Anesthesiology 2001; 95:271

Sedation Caused by Local Anesthetic Blockade

To the Editor:—I read with interest the article "Sedation during Spinal Anesthesia" by Pollock *et al.*[1] During the past 12 yr, while performing local anesthetic blocks for eye surgery without the administration of sedative medication, I have observed a considerable number of patients who become lightly sedated after block insertion. These patients note the sedative effect not infrequently and ask what has been administered to produce it. They do not attribute it to relief that a stressful procedure has been successfully completed. I have not noticed comparable sedation with any other peripheral local anesthetic blocks. The mass of local anesthetic is small, generally between 3–5 ml lignocaine, 2% (60–100 mg), but in excess of the quoted spinal doses in the article. Within its sheath and the layers of dura and arachnoid mater that surround it, the optic nerve is bathed in cerebrospinal fluid.

Brain stem anesthesia by direct injection into the subdural space around the nerve is a well-documented complication of retrobulbar blocks.[2] In most regional anesthetic techniques for eye surgery, the deposited local anesthetic gains access to the intraconal space, and there it lies in close contact with the optic nerve. This is particularly true of sub-Tenons and uncomplicated retrobulbar blocks. Is it possible that this unique epidural deposition of local anesthetic may have a similar mechanism of sedative action in these patients as occurs in spinal or classic epidural anesthetics?

Robert Andrew Fry, M.B., Ch.B., D.A. (U.K.), F.A.N.Z.C.A., Auckland Hospital, Auckland, New Zealand. RobF@ahsl.co.nz

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In Reply:—We appreciate the comments of Dr. Fry and are grateful for the opportunity to reply. Although we do perform retrobulbar blocks at our institution, all of our patients are sedated with propofol before block placement; therefore, we are not able to comment directly on Dr. Fry's observation of sedation associated with retrobulbar block. As mentioned in our article, there have been three theories proposed for the mechanisms of sedation associated with spinal anesthesia: increased serum concentrations of local anesthetics, rostral spread of local anesthetics into the brain, and decreased afferent input from the spinal cord. Of these three theories, only a delayed rostral spread of the local anesthetic might reasonably account for the sedation we observed in our volunteer group 60 min after spinal anesthesia. As Dr. Fry mentions, given the proximity of the optic nerve to the cerebrospinal fluid, it is not inconceivable that a similar effect may be responsible for his observation of sedation after retrobulbar block.

Julia E. Pollock, M.D.,* Spencer S. Liu, M.D., Joseph M. Neal, M.D.
*Virginia Mason Medical Center, Seattle, Washington. anejep@vmmc.org

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Thomas Drysdale Buchanan or Henry Isaiah Dorr: Give Credit to Both

To the Editor:—I read with great interest the correspondence "Henry Isaiah Dorr Was the First Person to Hold the Title Professor of Anaesthesia."[1] Lowenstein and Kitz state, "The earliest previously known appointment of a Professor of Anesthesia was that of T. S. Buchanan at the Flower School of Medicine in New York City in 1905." During the past 2 yr, I have researched the beginnings of the anesthesia department at New York Medical College (Valhalla, NY), in particular the career of Thomas Drysdale Buchanan, M.D. I will explain the difference between the Dorr and Buchanan appointments and, in so doing, give credit to the many areas in which Buchanan contributed to the recognition of the specialty of anesthesiology.

Buchanan graduated from The New York Homeopathic Medical College and Hospital in 1897 (originally chartered in 1860 as The Homeopathic Medical College of the State of New York in New York City) and then interned at the Metropolitan, Post Graduate, and Flower Hospitals in New York City. He was appointed anesthetist at Flower Hospital in 1898 and director of anesthesia at the Fifth Avenue Hospital in 1902.

Quoting from a memorial to Buchanan (read by Ralph Waters, M.D., at the International Anesthesia Research Society meeting in 1940) written by Paul Wood, M.D., and published in *Current Researches in Anesthesia and Analgesia*, "In 1904 he (Buchanan) established a precedent, still in vogue, of having a department of anesthesia headed by a full time professor whose work was limited to the field of anesthesiology. Although others had taught officially in medical schools, they had not so limited their work, and to T. D. Buchanan goes the credit for being the first in this field."[2]

To New York Homeopathic Medical College (through a series of name changes, it became New York Medical College and Flower Hospital in 1936) goes the distinction of being the first medical college to have both a department dedicated to the specialty of anesthesia and a full-time professor of anesthesia. Here in lies the difference between Henry Isaiah Dorr, M.D., D.D.S., who was appointed Professor of the Practice of Dentistry, Anaesthetics and Anaesthesia at the Philadelphia College of Dentistry and Thomas Drysdale Buchanan, M.D., Professor of Anesthesia at the New York Homeopathic Medical College.

The Long Island Society of Anesthetists, established in 1905 by nine physician anesthetists, limited membership to physicians from Brooklyn (Long Island), New York. At the constant urging of colleagues from Manhattan, the nine founders reluctantly agreed to increase charter membership to no more than 50 physicians. Buchanan and J. T. Gwathmey, M.D., were admitted early on, with P. J. Flagg, M.D.,

becoming the final charter member.[3] This organization became the New York Society in 1911, and with the urging of both Buchanan and Wood, the American Society of Anesthetists came into being in 1936, undergoing the last of its name changes, in 1945, to the American Society of Anesthesiologists. "He (Buchanan) was a Founder and Past President of the American Society of Anesthetists; and at the time of his death (1940) he was President of the American Board of Anesthesiology, both of which organizations his labors helped so materially to establish. The latter was one of the most difficult tasks, as not only was there the usual lack of interest, but in addition much active opposition on the part of the medical profession. His personality, persistence and good judgment finally overcame all opposition."[2] Buchanan, who was the first President of the American Board of Anesthesiologists (written communication, Francis P. Hughes, Ph.D., American Board of Anesthesiologists Executive Vice President, Raleigh, NC, February, 2001), Wood, and Emery Rovenstine, M.D., were the examiners for the first board examination in 1939. Board certificate No. 1 was granted to Buchanan.[4,5,6,7]

Why my interest in Thomas Drysdale Buchanan? I graduated from New York Medical College, Flower and Fifth Avenue Hospitals in 1950, completing both my internship and anesthesia residency at the Flower and Fifth Avenue Hospitals. Donald E. Brace, M.D. (American Board of Anesthesiologists certificate No. 32), was Professor and Chair of the Department of Anesthesia. Brace was a close personal friend of Buchanan and had succeeded him as department head. Buchanan remained a member of the anesthesia department until his death in 1940. Don Brace, who referred to Buchanan as "Buck," frequently spoke of him and the influence he had on our specialty. On one occasion, when meeting with the residents, he shared a letter he had earlier received from Buchanan, describing his training and introduction to the field of anesthesia—a letter from which Paul Wood quoted when memorializing Thomas Drysdale Buchanan. "I was a junior at the old Homeopathic College and at the time it was the practice to take on four seniors to administer the anesthetics for the clinics. Naturally, I was anxious to be selected as one of the four who were to anesthetize for old Dr. Helmuth's clinic. In trying to obtain this experience, I finally importuned a junior surgeon and asked him if he would allow me to give an anesthetic and he said, 'Yes indeed, you bring me a case for surgery and I will let you give the anesthetic.' So I did. That was about the only instruction I had in anesthesia, more than most internes received at that time."[2]

In our society, we place a premium on being first. Of equal if not greater importance is the impact that those we anoint with the title of first have on future events. Each in their own way, Dorr and Buchanan played significant roles in the growth and development of the specialty of anesthesiology.

Bernard V. Wetchler, M.D., University of Illinois College of Medicine, Chicago, Illinois. wetchler3@cs.com

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Airway Bleeding in Negative-pressure Pulmonary Edema

To the Editor:—I read with great interest the case report by Dr. Dolinski *et al.*[1] of a patient with severe pulmonary hemorrhage associated with negative-pressure pulmonary edema after an episode of postextubation laryngospasm. Last year, I reviewed six cases of negative-pressure pulmonary edema at my institution.[2] Hemoptysis was a feature in five of these, and one patient, like the patient of Dr. Dolinski *et al.*, had clinical and computed tomography findings consistent with alveolar hemorrhage. This latter patient was also investigated extensively for a vasculitic process to explain the airway bleeding, but no such pathology was found. It seems that airway bleeding is a feature of negative-pressure pulmonary edema. In fact, some have suggested that the condition should be renamed "negative-pressure injury" to reflect the pathophysiology more accurately.[3] The negative pressure seems to cause a more severe injury to the lung than could be explained by changes in hydrostatic forces across the pulmonary capillary basement membrane alone. To my knowledge, bronchoscopy has been performed twice during an episode of negative-pressure pulmonary edema. Schwartz *et al.*[4] found fresh blood in the airway in his case of negative-pressure–induced alveolar hemorrhage. Koch *et al.*[5] found hemorrhagic lesions lining the trachea and large airways leading to speculation that bronchial rather than pulmonary capillary disruption was to blame. Analysis of the edema fluid itself has consistently revealed it to be an exudate,[6,7] further evidence for disruption of the capillary basement membrane.

I agree that the incidence of this problem is higher than is commonly believed. The incidence of 0.1% of anesthetic procedures reported by Dr. Dolinski *et al.* is supported an incidence of 0.094% reported by Deepika *et al.*[8] in a much larger series (30 patients). My own smaller series suggested a figure of approximately 0.05%.[2] Because the condition can be life threatening and the usual consequence is intensive care admission and a prolonged hospital stay, negative-pressure pulmonary edema is an important cause of perioperative morbidity. I think it is underreported and often misdiagnosed as fluid overload, aspiration, or even bronchospasm.

Paul McConkey, M.D., UC Davis Medical Center, Sacramento, California. ppmcconkey@ucdavis.edu

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Precipitation of Thiopental by Rapacuronium

To the Editor:—Rapacuronium is marketed as an alternative to succinylcholine for rapid sequence induction, especially in the pediatric population. Recently, we had to administer anesthesia to a 5-yr-old child (weight, 22 kg) considered to have a full stomach. A general anesthetic was planned with a rapid sequence induction using thiopental and rapacuronium. Thiopental (4 mg/kg) was injected, and immediately after the 22-gauge intravenous line was visibly cleared of thiopental, rapacuronium (1.5 mg/kg) was injected. Rapacuronium and thiopental precipitated in the intravenous line, making it inoperative.

Precipitation has been observed with thiopental and other depolarizing and nondepolarizing muscle relaxants, including succinylcholine, rocuronium, vecuronium, and pancuronium.[1,2] The mechanism seems to be a pH-dependent decrease in the solubility of thiopental.[1,3] Similarly, the differing pH of rapacuronium (pH 3.9) and thiopental (pH 10.6) may explain the observed precipitation. The complications of this interaction are serious and we would like to inform our colleagues about it.

Ullrich Schmidt, M.D., Salvatore R. Basta, M.D., Erik S. Shank, M.D.* *Massachusetts General Hospital, Boston, Massachusetts. eshank@partners.org

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Hofmann, Schmofmann: Atracurium Undergoes Michael Elimination

To the Editor:—Cisatracurium and atracurium are short-acting because they undergo spontaneous decomposition under physiologic conditions. We have all heard of the process termed a Hofmann elimination. In pharmacology, elimination is a mechanism of removing the active form of a drug from the body. In chemistry, elimination is a reaction giving rise to a double bond. The pharmacologic elimination of atracurium isomers happens to involve a chemical elimination reaction, but is the reaction a Hofmann elimination?

Elucidating the structures of numerous alkaloids, August Wilhelm von Hofmann (1818–1892) broke down quaternary amines by heating them with a flame. Under the conditions of Hofmann, all of the neuromuscular blocking agents undergo decomposition. Clearly, the atracurium isomers possess some special chemical feature. They are unique because their nitrogenous leaving groups are situated in the "β position" with respect to a carbonyl group (fig. 1). Carbonyl compounds possess acidic α hydrogens and therefore undergo ready α,β elimination reactions. These facile reactions were first emphasized by Arthur Michael (1853–1942) and are often named after him.[1–3]

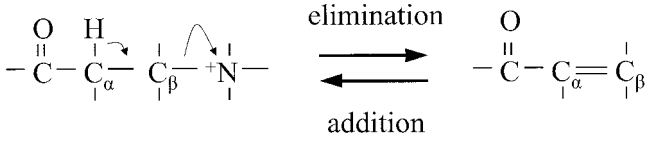

Fig. 1. Michael-type addition–elimination reaction.

The term Hofmann elimination correctly indicates that atracurium is a quaternary amino compound that is transformed into a tertiary product. However, the term does not enlighten as to why the other quaternary amino relaxants do not also undergo that reaction in the body. Although the term Michael elimination would be mechanistically informative, Hofmann elimination is probably here to stay. Michael did spend some student days in the Hofmann laboratory, and perhaps Michael would not have objected to this remembrance of a mentor—maybe.

Theodore A. Alston, M.D., Ph.D., Massachusetts General Hospital, Harvard Medical School, Boston, Massachusetts. talston@partners.org

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Use of the Esophageal Doppler with the *LMA-ProSeal*™

To the Editor:—Esophageal Doppler monitoring is a noninvasive method to determine stroke volume and cardiac output.[1,2] So far, it has been used in awake patients or intubated patients. Either the laryngeal mask airway (LMA) cuff made the introduction of the Doppler probe impossible or the fear that the insertion and possible intraoperative movement of the Doppler probe might displace the LMA or increase the risk for aspiration prevented its use. Recently, the *LMA-ProSeal*™ (Laryngeal Mask Company, Nicosia, Cyprus) has been made commercially available.[3] A comparison with a standard mask did not show any differences in insertion, trauma, or quality of airway.[4] The *LMA-ProSeal*™ incorporates a port (inner tube diameter: 7 mm for LMA size 5, 6.5 mm for LMA size 4; LMA size 3 is not available with a port) originally designed for insertion of a gastric tube to reduce gastric inflation and reduce gastric regurgitation during longer procedures.

We have inserted the esophageal Doppler probe (Deltex Company, Irving, TX; diameter: 6 mm) through the drainage port of the *LMA-ProSeal*™ successfully in 30 patients undergoing general surgery. It is important that the whole of the Doppler probe is well-lubricated to facilitate insertion and movement of the probe during the procedure. After induction of anesthesia and insertion of the *LMA-ProSeal*™, the Doppler probe was inserted in all patients in the supine position. The insertion was easily performed through the port of the *LMA-ProSeal*™ without displacement of the laryngeal mask. In masks of size 4, a resistance could be felt during insertion of the Doppler probe when passing the curve of the port within the LMA cuff but could easily be overcome and did not obstruct the movement of the Doppler probe. Regardless of the patient position (supine or lateral position), measurements of stroke volume and cardiac output were monitored throughout the procedure in all patients without additional repositioning of the probe. At the end of surgery, the Doppler probe was easily extracted, and the laryngeal mask was removed after the patient was fully awake. We conclude that the drainage port of the new *LMA-ProSeal*™ cannot only be used to insert a gastric tube but also to insert the Doppler probe. The *LMA-ProSeal*™ allows anesthesiologists to use esophageal Doppler monitoring—something that has previously been impossible.

Thomas M. Hemmerling, M.D., D.E.A.A., Centre Hospitalier de l'Université de Montréal Hôtel-Dieu Montréal, Hôtel-Dieu, Montreal, Quebec, Canada. thomashemmerling@hotmail.com

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