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Effects of Ventilation on Hemodynamics and Myocardial Blood Flow during Active Compression-Decompression Resuscitation in Pigs

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Background: **Active compression-decompression (ACD) improves hemodynamics and vital organ blood flow during cardiopulmonary resuscitation. The effects of intermittent positive pressure ventilation (IPPV) on ACD have not been studied. This study was designed to compare the effects of ACD with and without IPPV on gas exchange, hemodynamics, and myocardial blood flow.**

Methods: **After 30 s ventricular fibrillation, 14 tracheally intubated pigs were allocated to receive either ACD combined with IPPV (ACD-IPPV) or ACD alone. In animals treated with ACD-IPPV, the lungs were ventilated using a servo ventilator. Animals treated with ACD received 100% oxygen by a reservoir but ventilation was not assisted.**

Results: **Minute ventilation (median) was 6.5 and 6.1 l/min after 1 and 7 min of ACD-IPPV, and was 4.2 and 1.6 l/min after 1 and 7 min of ACD. In contrast to ACD-IPPV, Pa_{O_2} was less and Pa_{CO_2} was greater with ACD. Mean arterial (53 and 40 mmHg; $P < 0.05$) and mean central venous pressure (25 and 14 mmHg; $P < 0.01$) were greater during ACD-IPPV as compared with ACD. After administration of epinephrine 0.2 mg/kg, myocardial blood flow increased only in ACD-IPPV treated animals, and 5 min after epinephrine administration, myocardial blood flow was greater during ACD-IPPV (33 $ml \cdot min^{-1} \cdot 100\ g^{-1}$) as compared with ACD (15 $ml \cdot min^{-1} \cdot 100\ g^{-1}$; $P < 0.05$). Restoration of spontaneous circulation could be achieved only in animals subjected to ACD-IPPV.**

Conclusions: **Gas exchange was critically impaired during the late phase of ACD. Compared with ACD-IPPV, myocardial blood flow was less preserved with ACD and was too low to achieve restoration of spontaneous circulation. (Key words: Cardiac arrest: cardiopulmonary resuscitation. Gases: carbon dioxide. Hemodynamics: myocardial blood flow. Respiratory acidosis: acid-base status; hypoxia. Ventilation: mechanical.)**

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CARDIOPULMONARY resuscitation (CPR) using active compression-decompression (ACD) has been shown to improve hemodynamics and vital organ blood flow in animal[1–4] as well as human studies.[5–8] Clinical investigations comparing ACD to standard CPR in patients in cardiac arrest demonstrated an improvement in the immediate resuscitation success.[9–11]

When ACD was performed in humans in whom standard advanced cardiac life support had failed, a minute ventilation of 6.6 ± 0.9 l/min was observed.[5] The ventilatory aspects of ACD have raised interest in subsequent studies investigating ventilation generated by ACD *versus* standard and alternative CPR techniques.[12–14]

However, the effects of intermittent positive pressure ventilation (IPPV) on the potential of ACD to improve hemodynamics and to preserve myocardial blood flow have not been investigated as yet and it remains questionable whether ACD without IPPV is as effective as in combination with IPPV. Thus, the aim of this study was to compare the effects of ACD with and without IPPV on gas exchange, hemodynamics, and myocardial blood flow.

Methods and Materials

Anesthesia and Surgical Preparation

This study was approved by our institutional animal investigation committee. The animals were managed in accordance with the guidelines of the American Physiological Society. Fourteen pigs (24–25 kg) received 4 mg/kg intramuscular azaperone 1 h before surgical preparation. Anesthesia was induced with 10 mg/kg intravenous metomidate. The pigs were stabilized in the dorsal recumbent position and an endotracheal tube was inserted. Ventilation was performed with a servo ventilator with 65% nitrous oxide in oxygen at 20 breaths/min and with a tidal volume adjusted to maintain Pa_{CO_2} at 35 mmHg. Anesthesia was main-

tained using a continuous infusion of metomidate (0.5 $mg \cdot kg^{-1} \cdot h^{-1}$) and two doses of 0.024 mg/kg buprenorphine at the beginning of surgical preparation and before induction of cardiac arrest, respectively. Ringer's solution (6 $ml \cdot kg^{-1} \cdot h^{-1}$) was administered continuously. A lead II ECG was monitored. Fluid-filled catheters were advanced *via* femoral cutdowns into the abdominal aorta and right atrium. A pulmonary artery catheter was inserted *via* external jugular vein cutdown. A 7-French pigtail catheter was placed into the left ventricle for injection of radionuclide microspheres and iced saline. For the measurement of body temperature, a thermistor was placed into the aorta. Before cardiac arrest, 300 units/kg sodium heparin was administered intravenously to prevent intracardiac clot formation. All animals were autopsied to check correct positioning of the catheters, and to look for damage to the rib cage and internal organs.

Measurements

Aortic and right atrial pressures were measured *via* fluid-filled catheters using pressure transducers that were calibrated to atmospheric pressure at the level of the right atrium. Pressure tracings were continuously recorded using two monitors and a data acquisition/control unit. Measurements were performed at intervals of 30 s before cardiac arrest and at intervals of 1 s during CPR. An electronic substraction circuit was used to record arteriovenous pressure differences (aortic minus right atrial pressure difference), which reflect coronary perfusion pressure. Cardiac output was measured by thermodilution technique (5 ml iced saline injected into the left ventricle) using a cardiac output computer. Blood gases were measured with a blood gas analyzer and corrected for body temperature. Expired minute volume was sampled between min 1 and 3 and between min 7 and 9, respectively, after beginning of CPR through a nonrebreathing valve into a gas-tight reservoir and measured afterward with a spirometer.

Before and 90 s and 5 min after epinephrine administration during CPR, myocardial blood flow was measured with radiolabeled microspheres according to Heymann *et al.*[15] as previously described.[2] At the end of the experiment, the entire heart was removed. The left ventricular free wall including the endocardial and epicardial tissue layers was weighed, homogenized, and radioactivity from tissue and blood was measured with a gamma scintillation spectrometer.

Experimental Protocol

Before induction of cardiocirculatory arrest, hemodynamic parameters, and blood gases were measured. A 50-Hz, 60-V alternating current was applied *via* two subcutaneous needle electrodes to induce ventricular fibrillation. Cardiocirculatory arrest was defined as that point at which the aortic pulse pressure decreased to zero and the electrocardiogram showed ventricular fibrillation. Thirty seconds after induction of cardiocirculatory arrest, ACD was begun with a pneumatically driven automatic piston device (ACD Controller, Ambu International, Glostrup, Denmark). The chest compression rate was 80/min, and the duration of compression was 50% of the total cycle time. Unlike in humans in which a handheld compression/suction device provided with a silicone rubber suction cup (Ambu CardioPump, Glostrup, Denmark) is used for ACD, in the current study the compression pad was wired to the mid-sternum to ensure constant contact with the animal's chest wall and to prevent displacement of the pad during ACD. A velocity of compression of 7.48 inches/s was held constant. Based on experiments using the same animal model, the chest compression force was adjusted to produce 25% of sternal displacement of the pig's anteroposterior diameter. Active decompression was set to produce a sternal displacement of 10% greater than the resting anteroposterior diameter, for which a suction force of approximately 200 N was necessary. The decompression velocity was 7.48 inches/s.

During ACD with IPPV, mechanical ventilation with 100% of oxygen was performed at 20 breaths/min independently of chest compression at a tidal volume shown to result in a Pa_{CO_2} of 35 mmHg before induction of cardiac arrest. In animals without IPPV, 100% oxygen was supplied by an oxygen reservoir bag connected to the endotracheal tube.

After 5 min of CPR, all animals received epinephrine 0.2 mg/kg in a 10-ml volume of normal saline into the right atrium over a period of 5 s. Hemodynamic measurements and acquisition of aortic and mixed venous blood samples were performed before and 90 s and 5 min after epinephrine administration. After acquiring the last blood sample during CPR, direct current shocks were applied. Initially, up to three countershocks (3 J/kg) were administered. In cases of persistent ventricular fibrillation, epinephrine was administered at the same dose as the previous one, and after a further 90 s of CPR, up to three more countershocks (5 J/kg) were

administered. The same protocol (without defibrillation) was used if asystole or pulseless electrical activity occurred. Successful resuscitation was defined as the presence of coordinated electrical activity together with a systolic blood pressure above 80 mmHg for a duration of at least 5 min, during which no further resuscitative measures were applied.

Statistical Analysis

Values are expressed as median as well as 25th and 75th percentiles. For within-group comparisons, the Wilcoxon signed rank test was used. For comparisons between groups, the Mann-Whitney *U* test was used. Correlations between single parameters were analyzed by regression analysis. Statistical significance was considered at $P < 0.05$.

Results

There were no differences in baseline hemodynamics, myocardial blood flow, blood gases, or minute ventilation between animals in the ACD-IPPV or ACD groups before induction of ventricular fibrillation (tables 1–3). Whereas in ACD-IPPV-treated animals, spontaneous circulation could be restored within 20 s after removal of the last blood sample, in animals subjected to ACD alone, restoration of spontaneous circulation could not be achieved. Autopsy revealed no damage to the thoracic cage or the internal organs in either group.

Hemodynamics and Myocardial Blood Flow

Mean arterial pressure (median as well as 25th and 75th percentiles) was greater in the ACD-IPPV group compared with the ACD group before (44, 40–51 and 33, 26–37 mmHg; $P < 0.05$) as well as 90 s (73, 66–80 and 50, 48–57 mmHg; $P < 0.01$) and 5 min after epinephrine administration (53, 40–63 and 40, 33–43 mmHg) ($P < 0.05$). Similarly, mean central venous pressure was greater in ACD-IPPV-treated animals in comparison with ACD-treated animals before (22, 20–31 and 14, 10–18 mmHg; $P < 0.05$) and 90 s (27, 25–34 and 18, 15–21 mmHg; $P < 0.01$) and 5 min after administration of epinephrine (25, 21–33 and 14, 11–17 mmHg; $P < 0.01$). In both groups, mean arterial pressure and mean central venous pressure increased 90 s after epinephrine administration as compared with point in time before epinephrine admin-

Table 1. Hemodynamics Prearrest and during Active Compression–Decompression Resuscitation with and without Intermittent Positive Pressure Ventilation

	EPI			
	Prearrest	3 min Postarrest	6.5 min Postarrest	10 min Postarrest
Mean arterial pressure (mmHg)				
ACD-IPPV	108 (88–112)	44 (40–51)*	73 (66–80)†‡	53 (40–63)*
ACD	106 (92–120)	33 (26–37)	50 (48–57)‡	40 (33–43)
Mean central venous pressure (mmHg)				
ACD-IPPV	4 (2–4)	22 (20–31)*	27 (25–34)†‡	25 (21–33)†
ACD	3 (2–5)	14 (10–18)	18 (15–21)‡	14 (11–17)
Mean coronary perfusion pressure (mmHg)				
ACD-IPPV	98 (86–105)	24 (7–31)	49 (28–53)‡	28 (7–42)
ACD	104 (92–114)	19 (16–21)	33 (29–36)‡	23 (20–30)
Cardiac index ($ml \cdot min^{-1} \cdot kg^{-1}$)				
ACD-IPPV	164 (149–200)	27 (15–33)	19 (17–22)	20 (14–24)
ACD	184 (156–207)	26 (20–28)	17 (14–24)‡	20 (17–23)

Values are median as well as 25th and 75th percentile.

EPI = administration of epinephrine 0.2 mg/kg at 5 min postarrest; ACD = active compression–decompression resuscitation; ACD-IPPV = ACD combined with intermittent positive pressure ventilation.

*$P < 0.05$ *versus* ACD.

†$P < 0.01$ *versus* ACD.

‡$P < 0.05$ *versus* 3 min postarrest.

Table 2. Myocardial Blood Flow during Active Compression–Decompression Resuscitation with and without Intermittent Positive Pressure Ventilation

Myocardial blood flow ($ml \cdot min^{-1} \cdot 100\ g^{-1}$)	EPI		
	3 min Postarrest	6.5 min Postarrest	10 min Postarrest
ACD-IPPV	30 (19–43) [MAD 11.2]	55 (39–68)* [MAD 15.3]	33 (25–39)† [MAD 5.0]
ACD	25 (18–34) [MAD 3.5]	39 (20–60) [MAD 17.3]	15 (10–27) [MAD 6.2]

Values are median as well as 25th and 75th percentile. EPI = administration of epinephrine 0.2 mg/kg at 5 min postarrest; ACD = active compression–decompression resuscitation; ACD-IPPV = ACD combined with intermittent positive pressure ventilation; MAD = median absolute deviation.

* $P < 0.05$ *versus* 3 min postarrest.

† $P < 0.05$ *versus* ACD.

istration ($P < 0.05$). Mean coronary perfusion pressure was not significantly different between both groups before (24, 7–31 and 19, 16–21 mmHg) as well as 90 s (49, 28–53 and 33, 29–36 mmHg) and 5 min after epinephrine administration (28, 7–42 and 23, 20–30 mmHg). In both groups, coronary perfusion pressure

Table 3. Blood Gases Prearrest and during Active Compression–Decompression Resuscitation with and without Intermittent Positive Pressure Ventilation

	EPI			
	Prearrest	3 min Postarrest	6.5 min Postarrest	10 min Postarrest
Arterial P_{O_2} (mmHg)				
ACD-IPPV	378 (321–390)	164 (123–219)*	206 (168–246)†‡	156 (75–302)†
ACD	401 (364–453)	87 (66–106)	67 (58–81)	59 (48–66)
Mixed venous P_{O_2} (mmHg)				
ACD-IPPV	59 (47–55)	27 (23–35)	23 (20–39)	20 (18–24)
ACD	62 (58–67)	27 (22–30)	22 (17–28)	18 (26–20)
Arterial P_{CO_2} (mmHg)				
ACD-IPPV	34 (33–34)	25 (20–27)†	20 (18–22)†	26 (20–30)†
ACD	35 (34–36)	45 (40–53)	74 (67–80)‡	89 (85–98)
Mixed venous P_{CO_2} (mmHg)				
ACD-IPPV	41 (36–42)	44 (40–51)*	47 (50–55)†	56 (45–62)†
ACD	40 (40–45)	60 (51–66)	76 (70–88)‡	88 (84–92)
Arterial *p*H				
ACD-IPPV	7.51 (7.48–7.55)	7.62 (7.56–7.71)†	7.60 (7.53–7.70)†	7.50 (7.42–7.67)†
ACD	7.46 (7.44–7.48)	7.31 (7.26–7.38)	7.09 (7.04–7.14)‡	6.99 (6.95–7.03)
Mixed venous *p*H				
ACD-IPPV	7.48 (7.40–7.50)	7.41 (7.32–7.47)*	7.31 (7.26–7.40)†‡	7.23 (7.21–7.37)†
ACD	7.40 (7.36–7.42)	7.22 (7.18–7.31)	7.08 (7.05–7.18)‡	7.09 (6.96–7.05)
D_A-vP_{CO_2} (mmHg)				
ACD-IPPV	−6 (−3 to −9)	−22 (−16 to −26)*	−28 (−18 to −36)†‡	−28 (−21 to −42)†
ACD	−8 (−5 to −9)	−12 (−8 to −15)	−6 (−1 to −11)‡	−6 (−3 to −8)
D_A-v*p*H				
ACD-IPPV	0.02 (0.01–0.03)	0.24 (0.16–0.29)*	0.30 (0.18–0.35)†‡	0.20 (0.17–0.31)†
ACD	0.03 (0.01–0.04)	0.07 (0.04–0.11)	0.04 (0.02–0.05)‡	0.02 (0.01–0.04)

Values are median as well as 25th and 75th percentile. EPI = administration of epinephrine 0.2 mg/kg at 5 min postarrest; ACD = active compression–decompression resuscitation; ACD-IPPV = ACD combined with intermittent positive pressure ventilation; D_A-vP_{CO_2} = arterial-mixed venous P_{CO_2} gradient; D_A-v*p*H = arterial-mixed venous *p*H gradient.

* $P < 0.05$ *versus* ACD.

† $P < 0.01$ *versus* ACD.

‡ $P < 0.05$ *versus* 3 min postarrest.

increased 90 s after epinephrine administration as compared with mechanical measures alone ($P < 0.05$). Cardiac index was not significantly different between both groups before (27, 15–33 and 26, 20–28 $ml \cdot min^{-1} \cdot kg^{-1}$) as well as 90 s (19, 17–22 and 17, 14–24 $ml \cdot min^{-1} \cdot kg^{-1}$) and 5 min (20, 14–24 and 20, 17–23 $ml \cdot min^{-1} \cdot kg^{-1}$) after epinephrine administration. Ninety seconds after epinephrine administration, cardiac index was decreased as compared with preepinephrine levels. The decrease in cardiac index was significant only in the ACD group (table 1).

Myocardial blood flow was not different between both groups before (30, 19–43 and 25, 18–34 $ml \cdot min^{-1} \cdot 100\ g^{-1}$) and 90 s after epinephrine administration (55, 39–68 and 39, 20–60 $ml \cdot min^{-1} \cdot 100\ g^{-1}$). However, 5 min after epinephrine administration, myocardial blood flow was greater in animals subjected to ACD-IPPV (33, 25–39 $ml \cdot min^{-1} \cdot 100\ g^{-1}$) as compared with animals subjected to ACD alone (15, 10–27 $ml \cdot min^{-1} \cdot 100\ g^{-1}$; $P < 0.05$). Only in ACD-IPPV-treated animals did myocardial blood flow increase after epinephrine administration as compared to mechanical measures alone ($P < 0.05$) (table 2). In the ACD-IPPV group there was a significant correlation between coronary perfusion pressure and myocardial blood flow ($r = 0.68$, $P < 0.01$), but no such correlation was found in animals treated with ACD alone ($r = 0.36$, ns). Five minutes after epinephrine administration, seven animals in the ACD-IPPV group and two animals in the ACD group had myocardial blood flow values >20 $ml \cdot min^{-1} \cdot 100\ g^{-1}$ ($P < 0.05$, Fisher's exact test).

Blood Gases

Pa_{O_2} was higher before as well as 90 s and 5 min after epinephrine administration in the ACD-IPPV group as compared with the ACD group. Mixed venous P_{O_2} was not different between both groups at any point in time. Ninety seconds after epinephrine administration, Pa_{O_2} increased in ACD-IPPV-treated, but not in ACD-treated animals in comparison with preepinephrine levels, whereas mixed venous P_{O_2} did not change in any group between points in time before and 90 s after epinephrine administration. Pa_{CO_2} and mixed venous P_{CO_2} were less before as well as 90 s and 5 min after epinephrine administration in pigs subjected to ACD-IPPV in comparison with ACD treated pigs. While 90 s after epinephrine administration Pa_{CO_2} and mixed venous P_{CO_2} remained unchanged in the ACD-IPPV group, in the ACD group, Pa_{CO_2} and mixed venous P_{CO_2} increased in comparison with preepinephrine levels. Arterial and mixed venous pH values were higher before as well as 90 s and 5 min after epinephrine administration in ACD-IPPV-treated animals in comparison with animals treated with ACD alone. Ninety seconds after epinephrine administration, arterial pH decreased in the ACD group, but not in the ACD-IPPV group whereas mixed venous pH decreased in both groups in comparison with preepinephrine levels. The absolute value of the arterial-mixed venous P_{CO_2} gradient was greater before as well as 90 s and 5 min after administration of epinephrine in animals treated with ACD-IPPV in comparison with ACD-treated animals. The arterial-mixed venous P_{CO_2} gradient increased in the ACD-IPPV group and decreased in the ACD group 90 s after epinephrine administration in comparison with preepinephrine levels (table 3).

Ventilation

In the ACD-IPPV group, minute volume was 6.5, 6.2–6.8, and 6.1, 5.8–6.4 l/min after 1 and 7 min of CPR, respectively. In the ACD group, minute volume was 4.2, 2.3–4.6, and 1.6, 0.8–2.3 l/min after 1 and 7 min of CPR, respectively (fig. 1). Calculated tidal volume was 325, 310–339, and 305, 288–322 ml after 1 and 7 min of ACD-IPPV (minute volume divided by respiratory rate: 20/min) and was 52, 28–57, and 19, 3–29 ml after 1 and 7 min of ACD alone (minute volume divided by chest compression rate: 80/min).

Discussion

In comparison with standard CPR, ACD has been shown to improve hemodynamics, vital organ blood

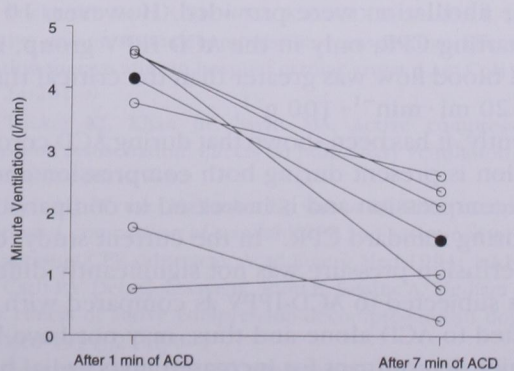

Fig. 1. Minute ventilation during active compression-decompression resuscitation without intermittent positive pressure ventilation. One minute after beginning of active compression-decompression, median minute ventilation is within the normal range of pigs weighing 25 kg (2.6–6.5 l/min), whereas 7 min after beginning of active compression-decompression, median minute ventilation is below this range. Filled circles indicate median minute ventilation.

flow, and immediate resuscitation success in animal as well as in human studies. Myocardial blood flow, which is one of the most important factors for restarting the arrested heart, was significantly greater when ACD was compared with standard CPR in pigs during cardiac arrest.[2,3]

Because ACD increases negative intrathoracic pressure, in addition to augmenting venous return and myocardial blood flow, ACD is able to increase minute ventilation independently of IPPV in comparison to other CPR techniques. The ventilatory aspects of ACD have raised interest in studies investigating ventilation generated by ACD *versus* other CPR techniques and the potential role of ACD as a substitute for rescue breathing during basic life support.[12–14]

Even with standard CPR, the necessity for ventilation during basic life support has been challenged recently by animal investigations. In dogs, it was observed that an arterial oxygen saturation greater than 90% was maintained for the first 5 min of ventricular fibrillation with chest compressions alone[16] and with concern to outcome, in swine, chest compressions alone appeared to be as effective as chest compressions combined with ventilation.[17] In contrast, in another study in pigs, greater hypoxia and hypercarbic acidosis as well as a lower rate of return of spontaneous circulation was found in animals in which the lungs were not ventilated compared with animals[18] in which the lungs were ventilated.

In the current study, in pigs receiving ACD without IPPV, otherwise ideal conditions including tracheal intubation, 100% oxygen, and a very short period of ventricular fibrillation were provided. However, 10 min after starting CPR, only in the ACD-IPPV group, myocardial blood flow was greater than the critical threshold of 20 $ml \cdot min^{-1} \cdot 100\ g^{-1}$.

Recently, it has been shown that during ACD coronary perfusion is present during both compression and active decompression and is increased in comparison to that during standard CPR.[2] In the current study, coronary perfusion pressure was not significantly different in pigs subjected to ACD-IPPV as compared with pigs subjected to ACD alone and thus, may not have been the major determinant for increased myocardial blood flow with ACD-IPPV at the end of the resuscitation phase.

After 10 min of CPR, there were marked differences in Pa_{O_2} and Pa_{CO_2} as well as in mixed venous P_{CO_2} levels between animals with and without IPPV. Myocardial hypercarbic acidosis was reported to reduce cardiac resuscitability independent of coronary perfusion pressure in a porcine model of cardiac arrest.[19] However, because we did not measure intramyocardial carbon dioxide we cannot conclude to which extent mixed venous P_{CO_2} represented intramyocardial carbon dioxide in the current study and whether mixed venous and intramyocardial carbon dioxide, respectively, affected myocardial blood flow.

During hypoxemia in lambs, metabolic acidemia attenuated the hemodynamic response to epinephrine.[20] In our study, mean arterial and coronary perfusion pressures were significantly increased in the ACD-IPPV as well as in the ACD group 90 s after administration of epinephrine. Thus, a diminished hemodynamic response to epinephrine administration seems unlikely to be the underlying cause for a diminished myocardial blood flow in animals without IPPV.

The lower arterial-mixed venous P_{CO_2} and pH gradients in the ACD group, which were also observed in a study in pigs whose lungs were not ventilated during CPR[13] indicate that an increase in Pa_{CO_2} did not lead to a proportional increase in organ venous P_{CO_2}. As a possible explanation for this phenomenon, it is suggested that under the conditions of CPR without IPPV the amount of carbon dioxide transported in arterial blood may be small in comparison to the total amount of carbon dioxide in organ venous blood and may not significantly contribute to mixed venous carbon dioxide content. In our study, cardiac index was equal in animals treated with ACD-IPPV and ACD and thus the arterial-mixed venous P_{CO_2} gradient did not reflect cardiac output.

In comparison with other animal studies on the ventilatory effects of ACD, in our study minute ventilation and the capability to maintain Pa_{O_2} and Pa_{CO_2} in physiologic limits was in the lower range. In tracheally intubated, not mechanically ventilated supine beagles weighing 10–15 kg, a minute volume of 4.9 l/min was achieved in 1-min trials of ACD.[12] Another investigation in tracheally intubated mongrel dogs (13–22 kg) placed in the lateral position showed that after a previous phase of 9 min of different CPR techniques in combination with IPPV, a minute ventilation of 13 l/min was generated in 3-min trials of ACD without IPPV.[14] In pigs receiving ACD without IPPV over 10 min after 6 min of ventricular fibrillation, minute ventilation was greater, and hypoxia and hypercarbic acidosis was less as compared to animals receiving standard CPR.[13] Differences in age, animal species (*i.e.*, regarding thorax configuration), and the methods used

may account for some of the different results in the literature and in the current study with respect to the amount of minute ventilation achieved. In our study, in animals treated with ACD without IPPV, minute ventilation and blood gases were acceptable at the beginning of ACD but deteriorated markedly with ongoing resuscitation. However, even at the beginning of ACD, as in other studies on the ventilatory effects of ACD, at 80 cycles/min the calculated tidal volume was low and the ratio between dead space and tidal volume must have been high. Further studies are needed to investigate the possible mechanisms how ACD improves gas exchange during CPR.

There are several potential limitations of this study. Because compliance was not measured, it cannot be concluded whether alterations in the compliance of the thorax and lung, respectively, may have contributed to the deterioration of ventilation after a longer period of ACD compared with ACD and IPPV. Because we did not investigate long-term survival or neurologic outcome, we cannot conclude whether IPPV would have influenced these variables in comparison to omission of IPPV. Because of radiation protection regulations we were not able to measure myocardial blood flow before induction of ventricular fibrillation and to control this variable for intergroup differences at this time. However, myocardial blood flow was not different before and 90 s after epinephrine administration and hence the observed difference at 5 min after epinephrine is unlikely to be caused by preexisting differences. Finally, by using 100% of oxygen before induction of a 30-s period of ventricular fibrillation and by choosing an epinephrine dosage of 0.2 mg/kg, our model was designed to improve oxygenation, hemodynamics, and resuscitation success rather than to simulate real clinical conditions.

In conclusion, the results of this study demonstrate that gas exchange was critically low during the late phase of ACD without IPPV. While myocardial blood flow was well preserved after 10 min of ACD combined with IPPV and administration of epinephrine, under the same conditions but without IPPV myocardial blood flow was impaired and was less than the critical threshold necessary for restoration of spontaneous circulation.

References

1. Cohen TJ, Tucker KJ, Redberg RF, Lurie KG, Chin MC, Dutton JP, Scheinman MM, Schiller NB, Callaham ML: Active compression-decompression resuscitation: A novel method of cardiopulmonary resuscitation. Am Heart J 1992; 124:1145–50

2. Lindner KH, Pfenninger EG, Lurie KG, Schürmann W, Lindner IM, Ahnefeld FW: Effects of active compression-decompression resuscitation on myocardial and cerebral blood flow in pigs. Circulation 1993; 88:1254–63

3. Chang MW, Coffeen P, Lurie KG, Shultz J, Bache RJ, White CW: Active compression-decompression CPR improves vital organ perfusion in a dog model of ventricular fibrillation. Chest 1994; 106: 1250–9

4. Wik L, Naess PA, Ilebekk A, Steen PA: Simultaneous active compression-decompression and abdominal binding increase carotid blood flow additively during cardiopulmonary resuscitation (CPR) in pigs. Resuscitation 1994; 28:55–64

5. Cohen TJ, Tucker KJ, Lurie KG, Redberg RF, Dutton JP, Dwyer KA, Schwab TM, Chin MC, Gelb AM, Scheinman MM, Schiller NB, Callaham ML: Active compression-decompression: A new method of cardiopulmonary resuscitation. JAMA 1992; 267:2916–23

6. Tucker KJ, Redberg RF, Schiller NB, Cohen TJ: Active compression-decompression resuscitation: Analysis of transmitral flow and left ventricular volume by transesophageal echocardiography in humans. J Am Coll Cardiol 1993; 22:1485–93

7. Shultz JJ, Coffeen P, Sweeney M, Detloff B, Kehler C, Pineda E, Yakshe P, Adler SW, Chang M, Lurie KG: Evaluation of standard and active compression-decompression CPR in an acute human model of ventricular fibrillation. Circulation 1994; 89:684–93

8. Pell ACH, Pringle SD, Guly UM, Steedman DJ, Robertson CE: Assessment of the active compression-decompression device (ACD) in cardiopulmonary resuscitation using transoesophageal echocardiography. Resuscitation 1994; 27:137–40

9. Cohen TJ, Goldner BG, Maccaro PC, Ardito AP, Trazzera S, Cohen MB, Dibs SR: A comparison of active compression-decompression with standard cardiopulmonary resuscitation for cardiac arrests occurring in the hospital. N Engl J Med 1993; 329:1918–21

10. Lurie KG, Shultz JJ, Callaham ML, Schwab TM, Gisch T, Rector T, Frascone RJ, Long L: Evaluation of active compression-decompression CPR in victims of out-of-hospital cardiac arrest. JAMA 1994; 271:1405–11

11. Tucker KJ, Galli F, Savitt MA, Kahsai D, Bresnahan L, Redberg RF: Active compression-decompression resuscitation: Effect on resuscitation success after in-hospital cardiac arrest. J Am Coll Cardiol 1994; 24:201–9

12. Tucker KJ, Khan JH, Savitt MA: Active compression-decompression resuscitation: Effects on pulmonary ventilation. Resuscitation 1993; 26:125–31

13. Idris AH, Wenzel V, Tucker KJ, Orban DJ: Chest compression ventilation: A comparison of standard CPR and active-compression/decompression CPR (abstract). Acad Emerg Med 1994; 1:A17

14. Carli PA, DeLaCoussaye JE, Riou B, Sassine A, Eledjam JJ: Ventilatory effects of active compression-decompression in dogs. Ann Emerg Med 1994; 24:890–4

15. Heymann MA, Payne BD, Hoffman JIE, Rudolph AM: Blood flow measurements with radionuclide-labeled particles. Prog Cardiovasc Dis 1977; 20:55–79

16. Chandra NC, Gruben KG, Tsitlik JE, Brower R, Guerci AD, Halperin HH, Weisfeldt ML, Permutt S: Observations of ventilation during resuscitation in a canine model. Circulation 1994; 90:3070–5

17. Berg RA, Kern KB, Sanders AB, Otto CW, Hilwig RW, Ewy GA:

Bystander cardiopulmonary resuscitation: Is ventilation necessary? Circulation 1993; 88:1907–15

18. Idris AH, Becker LB, Fuerst RS, Wenzel V, Rush WJ, Melker RJ, Orban DJ: Effect of ventilation on resuscitation in an animal model of cardiac arrest. Circulation 1994; 90:3063–9

19. Maldonado FA, Weil MH, Tang W, Bisera J, Gazmuri RJ, Johnson B, D'Alessio A: Myocardial hypercarbic acidosis reduces cardiac resuscitability. ANESTHESIOLOGY 1993; 78:343–52

20. Preziosi MP, Roig JC, Hargrove N, Burchfield DJ: Metabolic acidemia with hypoxia attenuates the hemodynamic responses to epinephrine during resuscitation in lambs. Crit Care Med 1993; 21: 1901–7