NEUROMUSCULAR BLOCKERS BEFORE THIOPENTONE

Sir,—I was interested to read of the negative findings in the article by Levack and Spence [1], since I have been firmly convinced, from experience, that giving tubocurarine before thiopentone results in more favourable intubating conditions with minimum doses of both drugs. Atracurium and alcuronium appear to behave similarly. This is a very common belief among experienced anaesthetists and the reason put forward for the difference is that both drug actions peak together. I would suggest that, in addition, the redistribution of cardiac output that occurs with unconsciousness, as a result of reduction of normal vasoconstrictor tone in the skin blood vessels, results in slightly less of an administered drug going subsequently to the muscles. It would be of interest to know the effect of 50% nitrous oxide, which they used, on skin blood flow, as this might divert some neuromuscular blocker from the muscles in the same way.

The writers took care to point out that the end point they sought was a 90% depression in T1, and not the earliest onset of intubation conditions. The degree of muscle paralysis is related to the frequency of impulses arriving at the muscle, which depends on inactivation of the central nervous system in addition to depression of the end-plate. While 90% depression in T1 was awaited, the depth of anaesthesia may have waned. A further source of error could be the late arrival of drugs in the wrist where T1 depression was estimated, compared with the area of interest in the neck.

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REFERENCES

I still feel that there is a worthwhile advantage to be gained by injecting the neuromuscular blocker first in selected cases, but recommend it only if the patient has two "good" veins and the anaesthetist has two syringes of thiopentone prepared.

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REFERENCE

HAEMODYNAMIC EFFECTS OF PROPOFOL

Sir,—The study by Claey and his colleagues [1] on the haemodynamic effects of propofol anaesthesia showed that hypotension was a prominent feature of the use of this agent, and that this pressure decrease was caused by peripheral vasodilatation. In some circumstances deliberately induced hypotension is a feature of anaesthesia, and propofol could have a place in such techniques, perhaps permitting the dose of vasodilator agents such as nitroprusside to be reduced. In particular, the use of propofol might confer an advantage since, despite the occurrence of systemic hypotension, this was unaccompanied by pulmonary arterial hypotension. Pulmonary arterial hypotension and attendant impairment of gas exchange is a common feature of the use of vasodilators such as sodium nitroprusside and, to a lesser extent, nitroglycerin, in normal patients [2].

Unfortunately, data on arterial oxygenation are not given in the paper by Claey [1], despite the sampling of arterial blood and reporting of other values from analysis of these samples. It is difficult to understand why oxygen tension values were not considered to be important, since the authors commented on moderate respiratory impairment. Such data would be of interest on clinical grounds alone, since the mean age of the patients was 62 yr, some were ASA III, and they breathed air during anaesthesia.

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REFERENCES

Sir,—The results of our study revealed arterial hypotension mainly as result of decreased systemic vascular resistance (SVR), without a compensatory increase in heart rate or cardiac output. No pulmonary arterial hypotension was observed and Pao2 remained unchanged (Pao2 before propofol: 10.59 ± 1.8 kPa; Pao2 at 45 min: 9.78 ± 1.77 kPa (mean ± SEM)). The hypotensive effect of propofol disappeared rapidly after cessation of the propofol infusion. In our study all patients breathed room air spontaneously, and the measure-