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Cardiac output during cardiopulmonary resuscitation at various compression rates and durations

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Abstract

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 and CPR, produced 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:

$$CO = CR \cdot SV_{\max} \left[\frac{DC}{k_1 CR + DC} \right] \cdot \left[\frac{1 - DC}{k_2 CR + 1 - DC} \right],$$

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 $CR = 126/\text{min}$ as well as 90-100% of maximal CO for $0.3 < DC < 0.5$ and $70 < CR < 150/\text{min}$. Such mathematical analysis may prove useful in the optimization of CPR.

Key words: cardiac arrest; cardiac arrhythmias; fibrillation, sudden cardiac death

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Introduction

Instructional guidelines (1), supported by some research (3, 8, 15), strongly imply that the frequency of sternal compression is critical to adequate performance of external cardiopulmonary resuscitation (CPR). Rhythm and counting to achieve the correct overall rate of 60/min are emphasized in practical CPR instruction. However, Taylor et al. (12) found no effects of the frequency of chest compression in the range of 30-80/min on the carotid flow velocity or arterial blood pressure during CPR. Earlier Del Guercio et al. (4), who studied patients during arrest using a mobile cardiac catheterization laboratory, found virtually zero correlation between cardiac index and compression rate in the range of 50-130/min for either external or internal cardiac massage. Rather than emphasizing compression rate, several authors (3, 4, 5, 12, 13) have pointed to the importance of sustained compression in generating cardiac output during CPR.

To develop a unifying concept for prediction of the optimum frequency and compression time for chest massage during CPR, we have conducted a systematic investigation of compression rate (i.e., frequency of compression) and duty cycle (compression duration divided by cycle time) during experimental CPR in dogs. In addition, we have developed and tested a simple mathematical model that predicts from limited experimental data the cardiac output generated by any combination of compression rate and duty cycle. This model may be useful in the validation and future refinement of CPR standards.

Theory

The output of pumps of the type sketched in Figure 1 is limited both by the time required for emptying and by the time required for filling of the pumping chamber. This statement applies to either the cardiac pump mechanism (Figure 1 A) or the thoracic pump mechanism (Figure 1 B) for blood flow during CPR (2). Consider an experiment in which only compression rate and duty cycle are varied, whereas other factors, such as compression force and ventilation pressure, are held constant. The functions relating pump output to filling time and to emptying time should be similar to those shown in Figure 2.

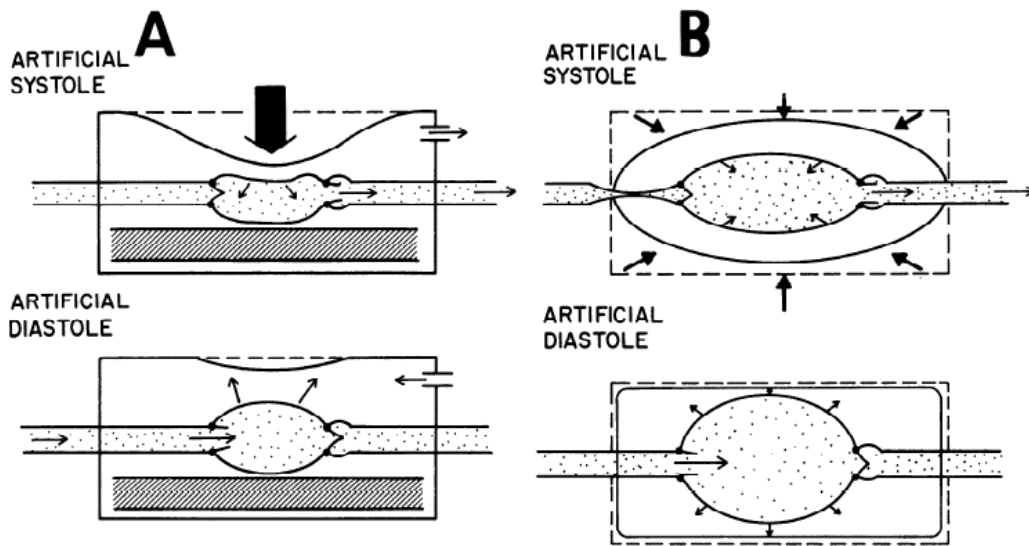


Figure 1. A: cardiac pump mechanism for generating blood flow during cardiopulmonary resuscitation (CPR). During artificial systole, central chest compression squeezes the heart against the spine, forcing blood out. Air is vented from the thorax via the trachea (top right). During artificial diastole, chest resiliency creates negative pressure for filling. Site of application of force (broad arrow) is critical. For simplicity, only 1 pumping chamber is shown. B: thoracic pump mechanism for generation blood flow during CPR. During artificial systole, thoracic compression or cough generates intrathoracic pressure, which is vented by arterial outflow to peripheral tissues. During artificial diastole release of intrathoracic pressure allows for filling. Site of application of force is not important. (Reproduced with permission from Crit. Care Med. 8: 192, 1980).

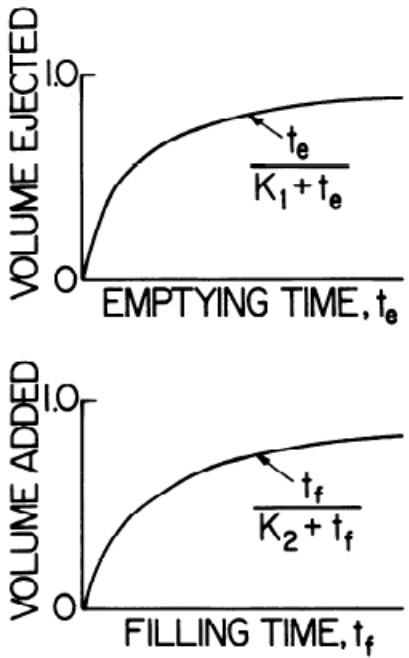


Figure 2. Hypothetical relationships between fractional emptying and filling of cardiac or thoracic pumps and time available for emptying (t_e) and time available for filling (t_f). Plateau levels represented by 1.0 indicate complete emptying or filling under given conditions.

According to this hypothesis, pump output is zero when either filling time or emptying time is zero. The contribution of emptying time to output rises to a plateau that represents complete emptying under the given conditions. Similarly, the contribution of filling time to output rises to a plateau that represents complete filling under the given conditions. The shapes of the functions sketched in Figure 2 may vary slightly from individual to individual. In general, however, a reasonable and mathematically tractable approximation to such functions takes the form $t/(k + t)$, for filling or emptying time (t) and filling or emptying constant (k). Such functions have value zero for $t = 0$, a maximal plateau value 1.0 for $t \gg k$, and value 0.5 when $t = k$, the time for 50% filling or emptying under the given conditions. In principle, then, a function to describe approximately the influence of compression rate and duty cycle on pump output can be written as

cardiac output = frequency \times stroke volume

or

$$CO = CR \cdot SV_{\max} \left[\frac{t_e}{k_1 + t_e} \right] \cdot \left[\frac{t_f}{k_2 + t_f} \right], \quad (1)$$

where CR represents compression rate, SV_{\max} represents the effective capacity of the pumping chamber, if it were allowed to completely fill and to completely empty, t_e and t_f represent ejection and filling times, and k_1 and k_2 represent ejection and filling constants. SV_{\max} is likely to depend on the size of the victim, the force of compression, the relative blood volume, venous filling pressure, and other constraining factors, such as the presence or absence of arterial collapse (16). The constant k_1 , equal to the time for half emptying of the pump, is likely to depend on the mechanical coupling between the chest wall and the pumping chamber and the outflow resistance. The constant k_2 , equal to the time for half filling of the pump, is likely to depend on the venous inflow resistance. Substituting $t_e = DC/CR$, and $t_f = (1 - DC)/CR$ in terms of the duty cycle (DC), for $0 < DC < 1$, we have

$$CO = CR \cdot SV_{\max} \left[\frac{DC}{k_1 CR + DC} \right] \cdot \left[\frac{1 - DC}{k_2 CR + 1 - DC} \right]. \quad (2)$$

Equation 2 concisely states our hypothesis concerning the effects of compression rate and duty cycle in CPR. This expression implies that the cardiac output is zero under four conditions: when rate is zero, when the emptying time is zero (which corresponds to duty cycle = 0), when the filling time is zero (which corresponds to duty cycle = 1.0) and when rate is very large so that both the time for filling and the time for emptying approach zero (vibration rather than compression of the chest). Accordingly this model predicts that there is an optimal intermediate compression rate and an optimal intermediate duty cycle at which cardiac output is maximal. With this conceptual framework in mind, we studied the influence of the rate and duty cycle of chest compression on cardiac output during CPR with the following objectives: 1) to measure cardiac output in a population of animals during CPR with a variety of compression rates and duty cycles, 2) to determine if the relative changes in output are described by Eq. 2, and 3) to determine if Eq. 2 has genuine predictive value: specifically, to determine if Eq. 2 with best-fit constants SV_{\max} , k_1 , and k_2 , evaluated for a limited number of observations in one population of animals, can predict relative cardiac output for entirely different compression rates and duty cycles in a different population of animals.

Methods

Animal preparation

Twenty mongrel dogs weighing 6-16 kg were used. Relatively younger animals with compliant chest walls and with ventral-dorsal vs. right-left thoracic diameters less than 1.6:1 were selected for the study. Each animal was anesthetized with 30 mg/kg i.v. pentobarbital sodium. The trachea was intubated with the largest possible cuffed endotracheal tube. The following catheters were inserted percutaneously: 1) a pigtail catheter advanced from a femoral artery into the left ventricle for injection of indicator to measure cardiac output; 2) a 30-cm-long 0.1-cm-ID catheter advanced from a femoral artery to the midthoracic aorta and attached to a motor-driven syringe for withdrawal of blood during inscription of dilution curves; 3) a catheter to monitor arterial pressure advanced 5-10 cm into the right brachial artery; and 4) a catheter to monitor venous pressure advanced via the right maxillary vein to a position in the superior vena cava. The catheters, which were used for arterial and venous pressure monitoring, were of the same length and caliber and were connected to matched Statham pressure transducers. Heparin (1 mg/kg i.v.) 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 that was fixed securely to the baseplate of a specially modified Thumper mechanical resuscitator (Michigan Instruments, Grand Rapids, MI). 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 downstroke 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) (10) to more closely model the situation of human resuscitation.

Physiological monitoring

A four-channel graphic record was inscribed using a Physiograph direct-inking recorder (Narco Bio-Systems, Houston, TX). Channels 1, 2, and 3 displayed the electrocardiogram, arterial blood pressure, and venous blood pressure, respectively. Electronically derived mean pressures could be obtained with front panel switches on the transducer drivers. Pressure channels were calibrated, and their linearity was confirmed by means of a mercury manometer. Channel 4 of the graphic record displayed indicator dilution curves for the measurement of cardiac output by the saline conductivity method (6, 7). This method uses 5% NaCl solution as the indicator and a calibrated flow-through conductivity cell as the detector. Two-milliliter aliquots of 5% saline indicator were injected forcibly into the left ventricle, and blood samples were withdrawn through the detector via the catheter placed in the thoracic aorta. This injection and sampling

configuration is similar to the right ventricular injection and pulmonary artery sampling scheme used in commercial thermodilution systems and permits adequate mixing of indicator in blood for accurate measurements of cardiac output, even under the very low flow conditions of CPR (6). Cardiac output values obtained with this technique during CPR agree with those obtained simultaneously with the Fick method (11).

To inscribe a dilution curve, blood was withdrawn from the thoracic aorta through the conductivity cell at a rate of 10 ml/min. This withdrawal rate was at most 15%, and typically about 5%, of the cardiac output during CPR. The internal volume of the conductivity cell used to measure indicator concentration was 0.1 ml. We considered this sampling rate fast enough to generate accurate dilution curves during CPR, yet slow enough so as to not alter significantly the measured cardiac output.

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 ECG and by loss of arterial blood pressure.

Immediately after confirmation of fibrillation, ventilation and chest compression were initiated with the mechanical chest compressor and positive pressure ventilator (Thumper). The 6 x 10 cm chest compression pad was centered in the midline with its caudal edge at the level of the xiphisternal junction. The Thumper was energized with 100% oxygen gas at 60 psi. The time constant for sternal displacement by the Thumper was 60-90 ms in preliminary experiments. Peak inspiratory pressure was 20 cmH₂O. The inspired oxygen concentration was approximately 80%. The force of chest compression was maintained at a constant value in the range of 30-50 pounds, which was determined in preliminary trials to produce the greatest arteriovenous pressure difference during ventricular fibrillation and CPR. Unless this step was taken to optimize compression force, animals did not survive experimental CPR with extremely unfavorable duty cycles (10 or 90%) in preliminary studies.

Ventilation and compression cycles of the Thumper were controlled by solenoid activated valves. The control unit, specially designed and fabricated by one of us (RWD), allowed quick selection of the desired compression rate and duty cycle. Ventilations were initiated independently of chest compression at a frequency of 12/min before, during, and after CPR. After 20 s of fibrillation and CPR, electronically derived mean blood pressures were recorded for 30-50 s. A dilution curve was obtained during this period, while blood pressures remained stable. Then a damped sine-wave defibrillator shock of 20-50 J was applied on the downstroke of the chest compression via the chest-to-back electrodes. After defibrillation the animal was allowed to recover until a stable pulsatile arterial blood pressure was attained.

Repeated 2-min episodes of ventricular fibrillation and CPR were studied in the same animal. In every animal, trials of standard compression rate and duty cycle (60/min, 50% duty cycle) were

studied, so that cardiac output data could be compared with the mean value generated under these standard, conditions. In this sense each animal served as its own control. At least two, and typically three, repeated trials of CPR at each combination of rate and duty cycle were studied in each animal according to a randomized sequence.

Manipulation of compression rate and duration: experiment 1

This experiment used 10 dogs. In one phase of the experiment, CPR was performed with a constant duty cycle of 50%, and compression rates of 20, 30, 40, 60, 80, 100, 120, and 140/min were tested in random order. In a second phase of the experiment, the compression rate was held constant at 60/min, and duty cycles of 10, 30, 50, 70, and 90% were studied. In half of the animals, the various rates were tested first and in the other half of the animals the various duty cycles were tested first.

Data analysis

First, to correct for animal-to-animal variation, all cardiac output values were divided by the mean value for standard CPR (60/min, 50% duty cycle) obtained in a particular dog. With these normalized cardiac output values, we conducted the following regression analysis to determine best-fit values of the constants SV_{max} , k_1 , and k_2 in Eq. 2. SV_{max} was determined from the y-intercept of the least-squares linear-regression function for the plot of $y = \ln(SV)$ vs. compression rate for DC = 0.5. The y-intercept indicates the SV for compression rate approaching zero, in which case there is maximal time per compression for both filling and emptying of the pump. Hence we could estimate SV_{max} as the y-intercept of this plot.

Knowing SV_{max} , and hence $CO_{max} = CR \times SV_{max}$ for this population of dogs, we calculated the best-fit values for k_1 and k_2 for trials in which compression rate

$$r = \frac{DC(1-DC)}{CR} \left[\frac{CO_{max}}{CO} - 1 \right] = k_1(k_2 \cdot CR + 1) - DC(k_1 - k_2). \quad (3)$$

This linear function of duty cycle (DC) is a rearrangement of Eq. 2. Numerical values for the emptying and filling constants, k_1 and k_2 were estimated from measured cardiac output data by calculating the least-squares linear regression coefficients for values of Eq. 3 as a function of the duty cycle, DC. If actual cardiac output data are predicted from Eqs. 1, 2, and 3, then a plot of

the calculated values, r , as a function of duty cycle will be a straight line with slope $-(k_1 - k_2)$ and intercept $k_1(k_2 CR + 1)$. From this plot, the theoretical values k_1 and k_2 were estimated from the simultaneous equations, slope = $-(k_1 - k_2)$ and intercept = $k_1(k_2 CR + 1)$, namely

$$k = \frac{1}{2 CR} \sqrt{s^2 + 4 \cdot \text{intercept} \cdot CR},$$

where $s = \text{slope} \cdot CR + 1$, and $k_2 = k_1 + \text{slope}$.

Knowing these least-squares estimates for SV_{\max} , k_1 , and k_2 , we could then calculate predicted values using Eq. 2 and compare them with measured flow values for the same animals.

Manipulation of compression rate and duration: experiment 2

The objective of this study was to determine if the optimum duty cycle was dependent on the compression rate and if the mathematical model represented by Eqs. 1, 2, and 3 would be successful in predicting the comparative flow rates in a new population of subjects under conditions not previously tested. In experiment 2 we compared measured cardiac output values in another 10 dogs with predicted values using the constants SV_{\max} , k_1 , and k_2 , determined from experiment 1. Methods were identical to experiment 1, except that the duty cycle and rate combinations tested were 10, 30, 50, 70, and 90% duty cycle at 30 compressions/min and 10, 30, 50, 70, and 90% duty cycle at 120 compressions/min.

Results

Figure 3A presents the effect of compression rate on relative cardiac output and stroke volume when duty cycle was maintained constant at 50% in experiment 1. Each data point represents the mean \pm SD of the 20-30 normalized cardiac output measurements obtained in the 10 dogs. The absolute value for cardiac output during standard CPR (42 ± 19 ml/min/kg) for the 10 dogs in experiment 1, is within the range of values previously reported by others in dogs and in humans (4, 9, 14, 15). As compression rate increased from zero to 140/min, cardiac output rose to a plateau. Relative stroke volume (relative cardiac output/compression rate) decreased steadily as rate increased (Figure 3B). Solid lines represent functions calculated from regression analysis of the data in Figures 3 and 4.

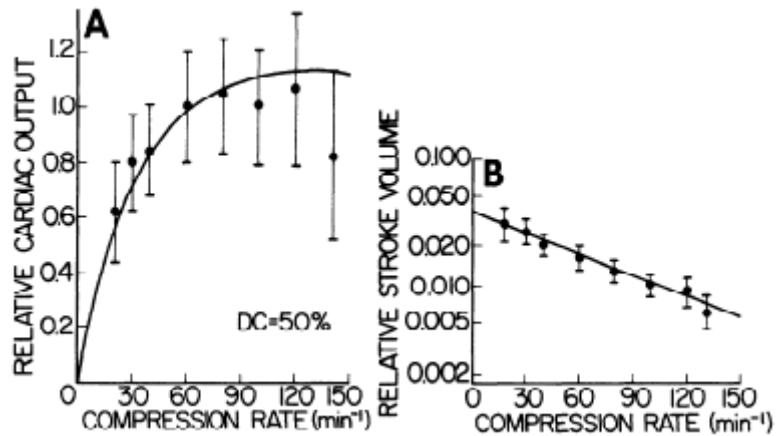


Figure 3. A: influence of compression rate on relative cardiac output. Means \pm SD of 20-30 determinations in 10 animals are plotted; 1.0 on ordinate represents 42 ml/min/kg, the measured flow during standard CPR (60/min, 50% duty cycle). Smooth curve represents regression function of the form of Eq. 2 evaluated with $SV_{max} = 0.038$, $k_1 = 0.00207$ min, $k_2 = 0.00707$ min. B: normalized stroke volume during CPR as a function of compression rate. Normalized, relative stroke volume was calculated as normalized cardiac output/compression rate and represents fraction of cardiac output during standard CPR generated per compression. Normalized stroke volume is a decreasing exponential function of compression rate. Least-squares line for semilog plot is shown. Value of stroke volume, extrapolated to zero compression rate, is taken as estimate of SV_{max} . Non-normalized value of SV_{max} is 19 ml.

Figure 4A presents the effect of duty cycle on relative cardiac output when compression rate was maintained constant at 60/min in experiment 1. At 60 compressions/min, the duty cycle for maximal cardiac output was approximately 40%. The smooth curve represents the function of the form of Eq. 2 calculated from regression analysis. Best agreement between measured and calculated values for cardiac output occurred when the regression analysis included only the three intermediate duty cycles: 30, 50, and 70%. Figure 4B shows that the r values (defined in Eq: 3) for these duty cycles fall along a straight line. The r values for the extreme duty cycles 10 and 90%, fall above the line, indicating a relatively lower cardiac output than expected from theory. These discrepancies for 10 and 90% compression are reasonable in view of the very short time period (100 ms) allowed for compression with 10% duty cycle and for chest recoil with 90% duty cycle. In practice, more than 100 ms was required for complete compression or recoil of the physical system tested. Indeed, the excursion of the Thumper pad was approximately 20% less (3.7 cm vs. 4.8 cm) for 10 and 90% duty cycles than for 30-70% duty cycles.

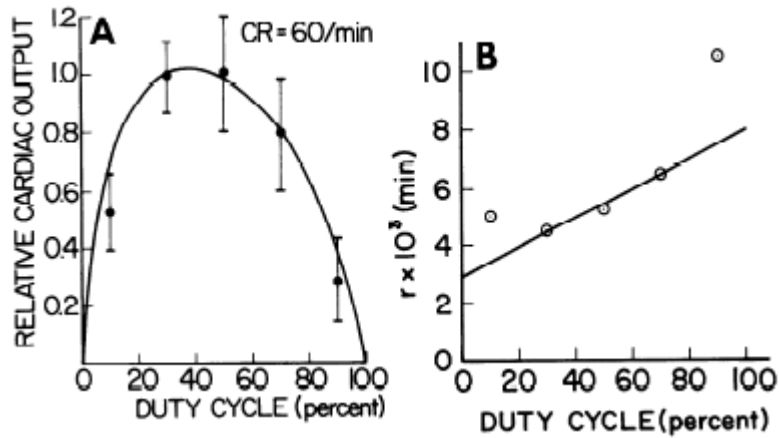


Figure 4. A: influence of duty cycle on relative cardiac output. Details similar to Figure 3A. Smooth curve represents regression function of the form of Eq. 2 evaluated with $SV_{max} = 0.038$, $k_1 = 0.00207$ min, $k_2 = 0.00707$ min. **B:** regression analysis for determination of emptying and filling constants k_1 and k_2 . Values of r plotted on ordinate were calculated as indicated in Eq. 3.

Mean arterial blood pressure varied with duty cycle in a manner similar to cardiac output (Figure 5). However, systolic blood pressure bore no relation to flow and was virtually the same for all duty cycles tested (Figure 5). Similarly, when duty cycle was held constant at 50% and rate was varied from 20/min to 140/min, mean arterial pressure rose from 33 mmHg at 20/min to a plateau of 49-53 mmHg at 80-120/min. Systolic arterial pressure, however, was not related to compression rate, with mean values for the 10 dogs ranging from 72 to 81 mmHg.

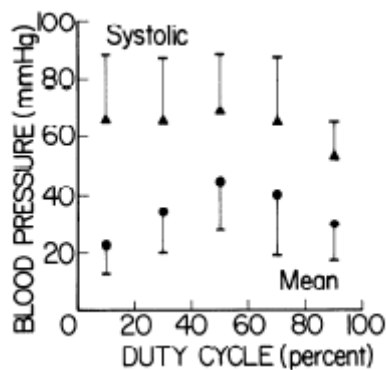


Figure 5. Systolic and mean arterial blood pressures during CPR at various duty cycles. Compression rate = 60/min. Details similar to Figure 3A.

Comparative cardiac output data from experiment 2 in which duty cycles from 10 to 90% were tested at 30 and 120 compressions/min are presented in Figure 6, A and B, respectively. As in Figures 3 and 4, the data are normalized by the values obtained during standard (60/min, 50%) CPR. Smooth curves represent the relative cardiac output values predicted from k_1 and k_2 calculated for the animals in experiment 1. These plots constitute a test of the predictive value of Eqs. 1, 2, and 3. In Figure 6A, the measured relationship between flow and duty cycle at 120 compressions/min is similar in shape to that predicted but slightly less in amplitude. In Figure 6B, measured and predicted cardiac output for all duty cycles at 30 compressions/min are less than those at 60 or 120 compressions/min. With compression rate equal 30/min and prolonged duty cycles of 70 and 90%, measured cardiac output is somewhat higher than predicted.

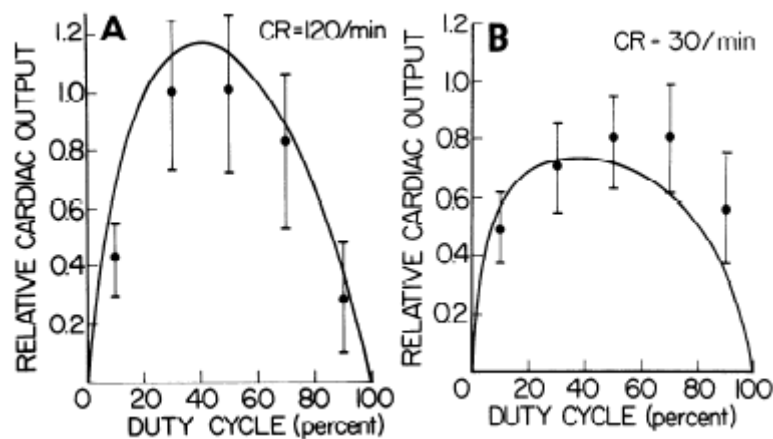


Figure 6. Measured and predicted cardiac output in experiment 2. A: compression rate = 120/min. B: compression rate = 30/min. 1.0 on ordinate represents value predicted for 60/min, 50% CPR. Smooth curve represents regression function of form of Eq. 2 evaluated with $SV_{max} = 0.038$, $k_1 = 0.00207$ min, $k_2 = 0.00707$ min, determined for dogs in experiment 1.

Discussion

These studies show that the effects of compression rate and compression duration on blood flow during CPR are largely understandable in terms of a simple conceptual model. Knowing the species constants determined from one experiment, one can predict reasonably well the cardiac output in a different population of subjects at various rates and duty cycles that were not previously tested.

At the extreme duty cycles of 10 and 90% and at the higher compression rates of 120/min and 140/min the measured cardiac output was less than that predicted. These discrepancies at shorter absolute compression or relaxation times may be related to the non-zero time constants for chest compression and recoil. A zero time constant indicates an instantaneous change in level. In actuality, the chest cannot be compressed and cannot recoil instantaneously. For example, if the time available is less than that required for complete compression, during 10% duty cycle, 60/min CPR, the mean amplitude of compression will be less than that at longer duty cycles. As a result, one would expect a reduced stroke volume compared to that predicted by our simple model in which constant compression amplitude (constant SV_{max}) is assumed. A similar argument can be made for 90% duty cycle CPR in which there is inadequate time for complete recoil of the chest. This fact was very evident to us in conducting these experiments with 90% duty cycle. For these reasons, the observation of cardiac output slightly lower than that predicted for extremely high or low duty cycles and extremely fast compression rates is not surprising and should not seriously limit the utility of the proposed model.

Figure 7 shows the predicted flow surface above the rate-duty cycle plane for dogs. The effect of duty cycle upon cardiac output is important at all rates. There is little effect of compression rate on cardiac output in the range of 50-120/min, because stroke volume decreases in approximately inverse proportion to compression rate. Along most of the ridge of the surface the compression rate is relatively unimportant in determining flow. This prediction is in agreement with our observations of Taylor et al. (12).

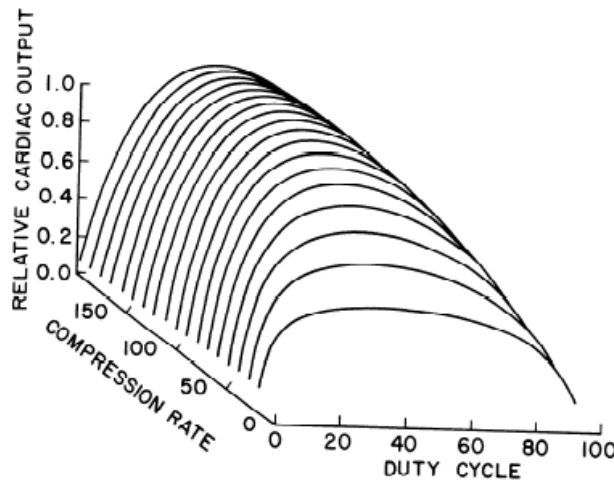


Figure 7. Predicted flow surface above rate-duty cycle plane, using Eq. 2 with the values $SV_{max} = 0.0380$, $k_1 = 0.00207$ min, and $k_2 = 0.00707$.

In particular, one can use this mathematical model to predict the rate and the duty cycle for maximal cardiac output in CPR. Setting partial derivatives of Eq. 2 with respect to rate and duty cycle equal to zero and simplifying, one finds that the compression rate for maximal cardiac output at a given duty cycle is

$$CR_{\max} = \sqrt{\frac{DC(1-DC)}{k_1 \cdot k_2}}, \quad (4)$$

and that duty cycle for maximal cardiac output at a given compression rate is

$$DC_{\max} = \frac{1}{k_1 - k_2} \left[Q - \sqrt{Q^2 - Q(k_1 - k_2)} \right], \quad (5)$$

where $Q = k_1 (1 + k_2 CR)$, or $DC_{\max} = 1/2$ if $k_1 = k_2$.

It is easy to find by numerical methods the simultaneous solutions of Eq. 4 and 5 for the pair of values CR_{\max} and DC_{\max} giving the best flow. Simply estimate CR_{\max} from Eq. 3, assuming $DC_{\max} = 0.5$ as an initial guess. Evaluate Eq. 4 using the estimate of CR_{\max} to obtain a revised estimate of DC_{\max} . Then evaluate Eq. 3 using the new estimate of DC_{\max} , etc. This method converges very rapidly to a unique solution in the proper quadrant of the rate-duty cycle plane. Using these equations, we found the rate and duty cycle for maximal cardiac output in 7 to 12 kg dogs are 126/min and 40%, values which are in agreement with experimental data (Figures 3 and 4).

Presumably, this paradigm is transferable to man. By studying as few as three rate, duty cycle combinations one can calculate the three constants SV_{\max} , k_1 , and k_2 for humans and then predict the theoretically best combination for human CPR. Extremely unfavorable combinations (such as 10 or 90% duty cycle) need not be tested in humans. Because the assumptions of our mathematical model are not stringent, it is reasonable to expect that a surface similar to that in Figure 7 exists for humans. Such a surface can be calculated if sufficient data are obtained by clinical investigators to calculate human values for k_1 and k_2 . Blood flow rather than blood pressure data are best for this purpose, because they directly indicate perfusion. In particular, systolic pressure is meaningless as an indicator of flow in a situation in which duty cycle is likely to change (Figure 3). This is because short duty cycle compressions produce pressure spikes, which are transient and ineffective in moving blood, a phenomenon previously documented in the CPR literature (3, 4, 5). If one is limited to pressure measurements, mean blood pressure computed from the pressure-time integral is a better indicator of flow.

The conceptual model presented in this paper is far from complete. Only the timing factors are explicitly included. Other important determinants of flow such as compression force, venous filling pressure, peripheral resistance, and the collapse pressure of arteries exiting the thorax are assumed constant and absorbed into the term SV_{\max} . In practice these variables may not be independent of rate, duty cycle, and flow as we have assumed. Peripheral resistance and venous inflow resistance are likely to depend on perfusion. Nevertheless, we believe the model presented in this paper describes the essential features of the influence of timing factors in CPR.

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