Awareness during Anaesthesia

SIR,—May I refer to the paper by Drs. J. Wilson and D. J. Turner (1 February, p. 280), in which the subject of awareness during anaesthesia was discussed. Out of a total of 1,328 cases that I have recently anaesthetized, five cases have volunteered details of their operative experience indicating awareness. Three of these cases received nitrous-oxide–oxygen and relaxant anaesthesia—two out of a total of 25 caesarean sections and one case of multiple injuries.

One of the cases of caesarean section was a shocked patient with placenta praevia who was being vigorously transfused. No premedication was given and she received oxygen in high concentrations. Awareness was indicated by the patient's ability to describe both auditory and tactile experience. The second case of caesarean section again received no premedication. The operation was prolonged to two hours and twenty minutes. Awareness was confirmed by the patient's ability to recall in detail a conversation occurring during the latter part of the operation. The case of multiple injuries received premedication with morphine 15 mg. four hours and forty minutes before operation. Awareness was revealed by detailed recall of experience of an underwater sealed drainage tube being inserted.

The further two cases able to recall experience received different types of anaesthesia. The first had a subglottic tumour removed. Premedication 45 minutes before operation consisted of pethidine 100 mg., perphenazine 5 mg., and atropine 0·6 mg. Anaesthesia was maintained with intermittent ventilation with nitrous oxide, oxygen, and halothane combined with intermittent thiopentone to a total of 650 mg. Awareness included recall of the surgeon's conversation and the sensation of having the laryngoscope manipulated. The second case required his throat packed prior to oral surgery. Premedication was with papaveretum 20 mg. and hyoscine 0·4 mg. given two hours and five minutes before surgery. Anaesthesia was induced with thiopentone 400 mg., and suxamethonium 50 mg. was given prior to nasal intubation. Consciousness was recovered unduly quickly and he had detailed recall of his throat being packed—an unpleasant and painful experience.

The incidence of volunteered awareness in the total series of 1,328 cases was 0·38%. In the 25 cases of caesarean section it was 8%. In the group of 133 cases receiving nitrous-oxide–oxygen and relaxant anaesthesia it was 2·3%.

Only the patient with his throat packed experienced pain. This was not otherwise a feature and accords with the experience of Parkhouse.[1] Several of the cases appear to have had adequate premedication, which does not agree with the conclusions of Wilson and Turner. It was felt that the awareness experienced in the case of placenta praevia was probably due to the high concentration of oxygen delivered—again in contrast to the conclusions of Wilson and Turner.—I am, etc.,

N. S. FAITHFULL.

British Military Hospital,
Rinteln, West Germany.

REFERENCE

1 Parkhouse, J., *Postgraduate Medical Journal*, 1960, **36**, 674.

SIR,—Some time ago one of our anaesthetic registrars told me of a personal experience he had when undergoing tonsillectomy in hospital. Because of inadequate anaesthesia he was fully aware that the endotracheal tube was being inserted into his trachea, but could not make his predicament known because he was paralysed with succinylcholine. He said it was the most horrifying and nightmarish ordeal he had ever experienced.

From personal observation and conversations with anaesthetic colleagues it seems to be a fairly general practice in most hospitals to use minimal amounts of intravenous thiopentone during induction of anaesthesia prior to intubation with succinylcholine or other relaxant, the thiopentone generally being administered in the half-strength 2½% solution. I do not suggest that the massive doses of intravenous barbiturates sometimes used for "blind" nasal intubation before the advent of muscle relaxants are either necessary or desirable in modern anaesthesia, but is it not possible that the pendulum has swung too far in the opposite direction? The routine use of 2½% thiopentone may provide adequate induction of anaesthesia in the majority of hospital cases, but there are times when "a little bit extra" is much better for the patient when endotracheal intubation is to be carried out—a remark which also applies to the administration of succinylcholine. I must confess that when confronted with a robust patient in good general condition I usually give an average dosage of 300 to 400 mg. of thiopentone followed by 50 to 70 mg. of succinylcholine prior to intubation. This, I am given to understand, is generally considered to be verging on the extravagant if not actually reckless by modern standards.

A method which used to be taught during the early days of intravenous anaesthesia is to estimate (or guess, if you like) the total amount of thiopentone appropriate for each individual patient. The dosage might be 50 or 500 mg. according to age and general physical condition. Half the amount is injected fairly quickly, followed by a short pause to observe the effect on the patient; the remainder is then injected at a slower rate. This is a safe and reliable method of induction, while the preliminary assessment of the dosage required minimizes the risk of overdosage.—I am, etc.,

PHILIP AYRE.

Department of Anaesthesia,
Royal Victoria Infirmary,
Newcastle upon Tyne.

Indications for Tonsillectomy

SIR,—I would have thought that it was relatively easy to understand the mechanics of a central mass of adenoids blocking the Eustachian tubes (1 March, p. 574). A central pituitary, when enlarged, may affect the optic pathway on either side—so an enlarged mass of adenoid tissue may block the tubes. The problem is, of course, not so facile; and more commonly infected, and, as, Mr. E. H. M. Foxen says (15 February, p. 442), not necessarily large adenoids cause Eustachian malfunction due to lymphatic stasis and mucosal oedema.[1] The importance of adenoids is not so much that they give rise to acute otitis but that they are largely responsible for conductive deafness and secretory otitis media, often with no history of acute episodes of infection. In one very large series 3·5% of all schoolchildren were deaf to a level of 15 db. or more.[2]

I quite agree with Dr. T. M. Banham (1 March, p. 573), who quite rightly says that adenoids will regress, but five years of continuous or intermittent deafness must retard education, and not all these ears return to normal. If conservative treatment fails adenoids should be removed and, in my opinion, the tonsils as well if, and only if, they are causing trouble. There is very good evidence that conductive deafness and secretory otitis are very common in childhood,[2 3] that secretory otitis is due to Eustachian malfunction[4 5] and that this in turn is very often due to adenoids, and will often resolve after complete adenoidectomy.[6]

I hope that no one will be misled into ignoring or underestimating the adverse effects of even a minor degree of conductive deafness on the education of schoolchildren at a vital period in their development, and the importance of audiometric testing and energetic treatment.—I am, etc.,

CHRISTOPHER HOLBOROW.

London W.1.

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Episodic Giddiness and Hyperventilation

SIR,—I found your leading article on episodic giddiness (22 February, p. 457) most informative, but was surprised to find your omitting anxiety[1] as a possible aetiological factor. In my experience it is often a symptom of hyperventilation,[2] responding well to explanation, reassurance, and desensitization to the underlying anxiety-producing situations. Propranolol is a useful adjunct.[3 4]

Professor M. Roth (22 February, p. 489) describes many symptoms of hyperventilation including "dizziness." I recently saw a woman who complained of "dizziness" of five years' duration, and who had consulted 25 doctors, including a variety of competent specialists, none of whom had explained the mechanism of her symptom (hyperventilation) to her. Wahl[5] justifiably describes the hyperventilation syndrome as one of the "commonly neglected psychosomatic syndromes."—I am, etc.,

HERZL LOWENSTEIN.

Durban,
South Africa.

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