EFFECT OF MAGNESIUM ON VENTRICULAR FIBRILLATION DUE TO HYPOTHERMIA

BY

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SUMMARY

Magnesium was intravenously administered in twenty-seven cases of ventricular fibrillation due to hypothermia. In two-thirds of the cases defibrillation took place following the magnesium administration, while in the remaining cases the effect of electric defibrillation was facilitated. The possible mechanism of the anti-arrhythmic influence of magnesium is briefly discussed.

Ventricular fibrillation in hypothermia develops at an oesophageal temperature of about 27°C. During heart surgery performed under cardiopulmonary bypass, fibrillation at this temperature is noted as a rule. Under such circumstances, however, this otherwise fatal irregularity, in fact, facilitates the work of the surgeon, since the operation can be performed on an immobile heart. Electrical defibrillation used during rewarming of the patient generally does not present difficulties. In certain cases, as in major myocardial injury accompanied by disorders of acid-base balance, and disturbances of the coronary circulation, defibrillation may be achieved with difficulty, and may be successful only after several attempts or may even sometimes be impossible. In an attempt to facilitate defibrillation, we put to trial the well-known anti-arrhythmogenic effect of magnesium in ventricular fibrillation (Antalóczy, 1965; Enselber, Simmonds and Mintz, 1950; Loeb et al., 1963; Surawitz, 1966; Szekeres, 1954).

METHOD

The temperature of the patients was lowered during the cardiopulmonary bypass to below 27°C, when hypothermia led to ventricular fibrillation. After the operation the temperature was gradually raised to 30°C and when this temperature was reached magnesium sulphate 0.1 g/kg was rapidly administered intravenously.

RESULTS

Defibrillation was noted in eighteen of twenty-seven patients (66.7 per cent) 3-9 minutes following the infusion of magnesium; in nine cases one single application of electric defibrillation* was also necessary. Comparing these data with those obtained from forty-one patients with ventricular fibrillation not treated with magnesium, it was noted that at a temperature of 30°C spontaneous defibrillation occurred only in eight cases (19.5 per cent). Of the thirty-three patients defibrillated with electricity, repeated defibrillation was necessary in fourteen cases: in one case four times, in six cases three times and in seven cases twice.

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\text{TABLE I}
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<th>Treated with MgSO₄</th>
<th>Untreated</th>
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<tr>
<td>No. of cases with ventricular fibrillation</td>
<td>27</td>
<td>41</td>
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<tr>
<td>Defibrillation without electric impulses</td>
<td>18 (66.7%)</td>
<td>8 (19.5%)</td>
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<tr>
<td>Defibrillation with electric impulses</td>
<td>9 (33.3%)</td>
<td>33 (80.5%)</td>
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Serum magnesium was measured in fifteen cases. It was observed that defibrillation took place when the magnesium level in the serum reached 4.5–5.0 m.equiv/l. Postoperatively the serum-magnesium level returned to its normal value of 1.7–2.0 m.equiv/l. after 18–24 hours.

The defibrillation due to magnesium shows a characteristic electrocardiogram (fig. 1). The fibrillation at high frequency and small amplitude

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gradually slows, the amplitude of individual fibrillation waves increase, and with the further decrease of frequency cardiac rhythm returns to normal.

30°C

N MgSO₄ 5 7 8.5 15 min

Fig. 1

**DISCUSSION**

The effect of magnesium on cardiac function is little known (Elkinton, 1957; Gabor and Soldi, 1953; Hoffmann and Cranefield, 1964; Laks and Elek, 1967; Rigó, 1965). Several authors observed that in hypomagnesia the Q-T time was prolonged, the T wave deformed, a U wave appeared, and the ventricular automatism sometimes showed an increase (Enselber, Simmonds and Mintz, 1950; Seta et al., 1966; Székely, 1946; Székely and Wynne, 1951). Loeb and associates (1968) observed during hypomagnesia a paroxysmal ventricular fibrillation in alcoholics and in patients treated with saluretics. Others noted cardiac arrest under the influence of stress and in alkalosis (Bates, Adamson and Pierce, 1966).

It is a well-known fact that in magnesium deprivation ventricular automatism increases; the ultimate cause of the appearance of the inferior pacemaker activity, however, is still an unsolved problem. Wang and Welt (1963) postulated that in hypomagnesia adenosine triphosphatase activity decreased, resulting in loss of intracellular potassium associated with an increased activity of the inferior pacemaker (Laks and Elek, 1967; Seta et al., 1966; Székely, 1946; Székely and Wynne, 1951).

Our trial of the use of magnesium in cases of hypothermic ventricular fibrillation was based on the observations cited. According to our experiences magnesium decreased the disturbed activity of the pacemaker and furthered defibrillation. The anti-arrhythmic effect of magnesium asserted itself also in the control of postoperative rhythm disorders and facilitated treatment with digitalis (Enselber, Simmonds and Mintz, 1950; Székely, 1946; Székely and Wynne, 1951).

**REFERENCES**

L'EFFET DU MAGNESIUM SUR LA FIBRILLATION VENTRICULAIRE, DUE A L'HYPOTHERMIE

SOMMAIRE
Du magnésium a été administré par voie intraveineuse dans vingt-sept cas de fibrillation ventriculaire, due à l'hypothermie. Dans deux tiers des cas, on observe une défibrillation après l'administration de magnésium, qui d'autre part facilite dans les cas restants la défibrillation électrique. L'auteur discute brièvement le mécanisme possible de l'effet antiarrhythmique du magnésium.

MAGNESIUMWIRKUNG UND KAMMERFLIMMERN WÄHREND HYPOTHERMIE

ZUSAMMENFASSUNG

CORRESPONDENCE

UNSTEADINESS AFTER RELAXANT ANAESTHESIA

Sir,—It has been our experience that considerable unsteadiness persists after the use of a non-depolarizing relaxant, even after neostigmine has produced good return of muscle power. This unsteadiness precludes the use of relaxants in outpatient anaesthesia. Therefore we have investigated the possibility that this ill-effect might be due to the persistence of a small concentration of relaxant, sufficient to block, preferentially, the gamma efferent nerve endings only, consequently upsetting the stretch reflexes.

This hypothesis was tested in one conscious subject (M.E.W.) by dropping a hammer from a standard height on to the patellar tendon. The amplitude of the resulting knee jerk was recorded on a card by means of a felt-tipped pen strapped to the foot. Gripping a dynamometer with the hand immediately before the elicitation of the knee jerk causes an increased discharge in the gamma motor neurones. This increases the sensitivity of the muscle spindles and an augmented jerk results (Bullen and Dornhorst, 1957). In a control series of recordings the mean amplitude of the knee jerk was 17.5 mm without reinforcement and 56.4 mm with reinforcement. Then gallamine 20 mg was injected intravenously, followed by another 20 mg 7 minutes later. The accumulated dosage was sufficient to paralyse the small muscles of the eyes, hand and throat. A further series of recordings showed that there was no significant change in the amplitude of the reflex, either with or without reinforcement (table I). This indicates that the gamma motor neurone endings were not blocked by the relaxant. Only one subject was used because of the nature of the experiment but the results were quite definite.

An unsteady feeling, reminiscent of motion sickness, was experienced for 2 hours after the injection while the subject attempted to carry out his normal activities. Throughout this period there was difficulty in maintaining visual fixation, as has been neatly demonstrated by Hannington-Kiff (1970). Therefore it would seem likely that the unsteadiness is due to ocular instability rather than to an effect on the muscle spindles.

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REFERENCES