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PEDIATRIC BRIEF REPORT

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Chest compressions versus ventilation plus chest compressions in a pediatric asphyxial cardiac arrest animal model

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Abstract *Objective:* To compare the ventilation achieved with chest compressions (CC) or ventilation plus compressions (VC) in a pediatric animal model of cardiac arrest. *Design:* Randomized experimental study. *Setting:* Experimental department of a University Hospital. *Methods:* Twelve infant pigs with asphyxial cardiac arrest. Sequential 3-min periods of VC and CC were performed for a total duration of 9 min. Tidal volume (TV), end-tidal CO_2 ($EtCO_2$), mean arterial pressure (MAP), central venous pressure (CVP), mean pulmonary arterial pressure (mPAP), and peripheral, cerebral, and renal saturations were recorded and arterial and venous blood gases were analyzed. *Results:* VC achieved a TV similar to the preset parameters on the ventilator, whilst the TV in CC was very low ($P < 0.001$). $EtCO_2$ with VC was significantly higher than with CC (14.0 vs. 3.9 mmHg, $P < 0.05$). Arterial pH was higher with VC than with CC (6.99 vs. 6.90 mmHg, $P < 0.05$). Arterial PCO_2 was lower with VC than with CC (62.1 vs. 97.0 mmHg, $P < 0.05$). There were no significant differences in the MAP; CVP; mPAP; peripheral, renal, and cerebral saturations; or lactate concentrations between the two techniques. *Conclusions:* VC achieves better ventilation than CC during cardiopulmonary resuscitation and has no negative effect on the hemodynamic situation.

Keywords Cardiac arrest · Children · Ventilation · Chest compressions · Resuscitation

Introduction

The new recommendations for cardiopulmonary resuscitation (CPR) are based on the fact that cardiac output and pulmonary blood flow during cardiac arrest (CA) fall by 10–25% compared to normal values, and the ventilation necessary to achieve adequate gas exchange would therefore also be lower than under normal conditions [1, 2].

Some authors recommend that only chest compressions (CC) should be performed during the initial phases of CPR to optimize the perfusion of vital organs. Some experimental studies in CA secondary to ventricular fibrillation (VF) have found a similar rate of recovery and a better neurological prognosis in animals that only received CC in comparison with those that were resuscitated with ventilation plus chest compressions (VC) [3–8]. In addition, no significant deterioration in the blood gas parameters was detected in the first 4–8 min of resuscitation with CC, maintaining a minimum respiratory flow due to the presence of agonal breathing and passive ventilation due to the chest compression–decompression [3–8]. In adults with out-of-hospital CA, ventilation did not improve the survival and the neurological prognosis [9–11].

However, the ideal ratio between ventilation and chest compressions in CA in children is under discussion. In

pediatric patients, the origin of CA is very often asphyxia and the blood flow is maintained for a certain time before cardiac activity ceases, reducing the oxygen tension and increasing the CO_2 tension. In addition, a higher rate of recovery with VC than with CC has been reported in some experimental models [12–14]. In experimental studies the tidal volume moved by chest compressions was less than the anatomical dead space [8, 15, 16].

The aim of our study was to compare the tidal volume and ventilation with CC and VC in an infant animal model of asphyxial CA.

Methods

Twelve Maryland pigs of 2 months of age and with a mean (SD) weight of 8.9 (1.7) kg were used in the study. International guidelines for the care of experimental animals were applied. The animals were intubated and connected to a mechanical ventilator (Drager SA2, Lubeck, Germany) with a respiratory rate of 20 breaths per minute, tidal volume of 10 ml/kg, FiO_2 of 50%, and PEEP of 3 cm H_2O. Sedation and relaxation (propofol 10 mg kg^{-1} h^{-1}, fentanyl 10 mg kg^{-1} h^{-1}, and atracurium 2 mg kg^{-1} h^{-1} as a continuous infusion) were maintained throughout the procedure, inhibiting the presence of agonal breathing. The following aspects were monitored: the ECG, peripheral oxygen saturation (Visconnet monitor, KGB Madrid, Spain), cerebral and renal saturation (INVOS Cerebral Oximeter monitor, Somanetics, Troy, MI, USA), and the respiratory volumes and pressures, FiO_2, and $EtCO_2$ via a spirometer connected to the endotracheal tube and an S5 monitor (Datex Ohmeda, Madison, WI, USA) with a volume-detection capacity of 7 ml. Arterial blood pressure, central venous pressure (CVP), and pulmonary arterial pressure (mPAP) were measured in the femoral artery and external jugular vein with a 5.5F Swan-Ganz catheter (Edwards Lifescience, Irving, CA, USA). Blood gases were analyzed using the GEM Premier 3000 blood gas analyzer (Instrumentation Laboratory, Lexington, KY, USA).

Cardiac arrest was induced by disconnection from the respirator for at least 10 min. Resuscitation was started after confirming the absence of spontaneous circulation. The animals were randomly distributed into two groups: in group 1, resuscitation was started with 3 min of manual chest compressions (100 compressions/min) and ventilation [20 breaths/min (bpm) with tidal volume of 10 ml/kg, via the respirator without interrupting chest compressions] (VC), followed by 3 min of chest compressions alone (CC), and terminating with a further 3 min of ventilation plus chest compressions (VC); in group 2, only chest compressions (CC) were performed initially, followed by ventilation plus compressions (VC), and terminating with only chest compressions (CC). We controlled continuously the number of chest compressions with the arterial blood pressure and EKG monitor.

The following parameters were recorded at baseline and at minutes 1 and 3 of each phase: inspiratory tidal volume (TV), $EtCO_2$, mean arterial pressure (MAP), CVP, mPAP, and peripheral, cerebral, and renal saturations. Arterial and venous blood gases and lactate measurements were performed every 3 min (Fig. 1).

The statistical study was performed with SPSS 16.0, using Student's *t* test, Friedman and Mann-Whitney *U* test for compared variables. Analysis of variance for repeated measurements test was used to study the evolution of the parameters during the experiment.

Results

The comparison of parameters in CC phases and VC phases is shown in Table 1. Using the VC technique, a

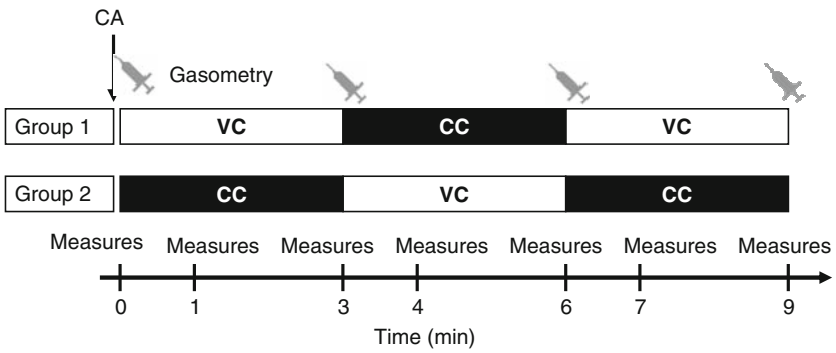

Fig. 1 Study protocol. The inspiratory and expiratory tidal volume, end-tidal CO_2, systolic blood pressure, diastolic blood pressure, mean blood pressure, central venous pressure, mean pulmonary arterial pressure, and peripheral, cerebral, and renal saturations were recorded at baseline and at minutes 1 and 3 of each phase. Arterial and venous blood gas analyses and measurement of lactic acid and base excess levels were performed only at the start of each phase and at 3 min. *CA* Cardiac arrest, *VC* ventilation and chest compressions phase, *CC* chest compressions only phase

Table 1 Comparison of hemodynamic, saturation, and respiratory variables between ventilation plus compressions phases (VC) and continuous chest compressions alone phases (CC)

	Basal values	VC phase	CC phase	*P*
Number of measures	12	36	36	
mAP (mmHg)	0.0 ± 0.0	20.6 ± 10.0	21.0 ± 11.7	0.88
CVP (mmHg)	5.6 ± 3.3	15.0 ± 9.4	17.5 ± 12.3	0.33
mPAP (mmHg)	19.1 ± 14.0	36.1 ± 17.6	39.1 ± 24.5	0.56
$SatO_2$ (%)	0.0 ± 0.0	72.5 ± 9.4	72.0 ± 12.6	0.87
Cerebral $SatO_2$ (%)	36.1 ± 8.7	42.3 ± 18.3	41.9 ± 16.0	0.92
Renal $SatO_2$ (%)	44.0 ± 18.8	46.8 ± 15.4	43.5 ± 13.4	0.38
$EtCO_2$ (mmHg)	0.0 ± 0.0	14.0 ± 8.0	3.9 ± 7.4	<0.05
Expired TV (ml)	0.0 ± 0.0	71.1 ± 21.6	2.8 ± 7.0	<0.05

Values are means ± SD. The comparisons include all values together from the first and third minutes of each phase
VC Ventilation plus compressions, *CC* chest compressions, *MAP* mean arterial pressure, *CVP* central venous pressure, *mPAP* mean pulmonary arterial pressure, *$EtCO_2$* end-tidal CO_2, *expiratory TV* expiratory tidal volume

tidal volume similar to the machine settings was achieved, whereas the tidal volume with CC was undetectable. The $EtCO_2$ was significantly higher with VC than with CC. There were no differences in the MAP, CVP, mPAP, or peripheral, cerebral, and renal saturations between the two techniques throughout the study (Table 1).

The arterial pH was significantly higher with VC than with CC (Fig. 2). $PaCO_2$ was significantly lower with VC than with CC (Fig. 3), and the PaO_2 was significantly higher (Fig. 4). The evolution of venous pH, PCO_2, and PO_2 was similar to the arterial values with significant differences between VC and CC (data not shown).

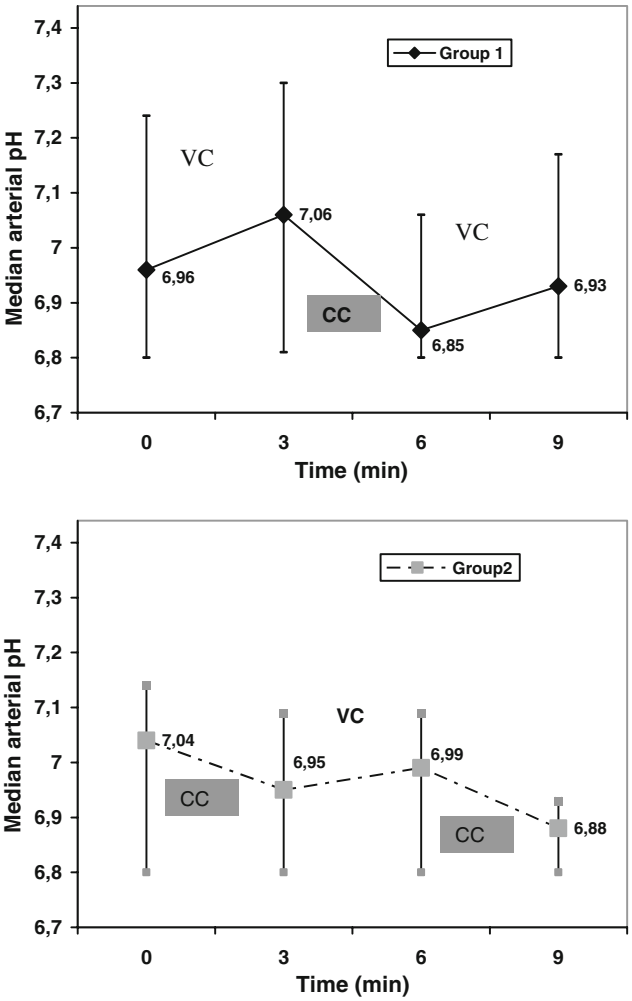

Fig. 2 Changes in the arterial pH with ventilation and chest compressions (*VC*) and with chest compressions only (*CC*). *Group 1*: Initial period VC, second period CC, third period VC. *Group 2*: Initial period CC, second period VC, third period CC. The arterial pH increased with VC and diminished with CC. The arterial pH was significantly higher with VC than with CC throughout the course of the experiment ($P < 0.05$)

Discussion

This is the first study of asphyxial CA in a pediatric animal model that has compared the ventilation achieved with CC and VC. In our study, the VC technique achieved a higher pH and PO_2 and a lower PCO_2 than CC. These findings agree with those other asphyxial CA experimental studies [12]. Studies using models of CA of cardiac origin have not found these differences [3–8]. This discrepancy may be due to the presence of agonal breathing, which has been shown to achieve a certain level of ventilation for a few minutes, and the reduction in oxygen consumption and CO_2 production immediately after spontaneous circulation ceases. Recent studies have demonstrated that the presence of gasping is one of the most important prognostic markers of survival [17]. In our study, we have inhibited gasping and simulate CA secondary to apnea. However, in prolonged models of ventricular fibrillation CC does not achieve sufficient ventilation, and the results are similar to those observed in our study [3–10, 18, 19]. Thus, in our pediatric model of asphyxial CA, ventilation achieves a marked improvement in the blood gases, although no improvement was observed in cardiac or cerebral oxygenation.

There are few studies that have analyzed the tidal volume achieved by CC. The majority of studies use CA of cardiac origin in intubated adult animals without muscle relaxation. In any case, the tidal volumes reported were smaller than anatomical dead space or decreased as the duration of CPR increased [8, 15, 16]. In our study, with CC, although the animals were intubated, the TV and the $EtCO_2$ recorded were close to 0. In contrast, adequate TV was achieved with VC, with a significantly higher $EtCO_2$, suggesting a more efficient alveolar CO_2 removal.

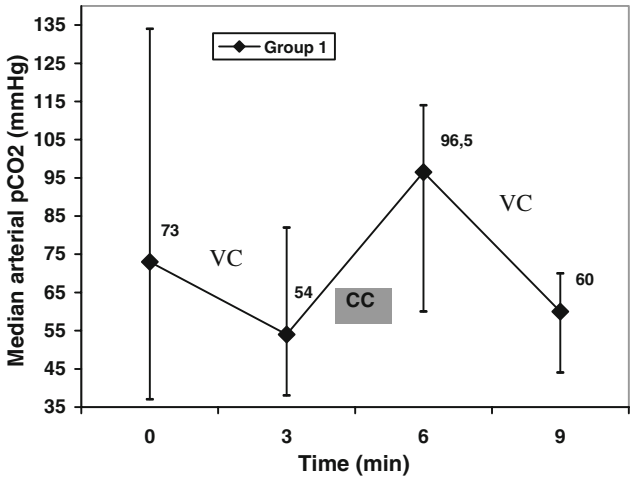

Fig. 3 Changes in the arterial PCO_2 with ventilation and chest compressions (*VC*) and with chest compressions only (*CC*). *Group 1*: Initial period VC, second period CC, third period VC. *Group 2*: Initial period CC, second period VC, third period CC. The arterial PCO_2 diminished with VC and increased with CC. The arterial PCO_2 was lower with VC than with CC throughout the course of the experiment ($P < 0.05$)

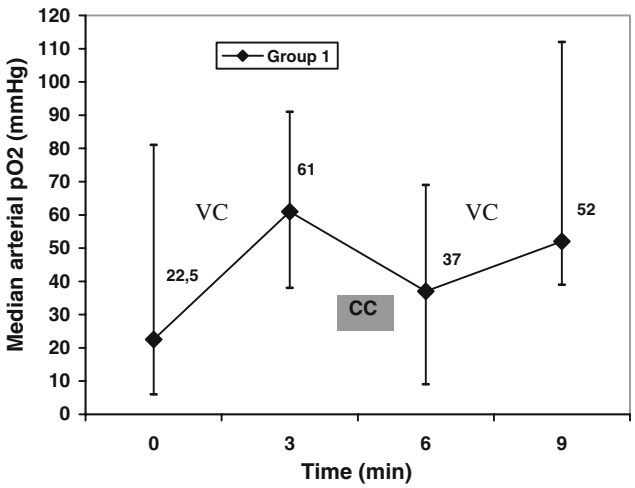

Fig. 4 Changes in the arterial PO_2 with ventilation and chest compressions (*VC*) and with chest compressions only (*CC*). *Group 1*: Initial period VC, second period CC, third period VC. *Group 2*: Initial period CC, second period VC, third period CC. The arterial PO_2 increased with VC and diminished with CC. The arterial PO_2 was higher with VC than with CC throughout the course of the experiment ($P < 0.05$)

It has been suggested that ventilation could cause hemodynamic deterioration due to the interruption of cardiac massage, decreasing cardiac output and the perfusion pressure of vital organs [3–8]. Moreover, ventilation could give rise to hypocapnia, with a secondary reduction in cerebral blood flow [3, 9–11]. However, other studies have found that poorer cerebral and coronary perfusion pressures are achieved with a low ventilatory frequency than with normal frequencies [20]. In our study, ventilation at 10 ml/kg and 20 bpm did not cause hyperventilation although the duration of CPR was short. Moreover, there were no significant differences in the hemodynamic parameters or in peripheral, cerebral, or renal saturations between the two techniques.

There are several limitations to our study. The animals remained intubated throughout the experiment, thus maintaining a patent permeable airway continuously. This would facilitate gas exchange. Cardiac massage was not interrupted during ventilation, and thus the study does not simulate initial bystander resuscitation. An FiO_2 of 50% was used for VC whereas during CC the FiO_2 was 21%. This could explain the differences in oxygenation found between the two techniques.

The design of our study enabled us to compare the two methods in a sequential manner in all animals, eliminating possible bias due to individual differences between the animals or in the cardiac massage performed by the rescuer. However, alternating the two methods has meant that it was not possible to detect more significant

differences in the results or to analyze the effect on survival. The evaluation of the quality of the chest compressions in the clinical setting is very difficult. In studies with manikins, it can be evaluated indirectly by the depth of chest compressions. However, these data can not be adequately evaluated in the clinical resuscitation or in our study due to the peak conformation of the infant pig chest. In our study, as in the clinical practice, the efficacy of the chest compression was evaluated in terms of the measured mean arterial pressure and indirectly by the peripheral and organ saturations.

We conclude that in asphyxial CA in an infant animal model, ventilation combined with chest compressions achieves better ventilation than CC without causing hemodynamic deterioration. The tidal volume achieved with CC is practically undetectable and the ventilation produced is insufficient to achieve adequate gas exchange.

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