clinical use and also adequate for research purposes provided that the ability to measure variations of less than 0.1 per cent is not important. It is necessary to make frequent adjustments of the zero and gain controls to insure accuracy. Concentrations of up to 10 per cent may be read directly from the meter by varying the normal standardization procedure. The meter may be connected to a 1 mv. recorder by means of a special adapter and permanent tracings obtained.

References

Case Reports

Muscle Rigidity Following Administration of Succinylcholine

James R. Cody, M.D.

This case is reported to call attention to the rare, potentially lethal, persistent generalized muscle rigidity which may follow intravenous succinylcholine and to speculate on its relationship to both myotonia dystrophica and idiopathic hyperpyrexia during anesthesia.

Case Report

A 10-year-old white boy weighing 54 pounds was admitted for surgical correction of bilateral cryptorchism. His general health had always been good and despite moderate idiopathic kyphoscoliosis, he played football and baseball. Routine laboratory studies disclosed hemoglobin, 13.3 Gm./100 ml.; hematocrit, 38 per cent; leukocyte count, 6,400 with a normal differential; and normal urine. On the day of operation secobarbital, 100 mg., was given by mouth at 7:30 A.M. and meperidine, 50 mg., and scopolamine, 0.4 mg., were given intramuscularly at 8:00 A.M. On arrival in the operating room, the patient was asleep but could be roused easily. His blood pressure was 90/40 mm. Hg.; pulse rate 84 beats/min. Induction of anesthesia was begun at 9:55 A.M. with 1.5 per cent halothane in 50 per cent nitrous oxide and oxygen, using a closed-circle system with carbon dioxide absorption and a 4 l./min. flow of gases. Intravenous infusion of 5 per cent dextrose in 0.2 per cent saline was started and at 10:00 A.M. succinylcholine, 40 mg., and atropine, 0.2 mg., were given to facilitate tracheal intubation. No fasciculations were noted, but within one minute the patient developed generalized tonic muscle spasms characterized by flexion and internal rotation of the arms, forearms, and hands and extension of the lower extremities with internal rotation of the feet. The abdominal muscles were rigid and the jaws could not be opened. A second injection of 20 mg. succinylcholine was given at 10:20 A.M. without altering the muscle spasm. By 10:05 A.M. blood pressure had risen to 110/40 mm. Hg. and pulse rate to 200 beats/min. The muscle contractions were so severe that the positions of the extremities could not be changed even with considerable force. Despite the muscle spasm and concurrent apnea, artificial respiration could be maintained without cyanosis, with the face mask and rebreathing bag. From 10:05 to 10:15 A.M. 2 per cent halothane was administered in an attempt

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to relax the muscles. This produced no relaxation but reduced blood pressure to 90/40 mm. Hg and pulse rate to 150 beats/min. The electrocardiographic monitor indicated that the tachycardia was sinus in origin. The operation was cancelled and halothane discontinued at 10:25 A.M. Spontaneous respirations returned at 10:30 A.M. Venous blood at this time contained calcium 5.8 mg./100 ml., phosphate 10.3 mg./100 ml., sodium 133 mEq./l., chloride 93 mEq./l., potassium 5.1 mEq./l., and CO₂ 16.5 mEq./l. All these values were within the normal range of our laboratory except phosphate (normal range 4–6 mg./100 ml.) and CO₂ (normal range 20–30 mEq./l.). By 10:40 A.M. spontaneous respiration was considered adequate and the patient was taken to the recovery room where his blood pressure was 110/50 mm. Hg, pulse rate 150 beats/min., and rectal temperature 99.4° F. His muscles remained in spasm. At 11:30 A.M. a second venous blood sample contained calcium 8.0 mg./100 ml. and phosphate 8.8 mg./100 ml. By 1:30 P.M., the child was awake and alert enough to complain through tightly clenched teeth that his muscles “felt sore.” He could not move his extremities voluntarily and complained of severe pain when passive movement was attempted. On the following day he was ambulatory but could open his jaws only partially, needed assistance in arising from bed, and could walk only on his toes. The muscle rigidity slowly disappeared. A full range of motion of the extremities was not possible until the third postoperative day.

Following this bizarre episode further studies were carried out. A muscle biopsy taken under local anesthesia was reported as “muscular dystrophy suggestive of myotonic dystrophy.” Electromyographic recordings were interpreted as compatible with primary muscle disease. No examiner could elicit delayed extension of the fingers following isometric contraction of the hand. Subsequent laboratory studies revealed no abnormalities. The cause of the increased blood phosphate was not established.

DISCUSSION

Including this patient, there are now published reports of 11 patients who developed some muscle rigidity following a depolarizing muscle relaxant injected intravenously. Three 1,2 of the 11 had known myotonia dystrophica and developed muscle rigidity after succinylcholine or decamethonum given during general anesthesia. Kaufman 1 reported 25 patients with myotonia dystrophica who underwent anesthesia and operation. Four were given succinylcholine and one developed spasm of the hand muscles lasting about a minute. The other three had no abnormal response. He mentioned another patient who developed generalized muscle spasm with respiratory distress after decamethonum. Patterson 2 reported a patient with myotonia dystrophica who developed severe generalized rigidity of all muscles after each of three consecutive injections of succinylcholine. Each period of rigidity lasted three to four minutes during which it was impossible to inflate the lungs.

Myotonia, characterized by a delay in muscle relaxation following a muscle contraction, often is associated with spasm of related muscle groups not involved in the original contraction. Orndahl and Stenberg 3 reported that patients with myotonia and those with denervated muscles are more sensitive to the stimulating action of depolarizing muscle relaxants than are patients with normal muscle, patients with primary muscle disease without myotonia, and patients with myotonia who have received gallamine or d-tubocurarine before succinylcholine. In this same group, Orndahl 4 studied the simultaneous mechanical (isometric) and electromyographic responses of myotonic and denervated muscle to depolarizing relaxants. He found that the electrical activity of the muscle diminished more rapidly than the contraction and in some instances disappeared completely while the contraction persisted for as long as four minutes. He concluded that the response was a contracture and suggested that this response occurred in muscle fibers with multiple and/or enlarged receptor surfaces, a condition present in myotonic muscle, during re-innervation of paretic muscle, and rarely in healthy subjects. In an unstated number of their patients these authors noted an increased resistance to inflation of the lungs after succinylcholine when given in doses greater than 20 mg.

Muscle rigidity following depolarizing mus-
<table>
<thead>
<tr>
<th>Reference</th>
<th>Age Yrs.</th>
<th>Diagnosis</th>
<th>Operation</th>
<th>Muscle Relaxant, min*</th>
<th>Duration of Rigidity, Min.</th>
<th>Maximum Temp, °F</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>42</td>
<td>Laceration of cornea</td>
<td>Repair of cornea</td>
<td>S.C. 60 C10</td>
<td>150</td>
<td>107</td>
<td>Expired</td>
</tr>
<tr>
<td>6</td>
<td>3</td>
<td>Arthrogyrosis Scoliosis</td>
<td>Achilles tendon lengthening</td>
<td>S.C. 15</td>
<td>80</td>
<td>107</td>
<td>Survived</td>
</tr>
<tr>
<td>6</td>
<td>5</td>
<td>Strabismus</td>
<td>None</td>
<td>S.C. 20</td>
<td>35</td>
<td>101.6</td>
<td>Survived</td>
</tr>
<tr>
<td>6</td>
<td>10</td>
<td>Thoracicolumbar scoliosis</td>
<td>Spinal fusion</td>
<td>S.C. 40</td>
<td>150+</td>
<td>108.5</td>
<td>Expired</td>
</tr>
<tr>
<td>7</td>
<td>5</td>
<td>Retarded growth</td>
<td>T &amp; A</td>
<td>S.C. 15</td>
<td>110</td>
<td>104.2</td>
<td>Expired</td>
</tr>
<tr>
<td>8</td>
<td>12</td>
<td>Thoracicolumbar scoliosis</td>
<td>None</td>
<td>S.C. 30</td>
<td>120</td>
<td>99.5</td>
<td>Survived</td>
</tr>
<tr>
<td>9</td>
<td>19</td>
<td>Hypertrophied T &amp; A</td>
<td>None</td>
<td>S.C. 40</td>
<td>75</td>
<td>103.6</td>
<td>Expired</td>
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<tr>
<td>This report</td>
<td>10</td>
<td>Cryptorchidism Kyphosecoliosis</td>
<td>None</td>
<td>S.C. 40</td>
<td>3 days</td>
<td>99.4</td>
<td>Survived</td>
</tr>
</tbody>
</table>

* S.C. = succinylcholine; C10 = decamethonium.
** Onset of rigidity after operation completed.

do not appear until the third and fourth decades of life, it seems reasonable to postulate that most of these patients had undiagnosed myopathy leading to a contracture response to succinylcholine. The heat production accompanying contracture, and undissipated because of the surgical drapes, might have led to the hyperpyrexia which may have been the cause of death in some patients.

From this collected clinical experience, some tentative therapeutic recommendations seem reasonable. It would seem unwise to persist in an elective surgical procedure in the face of an abnormal response to succinylcholine, because this has led to death in three of four patients whose operations were completed. Three of four patients whose operations were deferred survived. It would seem wise to monitor temperature in all children particularly those with musculoskeletal abnormalities, during anesthesia.

**Summary**

An episode of persistent generalized muscular rigidity in response to succinylcholine has been described and 10 other reported instances
of this abnormal response have been reviewed. It is suggested that this rigidity represents a contracture such as that seen in some patients with myotonia dystrophica after succinylcholine, and that patients exhibiting this response have undiagnosed myotonia. It is also suggested that the muscle contracture of undiagnosed myotonia is one cause of idio-
pathic hyperpyrexia during general anesthesia.

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Spinal Epidural Hematoma Following Continuous Epidural Anesthesia

TED F. GINGRICH, M.D.*

Recently, Bromage has written a compre-
hensive review of epidural anesthesia1 in which he indicates that further studies of the mechanism of action as well as applicability and complications of the technique are neces-
ary. This is a report of a serious, though rare, complication of lumbar epidural anesthesia.

CASE REPORT

A 73-year-old man was admitted to the hospital with a painful leg that had developed suddenly five days previously. He had a history of arterio-
sclerotic disease, and had had a Dacron prosthesis inserted at the aorto-iliac bifurcation three years prior to admission. Examination revealed a cold, blue, swollen left leg with no pulses palpable in the popliteal, dorsalis pedis or posterior tibial arteries. Faint pulsation was present in the femoral area. A line of demarcation was present at mid-
thigh level. Because of reasonable certainty that this clinical episode was due to thrombosis of the graft, it was decided to attempt a sympathtic block of the ex-
tremity to salvage as much of the leg as possible prior to amputation. An epidural catheter was placed between L3-L4 and 15 ml. of 1 per cent lidocaine (Xylocaine) were injected through the catheter. Within 20 minutes the leg began to warm and the line of demarcation receded about the level of the knee. The patient was obviously much more comfortable, so it was decided to leave the catheter in place and continue the block for two or three days. Sixteen hours after placement of the catheter, anticoagulation therapy was started, with intravenous heparin every four hours. After 24 hours, it was noted that Xylocaine had to be injected more frequently and in larger quantity to produce relief of pain. At the 48th hour, the patient developed severe pain in the lumbar area and was given both morphine and barbital. To check the position of the epidural catheter, 1 cc. of contrast dye was injected through the catheter and an X-ray was made. The tip of the catheter was located in the interspinous area, so it was removed. The patient had no more pain, but during the next

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rado Medical Center, Denver, Colorado.