Others are increasingly recognized now to be based on mild, transient depressive episodes of endogenous origin but equally stressful if only for their mysterious origin and still, sometimes, non-recognition. Your author's reference to non-specific symptoms is perhaps less valid in this context. The similarity is perhaps rooted in pathology.

The syndrome of mild depressive illness without significant or recognizable depression is frequently clear cut. Though no biochemical confirmation is yet available, by contrast with the lack of response with iron in "sideropenia," an objectively specific and subjectively clear response with antidepressant drugs is nowadays commonplace.

One may also wonder how much of the symptoms in controls and in those receiving drugs may be attributable to minor depressive states of subclinical "normal" intensity. Perhaps the exclusion of those with a history of depressive illness as such would be considered a logical step. To eliminate this variable more thoroughly seems impracticable, though sex and age differences, as your article mentions in a different connexion, are also relevant in depression.—I am, etc.,

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Metabolic Response in Ethnic Groups

SIR,—Dr. A. H. Rubenstein and his colleagues (22 March, p. 748) state that the difference in response of serum insulin, growth hormone, plasma-free fatty acids, triglycerides, and blood glucose to an oral glucose load, in carefully matched White, Indian, and African subjects "must be related to *genetic* and environmental differences among the three races" (my italics) while producing no evidence to support any genetic difference between the races. They compared seven African manual labourers with eight White clerks or traders who were 10 cm. taller and 13 kg. heavier than the Africans on average. Can these groups honestly be described as "carefully matched"?

The differences in metabolic response to a glucose load which they found can all be accounted for by the differences in diet between the groups. The Whites "enjoy a diet which is high in all nutrients" and must therefore include the refined sugar and products containing it which are found in the diet of all affluent societies, while the Africans' is "low in animal protein and fat and high in unrefined carbohydrates such as maize, bread, and sorghum." There is a wealth of good experimental evidence from all parts of the world summarized in two recent books[1 2] to show that refined sugar gives rise to unexplained but remarkable differences in serum values of glucose and fats compared with the same caloric intake of unrefined carbohydrates. In particular one study[3] shows an increased incidence of diabetes and coronary heart disease among Yemenite Jews who had been accustomed to a high intake of sucrose for many years compared with Yemenite Jews whose carbohydrate intake had been mainly of complex carbohydrates of approximately the same average amount. In addition obese non-diabetics eating an American type of diet were found to have greatly increased serum insulin levels,[4] and it is reasonable to suppose that the small differences in serum insulin levels found by Dr. Rubenstein and his colleagues can be accounted for by the absence of refined sugar from the diet of the African subjects.—I am, etc.,

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Ampicillin and Urticaria

SIR,—Dr. E. T. Knudsen's letter on skin reactions occurring in patients treated with ampicillin (29 March, p. 846) raises an issue of great importance. As a result of the continuous survey by Beecham Research Laboratories of adverse reactions to ampicillin reported in the literature, Dr. Knudsen reaches the conclusion that the majority of the erythematous rashes reported during ampicillin therapy "are apparently ampicillin-specific and do not indicate true penicillin hypersensitivity." Many clinicians seeing these rashes frequently would agree with this statement.

From our studies of hypersensitivity reactions to antibacterial drugs in patients with glandular fever,[1 2] my colleagues and I suggested that many hypersensitivity reactions to ampicillin were not true 6-aminopenicillanic acid hypersensitivity reactions.[3] This concept is a revolutionary one, since it has always previously been held that hypersensitivity is to the 6-APA "nucleus," and that since this "nucleus" is common to all penicillins there must be cross-sensitivity.[4] No one, to my knowledge, has tested this assumption by challenging patients hypersensitive to one penicillin with another penicillin—the spectre of sudden death from anaphylaxis has prevented such experimentation.

Ampicillin is one of the most prescribed drugs in the world and even accepting Dr. Knudsen's figure of 2·8% overall hypersensitivity (many reports have suggested the incident may be much higher[4–6]) it is apparent that many individuals are being labelled "ampicillin hypersensitive" and, in accordance with current practice, are being denied the use of all members of the most valuable group of antibiotics at present available. I suggest that the time has come to look upon the erythematous rashes occurring as a result of ampicillin treatment as specific to ampicillin, and that such hypersensitivity should not preclude the use of other members of the penicillin group of antibiotics.—I am, etc.,

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Advantages of General-Practitioner Hospitals

SIR,—I have had the privilege of working as a consultant at a general-practitioner hospital for over 23 years. The value of intimate practitioner and consultant co-operation, which would seem to exist under the N.H.S. only in such hospitals, is beyond assessment by those who have not experienced it, and the resulting benefit to the patient is enormous. The constant contact between the two branches of the profession is of mutual help. Both improve their medical knowledge, and certainly the consultant is a better doctor as the result of his intimate association with the working life and problems of a general practitioner.

Where the liaison exists a patient's minor symptoms are discussed with the consultant, and these apparently minor symptoms sometimes prove to be the precursors of severe illness. It is likely that many practitioners would hesitate to bother consultants with such apparent minor worries unless they work together in complete co-operation and unless they are constantly meeting each other at a professional level. The integration of practitioners into hospitals is probably the only way in which the hundreds of tragedies of missed diagnoses will be avoided.

The elimination of the cottage hospital seems to be the avowed intention of certain opinion both professional and lay. It can be argued with justification that a patient at such a hospital has the finest possible treatment. The consultant is rarely aided by registrars, so that every patient is seen and treated by him or her personally. Minor operations—hernias, appendicectomies, and varicose veins—are all dealt with by the consultant and his consultant anaesthetist. Such lists are seldom those of a consultant at a teaching hospital. Why therefore abolish these units, which serve a local demand with distinction?

It is true that such hospitals must have the backing of a large hospital to which the very seriously ill may be transferred, and where facilities for investigation are available at a more advanced level. Therefore every consultant appointed to a cottage hospital must be on the staff of a major unit to which the transfer of a patient can readily be facilitated.

Far from closing down the general-practitioner hospital their numbers should be increased. Their running costs are less than those of a major hospital, they are better adapted to run on a part-time nursing staff, and they are less dependent upon the limited number of resident doctors available in this country. Moreover, they offer the general practitioner access to a hospital and the continuity of supervision of their patients which

to the latter means much. These units could also be used for teaching purposes. At the general-practitioner hospital at which I attend students accompany me regularly. At the outpatients there they see more everyday cases than they would ever see at their teaching hospital, where referred cases tend to be of a highly specialized nature.—I am, etc.,

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Causes of Enuresis

SIR,—In your excellent leading article of the causes of enuresis (12 April, p. 63) you include among the things we "know" that do not cause the condition excessively deep sleep.

How do we know this? It is a statement that I have often heard or read, but I have never come across any proof of this opinion. On the other hand, 13 years ago I asked the mothers of 273 enuretic children the simple question, "Is he difficult to wake?" and 68% replied, "Yes." The same question was put to the mothers of 500 children attending outpatients for other reasons, and only 23% replied in the affirmative.[1]

Last year I saw 250 children at the Leicester School Clinic with congenital enuresis and 179 (72%) were said to sleep unusually heavily. Moreover, we have nine enuresis alarms in circulation, which cause a gratifying number of apparent cures in some very difficult cases. The failures are nearly all due to the alarm not waking the patient, although it wakes the parents and all the other non-enuretic members of the family. Lastly, I have this afternoon seen an enuretic girl of 10 who takes 15 mg. of dextroamphetamine a night—and still sleeps soundly.—I am, etc.,

Leicester. J. VERNON BRAITHWAITE.

REFERENCE

[1] Braithwaite, J. V., *Proceedings of the Royal Society of Medicine*, 1956, **49**, 33.

SIR,—Much as I hate writing letters, I feel I must comment on your leading article (12 April, p. 63) on this subject.

In 1953 I pointed out at a meeting of the urological section of the Royal Society of Medicine[1] that the combination of two factors was almost always associated with what I called then "simple" enuresis, and what is termed "primary" in your article. These two are deep sleep and a bladder which is accustomed to be emptied when it holds only an ounce or two (25–50 ml.) of urine. There simply is not enough capacity to go through the night, and our primitive habit of wetting our nappies (we all do it until we are "pot trained") is continued into late childhood or longer. Thus far "maturation" *is* a factor. The low social position of the parents acts, I suggest, because they in many cases could not be trusted to house-train a puppy.

Only the patient can cure himself, and this he can do if he can train his bladder to hold enough to go through the night. I usually aim at one ounce per year of age, and at the same time the bad habit of passing urine in the bed must be broken by *waking* the child *before* the bed it wet.

Anyone who wishes to verify these facts can do so by measuring how much an enuretic can hold before training—often less than a sherry glassful.—I am, etc.,

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REFERENCE

[1] Higham, A. R. C., *Proceedings of the Royal Society of Medicine*, 1953, **46**, 849.

Abortion Act in Practice

SIR,—As a general practitioner I have become increasingly concerned with the ethics of some members of the profession since the onset of the new abortion laws.

In one case I received a letter from a doctor about a patient of mine whom he had seen without my knowledge. I did not feel the case warranted abortion, knowing the full facts. These I explained to him in my letter, and expressed my opinion that an abortion was not indicated. Nevertheless, he obtained a second recommendation and carried out the abortion himself.

In another case I merely received a letter from a doctor saying he had seen my patient, referred her to a psychiatrist, and arranged termination. Apart from diagnosing this patient's pregnancy, I had no knowledge of any of the proceedings until I received the letter saying the patient had been aborted.

I have had several other patients treated like this. None of the doctors concerned are people I would normally consult and I would certainly not have referred any patient of mine to them. I always choose one of the several teaching hospitals locally. What can be done to stop this cashing in on our patients' predicaments, and raising our blood pressure with utter frustration and annoyance?—I am, etc.,

RICHARD W. PENNY.

London S.E.19.

Severe Self-poisoning

SIR,—I was interested to read Dr. G. R. Burston's account of the management of severe self-poisoning in Sunderland (15 March, p. 679). It is stated that 44% of the sample were discharged directly home to the care of their general practitioners. Does this imply that they did not receive psychiatric screening?

Self-poisoning carries not only an increased risk of further self-poisoning (as the paper shows), but further fatal episodes, regardless of the apparent seriousness of the initial attempt. For this reason the Hill report[1] recommends that all cases of self-poisoning should receive psychiatric attention. In the Bolton group of hospitals, with a case load last year of 260 attempted suicides, 92% of cases were screened. Certainly all cases of the severity described in the paper were dealt with by either inpatient or outpatient psychiatric services. It has thus been possible to achieve a virtually complete psychiatric service. This may be an advantage of having a psychiatric unit in a general hospital.—I am, etc.,

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REFERENCE

Scottish Health Services Council, *Hospital Treatment of Acute Poisoning*, 1968. Edinburgh, H.M.S.O.

Frusemide and Calcium Excretion

SIR,—Drs. J. A. Tambyah and M. K. L. Lim (22 March, p. 751) have shown that in the eight hours after oral frusemide there is an increase in urinary calcium excretion.

We have reported to the Renal Association[1] our preliminary studies on the effect of frusemide on calcium excretion. We have confirmed that both intravenous and oral frusemide causes a marked hypercalciuria in the four hours following administration. However, this is then followed by hypocalciuria so that the overall 24-hour calcium excretion may be little changed. It is probable that if Drs. Tambyah and Lim had studied their subjects for the next 16 hours a period of marked hypocalciuria would have been observed.

Before speculating on any prolonged effect from frusemide therapy it is important to observe the effect of frusemide on calcium excretion and absorption over the 24 hours, and also to consider whether changes persist during prolonged therapy.—We are, etc.,

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REFERENCE

[1] Renal Association, 1968, abstract to be published in *Nephron*, 1969.

Granules of Electrolytes for Infants

SIR,—Prompt oral treatment of infants who are vomiting or have diarrhoea can prevent or lessen the degree of dehydration and make emergency intravenous therapy unnecessary. In these conditions oral dextrose solution is contraindicated, as its use does not replace lost ions; an electrolyte solution is required. In reminding readers of this, you suggested in a leading article (13 January, 1968, p. 70) that it would be helpful to have a solution tablet containing the required replacement electrolytes, which could be used to make into feeds.

This idea was investigated here, but in the end it proved more practicable to prepare granules designed to be dissolved in sterilized distilled or boiled tap-water to produce a solution suitable for use as a "feed." The formula is based on one given by Talbot and his colleagues,[1] with the addition of dextrose and the minor modifications found necessary for the preparation of granules.