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Relationship of Blood Pressure and Flow During CPR to Chest Compression Amplitude: Evidence for an Effective Compression Threshold

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Abstract

This study was conducted to investigate the importance of the depth of chest compression in producing effective cardiopulmonary resuscitation (CPR) in animals, as indicated by cardiac output and mean arterial blood pressure. Cardiac output was measured by a modified indicator dilution technique in 8 anesthetized dogs, 6 to 12 kg body weight, during repeated 2-minute episodes of electrically induced ventricular fibrillation and CPR provided by a mechanical chest compressor and ventilator (Thumper®). Chest compression exceeding a threshold value (x_0) between 1.5 and 3.0 cm was required in each animal to produce measurable cardiac output. In particular, cardiac output (CO) was linearly related to chest compression depth (x) by an expression of the form $CO = a(x-x_0)$ for $x > x_0$, and $CO = 0$ for $x \leq x_0$. The mean value of x_0 was 2.3 cm. A similar threshold for measurable blood pressure was observed in 7 of the 8 dogs, with a mean value of 1.8 cm. For chest compression of 2.5 cm or greater, relatively modest increases in chest compression depth caused relatively large changes in cardiac output.

Key words: defibrillation, myocardial damage, toxicity, ventricular fibrillation, waveform

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Introduction

Chest compression during cardiopulmonary resuscitation (CPR) is intended to generate an artificial cardiac output. Theoretically, forward flow of blood occurs either because the cardiac ventricles are rhythmically compressed between the sternum and the spine [1] or because elevations in global intrathoracic pressure produced by thoracic compression are vented by outflow of arterial blood [2-4]. With either mechanism, the blood flow must be related in some way to the force, depth, or general vigor of chest compression. However, the vigor of manual

chest compression may vary widely among rescuers and may progressively diminish as a given rescuer tires. Yet to date no systematic investigation has been reported that describes optimum compression force or sternal displacement in animals or in man. We conducted a controlled study in dogs to determine the influence of these variables on cardiac output and mean arterial blood pressure during CPR.

Methods

Animal Preparation

Eight mongrel dogs weighing between 6 and 12 kg served as subjects. Younger animals with compliant chest walls and with ventral-dorsal versus right-left thoracic diameters less than 1.6:1 were selected for the study. Each animal was anesthetized with pentobarbital sodium (30 mg/kg IV). The trachea was intubated with the largest possible cuffed endotracheal tube. The following intravascular catheters were inserted: 1) a pigtail catheter advanced into the left ventricle through the femoral artery for injection of indicator to measure cardiac output; 2) a 30-cm long, 0.1-cm internal diameter catheter advanced into the mid-thoracic aorta through the femoral artery and attached to a motor-driven syringe for withdrawal of blood during inscription of dilution curves; 3) a catheter to monitor arterial pressure advanced 5 to 10 cm into the right brachial artery; and 4) a femoral venous catheter for administration of anesthetic. Heparin (1 mg/kg IV) was given to retard clot formation in the catheters, to permit reinfusion of blood withdrawn during inscription of dilution curves, and to diminish intravascular coagulation during periods of circulatory arrest.

The animal was then placed on a V-shaped board which was fixed securely to the baseplate of a specially modified Thumper® mechanical resuscitator (Michigan Instruments, Inc, Grand Rapids, MI). The limbs were tied securely to prevent lateral motion of the chest during CPR. Subcutaneous electrodes for recording the electrocardiogram (lead II) were secured in place, and wire mesh electrodes for sternal-to-back defibrillation were applied to the shaved skin of these regions with electrolytic gel. The V-shaped, 20 x 20-cm back electrode for defibrillation conformed to the animal board, and the rectangular sternal electrode was molded to the 6 x 10-cm chest compression pad of the Thumper®. With this electrode arrangement defibrillation could be accomplished easily on the down stroke of compression without interrupting CPR. The ventral-dorsal chest compression provided by this system was chosen rather than lateral chest compression (which is accepted practice in veterinary medicine) to more closely model the situation of human resuscitation.

Physiologic Monitoring

A four-channel graphic record was inscribed using a Physiograph® direct-inking recorder (Narco Bio-Systems, Houston, TX). Channels 1 and 2 displayed the electrocardiogram and arterial blood pressure. Intraesophageal pressure was recorded on channel 3 as a monitor of chest compression and ventilation. The amplitude of esophageal pressure pulses was used to monitor intrathoracic pressure in order to prevent traumatic over-compression. For this purpose, pressure was detected by a 25-cm long x 1-cm diameter soft rubber tube placed in the esophagus between the thoracic

inlet and the diaphragm and connected to a pressure transducer. Channel 4 of the graphic record displayed indicator dilution curves for measurement of cardiac output by the saline-conductivity method [5], which was specially modified for the low flow conditions of CPR [6]. This method employs 5% NaC1 solution as the indicator and a calibrated, flow-through conductivity cell as the detector. Its accuracy has been confirmed by comparison with the direct Fick method under conditions of CPR [7]. Two-milliliter aliquots of 5% saline indicator were injected forcibly into the left ventricle, and blood samples were withdrawn through the detector using the catheter placed in the thoracic aorta. This injection-sampling configuration permits adequate mixing of indicator in blood during CPR for accurate measurements of cardiac output [6]. Prior to trials of experimental CPR, control cardiac output was measured three times in each animal, while the heart was beating normally.

Experimental CPR

In a typical arrest/resuscitation sequence, ventricular fibrillation was produced by 60 Hz electrical stimulation of the left ventricular endocardium. A fine, 0.1-mm stainless steel wire threaded through the lumen of the left ventricular catheter facilitated conduction of electric current to the heart for this purpose. Ventricular fibrillation was confirmed by the presence of chaotic fibrillation waves in the electrocardiogram and by loss of pulsatile arterial pressure. Immediately after confirmation of fibrillation, ventilation and chest compression were initiated using the mechanical Thumper® energized with 100% oxygen at 60 psi. The 6 x 10- cm chest compression pad was centered in the midline with its caudal edge at the level of the xiphi-stemal junction. The effective inspired oxygen concentration was approximately 80% and the peak inspiratory pressure was 20 cmH_2O. The ventilation compression ratio was 1:5 and the overall compression rate was 62/min. The compression duration was always 50% of compression cycle time. After a 30-second stabilization period, electronically derived mean blood pressure was recorded at a chart speed of 0.1 cm/sec for 30 to 50 seconds, during which time a dilution curve was obtained. Then a damped sine wave defibrillator shock of 20 to 50 joules was applied during the down stroke of the chest compression via the chest-to-back electrodes.

After defibrillation the animal was allowed to recover until a stable, pulsatile blood pressure returned. Periodically arterial blood samples were obtained and analyzed immediately for pH, PCO_2, and PO_2 with the aid of an Instrumentation Laboratories Model 213 blood gas analyzer. These values were maintained as close as possible to normal by administration of sodium bicarbonate and by adjustment of airway dead space to counteract the tendency toward mixed metabolic acidosis and respiratory alkalosis.

On successive trials the force and depth of chest compression were varied to produce four to six different levels of peak esophageal pressure. In practice, compression amplitudes sufficient to develop either 20, 40, 60, 80, and 100 mmHg esophageal pressure or 10, 20, 30, 40, and 50 mmHg esophageal pressure were usually selected. The piston excursion of the Thumper® required to produce each level of esophageal pressure was also measured with the aid of a ruler. Cardiac output was calculated after each trial and plotted as a function of the peak esophageal pressure and of the sternal deflection generated by the Thumper®. As soon as the approximate range of cardiac output values achievable in a given animal was ascertained, further selection of

compression values to be tested was determined according to a random sequence. In this way each dog was taken through a total of 10 to 15 sequences of fibrillation-CPR-defibrillation at a variety of compression pressures and depths. At no time was a total force exceeding 100 lb applied to the sternum, and at no time was esophageal pressure in excess of 100 mmHg allowed to develop. At each level of sternal compression cardiac output and arterial pressures were measured in duplicate or triplicate, so that data representing 10 to 15 arrest/resuscitation sequences were obtained from each animal.

Postmortem Examination

At the conclusion of each experiment the animals were sacrificed by an overdose of barbiturates, and gross examination of the abdominal and thoracic cavities was conducted. Proper placement of catheters was confirmed. Special effort was made to record traumatic damage to the heart, liver, chest wall, lungs, pleura, and mediastinal structures as a result of chest compression.

Data Analysis

Cardiac output and blood pressure data were plotted as functions of the amplitude of chest compression, measured either as the distance traversed by the chest compression pad during one cycle or as the peak esophageal pressure developed during chest compression. To combine results from all the animals, linear regression analysis was performed on the data from each animal, and the value of cardiac output or mean arterial pressure for 5 cm chest compression was determined from the regressions. These values for CO (5 cm) and MAP (5 cm) were taken as normalizing values for each dog. The choice of 5 cm chest compression as a standard value was made because it is within the range recommended by American Heart Association standards for CPR [8].

A similar regression analysis was performed on CO and MAP data plotted as a function of peak esophageal pressure. Normalizing values for CO (50 mmHg) and MAP (50 mmHg) were determined by double reciprocal plotting and linear interpolation. Cardiac output and blood pressure data for each animal were then divided by these factors and plotted together to determine their relative dependence on chest compression amplitude in the series of animals.

Results

Cardiac Output

Cardiac output was dependent on chest compression amplitude in all animals; however, the range of cardiac output achievable varied from animal to animal. This individual variation is indicated by the normalizing values presented (Table 1). For example, the cardiac output achieved with 5 cm sternal compression ranged from 142 mL/min (10% of control) to 923 mL/min (42% of control). However, if the cardiac output data for each dog are divided by CO (5 cm) and plotted

together as a function of chest displacement, the results shown in Figure 1 are obtained. Cardiac output tended to increase linearly for chest displacements from 2.5 to 6 cm.

Table 1. Normalizing values in eight dogs subjected to experimental CPR

Body Weight (kg)	Beating Heart CO (mL/min)	CPR CO*	
		CO (5 cm) (mL/min)	CO (50 mm Hg) (mL/min)
10.4	1,400	145	109
11.3	1,860	159	120
8.5	884	236	303
10.6	2,210	923	569
10.5	2,080	690	939
8.9	1,470	142	181
7.2	662	281	184
8.0	1,140	162	227

*CO = cardiac output.

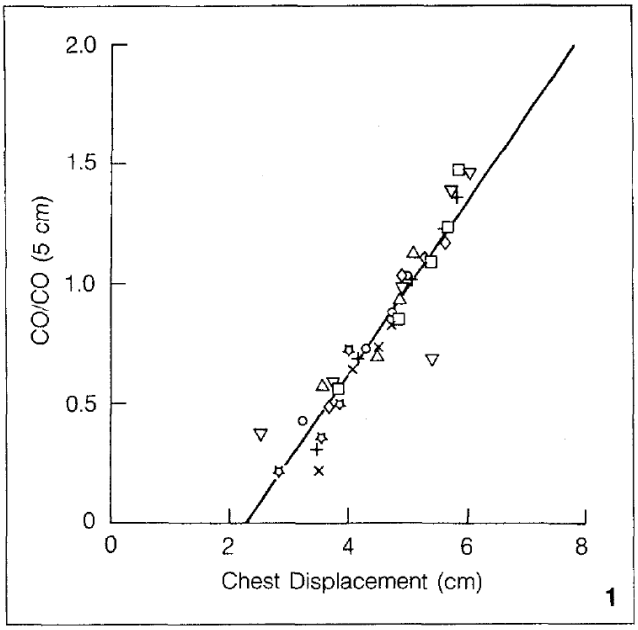

Figure 1. Relative cardiac output during ventricular fibrillation and CPR in dogs as a function of ventral-dorsal chest displacement. Displacement is represented in cm. Cardiac output is plotted as a fraction of the value obtained when the chest is compressed to 5 cm. Each data point represents the mean of two or three determinations in a given dog, and each symbol type represents a particular animal. The straight line is the least-squares, linear regression line.

The combined linear regression for all the dogs can be expressed in the form

$$CO/CO\ (5\ cm) = a\ (x - x_0),$$

for all values of compression depth greater than threshold value, x_0, and CO/CO (5 cm) = 0 otherwise. In this expression, "CO/CO (5 cm)" is the relative cardiac output, "x" is the chest compression in centimeters, "a" is the slope of the regression line, and "x_0" is the apparent threshold for effective compression. A similar analysis was done to calculate compression threshold values for individual dogs. The mean threshold was 2.3 cm and the standard deviation was 0.45 cm. The apparent threshold, x_0, is extrapolated rather than interpolated, and there are few data points for compression less than x_0 to confirm that subthreshold flow is zero. The reason that subthreshold data were not routinely collected was that animals would not survive repeated trials of CPR with such ineffective compression in preliminary experiments. Consequently such observations could not be routinely included in the data set.

A similar analysis was performed to show relative cardiac output during CPR as a function of the peak esophageal pressure developed during chest compression (Figure 2). CO/CO (50 mmHg) is the ratio of measured cardiac output to the cardiac output that would have been produced at a peak esophageal pressure of 50 mmHg. When peak esophageal pressure is used to define chest compression amplitude, the function does not indicate a threshold pressure for measurable cardiac output. Whenever peak intrathoracic pressure (as measured in the esophagus) was greater than zero, artificial measurable cardiac output was generated. Further increase in the amplitude of chest compression up to 50 mmHg esophageal pressure improved flow. More forceful compression than required to produce 50 mmHg esophageal pressure was effective in raising intrathoracic pressure, but ineffective in further improving flow.

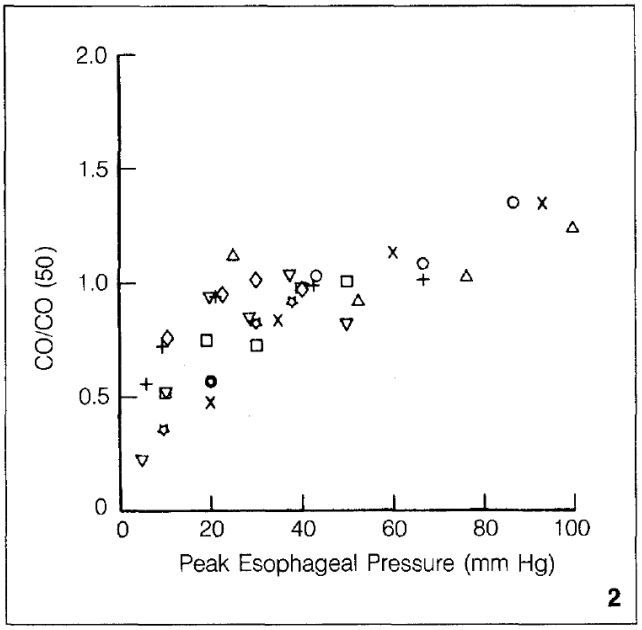

Figure 2. Relative cardiac output during ventricular fibrillation and CPR in dogs as a function of the peak esophageal pressure developed during sternal compression. Cardiac output is plotted as a fraction of the value obtained when the chest is compressed sufficiently to produce 50 mmHg peak esophageal pressure. Each data point represents a mean of two or three determinations in a given dog, and each symbol type represents a particular animal.

Mean Arterial Pressure

Similar analysis of mean arterial pressure (Figures 3 and 4) indicates an effective compression threshold of 1.5 cm for the study population. Linear regression analysis of individual data from seven of the eight dogs demonstrated threshold values, x_0, greater than zero. For these seven animals, the mean threshold for generation of non-zero arterial pressure was 1.8 cm, and the standard deviation was 0.85 cm.

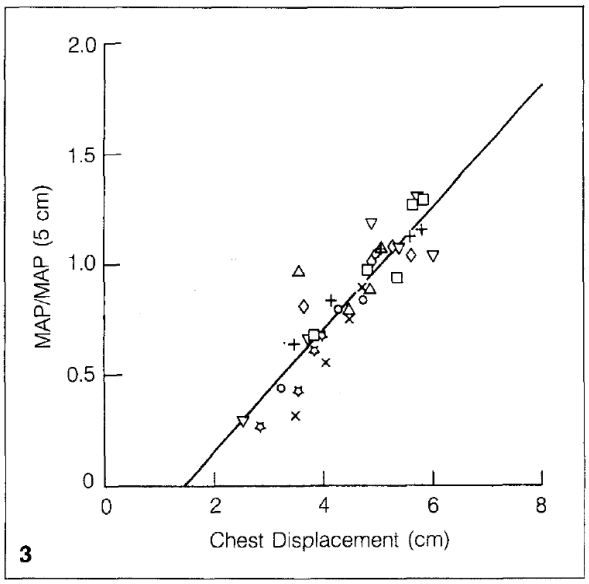

Figure 3. Relative mean arterial pressure during ventricular fibrillation and CPR as a function of sternal displacement. Details similar to Figure 1. Constants of the regression equation are $a = 0.28\ cm^{-1}$, $x_0 = 1.45$ cm.

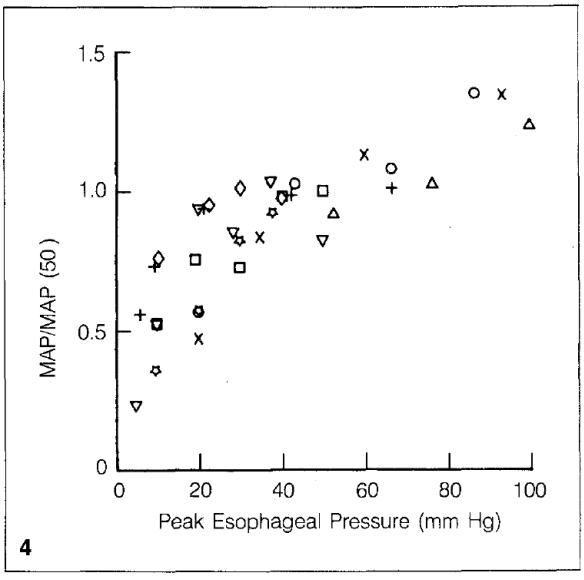

Figure 4. Relative mean arterial pressure during ventricular fibrillation and CPR as a function of peak esophageal pressure. Details similar to Figure 2.

Pathological Findings

Gross pathological findings in the animals studied are shown (Table 2). All had multiple rib fractures. About half had pulmonary contusion, mediastinal hemorrhages, and/or superficial cardiac hemorrhages in regions directly in the line of force applied during chest compression. None of the mediastinal hemorrhages appeared extensive enough, in our opinion, to produce cardiac tamponade. Because the cardiac output versus displacement curves for the two animals found to have hepatic laceration were not strikingly different from those of the other animals, they were not excluded from the study. Many of the pathological findings may have been related to some degree to heparin treatment, and thus their significance is difficult to assess.

Table 2. Gross pathological findings in dogs following 10 to 20 CPR trials*

Finding	Occurrence
Multiple rib fractures	8/8
Pulmonary contusion dorsal or ventral to heart	3/8
Epicardial hemorrhages	2/8
Pulmonary edema	1/8
Hepatic lacerations	2/8
Mediastinal hemorrhage	1/8
Hemothorax	1/8
Subendocardial hemorrhage	1/8

*Force on chest up to 100 lb. Developed esophageal pressure up to 100 mm Hg.

Discussion

Standards for cardiopulmonary resuscitation and emergency cardiac care [8] state only that in external cardiac massage the sternum should be depressed "a minimum of 1½ to 2 inches" (approximately 4 to 5 cm), and that "lesser amounts of compression are ineffectual." These statements are certainly supported by our study. However, the standards do not go on to emphasize the depth of chest compression as a critical variable. Rather, they seem to imply that any degree of chest compression within the prescribed range of 1½ to 2 inches is satisfactory. Such a recommendation would be rational if the true function relating cardiac output and compression depth were as shown (Figure 5, curve A). This hypothetical function rises to a plateau such that any degree of compression in the plateau region would be close to maximally effective.

Our study, however, argues strongly that the actual functional relationship is more like the line B in Figure 5 when sternal deflection is the measure of chest compression. In this situation flow is quite sensitive to small changes in sternal displacement, and for some displacements below a critical threshold value, cardiac output is virtually zero. Hence, under field conditions in which the compression force and depth may vary, it is unlikely that a given victim receives optimal CPR for the duration of the resuscitation effort.

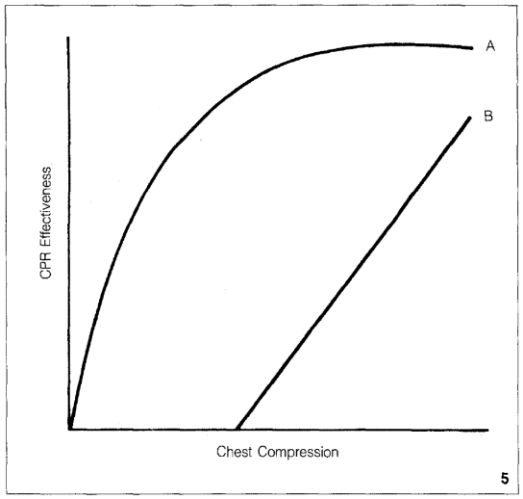

Figure 5. Hypothetical functions relating CPR effectiveness to chest compression amplitude. Horizontal axis is chest compression depth, and vertical axis is CPR effectiveness. Top function (A) indicates CPR effectiveness is insensitive to small changes in compression over a large range, as seems to have been implicitly assumed by framers of CPR standards. Lower line (B) is similar to results of this study when sternal deflection is used as the measure of chest compression. This line implies an effective compression threshold, below which CPR is totally ineffective, and indicates that CPR effectiveness is quite sensitive to small changes in compression depth. Interestingly, if peak esophageal pressure is used as the measure of chest compression, a curve similar to (A) is obtained experimentally.

Our experience in animals suggests that peak esophageal pressure may be a better indicator of effective chest compression than is sternal displacement. In particular, if either cardiac output or mean arterial pressure is taken as a measure of CPR effectiveness, and if peak esophageal pressure is taken as a measure of chest compression amplitude, then a curve more similar in shape to Figure 5-A is generated. If we were to measure esophageal pressure during prolonged resuscitations and maintain chest compressions so as to keep peak esophageal pressure slightly greater than 50 mmHg, we might be reasonably certain that near-maximal cardiac output were being generated. In conjunction with airways that incorporate an esophageal balloon, such as the esophageal obturator airway (EOA®), routine monitoring of esophageal pressure might be feasible in many arrest situations with little modification of existing equipment. Provision of such feedback might well lead to greater uniformity in the quality of chest compression and lessen the risk of chest wall or visceral damage due to over compression.

Conclusion

Our study should be interpreted as a plea for more careful monitoring of chest compression rather than a plea for more forceful chest compression. Previously Redding and Cozine [9] found that during closed chest massage in dogs, mediastinal hemorrhage, fractured ribs, and lacerations of the liver were frequently encountered when maximal force was applied to the chest sufficient to produce the greatest possible blood pressure. However, they quickly developed a moderately forceful technique, which generally avoided these complications.

Our pathological findings include hepatic laceration in two of eight animals, and certainly indicate that the risk/benefit ratio of more forceful chest compression deserves further evaluation. One should be especially cautious in drawing conclusions from this study regarding the risk/benefit ratio of CPR with more forceful compressions, because the animals were pre-treated with heparin, which alters the pathologic lesions of traumatic over compression and may have some influence on the outcome of resuscitation [10].

Our particular objective was to compare only relative benefits, as measured by cardiac output and mean arterial pressure, with each animal serving as its own control. Relative risks could not be assessed because more than one degree of chest compression was tested in each animal, and one cannot conclude which level of compression was critical for producing damage. A different experimental design will be required to address the overall risk/benefit ratio of CPR as a function of compression amplitude. Nonetheless, researchers and practitioners of CPR should be aware of the probable existence of an effective compression threshold and the potential benefits of monitoring chest compression more closely than has been done in the past.

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