base of modern medicine has been obtained from research which would not now be permitted. Should not the harm to patients which would result from refusing to publish such information be balanced against the likelihood that unethical research would be discouraged if the researchers knew that it is unlikely that they could find a publisher.

There are some things we simply do not have the authority to do, even to achieve a result which is in itself good. We do not have the authority to encourage harm to research subjects, even though the object would be to alleviate the suffering of patients. The last sentence in the Declaration of Helsinki is ‘In research on man, the interest of science and society must never take precedence over considerations related to the well-being of the subject’. The policy of the British Journal of Anaesthesia and other reputable journals should be to publish only ethical research: the use of placebos where an effective treatment exists and is available jeopardizes the well-being of the subject and is not ethical. I agree with Dr Clarke that such studies should not be published.

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2 Wilkinson D, Abdool Karim SS, Coovadia HM. Short course antiretroviral regimens to reduce maternal transmission of HIV. BMJ 1999; 318: 479–80

Propofol and epilepsy

Editor,—I am surprised at the requirement for an editorial on propofol and epilepsy. There is no new evidence to suggest that propofol causes epileptiform activity, as defined by EEG analysis. The nearest evidence clearly states the sub-anaesthetic doses used to provoke such EEG changes, in the same manner as with thiopental. Thus far only the onset of slow wave activity has been recorded, with a few patients showing dystonic reactions to propofol.

Indeed, in our own series now numbering 52, of patients undergoing ‘awake’ resections of dominant hemisphere epileptic foci under target-controlled infusions of propofol, we have had only two patients who did not recover from infusions, to allow accurate functional mapping, in less than 11 min. We suspect that these two patients may have suffered a complex partial seizure during the craniotomy phase but have no evidence to support this. Indeed, their outcome was no different from the rest of the group.

To quote an anonymous reference from a Data Sheet Compendium to support the argument for the conclusion that propofol should probably be avoided in epileptics with a driving licence is a little over zealous. The addition of the word ‘probably’ emphasizes the uncertainty of the conclusion.

Until such time as clear scientific evidence points to the propagation of convulsive EEG changes associated with dystonic movements on administration of inductive doses of propofol to patients, at appropriate rates, then propofol can be administered safely to epileptic patients. After all, there were a number of case reports in peer-reviewed journals of propofol infusions being used to control refractory status epilepticus in 1987–8. This is fact, not fiction.

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Editor,—I am grateful to Dr Huggins for his interest in my editorial. Propofol has been associated repeatedly with a range of excitatory events and sedative doses produced unequivocal increases in epileptiform activity in a proportion of patients during recordings from electrodes within the surface of the human brain. Clinical experience has shown propofol to be an effective anticonvulsant in animals and human. All of these points were clearly made in the editorial.

The key point overlooked by Dr Huggins is that epileptic patients will lose their driving licences as a consequence of any excitatory episode, including myoclonus and opisthotonus, regardless of the state of the EEG at the time. Therefore, it remains appropriate that clinicians should be guided by pragmatism in addition to science and not use this agent in epileptics who hold a current driving licence or have a reasonable prospect of doing so. To insist that propofol ‘can be administered safely to epileptic patients’ until there is evidence that convulsive EEG changes with dystonic movements can be demonstrated after induction doses of propofol, is a council of scientific perfection. An epileptic motorist who lost his licence as a consequence of such dogmatism would probably not appreciate the subtleties of this argument!

I stand by my recommendation that ‘in other epileptics and in status epilepticus, its use can easily be justified.’

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5 Boey W K, Lai FO. Comparison of propofol and thiopentone as anaesthetic agents for electroconvulsive therapy. Anaesthesia 1990; 45: 623–8
7 Kuilma M, Roine RO. Propofol in prehospital treatment of convulsive status epilepticus. Epilepsia 1995; 36: 1241–3
8 Yanny HF, Christmas D. Propofol infusions for status epilepticus. Anaesthesia 1988; 43: 514

Pulmonary artery catheter-induced right ventricular perforation during coronary artery bypass surgery

Editor,—Myocardial perforation is a rare complication of pulmonary artery catheterization. We describe a case of perforation of the right ventricle caused by a pulmonary artery catheter during coronary artery bypass surgery. A 71-yr-old man with three-vessel coronary artery disease and unstable angina pectoris was considered for coronary artery bypass surgery. Coronary angiography revealed total occlusion of the left anterior descending artery and the right coronary artery, and partial occlusion of the left circumflex artery. Left ventriculography showed no segmental wall motion dysfunction or aneurysm. Ejection fraction was within normal limits (0.61). There were no electrical or laboratory signs of myocardial infarction.

After induction of anaesthesia and tracheal intubation, a pulmonary artery catheter (7.5 FR VIP, Baxter, Irvine, CA, USA) was inserted via a percutaneous sheath introducer (8.5 FR, Arrow, Reading, PA) placed in the right internal jugular vein. The catheter was advanced to the 20 cm mark. The balloon was then inflated with 1 ml of air. Despite multiple attempts, the tip of the catheter could not be passed from the right ventricle into the pulmonary artery. The balloon was then deflated and the tip of the catheter was left in the right ventricle. There were no arrhythmias or haemodynamic disturbances during the catheterization attempts. The chest was opened with a median sternotomy incision. After opening the pericardium, the tip of the pulmonary artery catheter was seen protruding through the inferior wall of the right ventricle. The catheter was withdrawn into the right atrium, and the perfusion was closed with 3/0 sutures on pledgets; the patient subsequently underwent coronary artery bypass surgery. Recovery was uneventful and the patient was discharged 10 days after surgery with no residual complications.

Factors that predispose to ventricular perforation during catheterization include small chamber size, a stiff catheter, outflow tract obstruction and myocardial infarction. There was no evidence of pre-existing right ventricular weakness in our patient and perforation was most likely caused by a stiff catheter. The perforation potential of pulmonary artery catheters is increased by cooling and by the presence of multiple lumens.

Right ventricular perforation is a hazard of using the pulmonary artery catheter if the uninfrolled tip remains in the ventricle. Haemopericardium can occur unless the pericardium is opened. A catheter left in the right ventricle may also cause arrhythmia after cardiac surgery. Withdrawing the catheter to the right atrium will prevent this complication.

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Remifentanil and rapid sequence induction

Editor,—I read with interest the short communications on remifentanil and rapid sequence induction. As stated by the authors, ‘rapid sequence induction is often associated with significant haemodynamic changes which are potentially harmful’. The indication for performing rapid sequence induction is to obtain a secure airway with a cuffed tracheal tube, as quickly as possible, when there is a risk of regurgitation and aspiration. This necessitates the use of an induction agent and rapidly acting neuromuscular blocker (usually succinylcholine) given in rapid succession.

In this study, thiopental was given and cricoid pressure applied as consciousness was being lost. The study drug was then given as a bolus of 15 ml over 30 s, followed by succinylcholine, which was allowed another 60 s to take effect before laryngoscopy and intubation. Administration of remifentanil between thiopental and succinylcholine compromises rapid sequence induction and may increase the risk of regurgitation and aspiration.