Anesthesiology
48:155, 1978

Canadian Standards for Piped Gases

To the Editor:—Regarding the final paragraph of the article by Eichhorn *et al.*,[1] we would call attention to the recently approved revisions to Canadian Standards Association Standard Z305.1-1975, which include contaminant limits. There are 14 contaminants listed in the table, and limits are given for medical air, medical oxygen, and nitrous oxide. The revisions provide for testing the "source" only when the sample from the outlet exceeds the limits, and defines for testing purposes the point in the system where the "source" begins (looking from the outlet). The revisions also reference a new CSA Standard Z305.4, "Qualification Requirements for Agencies Testing Non-flammable Medical Gas Piping Systems." Readers who are interested in the details of the revisions should note that holders of Standard Z305.1 who have returned their coupons will be automatically advised when the revisions have been published. Orders for either of the Standards should be directed to the CSA Standards Sales Division, 178 Rexdale Boulevard, Rexdale, Ontario, Canada, M9W 1R3.

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(Accepted for publication September 21, 1977.)

Anesthesiology
48:155, 1978

Ketamine and Angina

To the Editor:—The article by Balagot *et al.*[1] attributed the postoperative occurrence of Prinzmetal's variant angina to: 1) failure to take nitroglycerin prior to the operation; 2) omission of atropine as part of the preoperative medication; and 3) inadequate spinal anesthesia. Although these factors were possibly contributory to the exacerbation of the condition, they still do not adequately explain the precipitating stress. The authors do not report the patient's vital signs after ketamine was given, nor do they consider that ketamine, with its inotropic and chronotropic effects,[2] may have precipitated an acute increase in myocardial oxygen consumption, with resultant ischemia. One should, in the case of a patient with known angina pectoris, be circumspect about the use of ketamine because of its known effects on increasing the determinants of myocardial oxygen consumption.

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(Accepted for publication August 11, 1977.)

Anesthesiology
48:155–156, 1978

To the Editor:—Dr. Balagot and his co-authors are to be complemented for their concise review of the pathophysiology of Prinzmetal's variant angina.[1] Their report calls attention to this disorder as one of the causes of ST segment elevation in the recovery room. Unfortunately, the brief presentation and analysis lack some important details. Elucidation of the patient's history of angina would have been helpful.

Was it suggestive of variant angina? What did his preoperative electrocardiogram show? Did nitroglycerine relieve the angina?

The sequence of anesthetic events also needs elaboration. Spinal anesthesia seemed to be an appropriate choice, provided that the patient could tolerate this without undue anxiety. The inclusion of "inadequate spinal anesthesia" among the possible causes of the postoperative ECG changes was certainly warranted, but the reasons for conversion to general anesthesia were unclear. Was the patient too apprehensive to permit a repeat spinal anesthetic? Tetracaine could have been injected into the subarachnoid space using a hypobaric technique, thereby not taking the patient out of the jackknife position.

General anesthesia consisting of ketamine, diazepam, nitrous oxide, and oxygen (by face mask?) was presumably selected in an effort to provide analgesia and amnesia without jeopardizing ventilation with the patient in the prone position. Ketamine, however, increases myocardial oxygen demand. It should be used with caution, if at all, in patients with coronary insufficiency. The increased oxygen demand is attributed to inotropic and chronotropic effects, and possibly to an increased left ventricular afterload.[2] Although the mechanism of these ketamine-induced changes remains controversial, a centrally mediated sympathetic response has been suggested.[3,4] If the pathophysiologic changes reviewed by the authors are correct, an alpha-adrenergic stimulus could precipitate an attack of variant angina. It would have been helpful to know how the patient's blood pressure and pulse responded to the incremental doses of ketamine. Indication as to which ECG lead was being monitored in the operating room would also have been useful information.

In summary, ketamine should be added to the authors' differential diagnosis of the cause of this patient's ECG changes. If general anesthesia had been/were elected, a technique that would have provided/would provide analgesia and anesthesia without increasing myocardial oxygen demand would probably have been preferable. These objectives could have been achieved by any of several face-mask techniques, if the operation had been performed with the patient in the lithotomy position.

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(Accepted for publication October 10, 1977.)

Anesthesiology
48:156, 1978

In reply.—Dr. Guerra's and Dr. Gravlee's suggestion that ketamine may have increased myocardial oxygen demand and contributed to the development of angina is certainly possible. However, the patient subsequently underwent a right pneumonectomy with removal of hilar and mediastinal lymph nodes for cancer. Anesthesia for this operation consisted of diazepam, 10 mg, for induction, succinylcholine, 80 mg, preceded by dimethyltubocurarine, 4 mg, to facilitate endotracheal intubation, and ketamine, 400 mg (total dose), and nitrous oxide-oxygen 50 per cent each for maintenance. ST elevation in the inferior leads developed postoperatively. It quickly subsided and was diagnosed as early depolarization rather than angina or myocardial infarction.

A repeat spinal anesthetic with hypobaric tetracaine would have mitigated the inadequate spinal anesthesia and would have been my initial choice. Unfortunately, I happened to be busy in another room when the spinal anesthetic was found to be unsatisfactory. There was no change in blood pressure and pulse rate (slight tachycardia before ketamine), as monitored with a standard EKG lead II. Use of the lithotomy position as suggested, and use of other agents that do not increase myocardial oxygen demand, might have obviated the variant angina.

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(Accepted for publication October 10, 1977.)