portion of my fellow medical students seem unaware that to be a student of medicine is a very great privilege.—I am, etc.,

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SIR,—Can Mr. A. R. W. Forrest (1 May, p. 281) give any justification for the implication that medical students during their training actually earn anything at all—not to mention £500 + per annum? Would he like to join the student laboratory technician in countless days of routine similar estimations? Perhaps the thankless back-breaking grind of a student nurse is more to his taste.

Medical students may need more money—but it is not their unquestionable right.—I am, etc.,

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Vascular Lesions of Eclampsia

SIR.—Disseminated intravascular coagulation¹ is being increasingly and unnecessarily invoked as an important pathogenetic mechanism in a whole variety of diseases. This tendency is particularly to be deplored in such conditions as renal cortical necrosis, malignant hypertension, and eclampsia, because the vascular lesions in these are due to ischaemia produced by obliterative arterial and arteriolar spasm. The photomicrograph given by Dr. J. Bonnar and his colleagues (3 April, p. 12) does not merely show fibrin thrombi in the vessels but presents the classical vascular lesion of "ischaemia with failed reflow" as seen in human and experimental renal cortical necrosis.

The vascular lesions of malignant essential hypertension have been shown² to be identical in nature with those of "ischaemia with good reflow" and "ischaemia with failed reflow," with the obvious inference that such vessels have undergone sustained functional obliteration with subsequent reflow—a process which could only be explained on the basis of spasm. If vascular obliteration lasts less than an hour no structural lesions result, though the patient has the renal and cerebral symptoms of malignant hypertension. With obliteration of one to three hours' duration there is medial necrosis with intramural fibrin deposition, but without mural or occlusive thrombosis. With obliteration of over three hours' duration medial and endothelial necrosis ensues, with intramural fibrin deposition and intravascular thrombosis.

These observations are equally applicable to eclampsia, in which pregnancy-induced hypertension reaches a malignant phase. In some cases of eclampsia the obliterative spasm may not be of a duration sufficient to produce any vascular lesions, so that at necropsy only minimal lesions may be found. In others, there will be the vascular lesions of ischaemia with good or failed reflow, varying in their severity and extent.

It must be obvious that the fits of eclampsia cannot be ascribed to any sudden intravascular clotting, but are due to cerebral ischaemia during prolonged obliterative spasm of the small arteries and arterioles. Fits will occur from such ischaemia, even when the obliterative spasm causing the ischaemia is not prolonged enough to render the vascular wall necrotic. When vascular

necrosis with luminal thrombosis does occur, this merely indicates the excessive prolongation of the operative spasm. Thrombosis does not originate the episode of ischaemia but postdates it, albeit its development will convert the original functional vascular obliteration into a permanent organic one.—I am, etc.,

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¹ McKay, D. G., *Disseminated Intravascular Coagulation*, New York, Harper & Row, 1965.

² Sanerkin, N. G., *Journal of Pathology*, in press.

³ Sheehan, H. L., and Davis, J. C., *Journal of Pathology and Bacteriology*, 1959, **78**, 105.

⁴ Sheehan, H. L., and Davis, J. C., *Journal of Pathology and Bacteriology*, 1959, **78**, 351.

Long-acting Phenothiazines in Schizophrenia

SIR.—We are interested by your leading article "Long-acting Phenothiazines in Schizophrenia" (23 January, p. 189), in which fluspirilene was mentioned.

One aspect of depot-neuroleptics is the duration of antipsychotic action; another aspect is their side effect liability. Villeneuve and co-workers¹ have found that extrapyramidal side effects occur less frequently with fluspirilene than with fluphenazine enanthate. Recently we have found that patients receiving other long-acting neuroleptics also suffer from more extrapyramidal side effects than patients receiving fluspirilene. We hope to publish more about these phenomena within a short time.

It should be suggested therefore that, at least as far as side effects are concerned, new depot-neuroleptics should be compared to fluspirilene.—We are, etc.,

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¹ Villeneuve, A., Dogan, K., Lachance, R., and Proulx, C., *Current Therapeutic Research*, 1970, **12**, 819.

Postpartum Coagulation Failure

SIR.—I have recently had to manage two cases of coagulation failure (presumably hypofibrinogenaemia) in pregnancy with a limited supply of blood, no fibrinogen, and no anti-fibrinolytic agents. One followed a macerated intrauterine death and the other a central placenta praevia in which the placenta had been perforated to deliver a recently dead baby vaginally. The diagnosis in each case was made on clinical grounds owing to lack of facilities and time. The blood issuing per vaginam did not clot.

The patients were treated on the assumption that two problems coexisted: coagulation failure and uterine hypotonia, and that the latter was the dominant problem (as, indeed, we were helpless to deal with the former).

Ergometrine was given, oxytocin drips (40 u/l.) set up and bimanual compression of the uterus instituted—to no avail; so the uterus, cervix, fornices, and vagina of each patient were tightly packed digitally. After this, continuous external digital stimulation of the uterus was maintained, for hours (in one case for six hours approximately), because despite the treatment mentioned the

uterus of each patient still tended to relax. This was done quite simply by sitting beside the patient's bed and continuously rubbing the uterine fundus with the finger tips. The patients were slightly under-transfused to maintain the blood pressure not above 90 mm Hg to obviate a high perfusion pressure and to encourage stasis in the uterine circulation. A slight and worrying trickle of blood (as the blood bank was almost depleted) persisted in both patients for some time, as did the tendency to uterine relaxation, but gradually both problems were overcome. The packs were removed after 24 hours and both patients did well.

These clinical observations support the idea of a dual problem in cases of coagulation failure, hypotonia possibly being the more significant clinically. Bleeding should not occur if the uterus contracts properly, and the hypotonia in these cases was particularly resistant to normal treatment. The external uterine stimulation was applied because it proved effective in counteracting the tendency of the uterus to relax, which was apparent for a considerable time after packing. This tendency gradually diminished and when the packs were removed both uteri contracted firmly and there was no bleeding whatever. The only alternative for these patients would have been a hazardous hysterectomy.—I am, etc.,

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Prolonged Fever in Bacterial Meningitis

SIR.—In your leading article (27 February, p. 474) you state "Sulphonamides, which are often and correctly used for the treatment of meningococcal meningitis . . ." Sulphonamides have been of great value in the treatment of meningococcal infections. Unfortunately owing to the emergence of sulphonamide-resistant strains these drugs should not be used alone for treating meningococcal disease but should be used in combination with penicillin.

The resistance of meningococci to sulphonamides was first recognized in the United States¹ but has since been seen in many parts of the world. In Scotland this laboratory has, since 1964, maintained a programme of surveillance of meningococcal strains isolated both here and in other laboratories, and we have examined these strains for their serogroup and sensitivity to sulphadiazine. Until recently no sulphadiazine-resistant strains were noted out of 157 examined. However, since late 1970 four strains resistant to 1 but sensitive to 5 mg sulphadiazine per 100 ml medium have been seen, one each from Inverness, Greenock, Fife, and Leeds. Preliminary studies suggest that all these strains are related to serogroup 135 which was first described in the United States² as an uncommon cause of disease.

Sulphonamide-resistant strains have previously been reported from studies in England³ and no case of suspected meningococcal disease occurring in the U.K. should therefore be treated by sulphonamides alone.

I am grateful to Dr. H. Williams, Inverness; Dr. J. Goudie, Greenock; Dr. J. Calder, Fife, and Dr. J. Stevenson, Leeds, for the strains of *N. meningitidis* and to Mr. W. Brown,