DEAR SIR,

We offered our analyses in a context of exploration and discovery, and we are pleased when others give our methods and results close scrutiny. We agree with Janes and Hasselbrock that it may be useful to consider the probilities of hits for high-risk (h-r) and control children separately, though it is not always possible to do so. We disagree with some of their arithmetic (e.g. 9/30 ≠ ·33; the probability of 3 hits for controls by their method is ·0017, not ·13).

To obtain their estimates of expected hits, Janes and Hasselbrock apparently assumed that the indicators remain statistically independent when the h-r and control samples are considered separately. We had shown the assumption of independence was not seriously violated for all groups combined (N = 116). For the h-r group (N = 30) alone, the assumption is clearly violated, and the calculation of joint probabilities as the product of the individual probabilities leads to erroneous expectations. Furthermore, Janes and Hasselbrock's arguments treat the joint probabilities of two hits as if they were conditional probabilities of hitting on two but not three indicators. Their calculations show that about 11 h-r children are expected to hit on at least two. In our h-r sample, 8 children hit on at least two (3 h-r children hit on only two and 5 hit on three). Even if 11 hits on two indicators is a valid expectation, we doubt the difference of 8 vs. 11 is significant. Because of our prior maximizing of χ^2, the χ^2 distributions for our results are not known and precise tests of significance are not possible.

The goal of our analyses was to estimate which individuals among the children of schizophrenics are the 'true' h-r subjects. The case histories appended to our article suggest that our methods are at least partially successful. We hope the complexities of within-group analyses for small samples do not deter other h-r researchers from pursuing similar goals. It should be obvious that unreported studies cannot be replicated.

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RESPIRATORY VENTILATION

DEAR SIR,

It was with great interest that we read the article by Mora *et al* on respiratory ventilation (*Journal*, November 1976, **129**, pp 457–64). We also are studying CO_2 sensitivity in relation to breathing controls in normals and some pathological states (Guz *et al*, 1977). We were worried by some aspects of their interesting paper.

(1) There is considerable doubt as to the accuracy of using intranasal catheters as a means of measuring end tidal values. In particular, the authors do not state whether the catheter is down the back of the mouth or whether it is positioned at the front of the nose. They do not mention whether the patients are mouth or nose breathers. The site of the catheter and the breathing mode of the subjects are known to provide sampling errors.

(2) We wondered what the effect of an intra-nasal catheter would be on the respiratory variables and mental state of a subject who was already in a 'nervous' state.

(3) There seems to have been no study of intra-patient variability during any one test; we have realized that this may be a source of error in normal subjects.

(4) Capillary blood was taken from the finger and seems to correlate very poorly with the end tidal results which the authors claim to be satisfactory. This is surprising. The literature contains much work which suggests that the ear lobe is the only acceptable source of arterialized capillary blood which bears any reasonable comparison with end tidal measurements. Of course, there is no substitute for measurement of arterial blood itself.

(5) It is surely well established that benzodiazepines do not have a short duration of action; it has been shown that a single dose of diazepam, because of its slow detoxication and active metabolites, may act for up to 48 hours (The *Benzodiazepines*, Garattini *et al*, 1973).

(6) The authors do not state what criteria they used to conclude that their subjects were free from respiratory disease. It has been shown by Gregg *et al* that absence of symptoms provides little evidence of absence of respiratory disease.

(7) It was interesting to note that normal subjects had end tidal pCO_2 varying between 27–46 mmHg. The usual normal range is between 36 and 42. Perhaps some people are more normal than others?

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(8) End tidal CO_2 values were estimated for 5 minutes but only one value was quoted. What happened to the end tidal CO_2 over this 5 minutes?

We feel that there is insufficient evidence at present in the general metabolic and psychiatric literature for the authors to make the statement that 'some chemical differences between most depressed patients and controls are easily explained', and their conclusion that response to drug treatment may be due to effects on the blood brain barrier of CO_2 levels is premature in the extreme. It would be sad if work which could be extremely valuable in evaluating specific relationships between control mechanisms at, or below, limbic system level and cortical function were obscured by hasty conclusions from enthusiastic workers.

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MOURNING

DEAR SIR,

Gardner and Pritchard's article ('Mourning, Mummification and Living with the Dead' (*Journal*, January 1976, **130**, pp 23–8) describes some extreme examples of unresolved grief reactions. Their use of the term 'mummification' is certainly appropriate.

In their article they cite milder examples of abnormal reactions to bereavement, e.g. Queen Victoria's case, and these are in fact more commonly observed in practice. They do not mention an alternative term, which to my knowledge was first used by Macdonald Critchley the 'Miss Havisham syndrome' (1). The name is, of course, taken from Charles Dickens' *Great Expectations*. Miss Havisham in the novel was jilted by her fiancé on their wedding day and from that time became a recluse, wearing the fading satin of her bridal dress and 'freezing' the house as it was on that day.

Critchley says that Miss Havisham was a fictional character. Yet it has been suggested (Tyrrell (2)), that Dickens dramatized a story told to him of a Miss Donnithorne. This lady was the daughter of Judge James Donnithorne of Sydney. In her case too, the bridgegroom did not appear, and thereafter all the wedding decorations, etc, were kept as they had been arranged. Except for the doctor and the solicitor, the house was locked against all visitors; it was said to be haunted and was avoided by the children of the suburb. Miss Donnithorne died in 1886 at an advanced but unknown age, while Dickens wrote his novel in 1860. The dates would allow the real event to have taken place before the writing of the novel, though I believe that Dickensians hotly dispute this.

Though eponyms are now frowned upon in medical terminology, the term 'Miss Havisham syndrome' may be an appropriate one in describing a situation where an individual has not faced or 'worked through' a loss (whether death of a loved one or disappointment of any kind) but has attempted to maintain an inappropriate *status quo*.

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SPEECH IN DEPRESSIVE STATES

DEAR SIR,

We are grateful to Christopher Ounsted (*Journal*, March 1977, **130**, p 315) for drawing our attention to a case report which describes the changes in spontaneous speech in a manic-depressive patient. We have to comment, however, on his surprising statement that the detailed analysis of tape-recorded counting in depressed patients undertaken by us (Szabadi, Bradshaw and Besson, 1976) 'is not required'.

1. *Counting vs spontaneous speech.* The use of a sample of 'automatic speech' (Hughlings Jackson, 1878), such as counting, has advantages compared to the recording of 'spontaneous speech'. It is easy to standardize the recording of counting and it is not subject to the contents of the patient's thoughts, as spontaneous 'propositional' speech (Hughlings Jackson, 1878) is likely to be. Furthermore, counting speed is less likely to be inadvertently manipulated by the examiner's prompting questions.

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