EFFECTS OF CALCIUM ON NEUROMUSCULAR BLOCK BY SUXAMETHONIUM IN DOGS

BY

R. P. BADOLA, S. CHATTERJI, K. PANDEY AND S. KUMAR

SUMMARY

The effects of intravenously administered calcium chloride on partial neuromuscular block produced by small and large doses of suxamethonium, tubocurarine and gallamine have been studied in dogs. Calcium chloride brought about an improvement in twitch tension, and this depression lasted for 5–15 minutes. The possible mechanisms of the block produced by small doses of suxamethonium. In the presence of a complete phase II block produced by suxamethonium, however, calcium chloride depressed the twitch tension, and this depression lasted for 5–15 minutes. The possible mechanism of the varying effects of calcium on different types of neuromuscular block are discussed and it is suggested that the depression of twitch by calcium in the presence of desensitization block may be due to a “membrane stabilizing action”. It is possible that this observation may prove useful clinically in differentiating desensitization block from other varieties of neuromuscular block.

The importance of calcium in synaptic transmission, particularly in neuromuscular transmission, is well recognized (Locke, 1894; Feng, 1936; Harvey and MacIntosh, 1940; del Castillo and Katz, 1954a, b). An anti-curare effect of ionic calcium was reported by del Castillo and Stark (1952). It is also known that calcium effectively antagonizes the myoneural blocking action of an excess of magnesium (del Castillo and Engbaek, 1954) and the neuromuscular block resulting from administration of such antibiotics as streptomycin, neomycin and kanamycin (Brazil and Corrado, 1957; Brazil, Corrado and Berti, 1959; Corrado, Ramos and DeEscobar, 1959; Jindal and Despande, 1960; Pandey, Kumar and Badola, 1964). Little is known, however, about the effect of calcium on the neuromuscular blocking action of depolarizing muscle relaxants, although Irwin, Wells and Whitehead (1956) studied the effect of the removal of ionic calcium by the administration of EDTA (ethylene-diamine-tetra-acetic acid) on the duration of apnoea induced by suxamethonium in dogs and rats.

In this paper an investigation into the effect of calcium on the myoneural block produced by suxamethonium is described, and the results are compared with the effect of calcium on the neuromuscular block due to tubocurarine and gallamine, under identical experimental conditions.

METHODS

Experiments were performed on mongrel dogs weighing 5–15 kg. Anaesthesia was produced by pentobarbitone sodium 30 mg/kg, given intravenously. The tibialis anterior muscle-nerve preparation was set up and muscle contraction recorded kymographically according to the method described by Pandey, Kumar and Badola (1964). Indirect stimulation of the muscle was produced by an electronic stimulator which delivered supramaximal square wave pulses.

After an initial recording of the response of the muscle to twitch (0.2, 0.4, 0.6, 0.8 and 1/sec) and tetanic (50/sec) rates of stimulation, suxamethonium, tubocurarine or gallamine was injected intravenously. The effect of calcium on the neuromuscular block produced by these agents was studied according to the following plan:

(1) In six experiments a small dose of suxamethonium (1–3 mg/kg) was given intravenously.

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This always abolished the twitch response of the muscle. When contractions reappeared, the responses to twitch and tetanus were recorded. Calcium 200–400 mg (2–4 ml of 10 per cent solution calcium chloride) was then injected intravenously while the muscle was being stimulated indirectly at a rate of 1/sec.

(2) In sixteen experiments a total of 5.5–13 mg/kg of suxamethonium was given in single or divided doses. The responses to twitch and tetanic rates of stimulation were recorded after each dose, and further doses were given until some or all of the following myographic features of a dual (or phase II) type of block appeared: (i) ill-sustained tetanus (1ST); (ii) post-tetanic facilitation (PTF); (iii) a progressive diminution of the amplitude of the successive responses at twitch rates of stimulation (fade).

As the effect of antagonists on dual block has been reported to vary with the degree of PTF and fade (Churchill-Davidson and Wise, 1963b) these were graded as follows:

Gradation of PTF: (1) mild when the amplitude of the post-tetanic twitch response was not more than 1.5 times the pre-tetanic response; (2) moderate when the response was greater than 1.5 times but less than twice the pre-tetanic response; (3) marked when the response was two or more times the pre-tetanic response (Churchill-Davidson and Katz, 1966).

Gradation of fade: (1) mild if the progressive decline in the amplitude of successive twitch responses was elicited only at some of the relatively fast twitch rates of indirect stimulation; (2) moderate if fade was elicited in the entire range of slow rates of stimulation, but the criterion for its being described as “marked” was not fulfilled; (3) marked if, in addition to being elicited at all the slow rates of stimulation, fade was so marked that the amplitude of the third to sixth response in any particular train of responses was reduced to between two-thirds and half of the first.

When IST, PTF and fade were observed together, the combination was termed a “triad” which was classified as “typical” when both PTF and fade were moderate or marked, and “atypical” when either of these was only mild.

Calcium chloride 200–400 mg was given intravenously in the presence of various combinations of the myographic features of a dual or phase II type of neuromuscular block during continuous indirect stimulation of the muscle at the rate of 1 or 0.8/sec. After allowing time for the effect of calcium to subside (5–15 min) neostigmine 0.5–1.5 mg was given intravenously with atropine sulphate 0.6 mg.

(3) In five experiments calcium chloride 200–400 mg was given after producing a partial neuromuscular block by tubocurarine. In five other experiments a similar dose of calcium chloride was given after producing partial neuromuscular block with gallamine triethiodide.

**RESULTS**

(1) **Effect of calcium on neuromuscular block produced by small doses of suxamethonium.**

The responses of the tibialis anterior, on partial recovery, after suxamethonium 1–3 mg/kg did not show any marked features of a dual type of neuromuscular block except for a mild degree of PTF in six experiments and an ill-sustained tetanus in three experiments. Calcium chloride did not appear to influence the rate of recovery of the muscle response (fig. 1).

(2) **Effect of calcium when there were features of a dual type of response.**

The “typical triad” resulted as often after single large doses of suxamethonium (table I, experiments 3, 12, 14, 15 and 16) as after repeated doses (experiments 6, 7, 9, 10 and 13). Repeated doses did not necessarily produce a more intense degree of phase II block (as in experiment 5). The different combinations of myographic characteristics of dual block, after relatively large doses of suxamethonium, which were present at the time of administration of calcium chloride are shown in table II.

Table III summarizes the observations on the effects of calcium chloride on the neuromuscular block in the presence of the various myographic features listed in table II. In the presence of a typical triad calcium chloride potentiated the existing neuromuscular block in all of ten experiments (fig. 2). The depressant action of calcium on twitch tension lasted for 5–15 minutes. In two of three experiments in which an atypical triad was present calcium potentiated the block (fig. 3) while in one no such effect was seen. Potentiation of the block after administration of calcium
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FIG. 1
(a) Tibialis anterior contractions (M) and arterial pressure (BP) recordings from an experiment in which suxamethonium 2 mg/kg was given at (S). There is no fade of twitch tension at various slower rates of stimuli. The tetanus at (T) is ill-sustained. There is no PTF.
(b) Calcium chloride (200 mg) given during continuous indirect stimulation at the rate of 1/sec produced no effect on the twitch tension. Administration of neostigmine methylsulphate (NMS) also did not influence the rate of recovery of the twitch tension.

FIG. 2
Recording of the tibialis anterior contractions of a dog which was given suxamethonium (Sux) 10.5 mg/kg in a single dose. On partial recovery a marked fade of twitch tensions at slow rates of indirect stimulation (0.2–1/sec) is seen. The tetanic response is hardly discernible (very ill-sustained). There is a marked PTF. This combination of marked fade, marked PTF and ill-sustained tetanus (IST) was termed the typical triad. Calcium chloride 300 mg given later depressed the twitch tension. Two doses of neostigmine methylsulphate (NMS) had no effect.

FIG. 3
Tibialis anterior contractions in response to indirect supramaximal stimulation. On partial recovery from suxamethonium 10 mg/kg, mild fade, mild PTF and an ill-sustained tetanus are seen (an atypical triad). Calcium chloride caused a depression of the twitch tension. Neostigmine methylsulphate (NMS) had no effect.

FIG. 4
Recordings of tibialis anterior contractions (M), intratracheal pressure excursions (R) and time (T) (10 sec), from an experiment in which a total dose of 10.8 mg/kg of suxamethonium had been given in divided doses. A mild fade of twitch tensions at 0.8 and 1/sec is seen (a). The tetanic response (T) is ill-sustained. There is no post-tetanic facilitation (PTF). Administration of calcium chloride during stimulation of the muscle at 0.8/sec caused depression of twitch tension. Neostigmine methylsulphate produced no effect (b).
### Table I

Myographic characteristics after large single or divided doses of suxamethonium and the effects of calcium and neostigmine in presence of these characteristics.

<table>
<thead>
<tr>
<th>Experiment No.</th>
<th>Wt. of dog (kg)</th>
<th>Pre-treatment muscle response</th>
<th>Initial dose of suxamethonium (mg/kg)</th>
<th>Duration of total unresponsiveness of the muscle (min)</th>
<th>Total dose of suxamethonium (initial and subsequent doses) (mg/kg)</th>
<th>Myographic characteristics on recovery from last dose</th>
<th>Myographic characteristics on administration of calcium</th>
<th>Combination of myographic characteristics prior to administration of calcium</th>
<th>Dose of calcium chloride (mg)</th>
<th>Effect of calcium on neuromuscular block</th>
<th>Dose of neostigmine (mg)</th>
<th>Effect of neostigmine on twitch tension</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>15.00</td>
<td>fade ist PTF</td>
<td>6.6</td>
<td>80</td>
<td>13.3</td>
<td>++ + Yes +</td>
<td>+ + + Yes +</td>
<td>Atypical triad</td>
<td>300</td>
<td>P</td>
<td>2.0</td>
<td>NC</td>
</tr>
<tr>
<td>2</td>
<td>9.25</td>
<td>fade ist PTF</td>
<td>8.1*</td>
<td>60</td>
<td>13.3</td>
<td>++ + Yes +</td>
<td>+ + + Yes +</td>
<td>Typical triad</td>
<td>300</td>
<td>P</td>
<td>1.5</td>
<td>NC</td>
</tr>
<tr>
<td>3</td>
<td>8.50</td>
<td>fade ist PTF</td>
<td>5.8*</td>
<td>60</td>
<td>9.3*</td>
<td>++ Yes +</td>
<td>+ + + Yes +</td>
<td>Typical triad</td>
<td>300</td>
<td>P</td>
<td>1.5</td>
<td>NC</td>
</tr>
<tr>
<td>4</td>
<td>8.00</td>
<td>fade ist PTF</td>
<td>9.3*</td>
<td>40</td>
<td>12.5</td>
<td>++ Yes +</td>
<td>+ + + Yes +</td>
<td>Typical triad</td>
<td>300</td>
<td>P</td>
<td>1.0</td>
<td>NC</td>
</tr>
<tr>
<td>5</td>
<td>8.00</td>
<td>fade ist PTF</td>
<td>11.1</td>
<td>45</td>
<td>21.8</td>
<td>++ Yes +</td>
<td>+ + + Yes +</td>
<td>Typical triad</td>
<td>300</td>
<td>P</td>
<td>1.0</td>
<td>NC</td>
</tr>
<tr>
<td>6</td>
<td>9.00</td>
<td>fade ist PTF</td>
<td>5.5*</td>
<td>40</td>
<td>11.1</td>
<td>++ Yes +</td>
<td>+ + + Yes +</td>
<td>Typical triad</td>
<td>300</td>
<td>NC</td>
<td>1.0</td>
<td>NC</td>
</tr>
<tr>
<td>7</td>
<td>9.00</td>
<td>fade ist PTF</td>
<td>5.5</td>
<td>20</td>
<td>13.3</td>
<td>++ + Yes +</td>
<td>+ + + Yes +</td>
<td>Typical triad</td>
<td>300</td>
<td>P</td>
<td>0.5</td>
<td>NC</td>
</tr>
<tr>
<td>8</td>
<td>5.00</td>
<td>fade ist PTF</td>
<td>5.0</td>
<td>80</td>
<td>6.2</td>
<td>++ Yes +</td>
<td>+ + + Yes +</td>
<td>Typical triad</td>
<td>200</td>
<td>P</td>
<td>0.5</td>
<td>NC</td>
</tr>
<tr>
<td>9</td>
<td>13.00</td>
<td>fade ist PTF</td>
<td>7.6*</td>
<td>40</td>
<td>10.5</td>
<td>++ + Yes +</td>
<td>+ + + Yes +</td>
<td>Typical triad</td>
<td>400</td>
<td>P</td>
<td>1.0</td>
<td>NC</td>
</tr>
<tr>
<td>10</td>
<td>13.00</td>
<td>fade ist PTF</td>
<td>7.6</td>
<td>35</td>
<td>13.3</td>
<td>++ + Yes +</td>
<td>+ + + Yes +</td>
<td>Typical triad</td>
<td>400</td>
<td>P</td>
<td>1.0</td>
<td>NC</td>
</tr>
<tr>
<td>11</td>
<td>12.00</td>
<td>fade ist PTF</td>
<td>8.3*</td>
<td>50</td>
<td>10.5</td>
<td>++ + Yes +</td>
<td>+ + + Yes +</td>
<td>Typical triad</td>
<td>300</td>
<td>P</td>
<td>1.0</td>
<td>NC</td>
</tr>
<tr>
<td>12</td>
<td>8.50</td>
<td>fade ist PTF</td>
<td>5.8*</td>
<td>60</td>
<td>50</td>
<td>++ Yes +</td>
<td>+ + + Yes +</td>
<td>Typical triad</td>
<td>300</td>
<td>P</td>
<td>1.0</td>
<td>NC</td>
</tr>
<tr>
<td>13</td>
<td>8.00</td>
<td>fade ist PTF</td>
<td>6.2*</td>
<td>40</td>
<td>10.5</td>
<td>++ + Yes +</td>
<td>+ + + Yes +</td>
<td>Typical triad</td>
<td>400</td>
<td>P</td>
<td>1.0</td>
<td>NC</td>
</tr>
<tr>
<td>14</td>
<td>Yes</td>
<td>fade ist PTF</td>
<td>5.8*</td>
<td>60</td>
<td>13.3</td>
<td>++ + Yes +</td>
<td>+ + + Yes +</td>
<td>Typical triad</td>
<td>300</td>
<td>P</td>
<td>1.0</td>
<td>NC</td>
</tr>
</tbody>
</table>

**IST**—ill-sustained tetanus.

**PTF**—post-tetanic facilitation.

**P**—potentiation of the existing neuromuscular block.

**NC**—no change in twitch tension attributable to the treatment.

* + = mild, ++ = moderate and +++ = marked fade or PTF.

* Single dose only.
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TABLE II

<table>
<thead>
<tr>
<th>Dose of suxamethonium (mg/kg)</th>
<th>No. of dogs</th>
<th>Typical triad*</th>
<th>Atypical triad*</th>
<th>IST and PTF</th>
<th>IST and fade</th>
<th>PTF and fade</th>
<th>Only IST</th>
<th>Only PTF</th>
<th>Only fade</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.0–6.0</td>
<td>5</td>
<td>4</td>
<td>1</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>6.1–8.0</td>
<td>6</td>
<td>4</td>
<td>2</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>8.1–10.0</td>
<td>3</td>
<td>1</td>
<td>–</td>
<td>2</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>10.1 or more</td>
<td>2</td>
<td>1</td>
<td>–</td>
<td>1</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

IST = ill-sustained tetanus.
PTF = post-tetanic facilitation.
* For explanations of typical and atypical triads see description in section on methods.

TABLE III

Effects of calcium chloride and neostigmine methylsulphate on neuromuscular block by suxamethonium, showing various combinations of the myographic characteristics of a dual type block.

<table>
<thead>
<tr>
<th>Combination of myographic characteristics</th>
<th>Number of dogs</th>
<th>Effect of calcium on the block</th>
<th>Effect of neostigmine methylsulphate on the block</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical triad*</td>
<td>10</td>
<td>Potentiation 10</td>
<td>Reversal 3</td>
</tr>
<tr>
<td>Atypical triad*</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Fade and IST</td>
<td>3</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

* See section on method for explanation of the terms typical and atypical triads.
** Neostigmine methylsulphate was not given after calcium in one of these experiments.
IST = ill-sustained tetanus.

chloride occurred in presence of IST and fade in two of the experiments where this combination was present (fig. 4).

The combination of IST and fade in three experiments reported here conflicts with the statements of previous workers that IST and PTF appear in that order much earlier than the fade of the twitch responses (Churchill-Davidson and Christie, 1959; Churchill-Davidson, Christie and Wise, 1960; Churchill-Davidson, 1963; Churchill-Davidson and Wise, 1963a, b; Katz, Wolf and Papper, 1963).

In fifteen of sixteen experiments neostigmine given 5–15 minutes after the administration of calcium chloride did not appear to exert any influence on the rate of recovery of the twitch response nor did it potentiate the existing neuromuscular block (table III; figs. 2, 3, 4).

(3) Effects of calcium on the block produced by tubocurarine and gallamine triethiodide.

Calcium always brought about an appreciable improvement in the twitch tension in the presence of neuromuscular block produced by either of the non-depolarizing relaxants (figs. 5, 6). However, this improvement was never as marked as has been reported after administration of calcium chloride in the presence of neuromuscular block caused by neomycin (Corrado, Ramos and De Escobar, 1959) or streptomycin (Pandey, Kumar and Badola, 1964).

DISCUSSION

The importance of calcium in synaptic transmission and in regulation of effector cell excitability is widely recognized but the mechanism of its effect has not been fully elucidated. A review of the literature (Brink, 1954; Shanes, 1958a, b; Katz and Miledi, 1965; Ritchie and Greengard, 1966; Karczmar, 1967) suggests that ionic calcium could modify muscle twitch tension in three different ways under different conditions. First, ionic calcium might promote acetylcholine (ACh) release by activating the discharge of the contents of the synaptic vesicles after arrival of an action potential (NAP) at the terminal axonal membrane (Koelle, 1965). Secondly, calcium could modify effector cell response through its regulatory action.
FIG. 5
Tibialis anterior contractions (M), blood pressure (BP) and time, every 5 sec (T) recordings from an experiment in which two doses of tubocurarine (1.25 mg) were given at arrows. Partial neuromuscular block was produced. Calcium chloride and neostigmine methylsulphate (NMS) both produced improvement of twitch tension.

on the permeability of the post-junctional membrane to sodium and potassium ions, the conductance of these ions across the post-junctional membrane varying in inverse proportion to the concentration of ionic calcium in the external medium. Thus an excess of calcium in the external medium would raise the threshold of excitation of the post-junctional membrane in response to the presynaptically released ACh; the endplate potentials following ACh release are not able to attain the threshold value required for the generation of a propagated muscle action potential. This action of calcium has been termed the membrane stabilizing action. Thirdly, calcium has also been stated to be of vital importance in excitation-contraction coupling. It is assumed that after depolarization of the muscle fibre membrane, ionic calcium is released from intracellular binding sites, enters muscle cytoplasm and sets off a chain of as yet unelucidated reactions resulting in the shortening of the contractile protein actomyosin (Sandow, 1965).

An explanation for the varying effects of calcium chloride on the indirectly elicited twitch tension under partial neuromuscular block of different types could be offered in the light of some of these known actions of ionic calcium on junctional transmission and effector cell excitability.

A facilitatory action of calcium on the presynaptic release of ACh might overcome any depression of the twitch tension resulting from a competitive receptor blockade or from an interference with the actual process of release of the transmitter. On the basis of this action of calcium the antagonistic action of this ion towards nondepolarizing relaxants was explained by del Castillo and Stark (1952). During the present experiments, however, the reversal of non-depolarization block by calcium was not very prominent, probably because there was little further scope for facilitation of ACh release while using supramaximal stimulation of the motor nerve. On the other hand, if the neuromuscular block is caused by drugs and ions which directly interfere with the process of the release of ACh (e.g., streptomycin and magnesium) the improvement in the twitch tension is very marked (Pandey, Kumar and Badola, 1964; Engbaek, 1952).

The depression of the twitch tension by calcium in presence of typical myographic features of dual block induced by suxamethonium requires explanation. The theory of the mechanism of neuromuscular blocking action of suxamethonium envisages that whenever a muscle is exposed to a depolarizing agent the endplate membrane is initially depolarized, and as long as the post-junctional membrane depolarization lasts the muscle fibre remains absolutely unresponsive to presynaptic stimulation (Thesleff, 1955; Paton, 1956; Gissen and Nastuk, 1966; Nastuk, 1966, 1967). The potential difference across the post-junctional membrane is then gradually restored despite the continued presence of the depolarizing agent. This restoration of the potential difference, however, is not accompanied by a parallel restoration of muscle responsiveness to presynaptic stimulation. The continued presence of the depolarizing agent makes the post-junctional membrane increasingly insensitive to the depolarizing action of the natural transmitter, ACh (Thesleff, 1955; Gissen and Nastuk, 1966). It has been shown that during a continuous intra-arterial infusion of suxamethonium at a constant rate the twitch tension...
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Tibialis anterior contractions (M) and blood pressure recordings from an experiment in which neuromuscular block was produced by two consecutive doses (1 mg/kg each) of gallamine triethiodide given at arrows marked G. Typical myographic features of a non-depolarization block are seen. The first dose of calcium chloride (200 mg) given at C did not cause any remarkable change in the twitch tension. The second dose produced an appreciable improvement. Even neostigmine methylsulphate (NMS) did not produce any dramatic change in the twitch tension. Time interval between the end of upper and the beginning of lower tracing = 7 minutes.

FIG. 6

The depression of twitch tension by calcium, observed in this study, can be explained on the assumption that this depression occurred in experiments where there was some desensitization of the post-junctional membrane to the depolarizing action of ACh. On this basis it has been suggested that "desensitization block" would be a better term to indicate the later phase of the neuromuscular block by suxamethonium.

The work of Nastuk (1966) and Manthey (1966) indicates that the concentration of calcium in the external medium plays an important role in the develop-

FIG. 7

Muscle twitch tension during continuous exposure to a depolarizing agent (e.g., suxamethonium). The tension output shows an initial steep depression followed by partial recovery and a secondary phase of depression. (Modified from Gissen, A. J., and Nastuk, W. L. (1966). Ann. N.Y. Acad. Sci., 135, 185, fig. 1; reprinted by permission of The New York Academy of Sciences.)
ment of desensitization of the receptors for acetylcholine at the neuromuscular junction.

Antagonism of calcium towards pure depolarization block has been claimed to occur by Irwin, Wells and Whitehead (1956) but could not be confirmed in the present study. The rate of recovery of the twitch tension after a small dose of suxamethonium was so fast that a slight degree of antagonism could not possibly be detected with the experimental technique employed by us.

The lack of any considerable antagonism to or potentiation of established desensitization block by neostigmine is also noteworthy. The use of this agent in the management of suxamethonium apnoea has been reported to have produced varying effects on the state of neuromuscular transmission (Vickers, 1963).

Three factors may account for the failure to demonstrate any reversal of desensitization block by neostigmine in the present study: first, too small a dose of neostigmine; secondly, its administration too late during recovery; or thirdly, the junctional stabilizing action of calcium previously administered. As the doses of neostigmine were considerable and the recovery of twitch tension in most experiments was far from complete at the time of administration of neostigmine, only the last factor appears likely.

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EFFETS DU CALCIUM SUR LE BLOCAGE NEUROMUSCULAIRE INDUIT PAR LE SUXAMETHONIUM CHEZ LE CHIEN

SOMMAIRE
On a étudié chez le chien, les effets de l'injection intraveineuse de chlorure de calcium sur le blocage neuromusculaire partiel induit par des doses minimales et importantes de suxaméthonium, de tubocurarine et de gallamine. Le chlorure de calcium entraîne une amélioration de la tension spastique en présence d'un blocage non dépolarisant, mais n'exerce aucun effet notable sur le blocage induit par de faibles doses de suxaméthonium. En présence d'un blocage complet à la phase II, induit par le suxaméthonium, le chlorure de calcium a néanmoins exercé un effet dépressif sur la tension spastique et cette dépression a duré 5 à 15 minutes. Le mécanisme possible des effets variables du calcium sur différents types de blocage neuromusculaire fait l'objet d'une discussion et il est suggéré que la dépression du clonus sous l'effet du calcium, en présence d'un blocage désensitif, peut être due à un "effet stabilisant sur la membrane". Il est possible que cette observation s'avère être utile sur le plan clinique, en vue de la différenciation d'un blocage désensitif par rapport aux autres variétés de blocage neuromusculaire.


Effekts del calcio sobre el bloqueo neuromuscular por suxametonio en perros

RESUMEN
Los efectos del cloruro de calcio administrado intravenosamente sobre el bloqueo neuromuscular parcial producido por dosis pequeñas y grandes de suxametonio, tubocurarina y gallamina fueron estudiados en perros. El cloruro de calcio produjo una mejoría en la tensión de contracción en presencia de un bloqueo no despolarizante, pero no ejerció ningún efecto evidente sobre el bloqueo producido por pequeñas dosis de suxametonio. Sin embargo, en presencia de un bloqueo completo de fase II producido por suxametonio, el cloruro de calcio deprimió la tensión de contracción y esta depresión duró 5–15 minutos. Es discutido el posible mecanismo de los efectos variables de calcio sobre tipos diferentes de bloqueo neuromuscular y se postula que la depresión de la contracción por el calcio en presencia de bloqueo por desensibilización pudiera ser debida a una "acción estabilizadora de membranas". Es posible que esta observación resulte ser clínicamente útil para diferenciar el bloqueo de desensibilización de otras variedades de bloqueo neuromuscular.