Clinical and Experimental Studies on Electromechanical Dissociation

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SUMMARY  Electromechanical dissociation (EMD) is the most frequent cause of unsuccessful cardiac resuscitation in critically ill patients. In a clinical study of cardiac arrest, including 54 episodes in 50 fully monitored patients, 14 episodes of ventricular fibrillation were observed and seven were reversed. In the remaining 40 instances, 36 cases of EMD were initially observed. Four patients had asystole. None of the patients with EMD or asystole were successfully resuscitated.

For objective study of EMD and its treatment, we developed an experimental model in which ventricular fibrillation was induced in mechanically ventilated dogs. EMD was predictably observed when, after an interval of 120 seconds, ventricular fibrillation was reversed with an external countershock. Neither metabolic acidosis nor metabolic alkalosis modified the incidence of EMD. A few dogs were pretreated with glucose-insulin-potassium or pharmacologic doses of methylprednisolone, but this did not clearly reduce the incidence of EMD. However, the onset of EMD was delayed when the body temperature of the animal was spontaneously reduced.

THE CESSATION of effective contractions of the heart (cardiac arrest) is usually associated with ventricular fibrillation or with cardiac standstill. Electromechanical dissociation (EMD), in which electrocardiographic complexes persist in the absence of effective cardiac output, has been viewed as an uncommon but usually fatal cause of cardiac arrest. EMD is typically due to a significant reduction in preload, as in exsanguinating hemorrhage or pericardial tamponade, or to a marked increase in afterload, as in instances of massive pulmonary embolism. However, EMD may also reflect severe dysfunction of the cardiac pump due to a variety of mechanical or metabolic causes. It is usually observed as an end stage of cardiac arrest in the form of an agonal rhythm. In contrast to instances of catastrophic cardiac arrest that conform to the criteria of sudden death, we observed that critically ill patients are more likely to have cardiac arrest in which the mechanical function of the heart is disabled, although a viable electrical rhythm persists. This experience prompted our group to undertake both clinical and experimental studies on the incidence, mechanisms and therapeutic options for management of EMD.

Clinical studies included a retrospective comparison of ECGs and arterial pressure in critically ill pa-


Materials and Methods

Clinical

We reviewed the clinical data of 50 patients who have had at least one episode of cardiac arrest during detailed hemodynamic, pulmonary and metabolic monitoring in our critical care ward between October 1975 and December 1978. Patients in whom respiratory arrest preceded cardiac arrest were excluded from the study. The patient group included 27 men and 23 women, ages 46–91 years (median 68 years). Only three of the patients were discharged alive from the critical care ward and one survived hospitalization. Primary diseases included complications of myocardial infarction (28 patients), bacteremia with shock (14 patients), obstructive shock due to pulmonary embolism (four patients) and hypovolemia (four patients).

In each instance, a #7F balloon-tipped pulmonary artery catheter (Swan-Ganz catheter, model 93A-118-7F, Edwards Laboratories) was advanced into the pulmonary artery and a Teflon catheter (Becton, Dickinson and Co.) was introduced into the femoral artery and advanced to the iliac artery. Arterial pressure, pulmonary artery pressure, central venous pressure and ECG were continuously recorded on an eight-channel recorder (model 7878A, Hewlett Packard) by techniques previously described.* Mechanical ventilation had been instituted before cardiac arrest in 43 of the 50 patients and 33 patients had been treated with a vasopressor agent (dopamine).

Cardiac resuscitation was performed in accordance with the standards of the American Heart Association.† The administration of sodium bicarbonate, guided by serial measurements of arterial blood gases, was titrated to maintain the arterial pH at 7.25 units or greater in the absence of respiratory acidosis. A transvenous pacemaker had been inserted before the cardiac arrest for management of atrioventricular block in seven patients and was inserted during the course of cardiac resuscitation in three additional patients.

When systolic arterial pressure was reduced to less than 40 mm Hg and the pressure pulse demonstrated less than 10 mm Hg difference between systolic and diastolic pressure, the patient was regarded as having EMD.

Experimental

Twenty-nine healthy mongrel dogs (18 male and 11 female) that weighed 14–41 kg (mean 24 ± 7 kg) were anesthetized by i.v. injection of 25 mg/kg of pentobarbital. Supplemental aliquots of 25–100 mg of pentobarbital were administered at intervals of 1–4 hours to maintain anesthesia during the experiment. Standard electrocardiographic leads were attached to the four limbs. After endotracheal intubation, dogs were mechanically ventilated on control mode with a Bennett MA-1 ventilator at a frequency of 12 breaths per minute. Oxygen concentration of the inspired gas was 50%. The Radford nomogram* was used for initial determination of the tidal volume and was adjusted to maintain the arterial carbon dioxide tension between 35 and 42 mm Hg.

A balloon-tipped, triple-lumen, thermistor-equipped pulmonary artery catheter (Swan-Ganz catheter 702-027-7F, Edwards Laboratories) was surgically inserted into the right femoral vein and flow-directed into the pulmonary artery until a pulmonary artery occlusive (wedge) pressure was obtained. The balloon was then deflated. The thermistor was used for measurement of blood temperature in the pulmonary artery. A 14-gauge polyethylene catheter (Intracath Deseret) was advanced from the right femoral artery into the abdominal aorta. A #7F Swan-Ganz catheter (model 93A-118-7F, Edwards Laboratories) of which the distal 50 cm were amputated, was surgically advanced from the right external jugular vein into the right ventricle. One port was used for right ventricular pressure measurement and the second port as a route for a flexible wire (Teflon-coated 0.035-inch wire guide, Cook, Inc.). The wire was connected to the V lead of the ECG and slowly advanced until a typical injury current indicating the endocardial contact of the wire was recorded. The experimental arrangement is shown in figure 1. One pole of an AC fibrillator (model 2039A, Medtronic) was connected to the right ventricular wire and the other to a conductive needle in the anterior chest of the dog. An electrical current of 0.2–2 mA (mean 0.9 ± 0.6 mA) was then applied to the flexible wire for induction of ventricular fibrillation. The alternating current was discontinued after arterial pressure pulses disappeared and when the mean arterial pressure decreased to less than 50 mm Hg. No mechanical cardiac compression was attempted during cardiac arrest.

After a predetermined interval, an external counter-shock was delivered with a standard DC defibrillator (Corbin-Farnsworth Inc.). Countershocks of 40, 80, 200 and 320 J were successively delivered until ventricular fibrillation was reversed.

Aortic pressure, pulmonary artery pressure, pulmonary artery occlusive (wedge) pressure and right ventricular pressure were measured with electrically isolated transducers (model 4-327-1, Bell and Howell). Appropriately calibrated pressures and ECG lead 2 were continuously recorded on a four-channel recorder (Sanborn model 7700, Hewlett Packard). The zero pressure reference was at the midchest position of the dog, in the third intercostal space. The pulmonary artery occlusive pressure, cardiac output and pulmonary artery blood temperature were measured before ventricular fibrillation and at intervals of 1, 4, 9, 14 and 19 minutes after defibrillation. Cardiac output was measured in duplicate by thermodilution using a cardiac output computer (model 9510, Edwards Laboratories) and recorded on a strip-chart recorder. When the morphology of the thermodilution curve indicated faulty injection or mixing of cold
(3°C) indicator the procedure was repeated. Pulmonary and systemic vascular resistances were calculated with standard formulas.4

Blood samples were simultaneously withdrawn from the aorta and the pulmonary artery before ventricular fibrillation and 5 and 20 minutes after defibrillation for measurements of arterial and mixed venous blood gases, oxygen saturations, hematocrit, hemoglobin concentration, electrolytes and lactate concentrations and plasma colloid osmotic pressure. Blood gases were measured by an automated analyzer (model IL 813, Instrumentation Laboratory) and hemoglobin concentration and oxygen saturation by a cooximeter (model IL 282, Instrumentation Laboratory). Colloid osmotic pressure was measured by an oncometer (model IL 186, Instrumentation Laboratory) and the blood lactate concentration by a semi-automated method developed in our laboratory.7 Oxygen consumption was computed from the cardiac output and the difference between oxygen contents of blood sampled simultaneously from the aorta and the pulmonary artery.

Five groups of dogs were studied (table 1). Control studies were performed on eight dogs. (group 1). In five dogs, after initial hemodynamic and metabolic measurements had been completed, a 0.15-M solution of hydrochloric acid (HCl) was intravenously infused over an interval of approximately 1 hour until arterial pH had been reduced to 7.15 (group 2). In five dogs, a solution of 0.6 M sodium bicarbonate was administered over an equivalent period of time until arterial pH was increased to 7.60 (group 3).

In five dogs, a solution of dextrose 50% in water with 103 units of regular insulin and 210 mEq of KCl per liter (GIK) was infused into a centeal vein at a rate of 1.5 ml/kg/hour according to the protocol of Maroko et al.8 This “polarizing” solution was administered over an interval of 2 hours before fibrillation and continued throughout the remainder of the experiment (group 4).

In six dogs, a single dose of 30 mg/kg of methylprednisolone (Solu-Medrol) was administered as an i.v. bolus injection over 30 seconds, 1 hour before ventricular fibrillation (group 5).

In the initial design of the study and after preliminary trials, we sought to differentiate between

TABLE 1.  Electromechanical Dissociation After 2 Minutes of Ventricular Fibrillation in 29 Dogs

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Pretreatment</th>
<th>CV Infusion</th>
<th>Dose</th>
<th>EMD at 2 min</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8</td>
<td>Control</td>
<td>—</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>2</td>
<td>5</td>
<td>0.15 M HCl</td>
<td>to pHa 7.15 (7.14 ± 0.02)</td>
<td>424 ± 89 ml</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>5</td>
<td>0.6 M NaHCO₃</td>
<td>to pHa 7.60 (7.60 ± 0.01)</td>
<td>392 ± 38 ml</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>5</td>
<td>GIK</td>
<td>D 50/W + 103 U Ins. + 210 mEq KCl/l</td>
<td>1.5 ml/kg/hour</td>
<td>3</td>
</tr>
<tr>
<td>5</td>
<td>6</td>
<td>Methylprednisolone</td>
<td>Bolus (30 seconds)</td>
<td>30 mg/kg</td>
<td>3</td>
</tr>
</tbody>
</table>

Abbreviations: EMD = electromechanical dissociation; CV = cardiovascular; GIK = glucose-insulin-potassium; pHa = arterial blood pH.
three intervals: less than 60 seconds, less than 120 seconds or more than 120 seconds of ventricular fibrillation. In addition, three of the animal groups were defibrillated at 30 seconds and two at 90 seconds. All groups were fibrillated and fibrillation was continued for 30 seconds (group 1, 2 and 3), 60 seconds (all groups), 90 seconds (groups 4 and 5) and 120, 150 and 180 seconds (all groups) before defibrillation.

In 13 dogs in EMD, we attempted to restore effective mechanical systole by intraventricular injection of atropine, 0.5 mg (three dogs), isoproterenol, 1 mg (one dog), epinephrine, 1 mg (five dogs), and calcium chloride, 1 g (four dogs).

At the end of the experiments, an autopsy was performed and the position of the flexible wire in the right ventricle was recorded together with gross pathologic changes which were present on examination of the heart and lungs.

The statistical analyses were performed using the t test for paired and unpaired data.

Results

Clinical Study

There were 54 episodes of cardiac arrest in 50 critically ill patients (table 2). Cardiac resuscitation with restoration of effective myocardial contraction was achieved in only seven (13%) instances. Each of these seven patients had ventricular fibrillation that was reversed after external defibrillation and i.v. injection of lidocaine.

Among the 47 patients who died, cardiac arrest was due to EMD in 36 patients (77%), ventricular fibrillation in seven (15%) and asystole in four (9%). The electrocardiographic rhythms documented in the 36 patients at the onset of EMD are summarized in table 3. Though the rhythm was initially from a supraventricular focus during EMD in a majority of patients, the incidence of atrioventricular block and subsequently idioventricular rhythm increased in the later stages of attempted resuscitation. We observed no effective arterial pulsation after treatment with sodium bicarbonate, atropine or adrenergic amines. Transvenous cardiac pacing, initiated during cardiac resuscitation in three patients, failed to reverse EMD.

In two of the four patients who presented with asystole, we observed EMD with either sinus rhythm or complete atrioventricular block. In the seven fatal cases due to ventricular fibrillation, successful defibrillation was, in each instance, followed by EMD, including junctional rhythm in three patients, atrioventricular block in three patients, and idioventricular rhythm in one patient. Therefore, each of the 14 patients in ventricular fibrillation was defibrillated, but seven developed fatal EMD.

Experimental Study

Ventricular Fibrillation Followed by Hemodynamic Recovery

When mechanical systole was restored after defibrillation, the hemodynamic course was similar for the five groups of dogs. After onset of ventricular fibrillation, arterial pressure rapidly declined to expected closing pressures (figs. 2 and 3). Right ventricular and pulmonary artery pressure increased mildly to levels corresponding to that of the closing pressure.

After defibrillation, right ventricular pressure, pulmonary artery pressure, pulmonary artery occlusive (wedge) pressure and aortic pressure increased (fig. 3). However, the severity of this hypertensive response was variable. Within 1 minute after defibrillation, a significant increase in stroke volume was associated with bradycardia, which explained why cardiac output was only moderately increased (fig. 3).
Within 10 minutes after defibrillation, the hemodynamic status had returned to that observed during the control period. There was no significant decrease in arterial pH and almost no increase in arterial blood lactate concentration throughout the experiments (table 4). Oxygen consumption did not change significantly when measured at 5 and 20 minutes after reversal of ventricular fibrillation.

**Effect of Pretreatment**

After the infusion of hydrochloric acid or sodium bicarbonate, cardiac output was increased and systemic vascular resistance was reduced. (table 5). After bicarbonate administration, we observed moderate lactacidemia, which persisted throughout the course of observation (table 4). Increases in serum osmolality were related to infusion of hypertonic sodium bicarbonate with an average increase in sodium concentration from 150 ± 2 to 169 ± 2 mEq/l (p < 0.001) after the infusion (table 4). Arterial carbon dioxide tension increased transiently after bicarbonate, from 36 ± 1 to 43 ± 1 mm Hg (p < 0.01).

During the 2-hour interval of GIK infusion that preceded ventricular fibrillation, cardiac output progressively decreased and systemic vascular resistance increased (table 5). Serum osmolality and potassium concentration were unaltered.

Immediately after injection of 30 mg/kg of methylprednisolone, we observed a marked decrease in arterial pressure and systemic vascular resistance (table 5). Heart rate was significantly reduced, although stroke volume increased. Cardiac output was unchanged. These hemodynamic effects were transient...
TABLE 4. Arterial Blood Gases, Plasma Osmolality, Lactate Concentration and Oxygen Consumption During the Experiments in the Three First Groups of Dogs

<table>
<thead>
<tr>
<th></th>
<th>Fibrillation (duration)</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>30 seconds</td>
<td>60 seconds</td>
<td>120 seconds</td>
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<tr>
<td></td>
<td></td>
<td>5 min after DF</td>
<td>20 min after DF</td>
<td>5 min after DF</td>
<td>20 min after DF</td>
<td>5 min after DF (EMD)</td>
<td></td>
</tr>
<tr>
<td><strong>Control (eight dogs)</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>pHa (U)</td>
<td>7.35 ± 0.01</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>7.50 ± 0.05</td>
<td></td>
</tr>
<tr>
<td>PaCO₂ (mm Hg)</td>
<td>39 ± 2</td>
<td>40 ± 2</td>
<td>39 ± 2</td>
<td>37 ± 2</td>
<td>39 ± 2</td>
<td>20 ± 2</td>
<td></td>
</tr>
<tr>
<td>HCO₃ (mEq/l)</td>
<td>22.6 ± 0.8</td>
<td>20.8 ± 0.4</td>
<td>20.0 ± 0.4</td>
<td>18.6 ± 0.6</td>
<td>19.5 ± 0.5</td>
<td>17.2 ± 1.1</td>
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</tr>
<tr>
<td>P. osmol (mosm/l)</td>
<td>320 ± 4</td>
<td>319 ± 3</td>
<td>321 ± 4</td>
<td>320 ± 6</td>
<td>320 ± 5</td>
<td>319 ± 7</td>
<td></td>
</tr>
<tr>
<td>Lactate (mmol/l)</td>
<td>2.0 ± 0.1</td>
<td>2.5 ± 0.2</td>
<td>2.1 ± 0.2</td>
<td>2.1 ± 0.1</td>
<td>2.1 ± 0.2</td>
<td>2.5 ± 0.3</td>
<td></td>
</tr>
<tr>
<td>VO₂ (ml/min)</td>
<td>129 ± 14</td>
<td>122 ± 28</td>
<td>138 ± 18</td>
<td>159 ± 24</td>
<td>134 ± 18</td>
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<tr>
<td><strong>Alkalemia (five dogs)</strong></td>
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</tr>
<tr>
<td>pHa (U)</td>
<td>7.37 ± 0.02</td>
<td>7.60 ± 0.01</td>
<td>7.51 ± 0.02</td>
<td>7.54 ± 0.03</td>
<td>7.50 ± 0.02</td>
<td>7.51 ± 0.02</td>
<td>7.80 ± 0.09*</td>
</tr>
<tr>
<td>PaCO₂ (mm Hg)</td>
<td>36 ± 1</td>
<td>43 ± 1</td>
<td>42 ± 2</td>
<td>45 ± 2</td>
<td>41 ± 2</td>
<td>19 ± 3</td>
<td></td>
</tr>
<tr>
<td>HCO₃ (mEq/l)</td>
<td>21.2 ± 0.7</td>
<td>43.4 ± 1.9</td>
<td>35.3 ± 2.2</td>
<td>38.2 ± 3.8</td>
<td>35.5 ± 1.1</td>
<td>33.7 ± 1.9</td>
<td>28.9 ± 1.9</td>
</tr>
<tr>
<td>P. osmol (mosm/l)</td>
<td>320 ± 5</td>
<td>339 ± 9</td>
<td>339 ± 5</td>
<td>339 ± 6</td>
<td>340 ± 11</td>
<td>325 ± 3</td>
<td></td>
</tr>
<tr>
<td>Lactate (mmol/l)</td>
<td>2.6 ± 0.1</td>
<td>3.2 ± 0.3</td>
<td>3.8 ± 0.3</td>
<td>3.9 ± 0.3</td>
<td>4.3 ± 0.2</td>
<td>6.0 ± 0.1</td>
<td></td>
</tr>
<tr>
<td>VO₂ (ml/min)</td>
<td>118 ± 10</td>
<td>154 ± 17</td>
<td>153 ± 21</td>
<td>144 ± 19</td>
<td>136 ± 19</td>
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</tr>
<tr>
<td><strong>Acidemia (five dogs)</strong></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>pHa (U)</td>
<td>7.35 ± 0.02</td>
<td>7.14 ± 0.02</td>
<td>7.15 ± 0.01</td>
<td>7.16 ± 0.01</td>
<td>7.13 ± 0.01</td>
<td>7.14 ± 0.01</td>
<td>7.28 ± 0.05</td>
</tr>
<tr>
<td>PaCO₂ (mm Hg)</td>
<td>40 ± 1</td>
<td>41 ± 2</td>
<td>40 ± 2</td>
<td>41 ± 2</td>
<td>40 ± 2</td>
<td>29 ± 3</td>
<td></td>
</tr>
<tr>
<td>HCO₃ (mEq/l)</td>
<td>22.0 ± 1.1</td>
<td>13.2 ± 0.8</td>
<td>15.1 ± 0.3</td>
<td>14.0 ± 0.3</td>
<td>13.6 ± 0.4</td>
<td>13.6 ± 0.8</td>
<td>13.4 ± 0.9</td>
</tr>
<tr>
<td>P. osmol (mosm/l)</td>
<td>326 ± 3</td>
<td>325 ± 3</td>
<td>309 ± 4</td>
<td>306 ± 5</td>
<td>308 ± 4</td>
<td>309 ± 4</td>
<td>307 ± 7</td>
</tr>
<tr>
<td>Lactate (mmol/l)</td>
<td>2.3 ± 0.2</td>
<td>2.4 ± 0.2</td>
<td>2.4 ± 0.2</td>
<td>2.4 ± 0.2</td>
<td>2.3 ± 0.2</td>
<td>2.5 ± 0.2</td>
<td></td>
</tr>
<tr>
<td>VO₂ (ml/min)</td>
<td>130 ± 20</td>
<td>141 ± 15</td>
<td>127 ± 19</td>
<td>123 ± 18</td>
<td>142 ± 17</td>
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</tr>
</tbody>
</table>

*Four dogs. One alkalemic dog who did not develop EMD after 120 seconds of ventricular fibrillation was excluded.

Abbreviations: pHa = arterial blood pH; PaCO₂ = arterial carbon dioxide tension; P = plasma; VO₂ = oxygen consumption; DF = defibrillation.

and spontaneously reversed within 10 minutes after the injection (table 5).

Electromechanical Dissociation

EMD occurred only after ventricular fibrillation persisted for 60 seconds or longer. There was no predictable relationship between the electrical power required for defibrillation and the appearance of EMD. During EMD, right ventricular pressure, pulmonary artery pressure, pulmonary artery occlusive (wedge) pressure and aortic pressure all remained comparable at levels that represented closing pressures (fig. 4). In the absence of precordial compression, electrocardiographic activity persisted for more than 20 minutes in each dog. The rhythms observed at the onset of EMD are summarized in table 3. The initial rhythms after defibrillation were supraventricular in the majority of cases. Eighteen of the 29 dogs had sinus or junctional tachycardia with a ventricular rate of more than 100 beats/min. With prolongation of EMD, the heart rate slowed and 18 dogs developed complete atrioventricular block. Attempts to restore effective mechanical systole by the right ventricular injection of atropine 0.5 mg (three dogs), isoproterenol 1 mg (one dog), epinephrine 1 mg (five dogs) and calcium chloride, 1 g (four dogs) failed to reverse EMD.

Incidently observed during EMD was a significant increase in arterial pH associated with hypopcapnea and attributed to the continuation of the ventilation in the absence of perfusion of the lungs (table 4). Blood lactate concentration increased very little except for the bicarbonate-treated dogs. Ionized calcium concentrations were determined in the arterial blood of five dogs and ranged from 2.0–3.0 mEq/l; it was unaltered after ventricular fibrillation and defibrillation.

Time of Onset of EMD in Relationship to Pretreatment

Group 1. The eight control dogs had EMD after 120 seconds of ventricular fibrillation (table 2). In a ninth control dog, in a corollary experiment, ventricular fibrillation was induced for 120 seconds to exclude the possible adverse effects of multiple episodes of ventricular fibrillation. Once again, EMD occurred after 120 seconds of ventricular fibrillation.
Group 2. EMD occurred after 120 seconds of fibrillation in each of the dogs pretreated with hydrochloric acid.

Group 3. EMD was observed in four of the five alkalemic dogs after 120 seconds of fibrillation. In one dog, the onset of EMD was delayed until the duration of ventricular fibrillation was prolonged to 150 seconds.

Group 4. Of the five dogs pretreated with GIK, three developed EMD after 90 seconds of ventricular fibrillation. In the two remaining dogs, EMD occurred after 150 and 180 seconds.

Group 5. EMD was observed after 120 seconds of fibrillation in three of the six dogs pretreated with methylprednisolone. EMD occurred after 150 seconds in two dogs and after 180 seconds in one dog.

Thus, EMD was observed after 90–120 seconds of ventricular fibrillation in 23 of the 29 dogs.

Influence of Core Temperature

We related the time of onset of EMD to the spontaneous blood temperature of the dogs. The core temperature of the dogs in which EMD occurred after 90–120 seconds of fibrillation averaged 37.4 ± 0.3°C, whereas it was 34.4 ± 0.8°C in instances in which EMD was delayed for more than 120 seconds after onset of defibrillation (p < 0.001) (fig. 5). Therefore, the delay in the onset of EMD in six pretreated dogs appeared to be more closely related to the spontaneously lower blood temperature of these dogs and not to the pharmacologic interventions.

Autopsy

The autopsy was performed after slow (< 40 beats/min), wide (> 200 msec) and small (< 5 mV) idioventricular complexes (agonal rhythm) were recorded. Usually, the right atrium was still beating, but there was no mechanical contraction of the ventricles. No dog had evidence of pericardial tamponade and their lungs were grossly normal. The general appearance and consistency of the heart were unaltered except for a small area of purpura at the site of the endocardial contact of the stimulating wire.

Discussion

Turner,9 in his review of 45 patients (which antedated continuous ECG monitoring techniques), reported that ventricular fibrillation accounted for only 20% of the preterminal events. More recently, Raizes et al.10 observed that EMD accounted for 68% of inpatient monitored sudden deaths due to myocardial infarction. In the present study of 50 critically ill patients, 36 of the 54 episodes of cardiac arrests (67%) were due to EMD and all were fatal. Moreover, EMD or asystole were the preterminal events in each patient, even when ventricular fibrillation had initiated the cardiac arrest. These data therefore indicate that unsuccessful cardiac resuscitation is not usually caused by failure to restore electrical systole. In most
critically ill patients, it is due to contractile failure of the heart.

We therefore searched for a model in which the development of EMD could be predicted. Investigators have used toxicologic techniques, including the administration of calcium-free solutions, bivalent cations or various organic compounds in isolated hearts or open-chest animals, to study EMD. We selected the model of induced electrical fibrillation in a closed-chest dog to simulate more closely the clinical presentation of cardiac arrest. The initial rhythms recorded after onset of EMD after defibrillation were similar to those in critically ill patients.

Our observations on the sequence of hemodynamic and metabolic effects of ventricular fibrillation and successful defibrillation are in agreement with those of Pansegrau and Abboud. Transient increases in left-sided preload and afterload were associated with equivalent increases in pulmonary artery pressure and right ventricular pressure. However, these hemodynamic effects had entirely subsided within 10 minutes after defibrillation. Remarkably, there was no significant change in arterial blood gases and arterial blood lactate in these mechanically ventilated dogs even in the absence of precordial compression. The onset of EMD after only brief episodes of ventricular fibrillation is not common clinically. Although we have not ruled out the deleterious effects on the myocardium of repeated episodes of ventricular fibrillation and defibrillation, in one control dog there was no difference noted and EMD occurred after a single 2-minute episode of ventricular fibrillation. Hypoxemia was excluded by serial blood gas determinations during mechanical ventilation on control mode. No
significant changes in electrolytes or serum osmolarity were observed. However, the possibility that continued ventilation during EMD with accompanying hypocarbia may have played a role cannot be excluded. The possibility that the anesthetic agent was a significant factor must also be considered. However, pentobarbital has been frequently used in other experiments on ventricular fibrillation without a significant incidence of EMD. Kuhn et al.14 noted hemodynamic recovery after periods of ventricular fibrillation of more than 15 minutes in dogs under comparable pentobarbital anesthesia. In contrast to Kuhn’s model, we did not institute external cardiac massage after ventricular defibrillation, and this would best explain the early development of EMD in our model.

EMD is viewed as a result of absent coronary perfusion and myocardial ischemia. When ischemia is induced in isolated myocardium, decreases in intracellular creatine phosphate and adenosine triphosphate are observed and have been incriminated in the development of EMD.12, 18

We investigated the influence of arterial pH on the onset of EMD, because the administration of sodium bicarbonate constitutes one of the initial pharmacologic interventions during cardiac resuscitation.4 Neither acidemia nor alkalemia altered the time of onset of EMD. However, protective effects of acidosis during myocardial hypoxia were reported by Bing et al.16 and Bercot et al.17 Interestingly, a persistently higher concentration of blood lactate was observed after pretreatment with sodium bicarbonate, confirming the observations of Tobin.18

Maroko et al.9 observed that after experimental coronary occlusion with infusion of GIK, the size of the myocardial infarct was reduced. Improved subendocardial perfusion after anoxic cardiac arrest was observed by Hicks et al.19 when a GIK solution was infused before, during or after cardiopulmonary bypass. In our studies, three of five dogs developed EMD at the end of 90 seconds of ventricular fibrillation. We could not demonstrate statistically significant reductions in the incidence of EMD after pretreatment with polarizing solution. Furthermore, there was no corresponding control data for 90 seconds of fibrillation, so we cannot exclude the possibility that pretreatment with GIK could have actually increased the incidence of EMD after shorter periods of fibrillation. Pharmacologic doses of methylprednisolone are widely used before or during cardiopulmonary bypass or before cardiac transplantation on the assumption that these minimize the injury to myocardium after anoxic cardiac arrest.19-21 Stabilization of cellular membranes and decreased capillary permeability have been suggested as possible mechanisms for this beneficial action of the corticosteroids.19-21 The injection of 30 mg/kg of methylprednisolone 1 hour before ventricular fibrillation did not significantly delay the incidence of EMD, with three of the six dogs developing EMD after 120 seconds of fibrillation, two dogs developing EMD after 150 seconds, and 1 dog after 180 seconds of fibrillation. Without control data for 90 seconds of fibrillation, it is possible that a beneficial effect of steroids after 90 seconds of fibrillation would not have been detected.

A spontaneously lower core temperature in six dogs correlated with a delay in the onset of EMD. This fortuitous observation is consistent with findings in other studies. Myocardial oxygen consumption is decreased during hypothermia.22 In isolated rat hearts, Hearse and Stewart23 observed that hypothermia decreased the myocardial depletion of adenosine triphosphate and creatine phosphate and improved cardiac output after elective ventricular fibrillation. Both systemic and local hypothermia have been widely used along with cardioplegia in patients during cardiopulmonary bypass to minimize ischemic injury with open heart surgery.24 Abendschein et al.25 observed in dogs that hypothermia decreased injury after coronary ligation. However, the fibrillation threshold is decreased when body temperature declines, especially to levels of less than 31°C.26

The pathophysiology of EMD is unknown. It may represent a failure of myocardial contractility resulting from the depletion of myocardial high energy phosphate compounds. Thus, myocardial preservation techniques could preserve myocardial energy stores and prevent EMD. Accordingly, moderate hypothermia may be useful for reducing the incidence of EMD.

The numbers of animals in each subgroup of this study are small and all conclusions are necessarily tentative. Nevertheless, the model developed has potential. Future studies of methods of acutely lowering core temperature in vivo are anticipated to investigate the mechanisms and metabolic determinants of EMD. The animal model developed in this investigation may also be used to investigate other interventions that could rapidly decrease myocardial oxygen requirements and preserve myocardial contractile function, with a view to prevention or reversal of EMD.

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