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Inspiratory impedance threshold valve during CPR

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Abstract

The use of an inspiratory impedance threshold valve (ITV) during cardiopulmonary resuscitation (CPR) should reduce intrathoracic pressure during natural chest recoil or active chest decompression. This might in turn improve venous return and thereby organ blood flow. The haemodynamic effects during both standard CPR and active compression–decompression (ACD)-CPR with and without the ITV, therefore, were studied in a well-established porcine model with cross-over design. Sixteen pigs were randomised to one of four methods initially, changing the method every fifth minute during mechanical chest compression at 100 min^{-1}. Myocardial blood flow was doubled when the valve was added to standard CPR, median (q25–q75) 14 (3–47) versus 27 (9–51) ml min^{-1} 100 g^{-1} ($P = 0.001$). ACD-CPR caused a similar increase, while adding the ITV to ACD-CPR only tended to increase myocardial blood flow ($P = 0.077$). Varying the technique had no effect on cerebral, kidney or carotid blood flow, coronary perfusion pressure, expired CO_2 concentrations or blood gases. The valve is a promising new tool in CPR, but more independent studies of the device are needed.

Keywords: Inspiratory impedance threshold valve; CPR; Pigs; Organ blood flow; Haemodynamics

1. Introduction

In the new international guidelines for cardiopulmonary resuscitation (CPR) the principle of hindering passive inspiration by an inspiratory threshold valve (ITV) during the decompression part of chest compression/decompression is introduced: 'the impedance threshold valve is acceptable as an adjunct to be used with a cardiac compression/decompression device to augment haemodynamic parameters (Class IIb)' [1]. Class IIb is defined as an 'acceptable, safe, and useful' alternative to standard CPR. The ITV is connected to the tracheal tube and is always open during expiration, but prevents inspiratory flow during the decompression phase of CPR. Using the valve, therefore, should produce lower intrathoracic pressures during both passive and active chest decompressions. Since it is believed that a decreased intrathoracic pressure is associated with enhanced venous return to the heart, as seen with active compression–decompression (ACD)-CPR, this should improve the haemodynamic status of the patient.

We are aware of only four publications [2–5] dealing with this valve during CPR, all with the inventor as one of the authors. In agreement with his opinion, we have, therefore, conducted an independent study. We hypothesised that hindering passive inspiration secondary to the decompressions would improve vital organ blood flow both during standard CPR and ACD-CPR due to a lower intrathoracic pressure. Thus, the aim of the study was to compare the haemodynamics during standard CPR and ACD-CPR with and without the valve with all methods being performed in each animal for a paired comparison to avoid the influence of inter-subject variability.

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2. Materials and methods

2.1. Animal preparation

The study was approved by the Norwegian Council for Animal Research and performed in a well-established pig model [6–8]. Sixteen healthy pigs (Norwegian landrace, *Sus scropha domestica*) of either sex (20–24 kg, aged 6–8 weeks) were fasted overnight with free access to water. The pigs were anaesthetised with ketamine 30 mg kg^{-1} and atropine 1 mg intramuscularily (i.m.) in the pen. In the laboratory a catheter was placed in an ear vein for infusion of 30 ml kg^{-1} h^{-1} warmed Ringer acetate throughout the preparation period. A target mean aortic pressure (MAP) was set at 75 mmHg < MAP < 120 mmHg. The pigs were placed supine with the chest in a U-shaped trough and the limbs secured to prevent lateral displacement of the chest during CPR. A specially constructed pig mask was used for the inhalation of anaesthesia with end-tidal concentration of 15% desflurane (Suprane®, Baxter) before a tracheotomy was performed. Circular sutures placed firmly around the trachea sealed the airway. To achieve the reported MAC level for pigs [9], anaesthesia was maintained with 10% end-tidal desflurane measured in a sidestream by a multigas monitor (Datex Capnomac Ultima™, Helsinki, Finland) and was individually adjusted if required. Ventilation was performed with a Siemens Servo Respirator 900 B, with F_iO_2 of 0.5, a frequency of 16 min^{-1} and an initial tidal volume of 15 ml kg^{-1} adjusted to maintain the end-tidal CO_2 ($ETCO_2$) at 5.0 ± 0.5 kPa as measured by the gas monitor. Urine was drained continuously through a cystostoma, and the rectal temperature was kept at 39.0 ± 0.5 °C, using a heating pad.

A 7F micro-tip pressure transducer catheter (Model SPC 470, Millar Instruments, Houston, TX, USA) was introduced into the right femoral artery and advanced to the descending aorta, at the level of the heart, for arterial pressure recordings. Another 7F micro-tip pressure transducer catheter (Millar Instruments) was introduced into the left ventricle through the left common carotid artery to measure the left ventricular pressure and to infuse radioactively labelled microspheres into the ventricle through the sideport. To assess tissue blood flow at least 6.0×10^5 microspheres (^{113}Sn, ^{57}Co, ^{103}Ru, ^{95}Nb or ^{46}Sc; NEN-Trac™, Dupont) with a diameter of 15.5 μm suspended in 8-ml saline were infused at a rate of 3 ml min^{-1} for 130 s. Arterial blood was continuously withdrawn by a pump (Harvard Instruments, Cambridge, MA) from the aorta during and after each microsphere infusion at a rate of 1.5 ml min^{-1} (3 ml min^{-1} for the baseline infusion) for 280 s (190 s for the baseline situation) via heparinised polyethylene tubing 90 (ClayAdams, Becton Dickinson, New Jersey; internal diameter 0.78 mm) with multiple sideholes. Following this withdrawal of an additional 1 ml of blood was taken. The catheter was advanced 15 cm into the left femoral artery from the inguinal ligament and was flushed with saline between each sampling. No additional intravenous (i.v.) heparin was given.

Two 7F Swan-Ganz® catheters (model 131HF7, Baxter Healthcare Corporation, Irvine, CA, USA) were placed in the right atrium, one via the right femoral vein and one via the right external jugular vein for pressure recordings and mixed venous blood sampling. A polyethylene catheter was introduced into the right axillary artery for arterial blood sampling. After ligation of all visible branches of the right common carotid artery except the internal carotid artery, a transit time perivascular flowprobe (model 3SB880, Transonic Systems Inc, Ithaca, NY, USA) was placed on the artery. All invasive catheters were introduced using a cut-down technique.

The oesophageal pressure at the level of the heart was measured by a cylindrical air-containing rubber balloon, 5-cm long and 3.4-cm in diameter, glued to the end of a 7F stiff catheter with an open end and multiple side holes in the section inside the balloon [10].

The Swan-Ganz and oesophageal catheters were connected to fluid filled transducers (Statham® P23Dd, Gould Instruments, Hato Rey, Puerto Rico).

Inspiratory airway flow, respiratory pattern and $ETCO_2$ were measured continuously using a combined differential pressure pneumotach flow sensor and solid state CO_2 sensor in combination with a respiratory profile monitor (CO_2SMO Plus!, Novametrix Medical System Inc., Wallingford, CT, USA). The flow/CO_2 sensor was inserted in the circuit between the ventilator and an antibacterial filter connected to the tracheal tube. The monitor was connected on-line with a computer into which the data were downloaded, stored and analysed using Analysis Plus! Software package (Novametrix medical System Inc.). Pressures and carotid flow signals were sampled using PC-based real time data acquisition hardware (DaqBoards™ model 200A, IOtech, Inc., Clevland, OH, USA) supported with software for DASYLab version 5.1 (Datalog, National Instruments company, Moenchengladbach, Germany) and printed on an eight-channel thermal array recorder model TA 11 (Gould Instrument Systems, Inc., Ohio, USA).

A 6.0×11.0 cm hard rubber plate was fastened to the sternum with six screws and two wire sutures. All catheter placements were verified by pressure tracings and post mortem examination. Calibration of all catheters and the flow probe were made at the beginning of each experiment.

2.2. Machinery

Standard and active decompression were achieved using a modified automatic hydraulic double-acting chest compression/decompression device (Heartsaver 2000®, Medreco, Bodø, Norway) with equal compression–relaxation phases. The piston movement was photoelectrically controlled. Compression depth was set at 4 cm and active decompression at 2 cm, as recommended by Wik et al. [8]. Active decompression was accomplished by attaching the piston to the hard rubber plate secured to the sternum; 5–10 s was needed to attach and detach the connection. The piston was adjusted to touch the surface of the rubber plate at the end of passive decompression to ensure continuous compression and decompression depths were unaffected by changes in the chest configuration during the study.

2.3. Experimental protocol

After instrumentation, baseline (pre-VF) measurements were obtained for all variables. The i.v. infusion, heating, inhalation anaesthesia and ventilation were thereafter discontinued. Ventricular fibrillation (VF) was induced by a trans-thoracic current (using 90 V AC) for 3 s and confirmed by ECG changes and an abrupt fall in carotid flow. After 3 min of VF, 4 cm standard mechanical chest compressions were begun at a rate of 100 min^{-1} for 30 s to allow the initial changes in chest configuration to occur before the zero point for the piston was set.

Manual bag (Laerdal, Stavanger, Norway) ventilation with 100% O_2 was interposed after every sixth compression without interruption of the compression–decompression cycle. A previously described impedance threshold valve [2,11] (ITV Resusci-Valve, provided by CPR_xLLC, Minneapolis, USA) was placed between the Laerdal valve and the pneumotach flow sensor. Briefly explained the valve does not impede the expiratory airflow, only passive inspiratory flow secondary to natural chest recoil or active decompression. It can be set either in an open mode (no impedance) or an impedance mode where a pressure above 36–37 cm H_2O is required to open the valve, after which the valve offers virtually no resistance. This was verified in vitro both before and after the experiments.

CPR was performed using one of four different methods varied in a randomised, crossover design with each method being used for 5 min. The methods were, Standard CPR with the valve in impedance mode, standard CPR with the valve in open mode, ACD-CPR with the valve in impedance mode and ACD-CPR with the valve in open mode. The person ventilating the pig was blinded to the $ETCO_2$ values.

The microsphere infusion for measuring organ blood flows was initiated 20 s after the beginning of each CPR method. Carotid flow and all pressures were recorded at 120 and 240 s after the start of each CPR method. Arterial and mixed venous blood gases were sampled between 150 and 210 s, recorded as 210 s. $ETCO_2$ and airway flow were also recorded simultaneously and organ blood flow measured as described above. The randomisation list was written so that performance of the different methods was evenly spread as the first, second, third or fourth method.

After completion of the experiment, the CPR was stopped and the pig died. All pigs were autopsied to check for damage to the rib cage and internal organs and for verification of catheter placements. Samples from the following organs were obtained, weighed and placed into counting vials: heart (four regions), brain (two regions) and kidneys (one region).

2.4. Calculations

All haemodynamic pressures, oesophageal pressure and carotid flow were analysed by exporting the raw data into a specially constructed programme designed in a mathematics software package (MATLAB®, The MathWorks, Inc., Natick, MA, USA).

The chest compression rate during CPR was set at 100 min^{-1}. The sampling frequency for the pressure and carotid artery signal was 200 Hz, hence there were 120 sampling points for each compression–decompression cycle. As the duty cycle was 50:50, the decompression phase was defined as the period $\pm$ 30 sampling points from the middle point between two peak compressions. Early-, mid- and late-decompression were then defined as being the first, mid and last 20 sampling points of this period.

Coronary perfusion pressure (CPP) was calculated as the difference between thoracic aortic and right atrial pressures in the decompression phase using an electronic subtraction unit.

Tissue blood flow was measured with radioactively labelled microspheres according to the technique described by Heymann et al. [12] with modifications as described by Iversen and Nicolaysen [13] and evaluated during CPR by others [6,8,14,15]. Radioactivity of tissue and arterial blood was measured in a gamma counter (Auto-Gamma 5220, Packard, IL) and corrected for overlap between the different isotopes. Regional blood flow per 100 g was calculated based on the radioactivity in tissue and in the reference arterial blood [13]. We checked that insignificant radioactivity was present in the arterial blood taken directly after the reference sampling.

2.5. Statistical analysis

Data are presented as mean $\pm$ S.D. when normality test was passed and median (25–75 percentile) when it

failed. Comparisons were made between the four CPR methods. Each pig served as its own control and for continuous parametric data the one-way ANOVA repeated measures with pairwise multiple comparisons was used. For non-normally distributed data the Friedman one-way ANOVA repeated measures was used. Statistical significance was considered to be at the $P < 0.05$ level.

3. Results

Ventricular fibrillation (VF) was obtained in 15 pigs by the first trans-thoracic electrical shock, while one pig needed two electrical shocks due to insufficient electrode–skin contact. Seven pigs were excluded. Five of these were due to problems with the sampling catheter in the left femoral artery, and two had major haemorrhage due to rupture of thoracic vessels. In the remaining nine pigs no gross liver, lung, heart or other visceral damage was observed at post mortem autopsy although one had findings consistent with pericardial inflammation on inspection and one had a fracture of the sternum at the upper border of the rubber plate. Despite this the latter had adequate invasive pressures and carotid flow and no internal organ damage on autopsy.

Oesophageal pressure was not recorded in one of the pigs due to malfunction of the transducer amplifier. Right atrial pressure and coronary perfusion pressure are given for eight pigs due to inadequate recordings in one pig, probably due to clotting of the catheter. Arterial blood gases were not obtained in one pig due to unsuccessful sampling from the axillary artery.

One minute after start and after each change in the CPR method, blood pressures, carotid flow and $ETCO_2$ had stabilised. The total CPR time was 24 min 41 ± 47 s. The four different CPR methods were evenly distributed among the nine pigs as being performed first, second, third or fourth.

In most animals there were periodic attempts of spontaneous ventilation during CPR, easily detected in the respiratory profile program (Analysis Plus!) and paralleled with clinical observations of gasping and variations in the pressures and carotid flow curve. The respiratory profile program documented the function of the impedance valve (Fig. 1).

3.1. Blood flows (Table 1)

Mean carotid blood flow was approximately 35% of pre-arrest control during CPR with no significant differences between the various CPR methods.

Mean cerebral cortical blood flow was 59 ± 11 ml min^{-1} 100 g^{-1} pre-VF and was between 53 and 61% of this during CPR with no significant differences between the methods.

Mean myocardial blood flow was 61 ± 26 ml min^{-1} 100 g^{-1} pre-VF and was reduced to between 39 and 59% of this figure during CPR with significantly lower flow with standard CPR and with no significant differences between the three other methods.

Mean renal blood flow was 245 ± 45 ml min^{-1} 100 g^{-1} pre-VF and was reduced to between 11 and 14% of this figure during CPR with no significant differences between the methods.

3.2. Measured pressures (Table 2)

Mean aortic pressure was 83 ± 6 mmHg pre-VF and was halved during CPR, with no significant difference between the four methods for mean, peak compression or any decompression phase.

Mean right atrial pressure was 8 ± 8 mmHg pre-VF and increased to 42 ± 15 mmHg during standard CPR. Both with and without the impedance valve, early decompression right atrial pressures were significantly lower during ACD-CPR than during standard CPR. Mean decompression right atrial pressure with the valve was significantly lower during ACD-CPR than during standard CPR with valve.

Mean left ventricular pressure was 44 ± 7 mmHg pre-VF and was reduced by 25% during CPR with no significant difference between the four methods for mean and peak compression. It was significantly lower during ACD-CPR with the valve than without the valve for mean-, early- and mid-decompression and also lower than standard CPR with the valve in early decompression.

Mean oesophageal pressure was 1 ± 2 mmHg pre-VF and not significantly different during standard CPR (1 ± 2 mmHg). The peak compression oesophageal pressure was significantly higher during ACD-CPR than standard CPR both with and without the impedance valve. During mean-, early- and mid-decompression the oesophageal pressure was significantly lower for ACD-CPR with the valve than for any of the other three methods.

3.3. Calculated pressures

Despite some variations in the right atrial pressures, there were no significant differences in mean coronary perfusion pressures during the various decompression phases between the four methods (Table 3).

3.4. Expired CO_2 concentrations and blood gases

There were no differences in end-tidal CO_2 or blood gases between periods with different CPR methods (Table 4).

4. Discussion

In this study myocardial blood flow nearly doubled when an ITV was added to standard CPR so that passive inspiration was hindered during chest decompression. Active decompression CPR caused a similar increase, with an insignificant ($P = 0.077$) further increase in myocardial blood flow when the ITV was added. Varying the technique had no effect on cerebral or kidney blood flow.

There are only four previous publications dealing with the impedance threshold valve during CPR [2–5], and the present is the first independent study without the patent holder as one of the authors. One of the four

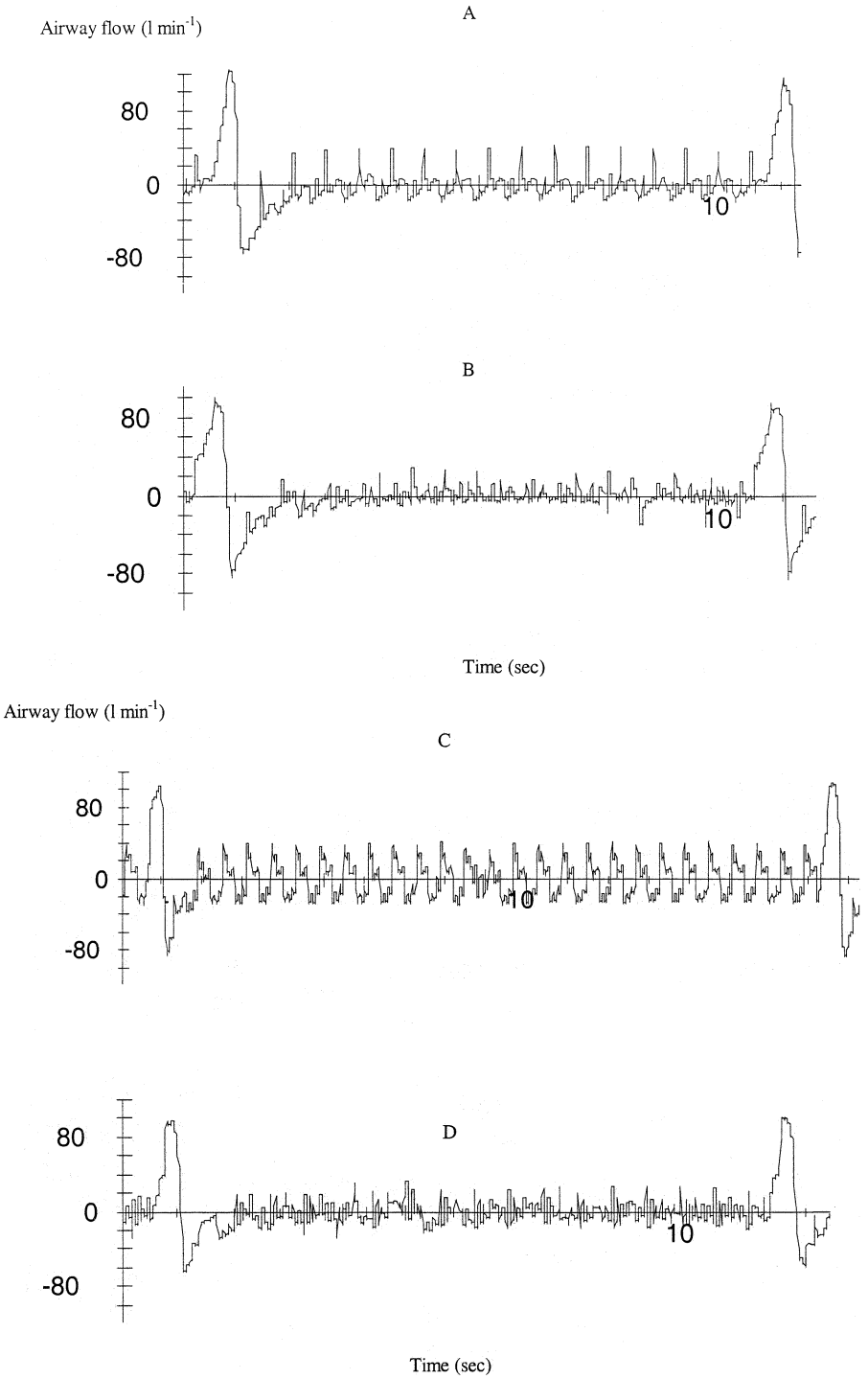

Fig. 1. Airway flow (l min^{-1}) generated with standard CPR (A), standard CPR with valve (B), ACD-CPR (C) and ACD-CPR with valve (D). Inspiratory flow is positive deflections. Passive airway flow is shown between two manual ventilations in four different runs. Inspiratory flow is damped when the valve is present (B, D), demonstrating the function of the valve.

Table 1
Internal carotid artery blood flow (ml min^{-1}) and regional blood flow (ml min^{-1} 100 g^{-1}) during pre-VF and generated with standard-CPR (S-CPR) and ACD-CPR with and without the inspiratory impedance valve

	Pre-VF	S-CPR	S-CPR w. valve	ACD-CPR	ACD-CPR w. valve
Carotid flow	135 ± 29	44 ± 26	45 ± 31	47 ± 21	52 ± 18
Brain (cortex)	59 ± 11	35 ± 31	36 ± 23	31 ± 18	36 ± 17
Heart	53 (40–76)	14 (3–47)	27 (9–51)[a]	29 (9–49)[a]	31 (17–49)[a]
Kidney	245 ± 45	28 ± 27	30 ± 23	35 ± 31	29 ± 15

Data presented as mean ± S.D. or median (25–75 percentile).
[a] $P = 0.001$ vs. S-CPR.

previous studies was a small human trial [2], while three were in pigs with standard-CPR [4], ACD-CPR [3] and one combined ACD-CPR with positive end-expiratory pressure [5].

The near doubling of myocardial blood flow with the valve during standard CPR appears slightly higher than the 39% increase reported by Lurie et al. [4]. It is interesting to note that in both studies the increase occurred without a significant effect on coronary perfusion pressure during the decompression phase, 'diastole'. While myocardial blood flow is normally considered to correlate with coronary perfusion pressure during CPR [16,17], both studies indicate that such a clear correlation is not present when the impedance valve is added. Mean diastolic coronary perfusion pressures were similar in the present study to that of Lurie et al. [4], 13 and 15 mmHg, respectively, for standard CPR without the valve and 13 mmHg in both studies for standard CPR with the valve.

While Lurie et al. [4] also found a positive effect on cerebral blood flow with the valve during standard CPR, we found no such effect. The reason for this difference is uncertain. It should be noted, however, that the increase reported by Lurie et al. [4] was small, from 19 ± 2 to 23 ± 2 (mean ± S.E.M.) ml min^{-1} $100g^{-1}$, with a significant difference only at two of four measurement points, and with half the animals showing a steady reduction in flow with time without any apparent effect of the valve. It could also be speculated that we had already obtained the highest cerebral blood flow possible with external CPR without the valve with 60% of pre-arrest cerebral blood flow versus 49% reported by Lurie et al. [4].

Contrary to Lurie et al. [3] we found no significant effect on myocardial or cerebral blood flow of using the valve during ACD-CPR. We cannot exclude that the lack of an effect on myocardial blood flow was due to a type II error as the P-value was 0.077 for these nine pigs. In addition there were significant differences in study design. While we used a cross-over design with each pig serving as its own control, Lurie et al. [3] compared two groups of animals. Furthermore autopsy for verification of catheter position or damage to the thoracic cage or the internal organs was not fully reported, and the number of excluded pigs, with reasons, was not documented [3,4]. There also seem to be some peculiarities in their results. While they stated that left ventricular myocardial blood flow was 56% of pre-arrest control during ACD-CPR with the valve, it appears to be only 44% calculated from the mean reported flow data (176 ± 38 vs. 77 ± 9 ml min^{-1} 100 g^{-1}). In addition the mean myocardial blood flow during ACD-CPR without the valve was only 23% of pre-arrest control while we recorded 55% of pre-arrest control.

We cannot explain confidently the difference in cerebral blood flow results in the two studies. The results by Lurie et al. [3] are somewhat difficult to interpret, however, as they report mean cerebral blood flows during ACD-CPR with the valve that were higher than pre-arrest controls. With the highest possible cerebral perfusion pressures (assuming intracranial pressures always below central venous pressures) of 11–17 mmHg during the compression phase and 27–31 mmHg during the decompression phase [3], well below the lower limit of cerebral autoregulation, it is difficult to understand how the cerebral blood flow could be higher than normal.

While we found no change in coronary perfusion pressure with the valve during ACD-CPR, Lurie et al. found a significant increase [3]. We cannot readily explain this difference in results either, but it should be noted that they only found a significant difference after 2 min, not after 7 min.

There are other possible reasons for the differences in results between Lurie et al. [3,4] and our present study. While we used pigs of both genders, they used only females that were approximately 4 weeks older and 10 kg heavier than those in the present study. It cannot be excluded that this might affect the haemodynamics.

Pentobarbital anaesthesia was used in both previous studies [3,4] versus desflurane in the present. Pentobarbital has a very long half-life, while desflurane, which has a very short half-life [18] was stopped at the time of cardiac arrest. Thus, the anaesthesia protocol in porcine CPR studies may have significant impact on haemodynamic variables [19,20]. An advantage of discontinuing a short-acting drug is that the model might mimic the non-anaesthetised cardiac arrest patient more closely.

Cardiac arrest is known to impede microcirculation due to activation of blood coagulation and formation of microemboli [21,22]. Heparin i.v., which was given before induction of VF in the previous studies [3,4] has previously been reported to improve recovery [23].

The radioactively labelled microspheres were injected as a bolus in the studies of Lurie et al., whereas we used a constant infusion rate over 2 min. A bolus injection will provide a more instantaneous picture of perfusion rates than a continuous one, which thus measures average flow more reliably.

With low cardiac output the reference sampling period must be prolonged and it should be controlled so that at the end of the period there are no further microspheres passing the end of the reference catheter. This was controlled in the present study by taking an additional sample through the catheter and checking that this sample did not contain significant radioactivity.

The compression rates were different, 80 versus 100 min^{-1} in the present study, the latter being recommended in the present guidelines [24] and we have previously found better flows with these higher rates in pigs [7]. The sternal displacement with compression and active decompression were also controlled differently, 25 and 10% of the resting anterior–posterior (AP) diameter of the chest wall respectively versus 4 cm down and 2 cm up from neutral position in the present study. The chosen compression-/active decompression length of 4/2 cm in the present study were based on

Table 2
Aortic, right atrial, left ventricular and oesophageal pressures (mmHg) during standard-CPR (S-CPR) and ACD-CPR with and without the inspiratory impedance valve

	S-CPR	S-CPR w. valve	ACD-CPR	ACD-CPR w. valve
Aortic pressure				
Mean	40 (35–56)	40 (33–62)	42 (31–50)	43 (32–53)
Peak compression	109 ± 50	116 ± 56	104 ± 37	119 ± 61
Mean decompression	24 ± 11	24 ± 7	24 ± 11	23 ± 7
Early decompression	25 ± 14	26 ± 11	21 ± 12	19 ± 9
Mid decompression	23 ± 12	23 ± 9	25 ± 10	23 ± 5
Late decompression	24 ± 9	24 ± 7	27 ± 10	28 ± 6
Right atrial pressure				
Mean	42 ± 15	47 ± 17	32 ± 8[a,b]	34 ± 10[a]
Peak compression	138 ± 57	156 ± 60	121 ± 37	139 ± 47
Mean decompression	10 ± 4	11 ± 5	10 ± 3	7 ± 4[c]
Early decompression	11 ± 7	15 ± 10	4 ± 5[a]	1 ± 5[a,b]
Mid decompression	9 ± 6	9 ± 7	11 ± 5	8 ± 3
Late decompression	10 ± 5	10 ± 5	13 ± 4	13 ± 5
Left ventricular pressure				
Mean	33 (31–49)	34 (27–52)	34 (28–36)	31 (23–38)
Peak compression	122 ± 42	131 ± 56	112 ± 29	122 ± 43
Mean decompression	10 ± 5	10 ± 4	11 ± 6	4 ± 3[d]
Early decompression	15 (8–20)	12 (10–21)	5 (1–10)	−2 (−6 to 0)[a,b,e]
Mid decompression	7 ± 5	6 ± 3	11 ± 6	4 ± 2[f]
Late decompression	8 ± 3	8 ± 5	16 ± 12	12 ± 8
Oesophageal pressure				
Mean	1 ± 2	1 ± 3	1 ± 3	0 ± 2
Peak compression	8 ± 4	9 ± 3	11 ± 3[a]	13 ± 4[a,b]
Mean decompression	−2 ± 3	−1 ± 4	−2 ± 3	−5 ± 3[g,h,i]
Early decompression	−1 (−3 to 0)	−2 (−5 to 1)	−4 (−5 to 0)	−5 (−9 to −3)[a,b,e]
Mid decompression	−1 (−4 to 0)	−3 (−5 to 1)	−4 (−5 to 0)	−6 (−10 to −4)[j,k,f]
Late decompression	−2 ± 3	−2 ± 4	0 ± 4	−3 ± 3

Data presented as mean ± S.D. or median (25–75 percentile).
[a] $P = 0.001$ vs. S-CPR w. valve.
[b] $P = 0.001$ vs. S-CPR.
[c] $P = 0.04$ vs. S-CPR w. valve.
[d] $P = 0.02$ vs. ACD-CPR.
[e] $P = 0.001$ vs. ACD-CPR.
[f] $P = 0.01$ vs. ACD-CPR.
[g] $P = 0.003$ vs. S-CPR.
[h] $P = 0.003$ vs. S-CPR w. valve.
[i] $P = 0.003$ vs. ACD-CPR
[j] $P = 0.01$ vs. S-CPR
[k] $P = 0.01$ vs. S-CPR w. valve.

Table 3
Calculated coronary perfusion pressure (CPP) (mmHg) at various phases in decompression (aortic-right atrial pressure) during standard-CPR (S-CPR) and ACD-CPR with and without the inspiratory impedance valve (mean ± S.D.)

	S-CPR	S-CPR w. valve	ACD-CPR	ACD-CPR w. valve
Mean decompression	13 ± 13	13 ± 9	14 ± 13	15 ± 8
Early decompression	13 ± 14	11 ± 10	16 ± 13	17 ± 10
Mild decompression	13 ± 13	14 ± 10	13 ± 14	15 ± 8
Late decompression	14 ± 14	15 ± 9	13 ± 11	14 ± 7

Table 4
End-tidal CO_2 ($ETCO_2$), arterial and mixed venous blood gases during pre-VF and the four different CPR methods (mean ± S.D.)

Blood gas	Pre-VF	S-CPR	S-CPR w. valve	ACD-CPR	ACD-CPR w. valve
$ETCO_2$ (kPa)	4.6 ± 0.3	2.5 ± 1.1	2.4 ± 1.1	2.3 ± 0.8	2.4 ± 0.8
Arterial					
pH	7.47 ± 0.04	7.32 ± 0.1	7.30 ± 0.1	7.32 ± 0.1	7.32 ± 0.1
p_{CO_2} (kPa)	4.9 ± 0.6	5.4 ± 1.7	6.1 ± 1.9	5.2 ± 1.8	5.6 ± 1.6
p_{O_2} (kPa)	31.0 ± 8.1	29.3 ± 23.9	21.6 ± 25.5	26.6 ± 20.6	23.4 ± 20.9
BE (mmol l^{-1})	3.1 ± 2.0	−5.9 ± 3.8	−5.4 ± 2.5	−6.6 ± 4.5	−5.4 ± 3.0
Mixed venous					
pH	7.42 ± 0.04	7.20 ± 0.1	7.17 ± 0.1	7.18 ± 0.1	7.18 ± 0.1
p_{CO_2} (kPa)	6.0 ± 0.5	8.6 ± 3.0	9.2 ± 1.6	9.1 ± 3.1	9.1 ± 1.4
p_{O_2} (kPa)	6.1 ± 0.7	3.6 ± 1.9	2.7 ± 0.7	4.0 ± 1.7	3.0 ± 0.6
BE (mmol l^{-1})	3.2 ± 2.8	−5.4 ± 4.0	−5.9 ± 2.4	−6.3 ± 4.1	−5.0 ± 3.4

previous positive haemodynamic effects of ACD-CPR versus standard CPR in the same model with no positive effects of increasing the compression or decompression lengths [8].

In the previous study of ACD-CPR [3], there is no description of baseline blood pressures, oesophageal pressure or other indexes of intrathoracic pressure. In the previous study of standard CPR [4] the pressure in the distal port of the tracheal tube was measured, but this cannot be extrapolated automatically to intrathoracic pressure in situations where there is flow in the bronchial tree, while a previous study has found a good correlation between oesophageal pressure and the pericardiac pressure [10].

The effect of the valve or not was compared after 2 and 7 min in the ACD-CPR trial [3]. In the standard CPR trial [4] it is stated, 'each time the ITV (inspiratory impedance threshold valve) was added to the respiratory circuit, the mean CPP remained constant'. This statement is not supported by the data, as the CPP was significantly reduced one out of three times according to Figure 2 in the paper.

End-tidal CO_2 and arterial blood gases (ABG) did not change with ACD-CPR alone as compared with standard CPR, in contrast to the work by Cohen et al. [25]. The absence of an increase in end-tidal CO_2 with increased flow, also previously reported from our laboratory [8], is probably due to the increased passive ventilation caused by the active decompression [8]. Thus end-tidal CO_2 is not a good parameter for evaluating changes in circulation when active decompression is added during CPR. When the impedance valve is activated, ventilation is affected, and this could explain the lack of an increase in end-tidal CO_2 if the total body blood flow increased in parallel to the increase in coronary blood flow. As expected, the arterial p_{CO_2} tended to increase, but not significantly when the valve was added both with standard CPR and ACD-CPR. As p_{CO_2} increases only slowly (calculated to approximately 0.5 kPa min^{-1} with discontinued ventilation during normal circulation and CO_2 production [26]), it is not surprising that there was no significant change in the 5–6 min each study period lasted with probably less CO_2 production than normal due to anaerobic conditions. Likewise, arterial pH and p_{O_2} did not decrease with the expected reduction in ventilation with the valve during these short study periods. This is in contrast to the findings by Lurie et al. with reduced arterial pH and p_{O_2} and higher p_{CO_2} values with the valve [3,4]. The reason is probably that the study periods in those experiments were longer, as they found no significant differences in blood gases with and without the valve during the first 2–3 min [3,4]. Airway flow measurements showed that the passive inspiratory deflections were damped by the valve (Fig. 1), indicating that the valve had functioned.

In conclusion the impedance threshold valve enhanced myocardial blood flow during standard CPR,

but not during ACD-CPR, and there was no effect on carotid or cerebral blood flow. The valve is a promising new tool in CPR, but more independent studies of the device are needed.

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Portuguese Abstract and Keywords

O uso de uma válvula com limiar de impedância inspiratória (ITV) durante a Reanimação Cardio-Pulmonar (RCP) deve reduzir a pressão intratorácica durante a retracção torácica natural ou descompressão torácica activa. Isto pode melhorar o

retorno venoso e portanto o fluxo sanguíneo dos orgãos. Os efeitos hemodinâmicos durante a RCP standard e durante a compressão-descompressão activa (ACD)-CPR com ou sem ITV foram estudados num modelo porcino com um desenho cruzado. Dezasseis porcos foram randomizados inicialmente para um de quatro métodos, mudando o método cada 5 minutos durante a compressão torácica a100 min. O fluxo sanguíneo miocárdico foi duplicado quando a válvula foi adicionada à RCP standard, mediana (q25–q75) 14 (3–47) versus 27 (9–51) ml min^{-1} 100 g^{-1} ($P = 0.001$). A ACD-CPR causou um aumento comparável enquanto a adição de ITV a ACD-CPR apenas tende a aumentar o fluxo sanguíneo miocárdico ($P = 0.077$). A variação da técnica não tinha qualquer efeito no fluxo sanguíneo cerebral, renal ou carotídeo, na pressão de perfusão coronária, na concentração de CO_2 expirado ou nos gases sanguíneos. A valvula é uma nova arma promissora na RCP, mas são necessários mais estudos independentes dos aparelhos.

Palavras chave: Valvula de limiar de impedância inspiratória; RCP; Perfusão de orgãos; Porco; Hemodinâmica

Spanish Abstract and Keywords

El uso de una válvula de umbral de impedancia inspiratoria (ITV) durante la resucitación cardiopulmonar (RCP)debería reducir la presión intratorácica durante la recuperación natural del tórax o la descompresión activa del tórax. Esto podría mejorar el retorno venoso y con ello el flujo sanguíneo a los distintos tejidos. Estudiamos los efectos hemodinámicos con y sin ITV durante RCP estándar y RCP con compresión y descompresión activa RCP-(ACD) en un modelo porcino bien establecido y con diseño cruzado. Dieciseis cerdos fueron randomizados a uno de 4 modelos inicialmente, cambiando el método cada cinco minutos, durante masaje cardíaco mecánico a 100 por $minuto^{-1}$. El flujo sanguíneo miocárdico se duplicaba cuando se agregaba la válvula al RCP estándar, con una mediana (q25–75) de 14 (3–47) versus 27 (9–51) ml min^{-1} 100 g^{-1} ($P = 0.001$). La RCP-ACD causó un aumento similar, al agregarle ITV a la RCP-ACD solo tendió a aumentar el flujo miocárdico ($P = 0.077$). Las variaciones de la técnica no tuvieron efecto en el flujo cerebral, renal, ni carotídeo, ni en la presión de perfusión coronaria, concentración de CO_2 expirado o gases en sangre. La válvula es una promisoria nueva herramienta para RCP, pero son necesarios mas estudios independientes de este dispositivo.

Palabras clave: Valvula de umbral de impedancia inspiratoria; RCP; Cerdos; Flujo sanguíneo a organos; Hemodinamia