EXPERIMENTAL PAPER

Effect of chest compressions only during experimental basic life support on alveolar collapse and recruitment[☆]

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Summary

Aim: The importance of ventilatory support during cardiac arrest and basic life support is controversial. This experimental study used dynamic computed tomography (CT) to assess the effects of chest compressions only during cardiopulmonary resuscitation (CCO-CPR) on alveolar recruitment and haemodynamic parameters in porcine model of ventricular fibrillation.

Materials and methods: Twelve anaesthetized pigs (26 ± 1 kg) were randomly assigned to one of the following groups: (1) intermittent positive pressure ventilation (IPPV) both during basic life support and advanced cardiac life support, or (2) CCO during basic life support and IPPV during advanced cardiac life support. Measurements were acquired at baseline prior to cardiac arrest, during basic life support, during advanced life support, and after return of spontaneous circulation (ROSC), as follows: dynamic CT series, arterial and central venous pressures, blood gases, and regional organ blood flow. The ventilated and atelectatic lung area was quantified from dynamic CT images. Differences between groups were analyzed using the Kruskal—Wallis test, and a $p < 0.05$ was considered statistically significant.

Results: IPPV was associated with cyclic alveolar recruitment and de-recruitment. Compared with controls, the CCO-CPR group had a significantly larger mean fractional area of atelectasis ($p = 0.009$), and significantly lower PaO_2 ($p = 0.002$) and mean arterial pressure ($p = 0.023$). The increase in mean atelectatic lung area observed during basic life support in the CCO-CPR group

remained clinically relevant throughout the subsequent advanced cardiac life support period and following ROSC, and was associated with prolonged impaired haemodynamics. No inter-group differences in myocardial and cerebral blood flow were observed.

Conclusion: A lack of ventilation during basic life support is associated with excessive atelectasis, arterial hypoxaemia and compromised CPR haemodynamics. Moreover, these detrimental effects remain evident even after restoration of IPPV.

Introduction

The International Liaison Committee on Resuscitation (ILCOR) recommends a ratio of 30:2 for chest compressions and mouth-to-mouth ventilation during basic life support (BLS). However, if a bystander is not trained appropriately in mouth-to-mouth ventilation or is reluctant to perform it, chest compressions without ventilation are advised.[1]

In animal studies, the outcome of cardiopulmonary resuscitation (CPR) without ventilation compared with that of intermittent positive pressure ventilation (IPPV)-CPR depends on the arrest model used. When a model of ventricular fibrillation is utilized, IPPV-CPR is not superior to CCO-CPR.[2–4] In asphyctic models, by contrast, a better outcome is achieved with IPPV-CPR.[5–7]

There is a paucity of data reporting the outcome of CPR without ventilation with that of IPPV-CPR in humans. Retrospective analyses of CPR registries show either no advantage of CPR without ventilation,[8,9] or a benefit of ventilation during BLS.[10] The first randomized controlled trial comparing both methods, by Hallstrom et al., revealed no difference in outcome between telephone-guided CCO-CPR and IPPV-CPR.[11] By contrast, a recent prospective multicentre observational study showed CCO-CPR to be superior to IPPV-CPR in some patient subgroups in terms of neurological outcome 30 days after cardiac arrest.[12] However, there remains a substantial lack of understanding about the mechanisms of different ventilatory strategies on atelectasis, haemodynamics and oxygenation during BLS.[13,14]

The aim of this study was to assess the effects of CPR without ventilation during fibrillatory arrest and BLS on the development of atelectasis and its sequelae, using conventional IPPV-CPR as the control. Dynamic computed tomography (CT) was used to visualize the primary end-point of atelectasis formation and recruitment during IPPV in a time-resolved manner. Changes in haemodynamics, pulmonary gas exchange and regional organ blood flow were addressed as secondary endpoints.

Materials and methods

Anaesthesia and instrumentation

The study was performed in 12 pigs (26 ± 1 kg), with approval of the state animal care committee and following the guidelines of the American Physiological Society. Anaesthesia was induced and maintained using a combination of piritramide (Dipidolor®, Janssen-Cilag Pharmaceuticals) and thiopentone. After endotracheal intubation with a cuffed tube, animals were ventilated with IPPV at an FiO_2 of 0.3, a tidal volume of 10 ml kg^{-1}, and a positive end-expiratory pressure (PEEP) of 5.1 cm H_2O. The respiratory rate was adjusted to maintain an end-tidal PCO_2 of 4.7—5.3 kPa. Ringer's solution was infused at a maintenance rate of 5 ml/(kg h) i.v. Intravascular volume loss was replaced quantitatively by i.v. hetastarch solution (130,000/6% Voluven®, Fresenius, Germany). Temperature was maintained actively between 37.5 and 38.5 °C. Arterial and central venous pressures were monitored via catheters advanced into the aorta and the vena cava by femoral cut-down and connected to pressure transducers zeroed to the level of the right atrium. Continuous blood gas analysis was provided using a Trendcare®/Paratrend™7 intra-arterial sensor (Diametrics Medical Ltd., UK) introduced into the left femoral artery. For injection of fluorescent microspheres, a 5F angiographic catheter (Sidewinder®, Cordis, Germany) was advanced through the right carotid artery into the left cardiac ventricle. For reference blood sampling, an 18G polyvinyl catheter was advanced into the thoracic aorta via the left brachiocephalic artery and connected to a blood-withdrawal pump. Correct catheter positioning was verified by typical pressure waveforms, blood gas analysis (Radiometer® 500 and OSM® 3, Radiometer), and during post-mortem examination.

Dynamic CT

Dynamic CT provides lung imaging with a temporal resolution of 100 ms/image in one predefined transversal slice. Animals were placed in a chest compression device in supine position and moved into the gantry.[15] A topographic scan was used to predefine a transversal plane approximately 4 cm cranial to the diaphragm for dynamic imaging. Dynamic CT image series were acquired for periods of 15 s, using a high resolution reconstruction algorithm, a slice thickness of 1 mm, a matrix of 512×512, a tube current of 110 mA and a tube voltage of 120 kV (Somatom® Plus 4, Siemens, Germany). Quantitative image analysis was performed off-line using a dedicated software tool which has been described in detail elsewhere.[15,16] Briefly, an automated segmentation algorithm defines the lung borders in every single CT image, and the individual fractions of different lung compartments within the total cross-sectional lung area were calculated, using the following compartmental density ranges: −1024 to −910 HU for overinflated lung, −910 to −300 HU for ventilated lung and −300 to +200 HU for atelectasis. Image post-processing quantified the cyclic changes of regional lung area attained by each of these compartments, and the mean regional lung area during several respiratory cycles.[17]

Fluorescent microspheres technique

The measurement of regional myocardial, cerebral, and renal blood flow using tracer-labelled microspheres has been described in detail by several groups.[18–21] We used the fol-

lowing fluorescent colours in randomized order: blue-green, yellow-green, orange-red and crimson (Triton Technology Inc., Leiden, Netherlands). Microspheres were injected into the left cardiac ventricle. Thirty seconds prior to injection, reference blood withdrawal from the thoracic aorta was commenced at a rate of 2 ml/min and continued for 2 min. Thereafter, a separate blood sample of 2 ml was withdrawn and its fluorescence determined in order to ensure that a withdrawal time of 2 min was sufficient. After euthanization of the animals at the end of the experiment, the entire heart and brain were removed, and multiple samples of kidneys and jejunum were obtained. The myocardium was divided into left ventricular free wall, interventricular septum, and right ventricular free wall. The left ventricular free wall and interventricular septum were sectioned into three layers, and the right ventricular free wall into two layers.

Study protocol

After induction of anaesthesia and instrumentation, animals were transported to the radiological facility and placed in supine position on a specially designed V-shaped wooden board implemented in the chest compression device, as described previously.[15] The chest compression device was positioned in the gantry of the CT scanner. Fifteen minutes prior to induction of ventricular fibrillation the animals were ventilated in IPPV mode at an FiO_2 of 0.21 and a tidal volume adjusted to maintain an end-tidal PCO_2 of 0.6—0.7 kPa. At baseline, haemodynamic data were recorded, arterial and central venous blood samples were drawn, microspheres were injected, and a dynamic CT series over 15 s was acquired. Fifteen minutes thereafter, ventricular fibrillation was induced by an external A/C shock of 110 V. Circulatory arrest was confirmed by loss of pulsatile pressure tracings and VF in the ECG. After 3 min of untreated ventricular fibrillation, BLS was commenced with a chest compression rate of 100/min at a duty cycle of 50% (Thumper® 12715 Programmable CPR Controller, Michigan Instruments Inc., Grand Rapids, MI). At this point, the animals were randomly assigned to either the control group ($n = 6$) or the chest compressions only group (CCO-CPR; $n = 6$). Animals in the control group received IPPV during BLS (FiO_2, 0.21), using a respiratory rate of 20/min and an inspiration: expiration time of 1:2. Tidal volumes were maintained at levels present during spontaneous circulation prior to cardiac arrest. Animals in the CCO-CPR group received no ventilation during BLS. The airway was kept open with the endotracheal tube left in place but disconnected from the ventilator circuit. Both groups received IPPV during advanced life support (ALS; FiO_2, 1.0).

Following 4 min of BLS, haemodynamic data, arterial blood gas analysis (ABG, Paratrend™7), organ blood flow measurement and a dynamic CT series over 15 s were performed. Following 5 min of BLS, ALS was commenced by increasing FiO_2 to 1.0 and administering an i.v. bolus of epinephrine, 40 µg kg⁻¹. Following 3 min of ALS, measurements were repeated as above. Following 4 min of ALS, animals were defibrillated up to three times (bi-phasic shock of 150 J, Forerunner, Heartstream Inc., Netherlands). In case of return of spontaneous circulation (ROSC), chest compressions were stopped, artificial ventilation (IPPV) was resumed as prior to fibrillatory arrest, and measurements were repeated as above.

Statistical analysis

All data are given as median (minimum—maximum) values. Categorical data were compared using contingency table testing. Differences among groups were sought for by the Kruskal—Wallis test. Comparisons within each group were performed with the Mann—Whitney U-test. Differences were considered to be significant at $p < 0.05$.

Results

Cyclic changes in atelectasis during CPR

The two groups did not differ at baseline. With each respiratory cycle of IPPV, expiratory collapse of lung parenchyma and inspiratory recruitment of atelectasis were observed during BLS and ALS (changes of fractional atelectasis (%): 15.5 [7.2—36.7]). During CCO-CPR atelectatic lung area still cycled, however at a much higher frequency, dictated by the rate of the external chest compressions (changes of fractional atelectasis (%): 10.7 [6.5—13.6]; see Figure 1).

Mean fractional lung compartments

Dynamic CT-based measurements are summarized in Table 1, while the time course of atelectasis formation is illustrated in Figure 2. Values for mean atelectatic lung area, calculated as a fraction of the total lung area examined, did not differ between the two study groups at baseline. Further-
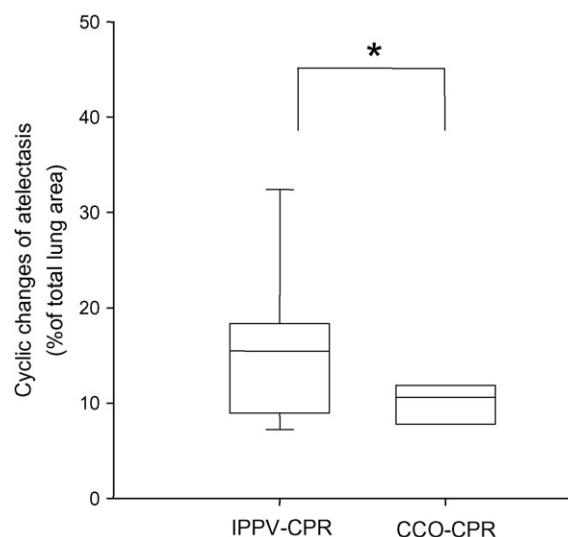
Figure 1 Cyclic changes of atelectasis, defined as difference between maximum and minimum of the fractional amount of atelectatic lung area during a 15 s dynamic computed tomography acquisition, at different ventilation regiments in cardiopulmonary resuscitation. *$p < 0.05$. CCO-CPR, chest compressions only during cardiopulmonary resuscitation; IPPV-CPR, intermittent positive pressure ventilation during cardiopulmonary resuscitation.
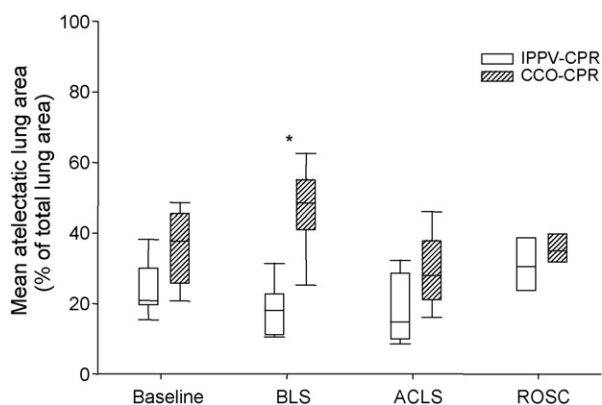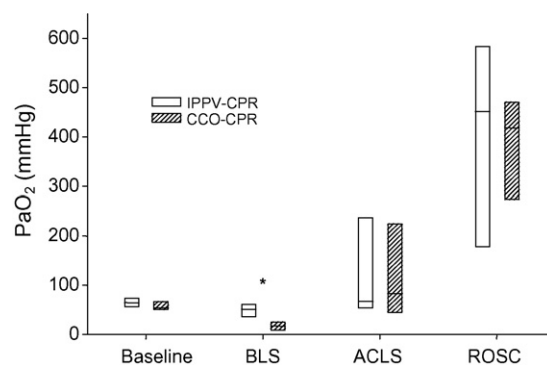
Figure 2 Mean atelectatic lung area in animals receiving no ventilation during cardiopulmonary resuscitation (CCO-CPR), compared with controls receiving intermittent positive pressure ventilation (IPPV-CPR). In the CCO-CPR group, the atelectatic lung compartment increased significantly during basic life support (BLS). There was only incomplete resolution of atelectasis during advanced life support (ALS) and after return of spontaneous circulation (ROSC). *$p < 0.05$ CCO-CPR *vs.* IPPV-CPR.

Figure 3 PaO_2 at each measurement point of the study protocol in animals receiving no ventilation during cardiopulmonary resuscitation (CCO-CPR), compared with controls receiving intermittent positive pressure ventilation (IPPV-CPR). PaO_2 decreases significantly in the CCO-CPR group during basic life support (BLS). There were no significant differences between the two groups during advanced life support (ALS) and after return of spontaneous circulation (ROSC). *$p < 0.002$ IPPV-CPR *vs.* CCO-CPR.

more, in the control group, values did not differ between the pre-arrest period and the BLS period. By contrast, the mean atelectatic lung area increased rapidly and significantly during BLS compared with pre-arrest measurements in the CCO-CPR group. The difference between the two groups in atelectasis during BLS was statistically significant ($p = 0.009$). The increase in mean atelectatic lung area observed in the CCO-CPR group remained clinically relevant throughout the subsequent ALS period and following ROSC. In addition, the ventilated lung area was also significantly reduced in the CCO-CPR group compared with the control group ($p = 0.016$). The CCO-CPR group showed significantly less lung overdistension than the control group during BLS ($p = 0.003$). No differences were observed between the two groups during ALS and after ROSC.

Arterial blood gasses

The arterial blood gas status is shown in Table 2, and the time course of PaO_2 is depicted in Figure 3. The two study groups did not differ at baseline. There was no change in PaO_2 between baseline and BLS in controls. By contrast, PaO_2 decreased from baseline in the CCO-CPR group. This decrease in PaO_2 in the CCO-CPR group compared with controls was statistically significant ($p = 0.002$). CCO-CPR was also accompanied by significant hypercapnia compared with controls ($p = 0.002$). During ALS, PaO_2 recovered in the CCO-CPR group, and no statistical differences were observed between the two study groups.

Haemodynamic data

Haemodynamic data are reported in Table 3, and mean arterial pressures are shown in Figure 4. Mean arterial pressures were lower in the CCO-CPR group than in controls during BLS ($p = 0.023$). Systolic arterial pressures remained lower in the CCO-CPR group than in controls even during ALS when both groups were ventilated alike ($p = 0.025$). However, coronary perfusion pressures did not differ significantly between the CCO-CPR group and the control group at any time point.

Table 1 Lung density compartments as determined by dynamic computed tomography

Parameter	Group	Baseline	BLS	ALS	ROSC
Overinflated lung (% of total cross-sectional area)	Control	6.3 (4.3—12.9)	8.8 (5.9—13.8)	9.3 (6.8—16.3)	9.2 (5.1—12.6)
	CCO	5.2 (4.2—8.1)	2.3 (0.4—5.7)	5.9 (2.7—9.5)	5.3 (4.7—8.6)
Ventilated lung (% of total cross-sectional area)	Control	70.7 (56.1—76.9)	73.9 (61.9—77.6)	74.0 (60.6—79.1)	61.0 (47.7—69.8)
	CCO	58.2 (46.1—75)	48.6 (35.4—72.7)*	64.0 (49.5—75.6)	58.5 (51.9—64.6)
Atelectasis (% of total cross-sectional area)	Control	20.9 (14.9—39.1)	18.1 (10.4—32.3)	14.8 (8.4—32.7)	30.5 (22.2—41.7)
	CCO	37.7 (19.6—48.8)	48.4 (22—64.4) *	27.9 (15.0—47.4)	35.0 (30.1—43.2)

Data are reported as median (minimum—maximum). *CCO *vs.* control $p < 0.05$. ALS, advanced life support; BLS, basic life support; NV, no ventilation; ROSC, return of spontaneous circulation.

Table 2 Arterial blood gas status

Parameter	Group	Baseline	BLS	ALS	ROSC
PaO_2 (kPa)	Control	8.5 (7.3—10.8)	6.8 (4.7—8.5)	8.9 (6.0—56.0)	60.0 (14.6—80.3)
	CCO	7.2 (6.0—9.0)	2.3 (0.8—4.0) *	11.0 (5.1—42.4)	55.6 (31.1—63.8)
$PaCO_2$ (kPa)	Control	4.7 (4.4—4.8)	4.0 (3.5—5.2)	3.7 (2.9—6.0)	7.2 (6.4—7.8)
	CCO	4.5 (4.4—4.7)	8.1 (7.3—9.4) *	5.2 (4.8—6.5)	6.5 (6.0—6.8)
SaO_2 (%)	Control	95.2 (93.8—96.9)	90.3 (76.8—96.0)	95.5 (83.0—99.9)	99.8 (97.3—99.9)
	CCO	93.7 (90.5—96.4)	20.9 (3.3—55.9)*	96.8 (74.1—99.8)	99.8 (99.4—99.9)

*CCO *vs.* control $p < 0.05$. ALS, advanced life support; BLS, basic life support; CCO, chest compressions-only cardiopulmonary resuscitation; $PaCO_2$, arterial partial pressure of carbon dioxide; PaO_2, arterial partial pressure of oxygen; ROSC, return of spontaneous circulation; SaO_2, arterial oxygen saturation.

Regional organ blood flow

Regional organ blood flows were determined in four out of six animals in each group (summarized in Table 4). Data from two animals in each group were excluded due to technical limitations (inaccurate timing of microsphere injection, failure of reference blood withdrawal under conditions of low blood flow). No inter-group differences were analyzed due to the reduced sample size. Left ventricular blood flow was determined separately for mid-myocardial, subendocardial, and subepicardial layers of the left ventricular myocardium (total left ventricular blood flow, Figure 5). No differences were observed between the two study groups in left and right hemispheric cerebral blood flow, even though the right carotid artery had been occluded for insertion of the left cardiac ventricle catheter. Within-group differences for cerebral blood flows were not significant. Right and left kidney blood flow showed no significant difference, thus indicating a homogeneous distribution of injected microspheres.

Return of spontaneous circulation

ROSC was achieved in 4/6 animals both in the control group and in the CCO-CPR group.

Discussion

The role of ventilatory support during cardiac arrest and basic life support is controversial. Results from this study show that ventilation during CPR as recommended by ILCOR recommendations[1] causes cyclic collapse and recruitment of substantial sections of lung parenchyma. Furthermore, entirely omitting ventilation during the initial minutes of BLS dramatically propagates the development of atelectasis and its negative sequelae.

In line with previous analyses, the current study used a dynamic CT imaging technique rather than the conventional static images. This novel technique allowed us to visualize and quantify atelectasis formation during cardiac arrest and CPR with different ventilatory strategies in a

Table 3 Haemodynamic measurements

Parameter	Group	Baseline	BLS	ALS	ROSC
Heart rate (min^{-1})	Control	95(75—170)	—	—	170(160—180)
	CCO	110(80—150)	—	—	150(120—150)
MAP (kPa)	Control	15.6 (10.8—17.7)	7.4 (4.8—8.5)	8.5 (4.8—10.1)	11.2 (10.8—11.7)
	CCO	14.0 (10.6—17.7)	5.6 (3.9—6.5)*	5.6 (4.0—7.0)	10.0 (9.6—10.9)
SAP (kPa)	Control	19.4 (14.4—21.7)	14.4 (8.2—14.9)	17.0 (9.6—19.4)	15.3 (14.5—16.4)
	CCO	18.6 (14.4—20.7)	10.0 (6.7—14.1)	11.7 (6.3—14.6)*	14.0 (13.6—15.7)
DAP (kPa)	Control	13.3 (8.9—15.6)	3.6 (2.7—4.0)	3.7 (2.3—4.8)	8.4 (8.1—8.9)
	CCO	11.4 (8.4—14.8)	2.0 (0.9—4.0)	2.5 (1.7—3.9)	7.8 (7.3—8.2)
CVP (kPa)	Control	2.7 (2.1—3.6)	3.1 (1.7—3.6)	3.1 (2.0—3.7)	2.1 (1.7—3.2)
	CCO	2.0 (1.3—3.1)	1.1 (0.1—2.9)*	1.3 (0.7—3.2)	2.1 (1.7—2.4)
CPP (kPa)	Control	10.3 (5.5—13.4)	0.5 (0—0.9)	0.7 (−0.1—1.3)	6.2 (5.2—6.8)
	CCO	8.4 (6.5—12.6)	1.1 (−0.9—2.1)	0.7 (−0.4—2.7)	5.7 (4.9—6.5)

*CCO *vs.* control $p < 0.05$. ALS, advanced life support; BLS, basic life support; CCO, chest compressions-only cardiopulmonary resuscitation; CPP, coronary perfusion pressure; CVP, central venous pressure; DAP, diastolic arterial pressure; MAP, mean arterial pressure; ROSC, return of spontaneous circulation; SAP, systolic arterial pressure.
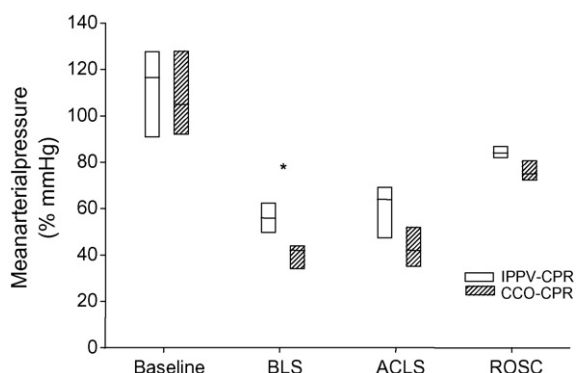
Figure 4 Mean arterial pressure at each measurement point of the study protocol in animals receiving no ventilation during cardiopulmonary resuscitation (CCO-CPR), compared with controls receiving intermittent positive pressure ventilation (IPPV-CPR). Mean arterial pressure decreased in the CCO-CPR group during basic life support (BLS), compared with control group, and did not recover even during advanced life support (ALS). *$p < 0.023$ IPPV-CPR *vs.* CCO-CPR.
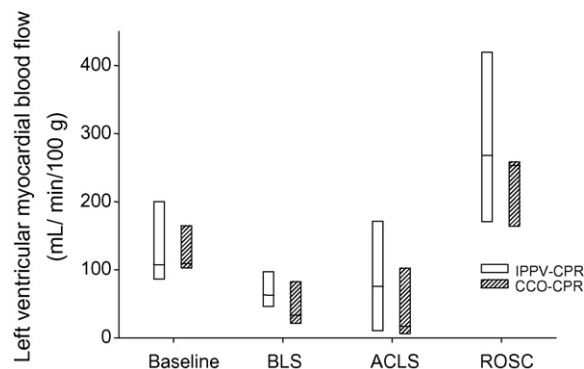
Figure 5 Left ventricular myocardial blood flow in animals receiving no ventilation during cardiopulmonary resuscitation (CCO-CPR), compared with controls receiving intermittent positive pressure ventilation (IPPV-CPR). No differences were noted between groups.

direct and time-resolved manner. In experimental models of ARDS, this as well as similar imaging techniques have already been applied successfully to assess cyclic recruitment of atelectatic lung regions.[22–25] In a pilot study, we described this technique with its adaptation to the specific requirements during experimental CPR.[15]

Cyclic collapse and recruitment of lung parenchyma likely leads to a shunt fraction varying over time and, consequently, to impairment of gas exchange. In a previous study, using the same imaging technique,[23] we demonstrated

a correlation between image-based determination of mean atelectatic lung area over time and the venous admixture. Dynamic assessment of the mean atelectatic lung area over time thus allows quantification and comparison of the amount of alveolar space capable of gas exchange provided by particular respiratory strategies or ventilator adjustments during CPR.

By relating imaging with gas exchange data, the present study demonstrated that the omission of ventilation during BLS leads to a dramatic increase of atelectases when compared with a control group ventilated conventionally. Our study confirms data described by others[2,7] showing that chest compression-only BLS is associated with several min-

Table 4 Regional organ blood flow

Region	Group	Baseline	BLS	ALS	ROSC
Myocardial blood flow					
Left ventricular subepicardial (ml/(min 100 g))	Control	97 (79–213)	59 (35–91)	64 (1–157)	249 (171–428)
	CCO	97 (87–174)	34 (17–119)	9 (1–89)	233 (144–173)
Left ventricular midmyocardial (ml/(min 100 g))	Control	112 (84–237)	75 (53–127)	89 (2–228)	269 (174–423)
	CCO	115 (105–187)	36 (13–180)	22 (2–136)	287 (179–296)
Left ventricular subendocardial (ml/(min 100 g))	Control	113 (75–241)	60 (29–101)	75 (2–187)	285 (165–409)
	CCO	119 (103–188)	34 (10–165)	20 (2–135)	216 (167–231)
Brain blood flow					
Left cerebral hemisphere (ml/(min 100 g))	Control	28 (17–32)	37 (35–41)	46 (30–129)	60 (58–71)
	CCO	33 (21–48)	22 (6–46)	21 (0–60)	77 (27–90)
Right cerebral hemisphere (ml/(min 100 g))	Control	23 (15–28)	33 (28–42)	40 (29–117)	54 (53–72)
	CCO	34 (20–66)	24 (5–39)	31 (0–61)	75 (27–80)
Renal blood flow					
Right kidney (ml/(min 100 g))	Control	149 (125–186)	48 (32–110)	8 (2–60)	197 (136–252)
	CCO	145 (128–267)	43 (16–79)	11 (0–22)	146 (90–199)
Left kidney (ml/(min 100 g))	Control	165 (109–189)	41 (32–111)	9 (1–49)	198 (151–233)
	CCO	160 (148–273)	42 (9–63)	8 (1–19)	152 (86–160)

ALS, advanced life support; BLS, basic life support; CCO, chest compressions-only cardiopulmonary resuscitation; ROSC, return of spontaneous circulation.

utes of exceptionally poor oxygenation. This may be due not only to a reduced fraction of ventilated lung, but also to impaired pulmonary perfusion, most likely as a result of increased pulmonary vascular resistance (PVR). Hypoxic pulmonary vasoconstriction and increased PVR in this setting can only be inferred based on physiological considerations, since data from such scenarios are scarce. However, well-recognized contributors such as alveolar and mixed venous hypoxia, acidosis, hypercapnia, alveolar collapse and hypothermia are typically present in the BLS setting. An increase in resistance to pulmonary blood flow may offer a ready explanation for lower mean arterial pressures in the CCO-CPR group.

It has been previously hypothesized that transmission of closed chest compression force to the intrathoracic conduit is improved when lungs are inflated at positive airway pressure.[26] Inflated lung parenchyma is thought to propagate compression force more homogeneously to the heart and intrathoracic vessels.[26] This promotes antegrade blood flow from the left cardiac ventricle into the aorta, and hence improves organ blood flow.[27] In support of this hypothesis, our study showed more favorable systemic haemodynamics in conventionally ventilated controls than in the CCO-CPR group.

The atelectatic lung fraction that developed in the CCO-CPR group during BLS was not reopened sufficiently even after resumption of constant-volume ventilation. Consequently, a larger amount of atelectasis and more haemodynamic impairment were observed even during ALS and after ROSC. These results indicate that ALS ventilation as recommended by current guidelines will not provide sufficient alveolar recruitment if chest compressions alone without ventilation were performed during the preceding BLS efforts. The proportion of animals achieving ROSC in the control group and in the CCO-CPR group was similar in our study. Whether a larger study that included more animals would be able to extract a difference between the two treatment groups remains to be shown.

However, there was a relatively broad variance of atelectasis between both groups at baseline. This variance could be explained by the fact, that the absolute amount of atelectasis is not only influenced by lung recruitment/derecruitment per se, but also by the specific cross-sectional imaging plane of the CT, which might have varied slighly between both groups. However, in the CCO group atelectasis increased from baseline to BLS by 12.1% (mean), whereas in the controls atalectasis decreased by 5.5%. This decrease follows by high intrapulmonary pressures during IPPV and chest compressions.

We consider these findings to be clinically relevant as they demonstrate that CCO-CPR and IPPV-CPR clearly differ in the capability of lung recruitment.

Data comparing the outcome of CPR without ventilation with that of IPPV-CPR in humans are inconsistent. A recent non-randomized, non-controlled observational study in out-of-hospital cardiac arrest cases reports similar frequencies of survival in patients receiving CCO-CPR and IPPV-CPR, both at hospital admission and at 30 days.[12] In addition, neurological outcome 30 days after cardiac arrest was also found to be similar in the two patient groups, and was even found to be superior with CCO-CPR in some patient subgroups.

Similar outcomes between telephone-guided CCO-CPR and IPPV-CPR were also observed in the trial by Hallstrom et al.[11] However, telephone instructions for IPPV-CPR took on average 1.4 min longer than for CCO-CPR, resulting in the arrival of emergency medical assistance before completion of the call in 26% of patients in the IPPV-CPR group, compared with only 13% of patients in the CCO-CPR group. This increased instruction time, during which victims were presumably without any form of CPR, may have led to the lack of an observed superiority of IPPV-CPR.[14]

In a study conducted by the Belgian Cerebral Resuscitation Group in more than 3000 out-of-hospital cardiac arrest events, survival after bystander CPR was shown to be virtually identical with IPPV-CPR as with CCO-CPR (16% *vs.* 15%, respectively). However, an updated follow-on study by the same group showed that while 16% of patients survived after IPPV-CPR, survival was only 10% after CCO-CPR.

We believe that our results are of enough concern to warrant further study in this important aspect of emergency medicine. It should be noted, however, that the difference between the control group and CCO-CPR group may have been less pronounced if the pigs had been normocapnic. It should also be noted that direct extrapolations from the BLS protocols used in animal studies to those used in the human patient emergency setting are, of course, not possible.

Conclusion

In conclusion, our findings demonstrate that avoidance of intermittent positive pressure ventilation during cardiac arrest and CPR dramatically increases the amount of atelectasis, leading to impairment in pulmonary gas exchange and haemodynamics once a critical amount of atelectasis is exceeded. Starting and continuing chest compressions without positive pressure ventilation provokes rapid alveolar collapse that is not readily reversible. The adverse effects persist even after restoration of positive pressure ventilation, due to insufficient alveolar recruitment.

Conflict of interest

None.

Acknowledgements

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