such incidents. It is current practice to treat those whose behaviour is dangerous in special settings, although the threshold for the use of such facilities is much higher than it used to be. HIV carrier status may be a critical factor in deciding whether a secure treatment environment is appropriate. Just as threats of violence are insufficient to warrant secure provision, so is supposed HIV carrier status. Assumption of innocence is a fundamental principle of English Law, and therefore when measures that involve significant infringement of liberty are to be taken, there must be proof of dangerousness. A positive HIV test combined with disturbed behaviour would constitute such proof. A general lowering of the threshold for secure provision would, in my opinion, be a greater evil than imposing HIV testing on disturbed patients. There are other benefits to be had from testing of disturbed patients, but as, individually, they would probably not warrant institution of testing, I shall not enumerate them here.

Dr Dunn asserts that HIV infection is relatively uncommon in this country. How does he know? Where are the valid epidemiological studies? What is certain is that the principal vectors, promiscuous homosexuals and intravenous drug abusers, are highly mobile groups, and even in our idyllic surroundings within the Dartmoor National Park we have seen several. The 'gay plague' hysteria has produced a quite violent backlash, and I fear this has gone too far. While it is quite right to disabuse the public of the notion that HIV can be contracted by merely being in proximity with a positive individual, it is wrong to understate the risk of infection by means other than sexual intercourse and transfusion, and this, unfortunately, is currently the case. It is only now that the problems of behavioural disturbance in HIV patients are being addressed – as a measure of the paucity of literature on the subject, I have received some 30 reprint requests from 12 different countries concerning two *letters* on the subject!

I hope that Dr Dunn's letter will go some way to initiating a debate on the subject out of which a professional consensus will emerge. In the last anaysis, however, it is the physician who determines whether a disturbed patient may be admitted, and the nurse who bears the brunt of the behaviour, and is therefore at risk. It is to the medical profession that the Courts turn for advice on ethical matters when there is a lack of precedent, and clearly psychiatrists will have a voice in determining the circumstances under which HIV testing can be undertaken in mentally disordered patients. Our profession has always accepted a responsibility which is more general than that to the patient alone, and it ill becomes us to retreat from this general reasonability to the safe isolationist position that Dr Dunn suggests. I would deny ignorance of the moral dimension of this problem; perhaps, rather, I am more aware of the complexities than Dr Dunn would appear to be.

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Molecular Genetics and Human Disease

SIR: Baron & Rainer's review (*Journal*, June 1988, **152,** 741–753) side-steps, once again, the most important aspect of recent and potential advances in molecular genetics. It seems increasingly likely that genetic markers will be identified which indicate increased vulnerability to subgroups of the functional psychiatric disorders. Despite the undisputed importance of environmental factors in the development of these illnesses, it is implicit in the writing of psychiatric geneticists that these advances could lead to prenatal diagnosis with selective termination of foetuses. This prospect has been explicitly advocated on at least one occasion (Wallace, 1986).

This issue is separate from the tragedy of individual women who feel unable to raise a severely ill child and therefore request prenatal diagnosis. The careful cost-benefit analyses of amniocentesis programmes (Chapple *et al,* 1987) and evangelistic approaches to these new methods of prevention (Milunsky, 1986) indicate, in my opinion, a hegemonic view within the profession that prenatal diagnosis should be encouraged for improvement of the public health and, indeed, the genetic pool. In the United States, the grotesque compensation claims against obstetricians for wrongfully permitting the birth of the plaintiffs (Shaw, 1986) emphasise the pressures in Western society to avoid the existence of handicapped or genetically abnormal people.

If predictions of advances in molecular biology are correct, the widespread acceptance that it is better for imperfect people never to have been born will provide an opportunity to reduce the frequency of disturbances of mood and behaviour by eugenic means. I would like to stimulate discussion of this difficult topic before we are overtaken by technological breakthroughs, as was the case with Huntingdon's disease.

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Psychophysiology and Expressed Emotion

SIR: I was most interested to read the study by Tarrier *et al* (*Journal*, May 1988, **152,** 618–624) on expressed emotion and psychophysiological responses, but am uncertain about the interpretation. Skin conductance can vary greatly with the clinical picture (Gruzelier, 1976) and also is altered by medication (Yannitsi *et al,* 1987). Neither of these factors were controlled for. In addition, Tarrier has already demonstrated an interaction between life events, the presence of a high expressed emotion relative, and skin conductance (Tarrier *et al,* 1979), but does not examine the interaction in this paper. Furthermore, altered skin conductance is associated with a poorer prognosis (Frith *et al,* 1979), as is a long past psychiatric history. However, neither the duration of past illness nor the number of past admissions was examined.

Overall it would be counterintuitive to expect that high expressed emotion does not have an impact on arousal. This paper certainly supports such a link, although the number of uncontrolled factors in the study means that this is not the only interpretation possible.

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SIR: The point Dr Cramer makes is reasonable; however, it does need to be put in context. We employed a within-subject design in which patients were tested with their relative being absent and then present. Hence, in the majority of our analyses patient factors were held constant and the presence of the relative was varied. Between-subject factors, which Dr Cramer cites as being important, are therefore greatly reduced in influence.

Dr Cramer cites a study by Yannitsi *et al* (1987) to demonstrate the importance of medication on electrodermal measures. This study compared patients while they were drug-free and while they received medication, and hence is not relevant here. Our patients were all receiving the optimal dosage of neuroleptics prescribed by the clinical team responsible for their care. This was clearly stated in the paper.

We did not record life events occurring prior to admission. However, data given by Leff & Vaughn (1980) would lead us to predict that patients with low-EE relatives would be more likely to experience a life event prior to relapse than those with high-EE relatives. Life events, therefore, might be expected to enhance the arousal levels of patients with low-EE relatives rather than explain the results we found.

The possibility that electrodermal measures are indicators of prognosis or vulnerability markers is a much more intriguing question. We have examined this problem in a longitudinal case study (Tarrier & Barrowclough, 1987), and we are presently looking at electrodermal vulnerability and episode markers in relation to our two-year follow-up data.

Although we are in full agreement with Dr Cramer that the factors he cites can be important influences on electrodermal measures, they are unlikely to explain the results that we presented.

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