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.

D. R. HANSON

Department of Psychology,
McMaster University,
Hamilton, Ontario L8S 4K1,
Canada

I. I. GOTTESMAN
L. L. HESTON

University of Minnesota,
Minneapolis, Minnesota 55455,
USA

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?