feel that the onus was upon Dr. Rollin to check the veracity of his facts.—I am, etc.,

R. H. S. LANE

St. Mark's Hospital,
London E.C.1

** We showed Mr. Lane's letter to Dr. Rollin, whose reply is printed below.—ED., *B.M.J.*

SIR,—I have always believed, and I am confident that a not insubstantial proportion of the medical profession also believed, that Sir Arbuthnot Lane was the subject of George Bernard Shaw's caricature portrayed by Cutler Walpole in his play "The Doctor's Dilemma." In the light of Mr. R. H. S. Lane's letter disclosing the correspondence between Shaw and Mr. T. B. Layton, of which I was totally unaware, it is obvious that this belief is both untrue and unfair. If I have caused offence by inadvertently perpetuating what must now be regarded as a myth, then I am extremely sorry.—I am, etc.,

H. R. ROLLIN

Horton Hospital,
Epsom, Surrey

Human Tissue Act

SIR,—As Scrutator reports (19 April, p. 151), Mrs. Barbara Castle recently indicated, in a letter to an individual correspondent, that where the deceased has signed a donor card the person lawfully in possession of the body (normally the hospital authority) is not under any legal obligation to contact relatives before authorizing the removal of organs for transplantation. Scrutator complains—rather churlishly, it might be thought—that Mrs. Castle's letter "fails to make clear . . . that it is not what a Government Minister declares but what the law says that matters," and that there are areas of doubt in the law.

In view of Scrutator's comment it is worth noting that the interpretation favoured by Mrs. Castle finds support in extrajudicial statements of the two of the present law lords who have expressed themselves on the subject.[1 2] The reasoning behind this view was stated in the recently published British Transplantation Society discussion document (1 February, p. 251). Mrs. Castle's interpretation is also in accordance with the general consensus of legal writers.[3-5]—I am, etc.,

P. D. G. SKEGG

Faculty of Law,
University of Oxford

1 Kilbrandon, Lord, in *Ethics in Medical Progress*, p. 51. London, Churchill, 1966.
2 Davies, Lord Justice E., *Proceedings of the Royal Society of Medicine*, 1969, **62**, 633,
3 Dworkin, G., *Modern Law Review*, 1970, **33**, 353.
4 Lanham, D. L., *Medicine, Science and the Law*, 1971, **11**, 16.
5 Speller, S. R., *Law Relating to Hospitals and Kindred Institutions*, 5th edn., p. 320. London, Lewis, 1971.

Diagnosis of Toxoplasmosis

SIR,—Dr. H. J. A. Longmore illustrates (12 April, p. 94) the frequency with which clinical toxoplasmosis may be diagnosed in general practice when the appropriate serological tests are carried out. He also unintentionally illustrates one of the several pitfalls that may occur in making a diagnosis.

His second case, in a newborn infant, appears to have been diagnosed as one of congenital toxoplasmosis on the strength of a dye-test titre of 1/256. However, in 23 cases of proved congenital toxoplasmosis shortly to be published[1] the lowest titre we obtained was 1/1024 and the titres in most of the cases were distinctly higher than this (geometric mean for all cases, 1/10 790). These findings were similar to those of other workers. Supporting our doubt about the diagnosis is the negative dye test at 18 months. Though the dye-test titre may be expected to fall over a period of years, perhaps encouraged by the early use of specific chemotherapy, we have never encountered a case becoming serologically negative in this way, nor to our knowledge has it been reported in the literature.

Now that a test for specific toxoplasma IgM antibody is becoming increasingly available it would be rash to diagnose toxoplasmosis in the newborn without this test giving a satisfactory positive result. The test can also be useful in suspected acquired cases, particularly where the dye-test titre is rather low—1/256 or 1/512. In such cases a negative IgM test is evidence against the current illness being due to toxoplasmosis. Our still rather brief experience on this has been that titres of 1/256 in current illnesses are usually accompanied by a negative or very weak IgM test, indicating that the dye-test titre is probably due to a past infection rather than the one in question.—We are, etc.,

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K. A. KARIM

Public Health Laboratory,
Leeds

1 Karim, K. A., and Ludlam, G. B., *Journal of Clinical Pathology*. In press.

Toxaemia of Pregnancy and Plasma Prolactin

SIR,—Dr. C. W. G. Redman and his colleagues (8 February, p. 304) have clearly demonstrated that among hypertensive women in the third trimester of pregnancy those with rising plasma urate levels had elevated plasma prolactin.

We have examined prolactin levels in hypertensive women (blood pressure (> 130/90 mm Hg) between 32 and 40 weeks pregnant who did not have proteinuria or gross oedema. None of the patients were taking hypotensive drugs; nearly all were having small doses of barbiturates, diazepam, or nitrazepam. The findings were compared with those in normotensive women having antenatal rest and similar sedatives for other reasons. Blood was taken at 09.00 hours from resting patients. Serum prolactin was measured by the double antibody radioimmunoassay, with prolactin 72/4/9 (Friesen) as standard, prolactin VLS No. 1 (N.I.H.) for labelling, and rabbit antiserum 65-5 (Friesen).

There was considerable between-patient variation, serum prolactin ranging from 48 to 273 μg/l, but values for an individual patient were relatively consistent (S.D. $\pm$ 33 μg/l) and showed no trend between 32 and 40 weeks maturity. The mean prolactin levels were: normotensive, 174 $\pm$ 45 μg/l (five patients); hypertensive, 176 $\pm$ 16 μg/l (12 patients). The standard errors cited represent between-patient variation; the difference between the two groups was not significant.

It therefore appears that elevated prolactin levels are not associated with pre-eclampsia or essential hypertension in pregnancy per se, but, as Dr. Redman and his colleagues have shown, with the renal effects of these diseases—reduced urate clearance, proteinuria, and oedema. As they suggest, it seems unlikely that prolactin is a primary aetiological factor in toxaemia of pregnancy and probably that metabolism or excretion of prolactin is affected when kidney function is impaired in this condition.—We are, etc.,

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Trasylol for Pancreatitis

SIR,—I would like to support Dr. M. L. Lewis's misgivings (22 March, p. 680) concerning the use of aprotinin (Trasylol) in pancreatitis. Though in theory aprotinin is ideal for the early stage of pancreatitis, in which there is kinin generation and increased fibrinolysis, yet without a battery of tests it is impossible to know whether the patient has passed on to a stage of fibrinolytic inhibition. Inhibition of fibrinolysis as a result of pancreatitis was documented by Gabryelewicz and Niewiarowski in 1968,[1] but confirmatory studies in man are still required. In 1967 Beller[2] showed that aprotinin predisposes to fibrin deposition in the kidneys by its inhibitory effect on fibrinolysis. Clearly an increase of fibrinolysis, as in early pancreatitis, is essential to the prevention of thrombus formation.[3] In my studies of pancreatitis in rats, which are animals which do not easily develop thrombi, inhibition of fibrinolysis gave rise to "shock lung,"[4] just as Dr. Lewis has described.—I am, etc.,

E. NIGEL WARDLE

Newcastle upon Tyne

1 Gabryelewicz, A., and Niewiarowski, S., *Thrombosis et Diathesis Haemorrhagica*, 1968, **20**, 409.
2 Beller, F. K., Mitchell, P., and Gorstein, F., *Thrombosis et Diathesis Haemorrhagica*, 1967, **17**, 429.
3 Kwaan, H. C., Anderson, M. C., and Gramatica, L., *Surgery*, 1971, **69**, 663.
4 Wardle, E. N., *Journal of Surgical Research*, 1973, **15**, 122.

Myeloid Leukaemia and Cot Deaths

SIR,—The preleukaemic state envisaged by me as a cause of sudden death of apparently healthy babies—either stillbirths[1] or cot deaths[2]—is unlikely to be associated with classical signs of myeloid leukaemia (Drs. E. Tapp and B. W. Otridge, 19 April, p. 140).

On the other hand, in-utero replacement of normal reticuloendothelial system cells with cells which look normal but behave abnormally (mutant cells) could (*a*) be caused by neoplasms of the reticuloendothelial system, provided they combined embryonic origins with short latent periods (for example, myeloid embryomas) and (*b*) produce intolerance of the anoxic conditions of childbirth (due to difficulty in replacing ϵ chains of haemoglobin with γ, β, and δ chains) and intolerance of the post-

natal environment (due to difficulty in replacing passive with active immunity).

Sudden and unexplained deaths of infants are often preceded by easy deliveries or a short second stage of labour.[3] So it is possible that cot deaths and unexplained stillbirths have a common aetiology, which should be explored by a combination of biochemical and histological tests. Also Drs. Tapp and Otridge have discovered an excess of plasma cells in the marrow of infants whose sudden and unexplained deaths were preceded by minor respiratory infections. So it is possible that infants who have a congenitally defective immune system have more difficulty in producing an effective leucocytosis (following infections) than in producing extra plasma cells.—I am, etc.,

A. M. STEWART

Regional Cancer Registry,
Queen Elizabeth Medical Centre,
Birmingham

1 Stewart, A. M., *British Journal of Cancer*, 1973, 27, 465.
2 Stewart, A. M., *British Medical Journal*, 1974, 2, 611.
3 Protestos, C. D., *et al.*, *Archives of Disease in Childhood*, 1973, 48, 835.

Genitourinary Medicine

SIR,—I was sorry to see in your leading article (11 January, p. 51) that the Department of Health and Social Security and the Royal College of Physicians have been advised to recommend that the specialty of venereology should in future be called "genitourinary medicine." Though venereologists do deal with conditions of the lower urinary tract, as it is inseparably linked with the genitalia, they should not presume to look after its upper part with its very different functions which are more the province of urologists and nephrologists, who have rightly protested in your columns against this intrusion. The discipline, layout, and objects of our clinics do not lend themselves to taking on this branch of medicine as well as the venereal diseases. At one time the treatment of venereal diseases was in the hands of dermatologists, gynaecologists, urologists, and surgeons (and still is in many countries) to its detriment, as it had become a neglected and unimportant, though lucrative, sideline of these specialties. In Britain we have succeeded in making the management of these diseases a separate specialty of its own so that they could be dealt with by experts in that subject only. It seems to be a retrograde step to link them now with another quite different branch of medicine, which may well in due course take priority over the venereal or "sexually transmitted" diseases and lead to their neglect.

The lack of recruits with higher qualifications to this specialty is said to be due to its limited academic scope. Rather than try to expand this scope by taking in other fields of medicine, would it not be better to modify the academic qualifications required so as to attract those clinicians who are more interested in the art of trying to help with the personal difficulties of the patient rather than in academic medicine only? The actual cure of most of the venereal diseases has become very easy since the advent of antibiotics and chemotherapy, but the patients' fears and worries remain much the same. In helping with the latter the venereologists can be of more real value to the patient than if he branches out into another area of medicine already fully covered by others.—I am, etc.,

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Uterine Rupture in Labour

SIR,—Experience of rupture of the uterus in Wolverhampton confirms that of Mr. M. Brudenell and Mr. S. Chakravarti (19 April, p. 122). During the past 12 years 23 of our patients sustained uterine rupture, an incidence of 1 in 2048. Of these cases, only two occurred in late pregnancy, both with classical scars. Four lower-segment scars ruptured in labour and only one fetus survived. Traumatic rupture occurred 11 times, due to internal version in four cases and to difficult forceps delivery in the remainder. Spontaneous rupture occurred six times and in three of these cases there were obvious predisposing causes—that is, severe congenital abnormality, previous uterine perforation, and previous manual removals of adherent placentae.

In this small series, with a maternal mortality of one patient and a fetal mortality of only 70%, at least half of the cases occurred during the last four years. Amniotomy was performed on 15 patients, nine of whom received oxytocin in addition. The Cardiff infusion system, introduced in the last three years, was not involved. Epidural analgesia was given to four patients, of whom three were grand multiparae and the fourth was a gravida-5 in whom the analgesia abolished pain caused by the rupturing uterus. It is relevant, however, that only seven patients had severe pain, while serious haemorrhage or shock were obvious features in 13. We now regard grand multiparity as a contraindication to epidural analgesia, though in general we favour its use, especially in breech birth, where it allows the obstetrician to assist delivery painlessly, without resort to general anaesthesia. The incidence of breech extraction has not been increased. Lower-segment scars are a contraindication to epidural analgesia in labour, but if this method of pain relief is considered essential, then continuous monitoring of the uterine contractions as well as the fetal heart rate is of greater importance than intermittent palpation of the lower-segment scar.

We have also found that the diagnosis is difficult. In one case laparotomy on the third day to perform sterilization revealed dehiscence in the upper segment in a patient who had had her seventh baby after a labour of two hours. The only symptom that she had had was a little pain associated with a little uterine tenderness and abdominal distension. The rent was firmly plugged by omentum and the cause was obviously uterine perforation, documented as occurring 12 months previously.

In our series hysterectomy for rupture of the uterus entailed an average of 2·7 l of blood transfused, while repair required only 1·8 l. One ureter divided during hysterectomy was successfully reimplanted and the overall morbidity of hysterectomy was greater than that of uterine repair.

I would disagree that rupture of the uterus always means loss of the mother's capacity to have further children. Ten of our patients had the ruptured uterus repaired. Six were not sterilized and four of these have had six living babies delivered by caesarean section. Our experience, however, leads me to agree with Mr. Brudenell and Mr. Chakravarti that amniotomy, oxytocin infusion, and epidural analgesia, so helpful to many patients, can become hazardous procedures in those with a weak or damaged uterus or who are grand multigravidae.—I am, etc.,

ALAN M. SMITH

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Wolverhampton

B.M.A.: Need for Radical Change

SIR,—In some intangible way the B.M.A. is felt to be failing its members. The membership is reported to be gradually falling away, especially on the hospital side, and to be perilously near the significant 50% figure. This must be largely due to its often praiseworthy but ineffectual attempts to cover all fields: to be trade union, employment agency, expert spokesman on all medical matters, educator, charity, and even, at local level, organizer of social functions. That it fails in many of the more important of these activities is inevitable, given this brief.

The time has come for radical change, and what more appropriate time than now, when our long-serving Secretary, Dr. Derek Stevenson, is within a year of retirement? There have been disquieting rumours that the succession has already been decided within the upper echelons.

There can be little doubt that what members need most is a good trade union in these times of great social change in the country. Ergo, we need a Secretary who will see that these trade union activities take precedence. The name of the leader of a rival trade union has become almost a conversational cliché in this respect: "We need a Clive Jenkins!" is the cry. We do not, of course, but do we actually need a doctor? The idea of two wings in the organization—a tough negotiating cadre and a bland advisory medical one, has been canvassed before. Why not have a tough negotiating Secretary anyway?

We must think very hard on this, and we must certainly advertise openly and widely for a new Secretary of the very highest calibre and not allow this to be settled in the corridors of power. The choice will be critical for the survival of the B.M.A. It cannot survive for much longer in its present form.—I am, etc.,

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Poole General Hospital,
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Scottish Assembly and the Health Service

SIR,—The Glasgow pathologists' fear (3 May, p. 280) that the 40-hour week will endanger our Scottish traditions of clinical laboratory and academic training is correct. The desire for a return to a salaried service is widespread in Scotland. The Scottish consultants' views on the consultant contract are also quite distinctive.