit the deleterious increase in right heart pressures and pulmonary edema. The optimal infusion time period and maximum volume are still unclear and require further study.

Our data suggest that concomitant administration of a vasoconstrictor with selective aortic arch perfusion may be required to optimize the effects of the infusion. The small infusion coronary perfusion pressure gradients seen in group 1 despite good flow rates suggest that profound vasodilation is present after ten minutes of total circulatory arrest. We were concerned that this might result in considerable shunting of the selective aortic arch perfusion infusate through the dilated microvascular beds without yielding maximal benefit to the ischemic tissues and lead to a more rapid rise in right heart pressures. Epinephrine was added to the group 2 infusion solution in an effort to restore vascu-

lar tone and possibly enhance the efficiency of the infusion. We thought that restoring vascular tone might limit shunting and attenuate the adverse increase in infusion right atrial pressure seen in group 1. A total infusion dosage of 0.12 mg/kg (about 3 mg) was chosen somewhat arbitrarily, although this dosage falls within the range used in recent studies investigating optimal adrenergic therapy during cardiac arrest.[15,16]

The results in group 2 demonstrated restoration of peripheral arterial resistance as expected, but the infusion right atrial pressure rise was not limited as had been hoped. It is unclear if the infusion right atrial pressure increase observed is due to excessive volume infusion, venoconstriction, or both. Also, it appeared that near-maximal hemodynamic benefit was seen after 1 to 2 mg (about 0.04 to 0.08 mg/kg) of epinephrine had been infused. Therefore, in addition to decreasing the infusion rate, infusion time, and total volume for group 3, the epinephrine dosage was reduced to 0.08 mg/kg (about 2 mg). The infusion midaortic arch pressure and infusion coronary perfusion pressure generated over the one-minute infusion in group 3 were comparable with those seen in group 2 while the infusion right atrial pressure was somewhat lower.

Our observations in groups 2 and 3 are consistent with the previously well-described α-adrenergic vasoconstrictor effects of epinephrine during CPR.[17,18] The aortic arch may prove to be a very effective route for the administration of epinephrine or other adrenergic agents. Recent preliminary work in our laboratory supports this hypothesis.[19] If used in combination with the selective aortic arch perfusion technique, adrenergic agents could be administered through the selective aortic arch perfusion catheter either separate from or as a component of the resuscitation solution.

The optimal oxygen-carrying solution to be used during selective aortic arch perfusion is unclear. In groups 1 and 2, crystalloid was used simply to assess the effects of volume infusion. Studies comparing perfluorochemicals, hemoglobin solutions, and other potentially acceptable blood substitutes will be required to determine which is most effective.

In groups 2 and 3, CPR was performed after selective aortic arch perfusion and the compression rate of 120 per minute was chosen arbitrarily based on Maier et al.[20] In both groups, CPR was in a range noted to correspond with restoration of spontaneous circulation.[4,5,21] Successful defibrillation was achieved only after CPR had been performed. This suggests that the brief infusions with the solutions used in this study were not adequate alone to allow successful defibrillation. It may be that the benefits of selective aortic arch perfusion will be to reperfuse the heart and brain initially, rapidly deliver pharmacologic agents, and make subsequent standard resuscitative efforts more effective.

There are several limitations to the data presented in this study. The experiments were not randomized or controlled, and the numbers in each group were small. It is therefore not possible to conclude from the data that selective aortic arch perfusion improves return of spontaneous circulation. Although conceptually the selective aortic arch perfusion technique was developed to improve neurologic recovery as well as promote restoration of spontaneous circulation, effects of selective aortic arch perfusion on neurologic recovery were not addressed in this study. Despite flow rates and pressure gradients that strongly suggest good perfusion, myocardial and cerebral flows were not quantitated. The left common carotid flow readings in group 1 indicated forward carotid flow but are likely quantitatively inaccurate because of changes in vessel size during arrest and hemodilution during infusion. Because of the changes made in the solution composition and the infusion parameters among the three groups, attributing hemodynamic differences to any specific variable is not possible. All the hemodynamic effects observed in this study may be due solely to the epinephrine administered. The value of perfluorochemicals cannot be addressed. These experiments were not undertaken to prove the efficacy of selective aortic arch perfusion but rather to demonstrate this new technique and provide preliminary data that may be helpful in designing controlled outcome studies.

Several aspects of the selective aortic arch perfusion technique and solution characteristics remain unaddressed. The appropriate temperature of the infusate has not been studied. A relatively hypothermic solution might be beneficial for facilitating neurologic recovery but may make defibrillation more difficult. The optimal positioning of the balloon within the thoracic aorta may be at the descending aortic arch or just above the diaphragm. The technique as presently described would largely prevent perfusion to much of the spinal cord during the selective aortic arch perfusion infusion. However, placement of the balloon lower in the thoracic aorta would shift some of the infusate into the thoracic perforating arteries and away from the heart and brain. It is not clear whether chest compressions should be performed during the selective aortic arch perfusion infusion. Compressions might augment flow and limit pulmonary congestion, or they might result in greater aortic incompetence and worsen pulmonary edema. The appropriate inflation pressure of the balloon has not been determined, and inflation of the balloon should probably be with a fluid rather than air.

The potential complications of this technique are several and include pulmonary edema, cerebral edema, aortic dissection, aortic rupture, catheter misplacement during blind insertion, air embolism (from balloon rupture or luminal injection), femoral arterial injury or thrombosis, and wound infection or hematoma. The relative risk of these complications must be weighed against the potential benefit of selective aortic arch perfusion.

Selective aortic arch perfusion involves isolation of the aortic arch with a balloon occlusion catheter, thereby allowing infusion of resuscitation solutions with some selectivity to the heart and brain. Inflation of pressure cuffs on both

upper arms would further limit nonvital organ flow although this was omitted in our study because of canine forelimb anatomy. Placement of the selective aortic arch perfusion catheter during a resuscitation would require either percutaneous or surgical access to a femoral artery. The selective aortic arch perfusion catheter would then be advanced blindly to the thoracic aorta, with the insertion length estimate based on approximation of body size. The time required to prepare the selective aortic arch perfusion solution must be short, and the preparation procedure must be simple. An apparatus allowing for rapid oxygenation will be required, and an infusion pump will most likely be needed. Further studies are needed to define the optimal selective aortic arch perfusion infusion parameters (ie, volume, infusion rate, temperature, etc) and the optimal composition of the selective aortic arch perfusion resuscitation solution to determine if this technique improves myocardial and cerebral viability.

CONCLUSION

Selective aortic arch perfusion is a technique that can be used during cardiac arrest to selectively perfuse the heart and brain. We have demonstrated that selective aortic arch perfusion is technically feasible, but optimal infusion characteristics, optimal resuscitation solution composition, and effect on resuscitation outcome must be established.

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