Increased Mitral Valve Insufficiency during Precordial “Whoop”**

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A patient with rheumatic mitral valve stenosis and regurgitation presented with shortness of breath and a “noise” in her chest. Cardiac auscultation revealed an intermittent late systolic “whoop.” An increase in severity of mitral valve insufficiency during the periods of “whoop” was observed by pulsed Doppler, hemodynamic tracings and left ventriculography.

Precordial “whoop” or “honk” is a high-pitched musical sound reminiscent of the “whoop” of pertussis or the honking sound of a goose. These sounds vary in intensity with respiration or body position and have been associated with mitral and tricuspid valve insufficiency, mitral valve prolapse, rheumatic mitral stenosis, left-sided pneumothorax, pacemaker catheter obstructing tricuspid valve closure and in patients without underlying heart disease. We present a patient with mild rheumatic mitral stenosis and insufficiency who had a marked increase in the severity of valve insufficiency during periods of precordial “whoop.” This was confirmed by pulsed Doppler, pulmonary capillary wedge tracings and left ventriculographic studies performed before and during the precordial “whoop.” Transient increase in severity of mitral insufficiency during periods of systolic whoop has not been observed before.

CASE REPORT

A 66-year-old white woman presented with increasing dyspnea on exertion and noted a peculiar sensation or noise in her chest when she turned to her left side. She had a history of hypertension and noninsulin-dependent diabetes mellitus, but no history of rheumatic fever.

Physical examination revealed no jugular venous distention. Cardiac auscultation in the supine position revealed normal first heart sound and loud pulmonary component of the second heart sound. A grade 2/6 holosystolic and a grade 1/6 diastolic rumble were heard at the apex. On assuming a left lateral posture, the patient produced symptoms of a “noise” in her chest. At that time, a grade 6/6 late systolic musical sound was observed. This disappeared as she assumed the supine position.

Electrocardiogram revealed normal sinus rhythm and evidence of left atrial enlargement. Phono- and M-mode echocardiogram performed simultaneously revealed a holosystolic murmur at the apex. On turning on her left side, a late systolic “whoop” was recorded (Fig 1). M-mode and two-dimensional echocardiography demonstrated a thickened and mildly stenotic mitral valve and an enlarged left atrium. Pulsed Doppler test performed when her chest was quiet revealed mild mitral insufficiency; during periods of precordial “whoop,” severe valve insufficiency was demonstrated by flow mapping techniques. Cardiac catheterization revealed normal resting right heart pressures. The pulmonary capillary wedge V wave increased from 22 mm Hg to 55 mm Hg during periods of “whoop.” The mitral valve area was calculated to be 1.6 cm². Left ventricular angiographic study performed at rest revealed mild mitral insufficiency; angiography repeated during precordial “whoop” revealed severe valve insufficiency (Fig 2). The left ventricular wall motion and coronary arteries were normal.

The patient underwent mitral valve replacement surgery. On gross examination, the valve was thickened and calcified with intact chordae tendineae. Microscopically, the excised valve had features consistent with chronic rheumatic disease process. Since surgery, her exercise tolerance has improved, and she no longer experiences the peculiar sensation or noise in her chest.

DISCUSSION

Precordial whoop was first thought to be an extracardiac sound, but subsequently has been shown to emanate from the mitral and tricuspid valve leaflets and chordae tendineae when they are set into vibrations at resonant frequency. Intermittent increase in severity of mitral insufficiency during periods of precordial whoop has not been reported previously. In our patient, this phenomenon was well documented by pulsed Doppler, pulmonary capillary V wave tracing and left ventriculographic images. The etiology and mechanism of increase in severity of valve insufficiency during periods of precordial whoop is not clear. Perhaps the rheumatic mitral valve prolapsed more in the left lateral position with severe insufficiency. Intermittent increase in severity of mitral insufficiency has been described in patients with a dilated poorly functioning left ventricle or during transient papillary muscle dysfunction. Our patient had normal left ventricular function and had no angiographic

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FIGURE 1. Phono- and M-mode echocardiogram of the patient before (upper panel) and during precordial “whoop” (lower panel). Note also thickened stenotic features of the mitral valve. MV = mitral valve; LV = left ventricle; RV = right ventricle; S = intraventricular septum.
Gas Flow Through a Bronchopleural Fistula*

Measuring the Effects of High-frequency Jet Ventilation and Chest-tube Suction

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High-frequency jet ventilation (HFJV) is FDA-approved for ventilating patients with bronchopleural fistulae (BPF), yet little is known about its effect on the fistula airleak. We quantitated a patient's BPF airleak during both conventional volume-cycled ventilation and HFJV. The effect of chest-tube suction (CTS) on BPF flow was also studied. Despite a significant reduction in peak airway pressure, the HFJV resulted in a 50-70 percent increase in BPF flow. CTS also significantly increased the airleak. HFJV may not always be the preferential method for ventilating patients with BPF and we recommend measuring the fistula airleak when attempting to optimize a patient's ventilatory parameters.

There is a paucity of information concerning the role of different ventilators and ventilatory parameters in managing persistent bronchopleural fistulae (BPF). Treatment is directed at decreasing the BPF leak in order to support ventilation of the intact lung, prevent pleural space infection and promote healing of the ruptured tissue.1,5 This goal can be approached in numerous ways, including: the application of local measures to seal the leak,2,6 the use of positive chest-tube pressure to counteract the gradient for flow,1,3 differential lung ventilation,7 and definitive surgical closure.8,9 Unfortunately, each of these measures requires technical skill, equipment and patient characteristics which are rarely available in the clinical setting. With the recent availability of high-frequency jet ventilation (HFJV), there has been growing enthusiasm for its use in the treatment of BPF.5,10,11 This report focuses on comparing the gas flow through a patient's BPF during conventional volume-cycled ventilation (VCV) and HFJV. Further, we describe the role of chest-tube suction (CTS) in promoting BPF flow and its ability to alter HFJV performance.

**CASE REPORT**

The patient is a 44-year-old man hospitalized for treatment of *Staphylococcus aureus* endocarditis complicated by septic emboli, ARDS, recurrent hemoptysis, tension pneumothorax, empyema and a persistent BPF. Long-term ventilation was provided through an 8.0 mm cuffed tracheostomy tube. Chest tubes were connected to a waterseal drainage system. We initially ventilated the patient with a Bear II ventilator in the assist-control (AC) mode and various manipulations were attempted in an effort to decrease the high peak airway pressures (PAP). First, the tidal volume (VT) was lowered from 700 to 500 ml, but the PAP was still 50 cmH2O. Next, we sedated the

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