house of the patient, and leprosy is so obviously a house disease, especially in poor squalid overcrowded huts, where in warm climates cockroaches abound. From one African hut no fewer than 2,500 cockroaches were caught in one night! It is not generally known that cockroaches bite man, and in doing so remove a piece of skin, which they swallow, leaving a small bleeding wound which later becomes a characteristic whitish depressed scar lasting for many months. Neither my African assistants nor I have ever actually seen a cockroach in the act of biting, but there is not the slightest doubt about it. The natives have reason to know the bites well.

In my leprosy hospital these wounds and scars have repeatedly been found to contain Hansen's bacilli, even when the organism could not be demonstrated elsewhere. Children are much more commonly bitten than adults, which probably explains why the disease generally develops in them.

Within the hospital about 23% of cockroaches taken from huts occupied by lepromatous patients are found to contain masses of Hansen's bacilli. Outside the hospital it is not usual to find acid-fast bacilli in cockroaches, though they have been found at times. The bacilli occur in large numbers in the faeces of cockroaches, and can pass from roach to roach by this means, since they feed on anything, from paper, clothing, and blacking to flesh and the dead bodies of other cockroaches. The feeding propensities would explain why, in one kraal from which nine patients had been admitted at various times, the last one five years earlier, acid-fast bacilli indistinguishable from Hansen's were found in a roach caught in a hut. The infection must have remained in that kraal in the shape of infected cockroaches and faeces. Hansen's bacilli have been demonstrated in faeces kept in a stoppered tube for 29 months, and were found to stain as deeply as when fresh.

Now, experimental inoculations of laboratory animals have failed to be of value, as have attempts at cultivation, so that man remains as the only object of experimentation. African natives do not fear the disease, but volunteers should be sought in other parts of the world, and should be inoculated (1) by allowing infected cockroaches to bite them, and (2) with infected faeces. One such experiment, has produced suggestive though not conclusive results.

—I am, etc.,

Kenya.

BERNARD MOISER.

Prognosis of Pleural Effusion

SIR,—May I at this late date reply to Dr. J. D. L. Reinhold's letter (June 7, p. 824) relating to my paper on this subject published in an earlier issue. The 190 patients concerned were, almost without exception, treated initially with prolonged bed-rest and a convalescent or sanatorium regime, and there was no evidence that differences in treatment of the original pleurisy affected the subsequent incidence of pulmonary tuberculosis. Although I continue to keep such cases in bed at least until their blood sedimentation rate becomes normal, I have the uneasy feeling that in this, as in other manifestations of the evolution of a primary tuberculous infection, its fate depends on factors probably outside of medical control. The essential thing is close observation during the ensuing 12 to 18 months, when most of the phthisis (i.e., tuberculosis that we can treat) occurs.

Dr. Reinhold and anyone else who is interested will find a much fuller account of this work, with details of the intra-thoracic lesions encountered before and after the onset of pleurisy with effusion, in the *American Review of Tuberculosis* of October, 1946.—I am, etc.,

Dunedin Hospital,
Dunedin, New Zealand.

BRIAN C. THOMPSON,
Medical Director, Tuberculosis Service.

"The Thalamic Syndrome"

SIR,—I was careful in my previous letter (July 19, p. 109) to limit my criticism of Dr. G. Tayleur Stockings's article to the statements with which he prefaces the account of his work on a euphoriant drug. Work of this type is always valuable if only for the light which it may throw on the mechanism of mental disorder.

My main criticism of Dr. Stockings's article is that he advances his theory that neurotic depression can be equated with thalamic dysfunction as though it were a clearly demonstrable fact. This criticism also applies to his claim that psychotic and neurotic depression can be differentiated by a specific response to "anoxic" therapy. I do not think the evidence so far adduced in support of this claim justifies its being presented to the readers of the *Journal* as though it were a well-recognized fact. Theories are useful where they lead to further research and

enable predictions which can be checked to be made. If, however, they are advanced as assertions they will not serve this purpose but will merely serve to confuse the interested reader who has no specialist knowledge of the subject.

In my simplicity I was unable to see any difference between "thalamic syndrome" and "thalamic dysfunction syndrome." I must confess that even after reading Dr. Stockings's explanation (Aug. 2, p. 187) I would still draw the inference if I saw the name of an organ attached to a syndrome (e.g., pituitary syndrome) that the symptoms which compose the syndrome were being attributed to dysfunction of that organ.

I think that the true role of cortex, basal ganglia, thalamus, and other parts of the brain and the relative importance of constitutional defects and external stimuli in the production of mental disorder can only be established as a result of very extensive research in which chemical agents will no doubt play a very important part. Meantime, in order to assess results accurately I believe it is very necessary to maintain clearly the distinction between experimentally proven fact and theories with little experimental support.—I am, etc.,

St. Mary Cray, Kent.

BRIAN H. KIRMAN.

"Mushroom" Poisoning

SIR,—The advice given by your annotator (Aug. 23, p. 302) that, unless we are experts, we should not eat any fungus which is not obviously a mushroom is sound, even if it deprives us of the edible fungi described by Mr. John Ramsbottom (p. 304). But unfortunately the really deadly fungus, *Amanita phalloides* (the Death Cap), may be easily mistaken for the edible mushroom and passes the popular (fallacious) tests of edibility. I think, therefore, that emphasis should be given to the fact that the gills of the Death Cap are permanently white, while those of the edible mushroom never are, and that the public should be told, "Never eat a 'mushroom' with white gills."

I have met only two cases of Death Cap poisoning and both patients were moribund when seen. Details of possible treatment were hard to find among the "accumulated mass of clotted bosh" which Mr. Ramsbottom rightly condemns. As "mushroom" poisoning may prove to be a very serious emergency, I suggest the following scheme for any future cases:

1.—If symptoms began within five hours of ingestion, wash out the stomach and leave in it 2 fl. oz. (57 ml.) of "white mixture."

2.—If there are blurred vision, sweating, salivation, or other symptoms suggestive of muscarine poisoning, give atropine gr. 1/50 (1.3 mg.) subcutaneously to an adult.

3.—If the onset of symptoms is delayed over five hours, the doctor should ask himself, "Is it Death Cap poisoning?" In deciding this point, have an expert opinion on any uneaten fungi and on the spores in the vomit. Failing this evidence, a delayed onset of symptoms should make the doctor decide in favour of Death Cap poisoning. There are three possible main lines of treatment, which should be given promptly: (a) Obtain and administer anti-phallic serum: Apply to the Director, Central Public Health Laboratory, London, N.W.9. (Telephone Colindale 6041). The dose will be indicated on the amount sent. (b) Commence treatment for failure of liver function (glucose, calcium, vitamin B₁, vitamin K, etc., and possibly blood transfusion). (c) The rabbit stomach-brain treatment of Limousin and Petit. This is based on the fact that, while cats die after eating *Amanita phalloides*, rabbits do not. But the juice of *Amanita phalloides* injected subcutaneously into rabbits is fatal, suggesting that the toxin is destroyed or neutralized in the rabbit's stomach. Cats fed on *Amanita phalloides* plus rabbit's stomach survive several days. If the rabbit's brain is given also, they recover completely. The treatment recommended is to give up to 5 uncooked rabbits' brains and stomachs minced up, daily for several days. Its practicability in a vomiting patient seems questionable, but the emergency is so desperate that it should be tried.—I am, etc.,

Enfield, Middlesex.

C. ALLAN BIRCH.

Tuberculosis and Malnutrition

SIR,—Dr. Agnes C. Clark (Aug. 9, p. 226) raises a very interesting point regarding tuberculosis and malnutrition. The usual teaching is that pulmonary tuberculosis occurs more frequently in an undernourished population and tends to be more severe. In her D.P. camp the exact incidence is not stated, but recovery appears to be very quick with the minimum treatment.

The obvious explanation is that the Poles and Ukrainians have some racial immunity to tuberculosis. This, however, has never been proved, and an epidemic of tuberculosis in a Siberian P.O.W. camp reported by Planner-Wildinghof (1921)¹ affected all races equally and was not influenced by any racial difference or any other form of selection.

The other probability is that an undernourished patient, contrary to the usual teaching, is not a suitable host for the tubercle bacillus. Study of the latest Austrian statistics recently acquired in Vienna seems to confirm this.

Pulmonary Tuberculosis in Austria

Year	Notification of Pulmonary Tb.	Deaths from Pulmonary Tb.
1940	8,611	6,086
1941	9,461	5,789
1942	11,777	6,179
1943	13,475	6,204
1944	11,775	7,027
1945	8,916	5,043
1946	12,467	4,900
1947 (first half) ..	6,759	2,118

The notification of cases is, I think, not sufficiently accurate for comparison each year, as cases are now more readily notified in order to obtain the dietary extras for such patients. The numbers of deaths are much more interesting. Until the end of 1944, Austria was presumably one of the better-fed countries of Europe. With the chaos in Austria before and after the cessation of hostilities in 1945 there was mass hunger for the first time, yet the deaths from pulmonary tuberculosis dropped by nearly 2,000 to just over 5,000. Another bad year nutritionally for Austria was 1946, yet the deaths from pulmonary tuberculosis dropped to under 5,000. This was in spite of gross overcrowding in the towns and a shortage of sanatorium and hospital beds. In 1947, after two years of severe malnutrition and a very hard winter, the deaths from pulmonary tuberculosis in the first six months were just over 2,000.

These figures, taken with Dr. Clark's experiences in D.P. camps, suggest that there may be something in the state of malnutrition which prevents the more serious and fulminating types of pulmonary tuberculosis from developing. It is known that by culturing the tubercle bacillus in unsuitable media an attenuated form of the bacillus will be produced (c.f. B.C.G.). The bacillus, for its optimal development and proliferation, requires an adequate supply of oxygen, glycogen, glycerol, and some amino-acids. Could there be some mechanism which interferes with the acquisition of these or other essentials from the depleted supply in the undernourished host? I feel that this subject has so many interesting aspects that it might well be profitable to carry out some intensive investigation in Austria into the problems of resistance to and recovery from tuberculosis, especially as the present conditions in that country will probably never recur.—I am, etc.,

A. MACFARLANE.

REFERENCE

¹ Quoted by Kayne, G. G., Paget, W., and O'Shaughnessy, L. (1939). *Pulmonary Tuberculosis*, p. 57.

Alkaline Haematin Method for Haemoglobin Determination

SIR,—An alkaline haematin method for the determination of haemoglobin was described by Dr. J. W. Clegg and myself in this *Journal* (1942, 2, 329). The standard recommended in this procedure consisted of a solution of crystalline haemin in sodium hydroxide. The colour of this solution was closely similar to, although not identical with, that of the alkaline haematin solution prepared from blood treated with sodium hydroxide. It suffered from the disadvantage of not being a permanent standard, and it was recommended that new standard solutions be made up at intervals of a few months.

The alkaline haematin method for haemoglobin has recently been re-investigated in the U.S.A. and has been recommended by B. L. Horerker (*J. Lab. clin. Med.*, 1946, 31, 589). The standard employed is a solution of crystalline haemin in borate buffer pH 9.4, which is claimed to be much more permanent than the Clegg and King standard of haemin in sodium hydroxide. The haematin standard in borate has the additional advantage of being more closely similar in colour to the alkaline

haematin solution formed when haemoglobin is treated with sodium hydroxide. I have investigated the Horerker standard and find the claims to be fully substantiated. Alkaline haematin standards prepared in borate buffer appear to remain unaltered for nine months, whereas those in sodium hydroxide are much less permanent (they appear now to be less permanent than we had originally found). The spectral curve of the alkaline haematin standard in borate is closely parallel to that of the alkaline haematin solution made from haemoglobin in sodium hydroxide. There is a quantitative difference in the depth of colour, similar to that found with the Clegg and King standard (1.3 times as much haemin is required to give the same colour as haemoglobin, on a molecular basis); but the spectral curve of the haemin in borate is of a similar shape to that of haemoglobin treated with sodium hydroxide, and does not show the differences seen in the curve of haemin in sodium hydroxide. Horerker compares his test and standard solutions with a yellow-green filter and finds a different factor than the 1.3 mentioned above for the quantitative relationship between the colour of haemin and haemoglobin. However, with the green light filters in use here (Chance OGR 1; Ilford tricolour green, spectrum green, and mercury green) the quantitative relationship between the colour of haemin in borate and in sodium hydroxide appears to be the same; and with these filters, therefore, the same quantity of haemin should be used in borate buffer as was specified by Clegg and King for use in sodium hydroxide solutions.

The findings for a series of solutions of different crystalline haemins in borate buffer and in sodium hydroxide solution are given in Tables I and II. It is seen that the average reading of

TABLE I.—Colour Intensities of Various Preparations of Haemin in NaOH and in Sodium Borate

(Colours read in a King (1942) photoelectric colorimeter with Chance and Ilford light filters, and in a Hilger absorptiometer with Ilford spectrum yellow-green light filter)

Preparation of Haemin		Specific Extinction Coefficients of Haemin Solutions. (Es for 1 mg. Haemin Iron per 100 ml. Solution)*							
No.	% Iron	Haemin in NaOH Solution†				Haemin in Borate‡			
		Chance Green Filter	Tri-colour Green Filter	Mercury Green Filter	Yellow-green Filter	Chance Green Filter	Tri-colour Green Filter	Mercury Green Filter	Yellow-green Filter
16	8.41	0.810	0.806	0.646	0.742	0.821	0.777	0.623	0.741
17	8.26	0.854	0.838	0.672	0.772	0.835	0.816	0.642	0.767
19	8.13	0.857	0.843	0.669	0.774	0.855	0.835	0.654	0.777
26	8.36	0.855	0.836	0.666	0.765	0.843	0.821	0.646	0.768
28	8.00	0.834	0.821	0.660	0.754	0.835	0.816	0.645	0.770
30	8.42	0.816	0.799	0.638	0.730	0.810	0.797	0.625	0.741
DGH	8.03	0.878	0.857	0.677	0.793	0.858	0.842	0.660	0.790
Mean	..	0.843	0.829	0.661	0.761	0.837	0.816	0.642	0.764
Coeff. of variation (%)		2.68	2.62	2.14	2.81	2.04	2.85	2.15	2.36

* The photoelectric readings (extinctions) of these solutions were appropriately factored by the weights of haemin taken and the percentages of iron in the haemins, to give the readings (specific extinction coefficients) which would have been obtained had 100 ml. of each standard solution contained an amount of haemin equal to 1 mg. of iron, and when the solutions were read in the colorimeter at a depth of 1 cm.

† Approximately 40 mg. haemin dissolved in 500 ml. 0.1 N NaOH.

‡ Stock solution: approximately 40 mg. haemin dissolved (overnight) in 50 ml. of Sorensen's borate buffer pH 9.4. Aged for two days in ice-box. Standard: 50 ml. of stock, diluted to 500 ml. with same buffer.

TABLE II.—Stability of Standard Solutions of Haemin in Sodium Borate

(Mean specific extinction coefficients of seven alkaline haematin standards read at various times after preparation)

Age of Solutions	Haemin in Borate Buffer pH 9.4			
	Chance Green Filter	Tricolour Green Filter	Mercury Green Filter	Yellow-green Filter
2 days	0.837	0.816	0.642	0.764
2 months ..	0.838	0.821	0.650	0.770
4 "	0.845	0.828	0.660	0.759
9 "	0.850	0.822	0.659	—
1 year	0.783	0.778	0.636	0.720

the colour for the different haemin solutions is closely similar for both the borate and sodium hydroxide solutions for any given light filter. The keeping quality of the haematin standard in borate buffer is certainly superior to that in sodium hydroxide solution.