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

R. H. S. LANE

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** 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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Public Health Laboratory,
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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.,

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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-