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Cardiopulmonary Resuscitation with Interposed Abdominal Compression after Asphyxial or Fibrillatory Cardiac Arrest in Pigs

Karl H. Lindner, M.D., Friedrich W. Ahnefeld, M.D.,† Ingrid M. Bowdler, M.D.**

The purpose of this study was to compare the efficacy of standard cardiopulmonary resuscitation and cardiopulmonary resuscitation with interposed abdominal compression for restoration of spontaneous circulation in an asphyxial and fibrillatory arrest model. Twenty-eight pigs weighing 19–27 kg were randomly allocated to two arrest groups. Each of these two groups was then subdivided into a treatment group and a control group resulting in four groups of seven pigs each. In the control groups standard cardiopulmonary resuscitation was performed with a pneumatically driven chest compressor at a rate of 80 beats per min. The animals' lungs were ventilated at a respiratory rate of 20 breaths per min independently of chest compression. In the treatment group, in addition to standard cardiopulmonary resuscitation, manual interposed abdominal compression was applied at the midabdomen in the second half of the relaxation phase using a blood pressure cuff to measure and standardize the compressions. Following asphyxial cardiac arrest of 3 min, none of the seven animals could be resuscitated with standard cardiopulmonary resuscitation, whereas all seven animals could be resuscitated with interposed abdominal compression and standard cardiopulmonary resuscitation after 240 ± 84 s. Following fibrillatory cardiac arrest of 4 min, none of the seven animals that received standard cardiopulmonary resuscitation and countershocks could be resuscitated. In the group that received standard cardiopulmonary resuscitation and interposed abdominal compression spontaneous circulation was achieved in all animals in 244 ± 117 s. End-diastolic arteriovenous pressure difference, which correlates with coronary blood flow, was significantly higher with interposed abdominal compression during resuscitation from both forms of cardiac arrest. The results of our study indicate that cardiopulmonary resuscitation with interposed abdominal compression in the second half of the relaxation phase improves diastolic arteriovenous pressure difference and resuscitation success in comparison with that following standard cardiopulmonary resuscitation. The use of interposed abdominal compression during basic cardiac life support should be investigated further in patients. (Key words: Heart, resuscitation: coronary perfusion pressure; interposed abdominal compression.)

THE ADDITION of interposed abdominal compression (IAC) during the release phase of chest compression to standard cardiopulmonary resuscitation (STD-CPR) has been shown to improve coronary perfusion pressure and cardiac output.[1,2] Previous studies have shown that coronary perfusion pressure correlates well with coronary blood flow produced during CPR[3] and with resuscitation success.[4–6] When external chest compression is applied, the oxygen requirements of the fibrillating myocardium can often not be met.[7] This is particularly so as resuscitation time increases. Voorhees *et al.* showed that abdominal counterpulsation led to an improvement of myocardial and cerebral blood flow measured with the radioactive microsphere technique.[8] In the investigation of Ohomoto *et al.*, survival rate in dogs was significantly improved,[9] whereas Kern *et al.* found no difference in recovery of cardiac function and survival with IAC-CPR.[10]

Because the results of the above-mentioned studies are at variance, and because in recent literature there are no reports on the efficacy of IAC-CPR in an asphyxial arrest model in which a severe oxygen deficit is already present at the beginning of the arrest period, we have compared IAC-CPR with STD-CPR for the management of asphyxial and fibrillatory cardiac arrest.

Methods

Animal Preparation

This investigation was performed in accordance with our institutional Animal Care Committee. We anesthetized 28 pigs weighing 19–27 kg (age 10–12 weeks) with an iv bolus injection of metomidate (12.5 mg/kg). The pigs were fasted for 6 h prior to surgery. The animals were placed on a V-shaped board and their trachea intubated during spontaneous respiration. Anesthesia was maintained with ketamine (10 mg/kg) iv, followed by an infusion of 7.5 $mg \cdot kg^{-1} \cdot h^{-1}$. Muscular relaxation was provided by pancuronium bromide (0.2 mg/kg). After instrumentation the ketamine infusion was stopped and the animals' lungs were ventilated for 10 min with 100% oxygen before cardiac arrest was induced. The infusion of ketamine was only resumed following successful resuscitation at the same dose as previously mentioned.

Electrocardiographic monitoring was performed using subcutaneous leads. Saline-filled catheters were advanced *via* femoral cutdowns into the right atrium and abdominal aorta. A 7-F quadruple-lumen flow-directed thermistor-tipped pulmonary artery catheter (Model SP5107, American Edwards Laboratories, Santa Ana, California) was inserted *via* right external jugular vein cutdown. Cardiac

* Doctor of Medicine.
† Professor, Doctor of Medicine.
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Address reprint requests to Dr. Lindner: Clinic of Anesthesiology, Ulm University, Steinhoevelstrasse 9, D-7900 Ulm (Donau), Federal Republic of Germany.

output was measured in triplicate by thermodilution technique with 10 ml iced saline using the Edwards Laboratories Cardiac Output Computer 9520 A. The intravascular catheters were connected to Statham P23Db transducers, which were aligned at the level of the right atrium and zero established to atmospheric pressure and calibrated using a mercury manometer. Catheter position was confirmed at postmortem examination. All pressures were recorded on a four-channel recorder (Model LRS 4 R Penless, Linseis, FRG). An electronic subtraction circuit was used to record end-diastolic arteriovenous pressure differences (aortic minus right atrial pressure difference). Intravascular pressures in the aorta and the right atrium were measured over a period of 90 s after starting mechanical measures.

Experimental Protocol

Asphyxial Arrest. In 14 pigs, asphyxial cardiac arrest was induced by clamping the endotracheal tube. Arrest was defined as that point at which the aortic pulse pressure decreased to zero. In most cases the ECG showed a slow idioventricular rhythm (<30 beats per min) or ventricular asystole. Cardiac arrest was allowed to continue for 3 min. After a 3-min period of cardiac arrest, external chest compression with a pneumatically driven chest compressor (Thumper® Model 1004, Michigan Instruments, Grand Rapids, Michigan) and ventilation (Servo Ventilator 900, Siemens, FRG) were begun. The rate of compression was 80 breaths per min corresponding to a cycle time of 750 ms. The duration of compression was 50% of the cycle time, the compression being directly measured at the animal's chest. Before arrest, the anteroposterior chest diameter was measured at the lower edge of the sternum in all animals. It ranged from 19–21.5 cm (mean 19.9 cm). The chest compression force, 80–100 lbs, was selected to produce 25% sternal displacement (expressed as a percentage of anteroposterior diameter) and was not changed thereafter during either STD-CPR or IAC-CPR. The animals' lungs were ventilated at a respiratory rate of 20 breats per min independently of chest compression and with that tidal volume which, prior to induction of cardiac arrest, had been shown to result in normal blood gas values.

In seven of the 14 animals, mechanical resuscitation was continued using the parameters described above until spontaneous circulation was resumed or for a maximum of 30 min from the beginning of resuscitation. In addition to the chest compression, manual interposed abdominal compression was applied to the midabdomen in the remaining seven animals using a folded blood pressure cuff with a pressure of 100–120 mmHg according to the technique described by Ralston *et al.*[1] Abdominal compression was applied to coincide with the second half of the relaxation phase directly preceding the next chest compression. Chest compression and interposed abdominal compression were continued until a spontaneous circulation was resumed or for a maximum of 30 min from the beginning of resuscitation. The animals were allocated for STD-CPR or IAC-CPR using random numbers during the period of arrest.

A spontaneous circulation was considered present when the ECG showed coordinated electrical activity, the systolic blood pressure was more than 90 mmHg, and the diastolic blood pressure more than 40 mmHg for at least 3 min during which neither mechanical measures nor drug therapy were necessary. The lungs of all resuscitated animals were ventilated and the pigs were observed over a 2-h period.

Fibrillatory Arrest

VF was induced in 14 animals by applying an alternating current of 50 Hz and 60 V *via* two subcutaneously

TABLE 1. Asphyxial Arrest. Hemodynamic Measurements during the Postresuscitation Phase

		Postresuscitation Phase (min)						
		5	10	20	30	60	90	100
HR (min)	STD-CPR	—	—	—	—	—	—	
	IAC-CPR	190.0 ± 19.9	165.7 ± 20.6	150.9 ± 20.8	150.6 ± 15.9	155.1 ± 16.1	142.7 ± 15.1	135.6 ± 15.2
AOS (mmHg)	STD-CPR	—	—	—	—	—	—	—
	IAC-CPR	157.4 ± 21.5	131.1 ± 22.1	103.6 ± 13.3	97.1 ± 10.1	100.3 ± 10.1	101.0 ± 7.2	106.6 ± 13.2
AOD (mmHg)	STD-CPR	—	—	—	—	—	—	—
	IAC-CPR	98.3 ± 31.9	79.7 ± 10.0	63.7 ± 20.4	61.4 ± 12.8	65.3 ± 6.4	67.1 ± 15.4	63.3 ± 12.1
End-diastolic	STD-CPR	—	—	—	—	—	—	—
A-V pressure	IAC-CPR	93.3 ± 32.2	75.6 ± 10.4	60.1 ± 20.7	58.0 ± 12.9	62.7 ± 6.5	64.0 ± 15.7	61.7 ± 12.2
difference (mmHg)								
Cardiac index	STD-CPR	—	—	—	—	—	—	—
($ml \cdot min^{-1} \cdot kg^{-1}$)	IAC-CPR	143.8 ± 17.9	140.5 ± 22.9	131.3 ± 15.7	125.7 ± 20.9	118.6 ± 23.5	129.3 ± 19.6	129.9 ± 16.3

Values are mean ± SD.

Abbreviations: STD-CPR = standard cardiopulmonary resuscitation. IAC-CPR = interposed abdominal compression cardiopulmonary resuscitation. HR = heart rate. AOS = aortic systolic pressure. AOD = aortic diastolic pressure.

TABLE 2. Asphyxial Arrest. Arterial Blood Gas Analysis during the Postresuscitation Phase

		Postresuscitation Phase (min)						
		5	10	20	30	60	90	120
Pa_{O_2} (mmHg)	STD-CPR	—	—	—	—	—	—	—
	IAC-CPR	350.3 ± 109.6	277.0 ± 111.5	266.0 ± 124.5	297.7 ± 124.5	346.0 ± 66.6	345.6 ± 72.2	344.4 ± 129.6
Pa_{CO_2} (mmHg)	STD-CPR	—	—	—	—	—	—	—
	IAC-CPR	50.0 ± 7.4	48.6 ± 6.9	47.5 ± 6.7	47.0 ± 9.1	45.1 ± 8.1	44.4 ± 6.9	43.9 ± 7.9
*p*H	STD-CPR	—	—	—	—	—	—	—
	IAC-CPR	7.16 ± 0.08	7.19 ± 0.08	7.21 ± 0.06	7.25 ± 0.09	7.32 ± 0.08	7.37 ± 0.08	7.40 ± 0.08
BEa (mmol/l)	STD-CPR	—	—	—	—	—	—	—
	IAC-CPR	−9.8 ± 3.4	−8.6 ± 3.3	−7.0 ± 3.3	−5.6 ± 2.8	−1.7 ± 2.2	0.1 ± 2.1	2.4 ± 2.1

Abbreviations as in table 1.

placed needle electrodes. Ventilation was stopped at the same time at which the electric shock was applied. After a 4-min period of ventricular fibrillation, seven animals each were managed by STD-CPR or by IAC-CPR for 90 s before defibrillation was carried out. The animals were randomized during the period of arrest. An external countershock of 4 J/kg (Lifepak 4, Physio-Control, Redmond, Washington) was applied. When the initial countershock failed to convert the ventricular fibrillation, the animal received another countershock of 4 J/kg 30 s later. Ventilation, chest compression, and interposed abdominal compression, respectively, were continued during and between attempts at defibrillation. When this second countershock was also unsuccessful, a third and further countershocks were administered at intervals of 90 s using 8 J/kg. After the sixth countershock the energy level was increased to 16 J/kg. Defibrillation was considered to be successful when ventricular fibrillation was changed to a sinus rhythm, electromechanical dissociation, or asystole. Resuscitation success was defined as mentioned above.

DATA ANALYSIS

Heart rate, aortic and right atrial pressures, and cardiac output were measured prearrest, during the period of asphyxia, and 5, 10, 20, 30, 60, 90, and 120 min after successful resuscitation. At the same time, arterial blood samples were obtained for analysis of blood gases and *p*H using a blood gas analyser (IL 1302, Instrumentation Laboratories Lexington, MA) corrected for body temperature.

In tables 1 and 2 intravascular pressures during CPR are displayed 60 s after starting mechanical measures. The Wilcoxon-Mann-Whitney test was used to determine differences between STD-CPR and IAC-CPR. Resuscitation rates for the two groups were analyzed using Fisher's exact test. Differences between intravascular pressures prearrest and during the period of asphyxia within the same study group were tested by analysis of variance with multiple comparison testing (Newman-Keuls). Statistical significance was considered at the $P < 0.05$ level.

Results

ASPHYXIAL ARREST

Four minutes after clamping the endotracheal tube the heart rate, aortic systolic and diastolic pressure and diastolic arteriovenous pressure difference were statistically higher in both groups than prearrest values (table 3). He-

TABLE 3. Asphyxial Arrest. Hemodynamic Measurements Prearrest, during the Period of Asphyxia, and during Cardiopulmonary Resuscitation (CPR)

		Asphyxia (min)				
		Prearrest	2	4	6	CPR
HR (min)	STD-CPR	121.6 ± 12.2	132.4 ± 11.6	164.7 ± 16.8*	62.4 ± 12.8*	—
	IAC-CPR	115.3 ± 9.3	124.9 ± 10.5	152.9 ± 7.5*	65.0 ± 15.5*	—
AOS (mmHg)	STD-CPR	116.8 ± 11.4	136.9 ± 17.4	158.4 ± 8.8*	74.4 ± 10.6*	68.3 ± 4.2
	IAC-CPR	109.7 ± 10.2	126.6 ± 15.5	154.3 ± 8.0*	66.6 ± 9.2*	92.4 ± 6.7†
AOD (mmHg)	STD-CPR	74.6 ± 16.8	89.7 ± 13.4	104.4 ± 18.8*	46.2 ± 6.0*	20.6 ± 2.8
	IAC-CPR	70.7 ± 17.1	82.9 ± 12.7	105.3 ± 14.9*	39.0 ± 7.4*	41.7 ± 4.5†
End-diastolic A-V pressure difference (mmHg)	STD-CPR	70.2 ± 17.0	81.9 ± 14.8	100.7 ± 16.4*	32.8 ± 8.8*	9.8 ± 3.6
	IAC-CPR	68.2 ± 17.5	80.6 ± 12.9	94.2 ± 15.1*	29.7 ± 7.6*	32.1 ± 6.3†
Cardiac index ($ml \cdot min^{-1} \cdot kg^{-1}$)	STD-CPR	149.0 ± 18.6	—	—	—	—
	IAC-CPR	134.3 ± 13.0				

Values are mean ± SD.
Abbreviations as in table 1.

* Statistically different from prearrest value in the same group.
† Statistically different from STD-CPR.

TABLE 4. Asphyxial Arrest. Arterial Blood Gas Analysis Prearrest, during the Period of Asphyxia, and during Cardiopulmonary Resuscitation (CPR)

		Asphyxia (min)			
		Prearrest	2	5	CPR
Pa_{O_2} (mmHg)	STD-CPR	419.4 ± 29.3	24.9 ± 12.3*	7.8 ± 2.6*	208.4 ± 76.8
	IAC-CPR	432.7 ± 49.0	31.1 ± 22.7*	6.0 ± 1.7*	196.3 ± 104.7
Pa_{CO_2} (mmHg)	STD-CPR	36.4 ± 2.4	64.8 ± 6.7*	84.3 ± 18.9*	33.6 ± 6.8
	IAC-CPR	38.1 ± 1.1	66.1 ± 6.9*	95.1 ± 23.2*	32.7 ± 5.4
*p*H	STD-CPR	7.49 ± 0.06	7.24 ± 0.04*	7.17 ± 0.10*	7.22 ± 0.16
	IAC-CPR	7.44 ± 0.04	7.25 ± 0.04*	7.10 ± 0.10*	7.24 ± 0.12
BEa (mmol/l)	STD-CPR	4.3 ± 2.4	3.0 ± 2.1	−1.1 ± 2.6*	−11.0 ± 3.0
	IAC-CPR	2.4 ± 2.8	1.8 ± 2.3	−2.8 ± 2.9*	−11.8 ± 3.8

Values are mean ± SD.
Abbreviations as in table 1.
* Statistically different from prearrest value in the same group.

modynamic depression was observed after 6 min of asphyxia. The hypoxemia led to a severe metabolic and respiratory acidosis (table 4). During prearrest and the period of asphyxia, no significant differences between the groups were found. The duration of asphyxia up to the point at which cardiac arrest occurred was 9.2 ± 1.6 min in the STD-CPR group and 8.8 ± 1 min in the IAC-CPR group (table 5). None of the STD-CPR-treated animals survived despite continuing CPR for 30 min. With IAC-CPR all seven animals could be resuscitated within 240 ± 84 s. Aortic systolic pressure and aortic diastolic pressure were significantly higher during IAC-CPR than with STD-CPR (table 3). End-diastolic arteriovenous pressure difference was 9.8 ± 3.6 mmHg with STD-CPR and 32.1 ± 6.3 mmHg with IAC-CPR ($P < 0.05$). Arterial blood gas analysis was not different between the two groups during CPR (table 4). The observed frequency of successful resuscitation between the two groups was significantly different ($P < 0.05$). All the animals that could be successfully resuscitated survived during the following 2-h period of observation in a stable hemodynamic condition.

Hemodynamic parameters and arterial blood gases in the postresuscitation phase are shown in tables 1 and 2.

FIBRILLATORY ARREST

Successful defibrillation did not always result in a stable spontaneous circulation (table 5). Even when defibrillation was successful, all those animals that were treated with STD-CPR failed to achieve a spontaneous circulation. Aortic blood pressure and end-diastolic arteriovenous pressure differences were significantly higher with IAC-CPR than with STD-CPR (table 6). After an average duration of 244 ± 117 s, restoration of spontaneous circulation was possible in all seven animals treated with IAC-CPR.

There was no significant difference in hemodynamic parameters and arterial blood gas analysis before induction of arrest between the two groups (tables 6 and 7). The measurements in the post-CPR phase are shown in tables 8 and 9.

STD-CPR and IAC-CPR did not induce regurgitation in any of the pigs. No significant gross trauma to intra-abdominal or intrathoracic organs was seen at postexperiment necropsy. In particular we found no lacerations of the liver nor the spleen. In one of the animals resuscitated with IAC a small amount of serosanguinous fluid was found in both pleural cavities.

TABLE 5. Resuscitation Results after Asphyxial and Fibrillatory Arrest

	Asphyxial Arrest		Fibrillatory Arrest	
	STD-CPR	IAC-CPR	STD-CPR	IAC-CPR
Number of pigs	7	7	7	7
Time to arrest (min)	9.2 ± 1.6	8.8 ± 1.0	—	—
Defibrillation successful	—	—	7/7	7/7
Resuscitation time (s)	—	240 ± 84	—	244 ± 117
Success of resuscitation	0/7	7/7*	0/7	7/7*

Values are mean ± SD.
Abbreviations as in table 1.
* Significant difference by Fisher's exact test between the STD-CPR and IAC-CPR groups ($P < 0.05$).

TABLE 6. Fibrillatory Arrest. Hemodynamic Measurements Prearrest and during Cardiopulmonary Resuscitation (CPR)

		Prearrest	CPR
HR (min)	STD-CPR	122.0 ± 14.6	—
	IAC-CPR	119.6 ± 10.4	—
AOS (mmHg)	STD-CPR	125.3 ± 16.2	72.4 ± 2.8
	IAC-CPR	116.1 ± 14.7	98.8 ± 4.7*
AOD (mmHg)	STD-CPR	74.5 ± 18.4	23.2 ± 3.6
	IAC-CPR	76.7 ± 16.6	44.8 ± 5.7*
Diastolic A-V pressure difference (mmHg)	STD-CPR	73.1 ± 18.5	12.4 ± 4.0
	IAC-CPR	75.6 ± 16.9	34.1 ± 6.4*
Cardiac index ($ml \cdot min^{-1} \cdot kg^{-1}$)	STD-CPR	132.7 ± 16.5	—
	IAC-CPR	125.9 ± 19.2	—

Values are mean ± SD.
Abbreviations as in Table 1.

* Statistically different from STD = CPR.

Discussion

When faced with the question as to which animal to use as a model of resuscitation from cardiac arrest there are undoubtedly a number of factors for and against the use of both dogs and the pigs. We used pigs in our study because the transverse shape of a pig's chest more closely resembles the human thorax than does the keel-shaped chest of the dog.[11,12] On the other hand, porcine coronary anatomy differs from that of the dog and from that of many older humans in that there are no well-developed collateral vessels.[13,14] However, dogs also have the disadvantage in that the healthy animals have no arteriosclerotic coronary stenoses and occlusions as are present in the aging human heart.

This study does have experimental limits: it was not possible to blind the investigator to such an obvious intervention as interposed abdominal compression. Despite this drawback, interposed abdominal compression seemed to be the sole reason for improved outcome because it was ensured that external cardiac massage led to a compression of 25% of the anteroposterior chest diameter in all animals.

It may be that mechanical compression with the Thumper® might to some extent have interfaced with chest wall recoil during relaxation phase in both groups. However, one would not expect to find this problem with manually applied massage.

We found a significant difference in the resuscitation success with IAC-CPR and STD-CPR techniques following both asphyxial and fibrillatory cardiac arrest. It has been shown that arteriovenous pressure difference, which correlates with myocardial blood flow,[6] could be used as a prognostic guide for successful resuscitation from cardiac arrest.[4,5] While the definition of coronary perfusion pressure is still controversial, both mid-diastolic and end-diastolic values are used to calculate it.[4,15,16] Because abdominal compression was applied in the later relaxation phase, we have recorded end-diastolic arteriovenous pressure differences, but are well aware of the fact that we have made no correlation with myocardial blood flow.

End-diastolic arteriovenous pressure difference was about 2.5 times greater in the fibrillatory arrest model with IAC than with STD-CPR. This is in agreement with the results of Voorhees *et al.* who found a diastolic arteriovenous pressure difference of approximately 14 mmHg with STD-CPR and approximately 26 mmHg with IAC-CPR.[2] Whole brain and myocardial blood flow increased 12% and 22.7% during IAC-CPR.[8] In these studies each subject was its own control and the authors did not try to regain spontaneous circulation.[2,8]

Two separate mechanisms are postulated to explain the positive effect of interposed abdominal compression. Similar to the effect that occurs during intra-aortic balloon counterpulsation, abdominal compression can compress the abdominal aorta and thus increase aortic diastolic pressure and coronary perfusion pressure. Abdominal compression has substituted for the arterial windkessel that normally maintains aortic diastolic pressure. The second mechanism postulated is an increase in venous return, as demonstrated by enhanced antegrade flow velocity in the inferior vena cava during the period of chest release and abdominal compression.[2]

TABLE 7. Fibrillatory Arrest. Arterial Blood Gas Analysis Prearrest and during Cardiopulmonary Resuscitation (CPR)

		Prearrest	CPR
Pa_{O_2} (mmHg)	STD-CPR	416.7 ± 39.8	295.2 ± 86.4
	IAC-CPR	443.4 ± 37.7	306.8 ± 74.3
Pa_{CO_2} (mmHg)	STD-CPR	36.4 ± 3.6	28.6 ± 7.9
	IAC-CPR	36.7 ± 4.1	29.0 ± 5.2
*p*H	STD-CPR	7.47 ± 0.05	7.36 ± 0.14
	IAC-CPR	7.47 ± 0.06	7.38 ± 0.16
BEa (mmol/l)	STD-CPR	3.5 ± 2.7	−7.6 ± 2.3
	IAC-CPR	3.9 ± 2.2	−7.1 ± 3.4

Values are mean ± SD.
Abbreviations as in table 1.

TABLE 8. Fibrillatory Arrest. Hemodynamic Measurements during the Postresuscitation Phase

		Postresuscitation Phase (min)						
		5	10	20	30	60	90	120
HR (min)	STD-CPR	—	—	—	—	—	—	—
	IAC-CPR	176.9 ± 22.7	146.1 ± 12.8	133.1 ± 6.3	120.1 ± 7.9	124.6 ± 9.5	125.3 ± 13.1	120.6 ± 16.4
AOS (mmHg)	STD-CPR	—	—	—	—	—	—	—
	IAC-CPR	163.7 ± 8.8	126.7 ± 14.2	93.0 ± 10.7	90.4 ± 13.2	101.0 ± 9.0	102.4 ± 14.7	100.9 ± 17.4
AOD (mmHg)	STD-CPR	—	—	—	—	—	—	—
	IAC-CPR	115.7 ± 12.0	80.1 ± 18.5	56.0 ± 14.5	52.7 ± 13.5	60.4 ± 17.4	57.0 ± 15.1	61.3 ± 11.5
End-diastolic A-V pressure	STD-CPR	—	—	—	—	—	—	—
difference (mmHg)	IAC-CPR	113.9 ± 12.3	78.6 ± 18.4	54.5 ± 14.6	52.1 ± 13.7	60.3 ± 17.5	57.4 ± 15.3	60.9 ± 11.5
Cardiac index	STD-CPR	—	—	—	—	—	—	—
(ml · min^{-1} · kg^{-1})	IAC-CPR	116.4 ± 34.9	87.1 ± 24.3	77.7 ± 23.5	74.8 ± 23.0	91.8 ± 19.7	98.9 ± 24.9	98.8 ± 19.8

Values are mean ± SD.

Abbreviations as in table 1.

In dogs, Kern *et al.* found no statistically significant difference in hemodynamics during resuscitation, in initial resuscitation success, or in 24-h survival rate with STD-CPR compared with that following IAC-CPR.[10] The authors presume that the more forceful chest compression used in their study is probably the reason why they could not demonstrate a benefit of IAC-CPR. In preliminary studies (unpublished) a more forceful STD-CPR (the sternum was depressed by 8 cm) did not produce a higher diastolic arteriovenous pressure difference. But recoil of the chest during the relaxation phase became more incomplete as compression force was increased because elasticity was compromised.

A coronary perfusion pressure of 20 mmHg has been reported to correlate with resuscitation success and survival.[4,5,15,17] We believe that we have chosen optimum compression force and sternal displacement in the present study. There are a number of investigations in which, despite optimal mechanical CPR technique, coronary perfusion prsssure is only between 10–15 mmHg.[2,18–20] It is therefore not surprising that none of the our animals could be resuscitated with mechanical measures only. Similar results have been obtained in dogs. In the study by Yakaitis, none of ten dogs could be resuscitated after a 5-min period of VF.[21] Implications with respect to lack of resuscitation success in the STD-CPR groups, are limited by the small number of animals used. Furthermore, there is a striking contrast between the observed poor results of CPR in animal studies and the results obtained in patients. A 0% survival rate is unexpected given the 40% short-term survival of human cardiac arrest victims who receive basic cardiac life support with 4 min and advanced cardiac life support within 10 min of the event.[22]

Even assuming that the pigs in our study and the dogs in the literature had inadequate sternal compression by the Thumper®, the addition of IAC may have changed the resuscitation efforts from inadequate to adequate.

The conflicting results reported in the literature are probably also due to the different duration of abdominal compression. There are no doubts about the fact that continuous abdominal binding during CPR leads to a decrease in the size of the perfused vascular bed by inhibiting subdiaphragmatic flow.[23] Despite a significant increase in arterial diastolic pressure, there was also a similar rise in right atrial pressure, and coronary perfusion pressure did not change significantly.

There could be two reasons why abdominal compression should be preferentially performed in the second half

TABLE 9. Fibrillatory Arrest. Arterial Blood Gas Analysis during the Postresuscitation Phase

		Postresuscitation Phase (min)						
		5	10	20	30	60	90	120
Pa_{O_2}	STD-CPR	—	—	—	—	—	—	—
(mmHg)	IAC-CPR	335.6 ± 66.9	368.6 ± 96.4	424.0 ± 51.4	428.7 ± 60.3	415.1 ± 56.2	408.1 ± 62.5	423.0 ± 60.9
Pa_{CO_2}	STD-CPR	—	—	—	—	—	—	—
(mmHg)	IAC-CPR	50.0 ± 5.8	45.1 ± 5.8	42.4 ± 5.6	40.2 ± 5.7	39.8 ± 3.7	39.8 ± 4.1	40.4 ± 5.3
*p*H	STD-CPR	—	—	—	—	—	—	—
	IAC-CPR	7.26 ± 0.07	7.30 ± 0.08	7.34 ± 0.07	7.36 ± 0.07	7.39 ± 0.05	7.39 ± 0.05	7.39 ± 0.05
BEa	STD-CPR	—	—	—	—	—	—	—
(mmol/l)	IAC-CPR	−4.2 ± 2.7	−3.2 ± 2.6	−2.0 ± 2.8	−1.5 ± 2.8	0.1 ± 2.7	0.1 ± 2.7	0.3 ± 2.2

Values are mean ± SD.

Abbreviations as in table 1.

of the relaxation phase prior to chest compression. First, when external cardiac compression is used, coronary blood flow is probably high during the first half of the relaxation phase after which it decreases.[23] Abdominal compression in the second half of the relaxation phase, which leads to a compression of the abdominal aorta, could increase coronary perfusion and hence myocardial blood flow during this phase with a higher total myocardial blood flow. Second, blood flow below the diaphragm mainly occurs during the early relaxation phase.[24] Pumping of blood into the thorax by abdominal compression can only be effective when there is still a minimal blood flow to the abdominal organs.

When epinephrine is given, IAC-CPR did not improve the results of cardiac resuscitation.[25] The addition of IAC did not increase the frequency of regurgitation as compared with STD-CPR.[25] Based on these observations, IAC-CPR may play a role in basic cardiac life support, provided that a third rescuer is available. Ventilation should be performed in a short pause between the chest compressions, when the abdomen is not compressed.

The results of our study indicate that the potential use of abdominal compression in basic cardiac life support should be investigated further in patients.

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