Sir,—Brock-Utne and Jaffe recommend the sitting position for fibreoptic intubation in the presence of a full stomach [1]. I would endorse this for most patients undergoing fibreoptic intubation, as there are several advantages in addition to those mentioned in their letter.

The sitting position is more natural for the patient and allows face-to-face communication with the anaesthetist. This makes for a more relaxed patient with less need for sedation and thus a truly "awake" intubation. Any blood or secretions drain from the pharynx or can be swallowed more easily, making for a clearer view. It is also a more physiological position for the patient with obesity or respiratory or cardiac disease. The only objection is that the view is "the wrong way round", but with a little practice and imagination this is not a problem.

Originally, I adopted the use of the sitting position for fibreoptic intubation because I copied the technique of physicians when performing fibreoptic bronchoscopy. On a recent visit to the Department of Anaesthesiology at the University of Michigan, I was encouraged to find that their "Difficult Airway Clinic" endorsed this practice [2]. Presumably, anaesthetists place their patients in the supine position from habit.

S. Dowson
Leeds

REFERENCES

APOLGY
Sir,—We wish to apologize for the error in the list of References in our article [1], which Dr Fassoulaki has called to our attention. The article written by Fassoulaki and co-workers (listed as reference No. 8 in [1]) was published in the January, 1983 issue of British Journal of Anaesthesia [2], not in the 1979 issue. We deeply regret any inconvenience this error may have caused the authors, editors and readers.

N. Saitoh
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REFERENCES

DOES PROPOFOL IN COMBINATION WITH ALFENTANIL PROVIDE STABLE ANAESTHESIA IN CARDIAC PATIENTS?

Sir,—We read with interest the article by Manara and colleagues [1] in which they reported eight patients undergoing CABG using a bolus dose of both alfentanil and propofol for induction followed by an infusion of each agent separately. The authors stated that induction was associated with significant reductions in SAP (−22%), MAP (−22%), DAP (−18%) and LSVSWI (−30%), with no significant haemodynamic response to tracheal intubation. These variables returned to baseline values gradually after skin incision, and there were no significant changes in PAP, CVP, PAWP, SVR and PVR. The patients all had ejection fractions greater than 0.5 and a left ventricular end-diastolic pressure less than 12 mm Hg, and the authors comment that their technique offered stable anaesthesia for CABG patients with good left ventricular function.

We disagree with this assumption, based on their own data and the data of Kaplan and colleagues [2], Lippmann, Paicius and Gingerich [3] and others [4, 5] who have shown, in both cardiac and non-cardiac patients, that propofol when used alone for induction is a myocardial depressant—an effect which is even more pronounced when propofol is used in conjunction with an opioid such as fentanyl [6]. The haemodynamic data in table I of the article demonstrate significant left ventricular depression from induction, lasting 5-8 min and probably longer. Together with a decrease in SAP, DAP and MAP, without a significant decrease in SVR, this demonstrates a pronounced myocardial depressant effect of the infusion combination, reflected by a decreased LSVSWI of up to 30%, not stable anaesthesia as the authors indicate. Return to baseline values occurred only after significant surgical stimulation.

Would these authors suggest the technique to be safe in CABG patients with poor left ventricular function and ejection fractions less than 0.5? We think not, and we would suggest that extreme caution be exercised in this subset of patients.

One additional point we wish to highlight is that the doses of propofol used in their study were extremely small. Without concomitant use of significant doses of alfentanil, most patients would not be fully anaesthetized. As shown by Vermeulen and colleagues [6], the use of anaesthetic doses of propofol in conjunction with an opioid produced, in a dose-related fashion, much more pronounced myocardial depression.

M. Lippmann
R. Ginsburg
Los Angeles

REFERENCES

Sir,—Professor Lippmann and Dr Ginsburg raise several points for discussion about the technique we described for use in patients with good left ventricular function undergoing coronary artery bypass grafting (CABG).

We fully appreciate that our data can be interpreted as showing myocardial depression, based on a reduction of LSVSWI without significant change in SVR. We also commented that these haemodynamic changes are similar to those reported previously in patients with coronary artery disease (CAD), by slightly smaller in magnitude, probably because of the smaller doses of propofol used in our study. The real issue, therefore, appears to be if myocardial depression is synonymous with unstable anaesthesia.

We would agree that propofol can depress myocardial contractility, although greater infusion rates and blood concentrations than those in our patients would be required to cause changes comparable to those produced by 1 MAC of the commonly used volatile agents [1-3]. More relevant is the question as to whether or not mild impairment of the ability of the ventricular muscle to develop force (from a given end-diastolic volume) is necessarily disadvantageous. Hamilton [4] wrote an eloquent defence of the
principle, and we agree with the view [5] that mild haemodynamic depression is desirable in these patients, provided that coronary perfusion pressure remains within limits which permit a reduction in myocardial work to influence myocardial oxygen balance favourably. Stephan and colleagues [6] have demonstrated that a 31% reduction in myocardial oxygen consumption accompanies a 26% reduction in myocardial blood flow during propofol anaesthesia for CABG. As arterial pressure was maintained at clinically acceptable values throughout the study, and did not overshoot resting awake values in any patient, we consider the technique to provide stable anaesthesia for CABG.

This stability may be enhanced, outside the constraints of a rigid research programme, by manual alteration of the rate of infusion of propofol as required. The only way of maintaining LVSWI in patients undergoing CABG is to avoid the use of i.v. and inhalation anaesthetic agents altogether and rely simply on large doses of opioids. However, this approach results in a high incidence of breakthrough hypertension [7], thus risking myocardial ischaemia [8]. Would this be stable anaesthesia simply because LVSWI is maintained? We think not. Furthermore, opioids alone cannot be relied upon to produce unconsciousness [9].

We would like to reassure Professor Lippmann and Dr Ginsburg that we exercise caution when anaesthetizing any patient, particularly those with poor LV function, irrespective of the anaesthetic technique used. Indeed, it has been shown that mortality following CABG is not influenced by the choice of anaesthetic [10, 11], and suggested that patients with good LV function do well regardless of anaesthetic agent, whereas those with poor LV function fare badly with any technique, no matter how carefully it is administered. However, we could not at present recommend the technique we described for use in patients with poor LV function until haemodynamic studies are completed in this subset of patients undergoing CABG. Initial experiences in this centre have been encouraging [unpublished observations].

We agree that the dose of propofol (3 mg kg$^{-1}$ h$^{-1}$) used in our study was small. This does not, however, imply that our patients were inadequately anaesthetized. The ED$_{50}$ and ED$_{95}$ of propofol to prevent movement in response to surgical incision are 2.94 mg kg$^{-1}$ h$^{-1}$ and 4.98 mg kg$^{-1}$ h$^{-1}$, respectively [12], when combined with the doses of alfentanil used in our study. In paralysed patients the ED$_{50}$ required to provide amnesia to prevent wakefulness is less than that required to prevent movement. The ability to control anaesthetic and analgesic components individually should result in a smaller incidence of awareness than techniques relying on high doses of opioids alone. Vermeyen and colleagues [13] showed reductions in SAP, DAP and LVSWI of 28%, 23% and 32%, respectively, which are dissimilar to the reductions that we observed. They altered the propofol infusion rate manually as required and did not perform a dose–effect study of propofol on haemodynamics. It is worth noting that these authors concluded that fentanyl–propofol anaesthesia was adequate for patients with good LV function undergoing CABG.

CORRESPONDENCE


POTASSIUM IN THE PERIOPERATIVE PERIOD

Sir,—I would like to correct an error made in my recent article “Potassium in the perioperative period”[1]. In the maintenance of anaesthesia section I wrote “Some substances, such as penicillins, are potassium salts”. What I should have written was “Some substances, such as some penicillins, contain potassium salts”. Most penicillins are, in fact, sodium salts.

I am grateful to Dr David Ryan of Newcastle upon Tyne for bringing this error to my notice.

I wish also to take this opportunity to state that Augmentin and Timpicin contain potassium clavulanate. In other parts of the world, I am informed, injectable phenoxymethyl penicillin and procapicillin are, in fact, potassium salts.

R. S. VAUGHAN
Cardiff

REFERENCE


REVERSAL OF EXTRADUALLY INDUCED HYPOTENSION

Sir,—I read with interest the article by Sharrock, Mimeo and Urban on haemodynamic effects of hypotensive extradural anaesthesia [1]. The authors demonstrated the importance and safety of extradurally induced hypotension during total hip arthroplasty in controlled hypertensive patients. Hypotension was induced by blocking the autonomic nervous system via extradural agents on outcome of coronary artery surgery? Anesthesiology 1989; 55: 1115–1120.

Although the conclusion of this study suggested that induced hypotension using extradural anaesthesia was a safe technique in controlled hypertensive patients, I see a potential danger in the method. During the long-lasting effects of bupivacaine neural block, extradurally induced hypotension cannot be reversed, but only controlled by catecholamine infusions. If the patient’s arterial pressure must be increased before the effect of bupivacaine terminates, increasing the dose of catecholamine would be the only efficacious treatment. Such a therapy, however, may increase the risk of untoward effects on the heart and vascular system in hypertensive patients.

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REFERENCES


