CPR-Dependent Consciousness: Evidence for Cardiac Compression Causing Forward Flow

We present the case of a patient with cardiac arrest and resuscitation in whom, consistent with direct cardiac compression, large aortic-to-right atrial systolic pressure gradients occurred. Forward blood flow during CPR was sufficient for the patient to maintain consciousness. Although aortic-to-right atrial diastolic gradients adequate to maintain coronary perfusion in experimental models were generated, in our patient, cardiac function could not be restored and the resuscitation was ultimately unsuccessful. [Lewinter JR, Carden DL, Nowak RM, Enriquez E, Martin GB: CPR-dependent consciousness: Evidence for cardiac compression causing forward flow Ann Emerg Med October 1989;18:1111-1115.]

INTRODUCTION

The mechanism of blood flow during closed-chest CPR has been the subject of controversy since the technique was introduced in 1960. Direct cardiac compression, providing selective increases in ventricular pressures, was initially believed to be the mechanism of forward flow (cardiac pump mechanism). However, early investigations with invasive monitoring during CPR revealed equivalent increases in all vascular pressures. It was shown that cardiac output could be generated by a pressure gradient between the intrathoracic and extrathoracic compartments (thoracic pump mechanism). Current CPR research is defining the relative contributions of each mechanism with the aim of refining CPR techniques to optimize coronary and cerebral blood flow.

We present a case of a patient with cardiac arrest and resuscitation in whom large aortic (Ao)-to-right atrial (RA) pressure gradients were recorded during mechanical CPR. The patient maintained consciousness only when CPR was in progress. The Ao-RA systolic pressure gradients suggest that the cardiac pump mechanism was operative in this case and was responsible for sufficient forward flow to maintain consciousness.

CASE REPORT

A 60-year-old woman presented to the emergency department with a three-hour history of retrosternal chest pain unrelieved by 12 sublingual nitroglycerin tablets. The pain radiated to both shoulders and was associated with marked shortness of breath and vomiting. Medical history was significant for a myocardial infarction two years before presentation. Current medications included topical nitrates, oral nifedipine, and sublingual nitrates as needed.

On admission, the patient's blood pressure was 71/55 mm Hg, pulse, 84, respirations, 40, and rectal temperature, 37.0 C. The patient was a thin, profusely diaphoretic woman who was alert but anxious. The cardiopulmonary examination revealed a thready, regular radial pulse, jugular venous distention to the angle of the mandible at 90 degrees, and rales to the midscapulae. Cardiac sounds were obscured by the pulmonary findings; however, no gross murmurs or rubs were appreciated. There was no peripheral edema.

The patient was placed on a cardiac monitor. A peripheral IV catheter was inserted, and a 5% dextrose in water infusion was begun. Oxygen was administered through a nonrebreather mask at 15 L/min, and a dopamine infusion at 5 µg/kg/min was started. An ECG was obtained (Figure 1), and

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Received for publication August 25, 1988.
Revision received February 27, 1989.
Accepted for publication June 7, 1989.

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FIGURE 1. Twelve-lead ECG obtained on admission to the ED.

FIGURE 2. Ao, RA, and Ao – RA pressure tracings, without CPR, obtained shortly after invasive monitoring was initiated.

an upright portable chest radiograph revealed cardiomegaly and pulmonary edema. Initial laboratory values revealed potassium of 3.4 mmol/L (3.4 mEq/L), carbon dioxide, 11 mmol/L (11 mEq/L), hemoglobin, 114 g/L (11.4 g/dL), creatine kinase, 73 U/L, and lactate dehydrogenase, 131 U/L. Arterial blood gas values were pH, 7.23; oxygen pressure, 46 mm Hg; carbon dioxide pressure, 32 mm Hg; and bicarbonate ion, 13 mmol/L (13 mEq/L).

The patient's blood pressure increased transiently to 120/107 mm Hg in response to the dopamine infusion. Despite continuous increases in the rate of dopamine infusion and subsequent 50-mL fluid challenges to support any decreases in intravascular volume, the patient failed to maintain adequate arterial pressures and became increasingly lethargic. Ventricular ectopy developed 30 minutes after arrival and was not suppressed by an IV lidocaine bolus of 100 mg and a continuous infusion of 4 mg/min.

Thirty-three minutes after arrival at the ED, pulseless ventricular tachycardia developed. Immediate countershock with 300 J resulted in a wide complex pulseless rhythm. The patient was intubated, and mechanical ventilation and compression were initiated (Thumper®, Michigan Instruments, Grand Rapids, Michigan). Ventilatory pressure was 40 cm H2O, and approximately 80 lb of force was used to compress the sternum 2 in.

In accordance with a protocol previously approved by the Human Rights Committee of Henry Ford Hospital, an arterial catheter [70 cm, 5.8F] was placed percutaneously in the right femoral artery by the Seldinger technique and advanced to the thoracic aorta distal to the subclavian artery. A central venous catheter (double lumen, 25 cm, 7F) was placed percutaneously in the right subclavian vein and then up the right arm. The catheters were connected to Gould Statham P-50 transducers, which were set to stretcher height and zeroed to atmosphere. Simultaneous pressure tracings were recorded [Figures 2 and 3] with Hewlett-Packard 78205D amplifiers and a Hewlett-Packard 7758 multi-channel recorder [Hewlett-Packard, Sunnyvale, California]. Continuous Ao, RA, and Ao – RA pressures were available throughout the resuscitation. Simultaneous arterial and central venous blood gases were obtained [Table 1].

When mechanical CPR was instituted, the patient became responsive and required repetitive small doses of IV morphine sulfate and diazepam for sedation. Whenever CPR was discontinued, the patient became unresponsive. Despite maximal pharmacologic intervention, it was not possible to maintain adequate systemic arterial blood pressure without mechanical chest compression. Ao and RA pressure tracings demonstrated deterioration over time [Table 2]. Two hours and ten minutes after arrival at the ED, an Ao balloon pump was placed in an unsuccessful endeavor to stabilize the patient's cardiac status.

It was the consensus of the emergency physicians, cardiologists, and cardiothoracic surgeons that partial cardiopulmonary bypass, cardiac catheterization, or prolonged maintenance on mechanical CPR were not...
TABLE 1. Simultaneous arterial and central venous blood gas results

<table>
<thead>
<tr>
<th>Time From Initiation of Resuscitation</th>
<th>One and One-Half Hours</th>
<th>One and One-Half Hours</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Arterial</td>
<td>Central Venous</td>
</tr>
<tr>
<td>Partial pressure of oxygen (mm Hg)</td>
<td>205</td>
<td>32</td>
</tr>
<tr>
<td>Oxygen saturation (%)</td>
<td>99</td>
<td>47</td>
</tr>
<tr>
<td>Partial pressure of carbon dioxide (mm Hg)</td>
<td>20</td>
<td>33</td>
</tr>
<tr>
<td>pH</td>
<td>7.27</td>
<td>7.19</td>
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feasible. Three hours and fifteen minutes after presentation, resuscitative efforts were terminated and, in accordance with family wishes, an autopsy was not performed.

DISCUSSION

Kuowenhoven et al, in their original description of the technique of closed-chest CPR, proposed that direct compression of the heart between the sternum and vertebral spine was responsible for blood flow during cardiac massage. Weale and Rothwell-Jackson questioned this theory after demonstrating equivalent elevations in arterial and venous pressures during external CPR and proposed that global increases in intrathoracic pressures and not cardiac compression were responsible for flow. This suggests that the geometry of the chest may determine the relative contributions of the cardiac or thoracic pump to systemic blood flow. However, a recent study in human beings that demonstrated a significant role for the thoracic pump mechanism concluded that thoracic geometry and body weight did not correlate with Ao or coronary perfusion pressures during CPR.

In experimental models, coronary perfusion pressure, as reflected by the diastolic Ao-RA pressure gradient, strongly correlates with coronary blood flow during CPR. Successful resuscitation from cardiac arrest in animals has been shown to correlate with the maintenance of Ao diastolic pressures of more than 30 mm Hg or with coronary perfusion pressures of more than 15 mm Hg. Maier et al demonstrated coronary blood flow 65% to 75% of baseline values during high-impulse manual compressions, while simultaneously recording significant differences between intracardiac and intrathoracic pressures.

Sanders et al found that increasing intrathoracic pressures with simultaneous compression-ventilation CPR and abdominal binding was detrimental to Ao diastolic pressure and coronary perfusion pressure and that, compared with standard CPR, animals treated with these modified techniques could not be resuscitated. Studies in human beings have failed to demonstrate adequate coronary perfusion pressures with conventional CPR or improve-
SUMMARY

Progress in improving resuscitation from cardiac arrest is limited by the inability to evaluate the effectiveness of CPR. Arteriovenous pressure measurements and venoarterial carbon dioxide gradients provide physicians with measures of the adequacy of resuscitative efforts. If progress is monitored and found to be inadequate early in the resuscitation effort, alternative therapeutic maneuvers such as internal cardiac massage or cardiopulmonary bypass may be considered.

We thank David M Kruger, MD, for his editorial assistance and computer support.

REFERENCES

17. Maier GW, Tyson GS, Olsen CO, et al: The

TABLE 2. Aortic and right atrial pressures

<table>
<thead>
<tr>
<th>Time From Initiation of Resuscitation</th>
<th>One-Half Hour + CPR</th>
<th>One-Half Hour - CPR</th>
<th>One and One-Half Hours + CPR</th>
<th>One and One-Half Hours - CPR</th>
<th>Two Hours + CPR</th>
<th>Two Hours - CPR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ao systolic pressure (mm Hg)</td>
<td>122</td>
<td>40</td>
<td>100</td>
<td>38</td>
<td>100</td>
<td>35</td>
</tr>
<tr>
<td>RA systolic pressure (mm Hg)</td>
<td>48</td>
<td>36</td>
<td>40</td>
<td>32</td>
<td>40</td>
<td>30</td>
</tr>
<tr>
<td>Ao-RA systolic gradient (mm Hg)</td>
<td>74</td>
<td>4</td>
<td>60</td>
<td>6</td>
<td>60</td>
<td>5</td>
</tr>
<tr>
<td>Ao diastolic pressure (mm Hg)</td>
<td>36</td>
<td>36</td>
<td>35</td>
<td>32</td>
<td>38</td>
<td>35</td>
</tr>
<tr>
<td>RA diastolic pressure (mm Hg)</td>
<td>15</td>
<td>17</td>
<td>12</td>
<td>17</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>Ao-RA diastolic gradient (mm Hg)</td>
<td>21</td>
<td>19</td>
<td>23</td>
<td>15</td>
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Ao, aortic; RA, right atrial; Ao-RA, aortic minus right atrial; +CPR, with CPR; -CPR, without CPR.

Systolic pressures were measured at peak systole, and diastolic pressures were measured at end diastole.


