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### Prevention of Hypoxic Gas Mixtures

*To the Editor:*—The “malfunction” detected and reported by Abraham and Basagoitia<sup>1</sup> is of particular interest because it involves failure of a device which is absent on many anesthesia machines and, in ideal practice, would not be necessary. Many users of the Ohio Modulus<sup>™</sup> have discovered that the chain coupling of its oxygen and nitrous oxide needle valves can be used to initiate 25% oxygen in nitrous flow by turning the blue knob, without touching the green. This step-saving habit courts catastrophe if attempted on any machine without a functioning chain.

One should *never* increase the flow of nitrous oxide without first confirming adequate gas flow through the oxygen rotameter; conversely, oxygen flow should never be reduced below a safe ratio with the existing

nitrous oxide flow. This approach (when raising flows, oxygen first; when decreasing them, nitrous oxide first) more reliably protects against a hypoxic gas mixture than does any device. Device failure often becomes “potentially lethal” only when safe practice is violated.

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### A Laboratory Test to Detect Antibodies to Protamine

*To the Editor:*—We are developing a laboratory test to detect antibodies to protamine in patient's blood. This test will be used to document that the patient has had a protamine reaction or to predict whether the patient may safely receive protamine in those previously sensitized to this molecule.

To both develop and validate this test, I will need serum from patients who have had such reactions. If you know of a patient who has had severe anaphylactoid

reaction and in whom it appears that the agent responsible was protamine, I ask you to contact me.

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### Apnea and Syncope Following Intravenous Guanethidine Bier Block in the Same Patient on Two Different Occasions

*To the Editor:*—Intravenous guanethidine for the treatment of reflex sympathetic dystrophy (RSD) was

first introduced by Hannington-Kiff, based on the concept that outgrowing sprouts from damaged axons are

extremely sensitive to the alpha agonist actions of adrenaline.<sup>1</sup>

Guanethidine acts selectively on peripheral sympathetic nerves to depress the function of postganglionic sympathetic nerves by causing the release and subsequent depletion of norepinephrine from the nerve ending, as well as by inhibiting depolarization produced by nerve stimulation.

There has been one documented case of apnea and syncope following intravenous guanethidine reported by Martin *et al.*\* We report a case of apnea and syncope occurring twice in the same patient after each of two successive Bier blocks with guanethidine.

The patient is a 22-yr-old white female with RSD who had injured her right fourth finger about 2 yr prior to treatment. During the interim, she had undergone multiple surgical procedures, a series of six stellate ganglion blocks, and three Bier blocks with reserpine and steroids. All treatments resulted in temporary relief.

Physical examination of the patient was unremarkable except for her right fourth finger, which was fixed in the flexed position, and which was hypersensitive to touch. The physical examination was consistent with RSD; however, we felt there may be a strong neuromatous component causing the dystrophic pain.

Blood pressure was 130/90, and the heart rate was 84. A 20-gauge iv was inserted in a vein in the dorsum of her right hand, and also in an antecubital vein in her left arm. A double cuff tourniquet was placed on the right upper arm and the arm was exsanguinated with an Esmarch bandage. The cuff was inflated to 300 mmHg. A solution of 15 mg of guanethidine plus 20 cc of 0.5% lidocaine was injected over a period of 60 s. After 20 min, the tourniquet was deflated slowly over a period of 5 min. Blood pressure was 120/70, and heart rate was 72. Vital signs were stable when she was taken to the recovery room, where, almost immediately, she became apneic, flushed, and unarousable. She was immediately ventilated with 100% oxygen. She remained apneic for approximately 60 s, and then spontaneous respirations returned. Ventilation was assisted for about 3 min after the spell. For approximately 90 min after the episode, she was confused and disoriented. There were no neurological sequelae.

Five days later, at the patient's request, because of the substantial pain relief she obtained from the block, she was given a second Bier block with guanethidine. Again, she developed apnea and syncope in the recovery room and was subsequently managed in the same way.

Guanethidine monosulfate (Ismelin®) is a selective inhibitor of the sympathetic nervous system and does not interfere with parasympathetic function. Intravenous doses of guanethidine suppress ganglionic transmission and nerve conduction. It does not cross the blood-brain barrier, but sedation has been reported with chronic parenteral use.

Even though the adverse reactions did not occur until approximately 10 min after the tourniquet was deflated, the most likely explanation for her apneic episodes is central nervous system sensitivity to the drug itself, the precise mechanism for which is uncertain. Lidocaine also may elicit a central nervous system reaction manifested as excitation and/or depression. The total dose of lidocaine administered was 100 mg. This amount could conceivably cause syncope when given as a bolus. However, in a young healthy individual with no major cardiovascular problems, this is unlikely, especially with a 5–10 min delay between intravascular release and apnea.

The major benefit from a guanethidine Bier block compared to a local anesthetic block is the length of time that pain relief is obtained with guanethidine.<sup>2</sup> Even though guanethidine is an investigational drug, it has been used quite extensively for a number of years. The known side effects and adverse reactions are, in general, preventable. We recommend that it not be used in patients with extensive cardiovascular or respiratory diseases, patients known to be allergic to the drug itself, and in patients with pheochromocytoma. Along with an intravenous catheter, all patients should be monitored with an electrocardiogram, a pulse oximeter, and a blood pressure cuff, and equipment required for emergency airway management should be immediately available.

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