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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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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