REFERENCES

1. Jain U, Gold MI: Monitoring respirations during regional anesthesia. ANESTHESIOLOGY 60:166, 1984
2. ASA physical status rating (new classification of physical status). ANESTHESIOLOGY 24:111, 1963

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Anesthesiology
61:624, 1984

On the Prevention of Hypoxic Accidents

To the Editor:—In a recent letter to the editor, Dr. Zorab suggested that the way to prevent hypoxic accidents during anesthesia is to abolish the use of hypoxic gases such as 100% nitrous oxide on the anesthesia machine.[1] Instead, he recommends that all gases delivered to the machine contain at least 20% oxygen. As an advocate of low-flow and closed-circle anesthesia, I must point out that this method is not foolproof. For example, it is perfectly possible to deliver 1 l/min of air to a circle system attached to a 100-kg patient. This flow will more than keep the bag full on a tight circuit, yet will produce a hypoxic mixture in the circuit. This occurs because 1 l of air provides 209 ml of oxygen, while the basal metabolic rate for oxygen in a 100-kg patient is approximately 316 ml/min.[2] This same situation would occur with an 80/20 mixture of nitrous oxide oxygen but would take longer to develop, because of the initial high rate of nitrous oxide uptake and the ensuing second gas effect.

Thus, a calibrated, working oxygen meter in the circuit is still the best insurance against hypoxic accidents during anesthesia[3] and "eternal vigilance is the price of safety."

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REFERENCES

1. Zorab JSM: A simple, foolproof method to prevent hypoxia accidents. ANESTHESIOLOGY 60:515–516, 1984
2. Brody S: Bioenergetics and Growth. New York, Reinhold, 1945
3. Rendell-Baker L, Meyer JA: Failure to use O_2 analyzers to prevent hypoxic accidents. ANESTHESIOLOGY 58:287–288, 1983

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A Simple Device for Testing Peripheral Nerve Stimulators

To the Editor:—While monitoring the neuromuscular junction with a peripheral nerve stimulator (PNS) the need sometimes arises (such as when broken lead wires are suspected) to determine if the electrical stimuli are actually present at the patient electrodes. One way to confirm presence of stimuli is to feel for the pulses with one's own fingers, but this can be unpleasant or even painful.

I have found a simple alternative using an inexpensive neon lamp (type NE-2), available from most electronic parts suppliers. The lamp is touched or clipped to the ends of the lead wires (fig. 1), and it should flash with each single pulse or stay lighted with a tetanic stimulus. The orange glow can be seen even in a brightly illuminated room, and the device will not harm the PNS.

A neon lamp typically fires above 95 volts dc, while most PNSs deliver a maximum voltage of about 300 volts. Hence, the test will work at all but the lowest output settings of the PNS. However, as shown in a recent report,[1] the output voltage of a PNS drops

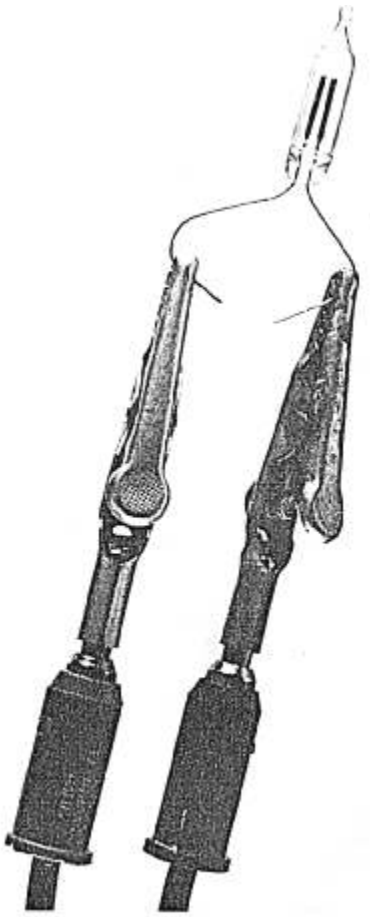

FIG. 1. Neon lamp (type NE-2) attached to clips from peripheral nerve stimulator.

drastically into an impedance load typical of surface or needle electrodes. Therefore, the neon lamp test will not work with both lead wires attached to the patient.

This test does not check for malfunction due to faulty electrodes, poor skin contact, or unusually high skin impedance, such as with xeroderma. Good clinical practice still dictates that a baseline patient response to the PNS be determined before giving neuromuscular blockers.

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REFERENCES

1. Mylrea KC, Hameroff SR, Calkins JM, Blitt CD, Humphrey LL: Evaluation of peripheral nerve stimulators and relationship to possible errors in assessing neuromuscular blockade. ANESTHESIOLOGY 60:464–466, 1984

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Bupivacaine: Cardiotoxicity or Anesthetic Technique?

To the Editor:—The possibility that bupivacaine exhibits greater cardiotoxicity than shorter-acting local anesthetics has made us all more aware of potential complications of major regional anesthetics. In a case we reported, a seizure and ventricular arrhythmias occurred while attempting epidural anesthesia with bupivacaine.[1] Subsequently, Batra *et al.*[2] and Knapp[3] were critical of our technique, and the former authors imply that such cases may lead to a short-lived rebirth of regional anesthesia.[2] We contend that the techniques utilized in this case, which occurred in March, 1981,[1] were not inappropriate at that time, and that such reports provide a basis for improving anesthetic practice. It is knowledge of techniques that may result in systemic toxicity, such as those reported by us[1] and others,[4–6] that can improve the safety of regional anesthesia. Current recommendations[7] concerning epidural administration of bupivacaine include the following: the use of a test dose containing epinephrine to detect intravascular injection, utilizing a "one-shot" technique instead of a catheter technique when giving 0.75% bupivacaine, and avoiding the injection of a large single dose by administering increments of 3–5 ml when 0.5% or 0.75% bupivacaine is used. We agree with the comments[2,3] that these procedures would have reduced the chance of systemic toxicity in the case we reported.[1]

Accepting the importance of the above recommendations, however, does not resolve the controversy as to