ELECTROMYOGRAPHIC RESPONSES TO SMALL DOSES OF SUXAMETHONIUM IN CHILDREN AFTER BURNS

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Cardiac arrest has occurred following the administration of suxamethonium to burned patients (Fleming et al., 1960), and recognition of this hazard has led to the avoidance of the use of suxamethonium in such patients. Bush (1964) postulated a number of possible mechanisms and, although it was suspected that hyperkalaemia might be the cause, this was not confirmed until Tolmie, Joyce and Mitchell (1967) reported repeated cardiac arrests associated with acute increases in serum potassium concentration in a badly burned soldier in Vietnam. Keneally and Bush (1974) reported a mean maximum change of 0.13 mmol litre\(^{-1}\) after suxamethonium 1.5 mg kg\(^{-1}\) in children—the only statistically significant change was in the group aged 5 yr and younger, in which there was a mean maximum increase of 0.23 mmol litre\(^{-1}\). Studies reported from our Burn Unit in 1970 showed that the increase in serum potassium concentration appeared to be related to the extent of the burn: an increase of 4.9 mmol litre\(^{-1}\) occurred on the 25th day post-burn in a child with 30% burns with only a very transient decrease in cardiac output as assessed by the intensity of heart sounds, while a boy with 10% burns showed an increase in potassium concentration of only 1.7 mmol litre\(^{-1}\) (Brown and Bishop, 1970).

Since the hazard of hyperkalaemia appeared to be related to the extent of the burn, and seemed to be greatest between 2 and 6 weeks after the burn, we set out to do a study (using suxamethonium 0.2 mg kg\(^{-1}\)) to assess the influences of the extent of the burn, and time following the thermal injury. Unexpectedly, one of the first patients became apnoeic following this dose. Thus we undertook this electromyographic study of responses of normal and burned patients to small doses of suxamethonium.

**PATIENTS AND METHODS**

Following the induction of anaesthesia with thiopentone two control groups (50 children each) were given suxamethonium, either 0.1 or 0.2 mg kg\(^{-1}\) diluted to 1 mg ml\(^{-1}\) in sterile water. The selection of burned patients was restricted to those who had an uninjured hand and forearm so that the EMG electrodes could be attached. Thirty-eight burned patients received suxamethonium 0.1 or 0.2 mg kg\(^{-1}\) on 59 occasions. Eleven patients who had repeated anaesthetics were studied on multiple occasions. In these patients the maximum response was used for comparison with the normal controls.

The integrated electromyograph (IEMG) was developed and constructed at the Department of Electrical Engineering at Monash University (Lam, Cass and Ng, 1981). Two stimulating electrodes were placed over the median nerve at the wrist, the recording electrodes over the opponens pollicis muscle and a reference electrode...
Injection 100 ~T 3
Time

FIG. 1. IEMG pattern showing how the measurements were made from the recording. 1 = Maximum depression in muscle activity (IEMG response). 2 = Time from injection to maximum IEMG depression. 3 = Time to 50% recovery. 4 = Time from 50% of total decrease in activity until recovery to the same level.

was placed on the forearm. A supramaximal stimulus at 1 Hz for 0.1 ms was used to stimulate the median nerve. The depression of the IEMG was recorded on a chart recorder. Measurements were made as illustrated in figure 1.

The non parametric Mann–Whitney U test was used for statistical analysis.

RESULTS

Thirty-eight patients were studied on 59 occasions. Suxamethonium 0.1 mg kg⁻¹ was given on 19 occasions (11 patients) and there was a statistically significant increase in paralysis (depression of muscle activity) and duration of action compared with controls (P < 0.001). There was a similar statistically significant difference between patients with burns receiving suxamethonium 0.2 mg kg⁻¹ compared with controls.

The results are summarized in table I and figure 2. The maximum responses were used for patients studied on multiple occasions.

Eleven patients were studied on two or more occasions. Only one of the eight patients given 0.2 mg kg⁻¹ had a maximum depression of muscle activity of less than 93%. Two patients given 0.1 mg kg⁻¹ had 83% and 93% maximum depression of muscle activity, respectively, while the third, in whom the decrease was 58%, was not studied until 16 days after the burn—which is later than the time of maximum depression seen in the other patients. This occurred between 4 and 12 days (mean 8 days).

Figures 3, 4 and 5 are illustrative of patients studied on multiple occasions. Figure 3 illustrates the depolarization response without any decrease in muscle activity following suxamethonium 0.1 mg kg⁻¹ in the early post-burn period. This phenomenon was also seen with this dose in some of the controls. Four days later the patient had a marked response to suxamethonium which lessened on subsequent occasions.

Figure 4 shows the marked responses on all occasions in an older patient with 10% burns receiving suxamethonium 0.2 mg kg⁻¹. Figure 5 shows the marked response to 0.2 mg kg⁻¹ on the first occasion, with a reduced response of shorter duration on the second occasion 16 days after the burn.

In general, the greater the degree of paralysis the longer was the duration of effect measured to the time of maximum depression, to 50% recovery and between the midpoints of the downslope and upslope of the IEMG traces (fig. 1).

**TABLE I. IEMG responses to suxamethonium in burns. Statistical significance compared with controls:**

<table>
<thead>
<tr>
<th>Group</th>
<th>Dose (mg kg⁻¹)</th>
<th>n</th>
<th>% max. depression in muscle activity</th>
<th>Time to max. depression (min)</th>
<th>Time to 50% recovery (min)</th>
<th>Time 50% dep. to 50% recovery (min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>0.1</td>
<td>50</td>
<td>5.3±1.6</td>
<td>0.22±0.21</td>
<td>0.43±0.15</td>
<td>0.37±0.11</td>
</tr>
<tr>
<td>Burns 20+ %</td>
<td>0.1</td>
<td>6</td>
<td>54.1±18.7***</td>
<td>0.79±0.18***</td>
<td>1.67±0.90</td>
<td>1.36±0.67</td>
</tr>
<tr>
<td>Control</td>
<td>0.2</td>
<td>50</td>
<td>56.6±22.2</td>
<td>0.63±0.19</td>
<td>1.23±0.39</td>
<td>0.99±0.34</td>
</tr>
<tr>
<td>Burns &lt; 10%</td>
<td>0.2</td>
<td>10</td>
<td>62.0±32.2</td>
<td>0.97±0.30***</td>
<td>2.19±1.29***</td>
<td>1.85±1.23***</td>
</tr>
<tr>
<td>Burns 11–19%</td>
<td>0.2</td>
<td>7</td>
<td>78.4±23.8*</td>
<td>0.94±0.37**</td>
<td>3.05±1.90**</td>
<td>2.72±1.91*</td>
</tr>
<tr>
<td>Burns 20+ %</td>
<td>0.2</td>
<td>10</td>
<td>76.4±24.2**</td>
<td>1.25±0.46**</td>
<td>3.61±1.70**</td>
<td>3.20±1.59***</td>
</tr>
</tbody>
</table>
SUXAMETHONIUM IN BURNS

Fig. 2. The mean IEMG recordings for control subjects and patients with burns after suxamethonium 0.1 mg kg\(^{-1}\) (dotted lines) and 0.2 mg kg\(^{-1}\) (solid lines). SD of maximum depression and time to 50% recovery shown.

Fig. 3. Degree and duration of paralysis in a 32-kg 6-yr-old (28% of burns) following suxamethonium 0.1 mg kg\(^{-1}\). It demonstrates the increase in sensitivity at 10 days after the burn.

DISCUSSION

Using small doses of suxamethonium, significant differences in response and duration of action were demonstrated in patients with burns compared with normal children. It was found also that patients with burns go through a phase of markedly increased sensitivity to suxamethonium some time between the 4th and 12th days after the burn. At this time the degree of paralysis following 0.2 mg kg\(^{-1}\) may cause apnoea and be sufficient to permit intubation of the trachea.

Viby-Mogensen and co-workers (1975a) demonstrated a decrease in plasma cholinesterase activity following thermal injury which correlated significantly with the extent of the burn. The decrease to minimum values occurred most rapidly in patients with a burn index greater than 30%. Most of this decrease occurred during the first 2 days after the injury. The duration of the depression in cholinesterase activity was some-
times prolonged and in some patients normal values were not regained for some weeks after full recovery.

Although the decrease in plasma cholinesterase concentration could result in an exaggerated response to suxamethonium, the fact that the maximum depression of IEMG activity occurs later suggests that it is not the only factor increasing the sensitivity to suxamethonium in burned patients.

Viby-Mogensen and co-workers (1975b) demonstrated a significant correlation between the maximum increase in serum potassium concentration and the extent of the burn when suxamethonium was given after the 14th day. They also noted that major increases before that time occurred only with very extensive burns. Our experience suggests that the greatest increases in serum potassium concentration occur during the 3rd and 4th weeks after the burn (Brown and Bishop, 1970). This is later than the time at which the maximum responses occurred in this study. In two of our patients who showed a markedly greater depression of muscle activity, blood samples taken each minute from 0 to 5 min after suxamethonium, showed no change in serum potassium concentration. In another child, with 30% surface area burns whose potassium concentrations were measured on nine occasions after suxamethonium 0.1 mg kg\(^{-1}\), plasma potassium concentration decreased until 15 days after the burn, when an increase of 0.3 mmol litre\(^{-1}\) occurred. The maximum increase in plasma potassium concentration (0.4 mmol litre\(^{-1}\)) occurred on the 26th day after the burn. The maximum depression of muscle activity (IEMG) occurred on day 12. It appears that the exaggerated neuromuscular blocking effects are not related to the hyperkalaemic response to suxamethonium.

We have shown that, after burns, children exhibit an increased degree and duration of paralysis following small doses of suxamethonium. They pass through a phase of exquisite sensitivity which occurs sometime between the 4th and 12th days after the burn, when they will become paralysed (and the trachea can be intubated) with doses as small as 0.2 mg kg\(^{-1}\). Although intubation can be performed with much reduced doses of suxamethonium—which will not cause dangerous increases in serum potassium concentration—it is not usually necessary to use this neuromuscular blocking drug, and it is generally advisable not to use suxamethonium in patients with large burns. Non-depolarizing drugs can be used, but larger doses than normal, particularly of tubocurarine, may be required (Brown and Fisk, 1979; Martyn et al., 1980).

The maximum changes in plasma cholinesterase activity, sensitivity to suxamethonium and potassium concentration after suxamethonium do not occur at the same times after the burn. Although the first two changes may have some interrelation, neither is related to the increase in the release of potassium.

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REFERENCES


