combination of impedance phlebography and radiofibrinogen leg scanning is reported to be of comparable sensitivity to invasive contrast venography.[9]

The application of non-invasive techniques in the early diagnosis of DVT has demonstrated the prophylactic value of low-dose heparin given subcutaneously. There is little alteration of clotting time and partial thromboplastin time, and little hemorrhagic hazard when administered in a dose of 5,000 units twice daily.[9] At present, the use of platelet function inhibitors such as aspirin and dipyridamole remains unclear.[10] Dextran, which also impairs platelet aggregation, is an alternative to low-dose heparin, but Smith *et al.*[11] have reported postoperative bleeding and allergic reactions, and it must be used with caution in patients with limited cardiovascular reserve due to the danger of fluid overload.[10]

In conclusion, we suggest that if delayed internal fixation requiring limb exsanguination is performed, a high index of suspicion for the presence of deep vein thrombus must be maintained. Active prophylaxis against the development of venous thrombosis should therefore be considered.

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Intravenous Verapamil to Relieve Pulmonary Congestion in Patients with Mitral Valve Disease

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In mitral valve disease, the development of pulmonary congestion is dependent on factors increasing resistance to flow in the left atrium, notably tachycardia (usually as the result of rapid ventricular response to atrial fibrillation), which shortens diastole during which the left atrium must be emptied.[1] Drugs that prolong AV nodal conduction time have been used in patients with atrial fibrillation to slow a fast ventricular response, *e.g.*, digitalis, propranolol, guinidine, and procainamide.

Cells in the AV node are probably slow-channel-dependent, and AV nodal conduction can be slowed by drugs that interfere with the slow inward current.[2,3] Verapamil (a calcium channel blocker) is effective in terminating supraventricular tachycardia or slowing the ventricular rate during atrial fibrillation.[3,4]

In two patients with mitral valve disease and pulmonary congestion due to the rapid ventricular response to atrial fibrillation, we administered verapamil

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(0.75 mg/kg), iv. In less than 3 min, the heart rate decreased substantially with concomitant hemodynamic and clinical improvement.

Reports of Two Cases

Case 1

A 56-year-old woman was hospitalized because of shortness of breath and a heart murmur. Congestive heart failure was diagnosed ten months ago and improved on medical therapy. However, she recently has increasing limitation of activity and shortness of breath on exertion. Cardiac catheterization revealed a moderately severe mitral stenosis (valve area, 0.667 cm^2) with mild insufficiency associated with a normal left ventricular systolic and left ventricular end-diastolic pressures with a marked gradient across the mitral valve (30 mmHg). The right ventricular and pulmonary artery systolic pressure (PAP) were 48–50 mmHg, and pulmonary capillary wedge pressure (PCWP) was 29 mmHg. Surgery for a mitral valve replacement was scheduled. She received maintenance doses of digitalis because of atrial fibrillation (0.125 mg daily with serum digoxin level 1.0 ng/ml). She was neither on propanolol nor quinidine.

Ten milligrams morphine sulfate and 0.4 mg scopolamine, im, were given one hour prior to arrival in the operating room when she was in acute respiratory distress associated with shortness of breath and wheezing. A radial artery cannula and a pulmonary artery catheter were inserted under local anesthesia. Arterial blood pressure was 95/70 mmHg and heart rate was 190 beats/min because of rapid response to atrial fibrillation. PAP was 60 mmHg and PCWP was 35 mmHg. Central venous pressure (CVP) was 7 mmHg.

Five milligrams verapamil were given iv over a period of 1 min to reduce the heart rate. In less than 3 min, the heart rate decreased from 190 to 75 beats/min. Arterial blood pressure rose from 95/70 to 120/80 mmHg. Cardiac output increased from 3.1 to 4.0 l/min. Anesthesia then was induced and maintained with iv administered fentanyl (100 μg/kg total). After induction of anesthesia, the heart rate further decreased to 65 beats/min. No other clinically significant changes occurred before cardiopulmonary bypass was initiated.

Case 2

A 65-year-old woman was hospitalized because of shortness of breath, palpitation, and a heart murmur. A ventriculogram revealed severe mitral regurgitation associated with moderate elevation of PCWP (14 mmHg). PAP was 28 mmHg and CVP was 13 mmHg. Arterial blood pressure was 130/60 mmHg and ventricular heart rate was varying between 60 and 130 beats/min because of atrial fibrillation. She had been taking 0.125 mg digoxin daily with a serum digoxin level of 0.9 ng/ml. She was receiving neither propanolol nor quinidine. She was scheduled for a mitral valve replacement.

Seven milligrams morphine and 0.4 mg scopolamine were given im one hour before induction of anesthesia. During the insertion of a radial artery cannula, the patient started coughing and wheezing. The heart rate was 180 beats/min. A triple-lumen thermodilution pulmonary artery catheter was inserted under local anesthesia. The PCWP was 37 mmHg with a huge V-Wave, PAP was 70 mmHg, and CVP was 13 mmHg. The arterial blood pressure was 90/70 mmHg with irregular beats typical for atrial fibrillation. Cardiac output was 2.4 l/min. Five milligrams verapamil were given iv over a period of 2 min. Within 2 min, the heart rate decreased from 180 to 65 beats/min. Arterial blood pressure rose from 90/70 to 110/70 mmHg. PAP decreased from 70 to 35 mmHg and PCWP decreased from 37 to 22 mmHg. The CVP did not change. Cardiac output increased slightly from 2.4 to 3 l/min. Anesthesia then was induced and maintained with iv fentanyl (100 μg/kg). The remainder of anesthesia and surgery proceeded without difficulty.

Discussion

Patients with mitral valve disease usually develop atrial fibrillation (about 43%).[5] When atrial fibrillation is associated with a slow ventricular rate, many patients are hemodynamically undisturbed by its presence. However, in some patients, a slow ventricular rate is often difficult to maintain despite an appropriate drug regimen with such drugs as digoxin or procainamide. Pulmonary congestion is due to the increased resistance to flow in the left atrium,[1] usually precipitated by tachycardia, which shortens diastole.

In patients with mitral regurgitation, augmentation of the blood flow from the left atrium into the left ventricle is facilitated by bradycardia[6] with prolongation of diastole. Conversely, compensation for mitral regurgitation may be impaired seriously by tachycardia[6] and shortening of the diastolic period. Therefore, tachycardia plays an important role in the development of pulmonary congestion in both mitral stenosis and regurgitation. Quinidine and digitalis have long been used either to restore sinus rhythm or to slow the AV conduction period to prevent a rapid ventricular response. Nifedipine (another calcium channel blocker) has been shown to be an effective afterload and preload reducing agent in pulmonary edema due to left ventricular failure.[7] Its main action, however, is vasodilation without altering AV conduction.[7] Nitrates also are effective for the treatment of pulmonary edema in patients with valvular heart disease.[8,9] However, neither nitrates nor nifedipine have any effect on supraventricular tachycardia or AV conduction. Verapamil, on the other hand, has the unique capability of treating pulmonary congestion by reducing the heart rate.[3,4]

Side effects, such as hypotension, bradycardia, and A-V block, may occur rarely and they usually are related to overdose.[10] Because of its tendency to produce A-V block, verapamil should be used cautiously in patients in whom A-V block is apt to develop[10] (patients taking digitalis) and should not be administered to patients treated with beta-adrenergic blocking agents.[10,11] Direct effect on vascular smooth muscle induced by calcium antagonists can be reversed by addition of calcium.[12] Further experience obviously is needed since our series of two patients was too small to draw any definite conclusions.

In our patients with mitral stenosis and regurgitation, pulmonary congestion was initiated by a rapid ventricular response to atrial fibrillation. We believe that the use of verapamil may be a valuable addition to existing drug regimens in relieving pulmonary congestion in

patients with mitral valve disease initiated by supraventricular tachycardia.

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The Influence of Endotracheal Tube Cuff Design and Cuff Lubrication on Postoperative Sore Throat

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Sore throat, while usually not an incapacitating postanesthetic problem, can be an uncomfortable one. Endotracheal tube cuff design, cleaning agents used on non-disposable endotracheal tubes, endotracheal tube lubricants (used on non-cuffed endotracheal tubes), and several other factors affect the incidence and severity of postoperative sore throat.[1–8] In this study, we evaluated the interaction of endotracheal tube cuff design and cuff lubrication on the incidence and severity of postoperative sore throat in 600 patients after anesthesia and surgery.

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Methods

The investigation was approved by the Human Research Committee. Informed consent to perform the study was obtained from 600 patients scheduled to undergo elective orthopedic and gynecologic operations at the time of the preoperative visit. Three standard commercially available endotracheal tubes and a new double contour cuffed tube were studied (fig. 1). The latter tube, provided by Ohio Medical Products, Inc., was designed to provide a large cuff volume, low intracuff pressure, and minimal tracheal-cuff contact area. The other tubes studied included National Catheter Company Lo Pro and Hilo Tubes and Portex Taper Cuff tubes.

Tube sizes varied from 7.0–8.0 ID. One hundred and fifty each of the four kinds of tubes were studied. The tubes were divided further into three groups of 50 each according to the lubricant used on the cuff. Tubes and cuffs were lubricated with 5% lidocaine ointment§

§ Astra, Inc., Worcester, Massachusetts (5% xylocaine in polyethyl glycol and propylene glycol; $pH = 5.5$).