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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REFERENCE

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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 post-operative 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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² Spencer, F. C., *Surg. Forum*, 1960, 10, 680.
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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.,

Hythe,
Southampton.

R. ROUSE.

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 neurological² and psychiatric³ 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 common-sense 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 tranquilizers 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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REFERENCES

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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

formation of anticoagulant fibrinogen degradation products aggravates the haematologic defect. Moreover, to control the replacement of fibrinogen, as is suggested, is difficult, since the laboratory tests commonly employed in acute fibrinolytic conditions (thrombin time, Fibrindex, etc.) are at best semi-quantitative. Even if a rapid method of fibrinogen estimation is used⁶ the findings can be misleading, for the blood level of fibrinogen may be higher than that indicated by the test owing to the presence of anticoagulant fibrinogen degradation products.⁷ In our experience in obstetrical patients^{4,5} the clinical condition of the patient may be a better guide to therapy than the fibrinogen level determined in the laboratory, and fresh whole blood is a better substitute than fibrinogen alone.

Lastly, the risk of E.A.C.A. causing intravascular thrombosis (apart from that presented by the original stimulus for defibrination) when it is given for a relatively short period (as in acute obstetrical conditions) is negligible. This is because the drug is excreted through the kidneys very rapidly.—I am, etc.,

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Polycythaemia and Chorea

SIR,—Your leading article (23 December 1967, p. 696) referring to Gautier-Smith and Pranker's two cases of progressive chorea due to polycythaemia rubra vera prompts me to summarize a similar case.

An old lady of 77 was referred to me at Barnet General Hospital in October with a six-months history of fidgety movements. Examination revealed that she was very arteriosclerotic, and the blood pressure was 190/100 mm. Hg. She showed grimacing movements of the face and writhing movements of the tongue, and had a moderately severe dysarthria. There were marked choreic movements to the trunk, leading to a lurching gait, and typical choreiform movements of the fingers and toes. Apart from extensor plantar responses the rest of the neurological examination was normal.

Regarding her as a probable case of senile chorea due to cerebral atheroma, treatment was started with thiopropazate (Dartalan) 10 mg. three times a day. At her next attendance one month later she was remarkably improved, and her daughter, an excellent witness, stated that the movements had ceased completely two days after the drug was commenced. However, the blood taken at the first attendance showed that the haemoglobin was 18.5 g./100 ml. and packed cell volume was 66%. A full blood count was

therefore ordered, and revealed: R.B.Cs 8,000,000/cu. mm., haematocrit 64%, M.C.H.C. 29%, M.C.V. 80 cu. mm., platelets 320,000/cu. mm., white blood count 8,800/cu. mm., neutrophils 58%, eosinophils 3%, basophils 2%, lymphocytes 36%, and monocytes 1%.

Re-examination at this time showed no enlargement of spleen, and this, together with a normal white cell count and platelet count, suggested that the polycythaemia was probably of secondary type, perhaps due to chronic bronchitis. It was thought advisable to withdraw 1 pint (0.6 l.) of blood, but this had no noticeable effect on the patient. Because of the side-effect of drowsiness the thiopropazate was progressively withdrawn, but at a dose of 10 mg. daily the choreiform movements returned, although not so severe as when she was first seen. She is now well controlled on thiopropazate 20 mg. daily together with orphenadrine 100 mg. daily.—I am, etc.,

London W.1. K. W. G. HEATHFIELD.

Transplant Lung

SIR,—We feel that the authors of the article on transplant lung (13 January, p. 80) are premature in describing this as a new syndrome. Previous reports by Rifkind¹ and Hamburger² have referred to "transplant pneumonia." The discussion by Mr. Slapak and others, however, on the pathogenesis of this syndrome in terms of alveolar-capillary block is extremely speculative for two reasons. Firstly, they produce no histological evidence of a thickened alveolar membrane; in the literature they have cited there is one illustration¹ showing hypertrophied alveolar cells in a case which also shows the alveoli filled with a fluid containing many cysts of *Pneumocystis carinii*. Secondly, their evidence that the dysfunction in lung physiology is a decrease in oxygen diffusion is weak because they do not report the effect of breathing 100% oxygen, a procedure which should overcome a diffusion block.³ In addition, it has been recognized for some time that the commonest cause of hypoxaemia, hypocapnia, and a low CO₂ diffusing capacity is imbalance of pulmonary ventilation and perfusion.⁴

The sentence "Our patient's vital capacity was 100% of the predicted value but his F.E.V.₁/V.C. was 78%, showing a mild degree of restriction," appears contradictory to us. Restrictive lung disease can only be diagnosed when the vital capacity is reduced. The F.E.V.₁/V.C. ratio is a measure of airways obstruction; the value quoted is normal.

We do not feel the authors have fully assessed the role of alveolar and interstitial oedema, especially since the chest x-rays of their cases are similar to those found in pulmonary oedema. The evidence for an immunological basis for this syndrome is extremely circumstantial. The decline in lung function was not accompanied by any deterioration in renal function, as might be expected if cross-reacting antibodies are involved. It is also surprising that investigators have so far been unable to reproduce this condition experimentally in the dog (and in our experience we have not encountered it either), since in many other respects the dog, after renal transplantation, has shown similar changes to those described in man.

There is little evidence for the conclusions these authors have reached or the hypotheses they have proposed.—We are, etc.,

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Rinsing the Dialyser

SIR,—I read with interest the paper by Dr. D. B. Evans and others (16 December 1967, p. 651) on different techniques of washing-back the blood from the two-layer Kiil dialyser at the end of dialysis. Evidence is presented that a rinse with 1 l. of normal saline (after tipping the dialyser vertically during half an hour) yields best results. However, with this technique the patient receives 500–700 ml. of 0.9% saline at the end of each dialysis. In patients on twice-weekly haemodialysis this equals an extra weekly sodium load of approximately 150–214 mEq. This seems to be highly undesirable in chronically dialysed patients who need rigorous dietary sodium restriction, which is (together with appropriate ultra-filtration during dialysis) the cornerstone in correction of hypertension.

I refer therefore to a recently published air-rinse technique¹ which yields equally excellent results without flushing extra water and sodium chloride into the patient. Results (approximately 5 ml. residual blood loss in the dialyser) were confirmed in our unit. Dangers of air embolism are virtually zero in nurse-supervised hospital dialysis and might be eliminated in home dialysis procedures by a rather simple device which blocks automatically the outflow bloodline immediately before the venous cannula when the first air bubble passes a photocell.—I am, etc.,

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REFERENCE

- 1 Pollard, T. L., Barnett, B. M. S., Eschbach, J. W., and Scribner, B. H., *Trans. Amer. Soc. artif. intern. Organs*, 1967, 13, 24.

Elastic Band Injuries

SIR,—I was interested to read the series of letters following the description of artifact ulcers produced by elastic bands by Mr. I. D. Kitchin and others (22 April 1967, p. 218). Dr. L. Cohen (6 May, p. 376) described the result of allowing an elastic band to slip over a tooth, and Dr. J. G. B. Thurston (same page) reminded us of his previous description of ulcers produced around fingers by elastic bands. Mr. J. P. Turney (13 May, p. 445) described an ulcer in the upper arm produced by a group of three elastic bands, and Dr. K. Dawson-Butterworth (20 May, p. 510) described skin ulceration with a deeply buried elastic band in the lower third of the