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Anesthesiology
1997; 87:461

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Tribute to Dr. Austin Lamont

To the Editor:—The beautiful article about Dr. Austin Lamont by Stanley Muravchick and Henry Rosenberg (ANESTHESIOLOGY 1996; 84:436–41), is one of several highly deserved tributes to this pioneer in academic anesthesiology. Having been an anesthesiology resident with him at the University of Pennsylvania in 1950–52, I would like to add some personal memories.

Because of an oversubscribed immigration quota for Austrians, my wife Eva and I went to Lima, Peru where in 1953 I initiated Peru's first academic department of anesthesiology. Without Lamont's and Dripps's interventions, we may not have obtained the then necessary special preference immigration visa (for needed specialists) to return to the US and become US citizens. Without Lamont's advice, I might not have gone to the Johns Hopkins Hospital (1954–55) and from there to Baltimore City Hospital as the first full-time chief of the department of anesthesiology (1955–61). Without these experiences, I would not have been prepared for the first chairmanship of the Department of Anesthesiology and Critical Care Medicine at the University of Pittsburgh Medical Center.

After all anesthesiologists of the Johns Hopkins Hospital division of anesthesiology resigned in the summer of 1955 because of the impossibility to develop an academic anesthesiology department and residency there, Lamont shared with me his experiences at the Johns Hopkins Hospital. Later, after an invitation by Johns Hopkins historian, Magee Harvey, Professor Emeritus of Medicine, I submitted my biased reflections on four historic attempts to establish a strong academic anesthesiology department at the Johns Hopkins Hospital—

under Lamont and Harmel in the 1940s; Proctor, Safar, and Bachman in the 1950s; Benson in the 1960s; and Nagel in the 1970s. In the 1980s, Rogers *et al.*, finally succeeded.

Austin was modest. In 1957, I showed him the Guedel airway I had modified with a mouthpiece for mouth-to-tube ventilation. He suggested replacing the straight mouthpiece with a turned around pediatric airway so it could also be used for children by turning it around. He asked for no credit.

Austin was compassionate. For our severely asthmatic daughter, he arranged for a consultation on the then not popular aerosol treatments. When she died in 1966, Lamont said: "When suffering becomes unbearable, nature (God) often lifts the burden." This applied to him at the end, when he died in 1969.

Austin was an academician with principles. He quietly influenced me and other residents to consider research careers by making Claude Bernard's book *An Introduction to the Study of Experimental Medicine* his farewell gift.

Austin was the best representative of American aristocracy. We miss him.

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(Accepted for publication April 25, 1997.)

Anesthesiology
1997; 87:461–2

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Lippincott-Raven Publishers

Possible Interaction of Esmolol and Nitrous Oxide

To the Editor:—In the recent article by Johansen *et al.*¹ describing the reduction of anesthetic requirements by esmolol, nitrous oxide is probably the primary anesthetic agent. It probably is the agent effecting the greatest contribution to the minimum alveolar concentration, or its equivalent, for intravenous anesthetic agents. This premise is supported by the reported propofol Cp_{50} of 3.85 $\mu\text{g/ml}$, with nitrous oxide compared to a Cp_{50} of 15.2 $\mu\text{g/ml}$ for propofol as the sole anesthetic agent for skin incision in tracheally intubated patients.² Although the patient groups in these two studies are not directly comparable, the absence of nitrous oxide results in an approximately 300% increase in the Cp_{50} for propofol.

If nitrous oxide is the primary anesthetic agent, then esmolol may affect its anesthetic action by inhibiting the sympathomimetic action of nitrous oxide. This increased sympathetic activity during nitrous oxide anesthesia has been found to antagonize both central nervous depression by isoflurane and isoflurane-induced suppression of learning.^{3,4} Any augmentation of the potency of nitrous oxide by the sympatholytic effects of esmolol would explain the reduction of anesthetic requirements in the study of Johansen *et al.* in humans,¹ Perel *et al.* in the rat,⁵ and the efficacy of esmolol as a narcotic substitute in previous studies.^{6,7}

Johansen *et al.* give no details of the temporal events during induc-

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tion of their patients. If esmolol does not effect its anesthetic action *via* nitrous oxide, did the authors notice a difference in the time to loss of consciousness between the propofol only, and the propofol plus esmolol groups, in the absence of nitrous oxide?

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1997; 87:462

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In Reply:—We appreciate Dr. Baranov and Dr. Bagshaw's constructive comments. The mechanism by which esmolol decreases anesthetic requirements cannot be deduced from our paper. Our goal was to document the existence of this interaction. The balanced-anesthetic technique used in the paper was complex, involving propofol, nitrous oxide, and morphine premedication. At the time, this technique represented the best clinical conditions for demonstrating the effect of esmolol. An interaction between any of the anesthetic components could be hypothesized. The experiment was specifically designed to examine the interaction under conditions approaching steady state. No attempt was made to evaluate differences in anesthetic induction between groups.

Currently, available clinical evidence suggests that esmolol potentiates opioid action. We have found that esmolol has no effect on isoflurane MAC alone but can potentiate MAC reduction by alfentanil.¹ This supports an earlier observation by Stanley *et al.*² that patients receiving long-term, propranolol treatment required 25% less fentanyl to produce loss of consciousness.

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(Accepted for publication April 26, 1997.)

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(Accepted for publication April 26, 1997.)