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Bradyarrhythmia induced by repeated dose of succinylcholine

To the Editor:

We read with interest the article by Nigrovic¹ concerning his hypothesis on the mechanism of adverse haemodynamic reactions to succinylcholine. When Nigrovic discusses bradyarrhythmia following a repeated dose of succinylcholine, he points out the study of Mathias *et al.*² who concluded that pre-curarisation with a non-depolarizing drug such as d-tubocurarine can prevent this phenomenon, an opinion usually accepted by the anaesthetist.³

We would like to emphasize that this prevention of bradyarrhythmia is not the case. We recently did a double blind study which included 24 women, ASA physical status I, in which we examined the efficacy of diazepam in preventing bradyarrhythmias induced by a second dose of succinylcholine (1 mg·kg⁻¹). The patients were randomly divided into three groups of eight patients each according to the pre-treatments received: G-I, intravenous diazepam (0.05 mg·kg⁻¹), G-II, d-tubocurarine (0.06 mg·kg⁻¹) and G-III, placebo (NaCl 0.9 per cent). Four cases of sinus arrest (3.2 to 14 sec) (G-I = 1, G-II = 2, G-III = 1) and two of bradycardia of less than 50 beats per minute (in Group III) followed the reinjection of succinylcholine. In fact, succinylcholine reinjection is a dangerous procedure and neither

intravenous d-tubocurarine nor intravenous diazepam provide adequate protection.

In view of the literature on this particular side effect of succinylcholine reinjection, we feel that there is at present no protection against this potentially harmful side-effect, except for the possible protective effect of glycopyrrolate.⁴

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REPLY

Thank you for the opportunity to answer the interesting letter of Drs. Martin and Léna.

When commenting on the explanation of Mathias *et al.*¹ about the mechanism by which non-depolarizing muscle relaxants alter cardiac effects of a repeated dose of succinylcholine, I pointed out² its shortcomings. It is my belief that this explanation cannot be the sole answer for the clinical observations of bradycardia or bradyarrhythmia.

On the other hand, published clinical reports, e.g., McLeskey *et al.*,³ and the data reported by Martin and Léna, show that "precuarization" does not reliably protect against the adverse cardiovascular effects seen on repeated administration of succinylcholine. These findings, however, can be easily interpreted in terms of the proposed hypothesis.² Inactivation of the nicotinic receptors on sympathetic terminals by (a) the previous administration of succinylcholine (see Fig. 2 in²) and/or (b) precuarization, leaves the succinylcholine-induced activation of the cardiac muscarinic receptors, with its