Aortic Pressure during Human Cardiac Arrest*
Identification of Pseudo-Electromechanical Dissociation

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We measured aortic pressure during clinically apparent cardiac electromechanical dissociation (EMD). Patients with pulse pressures were designated as having pseudo-EMD, those without, as having true EMD. Of the 200 patients studied, 54 presented with EMD, and 40 others developed it during resuscitation. Of the 94 with EMD, 39 were found to have pseudo-EMD. We compared the two types of EMD for electrocardiographic duration, return of palpable pulses, and response to standard- and high-dose epinephrine. The mean resting aortic pressure was 18 ± 11 mm Hg in patients with true EMD and 28 ± 11 mm Hg in those with pseudo-EMD. The mean pulse pressure in patients with pseudo-EMD was 6.3 ± 3.5 mm Hg. Patients with pseudo-EMD had a higher proportion of witnessed arrests, higher PaO2, and lower PaCO2 than patients with true EMD. Patients with pseudo-EMD had shorter QR and QRS durations than patients with true EMD. They had a better response to standard- and high-dose epinephrine than patients with true EMD. Many patients diagnosed clinically to be in EMD have mechanical cardiac activity; this should be considered when interpreting the results of cardiac arrest research.

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Electromechanical dissociation (EMD) is a common presentation for cardiopulmonary arrest. It has been defined as "electrical depolarization of the heart without synchronous myocardial fiber shortening and, therefore, without cardiac output," and is diagnosed clinically as "the presence of organized electrical activity on the electrocardiogram (ECG) but without palpable pulses." These criteria may cause to be grouped together as a single clinical entity what are actually heterogeneous pathophysiologic states. The prognosis of EMD has been grim, with rates of return of spontaneous circulation usually below 5 percent. Recently, EMD has been subdivided electrocardiographically, and it has been demonstrated that the subgroups have differing prognoses.

Echocardiography indicates that most patients in EMD may have myocardial wall and valve motion, and there is a preliminary report of arterial pulses in patients diagnosed as having EMD. Perfusion pressures are the driving force for hemodynamics during both spontaneous circulation and cardiac arrest. Echocardiographic studies are unable to measure these, and more than preliminary data are needed to understand hemodynamics during cardiac arrest. The relationship between the ECG and perfusion has also not been studied. The purpose of this study was to measure central aortic pressure in patients during EMD to determine if any had measurable pulse pressures. Patients were classified as those with and those without aortic pulse pressures, and the ECG findings and rates of return of spontaneous circulation in the two groups were compared.

METHODS

We prospectively studied consecutive adult patients suffering normothermic, nontraumatic cardiopulmonary arrest in the prehospital or emergency department setting who remained in arrest after placement of pressure-monitoring catheters. The study was limited to patients who died suddenly. Patients with preexisting multiple organ system disease were excluded. Cardiac arrest was diagnosed by the standard criteria: apnea and the absence of palpable carotid and femoral arterial pulses.

Prehospital care consisted of bag-valve-mask ventilation and external chest compression. Emergency department management was in accordance with advanced cardiac life support (ACLS) guidelines, with the exception of administration of high-dose epinephrine to a minority of patients as part of a concurrent research protocol.

Chest compression and ventilation were performed with a pneumatic device (Thumper; Michigan Instruments, Grand Rapids, Mich). A double-lumen 20-cm 7.5-F catheter (Cook, Bloomington, Ind) was placed in the right atrium through the subclavian vein. The proximal port was used for drug and fluid administration; the

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distal port, for pressure monitoring. A 60-cm 5.8-F catheter (Cook, Bloomington, Ind) was placed in the aortic arch by guide-wire technique through the femoral artery, either percutaneously or by surgical cutdown. The guide wire was inserted as far as necessary to reach the sternal angle. Placement of this catheter is a standard part of therapy during cardiac arrest in our department, and was approved by the Human Rights Committee of Henry Ford Hospital. Correct catheter position was confirmed by the presence of characteristic intrathoracic waveforms and appropriateness of arterial blood gas values (ie, arterial PO2 > 60 mm Hg). When needed, contrast radiography was performed to verify catheter location, and patients with malpositioned catheters were excluded. Previous studies in which all catheter positions were checked radiographically have demonstrated that this method results in correct catheter position in almost all patients. The catheters were connected to transducers (Abbott Systems, North Chicago, III) through a heparinized fluid-fush system that had been set up and calibrated in advance. The resulting signals were amplified (Hewlett-Packard 76205D). Simultaneously, C02 and pressure tracings were recorded (HP 7758 multichannel Hewlett-Packard) throughout resuscitation. Zero pressure was set at the midaxillary line after the patient's arrival. Patients with return of spontaneous circulation before measurement of aortic pressure were excluded. Placement of right atrial and aortic catheters allowed measurement of cardiopulmonary resuscitation (CPR) coronary perfusion pressure, which is the aortic pressure minus the right atrial pressure during the CPR relaxation phase.

Patients whose ECG showed regular, organized depolarizations but who lacked palpable carotid or femoral arterial pulses were designated to be in EMD. During resuscitation, chest compressions were occasionally discontinued for 5 to 10 s. Those patients with regular aortic pulse pressures less than 60 mm Hg that were synchronous with ECG depolarizations during these intervals were designated as having pseudo-EMD. Patients without aortic pulse pressures were defined as being in true EMD. Resting aortic pressure was measured at the ends of the intervals when compressions were discontinued. Return of spontaneous circulation was defined as a systolic blood pressure greater than 90 mm Hg, with palpable carotid or femoral pulses, that lasted more than 2 min.

The QR wave duration of the ECG was defined as the time from initial positive or negative deflection from baseline to return to baseline. The RS wave duration, if an S wave was present, was the time from secondary deflection from baseline to final return. The QRS duration was the sum of the QR and RS durations.

A concurrent study of the effect of high-dose epinephrine on CPR coronary perfusion pressure allowed an assessment of the effects of standard- and high-dose epinephrine on each type of EMD. Under this protocol, each patient first received standard AC1.5, including multiple administrations of 1 mg of epinephrine, before administration of a single right atrial bolus of epinephrine, 0.2 mg/kg of body weight. For each type of EMD we retrospectively compared the proportion of patients with return of spontaneous circulation after standard-dose epinephrine with that after high-dose epinephrine.

Data are reported as mean ± SD. Continuous variables were compared using a Mann-Whitney U test. The difference in the proportion of patients with return of spontaneous circulation and witnessed arrests for the two types of EMD was tested with a chi² statistic. Within each group the difference between the proportions responding to standard-dose and high-dose epinephrine was estimated with a 95 percent bias-corrected bootstrap confidence interval. Because not all patients in whom standard therapy failed had received high-dose epinephrine, we used a conservative test by assuming that patients who did not receive the higher dose would not have had return of spontaneous circulation had they received it. Statistical significance was prospectively set at p<0.05; no corrections were made for multiple inference.

RESULTS

Two hundred patients were studied. The mean age was 67 ± 16 years. Seventy-nine patients had a witnessed cardiac arrest. In these patients the time from cardiac arrest to measurement of aortic pressure was 26 ± 15 min (range, 0 to 74 min). Arterial blood gas measurements for all patients were as follows: PO2, 232 ± 157 mm Hg; PCO2, 35 ± 25 mm Hg; and pH, 7.20 ± 0.27. The coronary perfusion pressure for all patients was 10.0 ± 11.8 mm Hg.

Fifty-four patients presented in EMD, 27 in ventricular fibrillation, and 104 in asystole. In the remaining 15 patients, the initial rhythm was not recorded. Forty patients presented with non-EMD rhythms, but EMD developed at some time during their resuscitation before administration of high-dose epinephrine.

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Thirty-nine patients had pseudo-EMD at some time during their resuscitation before administration of high-dose epinephrine (Fig 1). Characteristics of patients with true EMD and pseudo-EMD are compared in Table 1. Patients with pseudo-EMD had a higher proportion of witnessed arrests, higher PaO₂ values, and lower PaCO₂ values than patients with true EMD. There were no significant differences in total arrest time and arterial pH.

The resting aortic pressure was 18 ± 11 mm Hg in patients with true EMD and 28 ± 11 in those with pseudo-EMD (p = 0.0001). The range of resting aortic pressures in patients with pseudo-EMD was 8 to 58 mm Hg, and the mean aortic pulse pressure was 6.3 ± 3.5 mm Hg (range, 2 to 14 mm Hg). The mean pulse rate of patients with pseudo-EMD was 65 ± 40 beats per minute (range, 15 to 180 beats per minute). Two patients considered clinically to be in EMD cardiac arrest were found to be in normotensive respiratory arrest with systolic blood pressures greater than 90 mm Hg.

The QR and QRS durations of patients with true EMD and pseudo-EMD are compared in Table 2. Patients with pseudo-EMD had shorter QR and QRS durations than patients with true EMD. Few patients in either group had P waves on their lead 2 ECG recordings. There were few patients in true EMD with a secondary deflection in their ECG; therefore, the sample size for comparison of R durations was small and not statistically significant.

Of the 200 patients studied, 58 had return of spontaneous circulation. Among the 94 patients who had EMD at some time during their resuscitation, 41 had return of spontaneous circulation. By comparison, among patients without EMD during resuscitation, only 3 of the 27 patients presenting in ventricular fibrillation and 14 of the 104 patients presenting in asystole had return of spontaneous circulation.

Eight of the 55 patients in true EMD and 14 of the 39 in pseudo-EMD responded to standard therapy, including epinephrine doses of 1 mg, with return of spontaneous circulation (p = 0.016). Six of the 26 patients in true EMD who received high-dose epinephrine had return of spontaneous circulation. Of the 18 patients with pseudo-EMD who received high-dose epinephrine, 14 had return of spontaneous circulation. This was significantly greater than the proportion with return of spontaneous circulation after standard doses (p = 0.003), and was the best outcome among all combinations of EMD type and epinephrine dose (p = 0.0001). All the patients with return of spontaneous circulation remained comatose and died after intensive therapy was withdrawn.

**DISCUSSION**

Our results demonstrate that many patients clinically classified as being in EMD have cardiac activity with pulse pressures and therefore a cardiac output. The belief of some authors that EMD represents electrical activity without associated mechanical activity is not universally correct. These data extend the work of Bocka and associates, who demonstrated that most patients in EMD have myocardial wall and valve motion, and indicate that cardiac arrest is not a uniform pathophysiologic state. Vincent and associates had previously identified similar hemodynamics during EMD, but their patients suffered an in-hospital cardiac arrest, were hemodynamically monitored before cardiac arrest occurred, and were not resuscitated. Since these patients were at the end stage of their disease, they may not represent the same population as out-of-hospital patients who die suddenly.

Our method does not preclude myocardial activity in patients with true EMD. These patients may have had myocardial contraction insufficient to open the aortic valve. However, cardiovascular, as well as neurologic, outcome from cardiac arrest is primarily a function of organ perfusion; therefore, patients without detectable peripheral pulse pressures are dependent solely on perfusion pressures generated by CPR and can be considered clinically to be in true EMD.

The data emphasize the inaccuracy of diagnosis based solely on the ECG and physical examination findings. In many patients, EMD is apparently not cardiac arrest, but rather a state of severe cardiogenic shock. This severe hypotension may propagate a positive feedback loop: hypotension causes decreased coronary perfusion, which impairs myocardial function, resulting in greater hypotension. This vicious circle, and our inability to interrupt it, most likely underlies the poor prognosis of patients in EMD.
Although the minimal perfusion present in patients with pseudo-EMD may be the cause of the better short-term outcomes, another possibility is that pseudo-EMD is a less deranged pathophysiologic state than true cardiac arrest and is more amenable to resuscitation. The younger age and higher coronary perfusion pressures in patients with pseudo-EMD are consistent with this conclusion. Time from arrest to resuscitation is probably the most important determinant of the condition of the myocardium. Although the total time from arrest to pressure measurement for patients with pseudo-EMD was not statistically different from patients with true EMD, these data were collected from bystanders and may be inaccurate. The greater proportion of witnessed arrests in patients with pseudo-EMD is suggestive of shorter arrest times in patients with pseudo-EMD.

Patients with pseudo-EMD were more likely to have return of spontaneous circulation with standard doses of epinephrine than were patients with true EMD, asystole, or even ventricular fibrillation. This is also consistent with a favorable myocardial state. Pseudo-EMD may explain some of the anecdotal reports of human resuscitation with epinephrine dosages that would appear, on the basis of better-controlled animal studies, to be inadequate. Future studies of cardiac arrest should analyze data from patients in EMD separately, since these patients may not be in cardiac arrest.

It would seem that the higher rates of resuscitation for ventricular fibrillation in other studies may be limited to patients defibrillated early in cardiac arrest. In our study, patients in ventricular fibrillation who remained in cardiac arrest long enough to allow measurement of aortic pressure were no more likely to have return of spontaneous circulation than patients presenting in asystole.

The higher rates of resuscitation for patients in pseudo-EMD did not translate to improved long-term survival for patients with pseudo-EMD. Almost all patients with return of spontaneous circulation succumbed to the sequelae of posts ischemic anoxic encephalopathy. This poor outcome demonstrates that pulse pressures in the range measured do not prevent postresuscitation syndrome, especially neurologic injury, and also may provide indirect support for the observation in animal models that "trickle" flow may be worse than no blood flow at all.

Auwerbe and associates, in reviewing a large series of patients, found that patients in EMD who were successfully resuscitated had QRS intervals that were shorter than those without return of spontaneous circulation. A narrow QRS may indicate a better metabolic state of the myocardium. During EMD, the QRS is often wide, with a bizarre waveform morphology. The observation that many patients have narrow, well-formed initial deflections was the basis for analyzing the parts of the QRS. The observation that patients in true EMD and patients in pseudo-EMD differed in their initial but not terminal QRS deflections indicates the importance of the initial deflection. Most of the difference in the total QRS intervals appears to result from prolongation of the initial deflection in patients with true EMD.

Although the total duration of the QRS, and especially the duration of the initial deflection, is shorter in patients with pseudo-EMD, the ECG alone is not a good predictor of myocardial contraction, as there is significant overlap between true EMD and pseudo-EMD (Table 1). However, in our sample, no patient in pseudo-EMD had a QRS interval or initial deflection in the QRS of more than 0.2 s. Others have noted the progressive deterioration in the ECG with time; our study suggests that in some cases this reflected a concurrent but incomplete deterioration in cardiac pump function.

The secondary deflection of the QRS, the RS duration, may not have differed between patients with true EMD and those with pseudo-EMD because patients in true EMD often lacked an Rs wave, making for a small sample size. Many patients in true EMD had wide monophasic QRS complexes.

Although the data concerning high-dose epinephrine were retrospective, they support the conclusion that patients in whom standard dosages failed may respond to high-dose epinephrine. These results are encouraging in that previous studies have failed to demonstrate clear efficacy for any specific therapy for EMD. Since high-dose epinephrine was not given until near the end of resuscitative efforts, it is not surprising that patients did not go on to long-term survival; severe neurologic injury would be a near certainty after such a prolonged ischemic episode.

The pathophysiologic processes that result in pseudo-EMD are probably identical to previously identified causes of impaired cardiac function, albeit to a more extreme degree. Tissue hypoxia, either locally secondary to myocardial ischemia or systemically secondary to respiratory failure, is likely to be the most common cause. So-called secondary EMD, such as occurs in cardiac tamponade, or a primary electrical disturbance, such as ventricular tachycardia, also may initiate the negative feedback loop already described. Numerous intracellular derangements have been theorized as underlying absent or decreased excitation-contraction coupling, but the specific mechanisms are not known. Ischemic myocardium has poor mechanical activity, and pseudo-EMD may represent a state of severe global myocardial ischemia. Whatever the cause, it seems likely that pseudo-EMD is a stage on the pathway to true EMD and asystole. In this respect, the lack of significant
difference in arrest times between patients in true EMD and those in pseudo-EMD is confusing. It may result from inaccuracy in the arrest times of patients with true EMD, since significantly fewer of these patients had witnessed arrests. Occasionally we observed loss of pulse pressures in patients with pseudo-EMD, demonstrating progression to true EMD. In some of these patients, pulse pressures would return with administration of epinephrine.

This study has several limitations. The nature of our emergency medicine system, and the protocol itself, resulted in considerable delay in obtaining data. This is indicated by the distribution of presenting ECG rhythms. The proportion of patients with ventricular fibrillation is lower, and that of asystole is higher, than in other series. In most emergency medical systems, the initial diagnosis of rhythm is obtained by paramedics in the field. Our patients were transported to the hospital by ambulances with only basic life support before an ECG was obtained. In some patients initially in ventricular fibrillation, their rhythm may have decayed to asystole during transport. Patients did not meet inclusion criteria until they had been in cardiac arrest long enough to be transported to the hospital and to undergo placement of aortic arch catheters. Thus, these results apply strictly only to patients in prolonged cardiac arrest. However, there is no reason to expect that pulseless patients would display even limited hemodynamics late in arrest. The opposite would be expected, and it is reasonable to hypothesize that the fraction of patients in pseudo-EMD would have been larger had it been possible to study patients with shorter arrest times. The data on epinephrine and outcome are retrospective and need to be confirmed in a prospective manner.

Although it has been demonstrated that coronary perfusion pressure is a good predictor of outcome during cardiac arrest, the significance of the difference in coronary perfusion pressure in patients with true EMD and those with pseudo-EMD is not clear. Although the aortic-to-right atrial pressure difference during CPR relaxation phase has been defined to be coronary perfusion pressure in the arrested heart. this may not be true during pseudo-EMD. It would seem likely that the spontaneous aortic pulse pressures would contribute to higher aortic relaxation phase pressures and higher coronary perfusion pressures.

The observation that a significant fraction of patients in EMD have myocardial activity makes it important that this subgroup be analyzed separately in future studies of cardiac arrest. This would prevent the mistake of concluding that a therapy was efficacious in cardiac true arrest when its effects may actually be limited to patients with beating hearts.

In summary, our study demonstrates that patients with a clinical diagnosis of EMD are in a spectrum of perfusion states that range from normotension to complete absence of perfusion. Patients with detectable aortic pulse pressures appear to have a different short-term prognosis and response to therapy than patients in true cardiac arrest. Administration of high-dose epinephrine may be considered in the treatment of narrow-complex EMD in hopes of improving what has been a dismal prognosis.

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REFERENCES
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Pediatric Cardiology Board Examination

The American Board of Pediatrics will administer the certifying examination in pediatric cardiology August 11 in three US cities. Registration will extend through January 31. For information, contact the ABP, 111 Silver Cedar Court, Chapel Hill, NC 27514-1651 (919:929-0461).