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What is This?
Impact of Modified Treatment in Echocardiographically Confirmed Pseudo-pulseless Electrical Activity in Out-of-hospital Cardiac Arrest Patients with Constant End-tidal Carbon Dioxide Pressure during Compression Pauses

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This study evaluated the ability of focused echocardiography (FE) and capnography to differentiate between pulseless electrical activity (PEA) and pseudo-PEA in out-of-hospital cardiac arrest, and the potential survival benefits with modified treatment. In PEA patients with stable end-tidal carbon dioxide pressure ($P_{et CO_2}$) during the compression pause and concomitant FE showing cardiac kinetic activity, the compression pause was prolonged for 15 s and an additional 20 IU vasopressin was administered. If pulselessness persisted, compressions were continued. Fifteen of the 16 patients studied (94%) achieved restoration of spontaneous circulation (ROSC); eight patients (50%) attained a good neurological outcome (Cerebral Performance Category 1 – 2). In an historical PEA group with stable $P_{et CO_2}$ values ($n = 48$), ROSC was achieved in 26 patients (54%); four patients (8%) attained Cerebral Performance Category 1 – 2. Echocardiographical verification of the pseudo-PEA state enabled additional vasopressor treatment and cessation of chest compressions, and was associated with significantly higher rates of ROSC, survival to discharge and good neurological outcome.

KEY WORDS: Cardiopulmonary resuscitation; Echocardiography; Capnography; Pulseless electrical activity; Vasopressin

Introduction

The ability to determine the presence or absence of a central pulse remains a key skill for healthcare providers in cardiopulmonary resuscitation (CPR), although some studies show that they perform it poorly.1 – 3 Life-support guidelines for healthcare...
professionals recommend a carotid pulse check of up to 10 s for each assessment (every 2 min in pulse-associated rhythm). Several consensus groups have evaluated and confirmed the usefulness of focused echocardiography (FE) in resuscitation management. Some studies have suggested the use of ultrasound detection of cardiac activity during the time available for pulse checks. This is especially important in the management of resuscitation-generated (secondary) pulseless electrical activity and in differentiating between pulseless electrical activity (PEA) and pseudo-PEA. In addition, clinical studies of capnography show a strong correlation between end-tidal carbon dioxide pressure (P\textsubscript{etCO}_2) and cardiac output, coronary perfusion pressure, cerebral perfusion pressure, restoration of spontaneous circulation (ROSC) and hospital discharge. Use of capnography throughout resuscitative efforts provides information regarding ROSC and response to resuscitation. The present study aimed to assess the value of echocardiographically confirming pseudo-PEA in out-of-hospital cardiac arrest (OHCA) patients with constant values of P\textsubscript{etCO}_2 during compression pauses, and the impact of modifying treatment.

**Patients and methods**

**STUDY SETTING**

All resuscitations were conducted in the city of Maribor and adjacent rural areas, encompassing a population of 200 000 inhabitants spread over an area of 780 km\(^2\). The emergency medical services are accessed through a single emergency number (112) and include two pre-hospital emergency teams with advanced life support (ALS) capability, two basic life support (BLS) teams and, during daytime from April to October, a rescuer on a motorcycle.

**PATIENTS**

The modified resuscitation protocol was only considered in patients aged between 18 and 80 years of age who had sustained non-traumatic, normothermic OHCA between November 2007 and October 2008. Exclusion criteria included: terminal illness; cardiac arrest secondary to trauma, drowning or hanging; severe hypothermia (< 30°C); spontaneous circulation regained before administration of a vasopressor agent. Outcomes were compared with those of an historical group with post-resuscitation PEA with stable P\textsubscript{etCO}_2 values (signalling some degree of spontaneous cardiac output), who received standard management without FE according to the 2005 European Resuscitation Council guidelines.

The modification of the resuscitation protocol was approved by the Ethical Board of the Ministry of Health, Republic of Slovenia. The need for informed consent was waived.

**RESUSCITATION PROTOCOLS**

**Standard protocol**

Cardiopulmonary resuscitation was initially attempted using regionally developed protocols, which incorporate the 2005 International Liaison Committee on Resuscitation (ILCOR) recommendations. The ALS team instigated the resuscitation effort if they arrived at the scene first or took over ongoing efforts by the BLS team. The ALS team placed an endotracheal tube, verified its proper position by capnography, and initiated positive pressure ventilation with a tidal volume of 6 ml/kg delivered at 10 times/min. In all cases where ultrasound was used, the ALS units were staffed with an emergency physician (an ALS instructor with basic training in the use of FE). Through intravenous (i.v.) access (external jugular or cubital vein), 250 ml of 7.2% saline in 6% hydroxyethyl starch (HyperHaes\textsuperscript{®} solution,
Fresenius Kabi (Bad Homburg, Germany) was administered as a first fluid, together with appropriate resuscitation drugs. Additional fluid, including 0.9% saline, was given intravenously at the discretion of the rescuers.

Cardiac rhythm and peripheral pulses were checked every 2 min. If ventricular fibrillation (VF) or pulseless ventricular tachycardia (pVT) were present, a single 150 J biphasic waveform electrical shock was delivered (LifePak® 12, Physio-Control, Medtronic, Minneapolis, MN, USA) and chest compressions resumed for another 2 min before re-assessing rhythm and pulse. If the patient remained in VF/pVT after the second defibrillation attempt, or if the patient had PEA or asystole as the presenting rhythm, 40 IU vasopressin (i.v. bolus) was given followed by 1 mg adrenaline (i.v. bolus) every 4 min. For shock-resistant VF/pVT, 300 mg amiodarone (i.v. bolus) was given between the third and fourth electrical shock. For PEA or asystole, 3 mg atropine (i.v. bolus) was given.

Patients who had ROSC in the field were started on 30 ml/kg 0.9% saline solution cooled at 4°C (i.v. infusion at 100 ml/min) and given 0.08 – 0.10 mg/kg vecuronium bromide (Norcuron®, Organon USA, Roseland, NJ, USA) (i.v. bolus injection) to initiate hypothermia while en route to the hospital. Patients were admitted directly to the intensive care unit (ICU) at the University Clinical Centre in Maribor and cooled to a core temperature between 32°C and 34°C by external means until they regained consciousness or had been hypothermic for 24 h.

**Modified protocol**

If PEA appeared after the initial resuscitation, it was classified as secondary or post-resuscitation PEA. In these patients, a subxiphoid view FE examination was performed during the subsequent pulse check, using a Titan portable ultrasound machine (SonoSite Inc., Bothell, WA, USA) to assess for the presence of cardiac kinetic activity (synchronous myocardial wall and valvular motion) and, therefore, pseudo-PEA. The FE examinations were undertaken in accordance with a modified algorithm for FE evaluation during resuscitation, as described by Breitkreutz et al.4 (Fig. 1).

All patients were continuously monitored using sidestream capnography. Patients with pseudo-PEA identified on FE examination who had a constant $P_{et}$CO$_2$ throughout the 10 s pulse check period were immediately given an additional 20 IU vasopressin and 0.9% saline (i.v. bolus injection), and the compression pause was prolonged for a maximum of 15 s. If the pulse remained absent after 15 s, chest compressions and standard ALS management were resumed.

**OUTCOME MEASUREMENTS**

Data were collected according to the Utstein style.20 The primary outcome was ICU admission. Secondary outcomes were ROSC in the field, survival at 24 h and survival to hospital discharge. In addition, neurological outcome was measured at hospital discharge using the Cerebral Performance Categories (CPC) Scale, a five-point grading where CPC 1 indicates good cerebral performance and CPC 5 indicates brain death, apnoea, areflexia etc.23

**STATISTICAL ANALYSES**

Data were presented as the mean ± SD. Odds ratios with 95% confidence intervals were calculated for the primary and secondary outcomes; both unadjusted and adjusted using multiple logistic regression. Differences between additional categorical variables were analysed using the $\chi^2$-test or Fisher’s exact test. Differences between continuous variables were analysed using
Echocardiographically confirmed pseudo-pulseless electrical activity

Prepare for FE and perform lung US
- Announce to the team during CPR
- Prepare and test US machine
- Prepare the patient (clothes, position)
- Using US exclude pneumothorax during CPR
- Have 20 IU vasopressin i.v. bolus injection ready
- When ready, announce “I will perform the US during next pulse check”

Electrocardiogram

VF/pVT/asystole

Continue CPR/ALS

PEA

Perform FE examination
- Tell the team to “count down from 10, while I check for pulse”
- Obtain subxiphoid view of heart, trying to see heart and valve motion

Cardiac wall and valve motion clearly discernible, no other specific cause found

Immediately give 20 IU vasopressin i.v. with flush

Not palpable

Continue CPR/ALS

Palpable

ROSC

Specific possible cause found (e.g. tamponade, PE, hypovolaemia)

Perform specific necessary action/therapy

No cardiac wall/valve motion visible

Continue CPR/ALS without delay

Wait 15 s (no chest compressions)

Pulse check

Prepare for FE and perform lung US as per AHA/ERC/ILCOR guidelines

FIGURE 1: Diagrammatic representation of a modified protocol for focused echocardiographic (FE) evaluation during resuscitation, following out-of-hospital cardiac arrest (BLS, basic life support; ALS, advanced life support; AHA, American Heart Association, ERC, European Resuscitation Council; ILCOR, International Liaison Committee on Resuscitation; US, ultrasound; CPR, cardiopulmonary resuscitation; VF, ventricular fibrillation; pVT, pulseless ventricular tachycardia; PEA, pulseless electrical activity; PE, pulmonary embolism; ROSC, restoration of spontaneous circulation)

the Student’s t-test, the Mann–Whitney test or the Wald test. A P-value < 0.05 was considered to be statistically significant. All data analyses were conducted using the SPSS® statistical package, version 15.0 (SPSS Inc., Chicago, IL, USA) for Windows®.
Echocardiographically confirmed pseudo-pulseless electrical activity

Results

During the study period (November 2007 – October 2008), 84 OHCA occurred; the initial rhythm was VF in 30 patients, ventricular tachycardia in two patients, asystole in 33 patients and PEA in 19 patients. In 16 patients (19%), pseudo-PEA with a stable $P_{et}CO_2$ was found during CPR and the modified protocol was administered.

In the historical group, 167 OHCA occurred; in 48 patients (29%) there was a stable $P_{et}CO_2$ in the absence of a pulse. The difference in the proportion of patients with a stable $P_{et}CO_2$ and PEA in the two groups was not statistically significant.

In the study series, five patients had an identifiable cause for the primary PEA: two patients had a massive pulmonary embolism and three patients had hypovolaemia. One patient with hypovolaemia and one with pulmonary embolism had pseudo-PEA during CPR and survived to ICU admission.

Overall, the modified management plan was well performed, despite different researchers leading the resuscitation team at any given time, with different paramedics manning the team. There were no cases where the presence of cardiac contractions could not be verified with FE (e.g. due to obesity, bright sunlight reflecting on the screen, technical problems).

Demographic and clinical characteristics for the study and historical groups are shown in Table 1. Both groups were adequately balanced with regard to gender, cardiac arrests happening within an urban area, cardiovascular aetiology, CPR performed by the BLS team, witnessed cardiac arrest, distribution of the initial cardiac rhythm, and mean time from call to CPR start. The study group was significantly younger than the historical group ($P = 0.044$).

Compared with the historical group, the study group received significantly fewer doses of adrenaline ($P = 0.009$) and significantly higher second doses of vasopressin ($P = 0.003$). In addition, the mean duration of the resuscitation effort was 12 min shorter in the study group.

The $P_{et}CO_2$ levels were significantly higher in the study group compared with the historical group at the moment of stable $P_{et}CO_2$ during the carotid pulse check (without compressions and with 20 IU vasopressin; Table 1). The difference in $P_{et}CO_2$ (i.e. the difference between the last value of PetCO2 before ROSC and the value at the moment of ROSC) at the pulse check was similar in both groups (2.8 mmHg; Table 1).

The initial $P_{et}CO_2$ (29 ± 19 mmHg versus $17 ± 9$ mmHg; $P = 0.022$, Table 1), the mean $P_{et}CO_2$ (36 ± 12 mmHg versus $19 ± 8$ mmHg; $P = 0.011$) and the final $P_{et}CO_2$ (39 ± 9 mmHg versus $22 ± 11$ mmHg; $P = 0.009$) were all significantly higher in the pseudo-PEA study group than in the historical group of patients. The mean additional time between pulse check and ROSC in the study group was 11.2 s (21.2 s together with pulse check).

Resuscitation outcomes are shown in Table 2. In the study series (16 cases), ROSC was achieved in 15 patients (94%), ICU admission in 14 patients (88%), 24-h survival in 13 patients (81%), survival to hospital discharge in nine patients (56%) and a good neurological outcome (CPC grade 1 – 2) in eight patients (50%). Compared with the historical group, the study group had significantly higher rates of ICU admission ($P = 0.009$), ROSC ($P = 0.006$), 24-h survival ($P = 0.002$) and survival to hospital discharge ($P = 0.001$). These four outcome differences remained statistically significant after adjustment for pre-treatment covariates ($P < 0.001$; Table 2).

At hospital discharge, statistically significantly better cerebral performance was
Echocardiographically confirmed pseudo-pulseless electrical activity

TABLE 1: Demographic and clinical characteristics of a study group with pseudo-pulseless electrical activity (pseudo-PEA) and an historical group with pulseless electrical activity (PEA) following out-of-hospital cardiac arrest

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Pseudo-PEA study group (n = 16)</th>
<th>PEA historical group (n = 48)</th>
<th>Statistical significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years), mean ± SD</td>
<td>59 ± 11</td>
<td>65 ± 13</td>
<td>P = 0.044^a</td>
</tr>
<tr>
<td>Gender, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>12 (75)</td>
<td>32 (67)</td>
<td>NS^b</td>
</tr>
<tr>
<td>Female</td>
<td>4 (25)</td>
<td>16 (33)</td>
<td>NS^b</td>
</tr>
<tr>
<td>Cardiac arrest in urban area, n (%)</td>
<td>13 (81)</td>
<td>37 (77)</td>
<td>NS^b</td>
</tr>
<tr>
<td>Witnessed arrest, n (%)</td>
<td>16 (100)</td>
<td>46 (96)</td>
<td>NS^b</td>
</tr>
<tr>
<td>Bystander CPR, n (%)</td>
<td>8 (50)</td>
<td>28 (58)</td>
<td>NS^b</td>
</tr>
<tr>
<td>Time from call to start of CPR (min), mean ± SD</td>
<td>5 ± 2</td>
<td>6 ± 3</td>
<td>NS^a</td>
</tr>
<tr>
<td>Cardiovascular aetiology of cardiac arrest, n (%)</td>
<td>14 (88)</td>
<td>42 (88)</td>
<td>NS^b</td>
</tr>
<tr>
<td>Initial cardiac rhythm, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VF/pVT</td>
<td>8 (50)</td>
<td>21 (44)</td>
<td>NS^b</td>
</tr>
<tr>
<td>PEA</td>
<td>2 (12)</td>
<td>4 (8)</td>
<td>NS^b</td>
</tr>
<tr>
<td>Asystole</td>
<td>6 (38)</td>
<td>23 (48)</td>
<td>NS^b</td>
</tr>
<tr>
<td>Duration of CPR (min), mean ± SD</td>
<td>19 ± 11</td>
<td>31 ± 13</td>
<td>P = 0.003^a</td>
</tr>
<tr>
<td>Drugs given, mean ± SD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adrenaline, mg</td>
<td>3.5 ± 2.6</td>
<td>6.0 ± 3.2</td>
<td>P = 0.009^a</td>
</tr>
<tr>
<td>Second vasopressin, IU</td>
<td>21.5 ± 12.8</td>
<td>6.7 ± 12.7</td>
<td>P = 0.003^a</td>
</tr>
<tr>
<td>(P_{et})CO(_2) (mmHg), mean ± SD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial</td>
<td>29 ± 19</td>
<td>17 ± 9</td>
<td>P = 0.022^a</td>
</tr>
<tr>
<td>1 min</td>
<td>38 ± 21</td>
<td>32 ± 19</td>
<td>NS^a</td>
</tr>
<tr>
<td>5 min</td>
<td>37 ± 11</td>
<td>35 ± 16</td>
<td>NS^a</td>
</tr>
<tr>
<td>10 min</td>
<td>38 ± 8</td>
<td>35 ± 15</td>
<td>NS^a</td>
</tr>
<tr>
<td>15 min</td>
<td>40 ± 12</td>
<td>34 ± 13</td>
<td>NS^a</td>
</tr>
<tr>
<td>20 min</td>
<td>31 ± 7</td>
<td>33 ± 12</td>
<td>NS^a</td>
</tr>
<tr>
<td>Stable with PEA</td>
<td>38 ± 14</td>
<td>30 ± 13</td>
<td>P = 0.037^a</td>
</tr>
<tr>
<td>Difference in (P_{et})CO(_2)^c</td>
<td>2.8 ± 1.0</td>
<td>2.8 ± 1.3</td>
<td>NS^a</td>
</tr>
</tbody>
</table>

^aUnpaired t-test or Mann–Whitney test.
^b\(\chi^2\)-test or Fisher’s exact test.
^cDifference between the last value of \(P_{et}\)CO\(_2\) before ROSC and the value at the moment of ROSC.

CPR, cardiopulmonary resuscitation; NS, not statistically significant (P > 0.05); \(P_{et}\)CO\(_2\), end-tidal carbon dioxide pressure; ROSC, restoration of spontaneous circulation; pVT, pulseless ventricular tachycardia; VF, ventricular fibrillation.

seen in survivors in the study group compared with the historical group (P < 0.001; Table 2).

Discussion
The present study demonstrated that FE in OHCA patients with PEA, with stable values of \(P_{et}\)CO\(_2\) during compression pauses, was able to identify patients with pseudo-PEA, i.e. a state of poor perfusion. Such patients may benefit from the administration of additional vasopressin: significantly improved ROSC, ICU admission, 24-h survival and survival to hospital discharge rates were seen when this was administered. This effect can be attributed to the recognition of pseudo-PEA and the use of a modified CPR protocol. These results highlight the importance of (i) using FE in
### TABLE 2:
Resuscitation outcomes in a study group with pseudo-pulseless electrical activity (pseudo-PEA) and an historical group with pulseless electrical activity (PEA) following out-of-hospital cardiac arrest

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Pseudo-PEA study group (n = 16)</th>
<th>PEA historical group (n = 48)</th>
<th>Statistical significance&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Odds ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (%)</td>
<td>n (%)</td>
<td>Unadjusted Adjusted&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Unadjusted Adjusted&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Primary ICU admission</td>
<td>14 (88)</td>
<td>24 (50)</td>
<td>( P = 0.009 ) ( P &lt; 0.001 )</td>
<td>8.6 (1.7 – 36.5) ( 22.4 (4.2 – 86.9) )</td>
</tr>
<tr>
<td>Secondary ROSC</td>
<td>15 (94)</td>
<td>26 (54)</td>
<td>( P = 0.006 ) ( P &lt; 0.001 )</td>
<td>11.3 (2.8 – 48.6) ( 28.4 (3.9 – 96.1) )</td>
</tr>
<tr>
<td>Survival at 24 h</td>
<td>13 (81)</td>
<td>20 (42)</td>
<td>( P = 0.009 ) ( P &lt; 0.001 )</td>
<td>7.7 (1.5 – 33.8) ( 19.8 (3.1 – 72.6) )</td>
</tr>
<tr>
<td>Survival to hospital discharge</td>
<td>9 (56)</td>
<td>7 (15)</td>
<td>( P = 0.002 ) ( P &lt; 0.001 )</td>
<td>17.5 (2.4 – 63.7) ( 31.4 (2.9 – 85.7) )</td>
</tr>
<tr>
<td>Good neurological outcome (CPC 1–2)</td>
<td>8 (50)</td>
<td>4 (8)</td>
<td>( P = 0.001 ) ( P &lt; 0.001 )</td>
<td>29.8 (2.8 – 82.8) ( 36.4 (4.8 – 115.4) )</td>
</tr>
</tbody>
</table>

<sup>a</sup>Analysed using the Wald test.

<sup>b</sup>Adjusted by covariates with known predictive value (age, male sex, witnessed arrest, time from call to start of cardiopulmonary resuscitation [CPR], bystander CPR, initial rhythm and initial end-tidal partial pressure of carbon dioxide).

CI, confidence interval; CPC, Cerebral Performance Category; ICU, intensive care unit; ROSC, return of spontaneous circulation.
OHCA patients (during pulse checks) in combination with capnography in the field, (ii) recognizing pseudo-PEA in patients with stable $P_{et}CO_2$ during CPR, and (iii) the role of vasopressin in the treatment of pseudo-PEA.

The carotid pulse check during CPR has been abolished by ILCOR for lay rescuers, but not for healthcare providers. Lapostolle et al.\textsuperscript{1} reported correct pulse detection in about 50% of pulseless situations and about 80% of situations with a weak pulse. In an analysis of 119 professionals during simulated scenarios, Albarran et al.\textsuperscript{2} reported correct pulse diagnoses in 48.5%. Similarly, Moule\textsuperscript{24} found that healthcare students made the correct pulse diagnosis in 38% of cases.

The results of the present study suggest the value of combining the carotid pulse check with FE examination during CPR by healthcare providers. Ultrasound examination is not an alternative to the pulse check, but provides an additional way to detect cardiac activity during the time available. In a study of 693 pulse checks in 226 patients, Blaivas\textsuperscript{10} confirmed discordance between pulse detection and emergent echocardiography findings in adult cardiopulmonary arrest patients. Salen et al.\textsuperscript{8,9} demonstrated the usefulness of combining capnography and sonography for evaluating and predicting outcome in cardiac arrest patients; these authors confirmed that the presence of sonographically identifiable cardiac kinetic motion was associated with ROSC and hospital admission, regardless of initial cardiac rhythm.\textsuperscript{8,9} Logistic regression analysis showed that prediction of survival using capnography was not enhanced by the addition of cardiac sonography.\textsuperscript{8,9}

The prognostic value of capnography has been demonstrated previously.\textsuperscript{20–22} End-tidal carbon dioxide levels should be monitored during CPR; they are considered to have useful prognostic value for determining the outcome of resuscitative efforts and indicating when to cease CPR in the field.

Blaivas and Fox\textsuperscript{15} reported that no patient with sonographically identified cardiac standstill survived to leave the emergency department, regardless of the initial electrical rhythm: cardiac standstill on echocardiogram resulted in a positive predictive value of 100% for death in the emergency department, with a negative predictive value of 58%. FE in OHCA improved diagnostic accuracy and treatment, and had prognostic value: Breitkreutz et al.\textsuperscript{4} and Niendorff et al.\textsuperscript{12} confirmed the usefulness of FE in patients with PEA arrest and integrating FE in the ALS response.\textsuperscript{25}

Cardiac arrest presenting as PEA has a very low survival rate. Many conditions underlying PEA, including cardiac tamponade, hypovolaemia and pulmonary embolism, are associated with specific findings.\textsuperscript{4} Niemann et al.\textsuperscript{18} compared the outcome of out-of-hospital post-shock PEA with primary PEA. ROSC and hospital admission were significantly more frequent and survival was greater in primary PEA, which may be related to myocardial electrical injury and/or increased aortic blood potassium concentration (which is 50% higher).\textsuperscript{17,19} The poor prognosis of secondary PEA underlines the importance of immediately identifying pseudo-PEA, which may be associated with a better outcome. Spreng et al.\textsuperscript{26} described an experimental study using a Doppler probe to make this differentiation following cardiac arrest. In 1994, White and Asplin\textsuperscript{27} reported on the use of $P_{et}CO_2$ observations for the differentiation of pseudo-electromechanical dissociation and electromechanical dissociation with stable carbon dioxide levels during pulselessness. Stable persistent $P_{et}CO_2$ values in combination with FE with evident cardiac kinetic activity (synchronous myocardial wall and valvular motion) were used for pseudo-PEA.
confirmation in the present study.

In the present study, patients with pseudo-PEA and minimal cardiac output were supported with an additional dose of 20 IU vasopressin (i.v. bolus). The favourable haemodynamic effects of vasopressin were associated with a 12 min shorter duration of CPR and fewer doses of adrenaline compared with the historical group, although two patients received additional doses of adrenaline after the second vasopressin dose without survival, in the field.

The experimental study of Wenzel et al. demonstrated that vasopressin, in comparison with adrenaline, significantly increased left ventricular myocardial and total cerebral blood flow during CPR. The beneficial effects of additional vasopressin have also been observed in other catecholamine-refractory shock states. The Innsbruck vasopressor strategy recommended, during CPR, alternating between bolus injections of 1 mg adrenaline i.v. initially, and 40 IU vasopressin i.v. subsequently, every 3 – 5 min if ROSC does not occur, regardless of the initial rhythm.

A study by Friesenecker et al. showed that, under normal physiological conditions, vasopressin exerted a substantially stronger vasoconstrictive action on large arterioles than noradrenaline. This observation could explain, in part, why vasopressin can be effective in advanced shock that is unresponsive to increased doses of catecholamines in standard shock therapy.

The concept behind the modified protocol we describe here was that of haemodynamic stabilization with peripheral vasoconstriction in patients with echocardiographically confirmed pseudo-PEA. Attaining ROSC in 94% and a good neurological outcome in half of the patients, despite a prolonged compression pause, adds weight to this hypothesis. The results of the present study suggest the value of echocardiographic assessment of OHCA patients with PEA to identify those with poor perfusion and to allow the use of a modified protocol. Further studies are needed to evaluate this approach.

In conclusion, this pilot study of 16 cases of echocardiographically identified pseudo-PEA with modified management showed encouragingly higher rates of ROSC and survival to discharge, with good neurological outcome, compared with an historical PEA group with a stable $P_{etCO_2}$ (but where a distinction between PEA and pseudo-PEA was not made echocardiographically). Echocardiographically confirmed pseudo-PEA in OHCA patients with constant values of $P_{etCO_2}$ during compression pauses may, therefore, indicate modified treatment.

**Conflicts of interest**

The authors had no conflicts of interest to declare in relation to this article.

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