Prolonged Anesthesia, Overcurarization, and Resection of the Abdominal Wall

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It has been noted that the administration, over long periods of time, of even small doses of some of the neuromuscular blocking agents, may cause the persistence of subtle degrees of paralysis. However, modern light general anesthesia, presumably involving the use of these neuromuscular blocking agents, has been advocated for patients who are to undergo prolonged operations.

CASE REPORT

A 57 year old, 73-kg. man was admitted, for carcinoma of the bladder with a fungating midline lower abdominal wall extension. An electrocardiogram revealed minimal anterolateral ischemia, otherwise there was no evidence of cardiopulmonary or neurologic disease.

A radical cystectomy, pelvic lymph node dissection, excision of an 8.5 x 4 cm. section of the abdominal wall, and construction of an ileal loop, were performed. A midline incision extended from the xiphisternum to the pubic rami. Anesthesia was induced with thiopental sodium (Surital 250 mg.), and maintained with nitrous oxide (4 liters per min.) and oxygen (2 liters per min.) after tracheal intubation using succinylcholine (60 mg.) apnea. A semi-closed carbon dioxide absorption circle system was used. D-tubocurarine (129 mg.) was given, in divided doses, to produce abdominal relaxation. Evidence of returning muscle power was neither sought nor observed before each dose of the drug. Respiration was controlled by a ventilator. The anesthetic lasted 11 hours, during which the vital signs were satisfactory. A total of 4,000 ml. of whole blood, 300 ml. of plasma, and 1,250 ml. of 5 per cent dextrose in water were given.

The last dose of d-tubocurarine (3 mg.) was given at 4:15 P.M. At 6:50 P.M., after the nitrous oxide was discontinued, consciousness and shallow diaphragmatic inspiratory efforts returned. Atropine (1 mg.) was injected intravenously followed by neostigmine (3 mg., in divided doses). The inspiratory force was then 22 cm. of water. However, the patient was unable to lift his head, had a distinct tracheal tug, poor thoracic excursions, and a marked lack of facial expression. His vital capacity was 250 ml. In view of this evidence of residual curarization the endotracheal tube was left in place and the patient was taken to the recovery room, where the use of a ventilator was reinstituted. The sodium was 113 mEq./liter, potassium 4.2 mEq./liter, chloride 77 mEq./liter, and carbon dioxide 25 mEq./liter. Normal sodium chloride (500 ml.) was infused. Although edrophonium (Tensilon 10 mg.) produced an increase in muscle power, additional antagonists or stimulants were not given. At 1:30 A.M. the diaphragmatic inspiratory efforts were out of phase with the ventilator. This situation was corrected by intravenous morphine (4 mg., in divided doses).

The following morning, 8:30 A.M., the inspiratory force was 27 cm. of water, and the vital capacity 650 ml. The patient was able to lift his head, had no tracheal tug, good thoracic excursions, and normal facial expressions. His sodium was 133 mEq./liter, potassium 4.6 mEq./liter, chloride 97 mEq./liter, and carbon dioxide 31 mEq./liter. Despite this evidence that the residual curarization had worn off, the patient was still unable to breathe adequately. Arterial blood analyses (table 1), during artificial ventilation, revealed that the P02 only rose to 235 mm. of mercury when 100 per cent oxygen was inspired, an alveolar-arterial oxygen tension difference of 434 mm. of mercury. The tidal volume, at this time, was 1,000 ml., and the physiologic dead space was measured to be

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### Table 1. Measurements of Respiratory Function

<table>
<thead>
<tr>
<th>Postoperative Day</th>
<th>Vital Capacity (ml)</th>
<th>Spontaneous Tidal Volume (ml)</th>
<th>Type of Ventilation</th>
<th>Arterial Blood Analyses</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Fio_2</td>
</tr>
<tr>
<td>0</td>
<td>250</td>
<td>250</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>1</td>
<td>650</td>
<td>250</td>
<td>Artificial</td>
<td>0.21</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1.0</td>
</tr>
<tr>
<td>2</td>
<td>800</td>
<td>350</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>1,300</td>
<td>500</td>
<td>Spontaneous</td>
<td>0.21</td>
</tr>
<tr>
<td>4</td>
<td>1,100</td>
<td>500</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>1,200</td>
<td>550</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>1,450</td>
<td>600</td>
<td>Spontaneous</td>
<td>0.21</td>
</tr>
</tbody>
</table>

Fio\_2 = fraction of inspired oxygen; Pa\_O\_2 = arterial oxygen tension; Pa\_CO\_2 = arterial carbon dioxide tension.

535 ml., a dead space to tidal volume ratio of 0.53. A tracheostomy was therefore performed, and the patient transferred to the respiratory unit, with artificial ventilation continued.

The patient improved rapidly during the next five days (table 1). Fifteen to 30 minutes of spontaneous respiration were tolerated each hour on the second postoperative day, and total independence of the ventilator on the sixth. A streaky area of atelectasis in the right mid-lung field was observed on radiological examination on the third postoperative day. The patient was discharged from the respiratory unit on the seventh postoperative day, and, after removal of the tracheostomy tube, from the hospital on the twenty-fifth postoperative day.

**COMMENT**

This case report illustrates four points: (1) The inadvisability of giving d-tubocurarine repeatedly in the absence of evidence of returning muscle power, and the fact that a 2 hour and 35 minute lapse, after the last injection of the drug, does not ensure that the patient will then be able to breathe adequately. (2) The value of applying several objective tests for residual curarization. It has been suggested that an inspiratory force of 20 cm. of water can usually be accepted as evidence of sufficient reserve of ventilatory effort. However, the inability of this patient to lift his head, the distinct tracheal tug, poor thoracic excursions, and marked lack of facial expression indicated residual curarization in spite of an inspiratory force of 22 cm. of water. The reason for the hyponatremia and hypochloremia, which was present at this time, and which might have contributed toward the muscle weakness, is not known. (3) The efficacy of the conservative method of treating over-curarization. In spite of the improvement after ecephalium, it was decided to treat this patient expectantly, even to the extent of giving morphine to reduce the spontaneous diaphragmatic inspiratory efforts, to allow the ventilator to function effectively. Persistence in the use of stimulants, in an attempt to induce adequate spontaneous ventilation in these patients, has been severely criticized. After 12 hours of artificial ventilation, and correction of the hyponatremia and hypochloremia, the objective tests indicated that the residual curarization had worn off. However, the patient was still unable to breathe adequately, presumably because of the "tight" abdominal wall. (4) The value of according a patient with respiratory failure the type of care given in a respiratory unit and of assaying his pulmonary status carefully and objectively. On the first postoperative
day, the large alveolar-arterial oxygen tension gradient, during ventilation with 100 per cent oxygen, indicated that there was considerable intrapulmonary shunting, presumably due to the perfusion of nonventilated air spaces.10 There was, at the same time, a marked increase in the dead space to tidal volume ratio. Such alterations in the relation of ventilation to perfusion are common in patients with respiratory failure and indicate the pattern of artificial ventilation and oxygenation which should be employed.10 On the sixth postoperative day the vital capacity was approximately 20 ml./kg., and the patient was able to sustain adequate spontaneous ventilation. Weaning from the ventilator is usually possible when the vital capacity has improved to three times the predicted normal tidal volume10 (to approximately 20 ml./kg.).

REFERENCES

Respiration

FIRE IN A RESPIRATOR A nurse moved a floor lamp, simultaneously a respirator in use on a critically ill patient suddenly emitted sparks and thick, black, acrid smoke. Reconstruction of the incident revealed that the lamp had a two-wire cord and at least three faults in the insulation of the wiring inside its metal structure. When the base of the lamp bumped against a grounded castor of the respirator, current flowed. The helical wire spring, having the greatest electrical resistance in the happenstance circuit, was heated to glowing and ignited in the oxygen-enriched atmosphere. Sparks that spewed from the respiratory were hot metal ejected from the burning housing. The acrid black smoke is characteristic of burning plastic. (Walter, C. W.: Fire in an Oxygen-Powered Respirator, J.A.M.A. 197: 44 (July) 1966.)