Traces obtained under identical circumstances as those in figure 1. Premedication with papaveretum and hyoscine. Methohexitone induction and maintenance of anaesthesia with 1 to 2 per cent halothane in oxygen. Healthy orthopaedic patients aged 16 to 34. In every case the arrhythmia followed a repeated dose of 25 mg suxamethonium. The trachea was not intubated in these cases. Paper speed in top three traces 10 mm/sec. Paper speed in bottom trace 25 mm/sec, with negative polarity of the exploring electrode. The bottom trace is from the only patient in this series who had been given atropine beforehand. Brief bradycardia and two ventricular arrests occurred. Note the artefacts during the 8-second cardiac arrest in the third trace due to pulmonary ventilation. The arrest followed by ventricular "escape", AV dissociation, ventricular "capture" and atrial arrest in the top two continuous traces are typical cholinergic effects of suxamethonium.

THE CARDIOVASCULAR EFFECTS OF NITROUS OXIDE IN THE DOG

Sir,—Two years ago (Brit. J. Anaesth. (1963), 35, 631) I reported that nitrous oxide, used with ether, or with halothane or methoxyflurane in low concentration (0.5 per cent), may produce appreciable depression of blood pressure and heart rate, reversible by withdrawing the nitrous oxide and replacing it with air or oxygen. Drs. Craythorne and Darby (Brit. J. Anaesth. (1965), 37, 560) administered nitrous oxide for 10 minutes at a time, to dogs, and failed to confirm these findings which, they say, are most difficult to interpret because of a large number of other variables such as premedication, surgical trauma, other anaesthetic agents, hypothermia, etc. I should like to make the following points:

(1) Their dogs were premedicated with atropine (0.8–1.7 mg), intubated under suxamethonium and anaesthetized with halothane (concentration unstated) for the purpose of placing in position tracheal, epidural, arterial, venous and right atrial catheters, and for suture of a strain gauge to the right ventricle via a right thoracotomy. When surgery (duration unstated) was completed, halothane was discontinued, and when the animals had fully recovered the administration of nitrous oxide was started. After each 10-minute experiment, dogs were ventilated for at least 20 minutes with room air and, presumably, were conscious before the commencement of each experiment. By contrast, none of my patients received surgery or had been operated upon more recently than 1–2 weeks previously, and observations were made, under stable conditions, 1–4 hours following induction of anaesthesia. The effects of withdrawing either nitrous oxide or the volatile gases were observed separately, both at normal temperature and during body cooling. In one instance (fig. 5, my paper) halothane was withdrawn as long as 52 minutes before nitrous oxide was withdrawn, yet little change occurred in blood pressure or heart rate until nitrous oxide was withdrawn.

(2) Ventilation during nitrous oxide administration in the dogs was maintained at that volume which had kept end-expiratory carbon dioxide constant during the control period. This was done because significant changes in myocardial contractility (i.e., in one of the parameters under investigation) can be produced by small changes in arterial Pco₂. However, since nitrous
oxide may depress ventilation (Bloch, in preparation) and thus would be likely to alter arterial PCO₂, the animals were being protected from a source of change the experiments were designed to measure.

(3) In those instances in which nitrous oxide was administered after sympathetic block (approximately one-third of the total) changes in contractility secondary to hypotension were prevented by maintaining blood pressure at normal levels with a slow infusion of methoxamine. Any effects of nitrous oxide in depressing blood pressure and heart rate, in this group of animals, would thus have been opposed and possibly obscured by the action of methoxamine.

(4) Whereas arterial blood takes up a high percentage of its nitrous oxide in 10 minutes, the same cannot be said of tissue nitrous oxide uptake, which requires rather longer. This must detract from the value of observations based on 10-minute nitrous oxide inhalation. The cardiovascular effects described are those produced by exposing initially conscious dogs, postoperatively, to nitrous oxide for 10 minutes at constant ventilation.

Maurice Bloch
London

A NEW LEASE OF LIFE?

Sir,—I am somewhat perturbed at the sentence in the editorial of your September issue which states: "At least one has proved relatively easy to give and relatively safe and so potent that the anaesthetist and surgeon no longer need to compete for the patient's airway."

I gather that this refers to the use of halothane as an adjuvant in general anaesthesia and probably particularly to outpatient and short cases, where endotracheal anaesthesia is not employed.

I cannot imagine that there is any condition in which a patient is rendered unconscious in which the airway can be neglected, as this sentence seems to suggest, and it would be indeed unfortunate if students (and this number of the Journal is addressed to students and postgraduates) were to think that the responsibility of the anaesthetist with regard to the airway had in any way been altered by the use of newer agents.

I do regret that I have to raise this point in what is essentially a valuable contribution to the postgraduate's library.

Victor Goldman
London

[We are grateful to Dr. Goldman for drawing attention to a sentence in the Editorial which could be misinterpreted. We would emphasize that there is not the slightest intention of suggesting that vigilance can now be relaxed by the anaesthetist. It was intended to point out that the uncertainties attending dental anaesthesia, and indeed during operative procedures at all levels in the airway, are greatly reduced by the use of an agent which can permit satisfactory anaesthesia despite a degree of leak.—Eds.]