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

J. A. LOURIE

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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.,

SUSAN MITCHLEY

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Vascular Lesions of Eclampsia

SIR,—Disseminated intravescular coagulation[1] 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 shown[2] 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-workers[1] 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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A. TANGHE

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Holland

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 States[1] 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 States[2] as an uncommon cause of disease.

Sulphonamide-resistant strains have previously been reported from studies in England[3] 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,

F.I.M.L.T., and Miss Diana Cox, R.T., for technical assistance.

—I am, etc.,

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Department of Pathology,
Ruchill Hospital,
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1 Millar, J. W., Siess, E. E., Feldman, H. A., Silverman, C., and Frank, P., *Journal of the American Medical Association*, 1963, **186**, 139.
2 Evans, J. R., Artenstein M. S., and Hunter, D. H., *American Journal of Epidemiology*, 1968, **87**, 643.
3 Abbott, J. D., Adams, D., and Collins, T. J., *Journal of Medical Microbiology*, 1970, **3**, 233.

Angina Pectoris

SIR,—I read with interest "Angina Pectoris—I" (27 February, p. 501) and "Angina Pectoris—II" (6 March, p. 545) and I thoroughly enjoyed the discussion.

May I be allowed to make a comment regarding "Mechanism" of angina pectoris? The causes of angina, brilliantly listed in the diagram, embrace everything but aortic valvular disease, which may be listed under provoking factors (B) and sub-heading—reduced coronary blood flow (5). Aortic stenosis may result in angina pectoris because of interference with coronary blood flow in the small vessels during systole due to high intraventricular pressure, and in aortic regurgitation coronary perfusion may be impaired by low diastolic pressure. In both forms of aortic valvular disease increased size and work of the heart may contribute to coronary insufficiency by augmenting the need for blood.

May I also have the opinion of the readers regarding the treatment of angina pectoris with β-blockers. At present three β-blockers are available: propranolol, practolol, and oxprenolol. Dr. J. C. Petrie has rightly mentioned that practolol is less likely to cause bronchoconstriction, but practolol is less potent than the other two and it has been used more for arrhythmias than for angina. Oxprenolol, as the manufacturers claim, has got intrinsic sympathomimetic activity and is less likely to cause cardiac failure. In our Unit during the last three months we have used oxprenolol in ten patients with angina pectoris. The results of treatment are extremely satisfactory, but three patients developed intractible cardiac failure even on very low doses of oxprenolol. How common is this unwanted side effect in others' experience?—I am, etc.,

M. AHMED

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Methyldopa and Associated Thrombocytopenia

SIR,—In their paper on methyldopa and associated thrombocytopenia (27 February, p. 494) Dr. S. M. Manohitharajah and others comment on the lack of reports of platelet antibodies in patients on methyldopa with a positive direct antiglobulin test.

I should like to report such a case recently admitted to this hospital with haemolytic anaemia. Although she had as yet not developed thrombocytopenia complement fixation test for incomplete platelet antibodies was weakly positive. Cessation of methyldopa has not yet been long enough for us to recheck her platelet antibodies. It is perhaps of interest that she is also a case of treated pernicious anaemia.—I am, etc.,

JEAN M. WEBSTER

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Blood Pressure and Bone Cement

SIR,—Since the correspondence last year on the subject of bone cement (22 August, p. 465; 29 August, p. 523; 5 September, p. 528; 19 September, p. 710; and 17 October, p. 176) we have been carrying out arterial blood pressure recordings during total hip replacement operations in which prostheses fixed with acrylic cement (Simplex Opaque) have been used. An arterial needle leading to a pressure transducer and amplifier with continuous recorder (Devices M.2) has been employed. So far, records of 36 insertions of cement have been obtained, and a fall in blood pressure has been seen in 34. The fall has averaged 7% of the pre-cement blood pressure after the insertion of cement into the acetabulum, and 11% in the femur. No catastrophic falls have been seen. The cement mixes have been prepared by a mixing technique known to eliminate up to 15% of the monomer by evaporation, and decompression of the femoral medulla with a polyethylene catheter has been routine. Maximum blood pressure fall occurred on average 165 seconds after the commencement of insertion of the cement into the femur. Other manoeuvres—for example, the use of rotating cutting tools in the femur and the acetabulum, or the reduction of the hip—often produced blood pressure changes as great as those following the insertion of the cement. This work suggests that a blood pressure fall occurs in nearly every case, but will be noted only by using continuous recording techniques.

Absorption of monomer from the cement surface has usually been regarded as being responsible for the blood pressure falls which have been noted in the past. This concept derives primarily from the experimental work of Homsy *et al.*[1] who showed that, in dogs, a fall of blood pressure was a regular sequel to the intravenous injection of monomer. Monomer is very insoluble in water or saline and a biphasic suspension is produced when monomer and blood or saline are mixed. In repeating some of Homsy's experimental work we have confirmed a regular fall in mean arterial blood pressure after the intravenous injection of monomer, but this has in every case (11 injections) been associated with a rise of central venous pressure which has started at least as soon as the blood pressure falls, and considerably outlasted it. Homsy commented on the appearance of "haemorrhagic lesions" in the lungs of his experimental animals and the lack of lesions in the other organs.

It is thus quite possible that the haemodynamic effect of monomer given by intravenous injection is due to pulmonary monomer embolism, and not to any specific pharmacological action of the monomer. It is by no means certain that the monomer in the free state is absorbed from the surface of the cement mix after implantation. Surprisingly, no attention has so far been given to the activation reaction as a possible source of compounds of monomer with local components of blood, fat or bone. Activated monomer (which is not present following the direct intravenous injection of monomer) is a highly unstable molecule, readily combining with a variety of substances (including, of course, polymer). It is possibly highly important with respect to the local toxic effects of the cement mix, and the experiments of Hulliger[2] suggest this may be so. Absorption from the cement surface after implantation is probably much more complicated than has been thought in the past, and at the present time it is not possible to say categorically that blood pressure changes following implantation of cement are *definitely* a consequence of toxic absorption from the cement surface.

We would suggest that more orthopaedic units using acrylic cement should monitor their cases by continuous arterial blood pressure recording, with, if possible, central venous pressure recording and E.C.G. This should be combined with a record of the method of the preparation of cement, the exact time of the commencement of the insertion of the cement, the duration of the insertion process, the time of the insertion of the prosthetic components, and the weight of cement inserted. This type of record is especially important in interpreting the exceedingly rare case in which cardiovascular collapse may take place: the absence of such data in this situation may lead to the misinterpretation of the cause of the collapse.—We are, etc.,

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M. L. JAMES

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Exeter

1 Homsy, C. A., Paper presented at Joint Workshop on Total Hip Replacement and Skeletal Attachment of American Academy of Orthopaedic Surgeons, 6 November 1969.
2 Hulliger, L., *Archiv für orthopädische und Unfall-Chirurgie*, 1962, **54**, 581.

Dyspareunia

SIR,—The article on dyspareunia by Mr. W. T. Fullerton (3 April, p. 31) has interested me. However, Mr. Fullerton seems to have overlooked an important type of dyspareunia in geriatric patients. Such patients are very prone to conceal this symptom. It is frequently owing to a perineal transverse skin ridge that may appear red and denuded, and becomes stretched during intercourse. Simple incision or excision of this tag cures this symptom.—I am, etc.,

N. G. MUSSALLI

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Streptococcal Meningitis

SIR,—I should like to contest the statement made by Drs. S. M. Hempling and M. de L. P. Coutinho (17 April, p. 166) that streptococcal infections are nowadays decreasing in frequency and severity.

I recently had occasion to survey streptococcal infections in patients attending the casualty department of the Leeds General Infirmary. The results of this study[1] confirmed an earlier subjective impression that, far from decreasing, these infections had increased in incidence by a factor of three over the previous five years. The lesions produced were of a wide range, and were not confined to minor skin sepsis.[1] On at least one occasion one of these patients almost succumbed to the infection and de-