

End Tidal CO₂ Monitoring in Condition of Constant Ventilation: A Useful Guide during Advanced Cardiac Life Support

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Abstract: Success of advanced cardiac life support (ACLS) depends on several factors: character and severity of the primary insult, time interval between cardiac arrest and effective basic life support (BLS) and the ensuing ACLS, patient's general condition before the insult, environmental circumstances and efficacy of BLS and ACLS. From these factors, only the efficacy of ACLS is under control of emergency personnel. The end tidal partial pressure of CO₂ (P_{ET}CO₂) has been shown to be an indicator of the efficiency of ACLS and a general prognostic marker. In this study P_{ET}CO₂ was monitored during out-of hospital ACLS in three cases of cardiac arrest of different aetiology. The aetiology included lung oedema, tension pneumothorax and high voltage electric injury. P_{ET}CO₂ served for adjustments of ACLS. In these three cases the predictive value of P_{ET}CO₂ monitoring corresponded to previously reported recommendations.

Introduction

Cardiac arrest is a dynamic process with many important pathophysiological consequences occurring within seconds and minutes. Many studies have been done with the aim of improving advanced cardiac life support (ACLS) in order to preserve the patient's prearrest level of neurological functions. Improvement of multifunctional monitoring devices, working in real time and storing data, helps to analyse the process at the scene as well as later on, when final outcome of the rescue effort is known.

Success of ACLS is determined by many factors: severity of the primary insult, time interval between cardiac arrest and effective BLS and ACLS, patient's general state of health, causes evoking cardiac arrest, environmental circumstances, BLS and ACLS efficacies. From these numerous factors, only ACLS efficacy is under control of its providers.

P_{ET}CO₂ reflects tissue metabolism, tissue and lung perfusion, and alveolar ventilation including its distribution. Capnometry, measuring P_{ET}CO₂, has been demonstrated as a useful tool for verification of correct orotracheal intubation [1], assurance of adequate ventilation [2], prognosis in patients with normothermic nontraumatic cardiac arrest, an indirect control of the cardiac output, the first clinical indicator of the restoration of spontaneous circulation (ROSC) [3], and indirect information about cerebral perfusion [4].

This paper analyses three cases of cardiac arrest due to very different aetiology in which P_{ET}CO₂ monitoring was used to control the efficacy of ACLS.

Materials and methods

A professional team with physician resuscitated three out-of-hospital patients suffering cardiac arrest. The team was working in a "Rendezvous system", i.e. two EMS (Emergency Medical Service) teams, consisting of a large ambulance (driver and paramedic) and a small rendezvous care vehicle (physician and paramedic), met at the scene.

All the patients were intubated and the lung ventilation was performed by an automatic device. A constant tidal volume and constant ventilatory frequency were held during all out-of-hospital care. Chest compressions were independent of the lung ventilation [5].

The end tidal $P_{ET}CO_2$ was monitored by “Zoll M series” instrument. The Zoll M series option continuously measures carbon dioxide and respiratory rate using a unique mainstream, solid-state infrared sensor Capnostat (Novamatrix Corporation). The Capnostat sensor is attached to an airway adapter connected to an endotracheal tube. Infrared light beams through the airway adapter to a detector on the opposite side of the airway. The CO_2 concentration in breathing gases is continuously recorded according to the light absorption. $P_{ET}CO_2$ is automatically indicated in millimetres of mercury (mmHg). The respiratory rate is calculated from intervals between CO_2 peaks.

Results

■ Case 1: Asystole – female – age 67

Diagnosis: pulmonary oedema and chronic renal insufficiency

Outcome: sent home without neurological disability

Course of ACLS:

08:40:00 emergency call: dyspnoea (after a few seconds the patient lost consciousness, persisting gasping)

08:46:00 emergency personnel, 4 members, at patient's side

Patient's condition – coma, apnoea, mydriasis and asystole, sitting on the armchair, no BLS prior to arrival of EMS had been started.

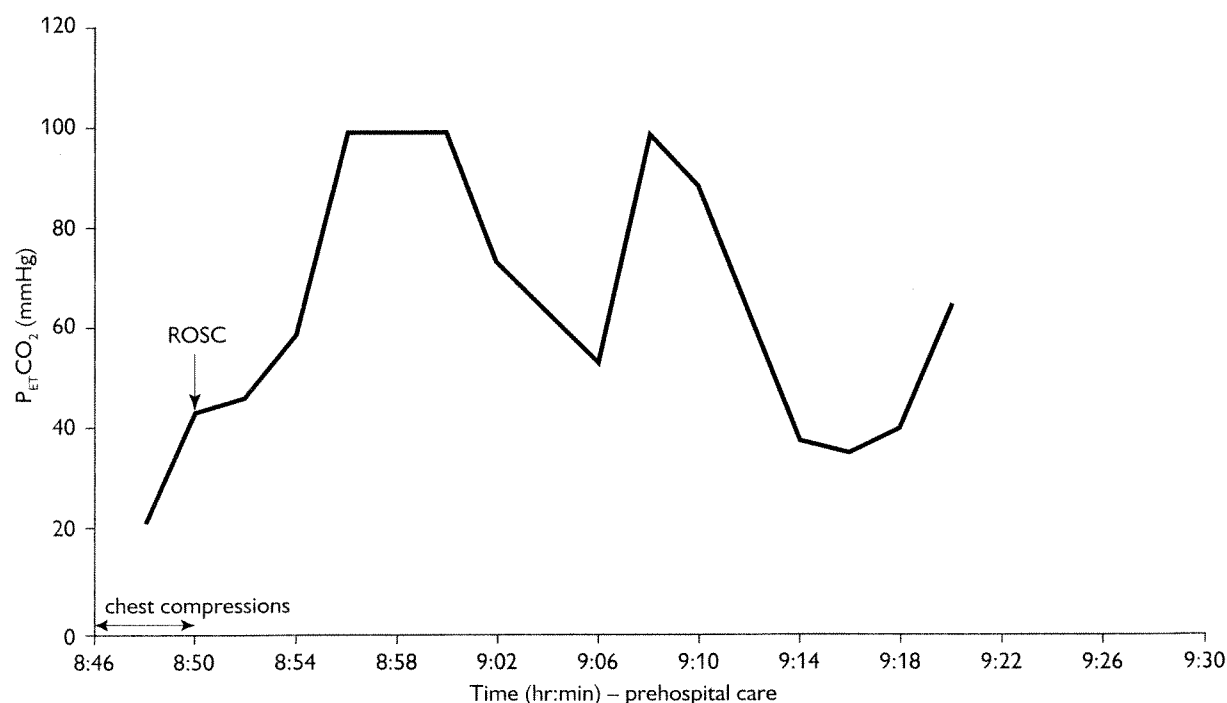


Figure 1 – Time course of resuscitation in the patient with pulmonary oedema.

Bystander history – patient didn't sleep all night, complained of dyspnoea for many hours, patient took several different medicines.

EMS details – the patient was placed in a horizontal position, the physician performed intubation (absence of laryngeal reflex) and started artificial ventilation, one paramedic started chest compressions and the second paramedic prepared i.v. line and prepared adrenalin, the driver fixed ECG leads and confirmed asystole.

08:47:20 $P_{ET}CO_2$ monitoring was started

08:48:04 adrenalin 2 mg i.v.

08:49:25 ECG: AV block III. degree, atropine 1 mg i.v.

08:50:03 ROSC: sinus rhythm, palpable peripheral pulse

08:54:30 restoration of spontaneous respiratory effort – application of a hypnotic and paralytic medication

08:55:00–9:30:00 transport to a hospital, haemodynamically stable during the transportation

09:30:00 admission to hospital (ICU)

14:00:00 consciousness restored

18:00:00 extubation

History added at the ICU: The patient had chronic renal insufficiency, anuric, dialysed three times a week, suffered first cardiac arrest with successful ACLS three years ago.

Description of $P_{ET}CO_2$ levels record (Figure 1): The first measured value of $P_{ET}CO_2$ was 21 mmHg (2,79 kPa) and there was a further increase during initial resuscitation. An additional significant rise of $P_{ET}CO_2$ occurred following ROSC. The decrease during the interval of 15 to 20 minutes occurred during the patient's transport down the stairs performed in the position "legs first". After restoration of a horizontal body position $P_{ET}CO_2$ value increased again to supranormal values.

Feedback from $P_{ET}CO_2$: The typical significant rise in $P_{ET}CO_2$ following ROSC has been shown. The change in patient's body position during transport caused a decrease in $P_{ET}CO_2$. This was attributed to a sudden redistribution of blood in the circulation compromising the lung perfusion.

■ **Case 2: Ventricular fibrillation – male – age 22**

Diagnosis: polytrauma and tension-pneumothorax

Outcome: death 8 hours after admission to the hospital

Course of ACLS:

16:54 emergency call: traffic accident, young man

17:10 arrival of EMS ambulance, aero-medical assistance requested

Patient's condition – coma, apnoea, mydriasis

Bystander history – motorcycle accident victim, found in a coma near the road in a rural area, circumstances of the accident and the accident time unknown

EMS details – ECG-ventricular fibrillation, immediately defibrillated, after

defibrillation asystole, intubation and artificial ventilation, chest compressions and adrenalin 3×2 mg i.v., Solu Medrol (suspected spinal injury), Hydroxyethylaminum solution 10% (suspected haemorrhagic shock), all these interventions were unsuccessful

17:36 aero medical physician at patient's side

17:36–17:40 treatment by chest compressions and mechanical ventilation, by auscultation there were quiet breath sounds on the right side and extremely quiet breath sounds at the left side, replacing of ET tube was without effect, reintubation was also without effect

17:38 $P_{ET}CO_2$ monitoring was started

17:40 suspicion of pneumothorax

17:42 chest tube inserted

17:43 adrenalin 2 mg i.v

17:44 ROSC

17:47 sinus rhythm, central and peripheral pulse palpable

17:47–18:20 transport to a distant hospital, patient was haemodynamically unstable and therefore dopamine, noradrenalin, crystalloids (500 ml sodium chloride solution 0,9%, 500 ml Ringer's solution) Haemacell 1000 ml and Hydroxyethylaminum solution 10% 500 ml were administered during transportation

18:20 admission to hospital

8 hours later death

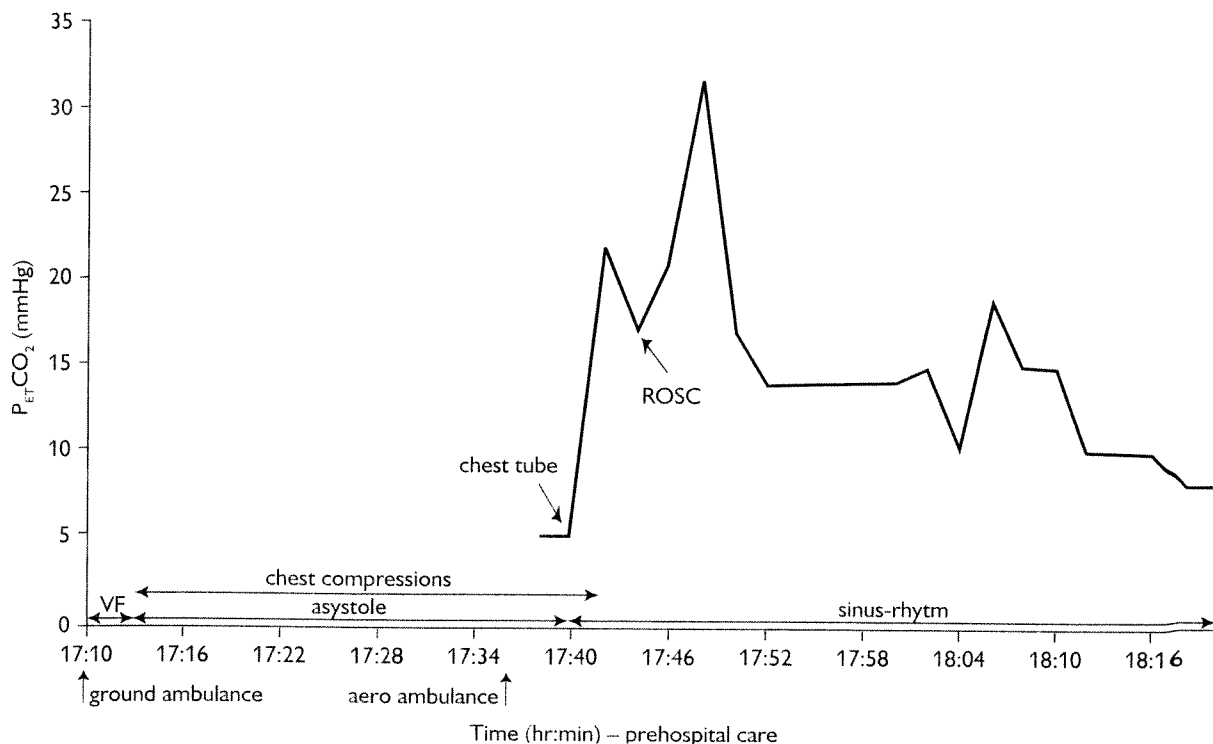


Figure 2 – Time course of resuscitation in the patient with tension-pneumothorax.

Description of $P_{ET}CO_2$ levels record (Figure 2): The first $P_{ET}CO_2$ value 5 mmHg (0,66 kPa) was low and there was no significant rise during initial ACLS interventions. $P_{ET}CO_2$ levels increased to 22–40 mmHg (2,93–5,33 kPa) after chest tube insertion and ROSC, but it decreased again and remained low.

Feedback from $P_{ET}CO_2$: The low initial $P_{ET}CO_2$ level indicated several possible problems (hypothermia, wrong intubation, inadequate ventilation, low cardiac output, a measurement failure). In cases of tension pneumothorax with automatic volume control ventilation, a low $P_{ET}CO_2$ may be caused by compromised blood return and, consequently a low cardiac output.

■ **Case 3: Ventricular fibrillation – male – age 17**

Diagnosis: electrocution and extensive burn of the chest and right arm

Outcome: discharge without neurological damage

Course of ACLS:

12:57 the accident occurred

12:58 emergency call

Patient's condition – patient in a horizontal position, fine ventricular fibrillation, mydriasis without photoreaction, extensive burns of the chest and on the right arm.

Bystander's history – electrocution by 22 kV at an electric substation, first aid provided by extrication from electric substation and BLS started

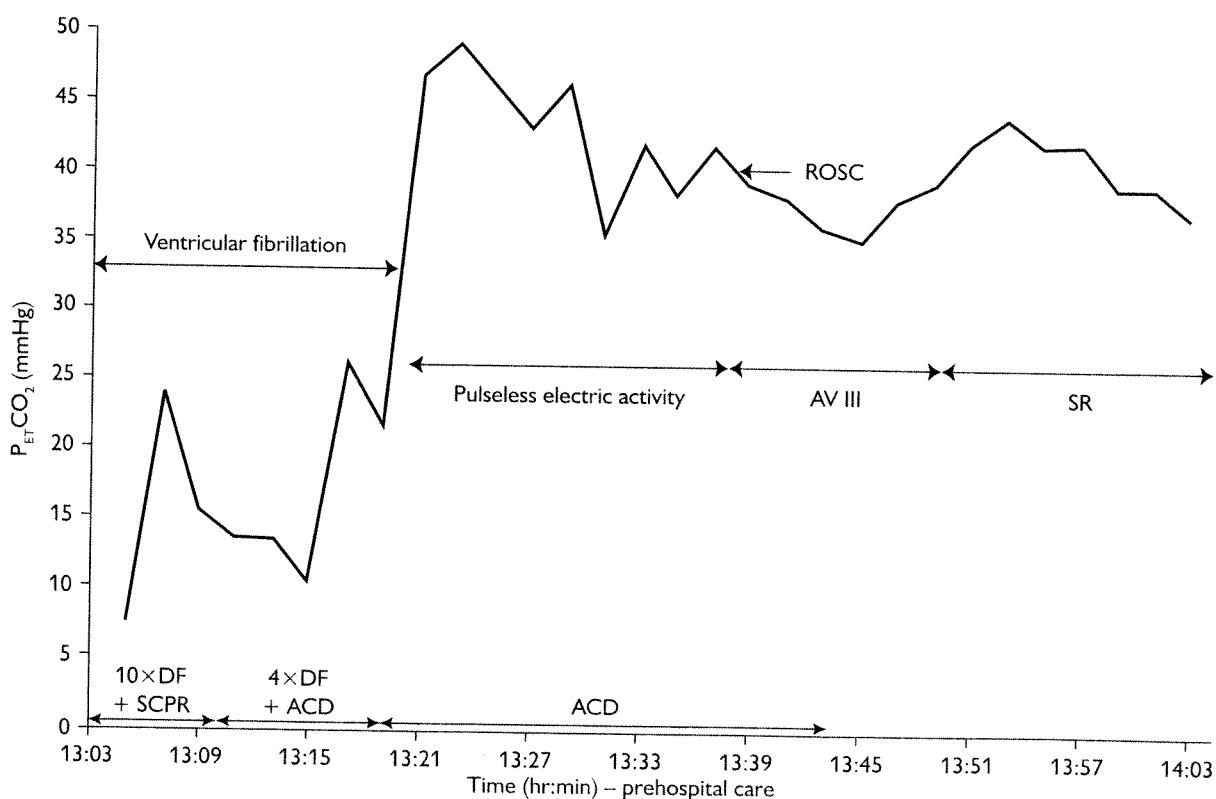


Figure 3 – Time course of resuscitation in the patient with electrocution.

13:03 EMS paramedics team arrived – standard chest compressions and ventilation by a self-inflating bag started

13:04 EMS physician team arrived

EMS details – (13:04 – 13:06) physician (1) intubation and mechanical ventilation and introduction of i.v. line through vena jugularis externa, driver (2) standard chest compressions, paramedic (3) adrenaline 2 mg via endotracheal tube and 2 mg i.v., paramedic (4) ECG monitoring (fine ventricular fibrillation) and defibrillator preparation, 3 defibrillations (a biphasic shock) were unsuccessful, mydriasis with lazy photoreaction still present, adrenaline 2 mg i.v.

13:05 $P_{ET}CO_2$ monitoring was started (7 mmHg, 0,93 kPa)

13:06–13:20 automatic volume mandatory ventilation, 11 repeated defibrillation shocks because of continuing ventricular fibrillation, pharmacotherapy supplemented with amiodarone, lidocaine, bicarbonate and $MgSO_4$

13:10 standard chest compressions converted to active compression/decompression by a cardio pump (ACD)

13:20–13:38 pulse-less ECG electrical activity, mydriasis and pharmacotherapy continued with atropine 1 mg, noradrenalin, infusion containing dopamine, body fluid replacement (500 ml sodium chloride solution 0,9%, 500 ml Hartman's solution, gelofusine 500 ml), ACD-chest compression/decompression was continued because if these were interrupted, central pulse was not palpable and immediately also $P_{ET}CO_2$ values dropped. [Image1]

13:38 ROSC (after 35 minutes) – ECG-AVIII, 13:50 – sinus rhythm and peripheral pulse palpable

13:42–14:05 transport to hospital

In the hospital: Myocardial ejection fraction was 40%, burns over 13% of the body surface of IIA and IIB degree, extubation 4 days later and full consciousness 6 days later

Description of $P_{ET}CO_2$ levels record (Figure 3): The first measured $P_{ET}CO_2$ was low but a rapid increase during initial resuscitation occurred, followed by low values again (the initial standard manual CPR was repeatedly interrupted by numerous DF attempts). The active compression/decompression by a cardio-pump was followed immediately by the significant rise of $P_{ET}CO_2$. Pulse-less electric activity first appeared at ECG after 19 minutes and there was a rise in $P_{ET}CO_2$ to a physiological value around 40 mmHg.

Feedback from $P_{ET}CO_2$: A low $P_{ET}CO_2$ and its decreases indicated that initial ACLS was not optimal. The change from standard chest compressions to cardio-pump (ACD) induced a significant increase of $P_{ET}CO_2$ and consequently ACD maintained a high level of $P_{ET}CO_2$. The following high $P_{ET}CO_2$ indicated that a satisfactory artificial circulation was achieved and thus ACLS was improved. This is a typical example of when the ACLS should not be discontinued, despite the long period without ROCS.

Discussion

$P_{ET}CO_2$ measurement (capnography) is a simple method that can be used immediately after orotracheal intubation. The only procedure is to place the detector into the endotracheal tube. Interpretation of $P_{ET}CO_2$ assumes that the lung ventilation is maintained constant because $P_{ET}CO_2$ is inversely related to the lung (alveolar) ventilation. Consequently changes in the total alveolar ventilation would interfere with the information regarding conditions of the circulation and of the tissue metabolism. Capnography in condition of constant ventilation provides integrated information on aerobic tissue energy metabolism, tissue perfusion, and the perfusion of ventilated lung alveoli (all variables tightly linked to the cardiac output). Several studies, both experimental [7], and clinical [8, 9, 10], have demonstrated a positive correlation between a wide range of cardiac outputs and $P_{ET}CO_2$ values. Capnography is thus a useful tool to monitor efficacy of the ACLS at the scene and has a strong predictive value for the ultimate outcome of it.

Callham and Barton [11] found that 14 patients out of 55 nontraumatic out-of-hospital cardiac arrest patients who achieved ROSC had a mean $P_{ET}CO_2$ value of 19 ± 14 (SD) mm Hg at the start of resuscitation while those who did not achieve ROSC had only 5 ± 4 (SD) mm Hg.

Asplin and White [12] related $P_{ET}CO_2$ values recorded 1 and 2 minutes after the start of resuscitation and the maximum values achieved with ROSC on-scene. The patients that achieved ROSC had all these values significantly higher than those who were not successfully resuscitated.

Wayne et al [13] tested the level of $P_{ET}CO_2$ of 10 mm Hg after 20 minutes of the resuscitation effort as a hypothetical measure of the survival among 90 patients with the out-of-hospital cardiac arrest with pulseless electrical activity. Although the initial values of $P_{ET}CO_2$ did not differ, with the mean value of 11.7 in survivals and 10.9 mm Hg in non-survivals, the values were significantly different after 20 minutes (mean value of 3.9 mm Hg in nonsurvivals against 31 mm Hg in survivals) and the tested value of 10 mm Hg after 20 minutes was found highly predictive for the survival. Later, the authors confirmed this in another study of 150 patients [14]. Cantineau et al [16] concluded from their prospective study of 120 patients with nontraumatic out-of-hospital cardiac arrest that $P_{ET}CO_2$ of 10 mm Hg, as a maximum values achieved during the first 20 minutes of resuscitation, could be used as a cut-off value to discriminate between patients that could achieve ROSC from those who did not. Ahrens et al [15] arrived at a similar conclusion, regarding the predictive value of 10 mm Hg of $P_{ET}CO_2$ after 20 minutes of resuscitation, in a study that enrolled 127 patients. Grmec and Kupnik [17] concluded, from a study including 246 patients with normothermic nontraumatic cardiac arrest, that initial and final values of $P_{ET}CO_2$ of less than 10 mm Hg (1.33 kPa) were incompatible with survival.

The three patients presented in this study suffered a cardiac arrest from a nontraumatic (case 1) and traumatic (case 2 and 3) causes. The monitoring of

$P_{ET}CO_2$ provided information on the effectiveness of ACLS measures that were, however, highly variable.

In the case of the pulmonary oedema already the initial $P_{ET}CO_2$ was relatively high that might be caused by a likely significant hypercapnia preceding the cardiac arrest and resulting from a long lasting period of severe dyspnoea. ROCS resulted in further significant improvements in tissue and lung perfusions as indicated with a rapid and steep increase in $P_{ET}CO_2$ that reached very high levels. The high supranormal values of $P_{ET}CO_2$ indicated an intensive oxidative energy metabolism in tissues due to their adequate perfusion. The supranormal values of $P_{ET}CO_2$ persisted for almost 20 minutes. With regard to long-term dyspnoea before cardiac arrest, it can be assumed that previously accumulated CO_2 had been washed out from the blood and tissues compartments and that accumulated lactic acid had been converted to CO_2 . This was predictive of the good outcome which was confirmed. $P_{ET}CO_2$ transiently decreased during the phase of transport down the staircase in a vertical position legs-first. This was attributed to a negative influence of the gravitation on the blood venous return and the cardiac output. $P_{ET}CO_2$ increased back as expected after the patient resumed a horizontal position. The assumption of influence of the gravitation was thus confirmed.

The second case was the trauma due to a road accident. The $P_{ET}CO_2$ monitoring started in the already intubated patient, when ACLS had been provided for about fifteen minutes. The initial value of $P_{ET}CO_2$ of only 5 mmHg (0,66 kPa) led to verification of the correctness of the intubation. The limited rise in $P_{ET}CO_2$ indicated continuing significant problems with the blood circulation and lung perfusion. Promptly after the tension-pneumothorax was diagnosed and a drainage of the pleural cavity introduced, the $P_{ET}CO_2$ values raised significantly. The following decrease of $P_{ET}CO_2$ below 10 mmHg (1,33 kPa) indicated worsening of tissue and lung perfusions and predicted an unfavourable prognosis.

The third case was an injury by a high voltage alternating electric current. The monitored rise of $P_{ET}CO_2$ indicated effective improvement of the ACLS particularly due to using a cardio-pump. The effort directed to achieve ROSC was repeatedly unsuccessful, probably due to a significant damage to the myocardium from the high voltage electric injury. However, the high values of $P_{ET}CO_2$ indicated that the oxidative tissue energy metabolism, the circulation and the respiratory gases exchange were sufficiently achieved by the ACLS. These high values of $P_{ET}CO_2$ were the main reason why the EMS team continued in life saving efforts in spite of all others indices leading to termination. The ACLS, taking more than half an hour, was finally fully successful.

Conclusion

The three cases demonstrate the usefulness of the $P_{ET}CO_2$ monitoring in evaluation of the efficacy of different procedures used during ACLS in patients suffering the cardiac arrest from different etiologies. $P_{ET}CO_2$ reflects tissue

metabolism, tissue and lung perfusion. Because $P_{ET}CO_2$ reflects alveolar ventilation as well, the condition of constant ventilation is crucial. It has been shown in the text that the $P_{ET}CO_2$ monitoring is a useful indicator of the efficiency of provided ACLS particularly during the period from its beginning till the restoration of spontaneous circulation (ROSC).

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