loss will be sustained by him. The very inelasticity of the envisaged method of payment will alone ensure this.

I would suggest that compensation to the extent of £5,000 for every ten years of specialist service, with a maximum of £10,000, should be the agreed damages for this loss of expectation. Such a course would imply the Government's confidence that its new scheme will save the contributor from paying out fees to specialists, and would underline its honesty.

If a specialist's private practice is largely to be abolished the corollary is that he will have to support himself equally largely out of the salary he is paid for his duties at hospitals to which he is attached. My second suggestion is that £500 per year per appointment is the absolute minimum rate of payment. In addition sessional opportunities for work at a rate of £250 per session should be made available to cope with the pressure of the extra work that in theory should arise, and also to permit the practice-starved specialist to survive without existing on his capital, even if augmented by the aforementioned damages.

Thirdly, I would suggest that the offer of alternative appointment should only be considered if strictly *equivalent* facilities and opportunities are offered. A take-it-or-leave-it condescension can find no room in the contracts to be signed.

Here I would recommend that a committee of assessors should be appointed. It should be constituted principally of specialists, and if the equivalence of a proposed alternative appointment was in dispute the matter should be submitted to them for adjudication, and their ruling should be binding on both parties of the contract. A similar body should be set up to advise on any differences of opinion between employer and employed. Their services will surely be needed in the future to restrain the commissars on our door-step.

I should like to submit these four points for consideration and incorporation in a specialists' "charter." But above all is there not sufficient time to devise and arrange for signature a mutual form of guarantee among specialists whereby we declare our solidarity in the defence of these principles and our singleness of purpose to explore ways and means to enforce them? We are fortified morally. The change-over means the exchange of freedom for thralldom and the replacement of the principle of reward for striving by the use of the big stick for the lack of effort.

Well turbaned our Minister may traverse our Harley Streets in his quest to exchange his lamp for Aladdin's own. Let the specialists recognize this so that they may live happily ever after.—I am, etc.,

London, W.1.

JOHN SOPHIAN.

Activity of Liver Extracts

SIR,—In your issue of April 10 (p. 701) a report is given of a discussion on the megalocytic anaemias at the Royal Society of Medicine on March 23, 1948. I was surprised to read that Dr. J. F. Wilkinson stated that he had never been able to obtain an active liver extract from the liver of fishes after the oil had been removed. I would therefore like to draw attention to the fact that I produced a very potent oral liver extract from fish liver in Aberdeen in 1932, and that a full report of the haematological findings of patients treated with this extract was published in the *British Medical Journal*.¹ Two of the cases were treated with this extract by Dr. Wilkinson himself in Manchester. He reported to me that my extract of fish liver had resulted in the blood count being quadrupled in one case in three weeks and trebled in another case in thirteen days. Acknowledgment was made in this paper to the independent testimony of Dr. J. F. Wilkinson regarding the efficiency of fish-liver extract. It is to be noted that an aqueous extract of fish liver has also been used successfully by Connery² in America. The introduction of parenteral liver therapy shortly after we had successfully produced active oral liver extract from fishes was the reason why this method of production was discontinued, as it is generally agreed by the majority of leading haematologists all over the world that parenteral therapy has considerable advantages over oral therapy with liver or hog's stomach preparations.

Dr. Wilkinson, when discussing megaloblastic anaemias refractory to highly purified liver preparations, stated that it was essential that the individual batch of the liver preparation to which the patient was refractory should have been tested for

potency on a classical case of pernicious anaemia. Without such knowledge the refractoriness might be due to an inactive batch of liver extract being used. I would entirely agree with this suggestion. During the 20 years which have elapsed since I first published a report on the testing of liver extract I have tested the activity of a large variety of liver preparations, both commercial and experimental, in many hundreds of cases of pernicious anaemia. I always have at my disposal batches of liver extract of proved potency, which are available for use in cases of megaloblastic anaemia which have failed to respond partially or totally to liver therapy.

Refractoriness to parenteral liver therapy occurs most frequently in nutritional megaloblastic anaemia, in megaloblastic anaemia associated with the sprue syndrome and pregnancy, and in a small number of cases of megaloblastic anaemia in which no cause for the refractoriness to parenteral liver therapy can be discovered. In all these groups free hydrochloric acid may or may not be present in the gastric juice. The degree of refractoriness depends partly on the type and potency of the liver preparation given and its route of administration, and partly on personal factors in the patient, some of which are known and others of which are unknown. It is therefore essential when defining refractoriness to liver therapy to state clearly the criteria used.

In a paper entitled "Refractory Megaloblastic Anaemia," which appeared recently in *Blood*,³ I have defined the term "refractory megaloblastic anaemia" as used by myself, and submitted records of a variety of cases of megaloblastic anaemia which failed to respond to potent preparations of purified liver extract and thereafter responded to folic acid or proteolysed liver. Anyone interested in refractory megaloblastic anaemia can consult this paper for further details.—I am, etc.,

Edinburgh.

L. S. P. DAVIDSON.

REFERENCES

- ¹ *British Medical Journal*, 1932, 2, 347.
- ² *Amer. J. med. Sci.*, 1930, 130, 603.
- ³ *Blood*, 1948, 3, 107.

* Dr. J. F. Wilkinson's paper, to which Prof. Davidson refers, was read at a meeting of the Royal Society of Medicine and only a shortened version was published in the *Journal*. To avoid misunderstanding we showed Prof. Davidson's letter to Dr. Wilkinson, who tells us that what he in fact said at the meeting was, "Extracts of fish livers (cod, halibut, salmon, etc.), and also whale liver, when prepared from fresh livers, are haemopoietically very active, both orally and parenterally, but commercial fish and whale liver discards (residues) after the extraction of their respective oils by superheated or live steam do not furnish active preparations at all . . . and I have yet to obtain an active liver extract from reptilian and amphibian livers."—ED., *B.M.J.*

"Alkalosis" and the Effects of Alcohol

SIR,—I was interested to read the medico-legal case concerning alkalosis and the effects of alcohol (May 1, p. 861). It immediately called to mind a case under my care in 1936 when I was a house physician.

My patient, aged somewhere in the late forties, had a radiologically proven active duodenal ulcer. My Chief ordered his routine treatment at that time, which included 1-2 hourly feeds, with alkalis 2-hourly, but not belladonna or atropine. Within a day or so of the onset of this treatment the experienced sister-in-charge said to me that the behaviour of this patient was a bit odd at times. The following morning the night report showed that the patient was getting out of bed and had threatened to commit suicide. In the morning when I spoke to him he was most confused; his concentration was very poor, as was his memory, and it was not possible to hold his attention for any length of time. He would keep looking all around him, was restless and fidgety. Of course delirium tremens leapt to mind. However, the mental confusion and the very furred and dry tongue reminded me of a case of uraemia that I had seen, and a blood urea estimation was made. This showed a blood urea of 144 mg. %.

A tentative diagnosis of alkalosis was made. Alkali administration was stopped at once and the patient given frequent meals of well-cooked, easily digestible, and palatable food. By the next day he was much better. Blood urea estimations were carried out every 2 or 3 days; they eventually dropped to within normal limits. As there was no other cause for the uraemia it was now presumed that the cause of this as well as the patient's abnormal conduct was alkalosis. I must add that there never was hypertension, nor any abnormal findings on ophthalmoscopy, and that the urine never showed any albumin, red cells, or casts. At no time had there

been oedema, diarrhoea, or vomiting. No other drugs had been given and the patient's conduct prior to the commencement of treatment in hospital had been normal.

The patient was now a pleasant and co-operative individual who was rather vague about what had been happening to him during the days when his behaviour was abnormal. He scarcely ever touched alcohol because of his disability and had no previous history of mental aberration.

It is not too difficult to imagine what might have happened had this patient over-indulged in alkalis as an out-patient and had one whisky in milk, as some individuals I know are apt to do, followed by a car accident. The examining doctor would have observed a smell of alcohol and abnormal conduct, especially with the excitement of a car accident and a medical examination in such circumstances. Might not a doctor be excused for insisting on a diagnosis of alcoholism when the abnormal mental conduct would be predominantly the result of alkalosis?

A colleague of mine informs me of a case of urinary infection who died from alkalosis in the pre-sulphonamide days, when massive alkaline therapy was the order of the day. A blood urea estimation was suggested by an astute and enthusiastic nurse who had been swotting Price. It would appear to me that alkalosis should be kept in mind in all cases where alkalis are administered, especially when massive alkaline therapy is adopted. I have never forgotten the case that I have just described, and it has served me well.—I am, etc.,

B.A.O.R.

JOHN MACKAY-DICK,
Lt.-Col. R.A.M.C.

The Mystery of the Eosinophil

SIR.—Some years ago I took considerable interest in the eosinophil, and it left me with the impression that it is part of a defence mechanism, and that the occurrence of eosinophilia not otherwise explicable suggests the presence of a known, unknown, or facultative allergen.

Morphologically, the polynuclear leucocyte and the eosinophil are practically identical. They differ only in their staining reaction and granularity. The production of eosinophils can be provoked experimentally by infestation with intestinal or blood helminths, and it appears that the resulting eosinophilia is a response to a specific stimulus, in effect a foreign protein—a modified polymorph, in fact. In true allergic bronchospasm the exudate from the upper air passages is crowded with eosinophils, and in sensitive persons the spasm may be induced by various air-borne allergens—which appears to be not without significance.

There is not space in a brief letter such as the present to discuss all the conditions in which eosinophilia is a marked feature, but a line worth following is based on the theory that the granules in eosinophils are possible precursors of lysins the object of which is to effect lysis of the responsible allergen. My work convinced me that eosinophilia is a *result* and not a cause, and when it is present the cause has still to be sought. I certainly cannot agree with the "overflow" theory of eosinophilia in lymphadenoma, the cause of which is still unknown. In "eosinophilic granuloma" of skin the name suggests that eosinophilia is the cause of the granuloma rather than the result, as also in "tropical eosinophilia" and eosinophilic disease of the lungs. I have seen individuals who give an abnormal eosinophilic response to trivial or comparatively trivial causes: for example, a 60% count in a case of sunburn with a so-called "herpetiform" eruption of the lip and cheek.

I am certain that further work on the subject is long overdue and would be amply repaid, just as I am convinced that eosinophilia is protective in intention and never causative, however high the count.—I am, etc.,

Brookwood, Surrey.

H. M. STANLEY TURNER.

Treatment of Phlebothrombosis

SIR.—Mr. Hamilton Bailey's article (March 27, p. 594) emphasizes the need of active treatment for patients with post-operative thrombosis and pulmonary embolism. There is certainly great need for this emphasis, since complete immobilization of the leg for several weeks is still a common practice. Nor is this surprising, since the majority of textbooks published during the last two years still advise this conservative treatment

for femoral thrombosis. The diagnosis of incipient deep calf thrombosis is rarely even mentioned.

Nevertheless I do not believe that ligation of the femoral vein is the solution to the problem for the following reasons. First, in certain cases of pulmonary embolism, especially following gynaecological operations, the site of origin of the embolus cannot be detected in spite of careful examination of the legs. Would Mr. Bailey here advocate the ligation of the inferior vena cava, a serious operation in an ill patient, however expertly it may be performed?

In the second place, the end-results of thrombosis of the deep leg veins must be considered. Bauer has shown that nearly four out of five of such patients eventually develop a leg ulcer. It is difficult to believe that the ligation of the saphenous and common femoral veins in a patient who already has obstruction to some of the deep venous channels will not increase venous stagnation and consequently the risk of developing an ulcer. As the sequelae of chronic venous stagnation may take ten years to develop, the end-results of venous interruption may be more severe than is at first supposed.

On the other hand the anticoagulant drugs have been shown to be at least as effective as femoral ligation in the immediate treatment of venous thrombosis and pulmonary embolism, as was demonstrated by Mr. Gavin J. Cleland (April 17, p. 755). They have the added advantages of controlling venous thrombosis wherever it is situated, and of preventing the spread of thrombosis to the opposite leg, which occurs in 30% of cases treated conservatively. Moreover, as Zilliacus has shown, anticoagulant treatment prevents the spread of thrombosis from the calf to the thigh and thereby the chronic swelling and ulceration which otherwise frequently occurs. From the practical standpoint heparin is usually not difficult to administer. I have found that patients prefer a continuous drip transfusion to intermittent injections, provided that the needle is inserted at the wrist and not at the elbow. The production of a suitable delayed-acting heparin, such as heparin (Pitkin), which is now being used in the United States, would make heparin treatment much easier still. The cost of treating a patient with heparin is often no more than that of keeping him for one week in hospital. As the use of anticoagulants reduces the stay of the patient in hospital by four to six weeks it is indeed economical.

Where facilities for the estimation of the prothrombin concentration of the plasma are available, a combination of heparin and dicoumarol is probably the best treatment available. I agree with Mr. W. M. Capper (April 24, p. 807) that vein interruption may be necessary for cases in which anticoagulants are contraindicated, but I believe they form only a small minority. For most cases the anticoagulants are completely effective and should, in my opinion, be considered as essential to the treatment of venous thrombosis as insulin is to that of diabetes.—I am, etc.,

London, N.W.10.

KEITH BALL.

Origin of Depression

SIR.—The publication of Dr. W. Clifford M. Scott's paper on "A Psycho-analytic Concept of the Origin of Depression" (March 20, p. 538), which he delivered to the Section of Psychiatry at the International Congress of Physicians last September, has provided an opportunity for more careful consideration of his views. In opening the discussion at the meeting I felt bound, though with some diffidence as laying no claim to be abreast of recent developments in psycho-analytic theory, to raise anew that often repeated doubt about the reliability of "memories" referred back to infancy, to which Dr. Scott and others of the Freudian persuasion attach a fundamental importance. Especially in such emotional states as depressions which are profound enough to call for intensive treatment, how can we ever really recall what went on in our minds at the tender and largely unmyelinated age of 2 or 3, unless indeed, as must often happen, we have been told by our parents at some later date what happened to us then and how we reacted?

Even when true memories are recalled under psychological investigation, the mood of the present, if sufficiently intense, influences the selection of what is recalled from the past and the significance attached to past experiences. Are we to assume that all depressions are determined by past experiences, rather than they determine what is recalled about the past? As no report of the discussion which followed Dr. Scott's paper is being published, I take leave, Sir, to make these observations.—I am, etc.,

Sydney.

W. S. DAWSON.