increased glucose utilization (lasting up to three months) occurred during re-feeding. If this interpretation is correct, the effect of carbohydrate deficiency on glucose-tolerance depends not only on the severity of the deficiency and the measures taken to correct it, but also on the phase of the under-feeding–re-feeding cycle at which a test is carried out. This explanation would account for the remarkable doubling in the value of K on re-testing in Case 8 in Herzberg *et al.*'s paper. In this subject a weight-gain of 5·3 kg. is recorded, whereas the weight-changes in the other 7 subjects are under 1 kg.

Two other observations in their paper are worthy of comment. The first is the large range in the values of K in their eight subjects. As normals they quote ten subjects (aged 16–65) from a paper by Marks and Marrack (1962) who gave a mean K of 1·72, range 1·15–2·32 (S.D. 0·41). The subjects with depression (aged 26–76) gave a mean of 2·4, range 1·0–4·5 (S.D. 1·27), and seven of the sixteen values of K were well above the upper limit found in the normal group. The authors, however, seem to regard these as normal findings.

The second observation, ignored by the authors, is the large difference in K on re-testing in three of their subjects, although Marks and Marrack (1962), whom they quote, commend the use of K in studies of glucose tolerance precisely because "it is reproducible in an individual subject". I have suggested that the high values of K on re-testing in Case 8 may be of nutritional origin. The large differences in the two other cases (11 and 12), however, are difficult to explain. In my data differences of this order seemed invariably to be associated with dietary factors; but smaller differences (up to 0·3 per cent. per minute) were associated with another phenomenon, seldom mentioned in glucose tolerance studies. This is the large scatter of blood-glucose values around an ideal regression line which is sometimes found in intravenous glucose tolerance curves. In my subjects with depression this was greater than could be accounted for by experimental error in 40 per cent. of curves (and compared with smoother curves was associated with a significantly greater decrease in K). Herzberg *et al.* presumably obtained the value of K by fitting the best curve by eye through five values of blood-glucose on semi-logarithmic paper. This procedure can give varying results if there is much scatter of individual blood-glucose values, and may possibly account for some of the inconstancy in their values of K on re-testing.

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A CLINICAL INVESTIGATION OF PHOBIAS

Dear Sir,

I enjoyed reading the article by R. P. Snaith (*Journal*, June, 1968, p. 673), and should like to make the following two comments:

1. In my experience agoraphobia often has its origins in an episode of hyperventilation:

   " 'Typically, these patients noted the sudden onset of subjectively inexplicable panic attacks, accompanied by hot and cold flushes, rapid breathing, palpitations, weakness, unsteadiness and a feeling of impending death' " (p. 674).

   " . . . True vertigo was rare but a feeling of unsteadiness of the legs, an illusion of walking on shifting ground, or a lightness of the head was the usual pattern" (p. 675).

2. Four patients with symptoms of agoraphobia were given diphosphopyridine nucleotide (1 g. in 1,000 c.c. 5 per cent. dextrose by intravenous infusion). In three there was a lessening of anxiety and phobic symptoms; in two this change was marked.

   Two of these four patients are brothers. Both brothers were given 1,000 c.c. 5 per cent. dextrose by intravenous infusion, subsequent to the drip containing the D.P.N. One brother, who showed only slight improvement after his gramme of D.P.N., felt no change after the 5 per cent. dextrose infusion, nor after a subsequent 1 gramme of D.P.N.

   The other brother responded markedly to 1 gramme of D.P.N., equally dramatically to 1,000 c.c. of 5 per cent. dextrose in water some months later, but with only a slight change for the better to the second dextrose in water infusion. He responded more favourably again to a subsequent gramme of D.P.N.

B-blocking agents reduce to some extent the somatic symptoms of anxiety (1, 2), and often to a marked degree those, e.g. tachycardia and palpitations, related to respiratory alkalosis.

Perhaps there is "a neuro-physiological basis" (p. 676) for anxiety and phobic symptoms!

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PSYCHODYNAMIC CHANGES IN UNTREATED NEUROTICS

Dear Sir,

In their paper (*Journal*, May, 1968, p. 525) describing varieties of psychodynamically suspect patients, Milan, Bacal, Heath and Balfour appear to accept the following three propositions:

1. At follow-up, symptom improvement is no greater in psychodynamically treated than in untreated patients.
2. Symptoms are a response to identifiable stress, which the patient cannot handle because of personality disturbances.
3. Psychodynamic therapy relieves personality disturbances, so that the patient can handle the identifiable stress in a new way, without developing symptoms.

At follow-up a similar proportion of treated and untreated patients will be subject to the identifiable stress (either to its continuance or recurrence).

At least one of the propositions must be incorrect.

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MANDRAX AND DICHLORALPHENAZONE

Dear Sir,

In the April, 1968, issue of the *Journal* (p. 465), there is an article by Ijaz Haider: "A comparative trial of Mandrax and dichloralphenazone". I should like to comment on certain aspects of this evaluation.

The study was designed so as to generate a number of preferences for one or other drug on as many pairs of nights. In other words, each subject was tested for three pairs of nights, one night of each pair being either dichloralphenazone or Mandrax. Forty-eight subjects were said to have been tested. Preferences, both subjective (patients) and objective (staff) were gathered.

The trouble is that in the author's Table I the paired preferences add up to the specified N in no single instance! As an example, for patient preferences, for the first pair of nights there is a total of only 41 preferences, for the second pair of nights there is a total of only 35 preferences, and for the third pair of nights there is a total of only 34 preferences. Since Dr. Haider did not specify anywhere in the text what happened to the remaining preferences (that one would have expected from a sample of 48 subjects), it is difficult to make any sense out of the Table. Did the missing preferences indicate that the unheard-from subjects had no preference, or did it mean perhaps that they were dropped from the study, or did it mean that the records were lost?

Even if we accept the total N of 41 for the first pair of nights, 35 for the second pair of nights, and 34 for the third pair of nights, as to the patients' preference, and then turn to Table III for the statistical analysis of difference, we find serious errors. Chi squares for the successive pairs of nights are not as stated in Table III but are rather ·61, ·71, and ·47, respectively. The significance in all these instances is $\cdot 30 < p < \cdot 50$, a result totally different from the author's Table III.

There may be other errors in this paper; I have not bothered to check the statistics in all of the tables. However, the extent of the ambiguity and statistical error in just this instance is enough to cast doubt on the remainder of the study.

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TREATMENT OF PREMATURE EJACULATION

Dear Sir,

I refer to an article "The use of methohexitone sodium in the systematic desensitization of premature ejaculation" by Tom Kraft and Ihsan Al-Issa (*Journal*, March, 1968, p. 351). It seems to suggest that this drug has some special advantage over thiopentone sodium in premature ejaculation. It is certainly safer than thiopentone and recovery time is quicker. On the other hand, we find that during administration of 1 per cent. solution of methohexitone pain at the site along the vein is intense, and is enough to cause anxiety and distraction in the majority of cases. Our hospital anaesthetist (1) has tried using a more diluted form of methohexitone than recommended (i.e. 0·5 per cent.) but reports that pain is still experienced in no less than 35 per cent. of cases.

Premature ejaculation differs only in degree from the majority of impotence cases, if we exclude the few in which the causes are deep rooted in the process of psychosexual development (although Kinsey *et al.* (2) do not agree). Impotence and premature ejaculation are quite common in this part of the country; in most cases the causes lie in the personality, lack of sex education and rarely in the