| | |
|---|---|
| Psychiatrically well, having been depressed when originally seen .. | 8 |
| Psychiatrically well before and after, although at risk .. .. .. | 1 |

In 9 of these patients, therefore, affective disturbance was still present. It does not follow from these findings, of course, that the terminations were not justified; on the contrary, the initial careful psychiatric interviews had revealed that they were. What is indicated, in a significantly large proportion of cases, is that the operation, while resolving a traumatic predicament, is as likely as not to leave the woman still unwell after six weeks.

In eliciting the patient's views and feelings, care was taken to confirm her in the decision which she and her doctors had made to terminate the pregnancy; her opinion was invited, however, on that decision as she viewed the *fait accompli* in retrospect. Three of the nine unwell women and two of the nine well women expressed unequivocal moral misgivings. This suggests that doctors are not wasting their time when they sound out the moral attitudes of candidates for abortion, for a timely ventilation of scruples may help to avoid self-reproach in later life. Unfortunately, many women improvise their philosophy of abortion only when already in a state of confusion and distress.

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## SUICIDE IN BRIGHTON

DEAR SIR,

Drs. Jacobson and Jacobson state that the drop in suicide in Brighton could not be due to the Samaritans as there was no organization operating in that area during 1964–68 (*Journal*, October 1972, **121**, p. 376). This is not quite true.

There was a Brighton branch with a Centre at St. Peter's Church, which disbanded during 1965. In the same year a service opened up in Eastbourne, 22 miles and one local telephone call away. The number of the Eastbourne branch was published in the Brighton Telephone Directory from that year, and Brighton clients built up steadily from only 2 in 1965 to 61 in 1968, after which an independent service developed in Hove. Befriending by Brighton volunteers of clients who rang Eastbourne was available throughout this period.

Furthermore, through the period of suicide decline in Brighton the original City of London branch of the Samaritans became increasingly well known nationally as a result of mass media publicity, and it has, since its earliest days, received calls from all over the country, including Brighton to which the links by road and rail have always been strong.

The possibility cannot be ruled out, finally, that a national organization radiating an 'ethos' of suicide prevention may have an effect in a town which does not actually have a branch of its own.

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## SEXUAL ASPHYXIA

DEAR SIR,

In his paper (*Journal*, October 1972, **121**, 437–8) John Edmondson stresses the feelings of sexual guilt which result in such vicious attacks upon the self. Symptoms, however, usually and perhaps always contain a positive element which it is important not to overlook. In this case it is I think the healthy, albeit distorted, attempt of this boy to achieve the oblivion of an abandoned orgastic experience.

PETER LOMAS.

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## LACTATE-INDUCED ANXIETY: HYPOTHESIS AND EXPERIMENTAL MODEL

DEAR SIR,

In his letter (*Journal*, September 1972, **121**, 338), Friedhoff writes: 'Grosz and Farmer (*Journal*, April 1972, **120**, 415–8) have reported the results of an interesting study showing that anxiety symptoms can be precipitated by the production of metabolic alkalosis. Unfortunately, they present their results as a refutation of the previous conclusions of Pitts and McClure (*New Eng. J. Med.*, 1967, **27**, 1329–36) that an anxiety state can be produced by elevating blood lactate concentrations . . .. These newer findings should be viewed simply as an extension and refinement of the hypothesis of Pitts and McClure.'

The reason for presenting our findings as a refutation of Pitts and McClure's conclusions is first, that the kind of study we did should have been carried out by Pitts and McClure as a necessary control experiment, and, secondly, that we do not believe that the results of our study impart any substantially new knowledge to the understanding of mechanisms underlying anxiety symptoms in general, or of anxiety neurosis in particular. In certain susceptible, anxiety-prone people almost any major electrolyte disturbance, or disturbance of acid-base balance of body fluids, whether respiratory or metabolic, seems

liable to provoke symptoms of anxiety. Common examples that come readily to mind are respiratory alkalosis associated with hyperventilation, respiratory acidosis associated with lung disease, metabolic acidosis associated with uraemia, hyper- or hypocalcaemia associated with parathyroid disorders, and so on. The common denominator seems to be some deviation from the norm in the patient's internal bio-physical environment. This leads us to believe that *anxiety-prone subjects, and perhaps anxiety neurotics, may essentially suffer from an excessive sensitivity or intolerance to disturbances in their internal bio-physical homeostasis.* They may react with symptoms of anxiety and with compensatory homeostatic efforts to changes that may remain unnoticed or ignored by others.

Viewed in this light (*Arch. gen. Psychiat.*, November 1969, **21**, 611–9), Pitts and McClure's experiment does little more than illustrate what happens when anxiety-prone subjects are suddenly exposed to major perturbation of homeostasis—in this case to major electrolyte and acid-base balance disturbances. The facts that anxiety symptoms can be induced with lactate infusions even without bringing about significant blood lactate elevations, and that similar symptoms can be brought about with bicarbonate infusions admittedly refute Pitts and McClure's hypothesis. However, these factors offer no ground for any 'extension or refinement of the hypothesis of Pitts and McClure', as Friedhoff suggests, nor for that matter do they provide support for any other hypothesized specific mechanism.

More recently, some experimenters have used sodium lactate infusions as an experimental model to test the effectiveness of anti-anxiety drugs. The rationale for this seems to be based on the erroneous belief that lactate-induced anxiety provides a sound laboratory model for clinical anxiety. Since lactate infusions induce anxiety symptoms that are associated with electrolyte, pH and body fluid disturbances the like of which are found in nature only in physically quite ill people, and ordinarily not at all in anxiety neurotics, the validity of using lactate-induced anxiety as an experimental model from which to extrapolate findings to clinical populations is more than doubtful. (In fact, as we will report elsewhere, some of the lactate-induced effects are not only unusual but also hard to explain.) *In fine*, Pitts and McClure's laboratory model can hardly generate clinically valid conclusions.

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## THIAMINE DEFICIENCY IN THE AETIOLOGY OF THE HALLUCINATORY STATE COMPLICATING ALCOHOLISM

DEAR SIR,

The paper by Blackstock *et al.* (*Journal*, October 1972, **121**, 357–64) on the role of thiamine deficiency in the aetiology of hallucinatory states complicating chronic alcoholism provides a welcome addition to the thiamine deficiency/alcohol withdrawal debate. Their findings are not so dissimilar to those from my own study (1) as one might suppose from the way they have presented their data.

Our results agree in concluding that hallucinatory states may be induced by alcohol withdrawal alone, in the absence of any clinical and biochemical evidence of thiamine deficiency (Blackstock *et al.*, 6/15 cases, Morgan 4/8 cases with hallucinations).

The precise role, if any, of thiamine deficiency remains problematic. My own findings, based on clinical assessment and pyruvate studies on a very small series, suggest that hallucinatory states which develop while drinking is continuing (and which I have termed 'subacute' because they may extend over several weeks) might be closely associated with thiamine deficiency. Blackstock *et al.*, on the basis of their pyruvate findings, clearly do not subscribe to this view. However, if the criteria of thiamine deficiency in their study are widened to include either pyruvate or transketolase abnormalities, or both, then their findings show a trend in favour of my hypothesis. I suggest that this has still not been put out of court and should remain a matter for further investigation.

There are certain very real problems besetting this particular field of study. Not only are the subjects unreliable as a source of information, but there is also a pressing need for more precise definition of the mental phenomena involved, as Blackstock *et al.* rightly point out. Further, the biochemical tests of thiamine deficiency need further development. The transketolase test, itself superior to the pyruvate tolerance test, is still an indirect method of thiamine assay. Of course, it does not follow that direct assay of thiamine is necessarily any better; for example, isolated blood thiamine estimations fluctuate very widely with the thiamine intake over the previous few days, and are of doubtful value in assessing states of true clinical deficiency of thiamine. Some kind of thiamine tolerance test may eventually prove to be the most sensitive index. Preliminary data using such a technique, with spectrofluorometric assay of thiamine, have been published by Dewhurst and myself (2), but it is not yet clear whether the method can distinguish thiamine-deficient from normal