not the press should expose and deal with blatant drug peddling and indiscriminate abortionist activities.

The present resistance to the annual fee is surely only a symptom of frustration at a body out of touch with the bedside. It should not be beyond the wit of the B.M.A. to persuade the G.M.C. to disclose its activities and expenses and in return support an appropriate annual fee. If both sides remain obdurate we will all lose. A large section of the profession will soon be debarred from practising and the Government will have to intervene. A temporary *Register* (inevitably to become permanent) will be compiled, doctors will continue to practise, and the G.M.C. will become defunct or suffer a ministerial takeover. Short-sighted doctors will chortle over their victory while the State assumes total control over the profession. Never will so much have been surrendered for a fiver.

It sickens me to see the lengths to which the profession is prepared to go against a section of itself. If this intransigent stance had been adopted on more important fronts we might not have had two successive Review Body awards reduced (the loss of which would more than have paid G.M.C. fees for life), a pension scheme still woefully wanting, and a Government contract for consultants and general practitioners that would make a solicitor gasp. The B.M.A. subscription is £21; that of the medical defence societies £25, and the G.M.C. retention fee has been pitched at £5. These are all realistic figures. What is absurd is the doctors' earning capacity in this country under the N.H.S. Sir Keith Joseph must be watching our present internal dispute with amusement and amazement but certainly with no anxiety. We could scarcely be affronted if he felt he could run our affairs more adroitly. We may overwhelm the G.M.C. only to find we were concentrating on the wrong target—I am, etc.,

J. M. LAURENT

Chorleywood, Herts

Abortion Deaths

SIR,—Dr. Josephine A. C. Weatherall (21 October, p. 176) suggests that Sir George Godber's comments on abortion deaths, in the preface to the Report of Confidential Enquiries into Maternal Deaths in England and Wales 1967-69,¹ were in part prompted by "knowledge of the complete figures." He himself said, however, "This inquiry is the only available, reliable source of information about this particular aspect of the working of the Abortion Act."

Like Professor H. C. McLaren (30 September, p. 826), I serve as a regional assessor in relation to the confidential inquiry and, like him, I find Sir George's use of the word "improvement" in the preface impossible to equate with the facts in the report. This is particularly unfortunate, as the preface is the part of such reports most widely quoted in the press and elsewhere.

The harsh statistic is that in 1967, the last year before the introduction of the Abortion Act in 1968, and in 1969, the first year after, the number of deaths due to induced abortion were precisely the same. In 1967 only 3% were associated with "legal" abortion, whereas in 1969 41% were

associated with "legal" abortions. This shift was achieved by performing over 1,000 abortions for every week of 1969. This might be regarded as "improvement" from the point of view of the load on the judiciary but certainly not from a medical angle. Had Sir George called for immediate repeal of the Abortion Act it would have been understandable: that he could produce a complacent statement is rather frightening.

We hear much these days about accountability in the health service of the future. The Confidential Enquiry on Maternal Mortality was a model of what can be achieved from critical analysis of clinical care with the co-operation of a team of clinicians, and Sir George in his own preface to the report says the inquiry "should be an incentive to those in other fields of medicine who are prepared to undertake the self-discipline of this kind of retrospective inquiry into the results of their work. . . . It is to be hoped that other comparable exercises will become part of the Health Service in future." Sir George's hopes will be realized only if his successors resist the temptation to cloak unfavourable statistics which may emerge. Otherwise clinicians will not be prepared to give their time and effort.—I am, etc.,

J. S. SCOTT

Department of Obstetrics and Gynaecology,
University of Leeds

¹ Report of Confidential Enquiries into Maternal Deaths in England and Wales 1967-69. Reports on Health and Social Subjects, No. 6. London, H.M.S.O., 1972.

SIR,—Mrs. Madeleine Simms is quite right (21 October, p. 176): in 1968 the Chief Medical Officer did indeed comment in his annual report¹ that for 1967 both abortion deaths at 34 and non-abortion maternal deaths at 140 looked "exceptionally low" by comparison with 53 and 169 in 1966 and probably also taking into account the 1968 figures, which were up again to 50 and 150 respectively. But he would hardly wish to stand by that comment now, since in 1969 non-abortion deaths were even lower at 120 and 114 in 1970, with abortion deaths given as 35 and 32 for those two years.

Maternal mortality has been falling very satisfactorily over the past 20 years and more, but until recently the non-abortion mortality was being reduced proportionately more than for deaths resulting from abortion, as the following figures will show:

Maternal Deaths (England and Wales)

Year	Abortion	Non-Abortion
1950 ..	103	517
1955 ..	66	382
1960 ..	62	248
1965 ..	52	169
1970 ..	32	114

From 1967 onwards, however, the reductions in recorded abortion deaths seem to have been catching up. But whatever the reason for this it can hardly be regarded as a consequence of the Abortion Act, which did not come into operation until 27 April 1968.—I am, etc.,

C. B. GOODHART

Gonville and Caius College,
Cambridge

¹ Department of Health and Social Security, *On the State of the Public Health, 1968, Annual Report of the Chief Medical Officer to the Department.* London, H.M.S.O.

Barbiturates in Pregnancy

SIR,—In my time as orthopaedic surgeon at Harrow and Watford I came across perhaps a dozen cases of typical carpal tunnel syndrome arising in the last three months of pregnancy and causing much loss of sleep from the usual mixture of pain, tingling, and numbness in one or both hands. Two of these young women were the wives of orthopaedic registrars, who had tried the usual conservative measures such as injections of hydrocortisone into the flexor tendon sheaths at the wrist and a plaster night splint. Some of the others were taking so much sedative that they felt drowsy for most of the day and incapable of caring for a baby properly. All of them were screened for early toxæmia; only one was so affected, and she was whisked away by an obstetrician harbouring the baseless notion that all incisions in the palm develop keloid.

For all the remainder I performed complete open division of the transverse ligament strictly in the midline (continuing the line of the tendon of palmaris longus) under local anaesthesia, using the safe dose of 10 ml plain lignocaine 1% and an arm-cuff to control haemorrhage. This is a simple outpatient procedure, which should not take more than five minutes from first prick of the needle and inflation of the cuff to the last of the half-dozen sutures required to close the short incision across the butt of the palm. One woman came back to have the other side done a fortnight later. All the patients were truly grateful for the rapid relief given.

This brings me to your leading article (14 October, p. 64) on disturbed neonatal behaviour in 15 infants born to mothers taking barbiturates throughout pregnancy or in its last trimester. Two of the four mothers who had taken the barbiturates as sedatives, not because of addiction or epilepsy, were "apprehensive and deeply fatigued." For me those words ring a bell.—I am, etc.,

KARL NISSEN

Sherborne, Dorset

Rh Isoimmunization in Ruptured Pregnancy

SIR,—The paper by Drs. J. Katz and R. G. Marcus on the risk of Rh isoimmunization in ruptured tubal pregnancy (16 September, p. 667) prompts me to comment.

The authors cite as the only evidence of fetomaternal haemorrhage the demonstration of acid-resistant cells in the maternal circulation. That these cells could be of maternal and not fetal origin is a possibility dismissed in the report with the contradictory statements "The incidence of hereditary persistence of the fetal haemoglobin gene in the South African Bantu has been estimated to be of the order of 1 in 1,000." and "There is therefore no evidence of an increased incidence of high levels of fetal haemoglobin in the local African population. If anything the incidence is possibly less than in other population groups. . . ." This particular circumstance apart, raised levels of Hb F are characteristic of sickle-cell anaemia and some of the thalassaemia syndromes. I have encountered grand multiparity in an African homozygous for Hb S, and contrary to the apparent belief of Drs. Katz and Marcus thalassaemia is recorded as occurring in

Africans, notably in West Africa.¹ It would have been of interest to have had an account of erythrocyte morphology, sickling tests, Hb electrophoresis, and Hb F determinations in the pregnant patients studied.

But to accept for the purpose of further criticism that the cells whose origin I hold in question were derived from the fetus. Though the time of appearance of Rhesus antigenicity in fetal tissues has, to my knowledge, not been accurately determined, it cannot be at an earlier stage in embryonic development than the start of haemopoiesis. Since angioblastic tissue differentiates in only the third week of life and blood cells become recognizable only sometime thereafter, it is hardly likely that even the total blood volume of a 6-10-week embryo after intraperitoneal dilution, as the authors propose, in what is often several litres of maternal blood could account for the scores given for acid-resistant cells in the intraperitoneal and maternal peripheral venous blood. Such an effectively infinitesimal volume of fetal blood cells coupled with probably incompletely expressed Rhesus antigenic determinants can scarcely be described as a potential stimulus to maternal isoimmunization.

Furthermore, it should be pointed out that though ectopic pregnancy is a commoner occurrence in some groups of Africans than in Europeans, probably owing to the high prevalence rate of gonorrhoea in the former,² only some 5 per cent of South African Bantu are Rh negative.³ Drs. Katz and Marcus unfortunately omit any comment on the Rhesus phenotypes of their patients. It was my recent experience in Kenya, where the prevalence in the indigenous population of Rh(D)-negative phenotypes is very similar to that in the South African Bantu, that haemolytic disease of the newborn was infrequently encountered and when it did occur it was more often due to ABO than to Rhesus incompatibility. A very low incidence of haemolytic disease of the newborn due to Rhesus incompatibility has been described in other populations in which the incidence of the Rh(D)-negative phenotypes approximates to 5 per cent.⁴ Indeed, not all Rh(D)-negative women who suffer fetomaternal haemorrhage from a Rh(D)-positive fetus will become immunized.

In the light of these comments it is surely small wonder that Drs. Katz and Marcus have discovered only one record of Rh isoimmunization supposedly resulting from tubal pregnancy.—I am, etc.,

RONALD D. BARR

Department of Medicine,
University of Aberdeen

- 1 Lehmann, H., 1972, personal communication.
- 2 Carty, H. J., Nzoiki, J., Verhagen, A. R. H. B., and McGlashan, H. E., *East African Medical Journal*, 1972, 49, 376.
- 3 Mourant, A. E., *The Distribution of the Human Blood Groups*. Oxford, Blackwell, 1954.
- 4 *Obstetrics and Gynaecology in the Tropics and Developing Countries*, ed. J. B. Lawson and D. B. Stewart, p. 295. London, Arnold, 1967.

Hereditary Nephritis

SIR,—I should like to expand on your leading article on hereditary nephritis (12 August, p. 367) since more recent information is available on several points it emphasized. With regard to the natural history of the syndrome, we have recently analysed the ages at death (or terminal renal failure) of males with Alport's syndrome and found a high degree of concordance within families with regard to this parameter.¹ Moreover, two populations of affected families could be distinguished—one in which males invariably develop terminal renal failure between 16 and 28 years of age, and the other in which the renal failure develops between

33.5 and 52.5 years. Statistical analysis suggests that these two groups differ by a single gene of large effect rather than by multifactorial phenomena. Both types of Alport's syndrome appear to be inherited in an autosomal dominant manner. Both our study¹ and that of Feingold and Bois² indicate that the prevailing notion of an excess of affected offspring of affected parents is the result of the bias of ascertainment of earlier studies. Segregation ratios for all but possibly male offspring of affected males are not statistically different from unity, as expected in simple autosomal dominant inheritance.¹⁻³

With regard to therapy and counselling, a sizeable number of individuals with Alport's syndrome (at least 83) have undergone renal transplantation.⁴ Short-term rates of patient survival and kidney function are certainly no worse than those in other groups of patients treated similarly for other causes of renal failure.⁴ In addition, it has been our experience that their usual lack of debilitation facilitates their rehabilitation. Certainly these data, in addition to the results of long-term follow-up studies of transplanted patients, must be considered when providing genetic counselling. The genetic counsellor must also remember that not all affected males develop renal failure at a young age, and that he can frequently use the family history to predict the approximate age at which transplantation will be necessary. Moreover, our ability to determine the fetal sex permits a possibly more rational alternative (for example, aborting all male fetuses) for an affected parent than having no children at all.—I am, etc.,

PETER V. TISHLER

Harvard Medical School,
Boston City Hospital,
Boston, Mass

- 1 Tishler, P. V., and Rosner, B., *Birth Defects Original Article Series* (the Fifth Conference on the Clinical Delineation of Birth Defects, 1972), in press.
- 2 Feingold, J., and Bois, E., *Humangenetik*, 1971, 12, 29.
- 3 Preus, M., and Fraser, F. C., *Clinical Genetics*, 1971, 2, 331.
- 4 Advisory Committee to the Renal Transplant Registry, *Journal of the American Medical Association*, 1972, 220, 253.

Cannabis and Recurrent Herpes Simplex

SIR,—A variety of stimuli are known to trigger a recurrence of latent herpes simplex infection. A rise in temperature, physical trauma, exposure to ultraviolet light, steroids, and anxiety with a presumed liberation of adrenalene are well-known provoking factors. Cannabis smoking has not, to my knowledge, previously been noted as a provoking agent causing recurrent herpes simplex.

A 30-year-old man was first seen in October 1971 complaining of recurrent herpes of the shaft of the penis. The attacks lasted for five to eight days. For the past two years they had occurred much more often. During that period he had been smoking cannabis, and within two to three days, never less, never more, of having a "reefer" he would get a recurrence of his genital herpes. The attacks were unrelated to intercourse. In the spring of 1971 almost continuous attacks had made him give up smoking cannabis for a while and the attacks became much less frequent. When he later started smoking cannabis again the attacks recurred. When seen in October at the beginning of a new recurrence Type 2 herpes simplex virus was isolated, confirming the diagnosis of genital herpes. The lesion healed quickly with

antiviral treatment and he had only one recurrence, in a different site and equally short-lived, until January 1972. During the interval he had not smoked cannabis. He thought the antiviral drug had solved his problem and started smoking cannabis again (2-3 cigarettes per day),—and within 48 hours of starting he had an attack in yet another site. Though the attacks were aborted by antiviral chemotherapy he had another outbreak in August 1972 after smoking a lot of cannabis. He had had only two recurrences in the interval, both very slight.

I have since seen three other cannabis smokers with genital herpes who gave a very similar history. I have asked colleagues who see a large number of students whether they had had a similar experience. Two had had patients who were heavy cannabis smokers in whom distressing recurrent genital herpes had been an outstanding feature which had puzzled them greatly. MacCallum and I have speculated¹ that a possible common mechanism for provoking a reactivation of herpes simplex virus may be vasodilation. I asked Professor W. D. M. Paton whether cannabis had properties which might provoke recurrent genital herpes. I am grateful for his comments which were: (1) Cannabis produces vasodilation, the best-known example being conjunctival injection, now known not to be due to irritant smoke but apparently, from animal work, to a reduction of sympathetic tone. (2) The psychically active principles are intensely lipophilic and could well be taken up by nervous tissue (where it is possible the dormant virus resides, in whatever form, between attacks). It is also known that tetrahydrocannabinol (the main principle) affects the fine nerve fibres (Auerbach's plexus) in the intestinal wall. (3) Crude cannabis in experiments investigating the question of chromosome damage has been found to reduce the mitotic index. It may interfere with local or general immunity.—I am, etc.,

B. E. JUEL-JENSEN

Radcliffe Infirmary,
Oxford

- 1 Juel-Jensen, B. E., and MacCallum, F. O., *Herpes Simplex Varicella and Zoster*, p. 61. London, William Heinemann, 1972.

M.I.F. Test in Halothane Jaundice

SIR,—A cause and effect relationship between halothane and postoperative liver dysfunction has not yet been established.¹ We do not wish carpishly to criticize the letter of Dr. W. Jones Williams and others (7 October, p. 47) detail by detail, but the evidence presented by Doniach² does not withstand careful scrutiny and the "use of lymphocyte transformation tests for detecting hypersensitivity," reported by Paronetto and Popper,³ has not been confirmed.⁴

We contend that normal subjects are unacceptable as controls for the investigation by Jones Williams and his colleagues. We suggest that appropriate controls would be, firstly, postoperative patients, since lymphocyte responsiveness is known to be abnormal during this period,^{5,6} and secondly, patients suffering from liver dysfunction who have not been given halothane. Alteration in cell-mediated immune reactivity has been shown in various liver diseases^{7,8} and these changes may reflect an autoimmune response to liver damage, the implication being that liver damage releases liver specific antigen to which the subject may not have acquired full immunological tolerance. In