the notion of synergy or true sensitization of the neuromuscular junction by a small pretreatment dose of atracurium.

REFERENCES


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Hypoxia Following Tricuspid Valve Resection

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Hypoxia after cardiopulmonary bypass (CPB) may result from a variety of causes, such as pulmonary edema from cardiac or noncardiac causes, excessive secretions, endotracheal tube malpositioned in a main stem bronchus, and equipment malfunction. We recently observed an unusual case of hypoxia associated with tricuspid valve resection.

REPORT OF A CASE

The patient is a 41-year-old man admitted for a antibiotic treatment of subacute bacterial endocarditis (SBE) involving the tricuspid valve. Because this was refractory to optimal antibiotic therapy, a tricuspid valvulectomy was scheduled. History was significant for intravenous drug abuse of cocaine and heroin over many years and no prior cardiac history or symptoms. Traumatic amputation of the left arm occurred several years before admission. Physical examination revealed a cachectic patient, lying flat in no acute distress. His weight was 55 kg, arterial blood pressure (BP) 110/80 mmHg, central venous pressure (CVP) 14 mmHg, heart rate 120 beats/min and regular, and temperature 38.8°C. There was no significant “V” wave on the CVP tracing. There was jugular venous distension of 3–4 cm at 30 degrees and on auscultation of the heart there was an apical systolic ejection murmur and soft S2. The lungs were clear. The remainder of the physical examination was within normal limits. On echocardiogram there was tricuspid insufficiency and extensive vegetations on the valve. A chest roentgenogram showed increased interstitial markings. Preoperatively $pH_{L}$ was 7.46, $PaO_2$ 74 mmHg, $PaCO_2$ 29 mmHg, $HCO_3^{-}$ 17 mEq/L, and oxygen saturation 99% (room air) (table 1). Serum electrolytes and coagulation profile were within normal limits. The patient was pre-
TABLE 1. Arterial Blood Gases

<table>
<thead>
<tr>
<th></th>
<th>$pH$</th>
<th>$P_\text{O}_2$ (mmHg)</th>
<th>$P_\text{CO}_2$ (mmHg)</th>
<th>$HCO_3^-$</th>
<th>$% \text{O}_2$ Saturation</th>
<th>$F_\text{IO}_2$ (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preoperative</td>
<td>7.46</td>
<td>29</td>
<td>74</td>
<td>17</td>
<td>95</td>
<td>21</td>
</tr>
<tr>
<td>After sternotomy</td>
<td>7.33</td>
<td>33</td>
<td>72</td>
<td>18</td>
<td>91</td>
<td>100</td>
</tr>
<tr>
<td>During first CPB period</td>
<td>7.38</td>
<td>26</td>
<td>514</td>
<td>15</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>After first CPB period</td>
<td>7.22</td>
<td>48</td>
<td>36</td>
<td>21</td>
<td>58</td>
<td>100</td>
</tr>
<tr>
<td>During second CPB period</td>
<td>7.46</td>
<td>47</td>
<td>624</td>
<td>20</td>
<td>99</td>
<td>100</td>
</tr>
<tr>
<td>After second CPB period</td>
<td>7.45</td>
<td>31</td>
<td>583</td>
<td>21</td>
<td>99</td>
<td>100</td>
</tr>
</tbody>
</table>

CPB = cardiopulmonary bypass; $F_\text{IO}_2$ = inspired oxygen concentration.

medicated with 10 mg diazepam orally 90 min before being brought to the operating room. On arrival in the operating room the arterial line and a central venous line (CVP) were inserted. The preoperative CVP was 14 mmHg. The right atrial systolic and diastolic pressures were not noted. Anesthesia was induced with sufentanil 250 μg and diazepam 20 mg iv. Vecuronium 10 mg iv was administered to facilitate endotracheal intubation, and ventilation was controlled with an $F_\text{IO}_2$ of 1.0. Hemodynamic stability was maintained during and after induction of anesthesia. After sternotomy, $pH$ was 7.33, $P_\text{O}_2$ 72 mmHg, $P_\text{CO}_2$ 33 mmHg, $HCO_3^-$ 18 mEq/l, and oxygen saturation 91.5%, while ventilation was controlled with a tidal volume of 550 ml, a respiratory rate of 10 breaths/min, and an $F_\text{IO}_2$ of 1.0. Positive end-expiratory pressure (PEEP) 5 cmH₂O was added, and $pH$ was 7.28, $P_\text{O}_2$ 80 mmHg, $P_\text{CO}_2$ 36 mmHg, $HCO_3^-$ 18 mEq/l, base excess 8.8, and oxygen saturation 93.5%.

The tricuspid valve was completely excised during hypothermic cardiopulmonary bypass, lasting 45 min, and organizing vegetations were removed. During cardiopulmonary bypass arterial blood gases were normal. An arterial blood gas immediately after separation from cardiopulmonary bypass revealed severe hypoxia with a $pH$ 7.21, $P_\text{O}_2$ 40 mmHg, $P_\text{CO}_2$ 46 mmHg, $HCO_3^-$ 18 mEq/l, and oxygen saturation 62%. Arterial blood pressure at that time was 80/60 mmHg with a heart rate of 110 beats/min. The trachea was suctioned, and minimal secretions were obtained. Positive end-expiratory pressure 10 cmH₂O was added, and 88 mEq of NaHCO₃ were given. In spite of increasing PEEP to 15 cmH₂O, and additional NaHCO₃, $P_\text{O}_2$ decreased to 36 mmHg, and the patient became more acidic over the next 30 min. Arterial blood pressure decreased to 50 mmHg, mean right atrial pressure went to 31 mmHg, and the patient became severely bradycardic to approximately 15–20 beats/min. Atropine 1 mg iv, epinephrine 5 μg iv, and external and internal cardiac massage were administered, and no response was obtained. Because these attempts were unsuccessful, cardiopulmonary bypass was re instituted. Because of this unexplained deterioration associated with severe hypoxia, the atrial septum was explored and a probe patent foramen ovale (PFO) was discovered. The defect was sutured closed and a Haemorrhage porcine hetrograft was inserted into the tricuspid position. This second run of CPB lasted 120 min. After valve replacement, CPB was terminated without complications. Arterial blood remained within the range 90/60–100/65 mmHg, HR was 110 beats/min, mean right atrial pressure was 10–12 mmHg and $P_\text{O}_2$ was 583 mmHg on $F_\text{IO}_2$ 1.0, and $pH$ and $P_\text{CO}_2$ were normal. Mannitol was included in the pump prime, and 80 mg of furosemide was administered iv during the first period of CPB. The patient remained stable in the intensive care unit (ICU), and his trachea was extubated on the first postoperative day. His subsequent course was uneventful.

**DISCUSSION**

That tricuspid valve endocarditis is more common in intravenous drug addicts than the nonaddicted population is borne out by a number of reports. The incidence of tricuspid endocarditis in different series of intravenous drug abusers with SBE has been found to be over 50%.1-4 In populations of patients with endocarditis who are not drug addicts, the incidence of tricuspid valve involvement has been found to be much lower, 6–11%.5,6 It is theorized that the higher incidence of tricuspid valve involvement in the former population is due to repeated exposure of the right side of the heart to iv injected particular matter.

Resection of the valve, without insertion of a prosthesis, is the recommended surgical procedure for tricuspid endocarditis unresponsive to antibiotic therapy.7 Because of the capacity of the right ventricle to compensate for the resulting volume overload, patients tolerate valve resection well. Replacement of the tricuspid valve with a prosthesis can be associated with serious complications. These include thrombosis and malfunction of the new valve.8,9 and the possibility of the prosthetic valve becoming infected after it has been inserted into an infected site.

Hypoxia after cardiac surgery is not an uncommon problem. Not infrequently it is a result of left ventricular failure, retained secretions, exacerbation of preexisting lung disease such as asthma or chronic obstructive pulmonary disease, or, less frequently, a hypersensitivity reaction to various administered substances resulting in noncardiogenic pulmonary edema.10-12 The relatively poor oxygenation preoperatively was attributed to diffuse interstitial pulmonary disease, which occurs commonly in intravenous drug addicts.13 Rarely it can occur as a result of intracardiac shunting through a previously closed foramen ovale. This has been reported in association with mechanical ventilation and PEEP.14-16 The mechanism for shunting is PEEP-induced elevation of pulmonary vascular resistance, causing an increase in right atrial pressure, favoring blood flow across a probe patent foramen ovale. In this patient, resection of the tricuspid valve led to accentuation of valvular regurgitation and increasing right atrial pressure further. The right-to-left shunt, which probably began when the patient developed tricuspid regurgitation from subacute bacterial endocarditis (SBE), became more pronounced as a result, worsening the hypoxia. This association between right-to-left
shunting, patent foramen ovale, and tricuspid insufficiency and valvulectomy has not been reported previously.

With removal of the tricuspid valve, the mean right atrial pressure increases and the right atrium and ventricle function as a common chamber. A right-to-left intracardiac shunt can develop if a previously closed foramen ovale becomes patent. This may result when the pressure in the right atrium is greater than that in the left atrium.

During fetal development, a patent foramen ovale is a normal and necessary part of the fetal circulation. Approximately 25% of the venous return from the inferior vena cava is shunted into the left atrium through the patent foramen ovale. At birth, pressures in the left side of the heart become higher than those in the right, thus favoring functional closure of the foramen ovale. Anatomic closure usually occurs within a few months. However, in a study on unslected autopsies, a probe patent foramen ovale was detected in 29% of "normal" adults. An isolated patent foramen ovale is usually clinically insignificant, and, if shunting occurs, it is usually left to right because of the pressure gradient that normally exists in this direction. Patients are usually asymptomatic, as was this patient before development of SBE and tricuspid regurgitation. However, when conditions favor elevation of right-sided pressures, a right-to-left shunt can be created. Anything that increases right-sided pressures can lead to this, such as pulmonary hypertension from pulmonary emboli and mechanical ventilation with PEEP. In this case, it was severe tricuspid regurgitation.

The progressive hypoxia and acidosis that resulted from right-to-left shunting exacerbated the blood flow through the patent foramen ovale by increasing pulmonary vascular resistance. Impedance to right ventricle outflow increased, causing more regurgitation across the tricuspid valve and higher right atrial pressure, and flow through the patent foramen ovale was enhanced.

With insertion of a prosthetic valve, the right atrium pressures decreased from 31 to 12 mmHg, and the patient's condition improved dramatically. Administration of fluids iv was necessary to maintain stable hemodynamics. Because of the increase in mean right atrial pressure, venous return was impaired. Postcardiopulmonary arterial blood gases were good with PaO₂ greater than 200 mmHg and normal acid–base status. The postoperative course was uneventful.

In view of the prevalence of intravenous drug abuse in society today, SBE involvement of the tricuspid valve can be expected to continue, as can the necessity for resection of the tricuspid valve. When evaluating causes of hypoxia in patients with tricuspid insufficiency, whether or not they have undergone valve resection, intracardiac shunting through a patent foramen ovale should be considered. Because of the hemodynamic alterations resulting from this lesion, it is possible that an intracardiac shunt previously flowing from left to right has reversed direction or a foramen ovale that was functionally closed has now become patent because of the elevated right atrial pressure.

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REFERENCES