control values (table 1). It is probable that sustained contracture of extraocular muscles is responsible for this initial increase in intraocular pressure.

The subsequent full dose of SCf and endotracheal intubation did not increase intraocular pressure. There have been reports that intraocular pressure increases after endotracheal intubation.\textsuperscript{11,12} Straining and coughing during endotracheal intubation, which could contribute to the increase in intraocular pressure, were not seen.

Glaucomatous patients showed comparatively large increases in intraocular pressure in response to pretreatment doses, suggesting that this technique should not be used for such patients.

Surprisingly, the problem of post-SCf myalgia did not arise. It is difficult to say whether the protective effect against SCf-induced myalgia resulted from the pretreatment dose of diazepam or other factors. It has been reported that even 5 mg SCf may produce myalgia,\textsuperscript{13} so perhaps diazepam was responsible for its prevention.

In conclusion, pretreatment with a small subparalytic dose of SCf before administration of the full dose prevents muscle fasciculations and increases in intraocular pressure. It is concluded that this sequence of anestheisa can safely be used in intraocular or perforating ophthalmic operations.

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Use of a Left Atrial Pressure Monitor to Diagnose a Malfunctioning Mitral Valve Prosthesis

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Intra- and postoperative monitoring of left atrial pressure (LAP) is routine for all cardiac surgical procedures at our center.\textsuperscript{1} The following case demonstrates the value of left atrial pressure monitoring to detect faulty prosthetic valve performance after mitral valve replacement.

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REPORT OF A CASE

A 61-year-old white male needed anesthesia for replacement of his mitral valve. He had had rheumatic fever at the age of 6 years, but had been asymptomatic until five months prior to admission. At that time he experienced atrial fibrillation and congestive cardiac failure that progressed despite optimal medical management with digoxin and furosemide. The patient was admitted with the diagnosis of mitral insufficiency.

The patient was anesthetized with diazepam, pancuronium, N2O/O2, and fentanyl. The anesthetic course prior to cardiopulmonary bypass was uneventful. Intravenous administration of nitroprusside was necessary to maintain mean left atrial pressure (LAP) at ≤20 torr. The mitral valve was replaced with a 21-mm Björk-Shiley prosthesis without difficulty during 38 minutes of ischemic cold cardioplegic arrest. Myocardial performance...
was initially satisfactory following cardiopulmonary bypass with a systolic pressure of 100 torr and LAP 15 torr. Ten minutes after termination of bypass systemic pressure decreased to 40 torr with LAP 35–50 torr (fig. 1). The surgeon was immediately informed that the mitral valve prosthesis apparently was malfunctioning. Palpation of the left atrium confirmed the diagnosis. Repeated manual manipulation was necessary to relieve the intermittent mitral valve prosthesis obstruction. During the 30-min interval prior to reestablishing cardiopulmonary bypass cardiac output gradually deteriorated, with one episode of ventricular fibrillation that necessitated electrical defibrillation.

Examination of the prosthesis during bypass failed to disclose the cause of intermittent obstruction. A 29-mm Björk-Shiley valve was placed during 36 minutes of ischemic cold cardioplegic arrest and the disc occluder oriented differently to minimize potential contact with the thickened myocardium. Cardiopulmonary bypass was discontinued. The patients' hemodynamic status remained satisfactory, with LAP 10–15 torr and systolic pressures ranging from 90 to 120 torr.

The following morning the patient was neurologically normal, cardiac index 2.2 l/min, and the trachea was extubated. The only postoperative complications were a febrile episode on the fourth postoperative day and recurrence of atrial fibrillation. He was discharged in satisfactory condition on the ninth postoperative day.

**Discussion**

The purpose of monitoring devices is the accumulation and presentation of important physiologic data, which, when properly interpreted, warn, advise and instruct the physician. LAP provides information regarding filling pressure, myocardial function, and valve performance of the left side of the heart. Techniques of placement during cardiac surgical procedures have been previously described.

This case illustrates the importance of continuous monitoring of left atrial pressure during mitral valve operations. The data from the LAP catheter facilitated the diagnosis of a malfunctioning mitral valve prosthesis. Hypotension following replacement of the mitral valve is not uncommon, and when associated with an elevated LAP it usually is the result of decreased cardiac output or excessive volume of infusion. However, it was apparent from both the arterial pulse contour and the left atrial tracing that this case was not a common one of hypotension resulting from pump failure, but rather, the systemic hypotension resulted from intermittent mechanical failure of the prosthetic valve mechanism. With each beat there is an increase in LAP with progressive systemic hypotension. The hemodynamic observations resulted from failure of the left atrium to empty, causing a progressive increase in left atrial volume, and therefore, pressure. The left ventricle, in turn, received little or no volume from the left atrium, and with each contraction ejected a progressively smaller stroke volume, causing hypotension. The valve occluder intermittently obstructed until it was manually opened or enough intramural pressure forced it open. Two possible alternative causes for similar hemodynamic disturbances could be arrhythmia or manipulation of the heart; both were easily ruled out in this case by the persistent normal sinus rhythm on the EKG and by observation of the surgical field.

Malfunction of the mitral valve prosthesis is a rare complication that usually necessitates immediate valve
replacement to restore adequate cardiac output. Had the cause of the hemodynamic disturbances not been immediately detected, it is doubtful that our patient would have survived.

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Cardiac Tamponade from Central Venous Catheters

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At least 34 cases of cardiac tamponade resulting from the use of central venous catheters, 78 per cent of them fatal, have been reported since March 1968.¹ We know of another 11 fatal cases that have not been reported in the medical literature, and we suspect, as do others,²–⁴ that such accidents often are not reported. Below we review the 34 reported cases. In many of them, the causes, symptoms, clinical courses, and pathologic findings were similar; we have summarized these common features. All investigators agree that a few simple precautions can prevent perforation by catheters or greatly reduce its incidence. We present their suggestions, as well as measures that they found successful in treating tamponade. It is our impression that many of the reported deaths could have been avoided, had the perforations and incipient tamponades been diagnosed more promptly.

CLINICAL COURSE

Of the 34 patients whose cases were reported, 19 had brachial venous catheters, 12 had subclavian-vein catheters, and three had external jugular-vein catheters. Sixteen of the 19 brachial venous catheters were long cannulas of rigid polyethylene with sharp tips inserted through venous cutdowns. The 18 other cannulas (three brachial and 15 jugular or subclavian) were tubings made of nylon or Silastic®, or commercial units of Teflon®, soft polyethylene, or polyvinylchloride.

Ten patients in whose cases autopsy showed myocardial perforation as the probable cause of death had died without apparent symptoms of tamponade. Four were found dead in bed,⁵–⁷ one died during anesthesia,⁸ and five conscious subjects experienced sudden cardiac arrest minutes to hours after cannulation.⁹–¹⁰ The other 24 patients had uneventful periods of hours or days (as long as eight days¹¹¹) from insertion of the catheter to onset of tamponade. These patients experienced sudden onset of cyanosis and venous engorgement of the face and neck, complained of nausea, dyspnea, retrosternal or epigastric pain, and became restless, confused, or comatose. Minutes to hours after the appearance of those early symptoms, tachycardia, arterial hypotension, paradoxical pulse, muffled heart sounds, and oliguria developed. The vital signs of those patients with untreated tamponade continued to deteriorate for as long as four hours until cardiac arrest occurred.

DIAGNOSIS

Several patients¹,⁵,⁸,¹²–¹⁰ suddenly manifested abnormally high central venous pressures (CVP) loss of respiratory fluctuations, and absence of venous backflow, but those signs were overlooked because of the absence of physical signs. Normal venous pressure and respiratory oscillations, however, persisted in one patient who had subclavian venous perforation and hydromediastinum.¹⁷ In only five cases in which pericardial effusion was suspected was the diagnosis confirmed by low electrocardiographic voltages,¹⁶,²⁹ enlarged cardiac outlines on roentgenograms,¹⁶,¹⁷,²⁹ or on echocardiograms,¹⁷ or injection of dye into the central venous catheter.¹⁶,¹⁰ The relevant medical literature thus shows that in spite of