anesthesia. She also stated that he usually took more pills than those prescribed in order to better control his spasticity.

Baclofen, a γ-aminobutyric acid (GABA) derivative, is believed to exert its antispastic effects by inhibiting monosynaptic and polysynaptic spinal reflexes through an action on GABAergic interneurons. Individualized dosage is required, but the total daily dose should not exceed 80 mg daily. Baclofen can cause CNS depression because of an action on supraspinal receptors that may be potentiated by other CNS depressants. General anesthetics may act via potentiation of GABA action on synaptic transmission. Binding sites for GABA, benzodiazepines, and barbiturates have been described to be allosterically coupled within a three-receptor protein complex. It has been suggested that baclofen may act through binding to either the central benzodiazepine receptor inside the GABA complex or to specific receptors.

Baclofen also has adverse cardiovascular effects. In the case we report, sinus bradycardia without hypotension persisted. Sil et al. recorded bradycardia and hypotension during general anesthesia associated with baclofen premedication (30 mg orally) and suggested disturbance of the autonomic control of the circulation mediated via a GABAergic, baclofen-sensitive system as a possible cause.

Physostigmine has been shown to reverse coma and other baclofen overdose symptoms, and intravenous flumazenil has been reported to counteract intrathecal baclofen-induced CNS depression. We did not administer any of these drugs to the patient.

In conclusion, baclofen often is used in patients with spasticity resulting from spinal injuries for which spinal anesthesia is usually avoided because of medicolegal considerations. When general anesthesia has to be administered, we suggest avoidance of other GABA agonists such as benzodiazepines or barbiturates to prevent additive effects.

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Alarm Signals Used in Anesthesia and Intensive Care

To the Editor.—Recently, Wallace et al. described the hearing acuity of 188 anesthesiologists. This important study showed that 66% of the subjects had an abnormal audiogram. The authors made reference to an American Society for Testing and Materials (ASTM) proposed draft specification for alarm signals used in anesthesia and respiratory care, dated August 27, 1991. This standard was approved on February 15, 1993, and published in June 1993. The International Standard ISO 9703-1, Anaesthesia and Respiratory Care Alarm Signals: Part 1. Visual Alarm Signals, was published on July 15, 1992, and the International Standard ISO 9703-2, Anaesthesia and Respiratory Care Alarm Signals: Part 2. Auditory Alarm Signals, was approved on January 8, 1994, and will be published later this year. The process of writing these standards took advantage of the information presented by Wallace et al. at the annual meeting of the American Society of Anesthesiologists in New Orleans, Louisiana, in October 1992, and papers presented at a symposium entitled “Operating Room and Intensive Care Alarms and Information Transfer” held in Zurich, Switzerland, in February 1991.

The cacophony of alarm signals in operating rooms and intensive care units must be decreased. The International Organization for Standardization (ISO) Technical Committee 121, Subcommittee 3 on Ventilators and Related Equipment, is preparing the proposed draft specifications, “Anesthesia and Respiratory Care Alarm Signals: Part 3. General Requirements” and “Anesthesia and Respiratory Care Alarm Signals: Part 4. Guidelines on Application of Alarms.” Recently, Hyman stated that “Operating Room and Intensive Care Alarms and Information Transfer” (ASTM STP 1152) should be required reading for anyone involved in designing operating or intensive care rooms, and the associated equipment. Similarly, biomedical engineering students should read this book as part of their design classes as an introduction to the human factors component of the design problem.” They also should read Wallace et al.’s study of how auditory alarms perform at present. Much still needs to be accomplished in improving alarm technology.

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How Should Vascular Resistance Indexes Be Computed?

To the Editor:—Hemodynamic measurements such as cardiac output commonly are scaled by dividing them by body mass or body surface area (BSA) to help reduce interindividual variability. Less commonly appreciated, however, is the fact that resistances such as pulmonary vascular resistance (PVR) are not properly scaled by dividing by BSA. Physically, PVR primarily is determined by the number of vessels, their diameter and length, and the viscosity of the perfusate. Because the number of vessels increases as a function of body size, it is logical that scaling resistance measurements by BSA should result in a larger, not smaller, value.

I have read with great interest the excellent study by Puybasset et al. on the influence of inhaled nitric oxide on PVR in patients with acute respiratory distress syndrome. I wish to point out an error in the units for PVR and systemic vascular resistance (SVR) values in table 1, table 3, and figure 3. It is not apparent whether these are typographical errors or due to the use of an incorrect formula to scale resistances to BSA. If the authors divided PVR by BSA to compute PVRI, as their units would indicate, then the data are incorrect. The correct formula for computing PVR index (PVRi) is: PVRI = PVR × BSA, with the units for PVRI being dyne · cm⁻² · m² (note the absence of a minus sign on the exponent of “m²”). The formula for SVRI is analogous. However, if the authors calculated PVRI as PVRI = ∆P/CI (where ∆P is the pressure drop across the pulmonary resistance vessels and CI is cardiac index, where CI = CO/BSA), then the values

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