response to that action. It must be remembered, however, that, in the late 1930s, specialty boards proliferated at a rapid rate, and with McMechan's death, another obstacle was removed.

Originally, the ASA was formed to sponsor the American Board of Anesthesiology (ABA). Although much can be made of internationalism *versus* isolationism of the late 1930s, it is my belief that American anesthesiology looked to form one large, powerful national organization upon the death of Francis McMechan. On July 20, 1939, 3 weeks after McMechan's death, Paul Wood wrote that

> Is there any possibility of amalgamating all the organizations? . . . I would be pleased personally to have the International, Associated, and American join in one organization under any name they see fit and any secretary they might desire to have. I am perfectly willing to give up the American Society since it has accomplished the goal for which it was primarily working, that is, recognition of the American Board of Anesthesiology.[1]

My point in speculating on what might have been had the ASA and the IARS merged was to illustrate the vacuum in anesthesiology that developed as the "new" organization—the ASA—developed and defined its role. I respectfully disagree with Lawson's assertion that any merger would have been shortlived. Had the proposed merger been able to satisfy all parties, especially Mrs. McMechan, one strong society could have emerged. The anesthesiology community at the time was small, and there is little doubt that a single voice for organized anesthesia, which we now have, would have been beneficial in the late 1940s and 1950s.

As a member of both the ASA and the IARS, I am glad that there are at least two avenues for publication of historical and scientific research, and two outstanding journals to read. It is a credit to the post-McMechan IARS and ASA leadership that this relationship has developed.

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(Accepted for publication August 1, 1994.)

Anesthesiology
81:1306–1307, 1994

Delayed Arousal after General Anesthesia Associated with Baclofen

To the Editor:—A 49-yr-old, 63-kg man with spinal cord injury due to giant spinal lipoma was scheduled for penile prosthesis. The lipoma extended from T11 to L5, affecting conus medullaris and cauda equina. Although spinal cord was compressed by the tumor in its whole perimeter, compression was predominantly posterior. The patient had a surgical history of a cervical arthrodesis and a lamininectomy from T12 to L4 for excision of the mentioned spinal lipoma 14 yr before. The lipoma had caused sexual impotence, neurogenic bladder, unsteady gait, and muscle spasms in the lower extremities. The latter was treated with oral baclofen, 25 mg three times per day. Preoperatively, the patient did not complain of drowsiness or respiratory difficulties. Heart rate was 45 beats/min. Previous to general anesthesia, 1 mg atropine and 1.5 mg midazolam were administered intravenously. Bradycardia persisted, and exaggerated sedation was observed. Anesthesia consisted of 300 mg thiopental, 100 μg fentanyl, and 1% isoflurane in oxygen and nitrous oxide; $F_{I_{O_2}}$ 0.5. Atracurium (30 mg) was given to accomplish tracheal intubation. Intraoperatively, heart rate remained 40–45 beats/min, and arterial blood pressure varied between 110/75 and 140/85 mmHg. Surgery lasted 30 min. Neostigmine (1.5 mg) and atropine (1.5 mg) were administered intravenously twice, but the patient remained unconscious and unresponsive 6 h after surgery. No other drugs were given, and the results of serial blood gas analyses were normal. Later, slight myoclonus of the patient's shoulders and chest were noted. Consciousness was progressively regained, muscle tone was restored, and separation from mechanical ventilation was performed. Postoperatively, the patient had total amnesia of this postoperative period.

Pharmacologic causes, metabolic disturbances, or neurologic injury have been involved as possible etiologies of delayed awakening after general anesthesia. In this case, no history of endocrine disease, perioperative hypoglycemia or hyperglycemia, electrolyte or blood gas disorders were recorded. Although the surgical procedure should not have led to central nervous system (CNS) dysfunction, intracranial problems could not be excluded. The excessive sedation found after premedication with midazolam and the fact the anesthetic agents used were ordinarily short-acting and given in small total doses caused us to suspect that an increased central sensitivity to anesthesia could have been a possible cause of the delayed arousal.

In the immediate postoperative period, the patient's wife related two prior instances of prolonged unresponsiveness after general

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 $GABA_B$ medullary interneurons.[1] 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[2] that may be potentiated by other CNS depressants. General anesthetics may act *via* potentiation of GABA action on synaptic transmission.[3] Binding sites for GABA, benzodizepines, and barbiturates have been described to be allosterically coupled within a three-receptor protein complex.[4] It has been suggested that baclofen may act through binding to either the central benzodiazepine receptor inside the GABA complex or to specific receptors.[5]

Baclofen also has adverse cardiovascular effects. In the case we report, sinus bradycardia without hypotension persisted. Sill *et al.*[6] 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,[7] and intravenous flumazenil has been reported to counteract intrathecal baclofen-induced CNS depression.[8] 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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(Accepted for publication August 3, 1994.)

Anesthesiology
81:1307–1308, 1994

Alarm Signals Used in Anesthesia and Intensive Care

To the Editor:—Recently, Wallace *et al.*[1] 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" [2] held in Zurich, Switzerland, in February 1991.

* American Society for Testing and Materials: Standard specification for alarm signals in medical equipment used in anesthesia and respiratory care, ASTM F 1463-93. Philadelphia: ASTM, 1993.

† International Organization for Standardization: Anaesthesia and respiratory care alarm signals: Part 1. Visual alarm signals, ISO 9703-1:1992. Geneva, ISO, 1992 (available from American National Standards Institute, 11 West 42nd Street, New York, NY 10036).

‡ International Organization for Standardization: Anaesthesia and respiratory care alarm signals: Part 2. Auditory alarm signals, ISO 9703-2:1994. Geneva, ISO, 1994 (available from American National Standards Institute, 11 West 42nd Street, New York, NY 10036).