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# Pseudo-pulseless electrical activity in the emergency department, an evidence based approach

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## ABSTRACT

**Introduction:** A great deal of the literature has focused specifically on true pulseless electrical activity (PEA), whereas there is a dearth of research regarding pseudo-PEA. This narrative review evaluates the diagnosis and management of patients in pseudo-PEA and discusses the impact on emerging patient outcomes.

**Discussion:** Pseudo-PEA can be defined as evidence of cardiac activity without a detectable pulse. Distinguishing pseudo-PEA from true PEA is important for emergency physicians as the prognosis and management of these patients differ. POCUS is the tool most commonly used to diagnose pseudo-PEA and there are varying treatment strategies to manage these patients. Identifying patients in pseudo-PEA can help guide resuscitation decisions, and ultimately impact emergency response systems, patients, and families.

**Conclusions:** The incidence of pseudo-PEA is increasing. Effective care of these patients begins with early diagnosis of this condition and immediate treatment to warrant the greatest chance of survival. There is a need for further prospective studies surrounding pseudo-PEA as evidenced by the lack of research in the current literature.

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## 1. Introduction

Confusion exists in the literature and clinical practice regarding “near-miss” events such as near syncope or near pulseless electrical activity (PEA). Near PEA, PEA-like status, and pseudo-PEA all refer to an electromechanical dissociation (EMD) that causes electrical activity of the heart, but the myocardium is not functional enough

to produce cardiac output to generate a pulse [1,2]. An observational prospective study by Tayal and Kline focused specifically on the differences between true PEA and pseudo-PEA. In their examination, pseudo-PEA referred to patients with absent pulse, but with evidence of cardiac activity through the utilization of bedside echocardiography [3]. Thus, point-of-care echocardiography has enabled us to draw a contrast between pseudo-PEA and true PEA, which is defined as the absence of cardiac activity on ultrasound [3,4]. Although this definition of pseudo-PEA is vague, this is the first instance that emergency physicians reported this condition [3].

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With pseudo-PEA, a significant pathophysiologic event has impaired the cardiovascular system's ability to perfuse. It can be considered as a state of cardiogenic shock, which does not adequately maintain perfusion pressure, ultimately resulting in a non-detectable pulse [4]. Although myocardial contractions are present, there is insufficient circulation throughout the body due to decreased myocardial function [5]. It has also been seen that cases of pseudo-PEA are frequently caused by profound hypovolemia as a result of hemorrhage and obstruction to forward flow secondary to pulmonary embolism, tension pneumothorax, or cardiac tamponade. Pseudo-PEA also involves tachydysrhythmias or hypocontractile states with poor vascular tone such as advanced anaphylactic or septic shock [6]. Furthermore, myocardial infarctions and asphyxia can play a role in the pathogenesis of pseudo-PEA. In the majority of these situations, tachycardia would generally be present, predominantly sinus tachycardia or atrial fibrillation with a rapid ventricular response [7]. It is imperative to treat pseudo-PEA immediately as pseudo-PEA can progress to true PEA, where electrolyte and metabolic disturbances will result in the termination of mechanical activity [4].

When reviewing the literature, it is apparent that pseudo-PEA is not clearly defined and there are no widely accepted guidelines for physicians to follow when diagnosing and treating patients with this condition. This topic has a profound impact on the healthcare community, as PEA is a frequent finding during cardiac arrest resuscitation [8]. Within the last two decades, it has been seen that the incidence of PEA has increased to 19–23% [7]. However, many of these cases may be pseudo-PEA rather than true PEA, which would alter treatment strategies [9]. Consequently, there is a need for more research to be done on pseudo-PEA in order for physicians to identify and manage patients with this condition more successfully. In this article, we conducted a review of the literature focusing on the diagnosis and treatment of pseudo-PEA, and ultimately the impact it has on the emergency department (ED) and patient outcomes.

## 2. Methods

To complete a review of the literature, we searched PubMed, MEDLINE, Scopus, and CINAHL databases for randomized clinical trials (RCTs), meta-analyses, systematic reviews, practice guidelines, and observational studies with the following search terms: "pseudo pulseless electrical activity", "pseudo PEA", "near PEA", "near pulseless electrical activity", and "pseudo electromechanical dissociation". A total of 37 articles were retrieved. Our search was further limited to English-language only studies. Two authors assessed titles and abstracts for appropriate articles, narrowing to 9 articles. All of the remaining articles were reviewed and their findings were discussed. The reference lists of selected articles were reviewed for additional studies. Abstracts, unpublished data, and duplicate articles were excluded. The search methodology is shown in Fig. 1.

## 3. Results

### 3.1. Diagnostic approach to patients with pseudo-PEA

It is becoming increasingly clear that the main modality used in the diagnosis of pseudo-PEA is ultrasound. For many years, bedside cardiac sonography has been the approach to assess cardiac arrest patients. Specifically, the use of transthoracic echocardiogram (TTE) was used to evaluate for the presence or absence of cardiac kinetic activity. A study by Salen et al. illustrated the benefit of using TTE to identify patients with pseudo-PEA [10]. Out of the 70 subjects that were enrolled in the study, 34 of them were in

PEA. Using TTE, it was then determined that out of the 34 subjects in PEA, 11 of them (32%) displayed the presence of cardiac kinetic activity, resulting in them being classified as subjects with pseudo-PEA [10].

Despite the effectiveness of TTE, the use of transesophageal echocardiography (TEE) has also recently shown promise in assessing patients with cardiac arrest. A study by Teran et al. examined the clinical impact of TEE during the ED evaluation of patients who experience out-of-hospital cardiac arrest [11]. A total of 7 patients presented to the ED with an initial rhythm of PEA. However, upon TEE evaluation, 2 of the 7 (28%) patients were found to have pseudo-PEA, as defined by organized cardiac mechanical activity visualized on TEE and organized electrical activity seen on the monitor. By providing serial real-time assessments of cardiac activity, TEE is able to identify patients with pseudo-PEA, thus allowing for therapy to be tailored to the degree of ventricular function. In addition, not only does TEE provide higher quality cardiac images, it offers diagnostic information without physically interfering with any ongoing resuscitation tasks [11].

A study by Larabee et al. further demonstrated the possible benefit of the utilization of a noninvasive Doppler ultrasound to differentiate pseudo-PEA from true PEA [12]. The Doppler ultrasound was used to detect low-flow cardiac output by measuring the blood flow velocities of carotid arteries. The use of a Doppler ultrasound identified 8 of the 9 cases (89%) of pseudo-PEA in swine. It was determined that pseudo-PEA would generate a low flow cardiac output, whereas true PEA would generate no cardiac output. There was good interobserver reliability with regards to the findings of pseudo-PEA ( $\kappa = 0.873$ ). From this study, the overall sensitivity of the Doppler ultrasound to detect carotid artery blood flow velocity was 89% with a specificity of 85% [12].

A recent study by Zengin et al. evaluated the sensitivity of cardiac ultrasound and doppler ultrasound for detecting pseudo-PEA. During initial pulse checks for a total of 137 patients, 37 patients were initially diagnosed with PEA (27%), of whom cardiac ultrasound detected cardiac activity in 7 patients, indicating that 19% of patients were in pseudo-PEA. Doppler ultrasound of the left femoral artery correctly identified pseudo-PEA in 2 of the 37 patients (5%). After 15 min of resuscitation, 6 of 29 patients (21%) of patients were found to be in pseudo-PEA on cardiac ultrasound, compared to 4 of 29 patients (14%) identified with Doppler ultrasound. Also of note, the pulse check times for cardiac ultrasound were much shorter than for either Doppler ultrasound or manual pulse palpation ( $p < 0.001$ ) [13] (Table 1).

### 3.2. Therapeutic approach to patients with pseudo-PEA

It is important to confirm the diagnosis of pseudo-PEA before treatment can begin as management strategies are different for these patients than those with true PEA. A study by Prosen et al. demonstrated that for patients with pseudo-PEA, despite prolonging the compression pause for 15 s and additional administration of 20 IU vasopressin displayed improved outcomes [14]. 94% of the patients with pseudo-PEA achieved restoration of spontaneous circulation (ROSC) and 50% achieved a good neurological outcome. There were also high rates of survival to discharge and a shorter duration of cardiopulmonary resuscitation (CPR) was required. The concept behind the use of additional vasopressin is related to the hemodynamic stabilization with peripheral vasoconstriction [14].

It has been noted that repeated cardiac chest compressions asynchronous with the patient's cardiac cycle could raise the mean intrathoracic pressure, resulting in a reduction in cardiac filling and ultimately in cardiac output [15]. Consequently, a study by Paradis et al. examined the effect of CPR with external chest compressions synchronized with pseudo-PEA cardiac systole and diastole in a porcine model [16]. It was found that during

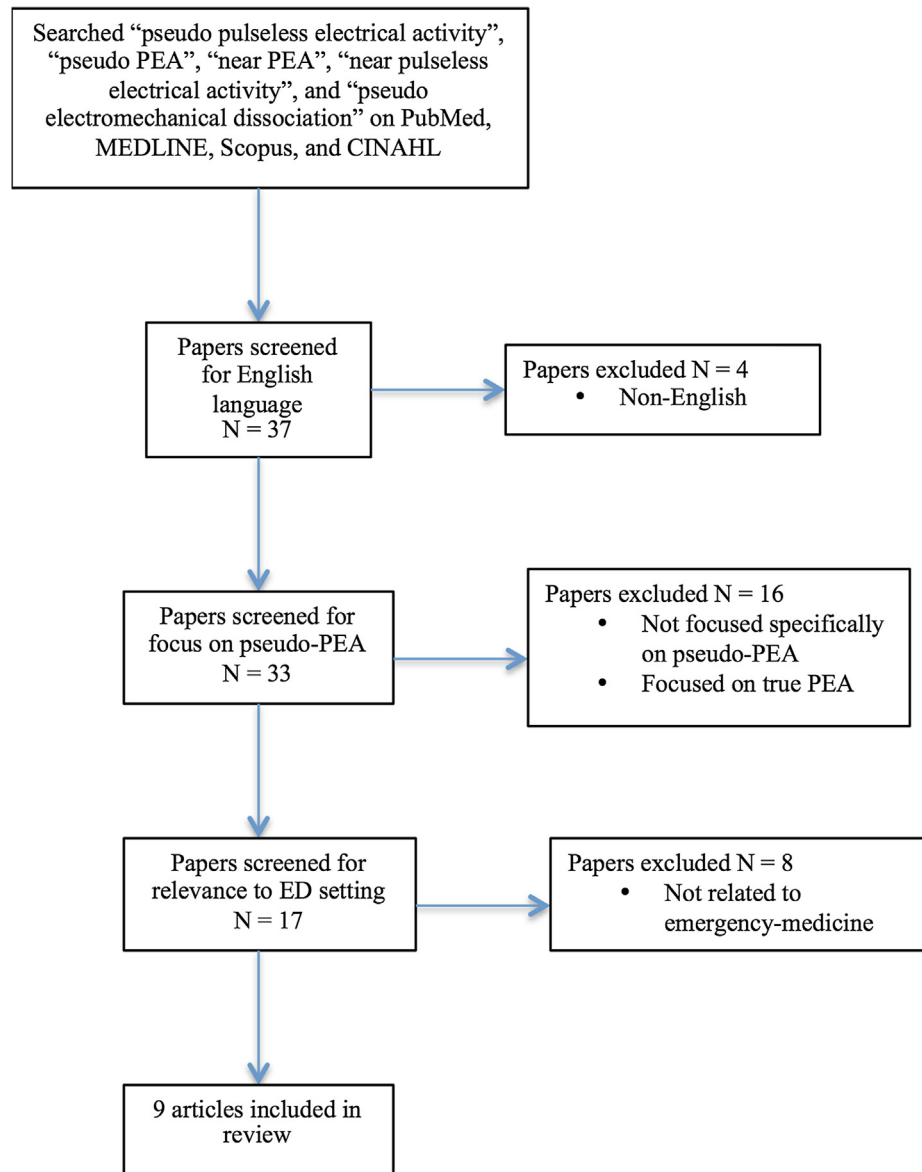


Fig. 1. The search methodology to include pseudo-PEA articles.

Table 1

Use of **ultrasound** in the **diagnosis of pseudo-PEA**.

Study	Design	No.	Findings	Conclusions
Salen et al. [10]	Prospective	70	Using TTE, 11 subjects with pseudo-PEA were identified	Visualization of cardiac kinetic activity can be made with the use of TTE, which helps to identify patients with pseudo-PEA
Teran et al. [11]	Prospective observational	33	Using TEE, 2 cases of pseudo-PEA were identified	TEE allows for characterization of cardiac activity, which includes the identification of pseudo-PEA
Larabee et al. [12]	Animal research	35	Using Doppler ultrasound, the blinded observers both recognized 8 of the 9 cases of pseudo-PEA and 22 of the 26 cases of true PEA	The Doppler ultrasound can reliably differentiate pseudo-PEA from true PEA over a wide range of aortic blood pressures
Zengin et al. [13]	Prospective	137	Cardiac ultrasound identified 37 patients with pseudo-PEA compared to doppler ultrasound identifying 7 patients	Cardiac ultrasound can shorten pulse check times and identify more patients in false-PEA than doppler ultrasound

pseudo-PEA, the **synchronization** of external chest compressions with cardiac systole resulted in a higher peak aortic pressure compared to synchronization with diastole. In addition, the aortic relaxation phase pressure was increased and the right atrial relaxation phase pressure was decreased with systolic synchronization.

The reasoning behind this is that the **external chest compressions are not interfering with ventricular filling**. All of these changes cause a higher coronary perfusion pressure with systolic synchronization, ultimately improving the low-flow state of those with pseudo-PEA [16] (Table 2).

**Table 2**  
Treatment strategies for patients with pseudo-PEA.

Study	Design	No.	Findings	Conclusions
Prosen et al. [14]	Prospective	16	With the use of <b>vasopressin</b> , ROSC was achieved in 15 patients, 24-hour survival in 13 patients, survival to hospital discharge in 9 patients, and a good neurological outcome in 8 patients	<b>Pseudo-PEA patients benefited</b> from the administration of additional <b>vasopressin</b> and the <b>prolongation</b> of the compression <b>pause</b>
Paradis et al. [16]	Animal research	8	Peak aortic pressure in systolic synchronization was 86.7 vs 69.3 in diastolic synchronization ( $P < 0.0001$ ), and coronary perfusion pressure in systolic synchronization was 37.6 vs 30.2 in diastolic synchronization ( $P = 0.0001$ )	<b>Synchronizing external chest compressions</b> with <b>cardiac systole</b> is more beneficial than diastolic synchronization for pseudo-PEA patients

**Table 3**  
Prognosis for patients with pseudo-PEA compared to true PEA.

Study	Design	No.	Findings	Conclusions
Chardoli et al. [17]	Prospective	50	Out of 39 patients with pseudo-PEA, 17 achieved ROSC, whereas out of 11 patients with true PEA, 0 achieved ROSC	<b>Survival</b> outcome for patients with <b>pseudo-PEA</b> is <b>higher</b> compared to patients with <b>true PEA</b>
Flato et al. [18]	Prospective observational cohort	32	Out of 27 patients with pseudo-PEA, 6 survived to hospital discharge, whereas out of 5 patients with true PEA, none of them survived to hospital discharge	<b>Better</b> prognosis for patients with <b>pseudo-PEA</b> compared to those with <b>true PEA</b>
Wu et al. [19]	Systematic review and meta-analysis	777	Pseudo-PEA patients more likely to obtain ROSC (RR 4.35, $p < 0.00001$ ). 15 patients survived to discharge (all pseudo-PEA).	<b>Better</b> prognosis with <b>pseudo-PEA</b> compared to those with <b>true PEA</b>

### 3.3. Prognosis of patients with pseudo-PEA

The **prognosis** for patients with **out-of-hospital cardiac arrest** is overall quite **poor**. Ventricular fibrillation/pulseless ventricular tachycardia has the best prognosis, followed by PEA, with asystole portending the worst prognosis. Although the prognosis for patients presenting with **PEA** is **poor**, the likelihood of survival for patients with **pseudo-PEA** is **greater** than those with **true PEA**. A study by Chardoli et al. examined the resuscitation outcome for PEA arrest patients, and they found that **43%** of the patients with **pseudo-PEA** achieved **ROSC**, whereas **no** patients with **true PEA** achieved **ROSC**. This indicates that all of the patients presenting with true PEA died [17]. In another study by Flato et al., it was seen that the rates of **ROSC** were **70%** for the patients with **pseudo-PEA** and **20%** for those with **true PEA**. Despite achieving ROSC, the patient with true PEA did not survive to hospital discharge, whereas **22%** of the patients with **pseudo-PEA** survived to **hospital discharge** and **15%** of them survived after 180 days [18]. A further study by Wu et al. demonstrated that although ROSC was achieved in some patients with pseudo-PEA and true PEA, only a few of the patients with pseudo-PEA survived to hospital discharge [19] (Table 3).

These results indicate that the **absence of cardiac activity** is an **accurate predictor of death**, as **all** of the patients with **true PEA** died, whereas **a few** of the patients with **pseudo-PEA** survived. Despite the **prognosis** of **pseudo-PEA** being **more favorable** than true PEA, the chance for **survival** for these patients is **still poor**.

## 4. Discussion

The purpose of this review was to examine what is in the literature about the condition known as “pseudo-PEA.” Specifically, this review looked at the prognosis of pseudo-PEA and how it is diagnosed and treated.

In order to describe pseudo-PEA, PEA must first be defined. However, PEA itself is **not** clearly **defined**. The American Heart Association (AHA) indicates that there is **no single unifying definition** for PEA. Nevertheless, the AHA describes PEA as spontaneous organized cardiac electrical activity, but **without blood flow sufficient** to maintain **consciousness** and **tissue perfusion** [7]. On the

other hand, **pseudo-PEA** can be defined as a condition that occurs **when cardiac electrical activity** is **present**, a **palpable pulse** is **absent**, and any of the following are present: **myocardial contractions** are demonstrated by an **echocardiogram**, such as with the utilization of **TTE** or **TEE**, or **carotid artery blood flow** is demonstrated by **Doppler ultrasound** [10–12].

Bedside ultrasound for the evaluation of the patient in cardiac arrest has become standard of care, although **concerns** have been raised in recent articles regarding **increased duration** of pulse checks that include bedside **ultrasound** compared to pulse checks without ultrasound [20,21]. However, it is **critical** to **differentiate pseudo-PEA** from **true PEA** as the **treatment strategies** and **prognoses** for the two conditions are **different**. With regards to prognosis, pseudo-PEA is associated with higher rates of ROSC and survival to hospital discharge [17–19]. Identification of the underlying etiology of cardiac arrest can lead to **expedited treatment of the severe shock state leading to pseudo-PEA** [22]. Understanding the patient's prognosis will help guide the decision of terminating resuscitation efforts. For instance, the identification of **true PEA** (cardiac standstill) could **predict the failure** of resuscitation and help inform the decision to **terminate CPR**, which allows for hospital resources to be utilized elsewhere. In **contrast**, the presence of **cardiac activity** in **pseudo-PEA** patients can **encourage** more **aggressive resuscitation** efforts and lead to interventions targeting specific ultrasound findings (e.g. empty hyper dynamic ventricle leading to fluid administration and search of cause of hypovolemia, or dilated right ventricle leading to the administration of thrombolytic therapy) [19]. Nevertheless, **whether the patient is in pseudo-PEA or true PEA, it is always important to remember that the outcome of cardiac arrest is still very poor** [4].

Regarding the treatment of patients with pseudo-PEA, a directed therapeutic approach to go along with aggressive resuscitation will provide patients with the best chance to survive. The administration of **additional vasopressin** is associated with a **survival benefit** and a **good neurological outcome** [14]. Moreover, external chest **compressions synchronized** with cardiac **systole** can result in a high coronary perfusion pressure improving the blood flow throughout the body [15].

Physicians have noted that **pseudo-PEA constitute 42% to 86% of the total PEA population** [10]. With the increase in the incidence of



pseudo-PEA, there is a need for future research to focus on creating new therapies that will address this clinical condition. It is seen that many of the studies with regards to pseudo-PEA are performed in animals. Consequently, application of these studies to humans should be the focus of future research studies.

## 5. Limitations

This review has several limitations. Some of the studies mentioned in this review represented only a small sample size, which would limit the generalizability of the findings. A larger sample size of subjects with pseudo-PEA would be required to confidently apply the conclusions of these studies to the general population. Also, some of these studies took place in a specific region; therefore the results may not be generalizable to other populations. In addition, the patients in these studies may have varying underlying causes for their pseudo-PEA, and this may affect the reliability of the studies.

## 6. Conclusions

The Advanced Cardiac Life Support (ACLS) guidelines are for physicians to follow when treating patients with true PEA. However, there are no such guidelines for pseudo-PEA. From this review, it is apparent that the topic of pseudo-PEA in the literature is very sparse. Consequently, further research should be done regarding this condition. It is imperative for physicians to be able to identify and treat patients who present with pseudo-PEA as their chance for survival is greater than those who present with true PEA.

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## Declaration of Competing Interest

The authors do not have a financial interest or relationship to disclose regarding this research project.

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