higher dosage and at the end of anesthesia.[12] In addition to its action on the chemoreceptor trigger zone, metoclopramide accelerates gastric emptying and increases lower esophageal sphincter tone, both of which should decrease the risk of developing aspiration pneumonitis. In a previous investigation utilizing a similar study population, we demonstrated decreased gastric volume following the iv injection of 10 mg of metoclopramide shortly before induction of anesthesia.[13] Despite the lack of antiemetic effect in the current investigation, patients did seem to benefit from metoclopramide by being able to sit, walk, and be ready for discharge earlier than other patients. Decreased postoperative dizziness also has been reported in previous studies[9,13] following administration of metoclopramide. The mechanism by which this agent accelerates recovery and ameliorates dizziness following anesthesia is unknown.

In summary, neither droperidol nor metoclopramide in the dose we employed was an effective antiemetic in this group of surgical outpatients. An increased dose of droperidol cannot be advocated in outpatients, because of its association with postoperative sedation and dizziness. Metoclopramide could be employed in larger doses, as it generally appears free from significant side effects. In these patients, in whom a narcotic technique is often selected in order to avoid the uterine relaxation that results from volatile inhalational agents, emetic stimuli may be too potent to reverse without causing undesirable side effects. Alternative anesthetic techniques utilizing diazepam or ketamine have proved less desirable for outpatient procedures. Despite the high incidence of emetic sequelae in our patients, discharge from the hospital was not delayed and no patient was incapacitated by prolonged nausea. Treatment of patients who experience protracted vomiting may be preferable to administering prophylactic antiemetics, with their potential adverse effects, to all patients.

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Anesthesiology
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Significant Sinus Bradycardia Following Intravenous Lidocaine Injection

ROXOLANA J. DEMCZUK, M.D.*

Lidocaine, when administered iv to suppress tracheal reflexes prior to endotracheal intubation,[1] usually has minimal cardiovascular effects. A case of sinus bradycardia associated with each administration of iv lidocaine is described below.

Report of a Case

A 79-year-old, 71-kg man, 4 weeks following an anterolateral myocardial infarction, was scheduled for insertion of an Austin Moore Prosthesis for his fractured hip. He had a history of left ventricular failure treated with digoxin and furosemide and ventricular ectopy controlled with procainamide. Preoperative 24-h ambulatory ECG monitoring (Holter monitor) and 12 lead electrocardiogram showed no evidence of sinus node disease. His serum digoxin level was 0.6

* Associate in Anesthesia.

Received from the Department of Anesthesia, Northwestern University Medical School, 303 East Superior Street, Room 360, Chicago, Illinois 60611. Accepted for publication June 17, 1983.

Address reprint requests to Dr. Demczuk.

Key words: Anesthetics, local: lidocaine. Complications: sinus bradycardia.

ng/dl. His serum potassium level was 4.1mEq/1. Procainamide was not given the morning of surgery. An arterial line and pulmonary artery catheter were inserted while the patient was awake. Following induction of anesthesia with iv diazepam and halothane, lidocaine 100 mg was given iv as a rapid bolus over 15 s in anticipation of endotracheal intubation. Thirty seconds later, heart rate fell precipitously from 92 to 60 bpm in normal sinus rhythm and the arterial blood pressure decreased from 160/90 to 80/50 mmHg. Following intubation of the trachea, which was facilitated by 80 mg succinylcholine iv, arterial blood pressure and heart rate rose only gradually to 140/80 mmHg and 90 bpm, respectively, and remained stable throughout the case. Upon arrival to the recovery room 150 min later, the patient was awake with an arterial blood pressure of 190/110 mmHg. His electrocardiogram showed ST segment depression and frequent ventricular premature beats. A 100-mg iv bolus of lidocaine was administered. Within a minute, the heart rate fell from 98 to 52 bpm accompanied by a decrease in systolic blood pressure to 76 mmHg. Sinus bradycardia was maintained. After 2–3 min and iv crystalloid administration, heart rate and blood pressure rose. A lidocaine infusion was initiated to treat new ventricular ectopy in the face of ST depression. When 50 mg had been given, heart rate again decreased to 47 bpm. The infusion was discontinued. The patient was observed in the intensive care unit where procainamide therapy was resumed, a myocardial infarction was ruled out, and he had no recurrence of bradycardia.

Discussion

Lidocaine can depress the sinus node.[2–8] Anesthesiologists frequently use lidocaine iv to depress laryngeal reflexes as well as to treat arrhythmias. Animal experiments suggest lidocaine suppresses sinus node activity only at toxic serum levels.[9] Certainly, conditions such as low cardiac outputs (shock) or congestive heart failure call for reducing the dose of lidocaine for fear of attaining toxic levels with standard doses.

Our patient had no clinical evidence of the above mentioned problems at the time of this operation. However, Roos[10] found serum levels to rise well above toxic even in normal subjects who received rapid (administered over less than 20 s) injections of therapeutic doses (1–1.5 $mg \cdot kg^{-1}$) of lidocaine. The levels peaked as high as 26.6 $g \cdot ml^{-1}$ at 60 s. Indeed, most of the reported cases exhibited bradycardia within 180 s from the start of a rapid injection, as did the patient in our report.

Interaction of lidocaine with high serum levels of digoxin may have played a role in suppressing the sinus node in some of the previously reported cases. Digitalis itself can cause sinus bradycardia or complete sinoatrial arrest when serum levels are excessively high by its direct and indirect effects. This patient had a normal serum digoxin and potassium level.

Thus lidocaine should be used judiciously, administered slowly, and dosages adjusted in the elderly in combination with other myocardial depressant drugs. Also, patients with preexisting sinus node disease, low cardiac outputs, congestive heart failure, and with fixed stroke volumes (rate dependent cardiac outputs) may be susceptible to lidocaine-induced bradycardia. Lidocaine suppression of the sinus node may result in complete asystole, as the drug also suppresses the latent nodal and ventricular pacemakers.

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