

WORKSHEET for Evidence-Based Review of Science for Veterinary CPR

1. Basic Demographics

Worksheet author(s)

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Date Submitted for review:

6/18/2011

2. Clinical question:

In dogs and cats with cardiac arrest (P), does the use of any specific rate for external chest compressions (I) compared with standard care (approximately 100/min) (C), improve outcome (ROSC, survival) (O)?

3. Conflict of interest specific to this question:

None

4. Search strategy (including electronic databases searched):

4a. Databases

MEDLINE via PUBMED (1950 to May 2009) (Performed May 2, 2011)

1. Chest Compression rate, CPR
2. External chest compressions
3. Animals
4. human
5. dog, cat

1 : X relevant hits out of 309
1 and 3: X relevant hits out of 120
2 and 3: 0 relevant hits out of 0
2 and 4: X relevant hits out of 26

-CAB (performed May 7, 2011)

1. Chest Compression rate, CPR
2. External chest compressions
3. Animals
4. human
5. dog, cat

No additional hits

4b. Other sources

GOOGLE
No additional hits

4c. State inclusion and exclusion criteria for choosing studies and list number of studies excluded per criterion

Inclusion criteria

Studies evaluating effect of external chest compression rates on CPR outcome
Animal studies, human studies using animal models, mathematical models, manikins

Exclusion criteria

Studies without chest compression evaluation, compression to ventilation studies, interposed or simultaneous abdominal/thoracic compressions, active compression/decompression, review articles, non-English articles, large animal studies

4d. Number of articles/sources meeting criteria for further review: 14

- 7 experimental dog studies (Feneley 1987; Newton 1988; Maier 1984; Fitzgerald 1981; Kern 1986; Halpern 1987; Fleisher 1987)
- 7 clinical prospective observational studies in adult humans (Christenson 2009; Abella 2005; Kern 1992; Fletcher 2008; Kellum 2006; Swenson 1987; Ornato 1988)

5. Summary of evidence

Evidence Supporting Clinical Question

Good			Feneley, 1987; A &E = 24 hour survival Newton, 1988; E=cardiac output, coronary perfusion pressure and brachiocephalic flow Maier, 1984; E=cardiac output Fitzgerald, 1981; E= cardiac output			<i>Christenson 2009; A</i> <i>Abella, 2005; A</i> <i>Kern 1992;</i> <i>E=ETCO₂</i>
Fair						<i>Fletcher, 2008;</i> <i>C</i>
Poor						<i>Kellum, 2006; D</i>
	1	2	3	4	5	6
Level of evidence (P)						

A = Return of spontaneous circulation
 B = Survival of event

C = Survival to hospital discharge
 D = Intact neurological survival

E = Other endpoint
 Italics = Non-target species studies

Evidence Neutral to Clinical question

Good						
Fair			Kern, 1986; A & D Halpern, 1987; E= perfusion Fleisher, 1987; E=cerebral blood flow			<i>Swenson, 1987;</i> <i>E=aortic pressure</i>
Poor						Ornato, 1988; <i>E=PeCO2 and BP</i>
	1	2	3	4	5	6
Level of evidence (P)						

A = Return of spontaneous circulation
 B = Survival of event

C = Survival to hospital discharge
 D = Intact neurological survival

E = Other endpoint
 Italics = Non-target species studies

Evidence Opposing Clinical Question

Good						
Fair						
Poor						
	1	2	3	4	5	6
Level of evidence (P)						

A = Return of spontaneous circulation
B = Survival of event

C = Survival to hospital discharge
D = Intact neurological survival

E = Other endpoint
Italics = Non-target species studies

DRAFT

6. REVIEWER'S FINAL COMMENTS AND ASSESSMENT OF BENEFIT / RISK:

Only one study was found using dogs which specifically measured the return of spontaneous circulation (ROSC) and 24-hour survival (Feneley, 1987) as end-points. A chest compression rate of 120/minute improved resuscitation and 24-hour survival compared to 60/minute rate. This study most closely answered the clinical question posed.

There are a number of variables related to chest compression rate which make studying the effect of this single element on ROSC during CPR difficult to assess. For instance, cardiac output and perfusion are often used as an endpoint in resuscitation studies. Since stroke volume is inversely related to heart rate, those factors which affect stroke volume such as force of compression (Ornato 1988), compression/decompression time and mechanisms of compression (thoracic pump versus cardiac pump, Halpern 1987), must be standardized to evaluate only the effect of rate.

Some authors were able show indirect supporting evidence for survival through improved cardiac output (Maier, 1984; Fitzgerald, 1981) and stable coronary perfusion and cerebral flow with compression rates as high as 150/minute in dogs (Newton, 1988). Other researchers attempted to determine the significance of specific rates by also using other end-points of resuscitation such as coronary perfusion, cerebral perfusion, neurologic outcome or 24-hour survival. More current studies in humans continue to support and correlate increased rates of at least 100/minute with increased survival from cardiac arrest (Abella 2005) and one suggested that rates as high as 150/minute may improve survival (Kellum, 2006). Most recently, human studies have demonstrated that increasing the proportion of time that chest compressions are performed per minute is crucial to survival (Christenson et al, 2009 and Fletcher et al, 2008). Kern et al. found ETCO₂ levels during CPR were higher at 120/minute (positive correlation with ROSC) than at 80/minute.

Several studies were identified that failed to demonstrate any advantage of increased compression rates in CPR. One such study reported no advantage to increasing compression rates from the old guidelines of 60/minute using cerebral blood flow in puppies (Fleisher, 1987). Another study compared rates of 120/minute, 60/minute or 60/minute with interposed abdominal compressions (Kern, 1986) and showed no difference in 24-hour resuscitation among these 3 variables. Halpern, 1987, demonstrated that rate had no effect on vital organ perfusion in dogs weighing 12-32kg receiving external chest compressions and Swenson et al., 1988, found only modest increases in aortic pressure at the 120/minute rate compared to conventional CPR.

There is substantial evidence demonstrating the benefits of faster chest compression rates (100/minute or more) compared to 60/minute. There is also evidence that suggests rates faster than 100/minute would be beneficial but only if the force of compression can be maintained at these more rapid rates. No studies were identified that evaluated the logistics of manually performing effective chest compressions at faster rates in dogs or cats. No specific rate for external chest compressions in dogs and cats, other than the current guidelines of 100/minute can be recommended at this time.

7. Conclusion

"DISCLAIMER: Potential possible wording for a Consensus on Science Statement. Final wording will differ due to other input and discussion."

Although no original veterinary studies were found evaluating the benefit of specific chest compression rates, a number of human studies were identified using dogs as research models. One randomized, controlled study specifically studying varying rates in dogs and using return of spontaneous circulation and 24-hour survival demonstrated that compression rates of 120/minute were beneficial compared to lower rates (Feneley, 1987, LOE3). In conjunction with the more recent human CPR studies, (Christenson 2009, Fletcher 2008, Abella,

2005 and Kellum, 2006) rates of at least 120/minute correlate with a higher survival rate. Further controlled veterinary studies in dogs designed to standardize body size, age and compressions strength would be required to demonstrate if rates higher than 100 to 120/minute would be beneficial (Newton,1988). No studies were found indicating that current recommendations of 100-120 chest compressions per minute decreased chance of survival.

8. Acknowledgement

None

9. Citation list

Influence of compression rate on initial success of resuscitation and 24 hour survival after prolonged manual cardiopulmonary resuscitation in dogs.

Feneley MP, Maier GW, Kern KB, Gaynor JW, Gall SA Jr, Sanders AB, Raessler K, Muhlbaier LH, Rankin JS, Ewy GA.
Circulation. 1988;77(1):240-50.

The influence of chest compression rate on initial resuscitation success and 24 hr survival after prolonged manual cardiopulmonary resuscitation (CPR) was investigated in 26 morphine-anesthetized dogs (17 to 30 kg). After placement of aortic and right atrial micromanometers and induction of ventricular fibrillation, manual CPR was commenced immediately and continued for 30 min. One group of 13 dogs underwent manual CPR at a compression rate of 60/min, and the other group at a rate of 120/min. The compression durations in the two groups were not significantly different (51.7 +/- 1.8% at 60/min vs 51.6 +/- 1.9% at 120/min). No drugs other than sodium bicarbonate were administered during CPR. A maximum of three attempts was permitted to defibrillate the heart. Successfully defibrillated animals were followed for 24 hr, during which time no treatment, other than naloxone, was given to reverse the effects of morphine. Arterial blood pH, PCO₂, and PO₂ were not significantly different in the two groups throughout the CPR period. When compared with the compression rate of 60/min, the compression rate of 120/min produced more successfully defibrillated animals (12/13 at 120/min vs 2/13 at 60/min, p less than .002) and more 24 hr survivors (8/13 at 120/min vs 2/13 at 60/min, p less than .03). All 24 hr survivors were conscious and able to sit, stand, and drink normally. One 24 hr survivor in each group had difficulty walking. Improved survival with the high-rate compression technique was consistent with the significantly higher mean aortic (systolic and diastolic) and coronary perfusion pressures attained with high-rate compressions (all p less than .002). Although the clinical applicability of these findings has yet to be demonstrated, they provide empirical support for the recent decision to increase the chest compression rate for manual CPR recommended by the American Heart Association, and indicate that the hemodynamic and survival benefits of faster compression rates in this experimental preparation were not dependent on covariant alterations in compression duration.

Key Points: LOE3, supporting, good: Rate of chest compressions of 120 improves immediate and 24-hour survival in dogs compared to 60/min.

A physiologic comparison of external cardiac massage techniques.

Newton J. R, D. D. Glower.

J Thorac Cardiovasc Surg 1988;95(5):892-901.

On the basis of recent investigation, controversy has arisen regarding which of several cardiopulmonary resuscitation methods optimizes hemodynamics. The present study was designed to compare five recently described chest compression techniques: high-impulse manual chest compression at 150/min, mechanical

compression at 60/min with simultaneous ventilation, mechanical compression at 60/min with simultaneous ventilation and either systolic or diastolic abdominal compression, and pneumatic vest compression at 60/min. Eight dogs were chronically instrumented with electromagnetic flow probes in the ascending and descending aorta while matched micromanometers measured aortic, left ventricular, and pleural pressures. At study, each dog was anesthetized with morphine, intubated, and the heart was fibrillated by rapid ventricular pacing. The five cardiopulmonary resuscitation methods were performed randomly in each preparation within 7 to 10 minutes of arrest. In four dogs, brachiocephalic blood flow was computed as total cardiac output minus descending aortic blood flow, and in all dogs coronary perfusion pressure was calculated as mean diastolic aortic pressure minus mean diastolic left ventricular pressure. Average cardiac output for seven studies was 662 +/- 61 ml/min with high-impulse manual compression, 340 +/- 46 ml/min with mechanical compression and simultaneous ventilation, 336 +/- 45 ml/min with mechanical compression and simultaneous ventilation with systolic abdominal compression, 366 +/- 52 ml/min with mechanical compression and simultaneous ventilation with diastolic abdominal compression, and 196 +/- 29 ml/min with vest resuscitation (high-impulse manual compression significantly greater than other techniques by multivariate analysis, p less than 0.05). Brachiocephalic blood flow generally followed cardiac output and was statistically the greatest with high-impulse manual compression at 273 +/- 47 ml/min (p less than 0.05). Finally, high-impulse manual compression provided the highest coronary perfusion pressure of 31 +/- 4 mm Hg (p less than 0.05) compared to 23 +/- 2 mm Hg for mechanical compression and simultaneous ventilation, 23 +/- 2 mm Hg for mechanical compression and simultaneous ventilation with systolic abdominal compression, 23 +/- 3 mm Hg for mechanical compression and simultaneous ventilation with diastolic abdominal compression, and 11 +/- 2 mm Hg for vest resuscitation. These data demonstrate that high-impulse manual compression generated physiologically and statistically superior hemodynamics when compared with other methods in this model of cardiopulmonary resuscitation.

Key points: LOE3, supporting, good: Supports compression rates at up to 150bpm to improve hemodynamic blood flow during CPR.

The physiology of external cardiac massage: high-impulse cardiopulmonary resuscitation

Maier G.W, G. S. Tyson, et al. (1984).

Circulation 1984;70 (1): 86-101.

In intact chronically instrumented dogs, left ventricular dynamics were studied during cardiopulmonary resuscitation (CPR). Electromagnetic flow probes measured cardiac output and coronary blood flow, ultrasonic transducers measured cardiac dimensions, and micromanometers measured left ventricular, right ventricular, aortic, and intrathoracic pressures. The dogs were anesthetized with morphine, intubated, and fibrillated by rapid ventricular pacing. Data were obtained during manual external massage with dogs in the lateral and supine positions. Force of compression was varied from a peak intrathoracic pressure of 10 to 30 mm Hg, and compression rate was varied from 60 to 150/min. Increasing force of compression increased stroke volume up to a peak intrathoracic pressure of approximately 20 mm Hg, beyond which stroke volume remained constant or declined. Stroke volume appeared to result primarily from direct transmission of manual compression force to the heart rather than from positive intrathoracic pressure because peak cardiac or vascular pressures or the change in these pressures were consistently two to four times greater than the corresponding intrathoracic pressures during manual compression. With increasing compression rate, stroke volume remained relatively constant, and total cardiac output increased significantly: 425 +/- 92 ml/min at 60/min, 643 +/- 130 ml/min at 100/min, and 975 +/- 219 ml/min at 150/min (p less than .05). Left ventricular dimensions decreased minimally at higher manual compression rates. In four patients undergoing CPR, systolic and diastolic arterial blood pressure increased with faster compression rates, correlating well with data obtained in the dog. Dynamic coronary blood flow in canine experiments decreased to zero or negative values during compression. Antegrade coronary flow occurred primarily during noncompression periods and seemed to be related to

diastolic aortic perfusion pressure; coronary flow at a compression rate of 150/min averaged 75% of control. Therefore stroke volume and coronary blood flow in this canine preparation were maximized with manual chest compression performed with moderate force and brief duration. Increasing rate of compression increased total cardiac output while coronary blood flow was well maintained. Direct cardiac compression appeared to be the major determinant of stroke volume during manual external cardiac massage.

Key Points: LOE3, supporting, good: Supports increased compression rates. Increased compression rates and moderate force improved cardiac output and maintaining coronary perfusion.

Cardiac output during cardiopulmonary resuscitation at various compression rates and durations.

Fitzgerald K.R, C.F. Babbs, et al.

Am J Physiol 1981;241(3): H442-8.

Cardiac output during cardiopulmonary resuscitation (CPR) was measured by a modified indicator-dilution technique in 20 anesthetized dogs (6-12 kg), during repeated 1- to 2-min episodes of electrically induced ventricular fibrillation, by a mechanical chest compressor and ventilator. With compression rates from 20 to 140/min and compression durations (duty cycles) from 10 to 90% of cycle time, cardiac output (CO) was predicted by the equation: $CO = CR \cdot SV_{max} \cdot [DC / (k_1 \cdot CR + DC)] \cdot [(1 - DC) / k_2 \cdot CR + 1 - DC]$, where CR is compression rate, DC is duty cycle, SV_{max} (19 ml) is the effective capacity of the pumping chamber, and k_1 (0.00207 min) and k_2 (0.00707 min) are ejection and filling constants. This expression predicts maximal CO for DC = 0.40 and CR = 126/min and 90-100% of maximal CO for 0.3 less than DC less than 0.5 and 70 less than CR less than 150/min. Such mathematical analysis may prove useful in the optimization of CPR.

Key Points: LOE3, supporting, good: Compression rate and duty cycle for maximum cardiac output is 126/min for dogs 7-12kg.

Chest compression fraction determines survival in patients with out-of-hospital ventricular fibrillation.

Christenson J, Andrusiek D, Everson-Stewart S, Kudenchuk P, Hostler D, Powell J, Callaway CW, Bishop D, Vaillancourt C, Davis D, Aufderheide TP, Idris A, Stouffer JA, Stiell I, Berg R; Resuscitation Outcomes Consortium Investigators.

Circulation. 2009;120(13):1241-7.

BACKGROUND:

Quality cardiopulmonary resuscitation contributes to cardiac arrest survival. The proportion of time in which chest compressions are performed in each minute of cardiopulmonary resuscitation is an important modifiable aspect of quality cardiopulmonary resuscitation. We sought to estimate the effect of an increasing proportion of time spent performing chest compressions during cardiac arrest on survival to hospital discharge in patients with out-of-hospital ventricular fibrillation or pulseless ventricular tachycardia.

METHODS AND RESULTS:

This is a prospective observational cohort study of adult patients from the Resuscitation Outcomes Consortium Cardiac Arrest Registry with confirmed ventricular fibrillation or ventricular tachycardia, no defibrillation before emergency medical services arrival, electronically recorded cardiopulmonary resuscitation before the first shock, and a confirmed outcome. Patients were followed up to discharge from the hospital or death. Of the 506 cases, the mean age was 64 years, 80% were male, 71% were witnessed by a bystander, 51% received bystander cardiopulmonary resuscitation, 34% occurred in a public location, and 23% survived. After adjustment for age, gender, location, bystander cardiopulmonary resuscitation, bystander witness status, and response time, the odds ratios of surviving to hospital discharge in the 2 highest categories of chest compression fraction compared with the reference category were 3.01 (95% confidence interval 1.37 to 6.58)

and 2.33 (95% confidence interval 0.96 to 5.63). The estimated adjusted linear effect on odds ratio of survival for a 10% change in chest compression fraction was 1.11 (95% confidence interval 1.01 to 1.21).

CONCLUSIONS:

An increased chest compression fraction is independently predictive of better survival in patients who experience a prehospital ventricular fibrillation/tachycardia cardiac arrest.

Key Points: LOE6, supporting, good: Increasing the proportion of time that chest compressions are performed per minute improves survival from CPR.

A study of chest compression rates during cardiopulmonary resuscitation in humans. The importance of rate-directed chest compressions.

Kern K. B, A. B. Sanders, et al.

Arch Intern Med 1992;152(1): 145-9.

A prospective, cross-over trial was performed comparing two different rates of precordial compression using end-tidal carbon dioxide as an indicator of the efficacy of cardiopulmonary resuscitation in 23 adult patients. A second purpose of this study was to determine the effect of audio-prompted, rate-directed chest compressions on the end-tidal carbon dioxide concentrations during cardiopulmonary resuscitation. Patients with cardiac arrest received external chest compressions, initially in the usual fashion without rate direction and then with rhythmic audiotones for rate direction at either 80 compressions per minute or 120 compressions per minute. Nineteen of 23 patients had higher end-tidal carbon dioxide levels at the compression rate of 120 per minute. The mean end-tidal carbon dioxide level during compressions of 120 per minute was 15.0 +/- 1.8 mm Hg, slightly but significantly higher than the mean level of 13.0 +/- 1.8 mm Hg at a compression rate of 80 per minute. However, end-tidal carbon dioxide levels increased rather dramatically when audiotones were used to guide the rate of chest compressions.

Key Points: LOE6, supporting, good: Mean ETCO₂ at a compression rate of 120bpm was slightly higher than at 80bpm.

Chest compression rates during cardiopulmonary resuscitation are suboptimal: a prospective study during in-hospital cardiac arrest.

Abella BS, Sandbo N, Vassilatos P, Alvarado JP, O'Hearn N, Wigder HN, Hoffman P, Tynus K, Vanden Hoek TL, Becker LB.

Circulation. 2005;111(4):428-34.

BACKGROUND:

Recent data highlight a vital link between well-performed cardiopulmonary resuscitation (CPR) and survival after cardiac arrest; however, the quality of CPR as actually performed by trained healthcare providers is largely unknown. We sought to measure in-hospital chest compression rates and to determine compliance with published international guidelines.

METHODS AND RESULTS:

We developed and validated a handheld recording device to measure chest compression rate as a surrogate for CPR quality. A prospective observational study of adult cardiac arrests was performed at 3 hospitals from April 2002 to October 2003. Resuscitations were witnessed by trained observers using a customized personal digital assistant programmed to store the exact time of each chest compression, allowing offline calculation of compression rates at serial time points. In 97 arrests, data from 813 minutes during which chest compressions were delivered were analyzed in 30-second time segments. In 36.9% of the total number of segments, compression rates were <80 compressions per minute (cpm), and 21.7% had rates <70 cpm. Higher chest

compression rates were significantly correlated with initial return of spontaneous circulation (mean chest compression rates for initial survivors and nonsurvivors, 90+/-17 and 79+/-18 cpm, respectively; P=0.0033).

CONCLUSIONS:

In-hospital chest compression rates were below published resuscitation recommendations, and suboptimal compression rates in our study correlated with poor return of spontaneous circulation. CPR quality is likely a critical determinant of survival after cardiac arrest, suggesting the need for routine measurement, monitoring, and feedback systems during actual resuscitation.

Key Points: LOE6, supporting, good: Study supports concept that rate of compressions is directly correlated to survival from cardiac arrest.

Basics in advanced life support: a role for download audit and metronomes.

Fletcher D, R. Galloway.

Resuscitation 2008;78(2):127-34.

An intention in 2003 to undertake a multicentre trial in the United Kingdom of compressions before and after defibrillation could not be realized because of concerns at the time in relation to informed consent. Instead, the new protocol was introduced in one ambulance service, ahead of the 2005 Guidelines, with greater emphasis on compressions. The results were monitored by analysis of electronic ECG downloads. Deficiencies in the standard of basic life support were identified but were not unique to our service. The introduction of metronomes and the provision of feedback to crews led to major improvements in performance. Our experience has implications for the emergency pre-hospital care of cardiac arrest.

Key points: LOE6, supporting, fair: Supports importance of maintaining increased compression rates of at least 100bpm, for increased survival

Cardiocerebral resuscitation improves neurologically intact survival of patients with out-of-hospital cardiac arrest.

Kellum M. J, K.W. Kennedy, et al.

Amer Jour of Med 2006;119: 335-340.

The neurologic status at or shortly after discharge was documented for adult patients with a witnessed collapse and an initially shockable rhythm. Patients during two 3-year periods were compared. During the 2001 through 2003 period, in which the 2000 American Heart Association guidelines were used, data were collected retrospectively. During the mid-2004 through mid-2007 period, patients were treated according to the principles of cardiocerebral resuscitation. Data for these patients were collected prospectively. Cerebral performance category scores were used to define the neurologic status of survivors, and a score of 1 was considered as "intact" survival.

In the 3 years preceding the change in protocol, there were 92 witnessed arrests with an initially shockable rhythm. Eighteen patients survived (20%) and 14 (15%) were neurologically intact. During the 3 years after implementation of the new protocol, there were 89 such patients. Forty-two (47%) survived and 35 (39%) were neurologically intact.

In adult patients with a witnessed cardiac arrest and an initially shockable rhythm, implementation of an out-of-hospital treatment protocol based on the principles of cardiocerebral resuscitation was associated with a dramatic improvement in neurologically intact survival.

Key Points: LOE6, supporting, poor: Supports increased chest compression rates for survival of cardiac arrest.

Twenty-four hour survival in a canine model of cardiac arrest comparing three methods of manual cardiopulmonary resuscitation.

Kern K. B., A. B. Carter, et al.
J Am Coll Cardiol 1986;7: 859-67.

Two new modifications of manual cardiopulmonary resuscitation, high impulse compression at a rate of 120/min and interposed abdominal compression at a rate of 60/min, have been reported to produce better hemodynamic responses than standard cardiopulmonary resuscitation at 60/min. However, the effect of these two new methods on initial resuscitation success and 24 hour survival is unknown. In this study, 30 mongrel dogs were divided into three equal groups, each treated with one of three types of manual cardiopulmonary resuscitation. Ventricular fibrillation was induced electrically in morphinized, endotracheally intubated dogs emerging from halothane anesthesia. After 3 minutes of circulatory arrest without intervention, one of the three techniques of manual cardiopulmonary resuscitation was begun, and continued for 17 minutes. Defibrillation was performed at 20 minutes. Successful resuscitation was defined as a mean arterial blood pressure of at least 60 mm Hg, without chest compressions, 10 minutes after the initial defibrillation attempt. Intensive care was provided for 2 hours, including hemodynamic and respiratory monitoring, and drug intervention when required. Twenty-four hour survival and neurologic deficit were used as critical measures of outcome. Ten of 30 animals survived 24 hours with a mean neurologic deficit score of 5% (normal = 0, brain dead = 100). There was no difference in initial resuscitation success, 24 hour survival or neurologic deficit of the survivors among the three manual cardiopulmonary resuscitation methods. Aortic diastolic and calculated coronary perfusion pressures were similar for all three methods. Well performed standard manual cardiopulmonary resuscitation is as effective as these modified versions (high impulse compression and interposed abdominal compression) when compared in the same animal model.

Key Points: LOE3, neutral, fair: No difference in survival or initial resuscitation with compression rate between 60/minute and 120/minute

Intrathoracic pressure fluctuations move blood during CPR: comparison of hemodynamic data with predictions from a mathematical model.

Halperin H. R., J.E. Tsitlik, et al.
Ann Biomed Eng 1987;15(3-4): 385-403

Whether blood flow during cardiopulmonary resuscitation (CPR) results from intrathoracic pressure fluctuations or direct cardiac compression remains controversial. We developed a mathematical model that predicts that blood flow due to intrathoracic pressure fluctuations should be insensitive to compression rate over a wide range but dependent on the applied force and compression duration. If direct compression of the heart plays a major role, however, the model predicts that flow should be dependent on compression rate and force, but above a threshold, insensitive to compression duration. These differences in hemodynamics produced by changes in rate and duration form a basis for determining whether blood flow during CPR results from intrathoracic pressure fluctuations or from direct cardiac compression. The model was validated for direct cardiac compression by studying the hemodynamics of cyclic cardiac deformation following thoracotomy in four anesthetized, 21-32-kg dogs. As predicted by the model, there was no change in myocardial or cerebral perfusion pressures when the duration of compression was increased from 15% to 45% of the cycle at a constant rate of 60/min. There was, however, a significant increase in perfusion pressures when rate was increased from 60 to 150/min at a constant duration of 45%. The model was validated for intrathoracic pressure changes by studying the hemodynamics produced by a thoracic vest (vest CPR) in eight dogs. The vest contained a bladder that was inflated and deflated. Vest CPR changed intrathoracic pressure without direct cardiac compression, since sternal displacement was less than 0.8 cm. As predicted by the model and opposite

to direct cardiac compression, there was no change in perfusion pressures when the rate was increased from 60 to 150/min at a constant duration of 45% of the cycle. Manual CPR was then studied in eight dogs. There was no surgical manipulation of the chest. Myocardial and cerebral blood flows were determined with radioactive microspheres and behaved as predicted from the model of intrathoracic pressure, not direct cardiac compression. At nearly constant peak sternal force (378-426 N), flow was significantly increased when the duration of compression was increased from short (13%-19% of the cycle) to long (40%-47%), at a rate of 60/min. Flow was unchanged, however, for an increase in rate from 60 to 150/min at constant compression duration. In addition, myocardial and cerebral flow correlated with their respective perfusion pressures.

Key Points: LOE3, neutral, fair: External chest compressions generate blood flow via intrathoracic pressure fluctuations and vital organ perfusion is independent on rate of compression.

Slow versus rapid closed-chest cardiac compression during cardiopulmonary resuscitation in puppies.

Fleisher G, C. Delgado-Paredes, S. Heyman.

Crit Care Med 1987;15(10):939-43.

Summary: The recommended rates for closed-chest cardiac compressions during CPR are based on physiologic variations with increasing age rather than experimental data. Using puppies, investigators compared mean arterial pressure, cardiac index (CI), and cerebral blood flow (CBF) at a slow (40/min, group 1) and a rapid (120/min, group 2) rate. CBF was measured in all experiments by N₂O uptake before and during CPR from cardiac arrest induced with KCl. Groups 1 and 2 were similar in terms of baseline mean weight, mean arterial pressure, CI, CBF, and arterial and venous blood gases. The values for mean CI and CBF (N₂O or microspheres) did not differ significantly at either rate of compression. The N₂O uptake and microsphere techniques for measuring CBF correlated closely during spontaneous cardiac contractions and mechanical chest compressions. Investigators concluded that a) closed-chest cardiac compressions at either rate studied provided inadequate CBF, and b) the N₂O uptake and microsphere techniques give similar measures of CBF under conditions of normal and low flow.

Key Points: LOE3, neutral, good: No difference in cerebral blood flow between compression rates of 40 and 120/ min

Hemodynamics in humans during conventional and experimental methods of cardiopulmonary resuscitation.

Swenson R.D, W. D. Weaver ,et al.

Circulation 1988;78(3): 630-9.

High-fidelity hemodynamic recordings of aortic and right atrial pressures and the coronary perfusion gradient (the difference between aortic and atrial pressure) were made in nine patients during cardiopulmonary resuscitation (CPR). Findings during conventional manual CPR were compared with those during high-impulse CPR (rate, 120 cycles/min with a shorter compression:relaxation ratio) as well as during pneumatic vest CPR with and without simultaneous ventilation and abdominal binding. Aortic peak pressure during conventional CPR averaged 61 +/- 29 mm Hg but varied widely (range, 39-126 mm Hg) among patients. Although the magnitude of improvement was modest, the high-impulse method was the only technique tested that significantly elevated both aortic peak pressure and the coronary perfusion gradient during cardiac arrest. During conventional CPR, aortic pressure rose from 61 +/- 29 to 80 +/- 39 mm Hg during high-impulse CPR, and the gradient rose from 9 +/- 11 to 14 +/- 15 mm Hg, respectively; p less than 0.01. The pneumatic vest method significantly improved peak aortic pressure but not the coronary perfusion gradient. Simultaneous ventilation and chest compression created high end-expiratory pressure and lowered the coronary perfusion

gradient. Abdominal binding had no significant hemodynamic effects. This evaluation of experimental resuscitation methods in humans shows that the high-impulse chest compression method augments aortic pressure over levels achieved during conventional CPR methods; however, the improvement in pressure is modest and may not be clinically important. Simultaneous ventilation as well as abdominal binding during CPR were associated with no benefit; in fact, simultaneous ventilation appears to adversely affect cardiac perfusion and, therefore, should not be used during clinical resuscitation.

Key Points: LOE6, neutral, fair: 120 compressions/minutes produces modestly higher diastolic and aortic pressures when compared to conventional CPR and may not be significant.

Effect of cardiopulmonary resuscitation compression rate on end-tidal carbon dioxide concentration and arterial pressure in man.

Ornato J.P., Gonzalez E.R., et al.
Crit Care Med 1988;6(3): 241-5.

The optimal rate of chest compression during CPR in man has been debated. Recently, the end-tidal carbon dioxide concentration (PetCO₂) has been shown to correlate with cardiac output during CPR in experimental animals. Eighteen prehospital cardiac arrest patients were studied to determine the effect of external chest compression rate on the PetCO₂ and BP in man when ventilation rate, ventilation inspiration time, applied compression force, and a 50:50 downstroke:upstroke ratio were held constant using a microprocessor-controlled CPR Thumper. Compression rate was increased from 60 to 140/min in 20 beat/min increments. The PetCO₂ was 1.7 +/- 0.2% at a compression rate of 60/min and did not change significantly at increased rates. Systolic BP fell progressively from 59 +/- 5 mm Hg at 60/min to 46 +/- 4 mm Hg at 140/min. Diastolic BP remained approximately 23 mm Hg at all rates studied. Using a CPR manikin, we found that greater Thumper compression force was necessary to sustain the same sternal displacement and to achieve the same applied sternal pressure when the rate was increased due to a rate-limited fall in the compression duration.

Key Points: LOE6, neutral, poor: An increased compression force was needed to maintain blood flow at increased rates in this manikin study.