reliance on mechanical ventilation until neuromuscular function is adequate for respiration would circumvent the possible adverse effects associated with the simultaneous administration of atropine and neostigmine in the patient taking propranolol. However, if in this clinical setting it is elected to reverse a nondepolarizing neuromuscular block by slowly titrating atropine against neostigmine, it should be done with careful monitoring in order to detect quickly possible adverse physiologic responses.

REFERENCES


Impaired Arterial Oxygenation Associated with Use of Bone Cement in the Femoral Shaft

TAMAS KALLOS, M.D.*

The use of acrylic (methylmethacrylate) bone cement for fixing prostheses in the femoral shaft has been associated with sudden cardiac arrest.1–4 The sequence of events leading to these accidents has not been clear. Hypotension produced by the absorption of the volatile methylmethacrylate monomer, a vasodilator,5 has been suspected to be the cause of the cardiovascular collapse.6 Recent studies have demonstrated that cement packing produces elevation of femoral medullary pressure, leading to embolization of medullary contents.6–10 The acute pulmonary fat and bone-marrow embolism resulting from insertion of cement and prosthesis in the femur may interfere with arterial oxygenation. If hypoxemia occurs under these conditions, it may contribute to the occurrence of cardiac arrest, especially when associated with systemic hypotension resulting from monomer absorption.

The present study was designed to determine whether cementing of prostheses into the femur is associated with impairment of arterial oxygenation. Since a decrease in arterial oxygen tension was found in every patient, possible ways of preventing hypoxemia were also studied.

MATERIALS AND METHODS

Twenty-four patients, ages 32 to 78 years, free of symptomatic cardiovascular or respiratory disease, were studied while undergoing Charnley total hip replacement. After induction with thiopental and tracheal intu-
bation with succinylcholine, anesthesia was maintained with nitrous oxide and halothane or enflurane using controlled respiration.

An 18-gauge Teflon catheter was inserted percutaneously into a radial artery after checking ulnar-artery flow by an Allen's test while the patient was awake. The catheter was continuously flushed with a slow drip of heparinized saline solution (3–4 drops/min). Arterial blood pressure was continuously recorded in 18 patients. Through the arterial cannula an indwelling continuous oxygen electrode (International Biophysics Corp., Irvine, Calif.) was passed into the arterial blood stream. After a period of 30–45 minutes for electrode stabilization, arterial oxygen tension was measured with an IL 213 electrode to calibrate the indwelling electrode. Calibration was rechecked with additional $P_o_2$ measurements intermittently during the study.

The patients were divided into three groups. Group A consisted of eight patients who received 33 to 50 per cent oxygen in the inspired gas during the study. The nine patients in Group B were ventilated with the same concentration of oxygen, but a vent hole was drilled in the distal femoral shaft. The vent consisted of a standard Craig needle biopsy cutter of 3-mm I.D. (for Patient 17 a vent of 6-mm I.D. was used). In five of the patients the vent was introduced with aid of a Steinmann pin used as a drill, without bone material being removed after drilling. In the other three patients the Craig needle biopsy set was used for drilling and the bone plug was removed. The patency of each vent was checked by aspiration. In one patient the intramedullary pressures during cement packing were recorded with aid of a plastic catheter connected to a pressure transducer and passed into thereamed-out femoral medullary canal.

Seven patients (Group C) were treated like Group A, except that five minutes before insertion of cement into the femur nitrous oxide was discontinued, the oxygen flow was increased to 6 l/min, and the inspired halothane (or enflurane) concentration was slightly elevated to compensate for elimination of nitrous oxide.

In six patients central venous pressure (CVP) was recorded with a catheter inserted through the internal jugular vein and checked by X-ray for location. Blood loss was estimated and normovolemia maintained with infusions of plasma protein fraction, whole blood, and lactated Ringer's solution.

To rule out hypotension as the cause of changes in $P_{a_o}_2$, ephedrine, 5–20 mg, was administered intravenously to five patients two minutes before insertion of cement.

To rule out a direct effect of methylmethacrylate on hemoglobin or on the oxygen electrode, blood was tonometered with gas of known oxygen tension. An IBC oxygen electrode continuously measured $P_o_2$ in the tonometered blood. $P_o_2$ values were also determined with an IL 213 electrode. Methylmethacrylate monomer was then introduced into the tonometered blood (in amounts to yield a concentration more than 100 times that expected in man during use of acrylic cement) and $P_o_2$ values were again recorded with both methods.

The $P_{a_o}_2$ values before and after insertion of cement and prosthesis in each patient were compared using paired t tests. $P < 0.05$ was regarded as significant.

**Results**

**Group A: Inspired Oxygen 33 to 50 Per Cent**

A significant decrease in arterial oxygen tension was observed in every patient in this group; mean $P_{a_o}_2$ values decreased from 131 torr in the control period to 122 torr ($P < 0.05$) one minute after the surgeon started to pack the cement into the femoral shaft (fig. 1). The mean $P_{a_o}_2$ value reached a nadir of 87 torr ($P < 0.01$) in four minutes and remained below control values for eight minutes or longer. In five of the eight patients, the $P_{a_o}_2$ decreased to below 80 torr; in one patient, it reached 50 torr and remained below 54 torr for 22 minutes.

**Group B: Vented Femurs; Inspired Oxygen 33 to 50 Per Cent**

The five patients in whom the vent was placed without removing a bone plug showed a decrease in $P_{a_o}_2$ from 126 to 109 torr in 90 seconds ($P < 0.05$); $P_{a_o}_2$ reached a nadir of 83 torr ($P < 0.01$) four minutes after
FIG. 1. Arterial oxygen tension changes in eight patients after packing bone cement into the femur in the absence of a femoral vent. Inspired oxygen 33–50 per cent.

FIG. 2. Arterial oxygen tension changes in nine patients after packing bone cement into the vented femur. Inspired oxygen 40–50 per cent.

the beginning of cement packing (fig. 2). The femoral medullary pressure measured in Patient 9 remained low during the initial part of cement packing, but suddenly rose to 208 torr and remained at this level for 30 seconds.

Patients who had a bone plug removed during insertion of the vent exhibited lesser decreases in $P_{a\text{O}_2}$. Two patients had no change in $P_{a\text{O}_2}$, and one patient showed a

FIG. 3. Femoral medullary contents drained through a vent during insertion of cement and prosthesis. On the left: as drained in a plastic tube; the light circles correspond to fat globules. On the right: same material after centrifugation; layers from the top: fat, bone marrow, plasma and erythrocytes.
decrease in Pao₂ from 160 to a low of 134 torr. The vents were open throughout cement packing and insertion of prosthesis in all patients except Patient 16, and drained various amounts of material (fig. 3). In Patient 16 the vent became occluded by a blood clot which was dislodged by intramedullary pressure when the prosthesis was inserted. This patient had the largest decrease in Pao₂ of this group (from a control of 144 to 108 torr after insertion of cement and prosthesis).

**Group C: Inspired Oxygen 98 Per Cent or More**

No decrease in Pao₂ was found following cement packing in two of the seven patients. The mean Pao₂ of 357 torr in the seven patients decreased to 321 torr two minutes after cement packing began ($P < 0.05$), a change which was statistically but not clinically significant. Six minutes after cementing began, mean Pao₂ was 293 torr, not significantly different from the pre-cement value ($P < 0.05$). In no patient did Pao₂ decrease to below 100 torr (fig. 4).

**Blood Pressure and CVP**

There was no correlation between changes in blood pressure and changes in arterial oxygen tension. In four patients (in group A) marked decreases in Pao₂ were observed even though arterial blood pressures did not decrease. Injection of ephedrine prior to packing of cement produced no change in Pao₂.

Central venous pressure (fig. 5) decreased slightly in the first three minutes in all six patients in whom it was measured. The decrease in central venous pressure was followed by a subsequent elevation from the

**Fig. 5.** Changes in central venous pressure in six patients following insertion of bone cement into the femur in the absence of a femoral vent. A slight decrease in the first three minutes preceded the elevation in every patient.
### Table 1. Arterial Oxygen Tensions in Patients after Packing Bone Cement into the Femoral Shaft (in Torr, Mean ± SEM)

<table>
<thead>
<tr>
<th>Number of Patients</th>
<th>Minus 2</th>
<th>Minus 1</th>
<th>Minus 0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>F&lt;sub&gt;1,n&lt;/sub&gt; = 0.33–0.5; no vent</strong></td>
<td>8</td>
<td>129.8 ± 13.3</td>
<td>129.6 ± 13.6</td>
<td>130.5 ± 13.8</td>
<td>121.9 ± 12.5</td>
<td>98.7 ± 10.0</td>
<td>88.9 ± 9.0</td>
<td>87.0 ± 10.6</td>
<td>91.3 ± 12.4</td>
<td>92.4 ± 12.8</td>
</tr>
<tr>
<td><strong>F&lt;sub&gt;1,n&lt;/sub&gt; = 0.4–0.5; vent; no plug removed</strong></td>
<td>5</td>
<td>126.0 ± 12.2</td>
<td>125.8 ± 11.0</td>
<td>125.0 ± 12.1</td>
<td>92.8 ± 10.1</td>
<td>85.4 ± 9.0</td>
<td>82.6 ± 6.1</td>
<td>87.8 ± 5.2</td>
<td>101.7 ± 10.8</td>
<td></td>
</tr>
<tr>
<td><strong>F&lt;sub&gt;1,n&lt;/sub&gt; = 0.4–0.5; vent; plug removed</strong></td>
<td>4</td>
<td>172.8 ± 33.2</td>
<td>171.5 ± 32.7</td>
<td>160.5 ± 35.6</td>
<td>156.5 ± 36.1</td>
<td>157.8 ± 36.6</td>
<td>150.0 ± 36.6</td>
<td>160.8 ± 34.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>F&lt;sub&gt;1,n&lt;/sub&gt; &gt; 0.98; no vent</strong></td>
<td>7</td>
<td>341.0 ± 19.5</td>
<td>356.6 ± 18.7</td>
<td>393.1 ± 18.0</td>
<td>321.1 ± 29.0</td>
<td>321.3 ± 27.9</td>
<td>297.4 ± 41.1</td>
<td>293.7 ± 45.9</td>
<td>292.3 ± 47.1</td>
<td></td>
</tr>
</tbody>
</table>

*Significantly different from time 0 (P < 0.05).
†Significantly different from time 0 (P < 0.01).

This study demonstrates a large decrease in arterial oxygen tension shortly after packing of bone cement and suggests that the presence of some type of oxygen embolism may have caused the elevation of pulmonary arterial pressure. Pak et al. ruled out the possibility that a decrease in Po<sub>2</sub> in the femoral artery was due to decreased pulmonary arterial pressure, as their data showed a decrease in arterial oxygen tension shortly after packing of bone cement.
duced during the use of cement, as found by Modig et al. Since the decrease in PaO₂ is not totally prevented by breathing high concentrations of oxygen, some of the decrease in PaO₂ should be produced by increased shunting or a decrease in cardiac output.

If the decrease in arterial oxygenation results from fat and bone-marrow embolism by elevated medullary pressure, it should be preventable by venting the medullary cavity. A plastic tube passed from the open end of the femur into the reamed-out shaft is effective in relieving the pressure and draining some of the medullary material; however, when it is removed as the prosthesis is inserted, the medullary pressure rises sharply. Vents drilled into the femoral shaft slightly below the area where cement will reach are effective in relieving the pressure. Holes in this location unfortunately weaken the femur and predispose to fractures. As a compromise, a vent hole may be made at the femoral condyle, where osteogenic fractures are less likely. With this type of vent, as the data of this study indicate, the size and patency of the hole and the resistance offered by the spongy medulla between the reamed area and the vent are critical. These factors will determine whether complete relief of medullary pressure elevation can be achieved. Since intramedullary pressures of several hundred torr have been measured during cementing of prostheses, the presence of a partially effective vent will not prevent pressure increases greater than the 4–8 torr necessary to produce pulmonary embolism. This seems to have been the case with the vents where the bone plugs were not removed and the one occluded by a clot.

Since no report of postoperative mobility due to delayed effects of pulmonary fat and marrow emboli after use of cement has appeared, the major problem seems to be the acute one, when hypotension from the absorbed monomer and hypoxemia from pulmonary fat and marrow emboli may occur. Attention should be paid, therefore, to avoiding the simultaneous occurrence of hypotension and hypoxemia. Hypotension can be minimized by careful maintenance of normovolemia and use of vasopressors if necessary. Hypoxia may be prevented by suctioning the embolizable material from the femoral shaft, the use of a plastic tube vent during packing of cement to relieve medullary pressure, and the administration of almost 100 per cent oxygen. Venting the femoral shaft with a large, well-placed hole may also prevent hypoxemia, but it introduces other potential problems (fracture, infection, hematoma, etc.), and its benefits to a given patient should be weighed against its risks.

The author thanks Drs. Martin L. Gold and Augusto Sarmiento for their help and encouragement, and International Biophysics Corp., Irvine, California, for supplying the indwelling oxygen electrodes.

REFERENCES

Failure of a High-Compliance Low-pressure Cuff to Prevent Aspiration

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Tracheal intubation with a cuffed tube permits effective positive-pressure ventilation and protects the lungs from aspiration. Ucleration of the tracheal wall, occasionally with hemorrhage or perforation, can occur with time when cuffs are inflated so that appreciable pressure is applied to surrounding tissues.1,2 Tubes sheathed with high-volume cuffs have been recently introduced. With these cuffs a seal can be created at low internal cuff pressures, minimizing the pressure that can be transmitted to tracheal mucosa.3 The following case report and supporting experimental observations indicate that large-volume cuffs, while affording an effective seal for positive-pressure ventilation, may not protect against aspiration in the spontaneously breathing patient.

REPORT OF A CASE

A 43-year-old Indian woman with a long, flamboyant history of alcoholism underwent operation for bleeding esophageal varices. A portacaval shunt was performed. Postoperatively the patient continued to vomit blood and started to bleed copiously from her nose as well. She was somewhat obtunded, with a depressed gag reflex. When blood was aspirated from the trachea, the trachea was intubated with a 8.0-mm I.D. Lanz endotracheal tube. This tube had a high-volume, high-compliance cuff which was inflated as recommended by the manufacturer so that the pilot balloon was approximately two thirds full, yielding a balloon cuff pressure of approximately 20 cm H2O. The patient was allowed to breathe spontaneously. Large amounts of blood continued to be suctioned from the endotracheal tube until the cuff was further inflated to pressures greater than 50 cm H2O, whereupon the ability to retrieve blood upon suctioning ceased. With deflation of the cuff to recommended pressures, blood was again obtainable by endotracheal suctioning. Positive-pressure ventilation at recommended cuff inflation pressures also prevented the appearance of blood in the trachea. The tube was replaced by a standard #9.0 Portex tube with a low-volume, high-pressure cuff. The bleeding was brought under control, and the trachea was extubated on the fifth postoperative day.

METHODS

Sizes #6 and #9 Lanz endotracheal tubes with large-volume cuffs were inserted into various models of the trachea, including excised dog tracheas of 20- and 30-mm diameter, as well as a glass cylinder of 20-mm diameter. cuffs were inflated as recommended by the manufacturer and cuff pressures were continuously monitored with a water manometer. Leakage of