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Difference in end-tidal CO_2 between asphyxia cardiac arrest and ventricular fibrillation/pulseless ventricular tachycardia cardiac arrest in the prehospital setting

Štefek Grmec, Katja Lah and Ksenija Tušek-Bunc

Center of Emergency Medicine, Prehospital Unit Maribor, Maribor, Slovenia

Correspondence: Katja Lah, katkalah@email.si

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Abstract

Introduction There has been increased interest in the use of capnometry in recent years. During cardiopulmonary resuscitation (CPR), the partial pressure of end-tidal carbon dioxide ($PetCO_2$) correlates with cardiac output and, consequently, it has a prognostic value in CPR. This study was undertaken to compare the initial $PetCO_2$ and the $PetCO_2$ after 1 min during CPR in asphyxial cardiac arrest versus primary cardiac arrest.

Methods The prospective observational study included two groups of patients: cardiac arrest due to asphyxia with initial rhythm asystole or pulseless electrical activity, and cardiac arrest due to acute myocardial infarction or malignant arrhythmias with initial rhythm ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT). The $PetCO_2$ was measured for both groups immediately after intubation and then repeatedly every minute, both for patients with and without return of spontaneous circulation (ROSC).

Results We analyzed 44 patients with asphyxial cardiac arrest and 141 patients with primary cardiac arrest. The first group showed no significant difference in the initial value of the $PetCO_2$, even when we compared those with and without ROSC. There was a significant difference in the $PetCO_2$ after 1 min of CPR between those patients with ROSC and those without ROSC. The mean value for all patients was significantly higher in the group with asphyxial arrest. In the group with VF/VT arrest there was a significant difference in the initial $PetCO_2$ between patients without and with ROSC. In all patients with ROSC the initial $PetCO_2$ was higher than 10 mmHg.

Conclusions The initial $PetCO_2$ is significantly higher in asphyxial arrest than in VT/VF cardiac arrest. Regarding asphyxial arrest there is also no difference in values of initial $PetCO_2$ between patients with and without ROSC. On the contrary, there is a significant difference in values of the initial $PetCO_2$ in the VF/VT cardiac arrest between patients with and without ROSC. This difference could prove to be useful as one of the methods in prehospital diagnostic procedures and attendance of cardiac arrest. For this reason we should always include other clinical and laboratory tests.

Keywords asphyxial cardiac arrest, end-tidal CO_2, prognosis

Introduction

Monitoring of end-tidal CO_2 has become a standard in the prehospital setting to ensure proper placement and function of the endotracheal tube and to help monitor the adequacy of ventilation [1]. In addition, it has been noted that cardiac arrest causes an abrupt fall in end-tidal CO_2 levels to values

CPR = cardiopulmonary resuscitation; $PetCO_2$ = partial pressure of end-tidal carbon dioxide; ROSC = return of spontaneous circulation; VF = ventricular fibrillation; VT = ventricular tachycardia.

near zero [2,3]. During cardiac arrest the partial pressure of end-tidal carbon dioxide ($PetCO_2$) falls to very low levels, reflecting the very low cardiac output achieved with cardiopulmonary resuscitation (CPR). It has been shown that the $PetCO_2$ achieved during advanced cardiac life support reliably predicts an outcome of cardiac arrest [2–12]. Higher levels of the $PetCO_2$ indicate better cardiac output, higher coronary perfusion pressure and a greater likelihood of successful resuscitation [13,14]. After the onset of cardiac arrest caused by ventricular fibrillation (VF), the $PetCO_2$ abruptly decreases to nearly zero and then begins to increase after the onset of effective CPR. Further increase is detected upon return of spontaneous circulation (ROSC) to normal or above-normal levels [2,3,9,12].

In an experimental animal model of asphyxial arrest during CPR, $PetCO_2$ levels were initially high (after the onset of arrest), then decreased to subnormal levels and then increased again to near-normal levels [15,16]. During a respiratory arrest, the cardiac output of pulmonary blood flow continues for some period of time prior to cardiac standstill. The CO_2 produced in the tissue during this period will continue to be delivered to the lungs, thereby increasing alveolar CO_2 (two-compartment hydraulic model of CO_2 kinetics). However, it is also important to recognize that it is not only the cessation of cardiac output alone that causes the fall of $PetCO_2$, but the cessation in conjuction with the washout of alveolar gas. This means that, in the absence of alveolar gas washout, CO_2 will remain in the lungs and probably that, as alveolar oxygen is being utilized, more CO_2 will be delivered.

On the basis of such a concept we built a hypothesis maintaining that the initial $PetCO_2$ should be higher in an asphyxial arrest model than in a VF/pulseless ventricular tachycardia (VT) cardiac arrest model. In the asphyxial cardiac arrest model there should also be no difference in patients with and without ROSC regarding the initial $PetCO_2$, since the initial $PetCO_2$ in this case reflects CO_2 cumulated in the alveolar compartment. This would suggest that the initial values of end-tidal carbon dioxide in asphyxial arrest do not have a prognostic value for ROSC as they do in VF/VT cardiac arrest.

If our results confirm both hypotheses, then this difference could be helpful in determining the mechanism of arrest in the prehospital setting.

Methods

This prospective observational study was conducted at the Center of Emergency Medicine, Maribor. The study included two groups of patients. The first group represented patients who suffered from heart arrest due to asphyxia. The causes of asphyxia included a foreign body in the airway, aspiration, suicide by hanging, drowning, edema or tumor of the airway, intoxication and acute asthma attack. The definitive cause of arrest has been confirmed in the hospital with further diagnostic and/or pathological report (autopsy). The initial rhythm was either asystole or pulseless electrical activity (all patients from this group with VT/VF as the initial rhythm were excluded). Patients with severe hypothermia (core temperature $<30°C$) were also excluded.

Table 1

Inclusion/exclusion criteria for the asphyxia group and the ventricular fibrillation/pulseless ventricular tachycardia (VF/VT) group of patients

VF/VT group
VF/VT initial rhythm
Age > 18 years
Core temperature > 30°C
Confirmed acute myocardial infarction and/or primary VF/VT (electrocardiogram, enzymes, autopsy, electrophysiological investigation)
Excluded patients with successful defibrillation in the first cycle
Excluded patients with acute myocardial infarction with asystole and pulseless electrical activity as the initial rhythm
Asphyxia group
Asystole and pulseless electrical activity as the initial rhythm
Excluded patients with VF/VT as the initial rhythm
Age > 18 years
Core temperature > 30°C
Excluded acute myocardial infarction as cause of arrest (clinical investigations and/or autopsy)
Etiology:
solid foreign body in the airway
aspiration
edema or tumor of the upper airway
hanging (excluded vasculatory or others causes of arrest – clinical investigations or autopsy)
Acute asthma attack (excluded cardiac causes of arrest)
Drowning (excluded cardiac causes of arrest)
Intoxications (excluded others causes of death – autopsy and/or added investigations in hospital

The second group included the patients with primary cardiac arrest (acute myocardial infarction or malignant arrhythmias). The initial rhythm was VF/VT (all patients from this group presenting with asystole or pulseless electrical activity were excluded). The definitive diagnosis (cause of arrest) was confirmed in the hospital (further diagnostic and/or pathological/autopsy report). The inclusion/exclusion criteria for asphyxia and VF/VT group are presented in Table 1.

The resuscitation procedures were performed by an emergency team (emergency medical doctor and two emergency

medical technicians or register nurses) in accordance with the International Liasion Committee on Resuscitation and European Resuscitation Council guidelines [17–19]. We used a manual technique to perform CPR. Pharmacologic interventions in individual patients were in accordance with the standards and guidelines of the International Liasion Committee on Resuscitation/European Resuscitation Council.

For management of VF or pulseless VT, direct-current countershocks were delivered by means of conventional techniques. $PetCO_2$ measurements were made by infrared sidestream capnometer (BCI Capnocheck Model 20600A1; BCI International Waukesha, WI, USA). Measurements for both groups were made immediately after intubation (first measurement) and then repeatedly every minute continuously. Endotracheal intubations were performed after two initial breaths with a valved bag at the beginning of CPR. Further ventilation was performed by mechanical ventilator (6–8 ml/kg at 10–12 breaths/min; Medumat Standard Weinmann, Weinmann, Namburg, Germany). The CO_2 cuvette was located in a connector between the mechanical ventilator and the endotracheal tube (it was applied to the endotracheal tube before intubation). Two patients were not intubated by the orotracheal technique because of complete obstruction of the upper airway, visualized by laringoscopy. In these two cases cricotireideotomy was performed using the traceoquick method (Tracheoquick Emergency Coniostomy Set; Willy Rüsch AG, Kernen, Germany). The procedure was performed in accordance with the instructions of the manufacturer, and both patients were successfully resuscitated and ventilated by mechanical ventilator.

The initial (first measurement after intubation), average (mean of all values obtained during a single resuscitation effort) and final (measurement at admission to hospital or discontinued CPR) $PetCO_2$ was detected for both groups. We performed the same procedure for the patients with ROSC and for those without ROSC.

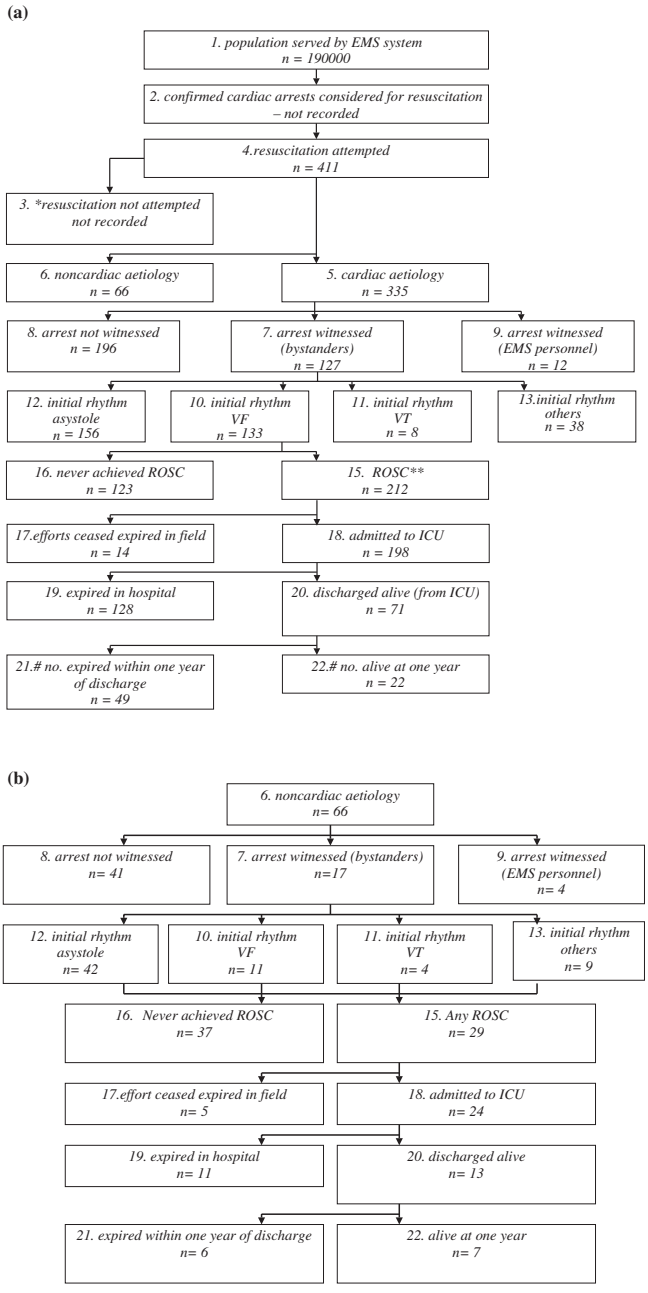
ROSC is defined as the return of spontaneous heartbeat or as palpable periferial arterial pulse and measurable systolic arterial pressure. As is seen from the Utstein style template, we distinguish intermittent ROSC, which is short in duration and a temporary event, from ROSC with hospitalization of a patient. In the present article, ROSC represents hospitalized patients.

The paired Student *t* test was used to compare initial and subsequent $PetCO_2$ values for each subject. For other parameters, both groups (asphyxial arrest group and VF/VT cardiac arrest group) were compared by Student's *t* test and the chi-squared test. Continuous variables are described as the mean ± standard deviation. $P < 0.05$ was considered significant.

Results

From February 1998 to October 2002 we analyzed 141 patients with primary cardiac arrest (initial rhythm VF/VT) and 44 patients with cardiac arrest due to asphyxia (initial rhythm asystole or pulseless electrical activity). The study environment, the prehospital environment and the characteristics of cardiac arrest and noncardiac arrest are displayed in Fig. 1a,b (Utstein style). The causes of asphyxial cardiac

Figure 1



(a) Cardiac arrests placed into the Utstein template. *It was not possible to determine the number of resuscitations not attempted because records for patients who were pronounced dead at the scene were not available. **Return of spontaneous circulation (ROSC). #Results before October 2002. **(b)** Non-cardiac arrests placed into the Utstein style. EMS, Emergency Medical Service; ICU, intensive care unit; VF, ventricular fibrillation; VT, ventricular tachycardia.

Table 2

Demographic and clinical characteristics of patients: a group with primary ventricular fibrillation/pulseless ventricular tachycardia (VF/VT) cardiac arrest and a group with asphyxial arrest

	Primary VF/VT cardiac arrest ($n=141$)	Asphyxial cardiac arrest (asystole and PEA) ($n=44$)	*P* value
Age (years)	65.8 ± 13.8	48.8 ± 20.1	< 0.05[b]
Gender (male/female)	82/59	27/17	0.83[c]
Response time (min)[a]	8.4 ± 5.7	8.9 ± 5.2	0.91[b]
Witnessed arrest (yes/no)	68/73	19/25	0.78[c]
Resuscitation by medical team (min)	28.3 ± 11.3	24.7 ± 13.4	0.76[b]
ROSC (yes/no)	101/40	18/26	< 0.05[c]
Discharged alive from ICU (yes/no)	38/103	7/37	< 0.05[c]
Average number of $PetCO_2$ observations	12.3 ± 3.4 (range, 7–22)	13.4 ± 2.8 (range, 9–28)	0.74[b]

ICU, intensive care unit; PEA, pulseless electrical activity; $PetCO_2$, partial pressure of end-tidal carbon dioxide; ROSC, return of spontaneous circulation.
[a]Time elapsed between the received 112 call to the arrival of Emergency Medical Service professionals at the patient's side.
[b]Student *t* test.
[c]Chi-squared test.

Table 3

The mean values for all patients of the initial, final, average and after 1 min of cardiopulmonary resuscitation (CPR) partial pressures of end-tidal carbon dioxide ($PetCO_2$) for arrest due to asphyxia and for ventricular fibrillation/pulseless ventricular tachycardia (VF/VT) cardiac arrest

	Initial $PetCO_2$ (mmHg)	$PetCO_2$ after 1 min of CPR (mmHg)	Average $PetCO_2$ (mmHg)	Final $PetCO_2$ (mmHg)
Asphyxial cardiac arrest	66.4 ± 17.3	29.1 ± 4.9	48.2 ± 10.1	27.3 ± 9.2
VT/VF cardiac arrest	16.5 ± 9.2	24.2 ± 5.1	17.3 ± 7.1	24.4 ± 10.3
P value (Student *t* test)	< 0.01	0.73	< 0.05	0.78

1 mmHg = 0.133 kPa.

arrest were solid foreign body in the airway (seven cases), aspiration (seven cases), edema or tumor of the upper airway (five cases), hanging (five cases), acute asthma attack (six cases), drowning (six cases) and intoxications with respiratory arrest (eight cases). Demographic and clinical characteristics for both groups are presented in Table 2.

The values of the $PetCO_2$ are presented in Table 3. In the group of patients who presented with arrest due to asphyxia there was no significant difference in the initial values of $PetCO_2$, even when we compared those with and without ROSC (70.1 ± 15.3 mmHg versus 62.8 ± 16.2 mmHg, $P=0.64$). On the contrary, in the group of patients who presented with VF/VT arrest there was a significant difference in the initial values of $PetCO_2$ between patients without and with ROSC (8.2 ± 4.3 mmHg versus 20.3 ± 6.2 mmHg, $P=0.04$). In all patients with ROSC the initial $PetCO_2$ was higher than 10 mmHg. The values of the $PetCO_2$ after 1 min of CPR did not differ significantly among the two groups. In both groups significantly higher values were achieved in patients with ROSC than in those without ROSC (asphyxial arrest group, 35.8 ± 8.6 mmHg versus 19.4 ± 8.7 mmHg, $P<0.05$; VF/VT arrest group, 30.2 ± 8.3 mmHg versus 14.2 ± 5.2 mmHg, $P<0.05$). The values of the final $PetCO_2$ in both groups were significantly higher in patients with ROSC than in the patients without ROSC (asphyxial arrest group, 31.2 ± 8.4 mmHg versus 7.2 ± 3.3 mmHg, $P<0.05$; VF/VT arrest group, 28.1 ± 4.8 mmHg versus 6.2 ± 2.8 mmHg, $P<0.05$).

Discussion

In the present study we confirmed that the $PetCO_2$ was markedly elevated during the first minute of CPR in asphyxial cardiac arrest. This study therefore confirmed the results of the studies that used animal models in which cardiopulmonary arrest was induced by asphyxia. In the present study the $PetCO_2$ values during CPR were initially high, then decreased to subnormal levels and then increased again to near-normal levels in patients with ROSC. This pattern of $PetCO_2$ changes is different from the pattern observed in

cardiac arrest caused by VF, since cardiac arrest from VF results in an abrupt cessation of cardiac output and pulmonary blood flow. Bhende and colleagues [15], Berg and colleagues [16] and von Planta and colleagues [20] concluded that, during the period of asphyxia, continued cardiac output prior to cardiac arrest permits continued delivery of CO_2 to the lungs, which (in the absence of exhalation) results in higher alveolar CO_2. This is reflected as increased $PetCO_2$ when ventilation is resumed.

Understanding the physiology of CO_2 production, delivery to the lungs and excretion are important in order to appropriately interpret $PetCO_2$ monitoring during CPR. The disposition of CO_2 can also be represented in a hydraulic model [21]. The large peripheral tissue compartment drains through a conduit (cardiac output) into the small central pulmonary compartment. The tissues produce CO_2, which empties into the peripheral tissue compartment. Carbon dioxide then flows by gravity (cardiac output) from the higher level tissue to the lower level pulmonary compartment. Alveolar ventilation, which equals expired ventilation minus ventilation of the anatomical dead space, and the effects of high ratio ventilation/perfusion matching eliminate CO_2 from the lung. In this model the cardiac output affects the distribution and total amount of CO_2 in the body and can help to understand the meaning of the $PetCO_2$ during CPR.

The present study discovered that we can trace the same pattern of $PetCO_2$ changes in the asphyxial arrest as were described in the animal models in the first minute after arrest [15,16], even after a longer period of time due to the access time. The inability to measure the $PetCO_2$ immediately after cardiac arrest was the main disability of this study.

We also concluded that the high initial values of the $PetCO_2$ in asphyxial arrest do not have a prognostic value for the appearance of ROSC as they do in the VT/VF cardiac arrest. On the contrary, the values after 1 min of CPR and also the final values of the $PetCO_2$ do have the prognostic value for ROSC. These data, like those from Berg and colleagues [16], suggest that the $PetCO_2$ during the initial phase of CPR of asphyxial arrest (1 min after intubation and cardiac massage) reflects alveolar CO_2 prior to CPR. In the asphyxial model, cellular respiration results in continued oxygen consumption and CO_2 production. The high pressure of CO_2 in the alveolar compartment is reflected in the high $PetCO_2$ during the initial phase of CPR.

The fast decline of the high values of the $PetCO_2$ can therefore only be interpreted by ventilation of the alveolar compartment, which then rapidly decreases the $PetCO_2$. However, in the next phase and with the beginning of CPR we can again detect the rise of the $PetCO_2$. This rise is achieved by successful cardiac massage, which washes the acumulated CO_2 out of the peripheral compartment [11,21–23].

Key messages

- $PetCO_2$ correlates with cardiac output and has a prognostic value for CPR
- The pattern of $PetCO_2$ changes in asphyxia is different from the pattern of $PetCO_2$ changes in VF/VT cardiac arrest
- Differences in the initial values of $PetCO_2$ can be useful in differentiating between the causes of cardiac arrest
- Initial values of $PetCO_2$ cannot be used as a prognostic factor for CPR in asphyxia arrest
- Values after 1 min of CPR in asphyxia arrest can be used as a prognostic factor for CPR

The acquaintance with this pattern of changes can be helpful in differentiation of cardiac arrest causes and in identification of mechanisms that led to cardiac arrest. This is very useful in the prehospital setting and can lead the course of action as hypoxia is a potentially reversible cause of cardiac arrest. The issue is potentially important when deciding upon the most effective sequence of resuscitation intervention. There is growing evidence that indicates positive pressure ventilation may be postponed for several minutes in instances of arrythmic arrest whereas it might be life-saving in instances of asphyxial arrest. The emergency medical doctor can therefore be orientated with greater reassurance towards the measures that are useful in asphyxial arrest [24–27]. However, one has to be aware that the initial values of the $PetCO_2$ in asphyxial arrest do not have the prognostic value for the outcome of CPR that they do have in VF/VT arrest [4–6,8–10].

Conclusions

The initial values of the $PetCO_2$ in asphyxial cardiac arrest are significantly higher than in VF/VT cardiac arrest. In asphyxial arrest there is also no significant difference in inital values of the $PetCO_2$ in patients with and without ROSC. In asphyxial arrest the initial values of the $PetCO_2$ therefore cannot be used as a prognostic factor of outcome of CPR, as they can be used in VF/VT cardiac arrest. This difference, together with other criteria, can therefore be useful for differentiation between the causes of cardiac arrest in the prehospital setting. For standard use of this difference in the $PetCO_2$ in the prehospital setting we suggest additional clinical research.

Competing interests

None declared.

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