Simplifying the diagnosis and management of pulseless electrical activity in adults: A qualitative review*

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LEARNING OBJECTIVES

On completion of this article, the reader should be able to:

1. Explain the implications of the diagnosis of pulseless electrical activity (PEA).
2. Describe the methodology to remember causes of PEA.
3. Use this information in a clinical setting.

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Objective: **The algorithms provided for advanced cardiac life support by the American Heart Association and the European Resuscitation Council Guidelines for Resuscitation for the diagnosis and treatment of pulseless electrical activity (PEA) correctly stress the importance of searching for potentially treatable causes, and suggest contributing factors that should be considered. This study sought evidence to support the factors that they mention in the algorithms.**

Data Source: **Human and animal studies in MEDLINE.**

Study Selection: **Putative causes of PEA along with the string "AND (PEA OR pulseless electrical activity OR electromechanical dissociation OR EMD)".**

Data Extraction: **Human studies documenting association and causation.**

Data Synthesis: **Qualitative.**

Conclusions: **Documentation for most putative causes is weak. Based on current documentation, a simplified rule is offered to direct resuscitators to treatable precipitants of PEA. It emphasizes the documented causes of PEA, has good pedagogical qualities, guides treatment, and is testable. Studies need to be performed to identify the best diagnostic and treatment strategies for PEA. (Crit Care Med 2008; 36:391–396)**

KEY WORDS: pulseless electrical activity; advanced cardiac life support; electromechanical dissociation; cardiac arrest; diagnosis; therapy

The current approach to the management of cardiac arrest has been well described by the American Heart Association (AHA) in its advanced cardiac life support (ACLS) guidelines and by the European Resuscitation Council guidelines (1, 2). Although initial attention to cardiopulmonary resuscitation is similar, their protocols then branch into further algorithmic approaches based on the type of rhythm and whether or not the patient has a pulse.

Pulseless electrical activity (PEA), previously known as electromechanical dissociation (EMD), occurs when a patient has organized electrical activity but no pulse. Dr. Paradis and colleagues (3) reported that the hearts of 41% of patients who presented with EMD had pseudo-EMD: mechanical cardiac activity that generated a low pressure that was inadequate to lead to a palpable pulse. The prognosis for PEA, even with the ACLS or European algorithm, is dismal. In the largest study of cardiac arrest in hospital patients, Dr. Nadkarni and colleagues (4) reported that of 11,963 adults with PEA, only 11% survived, and of these, only 62% had good neurological outcomes. For victims of out-of-hospital PEA, outcomes are even worse. A study of >1,000 such patients reported that only 15% were hospitalized and only 2.4% discharged (5). In another study, no patient with unwitnessed cardiac arrest or who was >80 yrs of age survived (6).

*See also p. 619.

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The reason for the miserable prognosis is that PEA is often caused by major damage to the left ventricle (7). Therefore, it is necessary not to miss the other less common but potentially treatable causes of PEA. Indeed, the AHA admonishes physicians that "the hope for resuscitation is to identify and treat a reversible cause," and implores resuscitators to "search for and treat possible contributing factors" that included hypovolemia, hypoxemia, hydrogen ion (acidosis), hypo- or hyperkalemia, hypoglycemia, hypothermia, toxins, (cardiac) tamponade (CT), tension pneumothorax (TP), thrombosis (coronary or pulmonary), and trauma." (1) By listing the causes in this fashion, the AHA appears to be proffering a mnemonic for resuscitators. It is probably based on an unreferenced article containing a similar (although not identical) "10-step training mnemonic" comprising conditions ("5 Hs and 5 Ts") felt to represent the "most likely and possibly treatable conditions that may be associated with pulseless QRS electrical activity" (8, 9). The European Resuscitation Council guidelines are even more explicit about proffering a memory aide. "For ease of memory," it divides potentially reversible causes into "the 4 Hs" (but discusses six that start with H—hypoxemia, hypovolemia, hyperkalemia, hypokalemia, hypocalcemia, and hypothermia—and one that starts with A, acidosis) and "the 4 Ts" (tension pneumothorax, tamponade, therapeutic or toxic substances, and thromboembolism) (2). Research on memory suggests that it is difficult for humans to recall more than seven items at one time. They do better with small chunks of information (10). Cardiopulmonary resuscitation team members may be fatigued and have high circulating catecholamine levels related to the stress of this life or (mostly) death situation (11). A code alarm and the following dash to the patient do not dispose to recalling a list, such as the one provided by the AHA, of 13 entities combined into 11 items that conflates two entirely separate causes (cardiac and pulmonary thrombosis) and combines two antipodal conditions (hypo- and hyperkalemia), even if it had been committed to retrievable memory (12).

LITERATURE REVIEW

This article analyzes the differential diagnosis provided by ACLS by performing MEDLINE searches of the associated conditions in the following manner: "[putative cause OR randomized controlled trial] AND (PEA OR pulseless electrical activity OR electromechanical dissociation OR EMD)." After relevant articles were found, the search was further expanded using the related articles link on PubMed's Web site. Based on the evidence, the article suggests an approach to diagnosis that is easier to remember, will not miss treatable causes of PEA, supports a treatment algorithm, and is verifiable.

Potentially Treatable Causes of PEA. Bleeding that leads to the severe hypovolemia needed to cause PEA must be massive. Most frequently, it is caused by external trauma or bleeding into the gastrointestinal tract, thorax, abdomen, and perhaps thighs (13). Obstruction to circulation may cause PEA, and by and large, there are three major obstructions that lead to PEA: massive pulmonary embolization (PE), CT, and TP. PE may be underdiagnosed as a cause of PEA. In a small series of 20 patients with PEA, nine had PE. Of the eight patients who were diagnosed before death, only two survived (14). CT obstructs circulation by blocking filling of the ventricles during diastole. Its major causes are rupture of the ventricle secondary to bullet or knife, myocardial infarction, and inflammation of the pericardium due to a number of infections or disorders that lead to inflammation (15).

TP, not the more common simple pneumothorax, blocks circulation by impeding the return of venous blood to the heart. Eminently treatable, it is nonetheless a rare cause of PEA. It should especially be considered in trauma patients, those with advanced emphysema, and patients who develop PEA while on a ventilator. A rare type of obstruction to venous return can occur in patients who are on a ventilator and develop air trapping (16). Removing the connection from the endotracheal tube to the ventilator should lead to prompt return of the pulse.

Other Putative Causes. What is the provenance of the eight other causes mentioned by the AHA? Although it is hard to be sure, they are probably modifications of the "10-step training mnemonic" (8). The causes listed in major textbooks are not entirely consistent with the AHA listing (1). *Hurst's The Heart* mentions eight without citation (17). The Harrison textbook adds drug overdose and massive myocardial infarction (18). *Critical Care* mentions only hypoxemia and acidosis, although it references the AHA algorithm (19).

Hypoxemia and acidosis are probably best considered not as initiators of PEA but as factors that exacerbate hypovolemia and pump failure by their negative inotropic and vasodilatory effects. They are accompaniments of any type of cardiac arrest. Their treatment is considered in the basic approach to ACLS that stresses oxygenation and discusses treatment of acidosis.

It is hard to determine the origin of the association between PEA and hypokalemia. Cardiac arrest leads to intracellular to extracellular movement of potassium secondary to ischemic acidosis and the problem should largely self-correct (20). A query for "hypokalemia" or "low potassium" and the study's search string produced two irrelevant studies.

The relationship between hyperkalemia and PEA is more complicated. Despite several case reports to the contrary (21), hyperkalemia should not cause PEA unless it is associated with peaked T waves, widened QRS complexes, or sinusoidal QRS pattern, and one may assume that if these are not seen on the electrocardiogram, other causes of PEA should be considered. However, increased QRS duration is not specific for hyperkalemia as an initiating cause of PEA. Animal studies suggest that the hypoxemic heart demonstrates shortening of the QT interval with gradual elevation of the J point and ultimate development of a monophasic complex (22). Human studies are further complicated by preexisting cardiac disease and medications that may influence the QRS duration. In a study of 503 patients who presented in the field with EMD not related to poisoning, initial rhythm strips showed that 61% had a widened QRS and 10% monophasic slurred complexes (23). While the study by Dr. Nadkarni and colleagues (4) does not report specific causes of PEA, it does list "metabolic and electrolyte disturbances" for 11%, although it does not mention hyperkalemia specifically. A search for "hyperkalemia" or "high potassium" resulted in no other relevant articles. Although it is recommended that calcium be given as an antagonist to the electrical abnormalities caused by hyperkalemia, its success with PEA due to hyperkalemia has not been reported.

The evidence for hypoglycemia causing PEA is scanty. Querying with "hypoglycemia" or "low glucose" and the search term led to one article on cardiac

arrest in patients with advanced cardiac disease who were awaiting transplant, but the article did not specify that the hypoglycemic patient had PEA (24).

Severe hypothermia is much more commonly associated with hypotension than PEA. "Hypothermia" and the search term led to 22 inapplicable articles. Hypothermia is not a cause of in-hospital cardiac arrest (25). In outpatient PEA, it should especially be considered in the winter in regions of the world with high altitudes or latitudes or for those who work in cold environments. Its diagnosis should not be occult if appropriately scaled thermometers are used and if Osborne waves are visible on the electrocardiogram. Nonetheless, its association with PEA is rare because the study by Dr. Engdahl and colleagues (5) of 1,069 outpatients with PEA did not report hypothermia as a cause, despite being conducted in Sweden over a 17-yr period.

Toxins and therapeutic agents may lead to PEA by increasing venodilation and decreasing cardiac contractility, but these are usually massive overdoses for which there is no immediate treatment unless the patient survives cardiopulmonary resuscitation. Searches for "toxin*" (with the asterisk representing any string of characters) or "overdos*" or "poison*" and the study's search string led to several relevant articles. In one study of 201 patients who had suicidal overdoses, three died of cardiogenic shock, EMD, and secondary acute respiratory failure resistant to therapy (26). Case reports implicated digoxin (27), atenolol (28), disopyramide (29), propranolol (30), amoxapine (an antidepressant) (31), and verapamil (32). PEA associated with beta-blocker overdosage may be responsive to intravenous calcium (30). In any case, these causes of PEA are quite unusual because the Dr. Engdahl and colleagues outpatient PEA study (5) reported that only 1% of 889 adults had "drug abuse" as a factor. The inpatient study by Dr. Nadkarni and colleagues (4) reported that 1% of inpatient cardiac arrests were caused by a "toxicological problem," but does not report specifically on PEA.

Trauma can certainly lead to PEA, but trauma is a precipitant, not a diagnosis. It leads to PEA by several mechanisms, including one or more of hypovolemia, pump failure due to contusion or laceration, TP, and CT. Although potentially treatable, blunt trauma leading to PEA is particularly ominous. In a study of 110 victims of blunt trauma with PEA, only 1% survived with secondary neurological impairment, leading the authors to conclude that "consideration should be given to allowing paramedics to declare blunt trauma victims with PEA dead at the scene" (33).

Treatment of Major Causes. The most easily treatable cause of PEA is TP. Needle thoracostomy on the involved side will immediately relieve the pressure that obstructs venous return. The pulse should return immediately, unless there is an accompanying process such as hypovolemia or CT. A chest tube can then be placed to correct the pneumothorax at a more leisurely pace, although air leakage may complicate matters (34).

The next most remediable cause of PEA may be thought to be volume depletion. However, PEA secondary to volume depletion suggests a major loss of circulatory volume, which may not be easy to stanch or replace or which already may have led to ischemic damage of critical organs, leading to the poor prognosis with PEA in trauma (33).

Similarly, needle drainage of a pericardial effusion or blood that causes tamponade may be considered straightforward. However, this procedure may be difficult to perform in the cardiac arrest setting. Although occasionally dramatically successful, pericardiocentesis may not be easily accomplished by the relatively inexperienced physicians who comprise many resuscitation teams, nor is it a skill requirement in many residency programs. In the setting of trauma, thoracotomy may be necessary to attempt to suture the rent in the myocardium caused by a bullet or knife wound.

Thoracotomy in the setting of myocardial infarction and PEA occasionally has been successful (35). Dr. Figueras and colleagues (36) reported that EMD developed in 72 of 1,487 (4.8%) consecutive patients admitted with a first myocardial infarction who were in Killip classes I and II; 62 (86%) did not respond to resuscitative efforts and an emergency thoracotomy was performed on 15 of them leading to two survivors (two of 15 surgical [13%]; two of 62 PEA [3.2%]). Of ten EMD patients and nine other hypotensive patients with free wall rupture who survived cardiac arrest and were treated medically, 15 (78.9%) survived a median follow-up period of 41 months, two after later cardiac surgery. Unfortunately, the study reports survival only for the combined group.

Massive PE may be an underdiagnosed cause of PEA. In a retrospective study of all 1,246 patients presenting to a university hospital with cardiac arrest, 60 patients (4.8%) were determined to have PE (37). Of these, 38 (63%) had PEA; 30 of them were correctly diagnosed as having PE. Two of 15 who received thrombolytic therapy survived (13%) compared with none of 15 who did not receive thrombolysis, calculated from Tables 3 and 4 in Dr. Kürkciyan and colleagues' paper (37). In a prospective study using echocardiography, Dr. Comess and colleagues (14) reported that of the 25 consecutive patients with PEA as the initial event, nine (36%) had PE; two survived hospitalization, one after surgery and one after thrombolytic therapy.

Dr. Abu-Laban and colleagues (38) demonstrated that general outpatient use of thrombolytic therapy in PEA patients does not lead to improved outcomes, but they did not specifically address PE. The efficacy of vigorous chest compression to break up a central obstructing clot has not been assessed. Dr. Courtney and colleagues (39) reported that 67.6% of patients with witnessed arrests and PEA had PE, and that this combination of findings had 94.5% specificity for massive PE.

The 3 and 3 Rule. The 3 and 3 rule simplifies the etiology of PEA into five major causes divided into two hierarchical chunks of three items each. The first list considers the three major mechanisms: 1) severe hypovolemia; 2) pump failure; and 3) obstruction to circulation. The next chunk considers the three major potentially treatable causes of obstruction to circulation: 1) TP; 2) CT; and 3) massive PE (Fig. 1).

A Proposed Algorithm Using the 3 and 3 Rule. A pulseless patient is identified. Cardiopulmonary resuscitation is begun and a cardiac monitor attached. If the patient has a rhythm other than ventricular fibrillation or tachycardia, PEA is confirmed. A pulse is then sought at the femoral artery during chest compression. There should be a pulse with effective chest compression. If there is a pulse, the patient probably has cardiac failure, although conductance of intrathoracic pressure along the femoral vein could give a false positive pulsation and obstruction to circulation is still a possibility. If chest compression is vigorous enough and no pulse is felt, the patient has severe volume depletion or obstruction to circulation. If neck veins are prominent (without chest compression), obstruction to circulation is likely. Auscultation after intubation and deviation of the trachea can aide the diagnosis of TP. Despite these suggestions, it must be mentioned that although the absence of a pulse

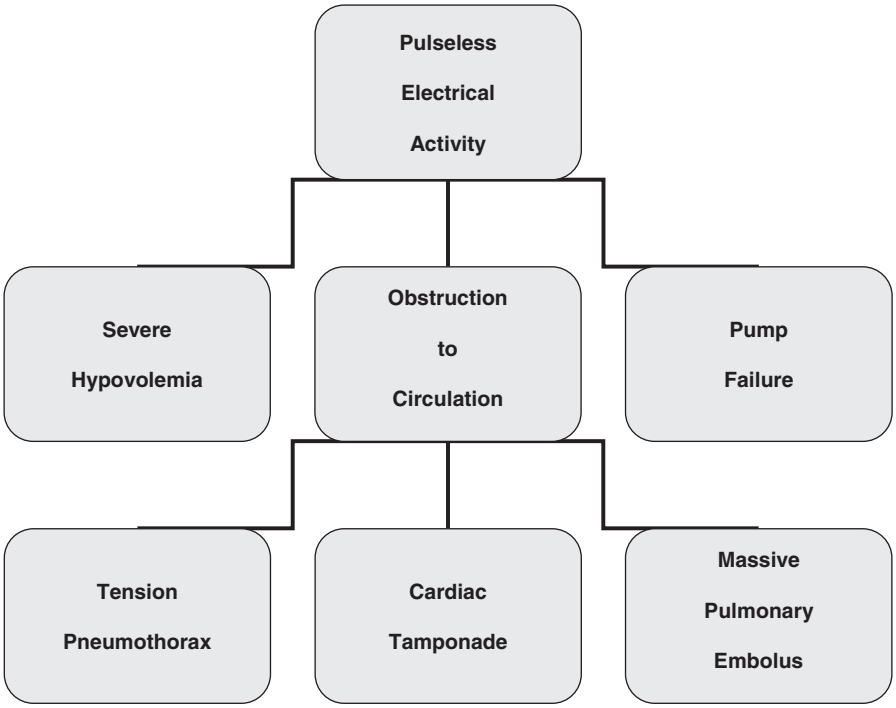

Figure 1. The 3 and 3 rule for patients with pulseless electrical activity. There are three major mechanisms of PEA. Obstruction to circulation can be further divided into three major causes. Most of the traditionally listed "causes" of PEA (hypoxemia, acidosis, hypokalemia, hyperkalemia, hypoglycemia, hypocalcemia, hypothermia, toxins, therapeutic agents) probably (although not exclusively) act by exacerbating pump failure. Trauma can lead to any of the first three major causes.

is a critical component of the definition of PEA, its use in the differential diagnosis and management of PEA has not been studied rigorously (see Limitations section).

If the patient has a pulse with chest compression and the QRS is wide or there are peaked T waves, or if there is a sinusoidal QRS, calcium chloride could be given intravenously. Although most patients will not respond to this therapy, if the QRS narrows or the T wave amplitude diminishes, the patient could be given bicarbonate and glucose and insulin. Potassium, other electrolytes, glucose, and serum creatinine should be ordered to guide further therapy. More studies are needed to specifically guide therapy in this situation.

Once TP has been excluded, the most likely remaining treatable diagnoses are massive PE or tamponade. An emergency echocardiogram should make the appropriate diagnosis. At this point, for a medical patient, pericardiocentesis has a small chance of helping. For a trauma patient in the ER, thoracotomy should be considered.

The Importance of Clinical Setting in PEA. The clinical setting can give an enormous amount of information about the likely cause of PEA. Knife- and gun-related chest trauma always should bring to mind hypovolemia, tamponade, and TP. Abdominal trauma should suggest hypovolemia. Recent surgery should evoke consideration of PE or hypovolemia. PEA in the setting of a transmural myocardial infarction should trigger thoughts of pump failure, rupture, and subsequent CT. In the setting of renal failure, all five causes (severe hypovolemia, pump failure, TP, CT, massive PE) need to be considered.

Benefits of the 3 and 3 Rule. A major benefit of the rule is that it is pedagogically very logical; it also nicely parallels present clinical teaching about renal failure, where the causes are grouped into prerenal (hypovolemia), intrarenal (pump failure), and postrenal (obstruction to circulation) (40). The rule can be taught as a list of five primary causes (severe hypovolemia, pump failure, TP, CT, massive PE), but it is probably better to teach it as two chunks of three items each (41) (Fig. 1). The explicit statement of obstruction to circulation and its tripartite division gives the resuscitator a further cue by linking its composite causes to a physiological mechanism. Under the stress of a cardiac arrest, this is a much easier chore than remembering, for example, a list of 11 items comprising 13 possible entities. As demonstrated, the rule can be incorporated into an algorithm that immediately directs treatment.

LIMITATIONS

The rule, based on clinical experience and the literature, still needs to be validated. In contradistinction to the present guidelines, it lends itself more readily to testing. It need not be perfect, but it must outperform the present guidelines in leading resuscitators to remember the most treatable causes of PEA. Such a study could be performed by testing resuscitators for the major causes of PEA after they have been taught by either the ACLS or European algorithm or the 3 and 3 rule. A recent study of a memory aid for EMD that used eight causes, color, positioning, numbering, clockwise sequence, and arrow cues led to a better recall of the causes of EMD than a "4 Hs and 4 Ts" approach (42). Individual components of the diagnostic and treatment algorithm need to be tested in practice. An example may be testing whether the ability to detect a femoral pulse with compression (a crucial component of the approach to PEA suggested above) leads to reliable diagnostic information.

The ultimate test of the algorithm based on the 3 and 3 rule would be that it led to better survival with acceptable morbidity. A partial approach to this problem would be a randomized simulation to see if using it more quickly identified the cause of PEA and gave more definitive therapy than the ACLS approach. Better yet would be an outcome study in which arrested patients were randomized to these different algorithms. A difficulty in undertaking such a study is the number of patients it would take to demonstrate a difference in outcome with PEA. It remains to be determined whether the use of technologies such as ultrasound will obviate the need for such a mnemonic (43).

Current Status of Research in the Diagnosis and Therapy of Causes of PEA. Review of the literature highlights the precariousness of the evidence base for the best approaches to the diagnosis and therapy of causes of PEA. The search string "PEA OR pulseless electrical activity OR electromechanical dissociation OR EMD" limited to randomized controlled trials returned only five studies: two in patients with EMD or asystole (44, 45) and two from the 1980s that each had ≤90 patients (46, 47). The one trial with

1,583 patients already has been discussed (37). More well-conducted randomized trials are sorely needed.

CONCLUSION

A new approach has been offered to guide the diagnosis and treatment of PEA. It appears to have advantages over the ACLS algorithm for PEA in that it: a) is short and should be easier to remember than previous, inconsistent approaches; b) stresses the potentially treatable causes of PEA; c) directs therapy; and d) has features of the razor favored by Occam. Its use lends itself to further testing of its pedagogical abilities. A terse, testable, and rememberable algorithmic approach to the diagnosis and treatment of PEA that emphasizes the major treatable causes of PEA has the potential to increase survival from this severe presentation of cardiac arrest.

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