et al., 1974). Nor would I care to recommend its use in all circumstances. For example, in certain cases of ankylosing spondylitis, trismus and carcinoma of the tongue the airway may be lost, never to be regained once general anaesthesia is induced. In such situations I would prefer to perform endotracheal intubation with the patient awake, and if unsuccessful proceed with an inhalation agent to induce the very lightest plane of anaesthesia before further attempts at blind nasal intubation. A surgeon, prepared to perform a tracheostomy if necessary, should be present during the induction of anaesthesia.

In drawing attention to these considerations, my aim is not to discourage anaesthetists from practising this technique. Indeed, it is only familiarity with the technique of blind nasal intubation which enables the anaesthetist to confront difficult cases with confidence. This familiarity must come from constant practice with straightforward cases using well-established techniques or modifications such as that employing propanidid described by Dr Oyegunle and previously by both Atkinson (1971) and myself.

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REFERENCES


ABSENCE OF SUXAMETHONIUM FASCICULATIONS IN PATIENTS WITH ATYPICAL PSEUDOCHELINESTERASE

Sir,—I would like to sound a note of caution after reading with interest Dr Baraka’s letter regarding the absence of fasciculations in patients with abnormal pseudocholinesterase (Baraka, 1975).

Recently, I anaesthetized a healthy, 32-year-old female for elective Caesarean section. Following pre-oxygenation, anaesthesia was induced with atripeine 0.6 mg, methohexitone 100 mg and suxamethonium 100 mg i.v. Such marked, generalized fasciculations occurred that I commented to the theatre technician on the classic display! Tracheal intubation was performed without difficulty and the patient was ventilated artificially. Muscle tone was not regained during the period of the operation and the patient remained paralysed for 2 hr 40 min. Artificial ventilation with nitrous oxide and oxygen was therefore continued for this period of time, and when the patient regained sufficient muscle power to ventilate adequately, the trachea was extubated. No nerve stimulation tests were performed at this time; edrophonium and neostigmine were not administered. Subsequently, she made an uneventful recovery. Questioning revealed that she had been anaesthetized only for dental extractions in the past and she knew of no resulting complications.

Biochemical investigations revealed the following results, compatible with abnormal pseudocholinesterase activity:

- Dibucaine no. 30
- Fluoride no. 5
- Chloride no. 70

Following this experience, I would be reluctant to follow Dr Baraka’s advice. In future I shall refrain from administering a non-depolarizing muscle relaxant after suxamethonium, until signs of returning muscle tone have become apparent, regardless of the presence or absence of muscle fasciculations.

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REFERENCE


Sir,—I read with interest the report of Dr Hunter on the generalized marked fasciculations which followed induction of anaesthesia with methohexitone and suxamethonium in a patient with atypical plasma cholinesterase activity. Such marked fasciculations can be attributed to the depolarizing activity of suxamethonium. However, this observation differs from our previous findings in five patients who were atypical homozygotes, and who did not show any fasciculations following the injection of suxamethonium (Baraka, 1975).

Muscular excitatory phenomena following the induction of anaesthesia with an hypnotic-suxamethonium sequence may result from the central excitatory effect of the hypnotic or the peripheral depolarizing activity of the suxamethonium, or both.

Induction of anaesthesia with methylated barbiturates, particularly in non-premedicated patients, can be associated with tremor, spontaneous involuntary muscle movements or hypertonus (Dundee, 1965). The patient reported by Dr Hunter was given methohexitone, and the marked excitatory phenomena observed may be related to this anaesthetic induction agent.

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REFERENCES
