Sir,—The work of Professor James and others in South Africa on nitrous oxide at high altitudes is of course well known; any review on awareness would certainly include it. Excluding this work (and some other important topics) from my article was solely a result of lack of space; an Editorial cannot be a complete review. Our objective is usually to select areas which are controversial, poorly understood or in need of further research.

However, one idea which follows from Professor James’s work is the possibility of pressurizing the operating theatre, similar to a jet aircraft. Even at sea level this could be useful. It would solve simultaneously the three problems of Caesarean section: fetal hypoxia, maternal awareness and unwanted effects of volatile agents. In recent years, progressively larger amounts of volatile agents have been used in the hope of achieving 100% success with the awareness problem; this may prove to be the solution but, as Moir [1] showed decades ago, some patients are put at risk from uterine bleeding and hypotension. Enhanced pressure could be useful in other situations where volatile agents are ruled out: critically low cardiac output and malignant hyperpyrexia. Implausible as it is in the current economic climate, a modest increase in pressure could conceivably be the simple answer in the future. The first anaesthetic may, in the end, be the best.

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Harrow


Plasma cholinesterase activity in elderly and young adults

Sir,—Although the physiological function of plasma cholinesterase is not known and patients with abnormal activity of this enzyme may be symptom-free and unaware of it, it is involved in the metabolism of several drugs used in anaesthesia, such as suxamethonium, mivacurium, procaine, chloroprocaine, tetracaine, cocaine, diamorphine, etc. [1].

Age-related changes in plasma cholinesterase activity are well described in neonates, infants and children [2]. At birth, plasma cholinesterase activity is low (approximately 50% of the adult value) and remains so until about 6 months of age. The activity increases so that between 3 and 6 yr of age, it is 30% greater than adult levels. Thereafter, it decreases and reaches the adult level at puberty. However, there is a paucity of information on enzyme activity in the elderly. We therefore estimated the enzyme activity in a group of elderly patients compared with the activity in a group of young adults. This was considered important in view of the increasing number of elderly in the population and the recent availability of the new neuromuscular blocker, mivacurium, which may be indicated in the elderly.

After approval from our Ethics Committee, 5 ml of venous blood was obtained for measurement of plasma cholinesterase from each subject (mean age 77.7 (range 70-93) yr) and 20 young adults (aged 29.6 (18-40) yr) patients admitted to hospital for elective surgery. None was receiving any medication. Plasma cholinesterase activity was measured colorimetrically using benzoylcholine as substrate. The results are shown in the table. The mean plasma cholinesterase activities in the elderly and young patients were 7.23 and 10.04, respectively (P < 0.05), but in the elderly (7.28 or 7.16 u. ml−1, respectively; P > 0.05).

This study shows that plasma cholinesterase activity was about 26% lower in the elderly, although still within the normal range. It is not known if this reduction is enough to prolong the duration of action of drugs such as suxamethonium and mivacurium. The duration of action of mivacurium has been reported to be increased by approximately 30% in the elderly [3]. However, it is probably not related to plasma cholinesterase activity as it has been suggested that the enzyme activity needs to be reduced by more than 70% to result in prolongation of the effect of suxamethonium [4]. Studies of other drugs which are metabolized by plasma cholinesterase may therefore be indicated in the elderly.

Table 1. Patient characteristics and plasma cholinesterase (PChE) activity in young adults and the elderly (mean (sd or range)).

<table>
<thead>
<tr>
<th></th>
<th>Young adults</th>
<th>Elderly</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>29.6 (18-40)</td>
<td>77.7 (70-93)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>166 (6.5)</td>
<td>166 (6.0)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>72 (9.0)</td>
<td>69 (8.5)</td>
</tr>
<tr>
<td>Sex (M:F)</td>
<td>11:9</td>
<td>11:9</td>
</tr>
<tr>
<td>PChE activity</td>
<td>9.11 (2.22)</td>
<td>7.23 (1.86)</td>
</tr>
<tr>
<td>(μ. ml−1)</td>
<td></td>
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Alfentanil and propofol for difficult tracheal intubation

Sir,—We were interested to read the letter by McDonald [1] describing the successful use of propofol and alfentanil in the management of difficult tracheal intubation. Despite the author’s enthusiasm, we have reservations on the use of this technique in the management of the difficult airway.

The role of neuromuscular blocking agents in the management of the patient in whom conventional laryngoscopy is impossible remains the subject of controversy. There is no doubt that the administration of agents to produce neuromuscular block may, in some patients, allow an otherwise impossible intubation to be achieved. However, in those patients whose lungs cannot be ventilated adequately by means of a face mask, the loss of spontaneous respiration may be lethal.

The advantages of an agent such as alfentanil, the effects of which may be antagonized in difficult cases, are obvious. It must be noted however that even under ideal circumstances, attempted intubation using propofol and alfentanil alone is associated with a failure rate of 10-17% [2, 3]. Increasing doses of propofol may reduce the failure rate at the expense of prolonged apnoea, unresponsive to opioid antagonists, should intubation prove impossible.

We would advise extreme caution in the use of this technique in any patient in whom it is impossible to ventilate by face mask under deep volatile anaesthesia. It is our opinion that in such patients the airway should be secured by means of elective cricothyrotomy before administration of neuromuscular blockers or respiratory depressants.

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L. A. WOODS
Northern General Hospital, Sheffield


Sir,—We wish to comment on the letter from McDonald [1] in which the management of a difficult airway is described using alfentanil and propofol. We are unclear why induction preceded fibreoptic intubation. It has been shown that awake fibreoptic intubation maintains the natural airway and there is easier identification of upper airway structures than when the patient is asleep [2]. Although propofol-alfentanil is useful for intubation without neuromuscular block in uncomplicated patients, in our experience the dose of alfentanil used produces a prolonged period of apnoea. As it was difficult to ventilate the lungs of this patient, the use of a technique likely to cause apnoea appears contraindicated. The argument that alfentanil can be antagonized safely with naloxone is unfounded as there are numerous reports of complications after opioid antagonism by naloxone [3]. We feel that in this case the technique of choice is awake fibreoptic intubation as, in the words of Benumof, "no bridges are burned" [4]. If this was unsuccessful we would then proceed to a retrograde intubation technique with the patient awake at all times.

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N. PACE 
J. J. HENDERSON
Western Infirmary 
Glasgow


Prediction of arterial from end-tidal $P_{CO_2}$

Sir,—The correspondence [1] between Dr Farmery and Dr Fletcher on prediction of arterial $P_{CO_2}$ from end-tidal $P_{CO_2}$ is interesting, both theoretically and practically. The observations of Dr Farmery in a young healthy subject of the difference between end-tidal and mean alveolar $P_{CO_2}$ in relation to tidal volume, are of interest, but I wish to sound a note of warning concerning the apparatus used.

The Hewlett-Packard capnometer (HP47210A) analyses carbon dioxide in the mainstream gas, by means of a window in an airway adapter inserted in the inspired gas pathway. In contrast with sidestream sampling devices, when gas has to pass from the sample port by way of a catheter to the analyser, one might assume that with the Hewlett-Packard instrument there is no delay in the analysis of this gas, and hence the signal output represents the gas composition in the adapter. Unfortunately, the Hewlett-Packard device does not provide a real-time measure of carbon dioxide in the inspired gas pathway. There is a degree of delay, presumably because the signal is manipulated digitally within the measuring device. I have attempted to measure the delay time and the response time of the capnometer and found that there do not seem to be unique values for these variables. In particular, there appears to be a considerable degree of smoothing of the analogue output signal and the $1:8$ ratio is altered.

The plot shown by Dr Farmery depends upon end-tidal carbon dioxide measurements and these may well be correct. However, he goes on to make measurements of $dP/dV$, which is the rate of change of $P_{CO_2}$ with exhaled volume, within a single breath. To do this accurately requires the exhaled volume at a particular instant to be related to the $P_{CO_2}$ in the airway at the same instant. Unless he has estimated the degree of delay introduced in the signal by this particular capnometer, such measurements may not be correct. This is because the rate of exhalation is not constant and the constant delay within the measurement device results in a varying volume discrepancy because of the varying expiratory flow. This may explain his observations that the pattern of expiration within the individual accounted for changes in $dP/dV$.

I do not wish to cast excessive doubt upon Dr Farmery’s conclusions, which appear at least in a young healthy subject to be consistent and dependent mainly on end-tidal estimates which will be unaffected by instrument delay. His hypothesis that the influence of tidal volume may be through variations induced in alveolar gas composition can be simply tested experimentally, for example with end-inspiratory pauses of different duration. The main purpose of my letter is to emphasise that the Hewlett-Packard capnometer, although apparently a "real-time" device, is far from being so in practice and within-breath measurements have to take instrument delay time into consideration.

G. B. DRUMMOND
Royal Infirmary, Edinburgh

Sir,—Dr Drummond is correct in pointing out that the Hewlett-Packard capnometer (HP47210A), although often regarded as a "real-time" apparatus, is not so in practice. The mainstream sampling device comprises a "black body" infrared radiation source and detector, between which are interposed an optical band filter and a rotating chopper wheel. This latter component houses three chambers which contain a carbon dioxide reference cell, a nitrogen reference cell and a cell vented to the atmosphere. The wheel rotates at 2400 rpm and data from the analogue front end are sampled via an analogue-to-digital converter (ADC) at 160 Hz. The real sampling resolution $\Delta f$, however, much less than this because the ADC samples four times per revolution and, as only one data bit can be obtained per revolution, the real sampling resolution (before smoothing) is approximately 40 Hz. This adds a time delay of up to 25 ms to that created by the smoothing and averaging process ($\sim 125$ ms), hence the delay time of $150 \pm 25$ ms quoted in the manufacturer’s specifications.

As my data acquisition and analysis were obtained by computer (with an ADC sampling also at 40 Hz), correction for this time lag was facilitated by simply delaying the flow signal by six samples. Neither signal is real time, but both are contemporaneous.

In order to verify the time lag for a given capnometer one needs to be able to measure the time difference between the presentation of $P_{CO_2}$ and the delay.