± SE).[1] When the same level of hypoxemia was induced in Temp *et al.*'s subjects abruptly, ventilation peaked at 26.2 ± 3.8 L/min and then settled to 15.9 ± 1.2 L/min over the subsequent 20 min.[2] The control responses of our subjects were typical for the particular conditions to which they were exposed,[5-7] but those of Temp *et al.*'s subjects appear unusually large.[8-10] (In the only studies closely comparable to that of Temp *et al., i.e.,* with the *identical* level of hypoxemia induced over a similar time period, together with eucapnia and normal levels of resting ventilation, the peak hypoxemic ventilation was 19.9 ± 2.0 L/min[8] and the 20-min values 9.8 ± 0.4[9] and 9.9 ± 1.0 L/min,[10] means ± SE.)

The apparently augmented control responses of Temp *et al.*'s subjects may have been due to extraneous stimuli (as suggested above) and/or to behavioral influences on breathing. Such biased responses could obscure an effect of isoflurane on the chemoreflex response *per se.* They also would explain why Temp *et al.* detected an effect of isoflurane when hypoxemia was induced with hypercapnia but not with normocapnia, because the more intense chemical stimulation of hypercapnic hypoxemia would reduce the relative impact of extraneous factors.

Statistical Inference. The conclusion of our study rejects the null hypothesis with an indicated type I error $P < 0.05$.[1] The conclusion of Temp *et al.* accepts the null hypothesis, but without an estimate of power or the probability of a type II error.[2] Temp *et al.* present 95% confidence limits for the effects of isoflurane on the control response in their subjects and infer that these limits "clearly exclude" our mean findings.[2] Unfortunately, this inference may not be valid, because the mean control response of Temp *et al.*'s subjects was above the 95% confidence limits for the control responses of our volunteers. Further, the mean control values of hypoxemic ventilation in Temp *et al.*'s subjects, both the peak value and that at 20 min, are well beyond the 95% confidence limits tabulated for the corresponding values from the most comparable investigations.[8-10]

Thus, these two studies differed in several important ways. One, performed in quiescent conditions, found that a typical response to hypoxemia was impaired by 0.1 MAC isoflurane consistently and inferred this effect of isoflurane with reasonable statistical probability. The other, performed with confounding extraneous stimuli, found that an unusually large response was affected variably and concluded the absence of an effect (as above) without statistical evidence. However one wishes to interpret each of these differences, they should not be ignored.

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In Reply:—Knill suggests we have accepted a null hypothesis, a conclusion based upon one phrase of a sentence in the abstract. Readers who went beyond the abstract found:

> Thus, the major finding of the study is the inability to reproduce the large, dramatic reductions of the acute hypoxic ventilatory response previously reported with 0.1 MAC concentrations of inhalational agents. A small to moderate reduction, however, can not be excluded by the data.[1]

In fact, we observed no significant effect of isoflurane on the acute hypoxic response, whether induced with normocapnia or with hypercapnia. The reduction of the *sustained* hypoxic response with hypercapnia, to which Knill alludes, also was substantially less than the 50–80% reductions previously reported *for the acute response.*[2] We noted that the acute and sustained responses are mediated somewhat differently and that Knill *et al.* has studied only the acute response.[2]

We went on to point out that "it seems probable that [Knill *et al.*'s] isoflurane confidence limits would overlap ours in the area of a 20–30% reduction" for the acute response.[1] And the final sentence of the manuscript begins, "The difference between our results and

those of Knill's group may indicate that subanesthetic isoflurane's effect on acute hypoxic sensitivity is less than previously reported . . ."[1]

Our *results* are indeed disparate with those of Knill *et al.*; any *conclusion* that those results prove an "absence of an effect," however, is solely Knill's interpretation.

Study Conditions. We do not fully agree with the assertion that measuring the hypoxic response requires "quiescent" environmental conditions. We[3–5,*] and others[6–9] have demonstrated successfully, for example, the effects of many drugs on the hypoxic response, despite music, videotape, bright lights, and/or "circuit motors." Such studies have found both augmentation[4,6,8] and depression[3,5,7,9,*] of the response.

We agree that our study and Knill *et al.*'s differed significantly in the way each handled the issue of subject state of consciousness. While we noted this in the manuscript, it is not clear how the biases, *which any approach to the problem will introduce,* should be interpreted.

For example, one might consider the propensity of human subjects "relaxed in a darkened and completely quiet room" to fall asleep, particularly while inhaling a subanesthetic volatile agent. Sleep in a quiet, dark room *in and of itself* reduces the hypoxic response by 30–70%.[10,11] If a sedative drug allows the subject to be *either* awake or asleep, it may be extremely difficult to separate a drug effect from a sleep effect.

We chose to have our subjects awake. It may well be that this choice alone explains the disparity between our results and those of Knill *et al.* We believe, however, that other differences in methodology, equipment, and techniques should be considered also.

Size of Control Response. Knill suggests "extraneous stimuli" may have augmented the control responses of our subjects. Subjects in two of "the only studies closely comparable to" ours, however, also were exposed to music[12,13] and, apparently, laboratory sounds.[13]

The control responses in our subjects were typical for our laboratory apparatus and technique and the hypoxic stimulus.[3–5,*] Moreover, Knill need look no further than Rebuck and Woodley,[14] Weiskopf and Gabel,[15] Javaheri and Guerra,[16] Mora *et al.*,[17] or Khamnei and Robbins[18] to find control hypoxic responses as large as ours. Further, both Douglas *et al.*[11] and Georgopoulos *et al.*[19] have performed subsequent studies in which the hypoxic response was as large.

Statistical Inference. We addressed above the issue of accepting a null hypothesis. We feel we must point out in addition, however, that the difference in control responses between Knill *et al.*'s subjects and our subjects is an irrelevant consideration to the confidence limits we have given. The measure being compared is a dimensionless ratio of the drug response to the control response and is, therefore, a valid comparison between laboratories, regardless of absolute magnitude of response.

In summary, Knill notes certain legitimate differences in study conditions, which we had noted in the manuscript. While we agree that these differences, and others, should not be ignored, our data nevertheless provide strong evidence that the effect of 0.1 MAC isoflurane is less than previously reported.

* Dahan A, Ward DS: The effects of nitrous oxide on the ventilatory response to sustained hypoxia in man (abstract). ANESTHESIOLOGY 73: A1168, 1990.

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A Right-angled Alligator Clip for Use with Insulated Nerve Block Needles

To the Editor:—Insulated needles with a nerve stimulator are used frequently to perform peripheral nerve blocks.[1] Typically, the anode is grounded to the patient, and stimulation of the needle tip is accomplished by attaching the cathode alligator clip to the metal hub of an insulated needle.

This needle assembly has a number of drawbacks. It is cumbersome and can be difficult to manipulate, particularly if the alligator clip is attached to a small needle. The wire from the alligator clip trails at a right angle from the needle, enabling it to enter the sterile field. Furthermore, the alligator clip has a tendency to move when the needle is manipulated during nerve blockade.

A solution to these problems is suggested by employing a right-angled alligator clip. This can be made by carefully breaking off that part of the alligator clip (Archer mini alligator clips 1-¼", cat no. 270-380A) that bears the "teeth" using pliers. The teeth are then soldered at a right angle to the ends of another alligator clip that have been shortened and flattened using pliers. When the teeth of the right-angled alligator clip grasp the hub of the block needle, the wire lies parallel to the extension tube and can be wound around it for added support (fig. 1). The right-angled alligator clip appears more compact and convenient to use than the standard alligator clip, particularly with short needles.

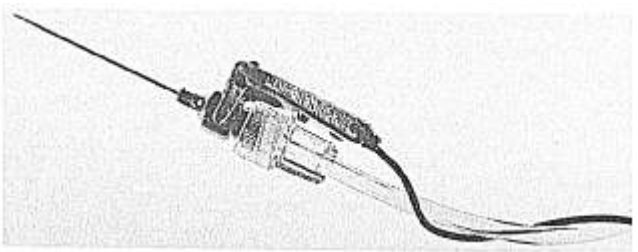

Fig. 1. Right-angled alligator clip attached to the hub of a 25-G sheathed pencil-point needle with extension set.

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Parents in the Operating Room?

To the Editor:—Many centers in which an appreciable number of pediatric surgical operations are performed allow parents or other responsible adults into the operating room during the anesthetic induction of children. Hopefully, this effects a less frightening and traumatic experience for the child by providing a familiar face in unfamiliar surroundings. The presence of parents in the operating room is not without its pitfalls, as illustrated by the following unusual experience.