Hypertension With or Without Hemodilution After Cardiac Arrest in Dogs

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We studied blood flow-promoting therapies after cardiac arrest in 18 dogs. Our model consisted of ventricular fibrillation (no blood flow) lasting 12.5 minutes, controlled reperfusion with cardiopulmonary bypass and defibrillation within 5 minutes, controlled intermittent positive-pressure ventilation to 20 hours, and intensive care to 96 hours. Group I (control, n=6) dogs were reperfused under conditions of normotension (mean arterial blood pressure 100 mm Hg) and normal hematocrit (>35%). Group II (n=6) and III (n=6) dogs were treated with norepinephrine at the beginning of reperfusion to induce hypertension for 4 hours. In addition, group III dogs received hypervolemic hemodilution to a hematocrit of 20% using dextran 40. There were no differences in the time to recovery of electroencephalographic activity among groups. All six group I dogs remained severely disabled; in groups II and III combined, six of the 12 dogs achieved good outcome (p<0.01). Some regional histopathologic damage scores at 96 hours were better in groups II and/or III than in group I (neocortex: p<0.05 group II different from group I; hippocampus: p<0.01 both groups II and III different from group I). Total histopathologic damage scores were similar among the groups. A hypertensive bout with a peak mean arterial blood pressure of ≥200 mm Hg beginning 1–5 minutes after the start of reperfusion was correlated with good outcome (p<0.01). Our results support the use of an initial bout of severe hypertension, but not the use of delayed hemodilution. (Stroke 1990;21:1178–1184)

Cardiac arrest (temporary complete global brain ischemia, no blood flow) and resuscitation are often followed by permanent brain damage.1–3 The degree of damage is influenced by prearrest factors, by the type and duration of the arrest, and, following reperfusion, by the “postresuscitation syndrome,”4 which we hypothesize consists of four interrelated components: 1) perfusion failure, 2) reoxygenation-induced chemical cascades leading to cell necrosis, 3) intoxication from postanoxic viscera, and 4) derangements of blood by stasis. The first component seems to comprise four stages: 1) initial, multifocal absence of reperfusion5–8; 2) transient, reactive global hyperemia9–14; 3) delayed, protracted global10–15 and multifocal12–14 hypoperfusion; and 4) resolution,14 a persistent low-flow state.15

We hypothesize that increasing reperfusion pressure and reducing blood viscosity by hemodilution can improve cerebral blood flow (CBF) and neurologic outcome after cardiac arrest. In 1976, using a dog model of cardiac arrest–cardiopulmonary resuscitation (CPR), we demonstrated that postarrest hypertension (mean arterial blood pressure [MABP] of approximately 150 mm Hg for 4 hours) induced by norepinephrine, immediate intracarotid hemodilution, and heparinization improves outcome.17 Hossmann18 showed that after 1 hour of global cerebral ischemia in cats, high reperfusion pressure correlated with recovery of electroencephalographic (EEG) activity. We recently showed that, after cardiac arrest in dogs, multifocal (local) CBF measured with stable xenon-enhanced computed tomography13 can be improved with hypertension and hemodilution.14 The objective of this study was to determine the effect of these treatments on outcome.

Materials and Methods

This project was approved by the Animal Care and Use Committee of the University of Pittsburgh School of Medicine. We used 22 healthy, custom-bred male hunting dogs (coon hounds) from the same
breeding colony, mean age 10 (range 8–12) months and mean weight 22 (range 18–25) kg.

The dog model includes ventricular fibrillation (VF) cardiac arrest lasting 12.5 minutes, controlled reperfusion by cardiopulmonary bypass (CPB) for ≤5 minutes, controlled intermittent positive-pressure ventilation (IPPV) to 20 hours, and intensive care to 96 hours. The same team conducted all experiments in 1988, in randomized sequence. We studied three groups: group I (n=6) was treated with normotension (MABP of 100 mm Hg) and had a normal hematocrit (Hct of ≥35%), group II (n=7) was treated with hypertension (MABP of >140 mm Hg) and had a normal Hct, and group III (n=9) was treated with hypertension and hemodilution. The duration of VF cardiac arrest was chosen on the basis of previous experiments.20-23 VF lasting 10 minutes occasionally led to good outcome,2,22,23 whereas normothermic VF lasting 12.5 minutes did not.

Briefly, after sedation with 10 mg/kg i.m. ketamine, the dogs were anesthetized for surgical preparation with 50%:50% N2O:O2 plus halothane via face mask until endotracheal intubation. Once intubated, the anesthetic was continued via IPPV, which was adjusted to control end-tidal Paco2 (Paco2) and Paco2. EEG was recorded from two frontal and two occipital scalp clips. Sterile cutdowns were performed for catheterization of the abdominal aorta, pulmonary artery, and vena cava. The superior vena cava (via the right jugular vein) and femoral artery were catheterized for CPB.23

We continuously monitored electrocardiogram, heart rate, MABP, central venous pressure, Paco2, core (pulmonary artery) temperature (Tpa), and EEG. We intermittently monitored arterial and mixed venous Po2, Paco2, pH, and base excess; Hct; blood glucose concentration; serum electrolyte concentrations; and cardiac output. We controlled MABP at 100±10 mm Hg (by adjusting the halothane concentration before and by using norepinephrine or trimethaphan after cardiac arrest), central venous pressure at 5–15 mm Hg, Tpa at 37.5±0.5°C, Paco2 at >100 mm Hg, Paco2 at 30–35 mm Hg, base excess at ±7 meq/l, and blood glucose concentration at 90–175 mg/dl (before arrest). The dogs were hydrated with intravenous Ringer’s solution without glucose and for 20 hours after cardiac arrest.

For the insult, under IPPV and paralysis with pancuronium, N2O and halothane were discontinued and IPPV was continued with 100% O2 for 1 minute, followed by room air for 4 minutes. VF was induced by an external transthoracic electric shock, and IPPV was stopped. (In pilot experiments without paralysis, this procedure did not cause limb movements.) The dogs were kept in VF for 12.5 minutes, then reperfusion was begun (resuscitation time 0 minutes).

Reperfusion was controlled with brief (≤5 minutes) closed-chest CPB by venoarterial pumping via a membrane oxygenator.23 For all dogs, the circuit was primed with 400 ml of 10% dextran 40 in normal saline and Ringer’s solution (50%:50%) plus 1.5 mg/kg heparin (150 units/kg) and 2 meq/kg NaHCO3. Reperfusion was started with CPB at maximal flow (>100 ml/kg/min). O2 flow through the oxygenator was adjusted to control Paco2 and Paco2. IPPV was restarted with 100% O2. At the start of CPB, 0.0125 (group I) or 0.025 (groups III and IV) mg/kg i.a. epinephrine was given, which intensified VF and increased the MABP generated by CPB. When total bypass achieved a MABP of 100 mm Hg, but not later than resuscitation time 3 minutes, an external defibrillating countershock was given at 100 J and repeated as needed at 200, 300, and 400 J. After restoration of a spontaneous heart beat assisted circulation was continued for 2–3 minutes, and all dogs were weaned from CPB by 5 minutes.

In groups II and III hypertension was achieved with 0.025 mg/kg i.a. epinephrine at the start of CPB, more if necessary, to achieve an MABP of 140 mm Hg. In group I, we expected CPB to generate an MABP of 100 mm Hg with the use of 0.0125 mg/kg i.a. epinephrine, more as needed to control MABP during CPB. After defibrillation and restoration of a spontaneous heart beat, epinephrine (which can cause refibrillation and hypermetabolism) was replaced by norepinephrine, given by titrated intravenous infusion, to control MABP at 100 mm Hg in group I and at 140 mm Hg for the first hour, 130 mm Hg for the second hour, 120 mm Hg for the third hour, and 110 mm Hg for the fourth hour in groups II and III. Thereafter, MABP was controlled at 100±10 mm Hg in all groups.

Initially after reperfusion, hemodilution was to be minimal in all three groups, from a Hct of approximately 40% to one of 30–35%, as a result of CPB with the circuit primed with plasma substitute. In groups I and II, at 5 minutes the bypass blood (approximately 15 ml/kg) was reinfused, which kept Hct above 35%. In group III, moderate hypervolemic hemodilution was achieved immediately after the restoration of a spontaneous heart beat (in pilot experiments earlier hemodilution interfered with MABP control) by simultaneously withdrawing 35 ml/kg blood and infusing 50 ml/kg dextran 40 in normal saline; this decreased Hct to 20–25%. Hemodilution was maintained for 2 hours, at which time the shed blood was slowly reinfused. The rationale for the Hct level chosen was adequate compensation with increased cardiac output24 and improved microcirculation.25

Intensive care was the same in all dogs; it included immobilization with pancuronium and IPPV with 100% O2 from 0 to 2 hours and with 50%:50% N2O:O2 from 2 to 20 hours. All dogs received 100 µg fentanyl every 4 hours from 6 to 20 hours to prevent immobilization stress (hypertension, dilated pupils). At 20 hours, the effect of pancuronium was reversed with neostigmine-atropine, and all dogs were weaned to spontaneous breathing by 24 hours.

Early neurologic recovery was evaluated as the time to recovery of the pupillary light reflex and as the times to recovery of any and sustained EEG
FIGURE 1. Mean arterial blood pressure (MAP) after ventricular fibrillation cardiac arrest (VFCA) in 18 dogs. By protocol MAP = 100 mm Hg in group I (n = 6), MAP > 140 mm Hg in group II (n = 6), and MAP > 140 mm Hg plus hemodilution in group III (n = 6). ROSC, restoration of spontaneous heart beat (circulation); BL, baseline. Vertical bars indicate standard deviation.

activity. Outcome was evaluated at 24, 32, 40, 48, 56, 64, 72, 80, 88, and 96 hours as neurologic deficit score (0%: best, 100%: worst) and overall performance category (1: best, 5: worst; categories 1 and 2 indicate good outcome while categories 3, 4, and 5 indicate poor outcome). Best neurologic deficit score and best overall performance category between 24 and 96 hours were recorded separately. No central nervous system depressants were given after 72 hours so as not to influence the final outcome evaluation at 96 hours. All outcome measures were determined by at least two investigators involved in life support. In addition, final outcome at 96 hours was determined by an observer not involved in life support who was unaware of the dog's treatment condition. Final outcome measures were the consensus of at least three observers. Secondary deterioration was determined as a final neurologic deficit score or overall performance category worse than the best values between 24 and 96 hours.

At 96 hours, under endotracheal anesthesia with N2O and halothane, the dogs were killed with a perfusion of paraformaldehyde and total necropsy, brain fixation, and preparation of the specimens were performed. Fifteen brain regions were examined by light microscopy, and the lesions were scored as described previously. A pathologist who did not know the dog's treatment condition evaluated the severity and extent of ischemic neuronal changes, infarcts, and edema. A total histopathologic damage score of approximately 100 reflects severe damage. Morphologic damage of the heart was evaluated by macroscopic quantification of ischemic lesions as a percentage of the total epicardial plus subendocardial surface area.

Data from experiments that did not follow protocol because physiologic variables were outside the prescribed limits (severe or prolonged hypotension, hypertension, acidemia, hypoxemia, hypercarbia, hypothermia, hyperthermia, prearrest hyperglycemia, uremia, and sepsis) and data from dogs that died due to primary extracerebral complications were to be excluded from outcome and histopathologic evaluation; data from dogs that died despite life support according to protocol were to be included. The means and standard deviations of physiologic variables and recovery and outcome measures were calculated and compared between and among the three groups. Continuous physiologic and outcome variables were examined by analysis of variance. Wilcoxon's rank test was used for the nonparametric variables, neurologic deficit scores, overall performance categories, and total and regional histopathologic damage scores. Fisher's exact test was also used for dichotomous comparison of dogs classified as having good or poor outcomes by best overall performance category. We used linear regression analysis to quantify the relation between final neurologic deficit score and early neurologic recovery (time to recovery of pupillary light reflex, time to recovery of sustained EEG activity) and total histopathologic damage score.

Results

We excluded four of the 22 dogs, one in group II (for spontaneous liver torsion and necrosis) and three in group III (one for ventilator failure and two for hypotension during reperfusion). All 18 dogs that followed protocol (six in each group) survived to 96 hours.

Baseline body weight, duration of anesthesia, MABP, central venous pressure, Tpa, blood gas values, Hct, blood glucose concentration, serum electrolyte concentrations, and cardiac output before arrest did not differ significantly among groups. Immediately before arrest, MABP was 111 ± 18, 109 ± 19, and 109 ± 12 mm Hg (Figure 1); Tpa was 37.6 ± 0.3°, 37.5 ± 0.4°, and 37.7 ± 0.4° C; Hct was 40 ± 4%, 40 ± 2%, and 41 ± 2%; and blood glucose concentration was 184 ± 48, 165 ± 43, and 218 ± 48 mg/dl in groups I, II, and III, respectively.
Resuscitation by CPB followed protocol. The number of countershocks (not shown) and time (Figure 1) required to restore spontaneous heart beat were 2.2±1.1 and 2 minutes, 35 seconds; 2.2±1.1 and 2 minutes, 34 seconds; and 1.2±0.4 and 2 minutes, 34 seconds in groups I, II, and III, respectively. At the time of restoration of spontaneous heart beat, MABP was 103±14 mm Hg in group I, 140±32 mm Hg in group II (p<0.01 different from group I), and 163±32 mm Hg in group III (p<0.01 different from group I). In groups II and III, the larger initial dose of epinephrine led to higher spontaneous reperfusion pressures. The amounts of additional epinephrine needed were 0.10±0.08, 0.24±0.10, and 0.35±0.17 mg in groups I, II, and III, respectively. From 5 minutes to 4 hours, MABP remained (nonsignificantly) higher in groups II and III than in group I because in group I MABP was unintentionally above normotension (Figure 1).

At resuscitation time 5 minutes Hct was 32±1%, 30±2%, and 22±4% (p<0.05 different from group I) in groups I, II, and III, respectively. In groups I and II, Hct was 29–43% at 30 minutes and 28–49% from 1 to 12 hours. In group III, Hct was 17–28% from 30 minutes to 2 hours (p<0.01 different from group I or II) and 22–35% from 2 to 12 hours (NS); thus, in group III Hct was <20% only until 2 hours.

Baseline cardiac output was 2.0–4.2 l/min, with no significant differences among groups. Cardiac output increased above baseline at 5 minutes after arrest and remained high until 2 hours. At 1 hour cardiac output was 5.2±1.1, 4.0±1.5, and 7.3±1.5 (p<0.01 different from group I) in groups I, II, and III, respectively.

Tpa remained at 37.5±0.5°C, with no significant differences among groups. At 30 minutes systemic mixed venous Po2 was significantly higher (p<0.01) in group III (82±47 mm Hg) than in groups I and II (51±8 and 69±6 mm Hg, respectively). Beyond 1 hour, however, mixed venous Po2 did not differ significantly among groups. During the first 2 hours, PaO2 was >300 mm Hg with an FiO2 of 100%, with no significant differences among groups. Arterial pH and base excess, as well as NaHCO3 requirements, did not differ significantly among groups. Four of six dogs in each group required only the 2 meq/kg NaHCO3 in the bypass circuit. The incidence of postarrest spontaneous hyperglycemia (in the absence of intravenous glucose) did not differ significantly among groups.

The time to recovery of the pupillary light reflex was 10±5, 14±1, and 14±2 minutes in groups I, II, and III, respectively. EEG activity began to recover at 18±10, 14±2, and 15±9 minutes and was sustained at 36±20, 35±11, and 30±11 minutes in groups I, II, and III, respectively. No dog showed evidence of seizure activity in EEG tracings or clinically. Most comatose dogs had transient opisthotonos and running movements.

Neurologic deficit scores were (nonsignificantly) lower in groups II and III than in group I. Best neurologic deficit scores (usually after 72 hours) were 36±3%, 33±12%, and 30±8% in groups I, II, and III, respectively. Final neurologic deficit scores were 40±5%, 34±13%, and 33±6% in groups I, II, and III, respectively. There was no significant correlation between final neurologic deficit score and time to recovery of the pupillary light reflex (r=0.13) or time to recovery of sustained EEG activity (r=0.23).

The overall performance category (Figure 2) showed severe disability (category 3) or coma (category 4) in all six dogs of group I. Three of six dogs in group II and three of six in group III showed moderate disability (category 2). Best performance categories in groups II and III combined, however, were significantly lower than those in group I.

Total histopathologic damage scores (Figure 3) were 122±20 (range 102–162), 100±19 (range 86–136), and 101±21 (range 80–136) in groups I, II, and III, respectively.

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**Figure 2.** Best overall performance category (BOPC) between 24 and 96 hours after ventricular fibrillation cardiac arrest lasting 12.5 minutes in 18 dogs. Arrows indicate secondary deterioration. Each dot represents one dog. MAP, mean arterial blood pressure; HCT, hematocrit; NS, no significant difference.
tively. Most lesions were ischemic neuronal changes, found predominantly in the neocortex, hippocampus, and cerebellum; there were no infarcts. Total histopathologic damage scores correlated significantly with neurologic deficit scores (r=0.61). Regional histopathologic damage scores (Figure 3) did not differ significantly among groups except in the neocortex and hippocampus. In the neocortex, scores were 12±4 (range 4–16), 10±3 (range 2–14) (p<0.05 different from group I), and 11±5 (range 4–26) (NS) in groups I, II, and III, respectively. In the hippocampus, scores were 14±2 (range 12–16), 8±3 (range 4–12) (p<0.01 different from group I), and 9±2 (range 6–12) (p<0.01 different from group I) in groups I, II, and III, respectively.

Macroscopic myocardial damage scores were 1.9±0%, 1.7±4%, and 1.8 ±2.5% in groups I, II, and III, respectively. Most lesions were in the right ventricular outflow tract.

Considering all three groups, we classified six dogs as having a good outcome (best overall performance category 2) and 11 as having a poor outcome (best overall performance category 3) (Figure 4). The final neurologic deficit scores of the two classes were 25±4% and 36±5%, respectively (p<0.01), and their total histopathologic damage scores were 87±4 and 117±21, respectively (p<0.01).

To determine the best pattern of reperfusion pressure, we compared dogs with MABPs of <100 mm Hg with those having MABPs of 100–150, 150–200, and >200 mm Hg during the reperfusion periods 0–5, 6–10, and 11–15 minutes, respectively, for the good and poor outcome classes. During the first period the good outcome class had a peak MABP of ≥200

![Figure 3](image-url) Total and regional histopathologic damage (HD) scores in brains of 18 dogs 96 hours after ventricular fibrillation cardiac arrest lasting 12.5 minutes. Six dogs in each group.

![Figure 4](image-url) Mean arterial blood pressure (MAP) after ventricular fibrillation cardiac arrest (VFCA) according to best overall performance category (BOPC) (2, good outcome, n=6; 3, poor outcome, n=11) in 17 dogs. ROSC, restoration of spontaneous heart beat; BL, baseline; CPB, cardiopulmonary bypass. BOPC 2 achieved ROSC at time not different from that of BOPC 3. Vertical bars indicate standard deviation.
mm Hg between 1 and 5 minutes in 11 of 30 determinations (37%); the poor outcome class achieved this degree of hypertension in only six of 55 determinations (11%) \( (p < 0.01) \) (Figure 4). After 5 minutes, MABP did not differ significantly between classes.

We also compared dogs with Hcts of <20% with those having Hcts of 20–25%, 25–30%, and >30% during the reperfusion periods 0–30, 31–60, and 61–90 minutes, respectively, for the good and poor outcome classes. There was no significant difference in Hct between classes. Other variables that might influence outcome \( (T_{pa}, P_{ao2}, P_{aco2}, \text{arterial pH, blood glucose concentration, and cardiac output}) \) also did not differ significantly between classes.

**Discussion**

The cardiac arrest model that we used has been well established; it meets the 10 requirements posed by our group for the evaluation of outcome in cerebral resuscitation research.\(^{20,21}\) Our results show a trend toward a neurologic deficit–mitigating effect of hypertension that is induced immediately after cardiac arrest (groups II and III). This trend was significant when groups II and III were combined and compared with group I (Figure 2) and when data from all dogs classified as showing good outcomes were compared with those of dogs showing poor outcomes (Figure 4). Regional histopathologic damage scores (Figure 3), which are generally considered to be more objective end points than functional outcome, were significantly lower in hypertension-treated dogs only in the hippocampus and neocortex. In our study, a brief hypertensive bout with an MABP of ≥200 mm Hg seemed beneficial (Figure 4). The small group differences in outcome might have been in part due to the extreme difficulty of controlling MABP immediately after such a severe no-flow insult; MABP in group I was higher than called for by protocol because investigators hesitated to use trimethaphan during this labile phase.

The rationale for induced hypertension is sound. Capillary compression and increased blood viscosity\(^5\)–\(^7\) might yield to increased reperfusion pressure. Hossmann\(^18\) has shown that an MABP of >140 mm Hg seems important for EEG recovery after 1 hour of global cerebral ischemia in cats. Beneficial effects of hypertensive reperfusion were also found after ischemia in models using small animals.\(^{28}–^{31}\)

The rationale for hemodilution after cardiac arrest is that blood flow is inversely proportional to blood viscosity, which increases in the microcirculation during cerebral ischemia.\(^32\) Hct is the main factor influencing blood viscosity. There is suggestive experimental evidence of a benefit from hemodilution in focal cerebral ischemia.\(^33\) Hemodilution\(^14\) might counteract the late postarrest vasospasm\(^11\) and adhesions of granulocytes,\(^34\) which are possibly initiated by endothelial damage.\(^35\) The initial no-reflow phenomenon\(^8\) seems to be a problem of hypotensive reperfusion\(^6\) and to be avoidable by normotension\(^7\) even without hemodilution.\(^14\)

Moderate hypervolemic hemodilution in group III was not performed instantaneously upon reperfusion (as this would be clinically impossible during CPR), but rather after the restoration of a spontaneous heart beat at resuscitation time ≥3 minutes. Hct was first measured at resuscitation time 5 minutes. Although dogs with good outcome had a lower mean Hct than those with poor outcome, the difference was not significant. Using stable xenon–enhanced computed tomography to monitor multifocal CBF,\(^13\) with the same model and standard therapy, we found protracted multifocal hypoperfusion; postarrest hemodilution (as in this study) normalized CBF.\(^14\) Thus, improving CBF does not necessarily improve outcome. For the entire organism, a Hct of 20–25% was not too low since it resulted in overcompensation by increasing cardiac output, as evidenced by increased venous \( P_{co2} \) values. Our current data are consistent with our previous results\(^17\) in which hypertension combined with hemodilution improved outcome, and we suggest that hypertension is the more important component. Heparin was used in both studies.

In this study, time to recovery of the pupillary reflex and time to recovery of EEG activity did not improve significantly with hypertension or hemodilution, although there was a slight trend of improvement. The incidence of recovery of EEG activity and evoked potentials after 1 hour of global cerebral ischemia in cats was improved by hypertensive reperfusion.\(^16\) This discrepancy might be explained by variable critical opening pressures of microvascular beds.\(^8\)

In studies since 1987, we have learned that a reduction of 2°C in brain temperature before, during, or after VF can mitigate brain damage.\(^2,20,22\) We monitored and accurately controlled \( T_{pa} \), which equaled deep brain temperature in pilot experiments; \( T_{pa} \) can be approximately 1°C higher than cortical epidural temperature.\(^20\) We achieved no significant group differences in \( T_{pa} \) nor was there a difference in \( T_{pa} \) between good and poor outcome classes. Blood glucose levels before arrest\(^36\) were controlled without insulin, by withholding glucose; after arrest, glucose administration was also withheld for 20 hours. Variable spontaneous (moderate) hyperglycemia after arrest was observed, not controlled, since the use of insulin was not considered reasonable.

The clinical use of hypertensive reperfusion is feasible. In this study in healthy dogs there were no deleterious cardiovascular effects of hypertension for 4 hours. High doses of epinephrine, preferably titrated, improve coronary perfusion pressure during external CPR,\(^37\) thereby enhancing the restoration of a spontaneous heart beat and increasing the likelihood of the desired hypertensive bout immediately thereafter. When the hypertensive bout does not occur spontaneously, it can be induced with titrated vasopressor therapy. The optimal duration of induced hypertension in patients remains to be defined.

Our results support the potential benefit of brief, severe hypertension induced during or early after
reperfusion from cardiac arrest, but they do not support the use of moderate hemodilution delayed by approximately 5 minutes after reperfusion. The multifactorial nature of posts ischemic-anoxic encephalopathy necessitates fine-tuning of the optimal MABP and Hct patterns after cardiac arrest with the use of reproducible animal outcome models.

Acknowledgments

H. Alexander and W. Stezoski coordinated the team of technicians and A. Abraham, A. Chandler, A. Pastula, and S. Wertheim helped with intensive care. R. Stone, PhD, helped with statistical analysis. L. Cohn helped with editing the manuscript. G. Foster and F. Mistrick helped prepare the manuscript.

References


Key Words • cardiopulmonary bypass • hemodilution • resuscitation • dogs
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Stroke. 1990;21:1178-1184
doi: 10.1161/01.STR.21.8.1178

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