It is plain that it is difficult to accept the deductions leading to the range of figures quoted as renal thresholds for glucose. Many of the points made in this paper are of the greatest importance in the detection and management of diabetes, and there is no wish to detract from these, but I do not think the renal threshold figures given are necessarily valid.—I am, etc.,

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1 Renschler, H. E., Weicker, H., and von Baeyer, B., *Germ. med. Monthly*, 1966, **11**, 237.

Source of Pulmonary Emboli

SIR,—Mr. N. L. Browse and his colleagues writing on the management of the source of pulmonary emboli (9 December, p. 596) are to be congratulated on their rational and aggressive approach to this problem. One hopes that their aggression is limited to extensive thrombotic states such as phlegmasia caerulea dolens.

Since December 1966 it has been my policy to precede or follow pulmonary embolectomy (according to the patient's condition) by disobliterating involved veins. In one instance this policy was not followed. That patient suffered a massive pulmonary embolus seven days after a hip operation. A successful embolectomy was performed, delivering an 18-in.-long (45-cm.) clot. For reasons outside our control simultaneous thrombectomy could not be done. The patient succumbed four days later to a second massive pulmonary embolus. It was of similar length and arose in the opposite leg. This case without doubt weighs in favour of pulmonary embolectomy and simultaneous venous thrombectomy. Similarly most cases of pulmonary embolism can be looked upon as an indictment of failure to adopt measures advocated by Mr. Browse and his colleagues.

While the most reliable form of treatment of extensive deep vein thrombosis is by thrombectomy, followed by heparin perfusion and long-term phenindione, one feels compelled to add that:

(1) When ligation or plication is considered, it should be confined to the superficial femoral vein only.

(2) When pulmonary embolism occurs, pulmonary embolectomy should be accompanied by thrombectomy of the affected veins.

(3) It is difficult to condone plication or ligation of the inferior vena cava. These procedures in themselves create conditions favouring further vein thrombosis. Ligation of the inferior vena cava should be limited to recurring septic emboli.[1] Spencer,[2] originator of plication of the inferior vena cava, reporting on long-term results on 39 cases,[3] states that there were six immediate postoperative deaths, a further three within two years, four had immediate and extensive vein thrombosis, nine of the surviving patients had leg oedema, and two had recurrent phlebitis.—I am, etc.,

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Psychotropic Drugs

SIR,—Dr. H. M. Flanagan (13 January, p. 119) is correct in pointing out the increasing use of the label "depression," and not only in psychiatry. It is the "nuciform sac" of the 1960s, taking the place of intestinal stasis or focal sepsis or visceroptosis as a diagnosis of the undiagnosable.

However, it is difficult to share his despondency about it. We should be delighted that medical fashion has for once settled for something whose treatment is fairly simple, reasonably effective, carries relatively little risk to the patient, and above all involves no permanent mutilation.—I am, etc.,

R. ROUSE.

Hythe,
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SIR,—I hope I may be given the privilege of replying to what has proved a long and, I hope, profitable correspondence. It was indeed gratifying to find how many clinicians there were prepared to confirm the value of the monoamine oxidase inhibitor drugs when used in suitable neurotic depressions and anxiety states. But such patients would very rarely agree to enter a mental hospital for prolonged "double-blind sampling," and they were therefore conspicuously absent from the group of severer depressions used to provide the misleading M.R.C. report.[1]

It was also not unexpected to learn that a group of four mental hospital doctors, including Dr. P. Leyburn (14 October, p. 108), working in the Newcastle area, had failed to find as yet the sort of patient benefiting so much from the monoamine oxidase inhibitor drugs, although both their neurological[2] and psychiatric[3] colleagues at the Newcastle General Hospital have already paid written tribute to their efficacy in properly selected patients. One of the troubles about doctors training and working predominantly in our mental hospitals is that they apparently see so many severe depressions, and not enough minor depressive and anxiety states, which are mostly seen in general practice and in general hospital psychiatric units. They also seem to have the idea that you can justifiably treat psychotic depressions with drugs, but neurotic depressions and anxiety states should preferably be given sex talks or group therapy. And if they do use any drugs they mostly use those which they have found to help their severer depressions but are of little use in the neurotic ones, and often even make them much worse.

It is paradoxically the general practitioners who are now learning to their delight that neurotic depressions and anxiety states often do very much better with selected antidepressant drugs than any amount of talk about sex or other matters. So often the time to talk to both the neurotic and psychotic patients about their problems is when they have responded to the drugs and not always during the illness itself. For so often, as the symptoms dissolve under drugs, the problems also resolve with them, and very little supportive psychotherapy is then needed of a commonsense nature.

In my 30 years in psychiatry I have seen the most refined statistics used time and again both to prove and to disprove the value of insulin treatments, E.C.T., leucotomy, and now all the new tranquillizers and antidepressant drugs. One is therefore constantly forced back to the bedside to find out what really works in selected patients and what does not. It seems that people working with statistics often tend to lump both neurotic and psychotic patients together in a very crude manner, and give the drugs equally crudely in their statistical tests. When statistics obviously *contradict* clinical findings I have now learnt to treat them with the very greatest suspicion, especially in psychiatry. I am certain that general practitioners are not quite as foolish as Dr. Leyburn thinks in using the new antidepressant drugs so intensively. For so many of them have found clinically what he does not seem to be able to find by his crude statistics, that the antidepressant drugs, properly used, have very great value for properly selected groups of patients. And they are certainly not mere placebos given for the doctors' own benefit, as he suggests in his last letter (18 November, p. 417).—I am, etc.,

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Use of Epsilon Aminocaproic Acid

SIR,—In congratulating your expert contributor on the concise and helpful account of the use of epsilon aminocaproic acid (23 December, p. 725), may I comment on points raised in connexion with the diagnosis and management of fibrinolytic states, particularly as they occur in pregnancy and labour?

Your contributor remarks that "primary hyperfibrinolysis is difficult to distinguish from other causes of the defibrination syndrome." However, a shortened euglobulin clot lysis time,[1] or dilute blood clot lysis time,[2] does usually indicate pathological fibrinolysis. These tests are rather complicated and time-consuming, which precludes their routine use in clinical practice. Besides, two relatively simple tests are available for the assessment of the fibrinolytic activity in emergency situations. These are a prolonged thrombin clotting time,[3] not corrected by a mixture of patient's and normal plasmas, and a positive agglutination test with the Fi-test reagent (Hyland Laboratories, California), using patient's serum.[4] Both these tests are indirect tests for fibrinolysis (dependent upon the presence of an excessive amount of fibrin degradation products), but the simplicity of these tests should appeal to the clinician.

Your contributor further states that the "Treatment [of defibrination syndrome] should *begin* [my italics] with controlled replacement of fibrinogen and other missing factors. . . ." However, the primary aim of the treatment should be to prevent further infusion of thromboplastins into the circulation and arrest the phase of hypercoagulability. This is achieved in patients with abruptio placentae by amniotomy and prompt delivery.[5] Until this is achieved transfusion of fibrinogen causes further intravascular coagulation or lysis of transfused fibrinogen, depending upon whichever process is operative. If the problem is one of lysis the