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Regional Blood Flow During Cardiopulmonary Resuscitation in Dogs

William D. Voorhees, BA; Charles F. Babbs, MD, MS, PhD; Willis A. Tacker, MD, PhD

ABSTRACT

To determine differences in regional blood flow during cardiopulmonary resuscitation (CPR) versus normal cardiac function, we measured regional blood flow to several organs in 19 pentobarbital-anesthetized dogs (6-12 kg). Regional blood flow was measured during sinus rhythm in 5 dogs and during electrically induced ventricular fibrillation with CPR in the other 14 dogs. Regional blood flow and cardiac output were measured using radioactively labelled polystyrene microspheres of $15 \pm 3\mu$ diameter, injected into the left ventricle. Adequacy of microsphere mixing at low cardiac outputs was verified by comparing flow rates to paired organs. Cardiac output was 175 ml/kg/min during sinus rhythm versus 47 ml/kg/min during CPR. Flow to all organs sampled was less during CPR, but the relative decrease varied widely. The ratios of regional blood flow during CPR to regional blood flow during sinus rhythm were 90% for brain, 35% for heart, 15% for kidneys, 17% for adrenal glands, 14% for pancreas, 3% for spleen, and 33% for small intestine. These results provide baseline values for regional blood flow during CPR which can be used

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to evaluate alternative CPR techniques and/or drugs which may improve perfusion of vital organs during CPR.

INTRODUCTION

Adequate perfusion of vital organs is essential for effective cardiopulmonary resuscitation (CPR). Due to high intrathoracic pressures and compression of great vessels, the distribution of blood flow during CPR may be different from that during normal cardiac pumping. Although data for flow in the carotid artery during CPR have been reported^{1,2,3,4}, total flow to the brain or other organs has not been described. We investigated regional blood flow during standard CPR in a canine model using radioactive tracer microspheres.

MATERIALS AND METHODS

Animal preparation

Nineteen mongrel dogs weighing 6-12 kg were used in this study. Regional blood flow was measured during sinus rhythm in 5 dogs and during CPR in the other 14 dogs. Relatively young animals with compliant chests and with dorsal-ventral versus right-left thoracic diameters in a ratio of less than 1.6:1 were selected, since preliminary experiments showed that it is difficult to develop adequate cardiac output in more keel-chested dogs.

After the animals were anesthetized with pentobarbital sodium (30 mg/kg i.v.) and intubated with cuffed endotracheal tubes, catheters were placed

- (1) in the left ventricle, via the right femoral artery, for injection of microspheres,
- (2) in the abdominal aorta, via the left femoral artery, for withdrawal of reference blood samples into a motor-driven syringe during each microsphere injection,
- (3) in the right brachial artery for monitoring arterial blood pressure, and
- (4) in the superior vena cava, via the right maxillary vein, for monitoring central venous blood pressure.

Heparin (1 mg/kg i.v.) was given to retard clot formation in the catheters and to reduce intravascular coagulation during periods of circulatory arrest.

Physiologic monitoring

A Physiograph^R direct-inking recorder (Narco Bio-Systems, Houston, Texas) displayed the electrocardiogram (lead II) and arterial and venous blood pressures. Intraesophageal pressure was also recorded as a monitor of chest compression and ventilation. For this purpose, a 25 cm long X 1 cm diameter soft rubber tube filled with water was placed in the esophagus between the thoracic inlet and the diaphragm and connected to a pressure transducer. To prevent mechanical trauma to the heart and lungs, peak esophageal pressure was not allowed to exceed 130 mmHg.

Induction of fibrillation and CPR

Ventricular fibrillation was produced by 60 Hz electrical stimulation of the left ventricular endocardium via a stainless steel wire (0.1 mm dia.) threaded through the left ventricular catheter. Fibrillation was confirmed by the presence of random fibrillation waves in the ECG and by loss of arterial blood pressure.

Chest compression and ventilation, provided by a specially modified mechanical Thumper^R (Michigan Instruments, Inc., Grand Rapids, Michigan), were initiated immediately upon confirmation of fibrillation. A 6 x 10 cm chest compression pad was centered in the midline with its caudal edge at the level of the xiphi-sternal junction. The pad was covered with a wire mesh which served as one defibrillation electrode. A second, V-shaped, mesh electrode was placed under the shaved back of the animal. Low resistivity electrode gel was applied between the electrodes and the skin surface. The resuscitator was powered by 100% oxygen at 60 psi and delivered an inspired oxygen concentration of approximately 80%. Peak inspiratory pressure was 20 cm H₂O. To model standard CPR technique⁵ as closely as possible, a ventilation:compression ratio of 1:5 with an overall compression rate of 62/min was used. The compression duration (duty cycle) was 50% of cycle time.

After blood pressures during CPR had stabilized (20-30 sec),
~~a 1 ml suspension of microspheres (5 x 10⁵ microspheres/ml 10%~~

~~dextran~~ ^{were} rapidly injected into the left ventricle to determine regional blood flow. After about 150 sec of CPR, the animal was defibrillated by a damped sine wave shock of 20-50 joules via the chest-to-back electrodes.

Microsphere technique

Radioactively labeled polystyrene microspheres (3M Co., Minneapolis, Minnesota) with mean diameter of $15 \pm 3 \mu$ were used to measure regional and total blood flow according to the method of Heymann⁶. Microspheres containing four different gamma-emitting labels (^{141}Ce , ^{85}Sr , ^{95}Nb , ^{46}Sc) were employed. These were suspended in a 10% dextran solution. One ml of well mixed suspension containing approximately 5×10^5 microspheres was injected into the left ventricle and the catheter was flushed with 10 ml of 0.9% saline. A reference blood sample was collected from the abdominal aorta, into a motor-driven syringe, at a rate of 10 ml/min, starting 10 sec before the injection and continuing for a total of 120 sec. This sample provided a known blood flow into a "surrogate organ" for the purpose of calculating regional blood flow in tissue samples.

At the conclusion of each experiment animals were euthanized by an overdose of barbiturate. The following whole organs were sectioned, weighed and placed in plastic counting vials: heart, brain, kidneys, adrenal glands, spleen, and pancreas. Several samples of small intestine were also taken from each dog. The radioactivity of these tissue samples and of the reference

blood samples was measured in a Beckman 8000 gamma-counter (Beckman Instruments, Inc., Fullerton, California). Regional flows were calculated from the count rate (CPM) data according to the relationship:

$$\frac{\text{TISSUE FLOW}}{\text{TISSUE CPM}} = \frac{\text{SURROGATE ORGAN FLOW}}{\text{SURROGATE ORGAN CPM}}$$

Total flow (cardiac output) was calculated from the relationship:

$$\frac{\text{TOTAL FLOW}}{\text{INJECTED CPM}} = \frac{\text{SURROGATE ORGAN FLOW}}{\text{SURROGATE ORGAN CPM}}$$

Statistical analysis

The null hypothesis that mean flow rates to the selected organs during CPR were equal to the mean flow rates during sinus rhythm was tested using the unpaired Student's t-test and a level of significance of P < 0.05.

RESULTS

Twenty trials of CPR were performed in 14 dogs; one each in 8 dogs, and 2 each in 6 dogs. To test the validity of the microsphere technique at low flow rates we calculated separately the blood flows to the right and left cerebral cortices and to the right and left kidneys. If mixing of the microspheres with blood were ^{adequately uniform} ~~adequate~~, members of each pair should receive comparable blood flow per gram of tissue. In 3 of the 20

trials, flows to paired organs were grossly unequal. In each case cardiac output was less than 15 ml/kg/min. For the remaining 17 trials the mean ratio (± 1 S.D.) of right to left organ blood flow per gram of tissue was 1.025 ± 0.033 for the cerebral hemispheres and 0.923 ± 0.075 for the kidneys, indicating good mixing during these trials.

Cardiac output in 5 dogs with normally beating hearts averaged 175.5 ml/kg/min. For the 14 dogs studied during CPR, mean cardiac output was 47.4 ml/kg/min, 27 percent of that in the normal controls. Regional flow to all organs sampled was less during CPR, however the extent of the difference varied over a wide range (Figure 1). Flow to the brain was not significantly different under the two conditions, differing by only 10 percent, while flow to the spleen during CPR was only 3 percent of control.

DISCUSSION

The microsphere technique allows accurate measurement of the distribution of blood flow during CPR, provided total flow exceeds 15 ml/kg/min. Using this technique, we compared measurements of regional blood flow in anesthetized dogs during standard CPR to measurements obtained in anesthetized dogs with normally beating hearts. This comparison revealed that cardiac output during CPR is about one fourth of normal, which is consistent with the results of previous investigators^{7,8,9}. Flow to the selected tissues, however,

varied widely.

The effectiveness of CPR undoubtedly depends upon adequate perfusion of vital organs, specifically the brain and heart. The present study indicates that the brain is preferentially perfused relative to other tissues during CPR. The heart appears to be perfused in proportion to cardiac output despite ^{the} high intrathoracic pressures produced intermittently by chest compression. Preferential perfusion of the brain is consistent with dilation of the cerebral vessels caused by increased carbon dioxide tension of the blood. It is also consistent with the phenomenon of venous-valving proposed by Chandra¹⁰ and associates at Johns Hopkins. According to this concept only a small arteriovenous (A-V) pressure difference is developed between the aorta and the superior vena cava by external chest compression, but a relatively large pressure difference is established between the aorta and the jugular vein, because the jugular vein is collapsed at the thoracic inlet by chest compression. The collapse of jugular veins ^{at the thoracic inlet} ~~in the thorax~~ produces a valve-like action which prevents central venous pressure spikes from being transmitted ^{to the periphery.} ~~into the jugular veins outside the thorax.~~ Figure 2 is a physiological recording during CPR in which the venous catheter was advanced from the jugular vein into the superior vena cava. No large venous pressure spikes are transmitted to the jugular vein, and a large pressure difference between the aorta and the jugular is evident. At the arrow the catheter tip was advanced into the chest. Large venous pressure spikes appear

and the positive A-V pressure difference is abolished. Evidently, collapse of the jugular veins at the thoracic inlet prevents pressure in the central venous system from being transmitted into the jugular veins. The resulting peripheral A-V pressure difference across the brain could account for the relatively good blood flow to the brain which we measured in this animal model. It is important to note that an A-V pressure difference must exist within the thorax at some time during CPR because the coronary arteries are perfused. This A-V pressure difference probably occurs during the "diastolic" component of the mechanical compression cycle.

The results of this study provide baseline values for regional blood flow during CPR in dogs. These values can be used to evaluate the effects of alternative CPR techniques as well as the efficacy of various drugs which may improve perfusion of vital organs during CPR.

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FIGURE LEGENDS

Figure 1. Tissue flows in anesthetized dogs with normally beating hearts and during standard CPR. Brain (BRN), Heart (HRT), Kidneys (KID), Adrenals (ADR), Pancreas (PAN), Spleen (SPL), and Small intestine (SI).

Figure 2. Arterial blood pressure (ABP), venous blood pressure (VBP), simultaneous arteriovenous pressure difference (A-V Δ P), and esophageal pressure (ESOPH PRESS) recorded during a CPR trial. A possible venous-valving mechanism is demonstrated by advancing the venous catheter tip from the jugular vein into the superior vena cava. The force of chest compression remains constant as shown by the esophageal pressure trace.

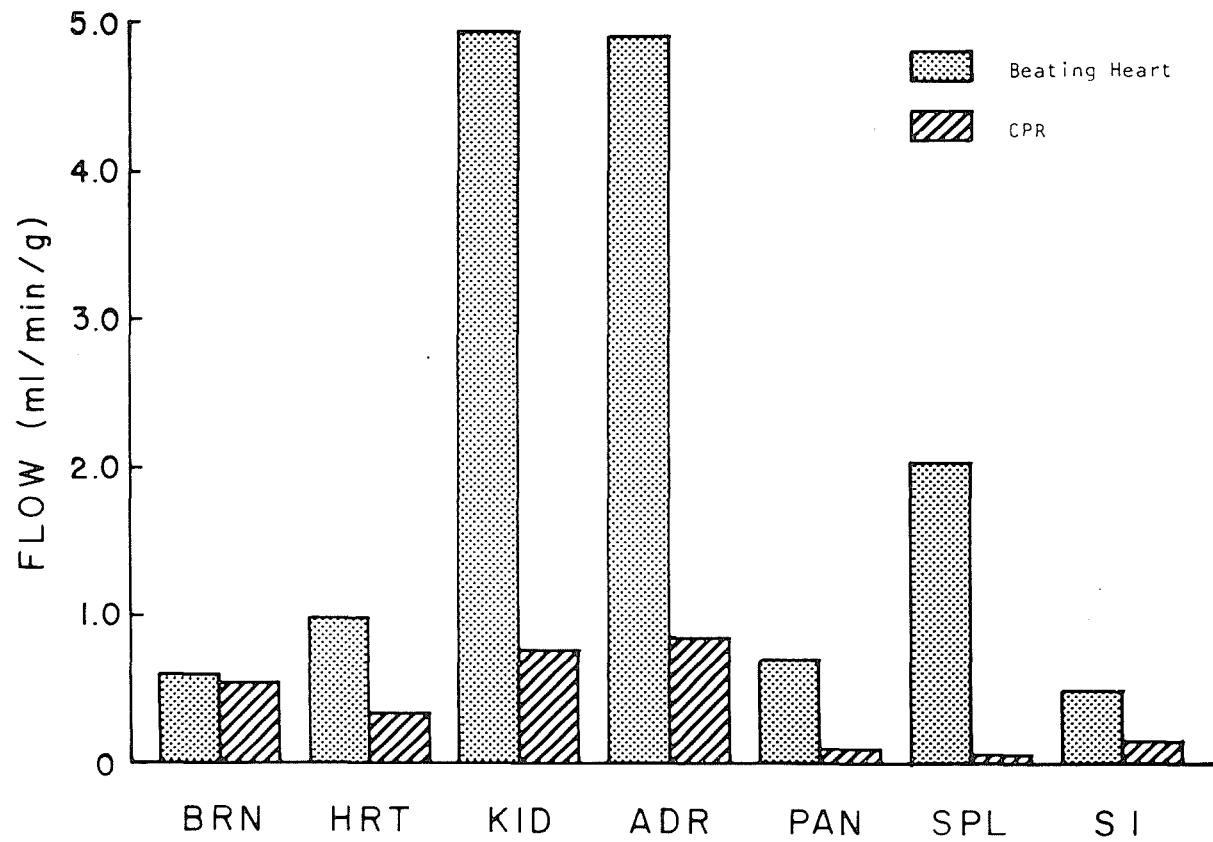
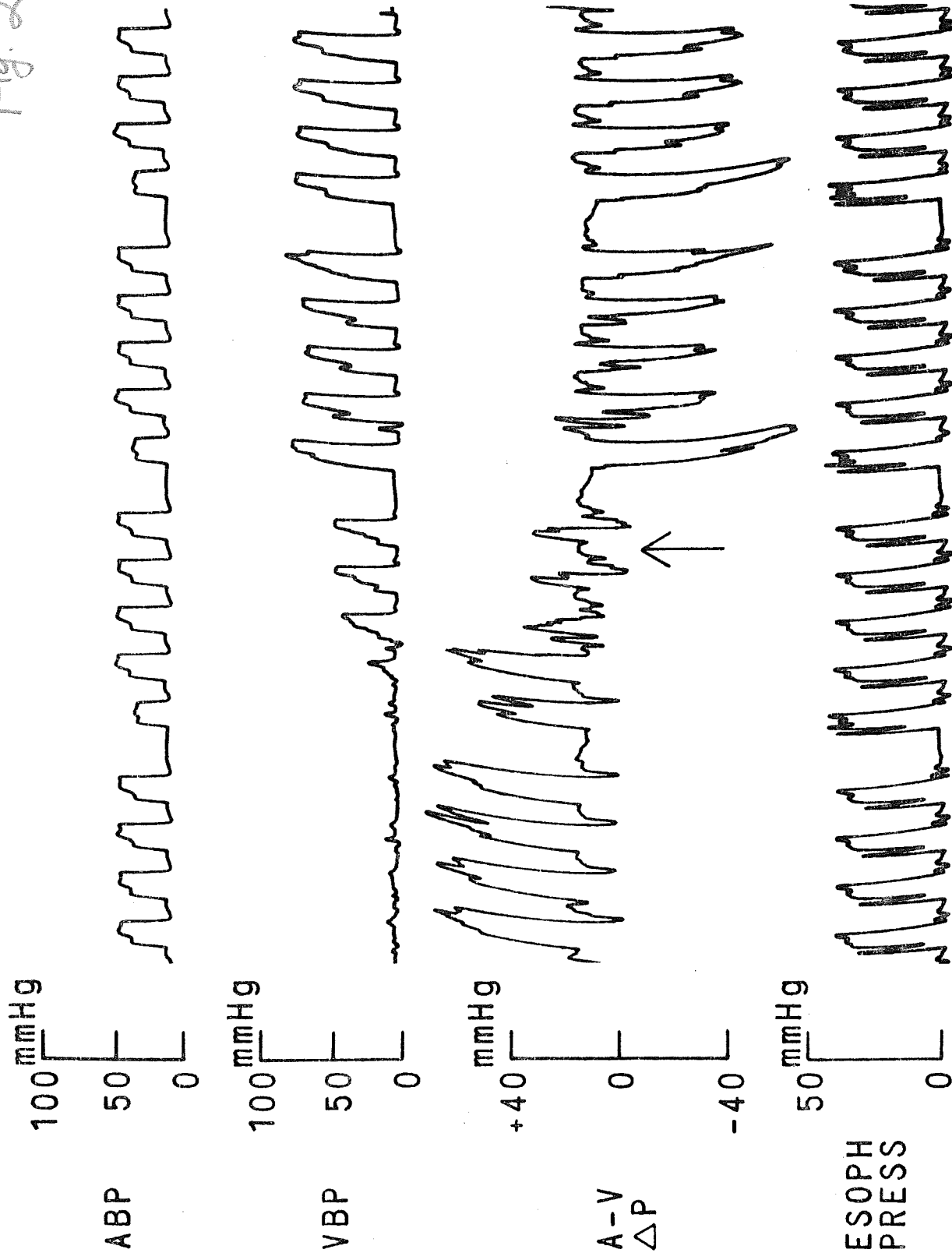


FIG. 1. Tissue flows in anesthetized dogs with normally beating hearts and during standard CPR. Brain (*BRN*), heart (*HRT*), kidneys (*KID*), adrenals (*ADR*), pancreas (*PAN*), spleen (*SPL*), and small intestine (*SI*).

Fig. 2.



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