

Emergency Closed Mitral Commissurotomy Using Ketamine Anesthesia: Report of a Case

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It is often necessary to perform emergency cardiac operations in patients in cardiogenic shock. Most such patients will benefit from the use of partial cardiopulmonary bypass instituted under local anesthesia. However, there are still patients for whom immediate closed cardiac surgical procedures are the best treatment. In these cases the proper selection and judicious administration of general anesthesia are of obvious importance.

This report describes the use of ketamine anesthesia in a moribund patient in profound cardiogenic shock secondary to far-advanced mitral stenosis who underwent closed mitral commissurotomy.

REPORT OF A CASE

A 41-year-old man who had been found unconscious was admitted to Harbor General Hospital on March 11, 1971. The patient was known to have had chronic pulmonary disease since childhood. A month prior to admission, the murmur of mitral stenosis had been detected, but the patient had refused further evaluation of his cardiac status. Previous pulmonary function studies had been compatible with severe obstructive pulmonary disease which improved with sublingual administration of isoproterenol, Quibron and expectorants. However, management of the case had been complicated by the patient's chronic intravenous use of heroin.

On physical examination the patient was unconscious, with a blood pressure of 75/30 mm Hg, a regular pulse of 140 beats/min, and a respira-

tory rate of 8/min. The chest was hyperresonant to percussion, with diffuse inspiratory and expiratory wheezes, rhonchi and bilateral basilar rales. The heart did not appear enlarged. The first heart sound was accentuated, but no diastolic murmur of mitral stenosis was heard. The abdomen was slightly distended and the liver was enlarged 5 cm below the right costal margin. Pitting edema extended to the mid-thighs bilaterally, and there was marked distention of neck veins at 30 degrees of elevation. Pertinent laboratory data revealed: BUN 65 mg/100 ml; serum Na 117 mEq/l; K 5.2 mEq/l; Cl 71 mEq/l; CO₂ 25 mEq/l. Arterial blood-gas values were: P_{O₂} 46 mm Hg; P_{CO₂} 80 mm Hg; pH 7.37. The remainder of the laboratory data were essentially normal. A chest x-ray was compatible with severe pulmonary edema.

The patient was treated with furosemide, iv, aminophylline and corticosteroids. In spite of controlled ventilation, blood gases and clinical status failed to improve. Twelve hours after admission, the patient underwent emergency combined right and transeptal left heart catheterization, which disclosed severe calcific mitral stenosis with a mean left atrial pressure of 38 mm Hg and a left atrial-left ventricular mean diastolic pressure gradient of 32 mm Hg. Because of the patient's failure to respond to treatment, it was decided to undertake immediate closed mitral commissurotomy. Despite preoperative continuous intravenous administration of isoproterenol and metaraminol, the patient had a blood pressure of 40/0 mm Hg and a normal sinus rhythm at a rate of 90 beats/min upon arrival in the operating room. He was semiconscious and responded to painful stimuli, which precluded immediate endotracheal intubation.

With the patient under local anesthesia, an arterial pressure line was placed in the right radial artery, a central venous pressure catheter was inserted in the right greater saphenous vein, and the electrocardiogram was placed to monitor lead I continuously during the surgical procedure. A rectal temperature probe was used to monitor the patient's temperature continuously.

Following intravenous administration of 150 mg of ketamine (2.0 mg/kg), endotracheal intubation was accomplished without difficulty. Respirations were controlled throughout the procedure using

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100 per cent oxygen. Intraoperative blood gases were not obtained. Fifteen mg of *d*-tubocurarine were administered over a 5-minute period. Ten minutes after the initial dose of ketamine, an additional 150 mg were injected intravenously. Additional doses of ketamine were given intravenously whenever the patient responded to the operative procedure. At the time of the left posterior lateral thoracotomy, blood pressure was 80/40 mm Hg, with a pulse rate of 60 beats/min. Vital signs remained unchanged until the commissurotomy was performed.

During the initial phases of the surgical procedure, an additional 200 mg of ketamine were given. Surgical manipulation of the heart resulted in multiple premature ventricular contractions which were controlled with 100 mg xylocaine, iv. Serum potassium was 4.8 mEq/l at this time, and the patient had not received any digitalis during the course of his illness. Following closed mitral commissurotomy, blood pressure rose to 110/60 mm Hg and pulse to 100 beats/min. Sinus rhythm remained normal, and all pressor and inotropic agents were discontinued. An additional 300 mg of ketamine and 12 mg of *d*-tubocurarine were given during the remainder of the surgical procedure. The total anesthesia time was 190 minutes.

The patient left the operating room with an endotracheal tube *in situ*, with assisted respiration, a blood pressure of 110/80 mm Hg, and a pulse rate of 90 beats/min. Postoperative serum electrolytes were within normal limits, and analysis of arterial blood obtained upon arrival in the intensive care area revealed P_{O_2} 190 mm Hg, P_{CO_2} 39 mm Hg, pH 7.58, and BE +13. The patient's postoperative course was uneventful, and he was discharged from the hospital on the fourteenth postoperative day.

COMMENT

General anesthetics may depress cardiovascular activity and cause cardiac arrhythmias. Patients undergoing emergency open-heart surgery with or without cardiopulmonary bypass have little cardiac reserve, and are unable to tolerate further depression of myocardial function. The use of ketamine may be particularly suitable for these desperately ill patients. Ketamine produces moderate to marked elevation of both systolic and diastolic blood pressures.¹ The reports of Virtue and associates² and Kreuzer *et al.*³ suggest that this elevation of blood pressure may result from an increased release of endogenous catecholamines which increases cardiac output while peripheral vascular resistance either remains un-

changed or decreases. Dowdy and Kaya^{4,5} have found evidence in laboratory animals suggesting that the pressor effect of ketamine may be the result of desensitization of the arterial baroreceptors. The antiarrhythmic effects of ketamine are thought to be due to a direct effect on the myocardium.⁴⁻⁶

The patient reported here remained hypotensive prior to completion of the mitral commissurotomy. Although anesthesia was initiated at a time when inotropic and pressor stimuli were maximal and cardiac response minimal, ketamine anesthesia did provide a suitable operative field, the opportunity to oxygenate the patient, and a reasonably stable hemodynamic state. Following completion of commissurotomy, the patient's hemodynamic condition was satisfactory. During the procedure he did not develop hypotension or cardiac arrhythmias. Based on this experience, it is felt that consideration should be given to the use of ketamine anesthesia for the hemodynamically unstable patient who needs emergency cardiac surgery.

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