of the leaflet connection to each of the mitral leaflets. However, as observed at the operating table, there was prolapse of both leaflets rather than flail leaflets.

We suggest that, despite chordae rupture, the echocardiographic picture is more compatible with mitral valve prolapse rather than with a flail posterior mitral valve leaflet.

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1. FEIGENBAUM H: Echocardiography. Philadelphia, Lee and Febiger, 1972, p 62, figs 4-21, 4-22

The authors reply:

As suggested by Drs. Sasse and Lightfoot, it may be difficult to distinguish prolapsing mitral valve leaflets due to ruptured chordae tendineae from prolapse due merely to long, redundant chordae and/or ballooning mitral valves. In two recent papers on the subject1,2 we have described those findings which most commonly distinguish these two types of prolapse.

In all the cases of ruptured chordae tendineae to the posterior leaflet confirmed at surgery, we have seen a paradoxical anterior movement of this posterior leaflet in early diastole. This abnormality is often associated with a hammock-like posterior bulging of the posterior leaflet during systole2. When this posterior leaflet is recorded throughout diastole, it remains primarily in an anterior position and moves posteriorly only in late diastole when it does so in a sharp manner. Probably because of increased flow across the mitral valve, the anterior leaflet tends to have a slightly increased magnitude and velocity of opening. As reported2, these echo characteristics were present in eight surgically confirmed cases of ruptured chordae tendineae to the posterior mitral leaflet and subsequent to that report in seven other cases operated at our institution.

We also agree with Drs. Sasse and Lightfoot that there may be hazards in interpreting echoes immediately anterior to the posterior left atrial wall. Currently, we identify posterior mitral leaflet as seen within the left atrium only if clearly defined valvular motion is recorded. More recently, we have found distinct valvular structures can occasionally be recorded during systole in the anterior portion of the left atrium near the posterior wall of the aorta. Upon changing the transducer position, this echo is found to be continuous with the paradoxically moving posterior mitral leaflet in early diastole.

Prolapsing posterior leaflets without ruptured chordae tendineae may also show a posterior sagging of the CD segment during systole and wide separation from the anterior leaflet echo. Recognition of this sagging in multiple echocardiographic views is considered mandatory in making this diagnosis in order to eliminate artifactual "false-positive" diagnoses. Also of note is that the echoes from the anterior mitral leaflet oftentimes show a rounded E point, forming a "roller coaster" appearance. In the absence of ruptured chordae, we have never seen either paradoxical anterior movement of the posterior leaflet echo at the onset of diastole or the leaflet within the left atrium. It should be recognized, however, that in some cases in which there is clear echo evidence of systolic prolapse of the mitral valve, it may be difficult to determine if there is accompanying rupture of mitral chordae tendineae.

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Wenckebach Periods and His-Purkinje Conduction

To the Editor:

Halpern and co-workers' demonstration of Wenckebach periods of alternate beats was thought provoking.1 We accept the authors' observations and interpretations in individual cases. We would like to question their over-all conclusion that AW are primarily a manifestation of depressed conduction in the His-Purkinje system (HPS). Their conclusion was based on 1) The occurrence of intraventricular blocks in all five cases with AW, 2) Pathological observations in one of their cases, with demonstration of lesions in the His-Purkinje system. 3) His bundle recording in one of their cases, with demonstration of AW distal to the His bundle. 4) Experimental production of AW with surgical trauma to the HPS.

Our thoughts regarding this evidence are as follows:
1) The presence of intraventricular conduction defects also implies a significant incidence of associated A-V nodal dysfunction.2,3 In our experience, progression of A-V nodal disease in patients with bifascicular block is not uncommon.4 Thus, the occurrence of AW in a patient with intraventricular block is consistent with AW in either the A-V node, HPS, or both. 2) The demonstration of pathological lesions in the HPS does not exclude the occurrence of major conduction block at the A-V node.5 The latter may be functional, without an apparent anatomic abnormality.6 3) In regard to the one case with His bundle recordings, we have recently seen a patient with somewhat different findings. Our patient had chronic bifascicular block and digitalis intoxication. The patient had

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