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Aetiologies of pulseless electrical activity in out-of-hospital cardiac arrests:

A retrospective study and analysis of specific causes

Etudiant Ludovic Beun, UNIL

Tuteur

Prof. Bertrand Yersin, Chef de service Service des Urgences, CHUV

Co-tuteur

Dr. Pierre-Nicolas Carron, Médecin adjoint Service des Urgences, CHUV

Expert

Prof. Joseph Osterwalder, Chefarzt Notfallstation, KSSG

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1. Abstract

Background: Pulseless electrical activity (PEA) cardiac arrest is defined as a cardiac arrest (CA) presenting with a residual organized electrical activity on the electrocardiogram. In the last decades, the incidence of PEA has regularly increased, compared to other types of CA like ventricular fibrillation or pulseless ventricular tachycardia. PEA is frequently induced by reversible conditions. The "4 (or 5) H" & "4 (or 5) T" are proposed as a mnemonic to asses for Hypoxia, Hypovolemia, Hypo-/Hyperkalaemia, Hypothermia, Thrombosis (cardiac or pulmonary), cardiac Tamponade, Toxins, and Tension pneumothorax. Other pathologies (intracranial haemorrhage, severe sepsis, myocardial contraction dysfunction) have been identified as potential causes for PEA, but their respective probability and frequencies are unclear and they are not yet included into the resuscitation guidelines. The aim of this study was to analyse the aetiologies of PEA out-of-hospital CA, in order to evaluate the relative frequencies of each cause and therefore to improve the management of patients suffering a PEA cardiac arrest.

Method: This retrospective study was based on data routinely and prospectively collected for each PEMS intervention. All adult patients treated from January 1st 2002 to December 2012 31st by the PEMS for out-of-hospital cardiac arrest, with PEA as the first recorded rhythm, and admitted to the emergency department (ED) of the Lausanne University Hospital were included. The aetiologies of PEA cardiac arrest were classified into subgroups, based on the classical H&T's classification, supplemented by four other subgroups analysis: trauma, intra-cranial haemorrhage (ICH), non-ischemic cardiomyopathy (NIC) and undetermined cause.

Results: 1866 OHCA were treated by the PEMS. PEA was the first recorded rhythm in 240 adult patients (13.8 %). After exclusion of 96 patients, 144 patients with a PEA cardiac arrest admitted to the ED were included in the analysis. The mean age was 63.8 \pm 20.0 years, 58.3% were men and the survival rate at 48 hours was 29%. 32 different causes of OHCA PEA were established for 119 patients. For 25 patients (17.4 %), we were unable to attribute a specific cause for the PEA cardiac arrest. Hypoxia (23.6 %), acute coronary syndrome (12.5%) and trauma (12.5 %) were the three most frequent causes. Pulmonary embolism, Hypovolemia, Intoxication and Hyperkaliemia occurs in less than 10% of the cases (7.6 %, 5.6 %, 3.5%, respectively 2.1 %). Non ischemic cardiomyopathy and intra-cranial haemorrhage occur in 8.3 % and 6.9 %, respectively.

Conclusions: According to our results, intra-cranial haemorrhage and non-ischemic cardiomyopathy represent noticeable causes of PEA in OHCA, with a prevalence equalling or exceeding the frequency of classical 4 H's and 4 T's aetiologies. These two pathologies are potentially accessible to simple diagnostic procedures (native CT-scan or echocardiography) and should be included into the 4 H's and 4 T's mnemonic.

Keywords: Out-of-hospital cardiac arrest; pulseless electrical activity; cardiopulmonary resuscitation; cause of death; change aetiologies.

2. Introduction

Pulseless electrical activity (PEA) cardiac arrest is defined as a cardiac arrest presenting with a residual organized electrical activity on the electrocardiogram (other than ventricular fibrillation - VF or pulseless ventricular tachycardia - VT) that would normally be associated with a palpable pulse. The absence of mechanical contractions and palpable pulse are due to either the absence of synchronous myocyte depolarisation, vascular failure, or alterations of myocyte contractil function.¹⁻³ Some patients present with residual mechanical myocardial contractions, but these are two weak to produce a detectable pulse or blood flow.⁴

In the last decades, the incidence of PEA among cardiac arrest has regularly increased compared to other types like VT and VF. This evolution could be explained by the constant increase of age and comorbidities in patients succumbing to cardiac arrest. The incidence of PEA in out-of-hospital cardiac arrests (OHCA) is now reported between 19% and 29%.⁵⁻⁸

Patients suffering out-of-hospital cardiac arrests (OHCA) presenting initially with PEA activity have a poor prognosis, with a survival rate at admission estimated about 26% and a survival rate to the hospital discharge estimated between 2% and 5%.^{7,9}

According to the European Resuscitation Council Guidelines and the American Heart Association recommendations, PEA is described as a non-shockable rhythm and his out-of-hospital management is mainly based on cardio-pulmonary resuscitation (CPR) – combining external chest compressions and assisted ventilation – and on early epinephrine administration.

These measures are intended to regain a palpable pulse and/or convert PEA activity into a "shockable rhythm", in order to administer a defibrillation shock and to improve the probability of a return of spontaneous circulation (ROSC). Simultaneously to the CPR measures, the identification of potential favouring causes is warranted.^{4,10}

PEA is frequently induced by reversible conditions and can be potentially reversed if those conditions are identified and corrected. The "4 (or 5) H" & "4 (or 5) T" are proposed as a mnemonic to asses for Hypoxia, Hypovolemia, Hypo-/Hyperkalaemia, Hypothermia, Thrombosis (cardiac or pulmonary), cardiac Tamponade, Toxins, and Tension pneumothorax. These causes are potentially reversible during the out-of-hospital period or shortly after the admission in the emergency department, thereby promoting the use of the concept of "reversible causes" for the 4 H & 4T. ^{4,10}

Nevertheless, these causes are not comprehensive and other pathologies have been identified as potential causes for PEA cardiac arrest. Potential aetiologies include intracranial haemorrhage, severe sepsis, myocardial contraction dysfunction, primary cardiac arrhythmia or other cardiac pathologies (valvular disease, graft rejection, congenital disease, and pacemaker dysfunction).¹¹⁻¹³

The respective probability and frequencies of these aetiologies are unclear. Until now, they are not included into the resuscitation guidelines, with the risk that they will not be suspected in OHCA, and that a significant number of critical, but potentially reversible conditions could be missed.

2.1 Objectives

The rising incidence of PEA cardiac arrests, the poor prognosis of this condition and the limited therapeutic strategies, emphasize the need to study the potential causes of PEA cardiac arrests, including aetiologies not included in the mnemonic H&T's. The aim of this study was therefore to analyse the aetiologies of PEA out-of-hospital cardiac arrests, in order to evaluate the relative frequencies of each cause. The results may help to suggest modifications of the mnemonic H&T's causes in resuscitation guidelines and improve the management of patients suffering a PEA cardiac arrest.

3. Materials and method

3.1 Setting and Design

This retrospective study takes place at the Lausanne University Hospital (CHUV). The CHUV is a 1000-bed university hospital located in western Switzerland. It is the primary hospital for the immediate catchment area comprised of about 300'000 persons, and serves also as the referral hospital and level 1 trauma centre for a population of over one million. The prehospital emergency medical service (PEMS) includes a unique emergency dispatch centre (EDC), staffed by trained nurses or paramedics, using a specific keyword-based dispatch protocol, including dispatcher-assisted cardiopulmonary resuscitation. Ambulances comprise trained paramedics or emergency medical technicians (EMT) and constitute the initial response of the PEMS. Prehospital emergency physicians may be dispatch on scene in the case of cardiac arrest, major trauma, respiratory distress, coma, or other life-threatening emergencies; or secondary at the request of the ambulance's paramedics on site.

Out-of-hospital pulseless electrical activity (PEA) cardiac arrest is defined as a cardiac arrest presenting residual electrical activity on the electrocardiogram other than VF or pulseless VT. The advanced cardiac life support algorithm used by the prehospital emergency medical services is in accordance with the European Resuscitation Council (ERC) Guidelines and includes cardiopulmonary resuscitation, intravenous or intraosseous access, epinephrine and amiodarone administration, defibrillation in case of a shockable rhythm, endotracheal intubation with end-tidal CO2 (EtCO2) monitoring According to the previous guidelines, atropine has been used systematically until the end of 2011.^{4,10}

No mechanical chest compression device was available during the study. In case of ROSC or successful resuscitation, the patient is transported to the hospital. PEA without ROSC could be transported to the hospital, particularly if pulmonary embolism, hypothermia, drug intoxication or cardiac tamponade are suspected. The decision is left to the discretion of the on-scene emergency physicians or paramedics.

3.2 Data collection

This study was based on data routinely and prospectively collected for each PEMS intervention. After each intervention, the paramedics and emergency physicians fill out a specific report. Ambulance reports comprise EDC data, evaluation of the patient on site (vitals, severity, life-saving measures), and actions undertaken (e.g. hospital transport, call of the SMUR, person let on site, or death on site). Emergency physician reports contain EDC data, clinical conditions, life-saving measures, treatments and procedures on site, transport indications and immediate outcome of the patient at time of hospital

admission. The list of diagnose, specific interventions (surgical intervention, thoracostomies, percutaneous coronary angioplasty) and immediate outcome are prospectively collected after 48 hours. In case of OHCA, the recorded data are in agreement with the Utstein recommendations for uniform reporting of cardiac arrest.¹⁴

3.3 Classification of PEA aetiologies

The description of the PEA aetiologies was retrospective and based on the patient's record, including the prehospital report, the hospital therapeutic and diagnostic data, the final diagnosis and the 48 hours survival rate.

The aetiologies of PEA cardiac arrest were classified into subgroups, based on the classical H&T's classification, supplemented by four other subgroups analysis: trauma, intracranial haemorrhage (ICH), non-ischemic cardiomyopathy (NIC) and undetermined cause. Usually acute coronary syndrome and pulmonary embolism are classified into the same group as T for "thrombo-embolic pathologies" but we decided to separate them in two distinct subgroups. In a second time, aetiologies were classified in 4 groups: H (H's pathologies), T (T's pathologies), O (other pathologies) and U (undetermined pathologies). (Table 2)

3.4 Population

All adult patients treated from January 1st 2002 to December 2012 31st by the PEMS for out-of-hospital cardiac arrest, with PEA as the first recorded rhythm, and admitted to the emergency department (ED) of the Lausanne University Hospital were included.

Patients were excluded either 1) if they presented with cardiac arrests initially due to pulseless TV, FV, or asystole as first recorded rhythm, 2) were declared dead on scene or during the transport, 3) suffered from an in-hospital cardiac arrest, 4) were aged less than 16, or 5) if the medical record was missing or double.

3.5 Statistical analysis

All individual data were de-identified and entered into an anonymous computerized database (Microsoft Excel Microsoft Corp., Redmond, WA). Categorical data are presented as counts and percentage frequencies. Continuous variables are shown as means ± standard deviations. Statistical analysis was performed using Stata Statistical Software Release 12.0 (Stata Corporation, College Station, TX).

3.6 Ethical aspect

For the purpose of the study, the data were collected into an anonymous database. The study protocol was agreed by the Ethical Committee of the University, as well as by the Health Care authority of the state.

4. Results

During the study period, there were 1866 OHCA treated by the PEMS. PEA was the first recorded rhythm in 240 adult patients (13.8 %). We excluded 96 patients: 76 were declared dead on scene, 8 were duplicates, 4 were less than 16 years old, 3 died during transport, 3 were in-hospital cardiac arrest and for 2 patients the medical records were missing. Finally, 144 patients with a PEA cardiac arrest admitted to the ED were included in the analysis (Annexe 1).

The mean age was 63.8 ± 20.0 years, 58.3% were men and the survival rate at 48 hours was 29%. Patients with trauma-related conditions were younger, benefited more frequently from dispatcher-assisted CPR, were less frequently administered adrenalin or defibrillation, and presented a worse survival rate at 48 hours. (Table 1)

We found 32 different causes of OHCA PEA for 119 patients that we have classified into 4 groups (9 subgroups). For 25 patients (17.4 %), we were unable to attribute a specific cause for the PEA cardiac arrest and the patients were thus classified as undetermined causes. (Table 2)

Hypoxia (23.6 %), acute coronary syndrome (12.5%) and trauma (12.5 %) were the three most frequent causes. Pulmonary embolism, Hypovolemia, Intoxication and Hyperkaliemia occurs in less than 10% of the cases (7.6 %, 5.6 %, 3.5%, respectively 2.1 %). Non ischaemic cardiomyopathy and intra-cranial haemorrhage occur in 8.3 % and 6.9 %, respectively. We had no case of isolated tension pneumothorax, cardiac tamponade or hypothermia in our population as the main cause of PEA cardiac arrest. (Table 3)

5. Discussion

According to these results, for PEA OHCA patients admitted to the ED, the mnemonic classical H's and T's represents only 54.9% of the aetiologies. Other aetiologies were suspected in 27.8 % of the cases (n = 40), and mainly involved trauma (12.5 %), non-ischemic cardiopathy (8.3 %), as well as intra-cranial haemorrhage (6.9 %). The results of our study confirm also a limited short-term survival rate in PEA OHCA.

The main result of this study is the important disparity of PEA causes as compared to the mnemonic classical H's and T's and the distribution of our cases reveal a high prevalence of specific aetiologies which are not specifically integrated into the classical H's and T's.

Hypoxia and cardiac pathologies represent the main cause of PEA OHCA in our study. These results are in accordance with other studies which show usually that hypoxia and cardiac ischemic pathologies are the main causes of PEA OHCA.^{6,7,15,16}

However, in our study, cardiac pathologies are represented by acute coronary syndrome (ACS) and non-ischemic cardiac diseases (NIC). NIC includes several different cardiomyopathies which could promote the occurrence of major cardiac dysfunctions and PEA cardiac arrest. These pathologies are heterogeneous by nature and their management require a specific diagnosis, making a diagnosis and therapeutic procedure difficult on site. However it is important to highlight the possibility of non-ischemic cardiac causes in the differential diagnosis of PEA OHCA. In the ED, a bedside echocardiography may be of major interest in these cases.^{17,18}

Except regarding hypoxia and cardiac causes, other classical aetiologies occur infrequently, with rates globally less than 10% for each H and T. Even pulmonary embolism, which is often described as the most frequent and iconic cause of PEA, occurs only in 7.6% of the cases.^{19,20} Similarly, cardiac tamponade, pneumothorax and hypothermia were not found in our study.

Traumatic pathologies deserve a particular commentary. Trauma is not a specific cause of PEA as it may be present in polytrauma patients who suffer from several injuries at the same time (severe bone fractures, hemo/pneumothorax, cardiac tamponade, flail chest, brain injury, medullar lesion, penetrating trauma, haemorrhage or visceral injuries). It is therefore frequently not possible to identify a unique traumatic cause of cardiac arrest and this category is frequently excluded from analysis about cardiac arrest. Trauma patients may therefore be assessed for hypovolemia, tension pneumothorax or cardiac tamponade.²¹ In previous publications, the prognosis of these patients appears not different from non traumatic cardiac arrests, probably due to the younger age of the patients and to limited risk of severe comorbidities.^{22,23}

Finally, we found a high prevalence of intra-cranial hemorrhage (ICH) in our study (6.9%). ICH is a recognized potential cause of cardiac arrest and PEA, with a postulated mechanism involving catecholamine's increase.^{11,24} ICH is a potential reversible cause and could be easily diagnosis with a native CT-scan in the ED.^{25,26}

This aetiology, as well as other factors like bacteraemia, sepsis or vascular dissections has been evoked in the literature as a potential cause of PEA OHCA, which should be assessed in the ED.^{12,15}

Several papers have tried to improve the management of PEA by introducing new diagnostic and therapeutic strategies. The PEA management guidelines are largely based on the 4 H's and 4 T's described the first time by Kloeck et al. in 1995.²⁷⁻²⁹ Since this publication, no paper has been published to prospectively evaluate the aetiologies of PEA OHCA and the related prevalence. The evidence supporting the theoretical 4 H's and 4 T's appears therefore to be limited.

5.1 Limitations

The main limitation of the study is the limited availability of information for patients declared death before arrival to the ED. We included only patients suffering from PEA OHCA and admitted alive into the ED.

These cases admitted in the ED may therefore represent a particular sample of the PEA population and the exclusion of patients who died on scene or during the transport, or those suffering from an intra-hospital cardiac arrest, could have create a selection bias and change the distribution of PEA causes.

The retrospective method warrants exhaustive evaluation of the cases, limiting the information in some cases and reducing the diagnosis accuracy. A previous study demonstrated that there might be several differences of diagnosis between clinical diagnosis and autopsy findings, especially in case of vascular ruptures or pulmonary embolism.¹⁵

In the ED, older patients admitted for OHCA and presenting with severe pathologies or comorbidities are frequently not thoroughly investigated during the first hours. For this reason, some specific pathologies may have been underdiagnosed before the patient's death.

They support the realisation of prospective studies based on systematic post-mortem analyses in order to re-evaluate the 4 H's and 4 T's.

6. Conclusion

According to the results of this study, ICH (intra-cranial hemorrhage) and NIC (nonischemic cardiac diseases) represent noticeable causes of PEA in OHCA, with a prevalence equalling or exceeding the frequency of classical 4 H's and 4 T's aetiologies. These two pathologies are potentially accessible to simple diagnostic procedures (native CT-scan or echocardiography) and should be included into the 4 H's and 4 T's mnemonic.

7. Conflict of interest statement

None to declare

8. Source of funding

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9. References

1. Ewy GA. Defining electromechanical dissociation. Ann Emerg Med. 1984 Sep;13(9 Pt 2):830-2.

2. Myerburg RJ, Halperin H, Egan DA, Boineau R, Chugh SS, Gillis AM, et al. Pulseless electric activity: definition, causes, mechanisms, management, and research priorities for the next decade: report from a National Heart, Lung, and Blood Institute workshop. Circulation. 2013 Dec 3;128(23):2532–41.

3. Mehta C, Brady W. Pulseless electrical activity in cardiac arrest: electrocardiographic presentations and management considerations based on the electrocardiogram. Am J Emerg Med. 2012 Jan;30(1):236–9.

4. Deakin CD, Nolan JP, Soar J, Sunde K, Koster RW, Smith GB, et al. European Resuscitation Council Guidelines for Resuscitation 2010 Section 4. Adult advanced life support. Resuscitation [Internet]. 2010 Oct [cited 2012 Dec 16];81(10):1305–52.

5. Parish DC, Dinesh Chandra KM, Dane FC. Success changes the problem: why ventricular fibrillation is declining, why pulseless electrical activity is emerging, and what to do about it. Resuscitation. 2003 Jul;58(1):31–5.

6. Engdahl J, Bång A, Lindqvist J, Herlitz J. Factors affecting short- and long-term prognosis among 1069 patients with out-of-hospital cardiac arrest and pulseless electrical activity. Resuscitation. 2001 Oct;51(1):17–25.

7. Väyrynen T, Kuisma M, Määttä T, Boyd J. Who survives from out-of-hospital pulseless electrical activity? Resuscitation. 2008 Feb;76(2):207–13.

8. Mader TJ, Nathanson BH, Millay S, Coute RA, Clapp M, McNally B. Out-of-hospital cardiac arrest outcomes stratified by rhythm analysis. Resuscitation. 2012 Nov;83(11):1358–62.

9. Kajino K, Iwami T, Daya M, Nishiuchi T, Hayashi Y, Ikeuchi H, et al. Subsequent ventricular fibrillation and survival in out-of-hospital cardiac arrests presenting with PEA or asystole. Resuscitation. 2008 Oct;79(1):34–40

10. Neumar RW, Otto CW, Link MS, Kronick SL, Shuster M, Callaway CW, et al. Part 8: adult advanced cardiovascular life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2010 Nov 2;122(18 Suppl 3):S729–767.

11. Mitsuma W, Ito M, Kodama M, Takano H, Tomita M, Saito N, et al. Clinical and cardiac features of patients with subarachnoid haemorrhage presenting with out-of-hospital cardiac arrest. Resuscitation. 2011 Oct;82(10):1294–7.

12. Coba V, Jaehne AK, Suarez A, Dagher GA, Brown SC, Yang JJ, et al. The incidence and significance of bacteremia in out of hospital cardiac arrest. Resuscitation. 2014 Feb;85(2):196–202.

13. Florance R, Tong N, Giubileo A, Lloyd C. Suggested change to Resuscitation Council guidelines on reversible causes of cardiac arrest: acute subarachnoid haemorrhage, and malignant tachyarrhythmia. Resuscitation. 2013 Jan;84(1):e17.

14. Recommended guidelines for uniform reporting of data from out-of-hospital cardiac arrest (new abridged version). The "Utstein style". The European Resuscitation Council, American Heart Association, Heart and Stroke Foundation of Canada, and Australian Resuscitation Council. Br Heart J. 1992 Apr;67(4):325–33.

15. Virkkunen I, Paasio L, Ryynänen S, Vuori A, Sajantila A, Yli-Hankala A, et al. Pulseless electrical activity and unsuccessful out-of-hospital resuscitation: what is the cause of death? Resuscitation. 2008 May;77(2):207–10.

16. Saarinen S, Nurmi J, Toivio T, Fredman D, Virkkunen I, Castrén M. Does appropriate treatment of the primary underlying cause of PEA during resuscitation improve patients' survival? Resuscitation. 2012 Jul;83(7):819–22.

17. Breitkreutz R, Walcher F, Seeger FH. Focused echocardiographic evaluation in resuscitation management: concept of an advanced life support-conformed algorithm. Crit Care Med. 2007 May;35(5 Suppl):S150–161.

18. Chardoli M, Heidari F, Rabiee H, Sharif-Alhoseini M, Shokoohi H, Rahimi-Movaghar V. Echocardiography integrated ACLS protocol versus conventional cardiopulmonary resuscitation in patients with pulseless electrical activity cardiac arrest. Chin J Traumatol. 2012;15(5):284–7.

19. Comess KA, DeRook FA, Russell ML, Tognazzi-Evans TA, Beach KW. The incidence of pulmonary embolism in unexplained sudden cardiac arrest with pulseless electrical activity. Am J Med. 2000 Oct 1;109(5):351–6.

20. Courtney DM, Sasser HC, Pincus CL, Kline JA. Pulseless electrical activity with witnessed arrest as a predictor of sudden death from massive pulmonary embolism in outpatients. Resuscitation. 2001 Jun;49(3):265–72.

21. Lockey, David J. et al. Development of a simple algorithm to guide the effective management of traumatic cardiac arrest. Resuscitation. 2012 Dec;84(6):738-742.

22. Hopson LR, Hirsh E, Delgado J, Domeier RM, McSwain NE, Krohmer J, et al. Guidelines for withholding or termination of resuscitation in prehospital traumatic cardiopulmonary arrest: joint position statement of the National Association of EMS Physicians and the American College of Surgeons Committee on Trauma. J Am Coll Surg. 2003 Jan;196(1):106–12.

23. Lockey D, Crewdson K, Davies G. Traumatic cardiac arrest: who are the survivors? Ann Emerg Med. 2006 Sep;48(3):240–4.

24. Lewandowski P. Subarachnoid haemorrhage imitating acute coronary syndrome as a cause of out-of-hospital cardiac arrest - case report. Anaesthesiol Intensive Ther. 2014 Sep–Oct;46(4):289–92

25. McCarron MO, O'Kane MJ. Accurate diagnosis of subarachnoid haemorrhage. Ann Clin Biochem. 2014 Nov;51(Pt 6):629–30.

26. Kidwell CS, Chalela JA, Saver JL, Starkman S, Hill MD, Demchuk AM, et al. Comparison of MRI and CT for detection of acute intracerebral hemorrhage. JAMA. 2004 Oct 20;292(15):1823–30.

27. Kloeck WG. A practical approach to the aetiology of pulseless electrical activity. A simple 10-step training mnemonic. Resuscitation. 1995 Oct;30(2):157–9.

28. Littmann L, Bustin DJ, Haley MW. A simplified and structured teaching tool for the evaluation and management of pulseless electrical activity. Med Princ Pract. 2014;23(1):1–6.

29. Desbiens NA. Simplifying the diagnosis and management of pulseless electrical activity in adults: a qualitative review. Crit Care Med. 2008 Feb;36(2):391–6.

10. Tables

	All (n=144)	Non-traumatic (n=126)	Traumatic (n=18)	Р
Age (years) mean (± S.D.)	63.8 (20.0)	65 (19.6)	52.9 (20.3)	P < 0.05
Male (gender) n (%)	84 (58.3)	73 (57.9)	11 (61.1)	NS
Witnessed n (%)	118 (83)	103 (82)	15 (88)	NS
B-CPR n (%)	60 (45)	47 (41)	13 (76)	P < 0.05
Adrénaline n (%)	121 (84)	109 (87.0)	12 (67.0)	P < 0.05
Defibrillated n (%)	45 (31)	42 (33.0)	3 (17.0)	P < 0.05
Survival rate at 48h n (%)	42 (29)	38 (30)	4 (22)	P < 0.05

Table 1: Characteristics of PEA OHCA population, trauma vs non-trauma conditions

Table 2: Description of the categories

Groups	Subgroups	Related aetiologies	
Н	Hypoxemia	Acute pulmonary edema	
		Hanging	
		Drowning	
		Pneumonia	
		Haemoptysis	
		Upper airways obstruction	
		Respiratory failure	
		Pulmonary aspiration	
	Hypovolemia	Traumatic hemorrhage	
		Ruptured aortic aneurysm	
		Upper gastrointestinal bleeding	
	Hyperkaliema	Hyper/hypokaliema	
Т	Toxic	Carbon monoxide	
		Methadone	
		Benzodiazepine	
		Opioid	
	Acute coronary syndrome	STEMI	
		NSTEMI	
	Pulmonary embolism	Massive pulmonary embolism	
		Non-massive pulmonary embolism	
0	Trauma	Motor vehicle accident	
		Fall	
		Traumatic brain injury	
	Intra-cranial hemorrhage	Intra-axial hemorrhage	
		Extra-axial hemorrhage	
	Non ischemic cardiomyopathy	Long QT	
		AV block	
		Pacemaker failure	
		Vascular disease	
		Graft rejection	
		Congenital heart disease	
U	Undetermined	Missing data	
		Diagnostic uncertainty	

Table 3: Aetiologies of the PEA OHCA

Aetiologies	n	%
Нурохіа	34	23.6
Undetermined	25	17.4
Acute coronary syndrome	18	12.5
Trauma conditions	18	12.5
Non-ischemic cardiopathy	12	8.3
Pulmonary embolism	11	7.6
Intra-cranial hemorrhage	10	6.9
Hypovolemia	8	5.6
Intoxication	5	3.5
Hyperkalemia	3	2.1
Total	144	100.0
Classical 4 H's	45	31.3
Other	40	27.8
Classical 4 T's	34	23.6
Undetermined	25	17.4
Total	144	100.0

11. Annexe

Annexe 1: Flowchart

