ORIGINAL RESEARCH

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Definitions and Treatment of Pulseless Electrical Activity, Pseudo-Pulseless Electrical Activity and Cardiogenic Shock: A Retrospective Video-Based Analysis

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Abstract

Objective: Cardiopulmonary arrest (CPA) rhythms are classified into shockable and non-shockable categories. Pulseless electrical activity (PEA) is a non-shockable rhythm defined as the absence of a palpable pulse despite organized electrical activity on the monitor. However, PEA encompasses a spectrum from complete cardiac inactivity to cardiogenic shock. Pseudo-PEA (p-PEA) represents an intermediate state, marked by electrical activity and varying degrees of myocardial motion. While PEA and p-PEA are treated in a similar manner to asystole, their distinct characteristics suggest a potential need for differential treatment, especially for p-PEA, which may benefit from positive inotropic therapy. This study aims to establish diagnostic criteria for PEA, p-PEA, and cardiogenic shock and assess their responses to inotropic therapy.

Materials and Methods: This retrospective, video-based study was conducted in the emergency department of a university hospital. Archived ultrasound (USG) video recordings from August 2017 to April 2021 were analyzed. Adult CPA patients with documented cardiac activity and positive inotropic therapy during cardiopulmonary resuscitation (CPR) were included. Data on demographic details, CPR characteristics, treatment interventions, and clinical outcomes were collected. Statistical analysis was performed using SPSS v24.0 with a significance level of $p<0.05$.

Results: Out of 94 patients, 12 met the inclusion criteria. Patients were divided into three groups: those with valvular motion alone (n=4), valvular and myocardial motion (n=6), and cardiogenic shock (n=2). Return of spontaneous circulation was achieved in all patients with both valvular and myocardial motion after inotropic therapy ($p=0.002$), but not in those with only valvular motion.

Conclusion: Patients with valvular motion alone were classified as PEA, while those with myocardial activity were defined as p-PEA. Positive inotropic therapy was effective in p-PEA but not in PEA. USG, including carotid and femoral examinations, can aid in differentiating cardiogenic shock from p-PEA, emphasizing the need for specific treatment protocols. Further research is essential to validate these findings.

Keywords: Pulseless electrical activity, pseudo-PEA, cardiogenic shock, positive inotropic therapy, echocardiography

Introduction

Cardiopulmonary arrest (CPA) rhythms are generally categorized into shockable and non-shockable rhythms [1,2]. To date, significantly more research has focused on shockable rhythms, including the development of defibrillation [3], with many guidelines providing more extensive coverage on this topic [4]. Shockable rhythms in CPA are characterized by irregular electrical activity accompanied by the absence of a palpable pulse, and defibrillation forms the cornerstone of their treatment. Non-shockable rhythms, on the other hand, are divided into asystole and pulseless electrical activity (PEA). Unlike shockable rhythms, PEA demonstrates organized electrical activity and frequently a certain degree of regular cardiac activity. However, its treatment follows the same protocol asystole, which lacks both electrical activity and cardiac motion entirely [4].

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PEA is defined as an arrest rhythm characterized by the absence of a palpable pulse despite the presence of an organized electrical rhythm on the monitor [5,6]. However, this definition is rather superficial, as PEA encompasses a wide spectrum ranging from a complete absence of cardiac activity to cardiogenic shock with the return of spontaneous circulation (ROSC), including various degrees of cardiac activity in between. This ambiguity is particularly notable in the absence of cardiac imaging, as routine use of cardiac ultrasound (USG) for rhythm assessment remains a topic of debate [2,7,8]. The intermediate condition, between PEA and cardiogenic shock, where varying degrees of valvular and myocardial activity accompany electrical activity, is often referred to as pseudo-PEA (p-PEA) [9]. p-PEA can only be diagnosed through cardiac USG. It is considered a more severe form of cardiogenic shock, in which perfusion pressure is inadequately maintained, ultimately resulting in an undetectable pulse [10]. As evident, p-PEA shares close ties with both PEA, and cardiogenic shock and lacks clearly defined boundaries. The differentiation among these entities fundamentally revolves around two factors: the presence or absence of a palpable pulse and the existence of cardiac activity. Despite numerous studies on pulse detection [11], manual palpation remains the standard method for assessing the pulse during cardiopulmonary resuscitation (CPR) in routine practice. However, manual pulse checks are subjective, varying between patients and practitioners [11,12]. The debate regarding whether the carotid or femoral artery is the most accurate site for pulse palpation further complicates the matter, as the optimal site may vary depending on the patient [12]. It is evident that a practitioner may not perceive identical pulse intensities in a morbidly obese versus an extremely thin patient, and even two practitioners assessing the same patient may arrive at differing conclusions. The second challenge lies in the confusion surrounding the distinction between PEA, p-PEA, and cardiogenic shock based on cardiac activity, even when cardiac USG is employed. For instance, in a patient with organized electrical activity and no detectable pulse, echocardiography may reveal no cardiac activity, isolated valvular motion, partial myocardial motion in addition to valvular movement, or coordinated myocardial motion involving the entire myocardium. This spectrum blurs the line between where PEA ends and p-PEA begins. In terms of treatment, PEA and p-PEA are currently managed in the same way as asystole. However, p-PEA, given its close relationship with cardiogenic shock, represents a distinct clinical condition that may benefit from adjunctive pharmacological therapies, such as positive inotropes, in addition to CPR, potentially leading to different outcomes [9,13]. Cardiogenic shock, on the other hand, is not a cardiac arrest state but rather a condition with a specific therapeutic approach, primarily involving positive inotropic agents. These entities represent clinical conditions with distinct treatment needs and outcomes, yet they are almost uniformly categorized as PEA in current guidelines [2]. Distinguishing among these definitions has significant implications for treatment, particularly in determining the need for ongoing CPR and the initiation of positive inotropic therapy, both of which are of critical importance.

This retrospective study, based on echocardiographic video recordings, aims seeks to establish the differential diagnosis of PEA, p-PEA, and cardiogenic shock with a treatment-focused approach, as well as to identify patients who may benefit from positive inotropic therapy.

Materials and Methods

Study Design and Setting

This study was conducted as a retrospective video-based analysis in the critical care unit of the emergency medicine department at a tertiary university hospital. The critical care area of this emergency department is equipped with 16 beds for vital monitoring, 6 mechanical ventilators, and USG devices, including two portable units, one fixed unit, and one with a transesophageal probe. All critical patients and those experiencing CPA are managed in this area. Bedside USG examinations of patients in the critical care unit are performed by senior residents under the supervision of five attending physicians or faculty members, and video recordings are systematically archived. The study retrospectively reviewed archived video recordings from August 2017, when regular video archiving began on the USG devices, to April 2021. Ethical approval for the study was obtained from the İzmir Katip Çelebi University Non-Interventional Clinical Research Ethics Committee on April 15, 2021 (decision number:0214, date: 15.04.2021).

Study Population

The study included patients aged 18 years or older who experienced in-hospital or out-of-hospital CPA and had cardiac USG imaging performed during CPR. The study population consisted of patients in whom any degree of cardiac activity was detected on these USG images, and who were subsequently initiated on positive inotropic therapy. Cardiogenic shock was defined as the presence of organized rhythm, echocardiographic evidence of myocardial activity, a weak palpable pulse with hypotension, and hemodynamic instability following ROSC. Patients were excluded if they had incomplete cardiac USG video recordings either before or after the initiation of positive inotropic therapy, if information was missing in physician or nurse observation forms, or if clinical outcome data were unavailable due to transfer to another hospital or incomplete contact information.

Data Collection and Study Protocol

During the study period, all recorded images from the three available USG devices were reviewed. All patients who underwent cardiac USG imaging during CPR were identified.

These patients were cross-referenced with the hospital's patient records; and those who received positive inotropic therapy during CPR were identified. The study population comprised patients with complete video recordings both before and after the initiation of positive inotropic therapy. Demographic data, chronic conditions, laboratory test results, CPR durations and outcomes, rhythms observed during CPR, treatment regimens, and interventions were applied, and, if ROSC was achieved, the return rhythm and vital signs were collected from the hospital's digital patient records and follow-up forms. These details were recorded on patient data forms. Patients who achieved ROSC were followed until hospital discharge or death, and data on 24-hour and in-hospital clinical outcomes were documented.

Statistical Analysis

Descriptive statistics were calculated, including frequency, percentage, median, minimum (min.), and maximum (max.) values. Counts and percentages were reported for categorical variables, while min. and max. values and interquartile ranges were determined for numerical variables The normality of the distribution for continuous variables was assessed using histogram curves, Kurtosis-Skewness values, and the Shapiro-Wilks test. Group comparisons were performed using the chi-square test. All statistical analyses were conducted with SPSS version 24.0 software using a 95% confidence interval, with a significance level set at $p<0.05$.

Results

During the study period, cardiac USG recordings of 94 patients who underwent CPR in the emergency department's critical care area were reviewed. Among these patients, 63 were identified as having received positive inotropic therapy during CPR. Of these, 11 had incomplete observation forms, and 40 lacked either pre- or post-inotropic therapy cardiac USG recordings. Consequently, 12 patients with complete cardiac USG recordings before and after positive inotropic therapy were included in the study. Among the included patients, 5 (42%) were female, and the mean age was 67±16 years. For 10 patients, dopamine and dobutamine were administered at a dose of 20 mcg/kg/min. during CPR, while 2 patients were classified as post-CPR cardiogenic shock and received 10 mcg/kg/min. of dopamine and dobutamine after CPR was completed. Patients in CPA at the initiation of positive inotropic therapy underwent CPR for a max. of an additional 30 minutes.

Of the patients with organized electrical activity on the monitor during CPR but no palpable pulse (Figure 1a-d) 4 (33%) exhibited only valvular motion on cardiac USG imaging obtained before positive inotropic therapy (Video 1a-4a). None of these patients showed significant changes in cardiac activity following inotropic therapy (Video 1b-4b). The general characteristics and CPR details for these patients are presented in Table 1.

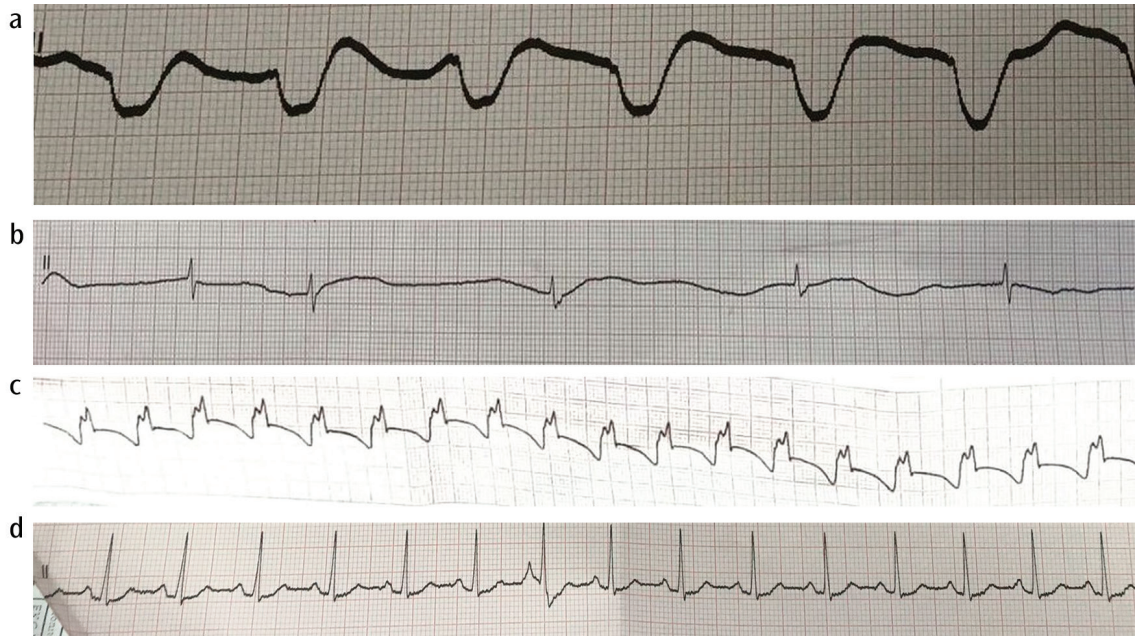

Figure 1 a). PEA rhythm in case 1 with only valvular motion on USG before inotropic therapy. b) Slow AF rhythm in case 2 with only valvular motion on USG before inotropic therapy. c) Wide-QRS SVT rhythm in case 3 with only valvular motion on USG before inotropic therapy. d) PEA rhythm in case 4 with pulmonary thromboembolism and only valvular motion on USG before inotropic therapy

USG: Ultrasound, PEA: Pulseless electrical activity, AF: Atrial fibrillation, SVT: Supraventricular tachycardia

Table 1. General characteristics and CPR data of patients who received positive inotropic therapy at CPR and in whom only valve motion was observed at pre-treatment cardiac USG

Case	Age (y)	♂♀	Clinical presentation	CPR course	Primary cause of CPA	Rhythms monitored in CPR	Cardiac USG (pre-positive inotrope)	Cardiac USG (post-positive inotrope)	Outcome of CPR and 24-hour survival
1	73	♂	This patient with sudden onset, dyspnea and confusion was brought to the hospital in an orthopneic state. CPA occurred 5 minutes after the start of NIMV treatment	At the 20th minute of CPR, a regular rhythm without a pulse was seen on monitor (Figure 1a). At the 20th minute of CPR, a regular rhythm with a wide QRS that did not produce a pulse, was seen on the monitor. Upon detection of cardiac activity at bedside USG, positive inotropes added to the treatment, and CPR continued	Unknown - possible PTE	Asystole -PEA - VF -asystole	Only valve motion (Video 1a)	Decreased valve motion (Video 1b)	ROSC could not be achieved after 30 minute of CPR and the patient was considered ex
2	88	♂	This bedridden patient had aspirated food during feeding. He was found at home with CPA by ambulance crews. He was brought to the hospital with supraglottic airway at the 25th minute of CPR	No pulse could be detected at the first pulse check (27th minute of CPR) in the hospital, but a rhythm compatible with AF with slow ventricular response was observed on the monitor (Figure 1b). A positive inotrope was added to the protocol after cardiac activity was observed at bedside USG and CPR continued	Aspiration pneumonia	Asystole - PEA - Asystole	Only valve motion (Video 2a)	Decreased valve motion (Video 2b)	ROSC could not be achieved after 40 minute of CPR, and the patient was considered ex
3	83	♂	CPA following sudden loss of consciousness at home. Relatives called an ambulance, and the patient was brought to the hospital with ETI at the 16th minute of CPR	After performing CPR for another 6 minutes in the hospital, a wide QRS tachycardic but non-pulsating rhythm was observed on the monitor. This was interpreted as SVT with bundle branch block with old ECGs (Figure 1c). A positive inotrope was added to the protocol after regular cardiac activity was observed at bedside USG, and CPR continued	Unknown	Asystole - PEA - asystole	Only valve motion (Video 3a)	Decreased valve motion (Video 3b)	ROSC could not be achieved after 30 minute of CPR and the patient was considered ex
4	77	♀	This patient, who was bedridden due to previous stroke, was brought to the emergency room due to sudden shortness of breath at home. The patient underwent pulmonary CT angiography after NIMV treatment and experienced CPA minutes later	While the rhythm was present on the monitor, the patient developed respiratory arrest, and no pulse could be detected. The patient was started on the CPR protocol, but there was still no pulse at the first pulse check, although the rhythm was still present on the monitor (Figure 1d). A positive inotrope was added to the treatment because of cardiac activity at bedside USG. Upon detection of PTE at thoracic CT angiography, thrombolytic therapy was started and the CPR protocol was maintained	PTE	PEA - asystole	Only valve motion (Video 4a)	Decreased valve motion (Video 4b)	ROSC could not be achieved after 40 minute of CPR and the patient was considered ex

USG: Ultrasound, CPR: Cardiopulmonary resuscitation, CPA: Cardiopulmonary arrest, PEA: Pulseless electrical activity, CVD: Cerebrovascular disease, NIMV: Non-invasive mechanical ventilation, CT: Computer tomography, AF: Atrial fibrillation, PTE: Pulmonary thromboembolism, VT: Ventricular tachycardia, VF: Ventricular fibrillation, MI: Myocardial infarction, ETI: Endotracheal intubation, GCS: Glasgow Coma Score, ROSC: Return of spontaneous circulation, ECG: Electrocardiogram, SVT: Supraventricular tachycardia

In 6 patients (50%) who exhibited organized electrical activity on the monitor but no palpable pulse during CPR (Figure 2a-g), cardiac USG imaging before positive inotropic therapy revealed valvular motion with varying degrees of myocardial motion (Video 5a-10a). All these patients demonstrated significant improvement in cardiac activity following inotropic therapy (Video 5b-10b); and ROSC was achieved in all cases, allowing CPR to be terminated. However, all these patients experienced recurrent CPA within 24 hours and succumbed in the clinical units where they were admitted. The general characteristics and CPR details of these patients are presented in Table 2. A statistically significant difference was found between the group with only valvular motion and the group with both valvular and myocardial motion in terms of response to positive inotropic therapy (ROSC, p=0.002).

In the remaining two patients, ROSC was achieved during formal CPR, evidenced by the presence of a weak pulse and low blood pressure, in addition to the organized rhythm observed on the monitor (Figure 3a, b). Positive inotropic therapy was initiated after CPR completion. Pre-therapy cardiac USG revealed valvular motion with varying degrees of myocardial motion (Video 11a and 12a). Post-inotropic therapy, the USG showed significant improvement in cardiac activity (Video 11b, 12b). Among these, case 11 achieved 24-hour survival and was discharged without sequelae, while case 12 experienced recurrent CPA but succumbed within 24 hours. The general characteristics and CPR details for patients in cardiogenic shock are presented in Table 3.

The contractile strength comparison of the contractile strength of cardiogenic shock cases (case 11 and case 12) with p-PEA cases (case 6 and case 9) through echocardiographic evaluation, it was observed that their findings were remarkably similar.

The odds of ROSC were 117 times higher in p-PEA patients, with phi coefficient of 0.998, indicating an exceptionally large effect size. Clinically, this strongly supports the importance of identifying myocardial motion via echocardiography during CPR and suggests that p-PEA patients should not be treated identically to PEA patients without myocardial activity. Positive inotropic therapy may have a markedly different and more favorable impact in pseudo-PEA cases (Table 4).

Discussion

Pulse assessment is one of the primary diagnostic tools guiding CPR and is currently performed predominantly through manual palpation, a rudimentary method. However, numerous studies have demonstrated that this technique is practitioner- and patient-dependent, with limited reliability in low-cardiac-output ROSC scenarios and susceptibility to various factors such as cold extremities and obesity [14-17]. For these reasons, although not yet part of routine practice, alternative methods such as carotid USG, femoral USG, and cardiac USG are gaining popularity [11,18]. Even with these instruments, the diagnostic boundaries between PEA, p-PEA, and cardiogenic shock remain unclear. Clinically, these entities exist on a spectrum. According to common understanding, PEA, characterized by a rhythm without any cardiac movement, lies at one end of the spectrum. p-PEA, where rhythm is accompanied by some degree of cardiac activity but no palpable pulse, occupies the middle. At the other end lies cardiogenic shock, characterized by rhythm, cardiac activity, a weak pulse, and low blood pressure [15,19]. However, these broad definitions leave gaps, and no consensus has been established among researchers. For instance, in their study, Wu et al. [18] did not classify patients with only valvular motion observed on cardiac USG into either the PEA or p-PEA groups. Conversely, Devia Jaramillo et al. [20] classified patients with valvular motion alone as p-PEA. Similarly, no clear data exist regarding the threshold at which myocardial contractility transitions from p-PEA to cardiogenic shock. This distinction is typically made using manual pulse palpation, a subjective and potentially unreliable method. Thus, under current definitions, PEA, p-PEA, and even cardiogenic shock are closely related entities with potential clinical overlap [10,21,22]. Furthermore, p-PEA patients with organized rhythm and some degree of myocardial contraction are treated in the same way as asystole patients, who lack rhythm and cardiac activity entirely. To our knowledge, this study is the first to evaluate the differential diagnosis of PEA, p-PEA, and cardiogenic shock, as well as their responses to positive inotropic therapy, through detailed echocardiographic analysis. Among the four patients with only valvular motion observed on cardiac USG, none responded favorably to additional positive inotropic therapy. Conversely, all six patients with valvular motion and varying degrees of myocardial motion demonstrated a positive response to inotropic therapy, achieving ROSC. These findings suggest that patients with only valvular motion should be categorized as PEA and treated accordingly. Meanwhile, patients with both valvular motion and varying degrees of myocardial motion should be classified as p-PEA, and their treatment may benefit from the addition of positive inotropes. Furthermore, carotid and femoral USG could be effective tools for distinguishing between p-PEA and cardiogenic shock in the differential diagnosis.

Importantly, this study does not aim to recommend a specific pharmacological agent, but rather to highlight a subgroup of patients (p-PEA) that may respond to inotropic support, and to stimulate further research in this area.

The most significant secondary finding of this study is the contribution of bedside USG to the diagnosis of patients. particularly in the p-PEA group, where ROSC was achieved with positive inotropic therapy allowing these patients to be accurately diagnosed and provided with specific treatment opportunities.

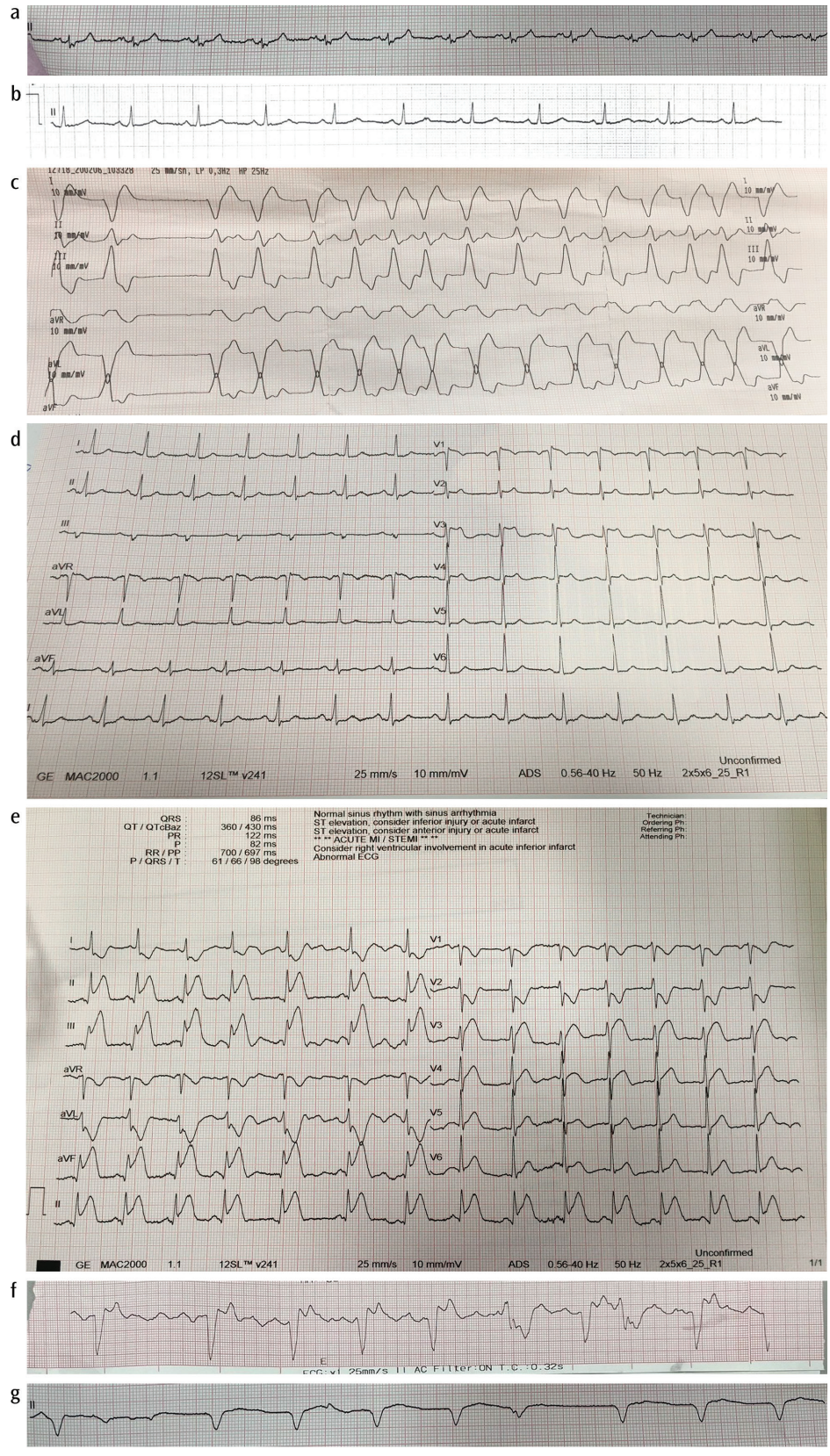

Figure 2. a) PEA rhythm in case 5 with myocardial + valvular motion on USG before inotropic therapy. b) PEA rhythm in case 6 with myocardial + valvular motion on USG before inotropic therapy. c) Wide-QRS tachycardia in case 6 after ROSC. d) PEA rhythm in case 7 with myocardial + valvular motion on USG before inotropic therapy. e) PEA rhythm in case 8 with myocardial + valvular motion on USG before inotropic therapy. f) PEA rhythm in case 9 with myocardial + valvular motion on USG before inotropic therapy. g) PEA rhythm in case 10 with myocardial + valvular motion on USG before inotropic therapy

USG: Ultrasound, PEA: Pulseless electrical activity, QRS:, ROSC: Return of spontaneous circulation

Table 2. General characteristics and CPR data of patients who received positive inotropic therapy at CPR and with myocardial motion in addition to valve motion at pre-treatment cardiac USG

Case	Age (y)	♂	Clinical presentation	CPR course	Primary cause of CPA	Rhythms monitored in CPR	Cardiac USG (pre-positive inotrope)	Cardiac USG (post-positive inotrope)	Outcome of CPR and 24-hour survival
5	76	♀	The patient was admitted to the emergency department with complaints of severe dyspnea and chest pain. The patient was anxious with unstable vital signs and experienced CPA during ETI	At the 12th minute of CPR, cardiac USG was performed on this patient, whose pulse could not be detected despite the rhythm on the monitor (Figure 2a). A positive inotrope was added to the treatment since cardiac activity was observed. At the 16th minute of CPR, the patient's pulse, which was confirmed by carotid USG, was taken, and CPR was stopped. Since thromboembolism was seen in the right structures, thrombolytic treatment was applied	PTE	Asystole - PEA - ROSC with AF with rapid ventricular response	Myocardium and valve motion (Video 5a)	Increase in myocardial and valve motion (Video 5b)	ROSC- ex within 24 hours
6	58	♀	The patient was admitted to an orthopneic state with severe dyspnea. The patient was unable to tolerate NIMV treatment and developed CPA at the 10th minute of admission	Although the rhythm was observed on the monitor (Figure 2b), no pulse was obtained, and CPR was started. Positive inotropic therapy was added to the treatment of this patient with cardiac activity at bedside USG. CPR was terminated when pulse was confirmed by carotid USG at the 12th minute of CPR. The patient stabilized after 1 hour, but experienced CPA again during CT angiography. The patient, who experienced continuous asystole during 20 minutes of CPR, died then	Unknown - possible PTE	PEA - ROSC with tachycardia with wide QRS (Figure 2c)	Myocardium and valve motion (Video 6a)	Increase in myocardial and valve motion (Video 6b)	ROSC- ex within 24 hours

Table 2. Continued

Case	Age (y)	♀♂	Clinical presentation	CPR course	Primary cause of CPA	Rhythms monitored in CPR	Cardiac USG (pre-positive inotrope)	Cardiac USG (post-positive inotrope)	Outcome of CPR and 24-hour survival
7	51	♂	The patient was brought to the emergency room by relatives due to the sudden onset of chest pain and unconsciousness. CPR was started when no pulse or rhythm could be detected at the time of admission. The duration of CPA was unclear	Rhythm was observed on the monitor (Figure 2d) after 8 minutes of CPR, but there was no pulse. A positive inotrope was added to the CPR protocol in this patient with cardiac activity at bedside USG. A pulse confirmed by carotid USG was palpated approximately 6 minutes later. Detailed cardiac ultrasound was performed and aortic dissection was detected (Video 7a). The patient was transferred for surgery	Aortic dissection	Asystole - PEA - ROSC with sinus tachycardia	Myocardium and valve motion (Video 7b)	Increase in myocardial and valve motion (Video 7c)	ROSC - Ex within 24 hours
8	74	♀	This patient was admitted with syncope after severe chest pain. He had a cold, pale appearance and agonal breathing. No pulse could be detected after several minutes, and the CPR protocol was started with advanced airway methods	Rhythm was observed on the monitor (Figure 2e) at the 15th minute of CPR, which was interpreted as asystole. Hemorrhagic pericardial effusion and cardiac activity were detected at cardiac USG performed due to lack of pulse. A pulse which was confirmed by carotid USG, 8 minutes after positive inotrope was added to treatment. The patient was transferred for surgery in an unstablecondition	MI and free wall rupture	Asystole - PEA - ROSC with sinus tachycardia	Myocardium and valve motion (Video 8a)	Increase in myocardial and valve motion (Video 8b)	ROSC- Ex within 24 hours
9	48	♂	This morbidly obese patient underwent 15 minutes of CPR from an external center with a diagnosis of ST elevation MI and was referred as ETI. VF developed while the patient was being received fromy the ambulance team, and the CPR protocol was started	After 10 minutes, a rhythm was seen on the monitor (Figure 2f), but because of doubt about the pulse in this morbidly obese patient, no beat was observed at femoral ultrasound (Video 9a) and CPR continued. Upon detection of cardiac activity at bedside USG, a positive inotrope was added to treatment. ROSC was achieved at the 16th minute, and the patient was transferred to the angiography laboratory	MI	VF- Asystole - PEA - ROSC with sinus tachycardia	Myocardium and valve motion (Video 9b)	Increase in myocardial and valve motion (Video 9c)	ROSC - Ex within 24 hours

Table 2. Continued

Case	Age (y)	♀	Clinical presentation	CPR course	Primary cause of CPA	Rhythms monitored in CPR	Cardiac USG (pre-positive inotrope)	Cardiac USG (post-positive inotrope)	Outcome of CPR and 24-hour survival
10	61	♂	The patient called an ambulance due to severe chest pain that started during exercise. The healthcare team detected elevation in the inferior and lateral leads of the patient's ECG. The patient developed CPA arrest during transport and was brought to the patient with 3 defibrillations and 10 minutes of CPR	After 5 minutes of CPR in the hospital, the rhythm was observed on the monitor (Figure 2g). Since the presence of a pulse was suspected, carotid ultrasound was performed, but no adequate pulse was detected at USG (Video 10a). positive inotrope was added to the CPR protocol due to cardiac activity at bedside USG. After a total of 20 minutes of CPR, ROSC was achieved, and the patient was transferred to the angiography laboratory	MI	VF - asystole-PEA - ROSC with sinus tachycardia	Myocardium and valve motion (Video 10b)	Increase in myocardial and valve motion (Video 10c)	ROSC - Ex within 24 hours

CPR: Cardiopulmonary resuscitation, CPA: Cardiopulmonary arrest, PEA: Pulseless electrical activity, CVD: Cerebrovascular disease, NIMV: Non-invasive mechanical ventilation, CT: Computer tomography, AF: Atrial fibrillation, PTE: Pulmonary thromboembolism, VT: Ventricular tachycardia, VF: Ventricular fibrillation, MI: Myocardial infarction, ETI: Endotracheal intubation, GCS: Glasgow Coma Score, USG: Ultrasound, ROSC: Return of spontaneous circulation ECG: Electrocardiogram, SVT: Supraventricular tachycardia

Bedside USG enabled the diagnosis of pulmonary thromboembolism (PTE) in case 5, aortic dissection in case 7, and free wall rupture in case 8, while also supporting the presumptive diagnosis of PTE in case 6. This study highlights that cardiac USG can be a valuable guide in cases where organized electrical activity is observed on the monitor during CPR, but the pulse is absent. Bedside USG provided these high-mortality patients with a chance for specific treatment, a finding consistent with earlier studies [23].

In this study, carotid and femoral USG, as well as cardiac USG, were utilized alongside manual pulse palpation for the differential diagnosis of p-PEA and cardiogenic shock. This was necessary because distinguishing these conditions based solely on echocardiographic visual assessment proved challenging. For example, no significant difference in contractile strength was observed between cardiogenic shock patients (case 11 and case 12) and p-PEA patients (case 6 and case 9). In morbidly obese patients like case 9, manual pulse detection might be difficult, as previous studies have indicated a high failure rate for manual pulse checks in obese patients [12]. Thus, even echocardiography may be insufficient for distinguishing p-PEA from cardiogenic shock, and combined methods such as carotid USG, femoral USG, or $ETCO_2$ may be necessary. In cases where none of these tools is available and a rhythm is present but no pulse is palpable, adding positive inotropic therapy may be beneficial.

In this study, all p-PEA patients with higher cardiac activity who were treated with positive inotropic agents achieved ROSC, while none of the PEA patients with less cardiac activity did. Väyrynen et al. [15] previously reported that the most significant factors influencing survival in PEA patients were the use of adrenaline and the presence of cardiac activity. These findings are consistent with our results. Mehta and Brady [10] previously reported achieving ROSC in a PEA patient during CPR by administering vasopressin, a vasopressor agent, in addition to standard resuscitation. Similarly, Wenzel et al. [24] demonstrated that vasopressin increased the ROSC rate in PEA patients during CPR. However, these studies did not differentiate between PEA and p-PEA, leaving the group classification of ROSC-achieved patients unclear. Prosen et al. [13] advanced this field by using capnography and USG to distinguish PEA from p-PEA, reporting that vasopressin was more strongly associated with ROSC and survival in p-PEA patients. In our study, consistent with Prosen et al. [13] findings, all p-PEA patients treated with positive inotropes, achieved ROSC, while no PEA patients did. Thus, cardiac activity during CPR may be a key determinant of treatment response. Supporting this, Wu et al. [18] reported that PEA patients with detectable cardiac activity had a 4.09-fold higher likelihood of achieving ROSC, compared to those without. Beyond vasopressin, Myerburg et al. [25] suggested that curcumin might be effective in achieving ROSC in PEA patients. While not all studies differentiated

between PEA and p-PEA, it appears that p-PEA patients with greater myocardial movement may have a higher likelihood of responding to certain drug therapies, warranting further targeted research.

Many experts believe that PEA has been an overlooked entity to date [9]. Extensive studies have been conducted on VF and VT, characterized by pulseless and irregular rhythms, leading to the development of specific therapies like defibrillation [26]. These therapies have significantly improved survival rates in these patients [27]. However, despite also being pulseless, regular rhythms like PEA and p-PEA have no specific therapeutic recommendations and are treated the same as asystole in routine CPR. Given the organized contractility and rhythm, especially in p-PEA patients, the ROSC and survival rates could potentially exceed those of irregular rhythms like VF. However, the superior survival rates of VF are primarily due to the availability of specific treatments like defibrillation. Similarly, the introduction of specific therapies for PEA and p-PEA patients could improve their survival outcomes. This study supports the hypothesis that positive inotropic agents, which are beneficial in cardiogenic shock, may also benefit p-PEA patients, who are in a condition similar to cardiogenic shock. Nevertheless, further research is required before this knowledge can be applied in routine practice

Study Limitations

The primary limitation of this study is its retrospective design, which means that data were accessed retrospectively. However, as these cases were recorded with the intent of being used for future training, detailed patient information and clinical data were systematically documented. This allowed access to most of the data necessary for the study. Another significant limitation is that it was conducted at a single center, which may restrict the generalizability of the findings. Furthermore, due to the retrospective nature and limited sample size, the study groups were not homogenous in terms of age or etiology of cardiac arrest, which may affect the comparability of the groups and limit the strength of conclusions.This missing data represents a significant limitation due to its known impact on survival outcomes.

Conclusion

According to the results of this study, valvular motion and myocardial motion can be used as references for the echocardiographic differentiation of PEA and p-PEA. Based on treatment response and outcomes, the group with valvular motion alone can be classified as PEA, while the group with valvular motion and in addition to varying degrees of myocardial motion can be classified as p-PEA. For the differential diagnosis of cardiogenic shock and p-PEA, carotid or femoral USG may serve as a useful tool. In terms of treatment, adding positive inotropic agents to the routine CPR protocol, may benefit patients with p-PEA, whereas such treatment may be ineffective for those in the PEA group. Further studies are required to validate these findings.

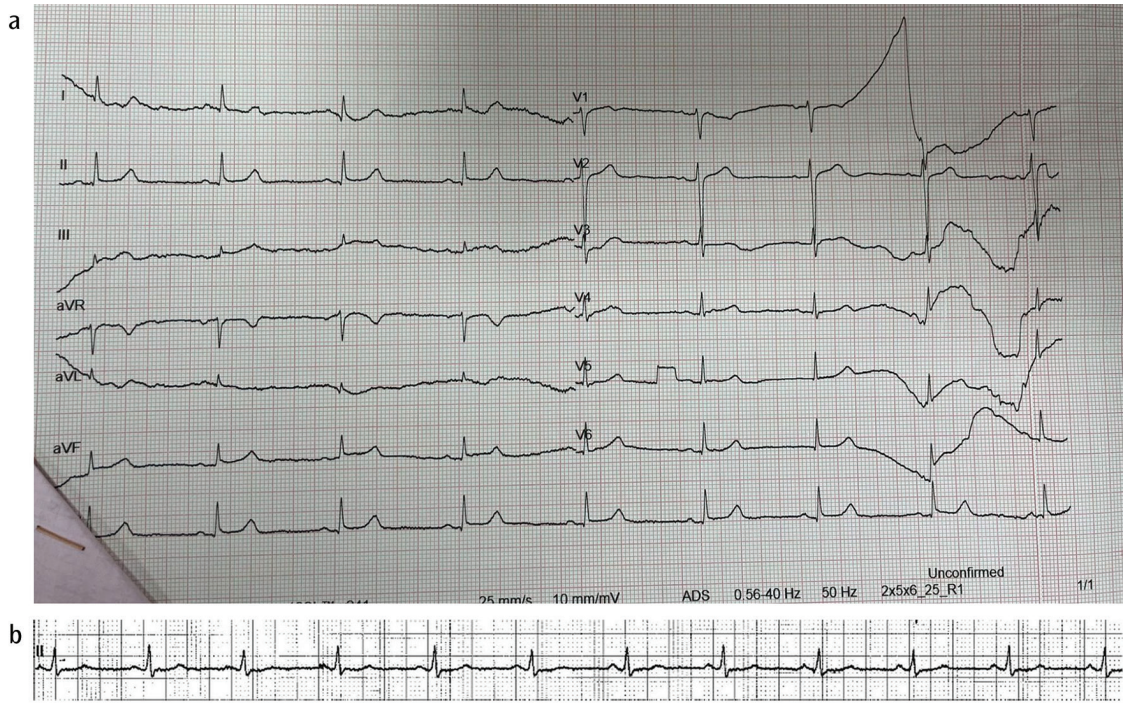

Figure 3. (a) Sinus tachycardia in case 11 with weak pulse and myocardial + valvular motion on USG in cardiogenic shock. b) Sinus rhythm in case 12 with weak pulse and myocardial + valvular motion on USG in cardiogenic shock

USG: Ultrasound

Table 3. General characteristics and CPR data of patients with post-CPR cardiogenic shock

Case	Age (y)	♀	Clinical presentation	CPR course	Primary cause of CPA	Rhythms monitored during CPR	Cardiac USG (pre-positive inotrope)	Cardiac USG (post-positive inotrope)	Outcome of CPR and 24-hour survival
11	27	♂	An ambulance was called by an ambulance by friends of the patient, who lost consciousness after using synthetic cannabinoids. The patient was brought to the emergency room with a GCS of 10 and developed VF 30 minutes later, at which the CPR protocol was initiated	A regular rhythm was observed on the monitor (Figure 3a) after a total of 12 defibrillations and 1.5 hours of CPR. A very weak pulse was detected from the carotid, and the presence of pulse was confirmed by carotid USG. Concomitant arterial blood pressure was 60/40. Cardiac activity was observed at cardiac USG, and the patient was evaluated as being in cardiogenic shock. CPR was terminated, and a positive inotrope was added to the treatment	Intoxication	VF - asystole - ROSC with normal sinus rhythm	Myocardium and valve motion (Video 11a)	Increase in myocardial and valve motion (Video 11b)	ROSC - survived
1 2	78	♀	This obese obese patient was brought to the emergency room by the ambulance team after a sudden onset of syncope at home. ETI was decided on for this unstable patient with deep hypoxia and hypocarbia. Although rhythm was seen on the monitor after ETI, arterial blood pressure could not be measured	CPR was started sine no pulse could be detected. In the third cycle, rhythm Figure 3b was observed, and a weak pulse was palpable. Bedside USG revealed a pulse in the carotid artery, and cardiac activity was present, and a thrombus was detected in the right atrium. The patient's arterial blood pressure was 70/30. CPR was terminated, and positive inotropic agents and thrombolytic therapy were added to the treatment	PTE	PEA - ROSC with normal sinus rhythm	Myocardium and valve motion (Video 12a)	Increase in myocardial and valve motion (Video 12b)	ROSC- ex within 24 hours

CPR: Cardiopulmonary resuscitation, CPA: Cardiopulmonary arrest, PEA: Pulseless electrical activity, CVD: Cerebrovascular disease, NIMV: Non-invasive mechanical ventilation, CT: Computer tomography, AF: Atrial fibrillation, PTE: Pulmonary thromboembolism, VT: Ventricular tachycardia, VF: Ventricular fibrillation, MI: Myocardial infarction, ETI: Endotracheal intubation, GCS: Glasgow Coma Score, ROSC: Return of spontaneous circulation, USG: Ultrasound

Table 4. ROSC Outcomes, effect size and odds ratio for p-PEA vs. PEA

Group	ROSC (+)	ROSC (-)	Total	Odds ratio (95% CI)	p-value	Effect size (φ)
Valvular + myocardial motion (p-PEA)	6	0	6	117.0 (1.35 -10,124.69)	0.002	0.998 (very large)
Valvular motion only (PEA)	0	4	4			

Odds ratios were corrected using the Haldane-Anscombe method. Effect size (phi coefficient) was derived from chi-square approximation with n: 10.0. According to cohen (1988), φ: 0.10 (small), 0.30 (medium), 0.50 (large); values above 0.80 are considered very large.

ROSC: Return of spontaneous circulation, PEA: Pulseless electrical activity, p-PEA: Pseudo-pulseless electrical activity

Ethics

Ethics Committee Approval: Ethical approval for the study was obtained from the İzmir Katip Çelebi University Non-Interventional Clinical Research Ethics Committee on April 15, 2021 (decision number:0214, date: 15.04.2021).

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: N.G.Ç.Y., A.Y., M.G.E., Concept: N.G.Ç.Y., A.Y., M.G.E., S.B., Design: N.G.Ç.Y., M.G.E., E.K., S.B., M.Ş., Data Collection or Processing: A.Y., M.G.E., E.K., S.B., Analysis or Interpretation: M.G.E., E.K., M.Ş., Literature Search: N.G.Ç.Y., A.Y., M.G.E., E.K., M.Ş., Writing: N.G.Ç.Y., A.Y., M.G.E.

Conflict of Interest: No conflict of interest was declared by the authors.

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Video 1a. Pre-positive inotropic therapy
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Video 1b. Post-positive inotropic therapy
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Video 4a. Pre-positive inotropic therapy
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Video 4b. Post-positive inotropic therapy
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Video 5a. Pre-positive inotropic therapy
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Video 5b. Post-positive inotropic therapy
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Video 6a. Pre-positive inotropic therapy
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Video 6b. Post-positive inotropic therapy
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Video 7a. Aortic arch from the suprasternal notch
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Video 7b. Pre-positive inotropic therapy
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Video 7c. Post-positive inotropic therapy
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Video 8a. Pre-positive inotropic therapy
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Video 8b. Post-positive inotropic therapy
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Video 9a. Femoral artery ultrasound
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Video 9c. Post-positive inotropic therapy
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Video 10a. Carotid artery ultrasound
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Video 10b. Pre-positive inotropic therapy
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Video 10c. Post-positive inotropic therapy
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Video 11a. Pre-positive inotropic therapy
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Video 12a. Pre-positive inotropic therapy
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Video 12b. Post-positive inotropic therapy
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