

1988

Myocardial Perfusion Pressure: A Predictor of 24Hour Survival During Prolonged Cardiac Arrest in Dogs

Karl B. Kern

Gordon A. Ewy

William D. Voorhees

Charles F. Babbs

Purdue University, babbs@purdue.edu

Willis A. Tacker

Follow this and additional works at: <http://docs.lib.purdue.edu/bmepubs>



Part of the [Biomedical Engineering and Bioengineering Commons](#)

Recommended Citation

Kern, Karl B.; Ewy, Gordon A.; Voorhees, William D.; Babbs, Charles F.; and Tacker, Willis A., "Myocardial Perfusion Pressure: A Predictor of 24Hour Survival During Prolonged Cardiac Arrest in Dogs" (1988). *Weldon School of Biomedical Engineering Faculty Publications*. Paper 87.

<http://docs.lib.purdue.edu/bmepubs/87>

MYOCARDIAL PERFUSION PRESSURE: A PREDICTOR OF 24HOUR SURVIVAL DURING PROLONGED CARDIAC ARREST IN DOGS

KARL B. KERN, GORDON A. EWY, WILLIAM D. VOORHEES, CHARLES F. BABBS and WILLIS A. TACKER

University of Arizona College of Medicine (KBK and GAE) and Purdue University Biomedical Engineering Center (WDV, CFB, and WAT)

[Resuscitation, 16 (1988) 241-250]

ABSTRACT

Myocardial perfusion pressure, defined as the aortic diastolic pressure minus the right atrial diastolic pressure, correlates with coronary blood flow during cardiopulmonary resuscitation (CPR) and predicts initial resuscitation success. Whether this hemodynamic parameter can predict 24-h survival is not known. We examined the relationship between myocardial perfusion pressure and 24-h survival in 60 dogs that underwent prolonged (20 min) ventricular fibrillation and CPR. Forty-two (70%) animals were initially resuscitated and 20 (33%) survived for 24 h. Myocardial perfusion pressure was significantly greater when measured at 5, 10, 15 and 20 min of ventricular fibrillation in the resuscitated animals than in the non-resuscitated animals ($P < 0.01$). Likewise, the myocardial perfusion pressure was also greater in the animals that survived 24 h than in animals that were resuscitated, but died before 24 h ($P < 0.02$). Myocardial perfusion pressure measured after 10 min of CPR was 11 ± 2 mmHg in animals never resuscitated, 20 ± 3 mmHg in those resuscitated that died before 24 h and 29 ± 2 mmHg in those that survived 24 h ($P < 0.05$). A myocardial perfusion pressure at 10 min of CPR of 20 mmHg or less is an excellent predictor of poor survival (negative predictive value = 96%). Myocardial perfusion pressure is a useful index of CPR effectiveness and therefore may be a useful guide in helping to optimize resuscitation efforts.

Key words: Cardiopulmonary resuscitation - Myocardial perfusion pressure - Cardiac arrest survival.

INTRODUCTION

The ultimate goal of cardiopulmonary resuscitation (CPR) is long-term survival without neurological deficits. A major problem in modern CPR is evaluating the effectiveness of ongoing resuscitation efforts. Aortic diastolic pressure and myocardial perfusion pressure, defined as the aortic mid-diastolic pressure minus the right atrial mid-diastolic pressure, measured during CPR have been shown to be predictive of defibrillation success and immediate resuscitation [1 - 11]. However, immediate resuscitation results do not always correlate with either long-term survival or neurologic outcome of survivors [2, 12]. Therefore, we analyzed retrospectively pooled data from our CPR research data base containing a series of 24-h survival experiments involving six different methods of external CPR, to determine if myocardial perfusion pressure measured during CPR could predict 24-h survival as well as immediate resuscitation.

MATERIALS AND METHODS

Animal preparations

All animal experiments were performed in accordance with the University of Arizona and Purdue University Animal Welfare Guidelines and according to the American Physiologic Society, Animal Research Guiding Principles. Sixty large mongrel dogs (23.5 ± 0.4 kg body weight) were anesthetized with morphine sulfate 2 mg/kg intravenously, then orally endotracheally intubated and given halothane 1-1.5% in oxygen. Under anesthesia, a 7-Fr. multipurpose catheter was inserted in the right femoral vein and advanced to the right atrium, while a 7-Fr. pigtail catheter with an intraluminal stainless steel wire was inserted via the right femoral artery into the ascending aorta. Subcutaneous leads were attached for electrocardiographic monitoring. The animals were subsequently weaned from anesthesia until the return of corneal reflexes was noted. Baseline hemodynamic pressures including ascending aortic, systolic and diastolic pressure and right atrial systolic, diastolic and mean pressures were obtained prior to full emergence from anesthesia. The aortic catheter was then advanced across the aortic valve into the left ventricle (LV). Before the animal awakened from anesthesia, ventricular fibrillation was induced electrically by passing a 60 Hz current through the wire in the LV catheter. After verifying ventricular fibrillation, the LV catheter was pulled back into the ascending aorta.

Experimental protocol

Following the onset of ventricular fibrillation, no intervention was performed for 3 min. After 3 min, one of six methods of CPR was performed for 17 min including: manual American Heart Association Standard [13], High Impulse Compression [14], Interposed Abdominal Compression [15], Thumper[®] performed American Heart Association Standard [13], Simultaneous Compression and Ventilation [6], or Vest CPR [8]. Each animal received only one form of CPR. All studies were performed under similar conditions in the same laboratory, albeit over a study period extending over two years. Aortic and right atrial pressures were measured at 5, 10, 15 and 20 min of ventricular fibrillation. Pressure recordings were calibrated prior to each measurement.

Myocardial perfusion pressure was calculated from the mid-diastolic aortic pressure minus the simultaneously obtained mid-diastolic right atrial pressure. At 20 min of ventricular fibrillation, electrical defibrillation attempts beginning with 100 J stored energy were performed. The stored energy setting was increased to 200 J and then to a maximum of 300 J, as needed, in an attempt to successfully defibrillate. If defibrillation was unsuccessful or if re-fibrillation occurred, a 10-min period of advanced cardiac life-support was performed. During this time, further CPR, defibrillation attempts (300 J), and use of intravenous epinephrine and atropine, were instituted in an aggressive attempt to resuscitate each animal.

At 30 min following the induction of ventricular fibrillation, CPR efforts were stopped and the aortic blood pressure measured. If the mean aortic blood pressure was greater than or equal to 60% of the mean baseline aortic pressure, the animal was considered “resuscitated.” Resuscitated animals entered an intensive care period for up to 2 h during which time all necessary ventilatory and hemodynamic support was provided with mechanical or manual ventilation and dopamine HCl. During this time all animals but one were successfully extubated. No animal required vasopressor support beyond this 2-h period. Animals were then placed in a maintenance cage for 24 h. Following 24 h, all survivors were euthanized. Necropsies were performed for all animals.

Data analysis

Pressure measurements were taken at mid-systole (compression phase) and at mid-diastole (relaxation phase of chest compression) during CPR. Hemodynamic data were recorded at 5, 10, 15 and 20 min of ventricular fibrillation. Animals were categorized into groups according to outcome: non-resuscitated; resuscitated but surviving less than 24 h; and 24-h survivors. Animals judged to have died from CPR-induced trauma deaths were then identified from the non-surviving groups and the data again analyzed.

Analysis of variance testing was performed to compare potential hemodynamic predictors among the different outcome groups. If a significant difference was found with the analysis of variance, then further testing using Bonferroni’s correction for multiple comparisons was performed. Sensitivity, specificity, positive predictive value, and negative predictive value were calculated in the standard fashion [16] to evaluate (post hoc) the premise that a coronary perfusion pressure (CPP) greater than 20 mmHg, measured at 10 min of CPR, predicts excellent survival. All data are reported as mean \pm S.E.M. A P-value less than 0.05 was considered significant.

RESULTS

Forty-two of 60 animals (70 %) were successfully resuscitated at 30 min. Twenty of 60 (33%) survived for 24 h. There were no differences in baseline hemodynamic values among the different outcome groups (Table I). Throughout the entire CPR episode, myocardial perfusion pressure was significantly greater for those animals resuscitated than for those animals not resuscitated (Table II). Myocardial perfusion pressure was also significantly greater for those animals that survived 24 h compared to those animals that, though resuscitated, survived less

than 24 h (Table III). Aortic diastolic pressure was significantly higher at 10 and 20 min of ventricular fibrillation for animals that survived the full 24 h versus animals that were resuscitated but did not survive 24 h (Table III). There was no significant difference between survivors and resuscitated animals that died prior to 24 h for aortic systolic pressures, right atrial systolic pressures, or right atrial diastolic pressures.

Significant differences in myocardial perfusion pressure, measured at 10 min of CPR, existed among the three distinct outcome groups. Myocardial perfusion pressure was significantly less in non-resuscitated animals than for animals that were resuscitated but died before 24 h and for animals that survived 24 h. Similarly, myocardial perfusion pressure in animals resuscitated that died before 24 h was significantly less than in animals that survived 24 h (Fig. 1). Myocardial perfusion pressure was related to outcome in these three groups. Eight of the 40 non-surviving animals had extensive CPR-induced trauma at necropsy. Three of these animals were never resuscitated and five were initially resuscitated but died within 4 h. After these animals were separated from the others and a fourth group designated, the data were re-analyzed. The mean myocardial perfusion pressure at 10-15 min of CPR for this group was significantly higher than any of the other groups, including the animals that survived 24 h. The mean myocardial perfusion pressure of the animals suffering fatal CPR-induced trauma is compared to the others in Fig. 2. The sensitivity, specificity, positive and negative predictive values of a myocardial perfusion pressure greater than 20 mmHg at 10 min of CPR are shown in Table IV.

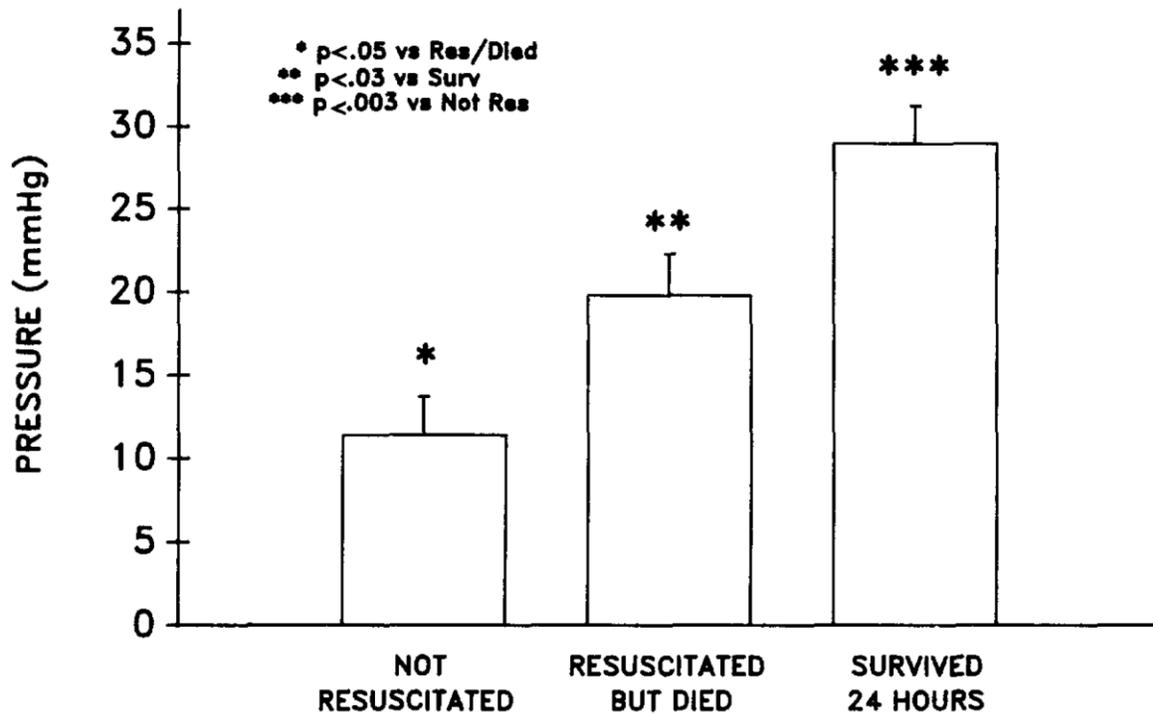


Fig. 1. Mean myocardial perfusion pressures at 10 min of CPR among non-resuscitated animals, animals that were initially resuscitated but died before 24 h, and animals that survived for 24 h.

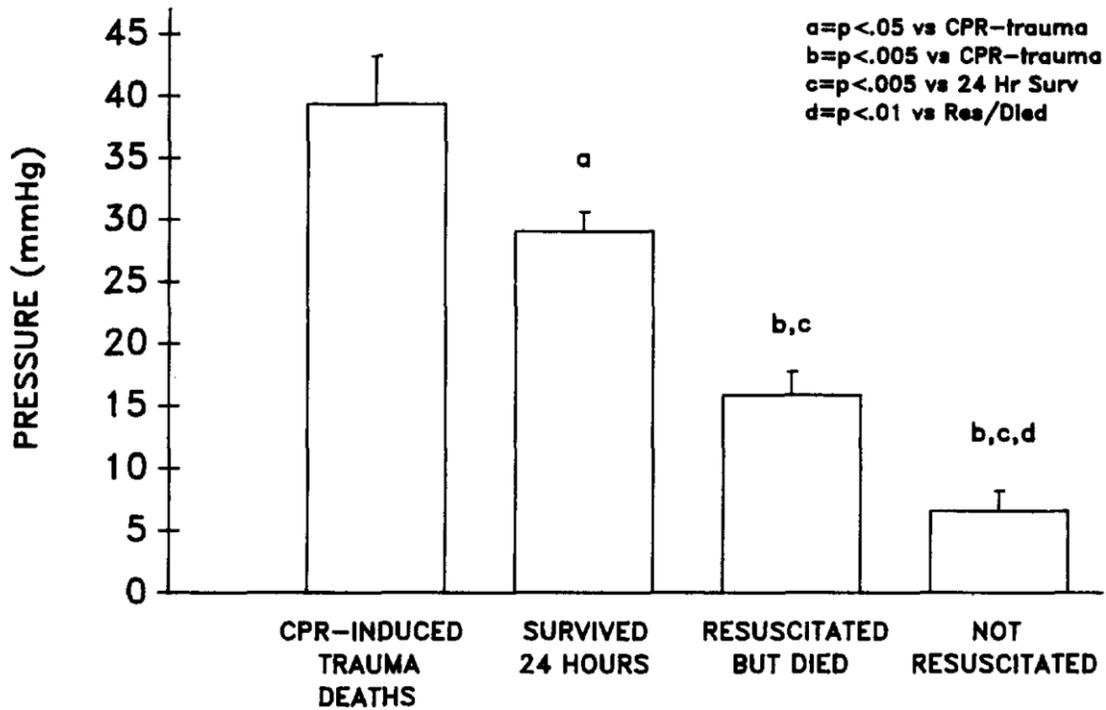


Fig. 2. Mean myocardial perfusion pressures between 10 and 15 min of CPR among animals that suffered CPR-induced trauma deaths, 24-h survivors, animals that were resuscitated but died nontraumatic deaths before 24 h, and animals that were never resuscitated.

TABLE I BASELINE (PRE-ARREST) HEMODYNAMICS AMONG THE DIFFERENT OUTCOME GROUPS

Pressures (mmHg)	Non-resuscitated	Resuscitated but expired	24-h survivors
Aortic			
Systolic	114.2 ± 3.4	113.2 ± 2.6	112.0 ± 2.4
Diastolic	82.0 ± 3.8	76.1 ± 2.7	73.6 ± 2.0
Right atrial			
Systolic	5.3 ± 1.1	3.6 ± 1.2	4.1 ± 0.6
Diastolic	0.7 ± 0.5	0.1 ± 0.1	1.1 ± 0.4
Coronary			
Perfusion pressure	81.0 ± 4.0	75.9 ± 2.8	72.3 ± 2.1

TABLE II HEMODYNAMIC COMPARISON BETWEEN RESUSCITATED AND NON-RESUSCITATED ANIMALS

AOD, aortic diastolic pressure (mmHg); AOS, aortic systolic pressure (mmHg); MPP, myocardial perfusion pressure (mmHg); RAD, right atrial diastolic pressure (mmHg); RAS, right atrial systolic pressure (mmHg).

	Ventricular fibrillation (min)			
	5	10	15	20
<i>Resuscitated (N = 42)</i>				
MPP	23.8 ± 1.6 ^a	24.2 ± 1.7 ^b	24.4 ± 1.9 ^b	19.9 ± 1.7 ^b
AOS	71.3 ± 3.9	79.3 ± 4.6 ^c	78.4 ± 4.0 ^d	76.5 ± 4.3
AOD	32.2 ± 1.9	73.0 ± 1.9 ^e	31.1 ± 2.2 ^e	27.0 ± 1.9 ^e
RAS	73.6 ± 7.6	69.8 ± 4.7	73.2 ± 5.1	70.8 ± 4.9
RAD	8.4 ± 0.9	7.8 ± 0.9	6.7 ± 0.8	7.4 ± 0.9
<i>Non-resuscitated (N = 18)</i>				
MPP	15.5 ± 2.7	10.8 ± 2.4	10.9 ± 3.0	8.6 ± 2.9
AOS	59.4 ± 4.8	61.2 ± 5.4	63.2 ± 6.1	60.1 ± 8.1
AOD	25.8 ± 2.7	20.4 ± 2.5	18.5 ± 3.1	15.3 ± 3.2
RAS	60.1 ± 6.4	65.9 ± 7.7	72.6 ± 8.4	64.7 ± 9.3
RAD	10.2 ± 1.9	9.1 ± 1.6	7.0 ± 1.3	6.3 ± 1.3

^a*P* < 0.01 vs. non-resuscitated.

^b*P* < 0.001 vs. non-resuscitated.

^c*P* < 0.025 vs. non-resuscitated.

^d*P* < 0.05 vs. non-resuscitated.

^e*P* < 0.005 vs. non-resuscitated.

TABLE III HEMODYNAMIC COMPARISON BETWEEN 24-H SURVIVORS AND RESUSCITATED ANIMALS THAT DIED BEFORE 24 H (Abbreviations as in Table II)

	Ventricular fibrillation (min)			
	5	10	15	20
<i>24-h survivors (N = 20)</i>				
MPP	26.5 ± 1.7	29.0 ± 1.6 ^a	29.1 ± 1.7 ^b	25.3 ± 1.5 ^c
AOS	69.7 ± 5.0	77.7 ± 4.5	76.5 ± 4.6	74.2 ± 3.6
AOD	35.0 ± 1.8	36.8 ± 1.9 ^b	35.0 ± 1.9	32.1 ± 1.8 ^b
RAS	63.4 ± 5.6	66.2 ± 6.6	71.1 ± 7.8	68.5 ± 7.9
RAD	8.6 ± 1.3	7.8 ± 1.3	6.0 ± 1.0	7.4 ± 1.3
<i>Resuscitated but expired before 24 h (N = 22)</i>				
MPP	21.4 ± 2.6	19.8 ± 2.6	20.1 ± 3.0	15.0 ± 2.6
AOS	72.8 ± 6.1	80.9 ± 7.7	80.1 ± 6.4	78.5 ± 7.7
AOD	29.7 ± 3.2	27.6 ± 2.9	27.5 ± 3.7	22.3 ± 3.0
RAS	69.3 ± 6.6	73.1 ± 6.8	74.8 ± 7.0	72.8 ± 6.1
RAD	8.3 ± 1.3	7.9 ± 1.3	7.4 ± 1.3	7.4 ± 1.4

^aP < 0.01 vs. Resuscitated but expired.

^bP < 0.02 vs. Resuscitated but expired.

^cP < 0.005 vs. Resuscitated but expired.

TABLE IV MYOCARDIAL PERFUSION PRESSURE > 20 mmHg AT 10 MIN OF CPR: A PREDICTOR OF 24-H SURVIVAL

	CPP < 20 mmHg	CPP > 20 mmHg
Dead at 24 h	$N_1 = 27$	$N_2 = 12$
Alive at 24 h	$N_3 = 1$	$N_4 = 19$
Sensitivity	= True positives/True positives + False negatives = $N_4/(N_4 + N_3)$ = 95%	
Specificity	= True negatives/True negatives + False positives = $N_1/(N_1 + N_2)$ = 69%	
Positive predictive value	= True positives/True positives + False positives = $N_4/(N_4 + N_2)$ = 61%	
Negative predictive value	= True negatives/True negatives + False negatives = $N_1/(N_1 + N_3)$ = 96%	

DISCUSSION

Presently, the best indicator of effective CPR that can be expected to produce successful resuscitation, is myocardial perfusion pressure [1 - 11]. Myocardial perfusion pressure correlates well with myocardial blood flow produced during CPR in experimental models [5, 6, 11]. Both myocardial perfusion pressure and myocardial blood flow correlate with resuscitation success [5, 6, 11]. Myocardial perfusion pressure and aortic diastolic pressure are also significantly greater in animals that are successfully resuscitated versus those not resuscitated after 20 min of CPR [7 - 9]. However, initial resuscitation success does not necessarily translate into long-term survival following cardiac arrest. Redding and Pearson were able to resuscitate 51 of 105 small dogs using external CPR, but only 24 of these 51 (47%) survived 24 h [2]. Likewise in our series of 60 large dogs, only 22 of 42 (52%) initially resuscitated animals survived 24 h. From both of these studies evaluating external CPR, only about 50% of animals successfully defibrillated with the restoration of a perfusing rhythm survived for 24 h. Whether myocardial perfusion pressure can predict 24-h survival, and not just successful defibrillation and restoration of spontaneous blood pressure, has not been previously examined.

Our finding that myocardial perfusion pressure is significantly higher in animals surviving 24 h than in successfully resuscitated animals that die before 24 h, suggests that myocardial perfusion pressure is a useful marker of 24-h survival. The importance of myocardial perfusion pressure in producing successful long-term survival is remarkable considering the many factors that influence 24-h survival post-cardiac arrest: the time before the initiation of CPR after cardiac arrest [17], time between the initiation of CPR and the arrival of definitive cardiac defibrillation [18, 19], total resuscitation time [20], hemodynamic status post-resuscitation [21], CPR trauma [22] and the clinical status prior to cardiac arrest [21].

In this experimental model, using a myocardial perfusion pressure greater than 20 mmHg to define the lower limit of adequate perfusion expected to provide good long-term results, we found that about one-third of those animals identified as probable survivors will not survive (positive predictive value of 61%). The most likely explanation is that some animals with good perfusion pressures suffer CPR-induced injuries that limit their long-term survival. Hence, though almost all survivors will have a myocardial perfusion pressure greater than 20 mmHg (sensitivity of 95%), not all animals with perfusion pressures above 20 mmHg will survive (specificity of 69%). Most importantly, if the myocardial perfusion pressure is less than 20 mmHg the likelihood for a successful long-term outcome is very poor (negative predictive value of 96%).

This information, obtained by arterial and right atrial pressure monitoring during the actual CPR effort, can be used to enhance the chances of success for each individual victim of cardiac arrest. Again within the confines of this cardiac arrest model, if the myocardial perfusion pressure is less than 20 mmHg at 10 min of CPR, then definite efforts should be made to alter the resuscitation therapy. Continuing in the same mode of therapy will almost never result in long-term survival. Changes in the resuscitation therapy can be as simple as increasing the force of compression or altering the site of compression. Other possibilities include increasing the use of

alpha agonists or open chest cardiac massage. The effect of such changes can be monitored by following the same hemodynamic parameter: the myocardial perfusion pressure.

The production of myocardial perfusion pressure with external CPR techniques is not without its problems. In an effort to maximize perfusion the use of excessive force with chest compressions can result in severe and life-threatening injuries. In this animal model of cardiac arrest, a myocardial perfusion pressure of 30 mmHg appears to be ideal for insuring long-term survival without a high degree of CPR-induced injury. Efforts with external chest compressions to raise this perfusion pressure above 30 mmHg do not seem justified because of the resultant CPR-induced trauma. The animals that died from CPR-induced injuries had a mean perfusion pressure of 39 ± 4 mmHg, while the animals that survived 24 h had a mean perfusion pressure of 29 ± 2 mmHg. These results suggest that myocardial perfusion pressure measured during CPR can also be used as a guide to avoid unnecessary injury.

Limitations

Myocardial perfusion pressure in this and other experimental CPR studies was measured in normal experimental animals. Most victims of cardiac arrest have coronary artery disease. Absolute values of myocardial perfusion pressure that produce resuscitation and 24-h survival in the healthy experimental animal free of coronary artery disease may not correlate with the human situation; nevertheless, the pattern of higher myocardial perfusion pressures producing better outcomes seems clear, up to the point where further efforts to improve myocardial perfusion pressure produce excessive trauma. Prospective study of myocardial perfusion pressure in humans to evaluate the optimal range for predicting survival seems indicated.

CONCLUSION

Identification of factors that accurately predict outcome during CPR is important, as alternative, more invasive approaches should be instituted only when it is known that continuing with the same approach will not be successful. Myocardial perfusion pressure can predict not only immediate resuscitation success but also 24-h survival, and can be used during the performance of CPR to guide the rescuer in his efforts to perform optimal CPR. If the myocardial perfusion pressure is less than 20 mmHg after 10 min of resuscitation efforts, then changes in the performance of CPR may be needed to improve successful long-term outcome.

REFERENCES

- 1 J.S. Redding and J.W. Pearson. Evaluation of drugs for cardiac resuscitation, *Anesthesiology*, 24 (1963) 203-207.
- 2 J.S. Redding and J.W. Pearson, Resuscitation from ventricular fibrillation, *J. Am. Med. Assoc.*, 203 (1968) 255-260.
- 3 R.V. Ditchey, J.V. Winkler and C.A. Rhodes, Relative lack of coronary blood flow during closed-chest resuscitation in dogs, *Circulation*, 66 (1982) 297-302.
- 4 A.B. Sanders, G.A. Ewy, C.A. Alferness, T. Taft and M. Zimmerman, Failure of one method of simultaneous chest compression, ventilation, and abdominal binding during CPR, *Crit. Care Med.*, 10 (1982) 569-513.
- 5 S.H. Ralston, W.D. Voorhees and C.F. Babbs, Intrapulmonary epinephrine during prolonged cardiopulmonary resuscitation: improved regional blood flow and resuscitation in dogs, *Ann. Emerg. Med.*, 13 (1984) 70-86.
- 6 J.R. Michael, AD. Guerci, R.C. Koehler, A.Y. Shi, J. Tsitlik, N. Chandra, E. Niedermeyer, M.C. Rogers, R.J. Traystmann and M.L. Weisfeldt, Mechanisms by which epinephrine augments cerebral and myocardial perfusion during cardiopulmonary resuscitation in dogs, *Circulation*, 69 (1984) 822-835.
- 7 A.B. Sanders, G.A. Ewy and T.V. Taft, Prognostic and therapeutic importance of the aortic diastolic pressure in resuscitation from cardiac arrest, *Crit. Care Med.*, 12 (1984) 871-873.
- 8 J.T. Niemann, J.P. Rosborough, R.A. Niskanen C.A. Alferness and J.M. Criley, Mechanical "cough" cardiopulmonary resuscitation during cardiac arrest in dogs, *Am. J. Cardiol.*, 55 (1985) 199-204.
- 9 J.T. Niemann, J.M. Criley, J.P. Rosborough, R.A. Niskanen and C. Alferness, Predictive indices of successful cardiac resuscitation after prolonged arrest and experimental cardiopulmonary resuscitation, *Ann. Emerg. Med.*, 14 (1985) 521-528.
- 10 A.B. Sanders, K.B. Kern, M. Atlas, S. Bragg and G.A. Ewy, Importance of the duration of inadequate coronary perfusion pressure on resuscitation from cardiac arrest, *J. Am. Coll. Cardiol.*, 6 (1985) 113-118.
- 11 H.R. Halperin. J.E. Tsitlik, A.D. Guerci, E.D. Mellits, H.R. Levin, A.Y. Shi, N. Chandra, and M.L. Weisfeldt, Determinants of blood flow to vital organs during cardiopulmonary resuscitation in dogs, *Circulation*, 73 (1986) 539-550.

- 12 K.B. Kern, A.B. Carter, R.L. Showen, W.D. Voorhees, C.F. Babbs, W.A. Tacker and G.A. Ewy, Twenty-four-hour survival in a canine model of cardiac arrest comparing three methods of manual cardiopulmonary resuscitation, *J. Am. Coll. Cardiol.*, 7 (1986) 859-867.
- 13 Standards and guidelines for cardiopulmonary resuscitation and emergency cardiac care (ECC), *J. Am. Med. Assoc.*, 244 (1980) 453-509.
- 14 G.W. Maier, G.S. Tyson, C.O. Olsen, K.H. Kernstein, J.W. Davis, E.H. Conn, D.C. Sabiston and J.S. Rankin, The physiology of external cardiac massage: high-impulse cardiopulmonary resuscitation, *Circulation*, 70 (1984) 86-101.
- 15 S.H. Ralston, C.F. Babbs and M.J. Niebauer. Cardiopulmonary resuscitation with interposed abdominal compression in dogs, *Anesth. Analg.*, 61 (1982) 645-651.
- 16 N.J. Fortuin and J.L. Weiss, Exercise stress testing, *Circulation*, 56 (1977) 699-712.
- 17 MS. Eisenberg, L. Bergner and A.P. Hallstrom, Cardiac resuscitation in the community, *J. Am. Med. Assoc.*, 241 (1979) 1905-1907.
- 18 M.S. Eisenberg, M.K. Copass. A.P. Hallstrom, B. Blake, L. Bergner, F.A. Short and L.A. Cobb, Treatment of out-of-hospital cardiac arrests with rapid defibrillation by emergency medical technicians, *N. Engl. J. Med.*, 302 (1980) 1379-1383.
- 19 K.R. Stults. D.D. Brown, V.L. Schug and J.L. Bean, Prehospital defibrillation performed by emergency medical technicians in rural communities, *N. Engl. J. Med.*, 310 (1984) 219-223.
- 20 R.S. Pionkowski, B.M. Thompson, H.W. Cruchow, C. Aprahamian and J.C. Davin, Resuscitation time in ventricular fibrillation -- a prognostic indicator, *Ann. Emerg. Med.*, 12 (1983) 733-738.
- 21 S.E. Bedell, T.L. Delbanco, E.F. Cook and F.H. Epstein, Survival after cardiopulmonary resuscitation in the hospital, *N. Engl. J. Med.*, 309 (1983) 569-576.
- 22 K.B. Kern, A.B. Carter, R.L. Showen, W.D. Voorhees, C.F. Babbs, W.A. Tacker and G.A. Ewy, CPR induced trauma: comparison of three manual methods in an experimental model, *Ann. Emerg. Med.*, 15 (1986) 674-679.