Improved Blood Flow During Prolonged Cardiopulmonary Resuscitation With 30% Duty Cycle in Infant Pigs

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**Background.** Sustained compression is recommended to maximize myocardial and cerebral blood flow during cardiopulmonary resuscitation (CPR) in adults and children. We compared myocardial and cerebral perfusion during CPR in three groups of 2-week-old anesthetized swine using compression rates and duty cycles (duration of compression/total cycle time) of 100 per minute, 60%; 100 per minute, 30%; and 150 per minute, 30%.

**Methods and Results.** Ventricular fibrillation was induced and CPR was begun immediately with a sternal pneumatic compressor. Epinephrine was continuously infused during CPR. Microsphere-determined blood flow and arterial and sagittal sinus blood gas measurements were made before cardiac arrest was induced and after 5, 10, 20, 35, and 50 minutes of CPR. At 5 minutes of CPR, ventricular and cerebral blood flows were greater than 25 ml·min⁻¹·100 g⁻¹ and were not significantly different between groups. When CPR was prolonged, however, myocardial and cerebral blood flows were significantly higher with the 30% duty cycle than with the 60% duty cycle. By 35 minutes, all myocardial regions had less than 5 ml·min⁻¹·100 g⁻¹ flow with the 60% duty cycle. In contrast, CPR with the 30% duty cycle at either compression rate provided more than 25 ml·min⁻¹·100 g⁻¹ to all ventricular regions for 50 minutes. By 20 minutes, most brain regions received 50% less flow with the 60% duty cycle compared with animals undergoing CPR with the 30% duty cycle (p<0.05). Cerebral oxygen uptake was better preserved with the 30% duty cycle. Chest deformation from loss of recoil was greater with the 60% duty cycle compared with the 30% duty cycle.

**Conclusions.** We conclude that the shorter duty cycle provides markedly superior myocardial and cerebral perfusion during 50 minutes of CPR in this infant swine model. These data do not support recommendations for prolonged compression at rates of 100 per minute during CPR in infants and children. (Circulation 1991;84:896–904)

Cardiopulmonary resuscitation (CPR) has been the subject of intense laboratory investigation during the past 20 years, and our understanding of the mechanism of blood flow during CPR has shifted from Kouwenhoven’s suggestion of direct cardiac compression¹ to include the intrathoracic pump mechanism.²,³ Although there remains some controversy about the precise mechanism of blood flow during CPR, the American Heart Association recently revised its guidelines for CPR by increasing the recommended compression rate.⁴ This would benefit blood flow by the cardiac compression model by increasing the number of compressions per minute, whereas it benefits blood flow by the intrathoracic pump model by decreasing the total cycle time and increasing the effective duty cycle (compression duration divided by total cycle time) for manual compressions of constant duration.

Conventional CPR has been ineffective for producing blood flow in numerous adult animal studies,⁵,⁷ and this led to studies that emphasized the intrathoracic pump mechanism. Augmenting intrathoracic pressure, either by using simultaneous compression and ventilation CPR or by using a thoracic vest increased myocardial and cerebral perfusion,⁵,⁶,⁸,⁹ and the physiological data have been found to match...
theoretical models based on an intrathoracic pump mechanism.9,10

Until recently there has been little information about CPR in an immature animal model that might be applicable to human infants and children. In contrast with results obtained in adult animals, we demonstrated that conventional CPR in 2-week-old piglets was quite effective in maintaining cerebral and myocardial perfusion for up to 35 minutes.11 Differences between these results and those obtained in adult dogs may reflect the different chest geometry of the two models.12 We found that augmentation of intrathoracic pressure with simultaneous compression and ventilation CPR did not improve blood flow compared with conventional CPR in piglets,13 presumably because conventional CPR was already highly effective. Piglets tolerate higher compression rates during CPR than older animals,14 suggesting that optimal compression rate and duty cycle in this model might differ from the optimal in adult animals.

Although conventional CPR with 60% duty cycle was effective in piglets, vital organ perfusion began to deteriorate at 20–30 minutes because of a decrease in aortic pressure.11 We currently hypothesized that shorter duration compressions might cause less ongoing deformation of the chest. This could lead to more effective prolonged CPR because refilling the thorax would not be impeded, total intrathoracic volume would be better preserved, and vascular channels would be less likely to be mechanically obstructed. The purpose of this study was to compare myocardial and cerebral perfusion during conventional CPR conducted for 50 minutes with a 30% duty cycle or a 60% duty cycle at compression rates of 100 and 150 per minute. We chose the young piglet because of its relatively deformable chest and because the configuration of the chest is broader than other nonprimate species. In addition, the chest size and stiffness of the piglet are more similar to the human infant than are those of most other animals. We used 50 minutes of CPR because prolonged CPR is relatively common in pediatric patients. Only one compression rate/duty cycle combination was used on each animal to evaluate the ongoing loss of chest recoil with repetitive sternal compressions.

Methods

These experiments were conducted according to the guiding principles of the American Physiological Society for studies involving experimental animals and were approved by the animal care and use committee of The Johns Hopkins University School of Medicine.

Preparation

All experiments were performed on 2-week-old piglets, ranging in weight from 3.5 to 6.8 kg (mean, 5.2 kg). The animals were anesthetized with 30 mg/kg i.p. sodium pentobarbital; supplemental anesthetic was usually required every 30 to 45 minutes, and 3–5 mg/kg i.v. was administered at these intervals. The last dose of anesthetic was always at least 30 minutes before onset of CPR, and no anesthetics were administered during CPR. The animals were ventilated via tracheostomy with supplemental oxygen by a volume-cycled ventilator before cardiac arrest. Fluid-filled catheters were advanced into the right atrium, left ventricle, and thoracic aorta via the femoral vein and arteries, respectively. Additional catheters were inserted via the axillary arteries into the proximal subclavian arteries for microsphere withdrawal and into the axillary veins for administration of fluids and epinephrine. A catheter was placed in the sagittal sinus with the catheter tip lying 1 cm anterior to the confluence of the sinus. This catheter was used to sample blood for determination of cerebral venous blood gases, pH, and oxygen content, and to measure sagittal sinus pressure. A pacing electrode was inserted into the right ventricle via a femoral vein, and was used to induce ventricular fibrillation. Heparin (1,000 units) was administered before cardiac arrest was induced. Early in these procedures, 60 ml of blood was withdrawn and replaced with 180 ml of Ringer's lactate; this blood was then reinfused during CPR to minimize changes in hemoglobin from the microsphere reference withdrawal samples.

Measurements

Regional blood flow was measured with the radiolabeled microsphere technique,15 which has previously been validated for use during CPR.5 Microspheres (15±1.5 μm diameter; DuPont–New England Nuclear Products, Boston) were vigorously shaken with a vortex shaker, sonicated for at least 20 minutes to disperse aggregates, and then vortexed again immediately before injection. Approximately 10⁶ spheres were injected in the measurement made before cardiac arrest, and 6×10⁵ spheres were injected for measurements made during CPR. A lower dose was used during CPR because most of the cardiac output is diverted to the brain and heart. These injections resulted in at least 1,000 spheres in each region of interest. The spheres were injected into the left ventricle over 10 seconds, then a 10-ml saline flush was given. The arterial reference withdrawal was obtained from an axillary arterial line with a withdrawal syringe pump (Harvard Apparatus, South Natick, Mass.) set at 3.82 ml/min before cardiac arrest and 1.91 ml/min during CPR. Withdrawal was continued for 2 minutes after the injection given before cardiac arrest and 5 minutes after each injection during CPR. After the experiment, the brain and heart were removed and fixed for 1–2 days in buffered formalin. The brain was dissected into cerebellum, medulla, pons, midbrain, diencephalon, cerebellum, caudate nucleus, and cerebrum. The cerebrum was further divided into middle cerebral, posterior cerebral, and watershed areas between the anterior and middle cerebral arteries and the posterior and middle cerebral arteries. The heart was dissected into the right and left atra, right ventricular subendocardium and subepicardium, left ventricular subendocardium, subepicardium, and midmyocard-
dium, and left, right, and middle sections of the interventricular septum. Multiple samples were also taken of the masseter muscle, tongue, skin, kidney, small intestine, and hind limb muscle. The samples were weighed, placed in vials, and counted with reference samples in a gamma counter (Minaxi model 5530, Packard Instruments, Dowers Grove, III.) equipped with a 3-in. through-hole crystal. The energy windows were (keV): $^{153}$Gd 68–170, $^{114}$In 174–230, $^{113}$Sn 360–440, $^{103}$Ru 450–560, $^{95}$Nb 690–820, and $^{46}$Sc 830–1,200. Spectra of pure isotopes were used to correct for overlap of counts using simultaneous equations.$^{15}$ Tissue blood flow ($Q_t$) was calculated as $Q_t= (C_1 \cdot Q_s)/ (C_1 \cdot W_s)$, where $Q_s$ is the reference sample withdrawal rate, $C_1$ is the reference sample count, $Q_1$ is the tissue sample count, and $W_s$ is the tissue weight. (Flows were normalized to ml · min$^{-1}$ · 100 g$^{-1}$ tissue wt.)

Arterial and sagittal sinus blood samples were analyzed for pH, PO$_2$, and PCO$_2$ with a Radiometer BMS3 electrode and analyzer system (Copenhagen). Oxygen contents and hemoglobin levels were measured with a CO-oximeter (model 282, Instrumentation Laboratory, Inc., Lexington, Mass.). All blood samples were analyzed immediately after drawing the samples. Cerebral oxygen uptake was calculated from the arterial-sagittal sinus oxygen content difference and blood flow to the cerebrum. Cerebral fractional oxygen extraction equals the arterial-sagittal sinus oxygen content difference divided by the arterial oxygen content.

Aortic blood pressure, right atrial pressure, and sagittal sinus pressures were measured with Statham db23 transducers and continuously recorded on an eight-channel Gould strip chart recorder (Glen Burnie, Md.). All transducers were referenced to the level of the right atrium, and true zero measurements were obtained at autopsy. During CPR, piston force measured with a strain gauge in the mechanical ventilator (Thumper, Michigan Instruments), and piston displacement measured with a sliding potentiometer were continuously recorded. Baseline chest measurements of each animal were obtained with a ruled caliper.

**Experimental Protocols**

Regional blood flow, arterial and sagittal sinus blood gases, and mean cerebral, right atrial, and sagittal sinus pressures were measured before cardiac arrest was induced and after 5, 10, 20, 35, and 50 minutes of CPR. Ventricular fibrillation was induced by passing alternating current (60 Hz) through the pacing electrode in the right heart. Immediately after fibrillation was induced, epinephrine was administered as a bolus through the femoral vein (40 $\mu$g · kg$^{-1}$) and a continuous infusion of epinephrine was then administered through an axillary vein throughout CPR (10 $\mu$g · kg$^{-1}$ · min$^{-1}$). Epinephrine was diluted in saline and infused at a volumetric rate of 1.91 ml · min$^{-1}$. In addition, blood was infused at a rate of 1.91 ml · min$^{-1}$ during CPR to offset the blood withdrawn for microsphere reference withdrawal sampling.

CPR was instituted 10 seconds after fibrillation using 100 compressions per minute and 60% duty cycle. External chest compression was delivered with a pneumatic chest compressor (Thumper). Compression force was adjusted to achieve 20% compression of the initial anteroposterior diameter and was not subsequently adjusted. The Thumper was then changed to one of four combinations of compression rate and duty cycle: 150 per minute, 30% $(n=7)$; 100 per minute, 30% $(n=6)$; 100 per minute, 60% $(n=7)$; and 150 per minute, 60% $(n=3)$. In the latter group, massive chest deformation occurred and no blood withdrawal was possible during CPR, so data from the first three groups are presented in this article. In all groups, the lungs were inflated after every fifth chest compression with 30 cm H$_2$O airway pressure of 100% oxygen using a ventilator that was controlled by the microprocessor, which controlled the Thumper.

**Statistical Analysis**

Data were analyzed with two-way analysis of variance with repeated measures over time. When the F ratio for within-groups effects was significant, linear contrasts were made between values before cardiac arrest and CPR values using the multivariate general linear model. When the F ratio for between-groups effects was significant, one-way analysis of variance was performed at each time point and differences among groups were compared by Tukey’s honestly significant difference test with Kramer’s modification. Statistical significance was set at $p<0.05$.

**Results**

There were no significant differences in weight, anteroposterior and lateral diameters, thoracic index (anteroposterior diameter divided by lateral diameter), chest circumference at the nipple line, or distance from the suprasternal notch to the xiphoid process between the three groups of piglets. The thoracic index was not significantly in excess of unity in any group.

**Brain**

Cerebral perfusion pressure (mean aortic minus mean sagittal sinus pressure) was similar among groups for the first 10 minutes of CPR (see Figure 1). With prolonged CPR, perfusion pressure declined in the 60% duty cycle group because of a decline in mean aortic pressure, whereas it was well maintained at a significantly higher level in both groups subjected to 30% duty cycle compressions. This difference in cerebral perfusion was not dependent on compression rate. Likewise, total cerebral blood flow was similar among groups during early CPR, but was better maintained during prolonged CPR in both 30% duty cycle groups in comparison with the 60% duty cycle group (see Figure 2). Flow did not differ by compression rate within the two 30% duty cycle groups. Cerebral oxygen extraction increased in all groups during CPR, but did not differ between
FIGURE 1. Graphs showing mean aortic pressure (open rectangles), mean sagittal sinus pressure (closed diamonds), and mean cerebral perfusion pressure (shaded area) before cardiac arrest and during cardiopulmonary resuscitation (CPR) with compression rate 150/min, duty cycle 30% (group I), compression rate 100/min, duty cycle 30% (group II) and 60% (group III), respectively. Values are mean ± SEM. In group III, the cerebral perfusion pressure was significantly decreased when compared with groups I and II at 20, 35, and 50 minutes of CPR (*p<0.05).

groups. Cerebral oxygen uptake was better maintained in both 30% duty cycle groups compared with the 60% duty cycle group by 10 minutes of CPR and remained preserved throughout 50 minutes of CPR (see Figure 2).

Regional cerebral blood flow values are available in tabular form from the authors. When CPR was conducted with 30% duty cycle at either compression rate, flows to many regions remained unchanged from values before cardiac arrest, whereas flow in all regions deteriorated below values before cardiac arrest during 60% duty cycle CPR. By 35 minutes of CPR, flow to most cortical regions was significantly lower in the 60% duty cycle group compared with either 30% duty cycle group. There were no significant regional flow differences between animals subjected to 30% duty cycle at 100 compressions per minute and 150 compressions per minute.

Heart

Pressures during diastole (before cardiac arrest) or the relaxation phase (CPR) were used to calculate the myocardial perfusion gradient (aortic minus right atrial pressure). Aortic pressure was significantly lower in piglets subjected to 60% duty cycle compared with either of the 30% duty cycle groups by 10 minutes of CPR and thereafter (see Figure 3). Similarly, left ventricular blood flow was better maintained with 30% duty cycle after 10 minutes of CPR (see Figure 4). The left ventricular subendocardial to subepicardial transmural flow ratio did not differ between groups, did not significantly fall below 1.0, and did not change significantly over the 50 minutes of CPR (1.15 before cardiac arrest, 1.17 at 50 minutes of CPR).

Regional myocardial blood flows are available in tabular form from the authors. In all regions and groups, flows were significantly decreased at all times during CPR when compared with values before cardiac arrest. By 10–35 minutes, however, flows were lower in most regions in the 60% duty cycle group compared with the 30% duty cycle groups. There were no significant regional flow differences between animals subjected to 30% duty cycle at 100 compressions per minute and 150 compressions per minute.
In all regions except the right atrium, blood flow exceeded 25 ml·min⁻¹·100 g⁻¹ for 35 minutes with 30% duty cycle, markedly better than with 60% duty cycle. This amount of flow would be expected to permit myocardial resuscitation following CPR.\textsuperscript{7,16}

**Other Organs**

Blood flow to the masseter muscle, tongue, skin, kidney, small intestine, and hind limb muscle decreased markedly in all three groups of animals during CPR (data not shown). There were no significant differences between the three groups.

**Blood Analyses**

There were no significant differences between groups with respect to arterial pH, PaCO\textsubscript{2}, PaO\textsubscript{2}, arterial O\textsubscript{2} content, or hemoglobin level (see Table 1). Sagittal sinus pH, sagittal sinus PCO\textsubscript{2}, and sagittal sinus O\textsubscript{2} content were significantly worse by 20–35 minutes during CPR conducted with 60% duty cycle compared with CPR conducted with 30% duty cycle and 150 compressions per minute. In all three groups, hemoglobin and arterial O\textsubscript{2} content decreased significantly from values before cardiac arrest by 10 minutes of CPR despite the continuous blood autotransfusion administered during CPR.

**Intrathoracic Compression Pressures and Chest Geometry**

Compression force was initially set to achieve a fractional pulsatile piston movement of 20%, and was not changed after the initial setting. Force remained constant during the experiment and did not differ between groups. In addition, aortic and right atrial peak compression pressures did not significantly differ between groups at any time during CPR. In the 60% duty cycle group, aortic peak pressure decreased significantly at 35 and 50 minutes from the value at 5 minutes, whereas right atrial peak pressure remained unchanged (tabular data available on request). Figure 5 shows representative vascular and airway pressure tracings at 5 minutes and 50 minutes of CPR at 60% duty cycles. The aortic and right atrial waveforms did not change shape between 5 minutes and 50 minutes of CPR, maintaining an essentially square wave configuration.

Pulsatile piston movement was adjusted to 20% of anteroposterior diameter before cardiac arrest at onset of CPR and remained constant during CPR in all three groups. During CPR, the relaxed position of the sternum became deformed from the dimension before cardiac arrest because of incomplete recoil of the chest wall in all three groups. Deformation increased over time, and by 35 minutes there were significant differences between groups. The least deformation developed in animals subjected to 30% duty cycle and 100 compressions per minute (20.2±2% of control diameter at 50 minutes), while the most severe deformation developed in animals subjected to 60% duty cycle (33.8±2% of control diameter at 50 minutes). Animals given 150 compressions per minute at 30% duty cycle

**Figure 3.** Graphs showing diastolic (relaxation phase) aortic pressure (open rectangles), right atrial pressure (closed ovals), and myocardial perfusion pressure (shaded area) before cardiac arrest and during cardiopulmonary resuscitation (CPR) with compression rate 150/min, duty cycle 30% (group I), and compression rate 100/min, duty cycle 30% (group II) and 60% (group III), respectively. Values are mean±SEM. In group III, the myocardial perfusion pressure was significantly decreased when compared with groups I and II at 10, 20, 35, and 50 minutes of CPR (*p<0.05).

**Figure 4.** Graph showing left ventricular blood flow (ml·min⁻¹·100 g⁻¹) before cardiac arrest and during cardiopulmonary resuscitation (CPR) with compression rate 150/min, duty cycle 30% (squares), and compression rate 100/min, duty cycle 30% (circles) and 60% (triangles), respectively. Values are mean±SEM. Left ventricular blood flow was significantly decreased with 60% duty cycle compared with either 30% duty cycle group at 10, 20, 35, and 50 minutes of CPR (*p<0.05).
TABLE 1. Blood Gas Analyses

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<th>Table 1. Blood Gas Analyses</th>
<th>Before cardiac arrest</th>
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<tr>
<td></td>
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<td><strong>Arterial blood gases</strong></td>
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<tr>
<td>pH</td>
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<td><strong>Pao2</strong> (mm Hg)</td>
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<td>Group 2</td>
<td>136±13</td>
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<td>214±15</td>
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<td><strong>Paco2</strong> (mm Hg)</td>
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<td><strong>CaO2</strong> (ml O2/dl)</td>
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Arterial and sagittal sinus blood gas values during cardiopulmonary resuscitation with compression rate 150/min, duty cycle 30% (group 1), and compression rate 100/min, duty cycle 30% (group 2) and 60% (group 3), respectively. Values are mean±SEM.

*Denotes deterioration from value before cardiac arrest, p<0.05; †denotes p<0.05 between group 1 and group 3. There were no other intergroup differences.

developed an intermediate amount of deformation (27.0±2% of control diameter at 50 minutes). Despite this deformation during CPR, there were no rib fractures noted at necropsy.

**Discussion**

In this study, we have demonstrated that 1) cerebral and myocardial blood flow are better maintained during 50 minutes of cardiopulmonary resuscitation with 30% duty cycle compared with 60% duty cycle; 2) maintenance of blood flow with 30% duty cycle is similar with 100 or 150 compressions per minute; 3) cerebral oxygen uptake and sagittal sinus blood gas values are better preserved with 30% duty cycle compared with 60% duty cycle; and 4) chest deformation develops over prolonged CPR but is lessened with 30% duty cycle when compared with 60% duty cycle.

Blood flow to the myocardium exceeded 25 ml·min⁻¹·100 g⁻¹ in all regions except the right atrium for as long as 35 minutes when 30% duty cycle
was used. With 60% duty cycle, the left ventricular free wall flow was 2 ml · min⁻¹ · 100 g⁻¹ after 35 minutes of CPR as diastolic coronary perfusion pressure fell below 20 mm Hg. Return of spontaneous circulation generally does not occur in adult humans when coronary perfusion pressure is below 20 mm Hg, a threshold that in adult dogs subjected to 1 hour of CPR corresponds to a left ventricular blood flow of 20 ml · min⁻¹ · 100 g⁻¹. Thus, the differences observed in this study between 30% and 60% duty cycle CPR would be expected to have direct impact on successful cardiac resuscitation after prolonged CPR.

Cerebral blood flow was better preserved with 30% duty cycle than with 60% duty cycle. This resulted in sustained cerebral oxygen uptake in the 30% duty cycle groups, consistent with preservation of cerebral metabolism and function. This major difference in perfusion between 30% and 60% duty cycle CPR would likely result in better neurologic outcome after prolonged CPR.

The mechanism of blood flow during CPR remains unclear, with data in support of either the intrathoracic pump model or the direct cardiac compression model. The controversy seems important because the two mechanisms can be accentuated by different interventions. The thoracic pump mechanism is augmented by prolonging compression duration, whereas the cardiac compression mechanism is augmented by increasing the rate of compression. Our results do not completely support either mechanism. If the thoracic pump were the predominant mechanism of blood flow generation, then 60% duty cycle should have resulted in superior cerebral perfusion when compared with 30% duty cycle. However, we observed no differences during early CPR and the opposite result during prolonged CPR. Because coronary blood flow occurs primarily during chest relaxation, the optimum duty cycle for coronary flow is probably less than 60%. It is also possible that the optimum duty cycle for cerebral blood flow lies between 30% and 60%. In adult humans who could not be resuscitated after prolonged CPR, however, Swenson noted that high-impulse, high-rate CPR resulted in better aortic pressure generation than conventional CPR. Our data are consistent with those results. If cardiac compression is the major mechanism of blood flow, then increasing the compression rate should have improved perfusion. Our data show no differences between 100 and 150 compressions per minute at 30% duty cycle. The lack of differences in early CPR can be reconciled with either pump mechanism by considering the impact on the relaxation time of the cycle. For example, regardless of which model is operative during CPR, one can consider a generic pump that has a time constant for ejection (τₑ) and refilling (τᵣ). Compression duration is not the only constraint on this pump; if the pump has inadequate refill time then forward flow will decrease. At a rate of 100 · min⁻¹ and a 30% duty cycle, relaxation time is 420 msec. Increasing the rate to 150 · min⁻¹ decreases relaxation time to 280 msec, whereas increasing duty cycle to 60% decreases relaxation time to 240 msec. Total relaxation time
(and therefore refilling time) per minute is unchanged by increasing the rate (42 sec \cdot min^{-1}) but drops to 24 sec \cdot min^{-1} when the duty cycle is increased; hence, the lack of improved perfusion during early CPR by increasing the rate or duty cycle is probably due to the brief filling time counterbalancing further emptying of the pump. Therefore, at the recommended rate of 100 compressions per minute for infant CPR, 30% duty cycle appears to be close to the optimum duty cycle. We do not believe that duty cycles shorter than 30% are advantageous because we have found poor levels of cerebral perfusion pressure when the duty cycle is reduced to 10–20% for brief periods, consistent with observations in adult humans.23

We do not have blood flow data in animals subjected to CPR with duty cycles between 30% and 60%, and it is possible that the optimal duty cycle lies between these two choices. Perfusion pressure data from our previous studies14 suggest a continuum of values between these two duty cycles, however, and we do not believe we have missed a more optimal, intermediate duty cycle.

With prolonged CPR, the deterioration in aortic pressure may be caused by a variety of mechanisms that can be divided into three categories: a change in the characteristics of the pump, a change in \( \tau_c \), or a change in \( \tau_r \). The characteristics of the pump would change if applied force or cyclic piston displacement decreased. However, neither of these changed during CPR, nor was there a significant difference between 30% and 60% duty cycle groups. Generated intrathoracic pressure would decrease if there was lateral flailing, bulging of the chest wall, or decreased lung volume.17 Decreased lung volume might be anticipated with 60% duty cycle, particularly during prolonged CPR. However, there was no significant change in peak right atrial pressure during CPR in the 60% duty cycle group, suggesting that intrathoracic pressure generation was unchanged. Figure 5 demonstrates that aortic, right atrial, and sagittal sinus pressure tracings were negligibly affected by interposed ventilations at 5 and 50 minutes during 60% duty cycle compressions; this suggests that decreased lung volume is not a major contributor to the deterioration we have seen with 60% duty cycle compressions. Chest deformation might decrease generated intrathoracic pressure, but we observed significantly more chest deformation at a compression rate of 150 per minute than at 100 per minute with 30% duty cycle; there was no difference between these groups in right atrial, aortic, or sagittal sinus pressures. In addition, myocardial and cerebral perfusion were not different between the 30% groups.

A second category of potential mechanisms involves time-dependent changes in \( \tau_r \) which in turn depends largely on arterial impedance. Although epinephrine was continuously infused, it is conceivable that repetitive long duty cycle compressions accelerate the loss of vascular tone in hypoxic tissue of the immature pig faster than short duty cycle compressions. The decline in peak aortic pressure without a decline in peak right atrial pressure (reflecting peak intrathoracic pressure) during prolonged CPR with 60% duty cycle supports this possibility. However, the aortic pressure waveform generally maintained a square wave shape even at 50 minutes (see Figure 5), suggesting that a significant arterial runoff was not present. Since the right atrial pressure and waveform did not deteriorate with time, this category is unlikely.

The third category involves time-dependent increases in \( \tau_r \) which depend on peripheral venous tone, blood volume, and the gradient for venous return. An increase in \( \tau_r \) would have the greatest impact in the 60% duty cycle group because this group has the shortest relaxation time. In addition, the long duty cycle itself may induce changes in \( \tau_r \) when CPR is prolonged by several potential mechanisms. First, repetitive distension of peripheral veins at long duty cycles may lead to greater mechanical creep and an increase in the unstressed venous volume. Second, the longer duty cycle may increase capillary hydrostatic pressure leading to plasma filtration in peripheral vascular beds. Third, the progressively greater chest deformation with 60% duty cycle may cause distortion of intrathoracic vessels and increase the resistance for venous return. Fourth, the loss of chest recoil may lead to greater intrathoracic pressure during the relaxation phase of the cycle and thereby decrease the gradient for venous return. Time-dependent increases in \( \tau_r \) would result in lower pump stroke volume. Decreased peak aortic pressure with preservation of peak right atrial pressure is consistent with this, since decreased ejection volume (from increased \( \tau_r \)) with a fixed arterial impedance would produce these findings. During early CPR, flow would be similar between 30% and 60% duty cycle groups but as \( \tau_r \) increased, filling time would become the limiting factor for flow generation in the 60% duty cycle group. We believe that this is the most likely explanation for the deterioration we observed during 60% duty cycle CPR.

The controversy between the cardiac compression and intrathoracic pump mechanisms becomes less important when it is considered that either mechanism must have properties of this generic pump.22 Clinical recommendations must assure that compression duration is adequate (given a particular \( \tau_c \)) to maximize output but does not limit relaxation time (given the corresponding \( \tau_r \)). Further, since \( \tau_r \) may become prolonged during CPR, different recommendations may be needed for prolonged CPR, as is commonly encountered in the pediatric setting. Finally, most of the coronary blood flow occurs during the relaxation phase of the CPR cycle. Models indicate that the optimum duty cycle for coronary flow is about 30–40%, consistent with results observed in humans.21 The higher coronary flow with 30% duty cycle in our study agrees with this prediction.

This swine model was developed to provide a suitable laboratory model of pediatric CPR. The
piglet was chosen because of its relatively compliant chest and because the thoracic index is more similar to humans than is that of most other animals, including dogs. The chest size, geometry, and stiffness are similar to those of human infants. We are able to produce good blood flow in this model with conventional CPR, setting it apart from other animal models in which conventional CPR yields poor results. We can therefore use this model to study possible improvements in conventional CPR used in the clinical setting or to examine experimental methods such as simultaneous compression and ventilation CPR.

We have demonstrated that 30% duty cycle provides markedly superior myocardial and cerebral perfusion when CPR is prolonged beyond 10 minutes in 2-week-old piglets. This animal model is similar in chest compliance, chest geometry, and behavior during conventional CPR to human infants and children. Loss of vital organ blood flow during prolonged CPR with a 60% duty cycle may be caused by several mechanisms associated with greater chest distortion, but our data support an increased $\tau$ as the predominant limitation of blood flow in this model. We suggest that blood flow during CPR in this model is primarily limited by venous return, not compression duration. Our results do not support prolonging compression duration beyond 30% duty cycle at the currently recommended rate of 100 compressions per minute in CPR of infants and children, nor do they support further increases in rate above 100 compressions per minute.

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