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.

LEWIS Sasse, M.D.
PAUL LIGHTFOOT, M.D.
Department of Internal Medicine
Southern California Permanente Medical Group
and Kaiser Foundation Hospital
Los Angeles and Fontana, California

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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 systole.2 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 reported,2 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 posterior 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 artificial "false-positive" diagnoses. Also of note is that the echos 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.

JUDY BURGESS
RALPH CLARK, M.D.
KEITH COHN, M.D.
Cardiopulmonary Laboratories
Presbyterian Hospital of Pacific Medical Center
San Francisco, California

References

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
Letters to the Editor

Wenckebach periods during sinus rhythm, 2:1 block at a paced rate of 130/min (fig. 1A), and AW at a paced rate of 140/min (fig. 1B). These conduction abnormalities were all localized proximal to the His bundle recording site, and reversed with atropine administration. Thus, our case appears to be an example of A-V nodal AW in a patient with bifascicular block. 4) The ability to provoke AW by surgical trauma to the His-Purkinje system does not mean that such AW could not also be provoked by experimentally induced A-V nodal dysfunction.

We would suggest that A-V nodal AW may be as common as AW in the HPS. We would also predict that some episodes of AW in patients with bifascicular block will reflect 2:1 block proximal to the His bundle (A-V nodal) with Wenckebach periods in the remaining functioning fascicle. His bundle recordings in patients with AW should help clarify the site or sites of this interesting phenomenon.

Ruben Chuquimia, M.D.
Kenneth M. Rosen, M.D.
Abraham Lincoln School of Medicine
University of Illinois
College of Medicine
Chicago, Illinois

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The authors reply:

Although Chuquimia and Rosen find our paper "thought provoking"—which is quite a compliment—and also accept our "observations and interpretations in the individual cases," they question "the over-all conclusion," which they state somewhat arbitrarily. Forced to choose an over-all conclusion, ours would be—for the sake of a better understanding—that "AW commonly occurs below the AV node" (as stated in the summary), and not that "AW are primarily a manifestation of depressed conduction in the HPS."

Regarding the four points of questioning, the following comments may be worthwhile: 1) They say that "the occurrence of AW in a patient with intraventricular block is consistent with AW in either the A-V node, HPS, or both." This is correct. However, when we discuss five patients, all of them with intraventricular block, the chances are obviously a predominance of the causative lesions at the intraventricular level, as indeed supported by the rest of the observations. 2) If a patient with right bundle branch block (RBBB), left anterior hemiblock, and AW has severe lesions in the HPS and none in the A-V node, to raise the possibility of a "major conduction block at the A-V node" of a "functional" nature is beyond our understanding, unless the philosophy here is "anything may happen anywhere." The case reported by Chuquimia and Rosen is certainly a nice example of AW at the nodal level in the presence of RBBB. However, in a patient with digitalis intoxication, and in whom the AW is suppressed with atropine administration, it is only natural to suspect that the AW, or at least part of it, occurs in the A-V node, despite the intraventricular block. In our study, cases like this were intentionally excluded. Incidentally, in our cases the AW was never abolished by atropine; on the contrary, it was commonly provoked with the same drug. This can certainly serve as a simple and nonaggressive approach.
Wenckebach Periods and His-Purkinje Conduction
RUBEN CHUQUIMIA and KENNETH M. ROSEN

Circulation. 1974;49:596-597
doi: 10.1161/01.CIR.49.3.596-a

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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