hallucinations, etc.). We are currently preparing an account which specifically addresses these problems of communication. We suggest that recent experiments on early childhood autism (implicating the lack of a 'theory of mind') may be relevant to those positive symptoms of schizophrenia that concern communication. Briefly, analogous to the problems of monitoring their intentions, the patients may also have problems in monitoring the intentions of others.

In addition to concisely pinpointing our weakness, Timney (*Journal*, February 1989, **154,** 268) implies that the symptoms we can explain are among the less common. However, in a survey of 242 patients with a first episode of schizophrenia carried out by our colleagues Eve Johnstone and Fiona Macmillan, delusions of control (34%) and of thought insertion (27%) were almost as frequent as verbal hallucinations to the subject (48%) and third person hallucinations (32%). We would agree with Ring (*Journal*, February 1989, **154,** 268) that depersonalisation and derealisation should be fundamental experiences in schizophrenia which increase with the increasing severity of positive symptoms. In Johnstone & Macmillan's survey they are indeed no more common than the classic positive symptoms (30% and 40% respectively). Maybe these experiences pale into insignificance beside the more extremely positive symptoms and are simply not reported.

Adams (*Journal*, March 1989, **154,** 416–418) describes an elegant hierarchy of monitoring symptoms derived from Hofstadter. She does not, however, indicate how this more complex account might be experimentally tested. She is wrong to suggest that in Frith (1987) questioned the validity of Crow's distinction between type I and type II schizophrenia. Indeed, it was stated that "negative symptoms represent a primary disease process rather than a secondary coping strategy".

In contrast, Klemperer (*Journal*, March 1989, **154,** 415–416) considers that our account is unnecessarily complicated. She suggests that we are wrong in believing that hallucinations are actions rather than percepts. It is by now well established that perception is not a passive process of stimulus reception, but an active one requiring, among other things, the generation and testing of hypotheses. Our account does not deny that hallucinations might be percepts. In fact, we are suggesting that hallucinations occur when the patient misperceives his own actions. Such misperceptions can also give rise to the false beliefs that are the basis of delusions. Klemperer suggests that a much simpler explanation of these phenomena is that they occur because of biochemical and structural abnormalities. We feel that this explanation has little predictive value and, furthermore, represents a form of dualism that is most unsatisfactory. It is implied there are independent physical and mental systems that communicate with one another. As a result, an abnormal signal from the physical system causes an abnormal experience in the mental system. We believe that the mental and physical systems are not independent entities, but different descriptions of the same system. Thus there are physical processes in the brain that exactly correspond with mental processes in the mind. Our approach to the neuropsychology of schizophrenia is an attempt to find ways of matching up these two descriptions.

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**Internal monitor defect in schizophrenia**

SIR: Frith & Done (*Journal,* October 1988, **153,** 437–443) put forward the theory of 'internal monitor defect' to explain the symptoms of auditory hallucination, delusion of control, and thought insertion in schizophrenia, and rejected the 'defective filter' theory. They postulated that these symptoms arose from the failure of the internal monitor system in registering the self-initiated actions (subvocal speech/act/thoughts) and hence labelling them as originating from an external agent. While I find this quite convincing, I have to point out that such a theory is no superior to the 'defective filter' theory is explaining other positive symptoms in schizophrenia, such as delusional perception, formal thought disorder, and delusion of reference. For delusions of reference at least, I find that the defective filtering out of insignificant and irrelevant external stimuli is a better explanation than the faulty labelling of "switch elicited by irrelevant stimulus". Symptoms like delusional perception and loss of reality testing indicate that there are defective thought processes, involving not only the labelling of ownership, but the actual logical deduction and interpretation of perception and thoughts.

Furthermore, I do not agree with the authors in saying that the monitor system is itself intact in schizophrenia. The classic symptoms of ambitendency (which Bleuler considered as a manifestation of the ambivalence of will), negativism, automatic obedience, and forced grasping seen in catatonic schizophrenia would be explicable only by a defective monitor failing to carry out its usual function of regulating the stimulus intention and the willed intention and deciding on which one to follow first.

Schizophrenics comprise a very heterogeneous group, and patients present with very different patterns of symptoms. There are probably more than several neuropsychological defects that give rise to different symptoms, and it is no wonder that no one theory can explain them all. All may be correct. Drs Frith and Done are on the right track in linking the symptoms of schizophrenia with neuropsychological theories and findings of malfunction in specific brain systems. One day it will lead us somewhere.

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**Naltrexone and Clonidine in Heroin Withdrawal Treatment**

SIR: We noted with interest Brewer *et al*'s study of naltrexone and clonidine in the treatment of opioid withdrawal (*Journal*, September 1988, **153**, 340–343). The search for a rapid and effective treatment for opioid withdrawal has been in progress for many years (Kolb & Himmelsbach, 1938). A treatment which promises to reduce the length of detoxification to less than three days with minimal drop-out will clearly appeal to many clinicians working in this field. We suggest, however, that the claims made by the authors are overstated and not supported by the results of the present study.

The authors state that the treatment was of "high acceptability" to patients. No evidence is put forward in support of this view such as patient's reports of acceptability or even an assessment of subjective symptoms or objective signs of opiate withdrawal. Furthermore, the treatment described was not compared with any other more commonly available treatment such as methadone or clonidine, leaving the authors' claims of effectiveness open to question.

Opiate withdrawal is now recognised to be subject to the influence of psychological factors including expectancy (Phillips *et al*, 1986). Thus, it is essential to conduct studies in double-blind design (Drummond *et al*, 1989), otherwise highly misleading results may be obtained.

The authors describe their treatment as the "naltrexone-clonidine technique". Closer examination of the method, however, reveals that in addition, patients received diazepam, nitrazepam, flurazepam, and hyoscine. This latter treatment was prescribed for "troublesome" abdominal cramping and nausea which were clearly not relieved by the naltrexone-clonidine combination. Furthermore, the dose of diazepam prescribed (up to 180 mg per day) was considerably higher than in studies of benzodiazepines used alone in the treatment of heroin withdrawal. Indeed, some subjects "experienced significant discomfort". This suggests that the basic naltrexone-clonidine combination was ineffective in controlling opioid withdrawal. The question arises as to whether any one of the drugs used in this combination regime would have been effective if taken alone in a sufficient dose. In a recent double-blind trial we found that cholordiazepoxide (250 mg daily) was as effective in controlling subjective withdrawal symptoms as a conventional methadone detoxification regimen (Drummond *et al*, 1989). The authors postulate that the mechanism of action of naltrexone in opioid withdrawal is that it "rapidly normalises the number and sensitivity of opiate receptors and reversed opioid induced central noradrenergic activity". While this tempting speculation adds a sense of scientific validity to the treatment, it is not supported either by evidence in this study or in the study cited in support of it (Kleber *et al*, 1987).

The authors suggest that the results of this study have "major implications" for National Health Service (NHS) treatment programmes, and question the need for specialist detoxification units and indeed specialised training in psychiatry or the addictions. To suggest that a highly selected group of private patients with major financial incentives for treatment is comparable to attenders at an NHS drug clinic or general practice is erroneous. Second, home withdrawal "with the help of telephoned instructions, a visiting nurse or an electronic sphygmomanometer" in our view hardly constitutes comprehensive treatment, represents a narrow view of the problem of heroin addiction, and does not amount to good value for money.

In a review of the early history of detoxification treatments, Klob & Himmelsbach (1938) observed "new treatments said to be specific [for heroin withdrawal] are advanced from time to time and then discarded as useless or even harmful". Champions of the naltrexone-clonidine technique would be well advised to subject this treatment to proper scientific scrutiny before making such assertions about its effectiveness. The methodology for such an investigation has been in existence for nearly half a century.

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