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.

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## 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 an 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 arbitary, untheoretical bits of verbiage that we might cling to when thought fails.

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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" . . ..'

This is not so: the hypothesis of inconsistency was advanced by us to explain, not schizophrenic thought disorder, but simply Dr. Bannister's experimental results—a rather different matter.

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## EYE-CONTACT AND DEPRESSION

DEAR SIR,

I was interested to read Derek Rutter's criticism of our study of Eye Contact in Depression in his review article 'Visual Interaction in Psychiatric Patients' (*Journal*, August 1973, 193–202).

I agree with him, there are major problems in controlling for interviewer behaviour and interview content without stylising the interview to such a degree to render its content artificial and useless. We were aware of these snags and indicated that we had found significant trends in our results. I agree that a more detailed breakdown of looking while listening and speaking are desirable. Another point that he raised was the effect of admission to a mental hospital on Eye Contact levels. In fact our depressed patients had been admitted to the psychiatric ward of a general hospital and were interviewed during the first week of their admission. Many were first admissions for psychiatric illness. Thus we avoided the stigma of admission to a large mental hospital, a point frequently mentioned with relief by the patients.

We also found wide variation in results between patient and control group, with some overlap in results. I would not attribute this so much to 'differences in aetiology and symptomatology' but to personality differences well known to influence levels of eye contact, such as dominance-dependency, introversion-extraversion, as well as sex differences. In our studies we encountered two female patients with total gaze avoidance. I attributed this to hostile defiant behaviour in hysterical personalities.

My overall impression of depressed psychiatric patients is that there is a marked and obvious reduction in eye-contact when intimate and painful topics are being discussed, that there is a general slowness of responsiveness in the eye-contact patterning compared with controls, but that this correlates with their psychomotor retardation.

We are also looking at eye-contact in the free discussion situation in order to avoid a discussion of symptoms. We are taking videotape recordings of depressed patients and their spouses in discussion and comparing eye-contact levels with those that the patient makes when talking to a normal stranger of the opposite sex. It seems that the anxious spouses are looking more than their depressed partners. The depressed patients become considerably more reactive and socially aware with the stranger and increase eye-contact.

In response to Rutter and Stephenson's results which indicate a similar level of reduced eye-contact in both schizophrenic and depressive groups I would suggest that there may be a minimum optimum level of eye-contact to which the emotionally disturbed individual retreats and that this protects him from a troublesome degree of social interaction and yet provides him with enough information about his interactant.

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