ing for a spontaneous return to normal mental functioning as euthyroidism was re-established following the withdrawal of the carbimazole. The previous history of post-partum depression, 14 months prior to the psychotic episode, also suggests that the mental dysfunction may have had other major determinants. As Drs. Herridge and Abey-Wickrama state, however, the continued psychiatric normality since euthyroidism has been re-established is suggestive of some association between the induced hypothyroid state and the psychotic illness.

Certainly, the mental symptoms described, a psychotic confusional state with depressive and paranoid features, are compatible with some of our recent findings published in detail elsewhere.² Careful objective psychological evaluation of persons referred with a diagnosis of thyroid gland dysfunction to the medical clinic of a general hospital revealed that in both hypo- and hyperthyroidism there is a profound disturbance of mental function usually unidentified by the referring physician. Abnormalities of cognitive function occur in both syndromes, but are greater in hypothyroidism, where considerable subjective awareness of the disability is frequently present (women can no longer remember their favourite recipes, are unable to accurately add up the grocery bill, etc.). Evidence suggests the disturbance of cognition increases with the deviation from the euthyroid state and that an increased reporting of bizarre thoughts and beliefs is correlated with this increasing confusion of intellectual function. Hence the "psychoses" occurring in disordered thyroid states are the result of an increasing delirium with the psychotic symptoms *per se* being a "final common path" of disturbance. That the bizarre thought improves as euthyroidism is re-established confirms this. When describing such syndromes it thus seems to me unnecessarily confusing to use the term "schizophrenic type," which implies a specific clinical picture of complex aetiology.

The suicide attempt and the depressive content of the delusions experienced by the patient again reflect our own experience and are of considerable theoretical interest. In our series of patients the hypothyroid individuals were frequently profoundly depressed in mood, but there was a striking absence of sadness in the hyperthyroid persons, suggesting perhaps that an excess of thyroid hormone protects the individual from such affect. Kleinschmidt³ has noted that depression of mood usually precedes the thyrotoxic state, and it has been our experience that depression sometimes follows successful treatment of thyrotoxicosis. Furthermore, thyroid hormones do appear to potentiate the speed of action of the tricyclic antidepressant drugs in the treatment of melancholia.

An explanation of these clinical observations may come from the metabolic links between the biogenic amines and the thyroid hormones. Both catechole and indoleamine metabolism have been implicated as abnormal in the affective disorders and there is now increasing evidence of a deficiency of 5-hydroxytryptamine (5-HT) (or/and nor-adrenaline) in the central nervous system in depression.^{4,5} Thyroid hormone is known to potentiate the effects of the catecholamines⁶ and to increase the level of 5-HT in

the brain.⁷ Thus, carbimazole, by reducing available thyroid hormone, may have an influence on biogenic amines within the central nervous system, thus imparting a depressive affect to any psychosis occurring due to the disordered thyroid state.

Drs. Herridge and Abey-Wickrama are to be thanked for an interesting and stimulating case report.—I am, etc.,

PETER C. WHYBROW.

Neuropsychiatric Research Unit,
Medical Research Council Laboratories,
Carshalton, Surrey.

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Local Hydrocortisone Injections

SIR,—Your expert (12 July, p. 102) gives helpful suggestions on the uses of corticosteroids. He also mentions certain contraindications to the use of intra-articular steroids, but at no point does he warn that all the theoretical work so far published indicates that intra-articular steroids have an effect, in the species studied, which would suggest that any intra-articular injection of hydrocortisone causes local damage to the articular cartilage.¹ The damage appears to be directly proportional to the dosage. Similar damage may occur after injection of hydrocortisone into tendons, and may lead to rupture of even the patellar ligament.²

I believe that if the very real dangers of this form of treatment are not well known then the complications will become more common and a valuable treatment fall into disrepute. I cannot think of an adequate indication for injecting any weight-bearing joint more often than three times a year.

Equally, however, I am among those who believe that hydrocortisone is particularly useful in conditions such as trigger finger and in de Quervain's tenosynovitis, where the tendon is invisible but the pain just as disturbing as in a more superficial tenosynovitis. If the area of tenderness is first infiltrated with local anaesthetic, a No. 12 needle can be inserted into the tendon without causing pain. That the needle is in the tendon can be shown by asking the patient with a trigger finger to flex the terminal interphalangeal joint—the proximal end of the needle then moves towards the tip of the finger (see Diagram). The syringe, containing 25 mg. of hydrocortisone acetate, is then fitted to the needle and pressure applied to the plunger. The needle is withdrawn slowly, with pressure maintained on the plunger, until it is possible to inject the fluid when it will slip between the tendon and the inflamed sheath. Pain relief within four days will occur in the great majority of patients, although painless triggering may persist, at a diminished level, for another month.—I am, etc.,

A. H. G. MURLEY.

Addenbrooke's Hospital,
Cambridge.

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Bacterial Endocarditis

SIR,—In their interesting memorandum on aortic stenosis produced by bacterial endocarditis (12 July, p. 97) Dr. P. V. Sacks and others mention that anticoagulants were given "in view . . . of pulmonary embolism." They also state "Two weeks after admission . . . the murmur of aortic incompetence had become louder and longer." Although the date of starting anticoagulants in relation to the above physical sign is not given, presumably they had already been given for some days at least. Such treatment, however, is contraindicated in bacterial endocarditis,¹ and retrospectively the increasing development of the aortic diastolic murmur was ominously suggestive of progressive disintegration of the valve.

I am sure Dr. Sacks and his colleagues will forgive me if I suggest that, in the presence of bacterial endocarditis, second-line defence measures such as vein ligation, urokinase, etc., would be safer, if faced by thromboembolic complications.

Their picture of the necropsy specimen shows what could be clot attached to the valve margins, but perhaps could also be due to intramural haematoma formation, which might increase the obstructive factor. Bacterial endocarditis was at one time also known as "ulcerative" endocarditis.—I am, etc.,

J. DE SWIET.

East Glamorgan General Hospital,
Nr. Pontypridd, Glam.

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