

## REFERENCES

1. MALLESON, A. G. (1973). 'Suicide prevention: a myth or a mandate?' *British Journal of Psychiatry*, **122**, 238-9.
2. BAGLEY, C. (1973). 'Suicide prevention: a myth or a mandate?' *British Journal of Psychiatry*, **123**, 130.
3. THE GAS COUNCIL (1973). 'Deaths due to accidental gas poisoning in England, Scotland and Wales.' Personal communication.
4. NIELSEN, and VIDEBECH, T. (1973). *Suicide Frequency Before and After Introduction of Community Psychiatry in a Danish Island*.
5. HICKS, R. C., and CHOWDHURY, N. 'Evaluation of an after-care service for patients repeatedly attempting suicide.' In preparation.

FATAL SELF-INJURIOUS BEHAVIOUR  
—A PRELIMINARY COMMUNICATION

DEAR SIR,

A recent publication by Stevens (1973) has emphasized the high risk of suicide in community-centred Day-Hospital/Industrial Rehabilitation Units for psychotic patients. On the other hand, to date, a review of relevant literature concerning the use of token-economy programmes with chronic psychiatric patients has failed to provide evidence of any report concerning the occurrence of suicide during or after programme participation, though there is evidence to suggest that during programme participation some patients are likely to threaten and show self-injurious behaviour (Schaefer and Martin, 1969). The purpose of this communication is to highlight the possible risk of fatal self-injurious behaviour in programmes dealing with chronic psychiatric patients and conducted in controlled prosthetic social-learning environments.

In rehabilitation studies using token reinforcement conducted by this correspondent, four patients out of a total of 56 made suicidal attempts which proved fatal. These four patients (three males and one female), all suffered from psychotic illnesses of long duration, and they committed suicide under the following circumstances: (a) one patient—over three months after successful programme completion, i.e. during the follow-up period, while resident in a half-way hostel and in sheltered employment; (b) one patient—over three months after successful programme completion, i.e. during the follow-up period and while in open employment for over two months, though still resident in hospital; (c) one patient—while out of hospital on a town visit; and (d) one patient—while out of hospital on a home visit accompanied by relatives. No environmental cause could be demonstrated in these suicides.

J. FERNANDÉZ.

30 Mountpleasant Square,  
Ranelagh, Dublin, 6, Ireland.

## REFERENCES

- SCHAEFER, H. H., and MARTIN, P. L. (1969). *Behavioural Therapy*. New York: McGraw-Hill.
- STEVENS, B. C. (1973). 'Evaluation of rehabilitation for psychotic patients in the community.' *Acta Psychiatrica Scandinavica*, **49**, 169-80.

POSSIBLE SIDE EFFECTS OF  
FLUPHENTHIXOL DECANOATE

DEAR SIR,

Many patients have benefited considerably from the regular medication provided by the depot neuroleptic drugs, and many of us have found a great use for them—particularly fluphenazine enanthate (Moditen), fluphenazine decanoate (Modecate) and more recently fluphenthixol decanoate (Depixol).

Side effects (apart from extrapyramidal effects) seem to have been relatively rare, but recently two patients of mine have shown quite remarkable weight increases whilst on Depixol. In one case, a female patient of 38, weight increase was so dramatic that she became facially unrecognisable and her ordinary clothes could not be worn. She put up with the weight increase for several months, but then refused further injections. Her weight has reduced and her figure and facial outline have returned to normal since injections were stopped and she reverted to trifluoperazine (Stelazine) by mouth. The other patient is a young man, aged 21, who after a severe psychotic episode and six months in hospital has done well. His psychosis is not now evident, he is working and is apparently doing well, but his weight increase (not apparently embarrassing to him!) is enormous.

We have found Depixol a useful drug, and I would be glad to know if any other clinicians have noted any similar effects. At least one observer (Gottfries) has noted weight reduction in a number of cases. I am not, at present able to offer any explanation how or why the weight increase occurs.

DAVID RICE.

Hellingly Hospital,  
Hailsham,  
Sussex BN27 4ER.

THE USE OF DISULFIRAM  
IMPLANTATION IN ALCOHOLISM

DEAR SIR,

A representative of the American company that manufactures disulfiram has informed me that the drug is absorbed into the blood stream via the lacteals of the gut and that absorption does not occur by other routes. Documentation for this assertion, however, was lacking, and I was interested in the article by Malcolm and Madden (*Journal*, July 1973,

123, 41-45) referring to their experience with disulfiram implantation in 70 alcoholics.

Of those alcoholics who drank after the implantation, only two reported a disulfiram-like reaction and returned to abstinence. The authors conceded that this might have been psychogenic, since the two patients were familiar with disulfiram reactions from previous experience with oral disulfiram.

The most compelling evidence, however, that disulfiram is absorbed in negligible amounts after implantation came from the observation of one patient whose wound became infected, sloughing four of the ten 100 mg. tablets implanted six weeks previously. About one-third of each tablet had dissolved. In short, about one-third of a gram of disulfiram had been absorbed over a six-week period. This would have resulted in infinitesimal blood levels (if indeed any was absorbed) and it is highly unlikely that alcohol ingestion would have produced a genuine disulfiram effect.

Since this point was not made in the article, I thought it should be commented upon.

DONALD W. GOODWIN.

Department of Psychiatry,  
Washington University School of Medicine,  
4940 Audubon Avenue,  
St. Louis, Missouri 63110.

#### INCONSISTENCY, LOOSE CONSTRUING AND SCHIZOPHRENIC THOUGHT DISORDER

DEAR SIR,

The Hayes and Phillips paper (*Journal*, August 1973, 123, 209-17) runs a curious course. It begins by proposing that in the grids of thought-disordered subjects lowering of Intensity (the level of correlation between constructs) means that minor fluctuations over time markedly lowers Consistency (the stability of the pattern of correlations from first to second grid). Thereby lower Intensity *causes* lower Consistency. Then follows a laboured experiment to show that it is lower Consistency that causes lower Intensity. All of which makes one fear for Messrs. Haynes and Phillips' Consistency, if not their Intensity. It were better to leave alone simple-minded notions of 'cause-effect' and regard Intensity and Consistency as interactive aspects of the total construct system.

Once out of the second growth underbrush of the experiment, we are invited to view my definition of loose construing as an illegitimate offspring of Kelly's original proposal. And well it may be but the question is not illuminated by their attempt to treat Kelly's view of 'loosening' as if it were an *ad hoc* bit of stray terminology rather than a concept entirely to be defined within the framework of personal

construct theory, from which it derives. In terms of the theory the argument runs as follows. If 'loosened construing' leads to 'varying predictions' (Kelly); if predictions are essentially specified by the *links between constructs* (of the type if A then B); then 'weakening of the relationships between constructs' (Bannister) is a fair, elaborative re-definition of loosening. (If Bloggs sees *Public School* as closely related to *honest*, then he firmly expects the old Harrovian to pay him back his £5; but if, for him, the relationship between these constructs weakens, then his prediction that he will get his £5 back begins to vary—it drifts between a hopeful guess and a doubtful hope.)

As their personal contribution to our understanding of thought disorder, Haynes and Phillips ask us to view it as 'inconsistency'—offering us thereby an *ad hoc*, non-explanatory, loosely defined, lay concept, about as useful as, say, 'disorganization' or 'vagueness' or 'confusion' or any other of a dozen arbitrary, untheoretical bits of verbiage that we might cling to when thought fails.

D. BANNISTER.

Bexley Hospital.  
Old Bexley Lane,  
Bexley, Kent DA5 2BW.

DEAR SIR,

Dr. Bannister's letter (by no means his first critical comment on our paper—see *Brit. J. soc. clin. Psychol.*, 1972, 11, 412-14, and in press), appears to us to consist only of abuse, and to advance no serious scientific arguments concerning our experiment. There would thus seem to be no need for more reply than this, were it not that in two places he (again, not for the first time—see the same references) gives an incorrect account of what we wrote.

Firstly, he states that our paper 'begins by proposing that . . . Intensity *causes* lower Consistency'. This is not so: in fact precisely the reverse is true. Our hypothesis (given in the second paragraph of our paper) is that inconsistency in thought-disordered schizophrenics lowers their Intensity scores. Two paragraphs later we mention that because Bannister's Consistency scores are contaminated by Intensity, 'it is also possible that low Intensity in thought-disordered schizophrenics was causing low Consistency scores, instead of the other way round'. However, this is not, as Dr. Bannister suggests, our hypothesis, but simply an alternative possibility that must be guarded against. Thus the inconsistency which he imputes to us is not in our paper, but is entirely of his own making.

Secondly, he writes: 'As their personal contribution to our understanding of thought disorder, Haynes and Phillips ask us to view it as "inconsistency" . . .'