warning sign of precancer, its prognostic value would have to be established over a longer period that that covered by the studies hitherto reported, for there is evidence that squamous and undifferentiated cancers may originate 10 years or more before death.  

Even if the early warning value of a cough were established it could help to prevent but a small proportion of cancers. Only half the cancers in the study of Boucot and colleagues occurred in coughers and only half of these may still have been precancerous when the cough began. Experience in antismoking clinics, attended by men who are specially keen to stop smoking, shows an average success rate of about 30%. Combining these percentages suggests that less than 10% of lung cancer could at present be prevented by trying to get coughing smokers to stop smoking. But this would not be the only benefit. A smoker's cough is an almost invariable preliminary to disabling bronchitis. If smokers who cough could be persuaded to stop smoking, disability and mortality from chronic bronchitis would certainly be diminished.

The practical problem of persuading people to stop remains the chief obstacle. We need more research into tobacco habituation to discover how more people may be freed from it. Meanwhile doctors should urge all their patients who have a cough to stop smoking forthwith. Only too often they are told to "cut down," and this is practically never possible.

Safety of Halothane

In most British hospitals halothane is now much the commonest vapour added to the gaseous anaesthetic mixture. But for some years a doubt has attended its administration. Is there a causal connexion between the drug and the post-operative jaundice and fatal hepatic necrosis that have occasionally followed anaesthesia with it?

The sporadic nature of the cases, the non-specific histological appearances of the liver, the presence of other possible causes, and the absence of clear animal experimental evidence of the hepato-toxicity of halothane in the circumstances of anaesthesia have kept this problem open. An obvious method of settling the answer is a prospective study. Apart from its magnitude such an investigation would not be too difficult to organize. However, opinions already expressed about the toxicity of halothane could present anaesthetists with an ethical dilemma, and plans for one large-scale prospective study in the U.S.A. were abandoned on these grounds. Anxieties were considerably allayed by the detailed and comprehensive retrospective studies by W. W. Mushin and colleagues1 and E. M. Slater and colleagues2 in 1964. These reports concluded that halothane was no more toxic to the liver than other anaesthetics. A further and very large retrospective study, called the National Halothane Study, was mounted in the United States, with results being analysed from 34 hospitals over a period of four years, and a summary of the report has recently been published.3

The important facts exposed by this study are the hospital mortality within six weeks after operation and the incidence of fatal hepatic necrosis. The study covered 856,000 patients, of whom 82 developed liver necrosis, or about 1 per 10,000 patients. Histological sections and clinical records were examined and a diagnosis made without knowledge of the details of the anaesthetic administered. The total death rate within six weeks of operation was found to be under 2%, but the death rate after administration of halothane was lower than this figure, and was midway among the rates for the several general anaesthetics used. The rate of fatal hepatic necrosis after halothane was lower than that after cyclopropane. The study by Mushin and colleagues1 did not show any increased liver damage when halothane was administered to patients with disease of the biliary tract. The present American study reports the same finding. Hepatic necrosis occurred after about 1 in 5,000 cases of cholecystectomy. However, in only one out of the six of these cases did it follow halothane anaesthesia, in spite of the fact that halothane was administered to one-third of the patients having biliary-tract operations.

There were in the American survey a few cases of hepatic necrosis—9 out of the 82—which could not be explained in any way other than by the nature of the anaesthetic. Seven of these patients had received halothane, and five of them had had one or more previous operations within the preceding six weeks. It seems, therefore, that if there is a connexion between halothane and liver damage—and the matter is still open—it is rare, but it might have some connexion with repeated administration of the anaesthetic.

The results of the American study, therefore, support in broad outline those obtained by Mushin and colleagues. It does not establish any causal relationship between halothane and hepatic necrosis, though it does not completely exclude the possibility. It gives the reasonable advice that, until the matter is finally settled, unexplained fever and jaundice in a patient after administration of halothane should contraindicate the subsequent use of the anaesthetic for that patient. Perhaps the most valuable outcome of this impressively large survey is a recommendation which is not directly concerned with halothane—namely, that mortality after surgery and anaesthesia should be looked at again, particularly with regard to any differences between institutions as well as between the various anaesthetics.

Walking Through Angina

The pain of ischaemic heart disease is one of the most common and most varied of visceral pains. It is usually precipitated by muscular exercise, but some patients get pain only after taking a heavy meal or exposure to cold, and a few others have attacks brought on by smoking. Once the pain has begun during exercise most sufferers find that it persists as long as they continue, and either the pain itself or fear of the consequences of carrying on often causes them to halt.

A few patients find that their pain wears off if they continue to exercise. The earliest known reference to this is in a letter read by William Heberden1 at the College of Physicians in London on 17 November 1772. Heberden had just published his account of angina pectoris in the second volume of the Medical Transactions of the College and had received

3 Summary of the National Halothane Study, J. Amer. med. Ass., 1966, 197, 775.