that deserves wider use, at least in the early part of training!

However, it must not be thought that "eternal vigilance" is the only monitor we need. Clinical investigation and experience show that the human senses can not detect hypoxia (Comroe and Bothelo, 1947) or the adequacy of ventilation (Semmes et al., 1985) with sufficient sensitivity or reliability. The use of monitors which can detect these factors, such as the capnograph and pulse oximeter, is thus not supplanting but extending our vigilance, supplementing the deficiencies in our clinical ability.

Oxygen analysers to monitor the inspired gases and ventilator alarms are widely advocated to improve the safety of anaesthesia (Franklin, 1979; Marks, 1983). However, they provide no physiological information and are thus unlikely to be scrutinized often enough to ensure accuracy and continued function. They are best regarded as solely monitors of anaesthetic machine function, since they give no guarantee of the patient’s well being (Weingarten, 1986).

We should also question the times during which we use monitors. Current practice is to establish monitoring only after the patient has been transferred from the induction room to the operating theatre and to discontinue it before the move to the recovery area. The logic of this practice is questionable, since many anaesthetic disasters occur during induction or recovery and during periods of change such as the movement from one area to another. Monitoring, then, needs to be available in these high risk areas and to be portable. We may also need to reconsider our cherished induction rooms and induce anaesthesia in high risk patients in the operating theatre.

Used uncritically, monitoring may be at worst a bane and at best a mixed blessing. Used appropriately, it can be a blessing for both the control of anaesthesia and its safety. Appropriate monitoring is the rational use of devices, throughout the period of risk, that supplement or enhance our clinical skills rather than encouraging them to wither. By the adoption of such principles, the priorities for the acquisition of monitors can be determined and a rational allocation made of the available finance. The question of the cost effectiveness of monitoring for patient safety will only be satisfactorily answered by observing the results of a consistent change in practice by a large group of anaesthetists working together. A code of practice for the United Kingdom, similar to the Harvard recommendations and those of the American Society of Anesthesiologists, and based upon a wide consensus is needed urgently.

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The identification of an antibody which interacts with halothane-altered rabbit hepatocytes is the product of some 10 years of research by scientists at the Liver Unit of King's College Hospital, London. Their work has been one of the more interesting developments in the field of anaesthetic toxicity. Initially, the antigen complex used was derived from a crude homogenate of hepatocytes from rabbits exposed acutely to anaesthetic concentrations of halothane (Vergani et al., 1978). Later methods were refined to allow testing with an antigen known to be on the hepatocyte membrane (Vergani et al., 1980). The ELISA (enzyme linked immunosorbent assay) now used provides a faster, more reliably quantitative, test which has enabled the King's College group to screen samples from patients outside their own liver service (which, in the nature of things, concentrates on patients with fulminant hepatis necrosis). Thus in their report Kenna, Neuberger and Williams (p. 1286 of this issue) have information on patients with a wide spectrum of severity of halothane hepatitis, whereas previous reporting has been confined to those with fulminant hepatic failure.

The recurrence of the "halothane" antibody is of interest for two reasons. First, there is the possibility that the antibody is, itself, a factor in the liver injury. While it is generally held that the halothane molecule alone cannot be antigenic, a halothane–liver cell membrane moiety as an antigen, coated with antibody, may attract T- lymphocytes ("Killer Cells") as an initiating process in liver necrosis. Dienstag (1980) has warned against this view on the ground that such a mechanism would result in a very early antibody response and early onset of liver injury. In some patients, however, antibodies did not appear until several weeks after exposure to halothane. This suggests that they are secondary to liver damage.

The other important matter is that the detection of antibody might offer a specific indicator of halothane as the cause of postoperative liver injury—a great improvement over diagnosing halothane-associated hepatitis by a process of excluding other causes of liver damage. In this respect, the latest report is disappointing. In at least one-quarter of the patients with presumed halothane hepatitis, the antibody could not be found. It might be that binding of antibody to liver tissue results in its absence in detectable quantities from serum. It is also possible, of course, that these patients have a reason other than halothane for their liver injury. The sensitivity of the test may be deficient, although that seems unlikely. Last, it is possible that there is more than one type of liver lesion associated with halothane; if this argument is sustainable it could give further credence to the hypothesis that the antibody may precipitate hepatic necrosis.

There is no clear association between the presence of antibody and any other features documented in the patients with liver injury. For example, the occurrence of antibody was similar in cases of fulminant and less severe liver injury. Age, sex, severity of operation, previous anaesthetic history, biochemical data and time to onset of jaundice were poor predictors of severity of hepatitis and of the likelihood of antibody. While these studies are applauded, it must be clear that the value of the antibody test in diagnosis is limited.

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