Humphreys and Delvin postulated a central action, yet when they emphasize the ineffectiveness of the drug it is certainly worth noting that eight of these patients, that is to say, nearly half of them, had a fall in supine diastolic pressure, in spite of the fact that they say that Jamaicans occupy an intermediate position on the scale and that they do not suffer from stress-induced hypertension which is frequently seen in Europeans. Therefore any "tranquillizing effect" of the drug would not be expected to show any beneficial results. There is therefore a certain degree of discrepancy in the fact that they consider the antihypertensive effect of the drug to be due to some form of tranquillizing action, yet stress-induced hypertension is not seen in Jamaicans.—I am, etc.,

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REFERENCES

1 Tewari, S. N., and Grant, R. H. E., *Postgrad. Med. J.*, (in press).
2 Prichard, B. N. C., *Proc. Roy. Soc. Med.*, 1968, Vol. 61, No. 5.
3 Prichard, B. N. C., and Gillam, P. M. S., *Am. J. Cardiol.*, 1966, **18**, 387.
4 Frohlich, E. D., Tarazi, R. C., Dustan, H. P., and Page, I. H., *Circulation*, 1968, **37**, 417.

Antibiotic Cover in Dentistry

SIR,—Mr. R. A. Peebles (22 June, p. 762) comments on the inference that "the onset of the disease was related to the performance of dental treatment" in our first patient with hypertrophic obstructive cardiomyopathy who developed infective endocarditis. Clinically there was no doubt that the symptoms of malaise with profuse night sweats became apparent after the dental manipulation. We therefore assumed that our patient may have developed a bacteraemia at the time of the dental treatment and that this led to endocarditis. We must point out that unfortunately we have no more details of our patient's dental treatment; we also do not know what the state of his teeth and gums was at the time.

Mr. Peebles is quite right to point out that fillings, in the absence of periodontal disease, are not usually a cause of bacteraemia, although a few cases have been reported in the literature[1,2] We agree that fillings alone are not normally dangerous. It would be a counsel of perfection but unnecessary and impracticable to cover all dental fillings, however minor, in susceptible subjects. None the less, failing to do so may rarely be followed by endocarditis. Positive blood cultures have also been reported after mere brushing of the teeth[3] and post-prandially,[4] so complete protection would involve wholesale dental clearance. We therefore advise antibiotic cover only for extractions, scalings, and extensive fillings or when there is periodontal disease.

We probably should have been more explicit in our paper and accept Mr. Peebles's implied criticism. Nevertheless in the regrettable absence of more details about the type of dental therapy we cannot say whether or not it was relevant to the subsequent illness. The point of mentioning it at all was really to help drive home the message of our paper that heart *muscle* disease unlike *valve* disease or *septal defect* is not generally recognized to be at risk from infective endocarditis and therefore in need of prophylactic antibiotics when appropriate.—We are, etc.,

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REFERENCES

1 Wagner, R. P., and Kruger, G. O., *41st Gen. meeting, Int. Assoc. Dent. Res.*, March 1963, edited by H. F. Osborne, p. 68, Abstract No. 135, Chicago.
2 Harvey, W. P., and Capone, M. A., *Am. J. Cardiol.*, 1961, **7**, 793.
3 Cobe, H. M., *Oral Surg. Oral Med. Oral Path.*, 1954, **7**, 609.
4 Murray, M., and Moosnick, F., *J. Lab. and Clin. Med.*, 1940–41, **26**, 801.

Hypothyroidism and ^{131}I Therapy

SIR,—There has been considerable criticism of the treatment of thyrotoxicosis by radioiodine (18 May, p. 427, and 27 July, p. 250) because of the development of myxoedema tending to occur over the years after the radioiodine has been administered. Some of this myxoedema must doubtless be considered inevitable owing to a gradual failure of the gland starting before the administration of radioiodine even though it has been preceded by the development of thyrotoxicosis.

In carrying out thyroid tests I have been struck by the fact that sometimes the function is partly physiological and partly pathological as indicated by the possibility of suppressing part of the function of the gland by the administration of triiodothyronine (T_3) before the test is carried out. The chief component of the radiation from ^{131}I is beta-radiation, and, judging from the fact that the use of external radiation is less efficient, it would appear that the effect of radioiodine in destroying the function of the gland is mostly by virtue of the beta component. The implication of this is that the effect of all the radiation will be very local and will not extend much, if at all, beyond the cells that have taken up the radioiodine. Assuming, therefore, that it is desirable to destroy pathological tissue and to leave physiological tissue intact it would seem reasonable to give the treatment dose of radioiodine after a course of T_3 so as to suppress any physiological thyroid tissue which is present. This procedure, for cases in which it is considered safe to use T_3 in the presence of thyrotoxicosis, would seem to offer the possibility of limiting hypothyroidism to those cases in which it may be unavoidable. Moreover, smaller repeated doses to treat the pathological cells should be a rational method of treatment.—I am, etc.,

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E.E.G. Signs of Death

SIR,—Your leading article (11 May, p. 318) and letters (1 June, p. 557; 22 June, p. 762; and 20 July, p. 185) prompt us to write of our experience of the E.E.G. in drug-induced coma. Since January 1968, among 48 poisoned patients studied electroencephalographically, 12 were deeply unconscious and unresponsive to any stimuli. Five of these 12 had flat electroencephalographic tracings for up to 11 hours. One patient died, and the remainder have made a complete clinical recovery.

As an example, one 63-year-old female who had taken a large dose of sodium barbitone was admitted deeply unconscious and unresponsive to all stimuli. Her E.E.G. showed complete electrical silence. Eight hours after her admission her E.E.G. for the first time showed slight activity but continued almost flat (Fig. 1). There was still no clinical response to stimuli. The E.E.G. activity gradually increased to generalized slow waves, as shown in Fig. 2. The E.E.G. one month later was within normal limits.

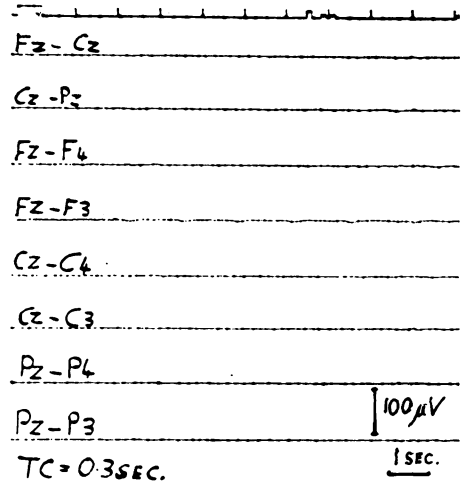

FIG. 1.—Eight hours after admission the E.E.G. continues almost flat.

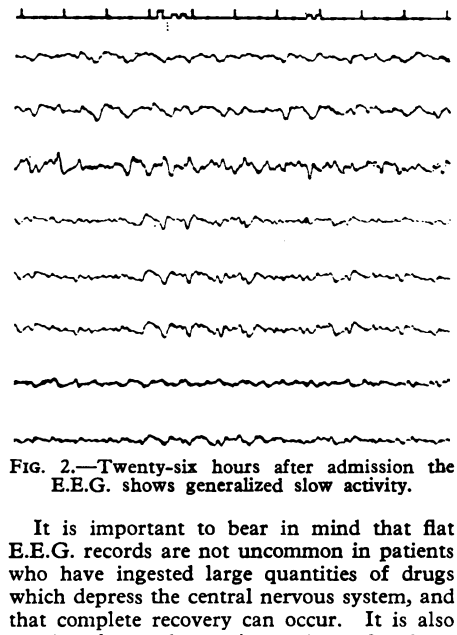

FIG. 2.—Twenty-six hours after admission the E.E.G. shows generalized slow activity.

It is important to bear in mind that flat E.E.G. records are not uncommon in patients who have ingested large quantities of drugs which depress the central nervous system, and that complete recovery can occur. It is also worthy of note that patients who at first have complete electrical silence may not show any clinical response to stimuli for as long as 15 hours, yet the E.E.G. may show progressive increase in electrical activity. Such a record can be of value prognostically.—We are, etc.,

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