Risks of Total Hip Replacement

SIR,—In your leading article (10 May, p. 296) on the risks of total hip replacement, you mention intraoperative circulatory collapse attributed to acrylic cement. Modig¹ says that the literature records "at least 30 deaths during the operative procedure and possibly there are many more unrecorded cases." The original reports which led to the Department of Health and Social Security setting up an inquiry in 1971 are now over four years old and during these four years the use of acrylic cement in hip therapy must have multiplied many times, but we no longer seem to hear of these complications in the scientific meetings of surgeons who are using this substance almost daily. This would appear to be the case even when used to cement femoral head prostheses in subcapital fractures of the neck of the femur, though special danger in this case was a feature of the literature to which the D.H.S.S. report² drew our attention.

I think I am right in saying that all experimental investigations have now exonerated methylmethacrylate monomer in the concentrations likely to be encountered in the blood stream during a surgical operation. The other suggestions, such as fat embolism, neurogenic impulses from raised intramedullary pressure, or microemboli of thrombotic products from the marrow, all seem unsatisfactory as explanations of sudden intraoperative collapse leading to death.

It is important from the historical point of view that we should try to put the record straight now that we have four years of hindsight. Are these complications still occurring in any numbers or even at all? Most especially, are they still occurring during total hip replacement? The possibility of collapse after suddenly removing a tourniquet when acrylic cement has been used in a knee is perhaps a rather different matter. I would very much welcome communications from those interested in this subject.—I am, etc.,

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¹ Modig, J., *Studies on the Etiology and Nature of the Pulmonary and Circulatory Reactions during Total Hip Replacement*. Acta Universitatis Upsalensis, Uppsala, Sweden, 1975.

² Department of Health and Social Security. *Report of Working Party on Acrylic Cement in Orthopaedic Surgery*. London, H.M.S.O., 1974.

Safer Cigarettes

SIR,—Your leading article (17 May, p. 354) will have given more comfort to the cigarette manufacturers than to the many now trying to escape from their subjection as direct or indirect victims. The "20 years of health education, treatment at clinics, and antismoking propaganda" to which you refer has been almost completely obscured by the false picture of smoking presented by those seeking only commercial gain. There is no effective treatment, and the education and propaganda has been conducted against at least 100 times the expenditure on promotion of the habit—and of course of sales. Indeed the real effort in health education has been made only recently. The medical profession is the only group in our population which is fully aware and almost wholly convinced, and smoking has been reduced

among us by about two-thirds. The B.M.A. and most other reputable medical bodies have eschewed smoking at major meetings.

Of course some people will still smoke because of their addiction, but that is no reason why they should smoke in public or anywhere save in the company of consenting adults. Every smoker in public is a potential recruiter of other smokers, even if he only smokes a pipe or cigar. He may not realize it, but he is offensive to non-smokers, who are half the community. Perhaps the smoker's most cruel effect is his threat to the resolution of the man or woman who is trying to quit because he or she knows the danger of the habit. The truly evil consequence is the recruitment of smokers among children, who copy adults in this foolishness. Substitute materials may reduce the risk to those who cannot stop, but they are hardly likely to merit the term safe and they still carry the threat of recruitment.

The Third World Conference on Smoking and Health, which does not open till 2 June, is hardly likely to welcome the reference you make to it. Dr. Hunter's address was given, I understand, to an occasion associated with the tobacco industry.—I am, etc.,

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** We regret the mistake in our leading article, which has already been corrected (24 May, p. 454).—Ed., *B.M.J.*

Beta-blockers and Fibrinous Peritonitis

SIR,—The recent report by Mr. W. O. Windsor and others (12 April, p. 68) implicates practolol as a cause of a fibrinous peritonitis leading to subacute bowel obstruction. We would like to draw attention to a similar case in a 72-year-old woman who had had practolol therapy for angina from October 1971 until June 1974, when propranolol was substituted. Her recent clinical presentation and operative findings were very similar to the case reported by Mr. Windsor and his colleagues. The symptoms were those of subacute intestinal obstruction and abdominal masses which varied from visit to visit. Eventually laparotomy was undertaken since she had had carcinoma of the breast in 1966 and we could not exclude intra-abdominal malignancy; this revealed the small bowel and transverse colon to be cocooned in a fibrous adhesive sheet but no evidence of carcinoma.

She had previously had eye symptoms similar to those described by Mr. P. Wright (8 June 1974, p. 560), but in addition she had bilateral parotid and submandibular enlargement similar to that of Sjögren's syndrome. Furthermore, she had developed transient pleural effusions for which no other cause was found and which subsided spontaneously. A disquieting feature is that the pleural effusions were first noticed some weeks after practolol therapy had been withdrawn and propranolol substituted; furthermore, the abdominal complications developed some six months after cessation of the practolol and while she was on propranolol. While it is likely that the pleural effusions and the dense fibrinous peritoneal adhesions were due to the practolol, progression of the polyserositis process appears

to have developed after withdrawal of the drug and its substitution by propranolol. A cautionary note must be sounded that lesions similar to those induced by practolol may be caused by propranolol, with or without prior administration of practolol.—We are, etc.,

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¹²⁵I-Fibrinogen Test in Diagnosis of Deep Vein Thrombosis

SIR,—The report by Mr. A. Kline and others on dextran in the prophylaxis of thromboembolism (19 April, p. 109) is a most important contribution. It is encouraging to see the protective effect of dextran, though incomplete, confirmed. It was valuable, too, to see evidence for what many workers have long suspected—namely, that it may be possible to protect against major embolism without necessarily abolishing every minor focus of peripheral thrombosis. However, when the authors discuss the results of the ¹²⁵I-fibrinogen test (I.F.T.) they are sowing unnecessary confusion. In view of the large number of clinical trials based on the I.F.T. it is important that its significance be spelt out.

The authors say their clinical diagnosis of deep vein thrombosis (D.V.T.) "was very accurate" and then show that out of a total of 52 diagnosed as D.V.T. by phlebography and/or the I.F.T. there were 36 or 69% clinical false negatives. The number of clinical false positives is not clear, but on the basis of the 20 phlebograms mentioned it was at least 8%.

In evaluating their I.F.T.s they rejected those which phlebography failed to confirm. This could be justified only if the radiologist had succeeded in outlining all the named veins or if some cause of a false positive I.F.T. was present, such as inflammation or trauma. In our experience the application of an anatomical score to each venogram underlines this point.¹ It is not disputed that the more extensive D.V.T.s would be detected by phlebography. One would like to know whether the difference between the treatment and control groups would have been significant without this interpretation.

In support of their claim that I.F.T.s are not relevant to serious thromboembolism they quote Mavor and Galloway.² These authors, while quite rightly emphasizing the importance of the iliofemoral segment as a source of major embolism, were not in a position to define the relationship between iliofemoral and peripheral thrombosis since they did neither prospective I.F.T.s nor routine ascending phlebograms. Secondly, they quote the study of Lahnberg *et al.*³ These authors found by lung scans in a general surgical population a remarkable incidence of pulmonary embolism of 44% in their controls and 16% in their heparin-treated group. Figures of this order really require confirmation, especially when only seven out of 33 had clinical signs or symptoms, and perfusion lung scans (without concurrent ventilation scans) are not easy to interpret. Mr. Kline and his colleagues have ignored a very large body of literature which suggests a contrary view.