0031-3998/89/2606-0558$02.00/0
PEDIATRIC RESEARCH

Vol. 26, No. 6, 1989
Printed in U.S.A.

Blood Flow during Cardiopulmonary Resuscitation with Simultaneous Compression and Ventilation in Infant Pigs

IVOR D. BERKOWITZ, TEERACHAI CHANTAROJANASIRI, RAYMOND C. KOEHLER, CHARLES L. SCHLEIEN, J. MICHAEL DEAN, JOHN R. MICHAEL, MARK C. ROGERS, AND RICHARD J. TRAYSTMAN

The Department of Anesthesiology/Critical Care Medicine, The Johns Hopkins Medical Institution, Baltimore, Maryland 21205

ABSTRACT. We determined whether the simultaneous chest compression and ventilation (SCV) technique of cardiopulmonary resuscitation (CPR) enhances cerebral (CBF) and myocardial (MBF) blood flows and cerebral O_2 uptake in an infant swine model of CPR as it does in most adult animal CPR models. We also tested whether SCV-CPR sustains CBF and MBF for prolonged periods of CPR when these flows ordinarily deteriorate. CPR was performed in two groups ($n = 8$) of pentobarbital anesthetized piglets (3.5–5.5 kg) with continuous epinephrine infusion (10 μg/kg/min). Conventional CPR was performed at 100 compressions/min, 60% duty cycle, 1:5 breath to compression ratio and 25–30 mm Hg peak airway pressure. SCV-CPR was performed at 60 compressions/min, 60% duty cycle and 60 mm Hg peak airway pressure applied during each chest compression. Peak right atrial and aortic pressures in excess of 80 mm Hg were generated during CPR in both groups. At 5 min of conventional and SCV-CPR, MBF was 38 ± 7 and 46 ± 7 mL·min^{-1}·100 g^{-1} (±SE), respectively, and CBF was 15 ± 3 and 13 ± 2 mL·min^{-1}·100 g^{-1}, respectively. However, as CPR was prolonged to 50 min, the sternum progressively lost its recoil and the chest became more deformed. Lung inflation at high airway pressure with SCV-CPR did not prevent this chest deformation. Aortic pressure gradually declined, whereas right atrial and intracranial pressure remained constant in both groups. Consequently, MBF and CBF fell less than 10 mL·min^{-1}·100 g^{-1} and cerebral O_2 uptake was markedly impaired during prolonged conventional and SCV-CPR. Therefore, SCV-CPR in an infant swine model does not enhance MBF and CBF during early CPR because intrathoracic pressure generation is already high with conventional CPR as reflected by the high right atrial pressure. In addition, SCV-CPR does not prevent the progressive chest deformation and the subsequent decline in CBF and MBF when CPR is prolonged, as is often required in pediatric resuscitation. (*Pediatr Res* 26: 558–564, 1989)

Abbreviations

CPR, cardiopulmonary resuscitation
SCV-CPR, simultaneous compression and ventilation cardiopulmonary resuscitation
CBF, cerebral blood flow
MBF, myocardial blood flow

Received October 5, 1988; accepted July 27, 1989.
Correspondence and reprint requests to Ivor D. Berkowitz, M.D., Department of Anesthesiology/Critical Care Medicine, The Johns Hopkins Hospital, 600 North Wolfe Street—Halsted 842, Baltimore, MD 21205.
Supported by USPHS NIH Grant NS20020.

The hypothesis that antegrade blood flow during closed chest CPR is due not to direct cardiac compression but to phasic increases in intrathoracic pressure (1, 2) has led to the investigation of alternative techniques of CPR in adult animals (3–5). According to this thoracic pump mechanism, external chest compression produces a generalized elevation of intrathoracic pressure that is transmitted to all thoracic vascular structures. Pressure in intrathoracic arteries is transmitted to extrathoracic arteries, but jugular venous valves and possibly collapse of veins at the thoracic inlet prevent full transmission of intrathoracic pressure to the extrathoracic veins. Consequently, uneven transmission of intrathoracic pressure to the extrathoracic arterial and venous system provides a gradient for antegrade blood flow during chest compression (1, 2).

In large adult dogs, conventional CPR using sternal compression generates low intrathoracic and vascular pressures, and correspondingly low CBF and MBF. However, changing to SCV-CPR generates higher intrathroacic and vascular pressures (3) and enhances cerebral and myocardial perfusion (4, 6), particularly when epinephrine is continuously infused (7). In man, SCV-CPR also increases blood pressure and carotid artery blood flow (8).

Less experimental data are available in pediatric animal models of CPR (9). In 6- to 12-wk-old dogs, cerebral blood flow levels with conventional CPR are reported to be as low as in adult dogs (10). In 2-wk-old pigs, in contrast, conventional CPR is effective in generating high levels of cerebral and myocardial blood flow, especially when epinephrine is infused (11). Age-related and possibly species differences in chest deformability and geometry permit the generation of higher intrathoracic pressure and myocardial and cerebral perfusion in immature pigs (12). These high blood flows, however, deteriorate after 35 min of CPR to levels lower than were achieved with prolonged CPR in adult dogs (11). The mechanisms of this decline are poorly understood. With prolonged CPR the compliant chest wall of the infant pig loses its recoil and progressive chest deformation develops (11, 12). This may distort intrathoracic veins and impede venous return. We postulated that lung inflation by ventilation at high airway pressure during SCV-CPR may prevent the progressive chest deformation and mitigate its interference with venous return. In addition, cyclic lung inflation itself can assist in propelling blood under certain conditions (13, 14).

The purpose of our study was to determine whether SCV-CPR

would further enhance cerebral and myocardial blood flow and cerebral O_2 uptake in an infant swine model of CPR, particularly during prolonged CPR when the conventional technique fails. Because resuscitation after cardiac arrest in children is frequently protracted (15), it is critical to be able to maintain an adequate vital organ perfusion until spontaneous circulation can be restored.

MATERIALS AND METHODS

Animal preparation. The protocol for these studies has been approved by the Animal Care and Use Committee of the Johns Hopkins Medical Institutions. All experiments were performed on 3.5 to 5.5 kg, 2-wk-old infant swine. The animals were anesthetized initially with pentobarbital (30–40 mg/kg intraperitoneally). Supplemental pentobarbital was administered intravenously as needed during surgery. Animals were ventilated via a tracheostomy by a Harvard animal ventilator (Harvard Apparatus, Co. Inc., Millis, MA), with an inspired O_2 of approximately 30%. End tidal CO_2 was monitored during the prearrest period to maintain arterial CO_2 tension ($Paco_2$) at 35–45 mm Hg. Saline-filled catheters were placed into the right atrium, thoracic aorta, and left ventricle via femoral vessel cannulation. Catheters were also placed into the axillary veins for fluid and drug administration and into the proximal subclavian artery via axillary arterial cannulation. A burr hole was made in the skull over the midline and a catheter was placed in the sagittal sinus with the tip positioned 1–2 cm anterior to the confluence of the sinus. A straight ventricular catheter (Cordis, Miami, FL) was placed through another burr hole into the lateral ventricle for intracranial pressure measurement. A bipolar electrode was placed via a femoral vein into the right heart to induce ventricular fibrillation. After the surgery was completed, pancuronium (0.2 mg/kg) was administered. Heparin (1000 U) was given before cardiac arrest was induced.

Measurements. Pressures were measured from the intrathoracic aorta, right atrium, sagittal sinus, and lateral ventricle with Statham P23Db transducers (Statham Instruments, Inc., Hato Rey, PR) referenced to the level of the right atrium. Arterial and sagittal sinus blood gases were measured on a Radiometer BMS3 electrode and analyzer system (Radiometer, Copenhagen, Denmark). Oxygen content was measured by Lex-O_2-Con fuel cell system (Lexington Instruments, Waltham, MA). Hb concentration was measured with a CO-Oximeter (model 282, Instrumentation Laboratory, Inc., Lexington, MA).

Regional blood flow was measured with microspheres labeled with ^{153}Gd, ^{51}Cr, ^{113}Sn, ^{103}Ru, ^{95}Nb, and ^{46}Sc (Dupont-New England Nuclear Products, Boston, MA). Use of the microsphere technique during CPR has been previously validated (4, 11). Approximately 1×10^6 microspheres (16 ± 0.5 μm diameter) were injected into the left ventricle for each prearrest measurement and 5×10^5 microspheres were injected for each of the postarrest measurements. Reference blood samples were withdrawn from the subclavian artery for 2 min after the control injection and for 4.5 min after each postarrest microsphere injection. The reference sample withdrawal rate was 3.8 mL/min for the prearrest measurement and 1.9 mL/min for each injection during CPR. This combination of injection doses and withdrawal rates ensured that there were at least 2000 microspheres in the reference sample before cardiac arrest was induced and at least 10 000 microspheres during CPR. Vials of blood and tissue were counted on a multichannel autogamma scintillation spectrometer (model 9042, Packard Instrument Co., Downers Grove, IL). Tissue blood flow was calculated by the standard simultaneous equation technique (16). It was assumed that the contamination of sagittal sinus blood from sources other than the brain, such as dura and skull bone is small, particularly during CPR when blood flow to these extracranial tissues is reduced (4).

At the end of each experiment, postmortem examination was performed to confirm catheter positions and to exclude the presence of a pneumothorax. The entire heart and brain were removed and tissue samples of kidney, jejunum, facial skin, facial muscle, and tongue were obtained. The heart was cut into sections of left ventricular free wall, interventricular septum, and right ventricular free wall. The brain was dissected into medulla, pons, midbrain, cerebellum and diencephalon, and cerebrum. Cerebral O_2 uptake was calculated from the arterial-sagittal sinus O_2 content difference and blood flow to the cerebrum. Cerebral fractional O_2 extraction equals the arterial-sagittal sinus O_2 content difference divided by the arterial O_2 content.

Experimental protocol. At least 90 min before CPR, 50 mL of blood was withdrawn from each animal and replaced with 200 mL of Ringer's lactate solution. This blood, diluted 1:1 with Ringer's lactate solution, was then infused at 3.4 mL/min when the reference samples were withdrawn during CPR to replace the blood volume. The animal was placed supine and secured to a V-shaped board that was attached to the baseplate of the mechanical chest compressor. Ventricular fibrillation was induced by passing a 60 Hz alternating current through the bipolar electrode in the right heart. External chest compression was begun about 15 s later with a pneumatic chest compressor (Thumper, Michigan Instruments, Grand Rapids, MI) equipped with a pediatric chest pad. This pad (8.0×5.5 cm) has a solid rubber hemisphere (3 cm diameter) molded in its center to apply the force over a smaller surface area than the adult pad. The pad was centered over the lower half of the sternum, 3–4 cm cephalad to the xiphoid. A pressure-limited ventilator was used during CPR. The pneumatic chest compressor and ventilator were synchronized by a microprocessor controller.

Animals were randomly assigned to conventional CPR or SCV-CPR groups. In the conventional CPR group ($n = 8$), CPR was performed at a compression rate of 100/min with a 60% duty cycle. Ventilation with 100% oxygen and peak airway pressures of 25–30 mm Hg was interposed after every fifth chest compression. In the SCV-CPR group ($n = 8$), chest compression was performed at 60 compressions/min with a 60% duty cycle. Ventilation was simultaneous with chest compression and peak airway pressures was set at 60 mm Hg. A ventilating gas mixture of 95% O_2–5% CO_2 was used in an attempt to maintain $Paco_2$ at comparable levels with the conventional CPR group. Applied chest force prevented overdistention of the lung at high airway pressures during SCV-CPR. In preliminary experiments, SCV-CPR performed at rates more than 60/min or at peak airway pressures greater than 60 mm Hg often caused pneumothoraces. In both CPR groups, the force of the Thumper was adjusted initially to produce a 20% displacement of the animals' baseline anteroposterior chest diameter, measured at the lower half of the sternum. Once this displacement had been achieved, no further force adjustments were made during the 55 min of continuous CPR. Force was measured by a strain gauge on the Thumper and piston displacement was measured by a sliding potentiometer built into the Thumper.

To better maintain perfusion pressures during CPR, each animal received a 40 μg/kg bolus of epinephrine through the left ventricular catheter at the onset of CPR, followed by a continuous right atrial infusion of 10 μg/kg/min epinephrine in saline. The rate of this infusion was 3.4 mL/min. Blood flow, pressure, and blood gas measurements were made pre-arrest and at 5, 10, 20, 35, and 50 min of CPR.

Statistical analysis. Data were analyzed with two-way analysis of variance with repeated measures over time during CPR. Mean values were compared by the Duncan new multiple-range test. Prearrest values were not included in the analysis because the variance of most measurements differed considerably from the variance during CPR. Prearrest values were compared between groups with the unpaired test. Statistical significance was set at $p < 0.05$. Values are presented as mean ±SEM.

RESULTS

Original records obtained during conventional CPR with intermittent ventilation and during SCV-CPR with high airway pressure are shown in Figure 1. Sternal displacement (the difference between the position of the Thumper piston at end-compression or "systole" and end relaxation or "diastole") was set at 20% of the initial anteroposterior chest diameter and remained unchanged for the duration of CPR. The chest did not completely recoil during the relaxation phase. The difference between the end-expiratory position of the Thumper pad resting on the sternum before arrest and the position at end-relaxation during CPR is defined as chest deformity. This increased progressively over the course of CPR with both types of CPR. Right atrial systolic pressure, a reflection of intrathoracic pressure, remained unchanged for the duration of CPR with both CPR modalities. The aortic systolic pressure, in contrast, progressively declined over time. Sagittal sinus pressure and intracranial pressure increased phasically with each chest compression, but to a lesser extent than the right atrial systolic pressure.

In the conventional CPR and SCV-CPR groups, cyclic sternal displacement was 19.4 ± 0.4 and 21 ± 0.8% of the baseline anteroposterior chest diameter, respectively. The corresponding compression force was 241 ± 20 and 232 ± 22 N. Chest deformity was 14 ± 2 and 13 ± 2% of baseline anteroposterior diameter at 5 min of CPR in the respective groups, and it increased 21 ± 2 and 20 ± 3%, respectively, at 50 min of CPR. There was no difference in sternal displacement, chest deformity or compression force at any time point between groups. Before arrest, chest shape as measured by the ratio of the anteroposterior to lateral chest diameter was similar in the conventional CPR (0.95 ± 0.03) and SCV-CPR (0.92 ± 0.02) groups.

Peak right atrial pressure was 83 ± 10 and 101 ± 6 mm Hg at 5 min and 81 ± 8 and 96 ± 10 mm Hg at 50 min in the conventional CPR and SCV-CPR groups, respectively. There was no augmentation at any time in peak right atrial pressure in the SCV-CPR group compared with the conventional CPR group and this pressure remained unchanged for the duration of CPR in both groups (Fig. 2). Peak aortic pressure, however, gradually declined to the same extent in each group with prolonged CPR (Fig. 2). At 5 min the aortic systolic pressure was 88 ± 9 and 101 ± 5 mm Hg in the conventional and SCV-CPR groups but declined to 64 ± 6 and 81 ± 8 at 50 min in the respective groups. There was no difference between the two groups. Aortic pressure during the relaxation phase declined progressively during the course of CPR from 37 ± 4 to 10 ± 2 with conventional CPR and from 32 ± 2 to 14 ± 3 mm Hg with SCV-CPR. Right atrial pressure during the relaxation phase remained unchanged. Thus, the pressure gradient for coronary flow declined during the course of CPR in both groups (*shaded area* in Fig. 2).

Substantial levels of total myocardial blood flow were obtained over the first 10 min of CPR, but flow declined when CPR was prolonged in both groups (Fig. 3). Blood flow to the left ventricle, right ventricle and septum all responded similarly (Table 1). There were no differences at any time point in total or regional myocardial blood flow between the CPR groups.

Total brain blood flow decreased progressively over the dura-

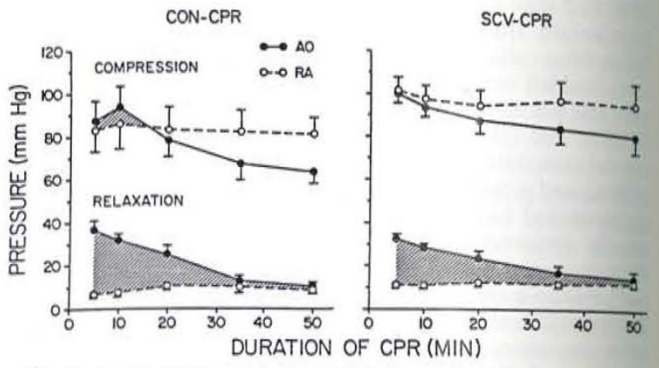

Fig. 2. Aortic (*AO*) and right atrial (*RA*) systolic (compression phase) and diastolic (relaxation phase) pressures during 50 min of conventional (*CON*) (*left*) and simultaneous compression and ventilation (*SCV*) CPR (*right*). *Shaded areas* reflect magnitude of positive aortic-right atrial pressure gradients for antegrade MBF.

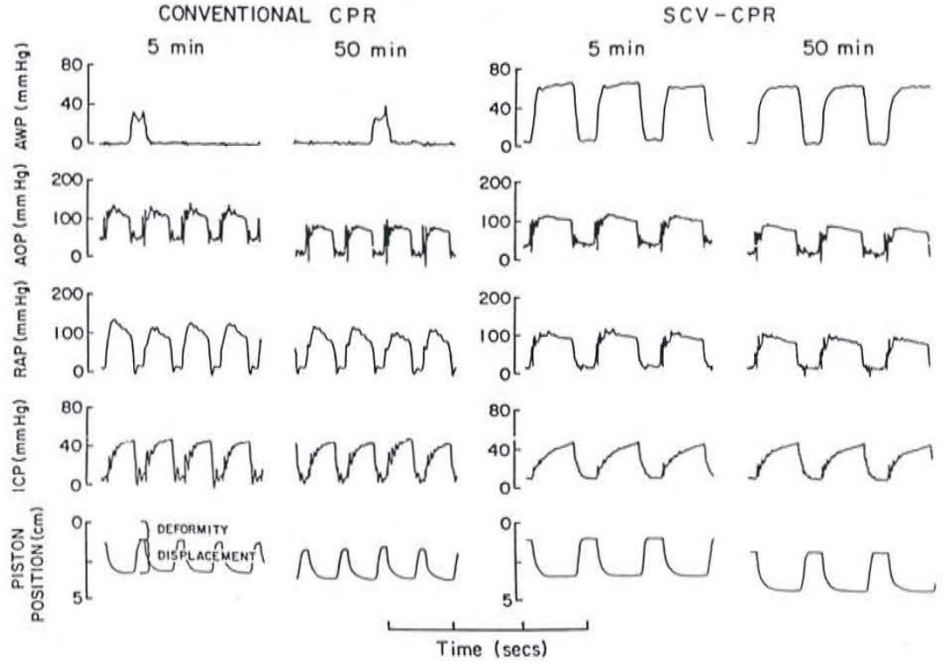

Fig. 1. Representative experimental tracings of airway pressure (*AWP*), aortic pressure (*AOP*), right atrial pressure (*RAP*), intracranial pressure (*ICP*), and piston position at 5 and 50 min of conventional and simultaneous compression and ventilation (*SCV*) CPR. Chest deformity is the difference between the position of the Thumper pad resting on the sternum before arrest (0 cm) and the position at end relaxation. AOP declined with prolonged CPR with both CPR modes, yet RAP, ICP and sternal displacement remained unchanged. Progressive chest deformity developed in both CPR groups with prolonged CPR.

tion of CPR and to the same extent in both CPR groups (Fig. 4). There was no difference between groups in regional blood flow to the cerebellum, pons, midbrain, or cerebrum. At 10 min of CPR, however, flow to the medulla was greater in the SCV-CPR group than in the conventional CPR group. Blood flow to infratentorial regions declined to a lesser extent than supratentorial regions in both groups during CPR (Table 1).

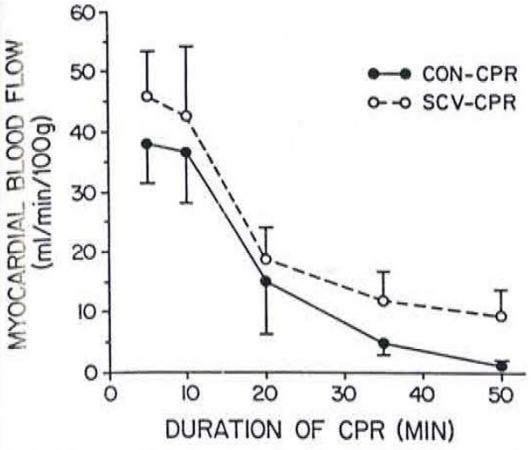

Fig. 3. MBF during 50 min of conventional (*CON*) and simultaneous compression and ventilation (*SCV*) CPR.

There were no differences between the CPR groups in arterial P_{O_2}, pH, Hb concentration, and oxygen saturation during CPR (Table 2). Arterial P_{CO_2}, however, was higher in the SCV-CPR group. In both groups, cerebral fractional O_2 extraction increased to near-maximum levels throughout CPR, but cerebral O_2 uptake fell progressively during CPR (Table 2). Cerebral O_2 uptake was not different between groups, except at 5 min when the value in the conventional CPR group was higher than in the SCV-CPR group. Mean intracranial pressure and mean sagittal sinus pres-

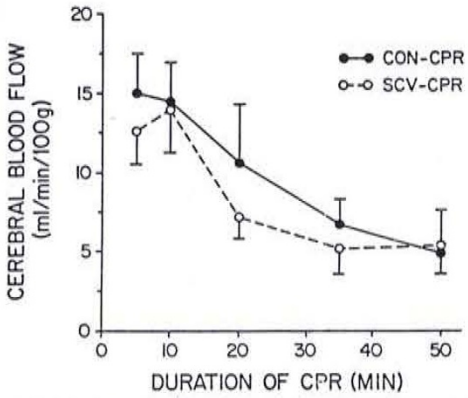

Fig. 4. CBF during 50 min of conventional (*CON*) and simultaneous compression and ventilation (*SCV*) CPR.

Table 1. *Regional blood flow (mL·min^{-1}·100 g^{-1}) before cardiac arrest and during prolonged CPR**

	Group	Before arrest	5 min	10 min	20 min	35 min	50 min
Brain							
Cerebrum	I	50 ± 8	13 ± 3	11 ± 2	8 ± 3	4 ± 2	2 ± 1
	II	53 ± 5	9 ± 2	9 ± 2	4 ± 1	2 ± 1	3 ± 1
Cerebellum	I	65 ± 8	22 ± 2	24 ± 4	17 ± 5	12 ± 3	8 ± 2
	II	72 ± 7	23 ± 4	27 ± 6	15 ± 3	10 ± 3	10 ± 4
Medulla	I	56 ± 7	22 ± 2	25 ± 2	26 ± 5	26 ± 3	21 ± 4
	II	71 ± 13	38 ± 6	48 ± 12†	31 ± 11	24 ± 8	23 ± 8
Pons	I	57 ± 11	20 ± 2	21 ± 3	18 ± 3	18 ± 3	14 ± 3
	II	54 ± 9	24 ± 5	31 ± 7	22 ± 5	18 ± 6	17 ± 6
Midbrain	I	62 ± 11	22 ± 2	22 ± 3	16 ± 4	14 ± 4	10 ± 3
	II	64 ± 9	18 ± 3	20 ± 3	12 ± 3	11 ± 4	12 ± 5
Diencephalon	I	46 ± 8	16 ± 2	16 ± 2	11 ± 4	9 ± 2	6 ± 1
	II	47 ± 5	12 ± 2	13 ± 2	8 ± 2	7 ± 2	8 ± 3
Heart							
Left ventricle	I	224 ± 43	44 ± 8	44 ± 11	15 ± 7	5 ± 2	2 ± 1
	II	271 ± 56	51 ± 9	46 ± 13	20 ± 7	13 ± 6	8 ± 5
Right ventricle	I	220 ± 43	44 ± 7	40 ± 7	16 ± 8	5 ± 2	2 ± 1
	II	233 ± 37	55 ± 9	54 ± 15	25 ± 8	14 ± 6	13 ± 6
Septum	I	277 ± 76	35 ± 7	33 ± 9	13 ± 9	4 ± 2	1 ± 1
	II	247 ± 44	43 ± 7	38 ± 11	16 ± 7	11 ± 5	8 ± 4
Cephalic tissues							
Tongue	I	6 ± 1	0.5 ± 0.1	0.5 ± 0.2	0.7 ± 0.2	0.9 ± 0.2	1.1 ± 0.2
	II	7 ± 1	0.3 ± 0.1	0.4 ± 0.1	0.5 ± 0.2	0.9 ± 0.2	0.8 ± 0.4
Facial skin	I	3 ± 1	0	0	0	0	0
	II	3 ± 1	0	0	0	0	0
Facial muscle	I	5 ± 1	0.1 ± 0.1	0.1 ± 0.1	0.2 ± 0.1	0.2 ± 0.1	0.3 ± 0.1
	II	5 ± 1	0.1 ± 0.1	0.1 ± 0.1	0.1 ± 0.1	0.1 ± 0.1	0.2 ± 0.1
Visceral organs							
Kidney	I	140 ± 17	0.1 ± 0.1	0	0.1 ± 0.1	0	0
	II	135 ± 18	0	0	0	0	0
Jejunum	I	37 ± 8	2.4 ± 0.7	3.3 ± 0.7	3.7 ± 1.4	1.6 ± 0.7	0.9 ± 0.4
	II	35 ± 5	1.3 ± 0.6	4.1 ± 2	3.0 ± 1	1.3 ± 0.5	0.5 ± 0.2

*Group I, conventional CPR; group II, SCV-CPR.
†$p < 0.05$ *versus* group I value.

Table 2. *Blood analyses, cerebral O_2 uptake, intracranial, and sagittal sinus pressures**

	Group	Before arrest	5 min	10 min	20 min	35 min	50 min
Arterial P_{O_2} (mm Hg)	I	133 ± 11	207 ± 30	171 ± 33	142 ± 32	120 ± 24	88 ± 16
	II	101 ± 5	283 ± 24	202 ± 32	191 ± 39	176 ± 40	163 ± 46
Arterial P_{CO_2} (mm Hg)	I	39 ± 1	20 ± 3	27 ± 3	32 ± 6	40 ± 7	52 ± 9
	II	39 ± 1	38 ± 6†	44 ± 7	59 ± 7†	72 ± 9†	89 ± 10†
Arterial pH	I	7.38 ± 0.01	7.53 ± 0.05	7.29 ± 0.05	7.08 ± 0.05	6.91 ± 0.05	6.73 ± 0.05
	II	7.39 ± 0.01	7.38 ± 0.01	7.17 ± 0.09	6.92 ± 0.05	6.77 ± 0.04	6.62 ± 0.04
Arterial Hb (g/dL)	I	10.2 ± 0.7	9.7 ± 0.6	8.4 ± 0.4	7.3 ± 0.5	6.2 ± 0.6	4.5 ± 0.4
	II	8.3 ± 0.8	8.6 ± 1.1	7.5 ± 1.1	6.3 ± 0.9	5.3 ± 0.8	4.1 ± 0.6
Arterial O_2 saturation (%)	I	100 ± 1	96 ± 4	97 ± 3	93 ± 3	85 ± 7	76 ± 9
	II	98 ± 1	100 ± 1	95 ± 5	91 ± 6	87 ± 7	78 ± 1
Arterial O_2 content (mL/dL)	I	13.9 ± 0.8	14 ± 1.2	11.9 ± 0.8	9.8 ± 0.9	7.6 ± 1.0	5.0 ± 0.8
	II	11.1 ± 1.0	12.3 ± 1.5	10.4 ± 1.6	7.8 ± 1.1	6.7 ± 1.1	4.7 ± 0.7
Sagittal sinus O_2 content (mL/dL)	I	8.5 ± 0.8	1.8 ± 0.3	1.5 ± 0.3	1.2 ± 0.3	1.1 ± 0.4	0.6 ± 0.2
	II	6.1 ± 0.8	1.8 ± 0.4	1.3 ± 0.3	1.2 ± 0.3	1.0 ± 0.4	0.7 ± 0.2
Cerebral O_2 uptake (mL O_2/min/100 g)	I	2.5 ± 0.3	1.4 ± 0.3	1.1 ± 0.2	0.7 ± 0.1	0.3 ± 0.1	0.1 ± 0.1
	II	2.6 ± 0.3	0.8 ± 0.1†	0.7 ± 0.2	0.3 ± 0.1	0.2 ± 0.1	0.1 ± 0.1
O_2 extraction	I	0.39 ± 0.04	0.86 ± 0.03	0.86 ± 0.03	0.87 ± 0.02	0.83 ± 0.04	0.84 ± 0.04
	II	0.45 ± 0.05	0.83 ± 0.03	0.85 ± 0.02	0.81 ± 0.03	0.87 ± 0.02	0.85 ± 0.02
Mean intracranial pressure (mm Hg)	I	5 ± 1	29 ± 4	28 ± 4	25 ± 3	24 ± 3	24 ± 3
	II	5 ± 1	27 ± 4	30 ± 3	26 ± 4	27 ± 5	26 ± 5
Mean sagittal sinus pressure (mm Hg)	I	5 ± 1	32 ± 5	32 ± 5	29 ± 2	30 ± 2	30 ± 2
	II	6 ± 1	28 ± 3	33 ± 3	32 ± 3	33 ± 4	32 ± 5

* Group I, conventional CPR; group II, SCV-CPR.
† $p < 0.05$ *versus* group 1 value.

sure averaged over the CPR cycle were equivalent (Table 2). These pressures remained unchanged during prolonged CPR and were not different between groups.

DISCUSSION

The major findings of this study are 2-fold. First, in contrast with studies in large adult dogs, SCV-CPR does not increase intrathoracic vascular pressures, and does not enhance either cerebral perfusion and oxygen uptake or myocardial perfusion when compared with conventional CPR. Second, SCV-CPR does not sustain cerebral and myocardial blood flow better than conventional CPR when CPR is extended for prolonged periods.

Several investigators have demonstrated in large dogs (>20 kg) that SCV-CPR, with peak airway pressures of 80–100 mm Hg, substantially increased intrathoracic and intravascular pressures when compared with conventional CPR (3, 4, 6). In the study by Luce *et al.* (6), SCV-CPR with ventilation at 80 mm Hg airway pressure increased intrathoracic pressure from 24 to 70 mm Hg when compared with conventional CPR. Koehler *et al.* (4) demonstrated that changing from conventional CPR to SCV-CPR with ventilation at 80–90 mm Hg airway pressure increased mean carotid arterial pressure from 27 to 64 mm Hg. However, studies with smaller dogs (7–12 kg) failed to demonstrate superiority of SCV-CPR (17, 18). In these latter studies there were no differences in the peak intrathoracic vascular pressures that were achieved by the two CPR modalities performed with conventional chest pads. It is notable in both these studies that intrathoracic vascular pressures generated in each CPR group exceeded the 55–70 mm Hg peak airway pressure used for the delivery of SCV-CPR.

Our investigation in piglets, the smallest animal that has been used for SCV-CPR research, also demonstrated that SCV-CPR did not increase intrathoracic pressure, as reflected by peak right atrial pressure, when compared with conventional CPR. Conventional CPR generated an intrathoracic pressure of 85 mm Hg, a value that exceeded the peak airway pressure of 60 mm Hg achieved during SCV-CPR. Frequent tension pneumothoraces with ventilation at peak airway pressures of more than 60 mm Hg precluded the use of high airway pressures in piglets. The use of SCV-CPR with peak airway pressures that are less than the intrathoracic pressure that can be generated by conventional CPR would not be expected to produce a further increment in intrathoracic pressure. Therefore, the lack of superiority of SCV-CPR in infant piglets, as in small dogs, is probably due to the relatively high intrathoracic pressure generated by conventional CPR in these models.

We believe that the reason for the high intrathoracic pressure achieved with conventional CPR in piglets is related to the shape and deformability of the chest of infant piglets. Because the cross sectional shape of the thorax in piglets and small dogs is more circular than that of large dogs, cyclic sternal displacement of similar percent of the anteroposterior diameter will produce a greater percent decrease in cross-sectional area in piglets and small dogs (12). Moreover, in piglets the chest does not fully recoil in between compressions and the chest shape becomes elliptical. Consequently, the 20% cyclic displacement on the minor axis of the ellipse produces considerably greater cyclic decreases in cross-sectional area and hence greater increases in intrathoracic pressure (12). Because human infants have deformable chests with an elliptical rather than circular cross-section, we anticipate that sternal displacement on the minor axis of the ellipse would produce high intrathoracic pressure when CPR is performed in the conventional manner. Thus, SCV-CPR is unlikely to provide a further advantage in human infants.

50 min
88 ± 16
163 ± 46
52 ± 9
89 ± 10†
6.73 ± 0.05
6.62 ± 0.04
4.5 ± 0.4
4.1 ± 0.6
76 ± 9
78 ± 1
5.0 ± 0.8
4.7 ± 0.7
0.6 ± 0.2
0.7 ± 0.2
0.1 ± 0.1
0.1 ± 0.1
0.84 ± 0.04
0.85 ± 0.02
24 ± 3
26 ± 5
30 ± 2
32 ± 5

In large dogs epinephrine administration during either conventional or SCV-CPR causes selective peripheral vasoconstriction in noncerebral and noncoronary beds, which raises aortic pressure but not right atrial pressure (7). Thus, the gradient for myocardial blood flow is enhanced. In our study with continuous epinephrine infusion, blood flow to noncerebral peripheral tissue was extremely low (Table 1). This peripheral vasoconstriction kept aortic pressure elevated in between chest compressions and provided a consistent gradient for myocardial blood flow during the chest relaxation phase of the CPR cycle. This accounted for the significant myocardial blood flow in both groups during early CPR. During prolonged CPR, peripheral tissue blood flow remained extremely low. This suggests that the progressive decline in aortic pressure and consequent decline in MBF and CBF is due to impaired venous return and not to peripheral vasodilation. This decline in aortic pressure in piglets is in marked contrast to the stable perfusion pressure and myocardial and cerebral blood flow seen in dogs with epinephrine infusion over a 50-min period of CPR (7).

Although SCV-CPR at an airway pressure of 60 mm Hg did not augment intrathoracic pressure, we postulated that SCV-CPR may be beneficial during prolonged CPR by preventing this apparent decline in venous return and pulmonary blood flow either by assisting chest recoil or by cyclically propelling blood through the lungs (13, 14). Progressive chest deformity that develops with prolonged CPR could obstruct pulmonary blood flow by distorting intrathoracic vessels or impede venous return into the chest. It is possible that with progressive chest wall deformity and distortion, chest wall compliance might be increased such that intrapleural pressure becomes positive during the relaxation phase of CPR. If this occurs in the presence of an unchanged right atrial pressure, as was demonstrated during CPR in both groups in this study, then the transmural pressure determining atrial filling might be compromised. No measurements of pleural pressure were, however, made in this study. Simultaneous ventilation at high airway pressure in this study did not, however, splint the chest wall or assist in chest recoil to prevent progressive chest deformity. Hausknecht *et al.* (13) demonstrated in an isolated, heart-lung model of CPR that lung inflation augmented cardiac output when swings in pleural pressure were low, but not when they were high. Because pleural pressure swings appeared to remain high when CPR was prolonged, cyclic compression of pulmonary capillaries by lung inflation does not appear to provide any significant benefit in this situation. Thus, it is more likely that there is some impediment to blood flow proximal to the pulmonary bed or that there is pooling of blood in abdominal veins. Repetitive cyclic stretch of splanchnic veins accompanying retrograde inferior vena caval blood flow during each chest compression may gradually increase venous capacitance, and this effect may be more prominent in immature pigs than mature dogs.

We used a compression rate of 100/min during conventional CPR as recommended by the American Heart Association guidelines for infant CPR (19), whereas a rate of 60/min was used for SCV-CPR. We found that animals subjected to SCV-CPR at higher rates developed pneumothoraces secondary to lung hyperinflation. Moreover, preliminary studies in our laboratory indicated that perfusion pressure gradients are relatively insensitive to compression rate between 60 and 100/min during conventional CPR in infant piglets (20). Thus, the difference in compression rates between groups is probably not a significant factor in affecting organ blood flow in this study. This study was not designed to elucidate the mechanism of blood flow during CPR and the results do not necessarily imply that the thoracic pump mechanism is responsible for antegrade blood flow. Direct cardiac compression remains a possible mechanism for flow generation in each of the CPR groups in this animal model.

In this study, cerebral blood flow and O_2 uptake were not higher with SCV-CPR than with conventional CPR. In adult dogs, intracranial pressure increases when airway pressure is increased during SCV-CPR, but the increase in aortic and carotid pressure is even greater (21), thereby enhancing cerebral blood flow (4). In contrast in this study, there was no difference between the CPR groups in either the upstream (aortic) or downstream pressures (either intracranial or sagittal sinus pressures) because intrathoracic pressure was equivalent. Under certain circumstances, aortic pressure may exceed carotid pressure because of collapse of the intrathoracic arteries at the thoracic inlet and cerebral perfusion pressure may be overestimated (7). However, CBF was similar in each group, suggesting that any overestimation of cerebral perfusion pressure by using aortic pressure was similar between groups.

The level of CBF achieved during conventional CPR in this study is lower than those from a previous study on piglets from this laboratory (11). However, in that study an adult chest pad was used, whereas in this study a pediatric chest pad was used. With the pediatric chest pad, less compressive force was required to produce 20% sternal displacement directly under the pad because the force was applied over a smaller surface area. However, this pad produces less displacement over the more rostral segment of the sternum because the hemisphere on the pediatric pad is not in contact with the rostral segment. Thus, the increase in intrathoracic pressure is somewhat less than that with the larger, flat adult pad. This may account for the slightly lower aortic pressures generated in this investigation than those achieved in the previous study.

Despite altering the concentration of inspired CO_2 to maintain the arterial P_{CO_2} uniform in both groups, P_{CO_2} was higher in the SCV-CPR group. In both groups, P_{CO_2} increased with prolonged CPR. The effect of hypercarbia and acidosis under circumstances of low CBF with prolonged CPR in this study is unclear. However, we suspect that differences between groups due to direct effects of CO_2 on cerebral vessels is small because the vessels are probably maximally vasodilated under circumstances of maximal fractional O_2 extraction (22). The increase in blood flow solely to the medulla at 10 min of SCV-CPR may, however, be accounted for by the increase in P_{CO_2} in that group. Hansen *et al.* (23) demonstrated in newborn piglets that there are regional differences in the response of cerebral blood flow to hypercarbia. In that study there was a greater increase in blood flow to the brain stem, cerebellum, and thalamus than to the cerebrum.

The Hb concentration in both groups of animals decreased by approximately 40% during 50 min of CPR. This was probably caused by hemodilution, because each animal received 220 mL of crystalloid infusion during resuscitation. There was no necropsy evidence of extravascular blood loss such as liver rupture that, in addition, might have contributed to the decline in hematocrit.

In summary, we have shown that SCV-CPR does not enhance cerebral and myocardial perfusion during early CPR in this infant animal model or CPR presumably because intrathoracic pressure generation is already high with conventional CPR. In addition, SCV-CPR does not prevent the progressive deformity of the infant animal chest when CPR is prolonged and does not mitigate the subsequent deterioration of CBF and MBF.

Acknowledgments. The authors thank Susan Hacker and Nikki Womer for their fine secretarial assistance in preparing this manuscript.

REFERENCES

1. Rudikoff MT, Maughan WL, Effron M, Freund P, Weisfeldt ML 1980 Mechanisms of blood flow during cardiopulmonary resuscitation. Circulation 61:345–362
2. Weisfeldt ML, Chandra N 1981 Physiology of cardiopulmonary resuscitation. Annu Rev Med 32:435–442
3. Chandra N, Weisfeldt ML, Tsitlik J, Vaghaiwalla F, Snyder LD, Hoffecker M, Rudikoff MT 1981 Augmentation of carotid flow during cardiopulmonary resuscitation by ventilation at high airway pressure simultaneous with chest compression. Am J Cardiol 48:1053–1063
4. Koehler RC, Chandra N, Guerci AL, Tsitlik J, Traystman RJ, Rogers MC, Weisfeldt ML 1983 Augmentation of cerebral perfusion by simultaneous chest compression and lung inflation with abdominal binding after cardiac arrest in dogs. Circulation 67:266–275

5. Halperin HR, Tsitlik JE, Guerci AD, Mellits ED, Levin HR, Shi AY, Chandra N, Weisfeldt ML 1986 Determinants of blood flow to vital organs during cardiopulmonary resuscitation in dogs. Circulation 73:539–550
6. Luce JM, Ross BK, O'Quin RJ, Culver BH, Sivarajan M, Amory DW, Niskanen RA, Alferness CA, Kirk WL, Pierson LB, Butler J 1983 Regional blood flow during cardiopulmonary resuscitation in dogs using simultaneous and nonsimultaneous compression and ventilation. Circulation 67:258–264
7. Michael JR, Guerci AD, Koehler RC, Shi AY, Tsitlik J, Chandra N, Niedermeyer E, Rogers MC, Traystman RJ, Weisfeldt ML 1984 Mechanisms by which epinephrine augments cerebral and myocardial perfusion during cardiopulmonary resuscitation in dogs. Circulation 69:822–835
8. Chandra N, Rudikoff M, Weisfeldt ML 1980 Simultaneous chest compression and ventilation at high airway pressure during cardiopulmonary resuscitation. Lancet 1:175–178
9. Zaritsky A 1986 Advanced pediatric life support: state of the art. Circulation 74(suppl IV):124–128
10. Fleisher G, Delgado-Paredes C, Heyman S 1987 Slow versus rapid closed-chest cardiac compression during cardiopulmonary resuscitation in puppies. Crit Care Med 15:939–943
11. Schleien CL, Dean JM, Koehler RC, Michael JR, Chantarojanasiri T, Traystman R, Rogers MC 1986 Effect of epinephrine on cerebral and myocardial perfusion in an infant animal preparation of cardiopulmonary resuscitation. Circulation 73:809–817
12. Dean JM, Koehler RC, Schleien CL, Michael JR, Chantarojanasiri T, Rogers MC, Traystman RJ 1987 Age-related changes in chest geometry during cardiopulmonary resuscitation. J Appl Physiol 62:2212–2219
13. Hausknecht MJ, Wise RA, Brower RG, Hassapoyannes C, Weisfeldt ML, Suzuki J, Permutt S 1986 Effects of lung inflation on blood flow during cardiopulmonary resuscitation in the canine isolated heart-lung preparation. Circulation 59:676–683
14. Passerini L, Wise RA, Roussos C, Magder S 1988 Maintenance of circulation without chest compression during CPR. J Crit Care 3:96–102
15. Nichols DG, Ketrick RG, Swedlow DB, Scott L, Passman R, Ludwig S 1986 Factors influencing the outcome of cardiopulmonary resuscitation in children. Pediatr Emerg Care 2:1–5
16. Heymann MA, Payne BD, Hoffman JR, Rudolph AM 1977 Blood flow measurements with radionuclide-labeled particles. Prog Cardiovasc Dis 20:55
17. Babbs CF, Tacker WA, Paris RC, Murphy FJ, Davis RW 1982 CPR with simultaneous compression and ventilation at high airway pressure in four animal models. Crit Care Med 10:501–504
18. Sanders AB, Ewy GA, Alferness CA, Taft T, Zimmerman M 1982 Failure of one method of simultaneous chest compression, ventilation, and abdominal binding during CPR. Crit Care Med 10:509–513
19. Standards and guidelines for cardiopulmonary resuscitation and emergency cardiac care. 1986 JAMA 255:2905–2932
20. Koehler RC, Dean JM, Schleien CL, Berkowitz ID, Michael JR, Atchison D, Rogers MC, Traystman RJ 1987 Effect of compression rate and duty cycle on cerebral perfusion pressure during cardiopulmonary resuscitation in swine. Anesthesiology 67:A152(abstr)
21. Guerci AD, Shi AY, Levin H, Tsitlik J, Weisfeldt ML, Chandra N 1985 Transmission of intrathoracic pressure to the intracranial space during cardiopulmonary resuscitation in dogs. Circ Res 56:20–30
22. McPherson RW, Eimerl D, Traystman RJ 1987 Interaction of hypoxia and hypercapnia on cerebral hemodynamics and brain electrical activity in dogs. Am J Physiol 253:H890–H897
23. Hansen NB, Brubakk AM, Bratlid D, Oh W, Stonestreet BS 1984 The effects of variations in $PaCO_2$ on brain blood flow and cardiac output in the newborn piglet. Pediatr Res 18:1132–1136

Annual Meeting of the Society for Adolescent Medicine

The Society for Adolescent Medicine will hold its annual meeting March 22–25, 1990, at the Hilton Hotel and Towers, Atlanta, GA. The meeting will address new material on a broad range of issues important to adolescent physical and emotional health: AIDS, teenage sexuality and pregnancy, eating disorders, depression, and risk-taking behaviors, including suicide and violence. Meeting presentations include all-day workshops, 3-h clinically oriented workshops, research networking breakfasts and luncheon seminars, scientific research paper presentations and poster sessions, as well as the prestigious Gallagher Lecture Series. Special symposia will be offered in conjunction with the Centers for Disease Control and the Society for Research on Adolescence. CME/CEU credit available. *For information contact* the Society for Adolescent Medicine, Suite 101, 10727 White Oak Avenue, Granada Hills, CA 91344, (818) 368-5996.

Abstract Deadline

The American Pediatric Society and The Society for Pediatric Research announce the abstract deadline for the 1990 Annual Meeting (May 7–11, 1990, Anaheim Hilton & Convention Center, Anaheim, CA) has been set as *January 4, 1990.*

For further information contact: 2650 Yale Blvd., S.E., Suite 104, Albuquerque, NM 87106 (505) 764-9099.

Hyperlipidemia in Childhood and the Development of Atherosclerosis

A conference on Hyperlipidemia in Childhood and the Development of Atherosclerosis will be held May 2–4, 1990, at the Hyatt Regency, Bethesda, MD. This conference will examine the role of hyperlipidemia and dyslipidemia in childhood in relation to the development of atherosclerosis. The program will focus initially on morphologic development of the atherosclerotic plaque, pathologic findings in pediatric autopsy series, biochemical correlates, and cellular models of atherosclerosis in the young. Distribution of plasma cholesterol and lipoprotein levels in children will be reviewed, including genetic, dietary, developmental, lifestyle, and pharmacologic influences on serum levels. Tracking of lipid levels over time will be reviewed, as well as international comparisons of lipids in pediatric populations. Cholesterol screening of children will be presented both from high-risk and population-based viewpoints. Pediatric office-based, school-based, and community-wide cholesterol screening activities and interventions will be described. Dietary and pharmacologic therapy of lipid disorders in children will be presented. Abstract deadline is *December 11, 1989.* The abstract, including title, author(s), and affiliation must be typed single space within a 5 × 4-3/8 inch rectangle, and sent to Dr. Christine L. Williams, Preventive Cardiology Center, New York Medical College, Valhalla, NY 10595. *For further information, contact* Conference Department, The New York Academy of Sciences, 2 East 63rd Street, New York, NY 10021, (212) 838-0230.