The Genesis of the Diastolic Murmur of Complete Heart Block

Phono-Echocardiographic Observations

MANSOOR JELVEH, M.D., MARVIN BERGER, M.D.,
AND EMMANUEL GOLDBERG, M.D.

SUMMARY A 72-year-old man with calcific aortic stenosis and complete heart block was noted to have an intermittent diastolic murmur. Using combined echocardiography and phonocardiography, it was demonstrated that the murmur occurred while the mitral valve was closing. This suggested that antegrade flow across a closing mitral orifice is of etiologic importance in the production of the murmur. Atrial sounds were also recorded and probably have a similar etiology. It is postulated that reduced ventricular compliance resulting in a more rapid rate of mitral valve closure was of etiologic importance in the genesis of the murmur.

ALTHOUGH DIASTOLIC MURMURS are common in congenital complete heart block,\(^1\)\(^2\) they are extremely unusual in elderly individuals with acquired complete heart block.\(^4\) We recently encountered an elderly patient with complete heart block who was noted to have an audible apical diastolic murmur. Simultaneous echocardiographic and phonocardiographic studies enabled us to correlate the murmur with mitral valve motion and thus elucidate some of the possible mechanisms involved in its production.

Case Report

A 72-year-old male was admitted to Beth Israel Medical Center for the first time on May 19, 1977, because of an infected pacemaker pocket. In 1965, after an acute myocardial infarction complicated by complete heart block, a permanent transvenous pacemaker was implanted. During the ensuing years, the patient remained free of cardiac symptoms, although replacement of the pacemaker generator was necessary on four occasions. After the last replacement in 1976, an abscess developed in the pacemaker pocket. There was no history of rheumatic fever. A cardiac murmur was known to be present for many years.

Physical examination revealed a regular pulse of 70/min and blood pressure of 116/90 mm Hg. The carotid pulse was of small volume with a slow upstroke. A sustained outward thrust was felt in the fifth intercostal space just lateral to the midclavicular line. The first heart sound was of variable intensity and the second sound was soft and single. A grade III/VI harsh ejection systolic murmur was audible over the entire precordium, and radiated to the neck. An intermittent, low-pitched diastolic murmur was present at the apex. The lungs were clear. There was no hepatomegaly or peripheral edema.

The electrocardiogram revealed a pacemaker rhythm with a ventricular rate of 70/min. The atrial rate which was sinus in origin was 50/min. Roentgenogram of the chest revealed slight cardiomegaly. Echocardiographic examination revealed concentric left ventricular hypertrophy. There were multiple dense linear echoes within the aortic root due to aortic valve calcification. It was felt that the patient had calcific aortic stenosis. To further clarify the mechanisms involved in the genesis of the intermittent diastolic murmur, a simultaneous echo-phonocardiographic examination was performed.

Echo-Phonocardiographic Observations

Figure 1 illustrates the relationship of the apical diastolic murmur to the P wave of the electrocardiogram and the mitral valve echogram. The murmur is present only when the P wave occurs late in ventricular systole or in early to mid diastole with a P to pacemaker stimulus interval (P-S interval) of 0.55-0.20 sec, and is associated with either premature closure or a more rapid rate of closure of the mitral valve. With longer P-S intervals the murmur tends to be of briefer duration and resembles a single or split sound (fig. 2, left and right). The initial vibrations of the murmur coincide with the beginning of a rapid closing movement of the mitral leaflets and its terminal vibrations end at the point of closure of the mitral leaflets as seen on the mitral echogram in figure 1. An interval of 0.20-0.25 sec is always present between the preceding P wave and the earliest vibrations of the diastolic murmur or sound.

The pattern of mitral valve motions bears a relationship to the intensity of the first sounds (fig. 1).

From the Department of Medicine, Division of Cardiology, Beth Israel Medical Center and the Department of Medicine, The Mount Sinai School of Medicine of the City University of New York.

Address for reprints: Mansoor Jelveh, M.D., Beth Israel Medical Center, 10 Nathan D. Perlman Place, New York, New York 10003.

Received January 26, 1978; accepted June 26, 1978.

Circulation 58, No. 4, 1978.
When the P-S interval is shorter than 0.20 sec, the mitral leaflets are fully open at the onset of ventricular systole, resulting in a relatively loud first heart sound. On the other hand, a P-S interval of 0.50-0.20 sec is associated with a mitral valve that is closed at the onset of systole and an absent or markedly reduced first heart sound. When the P-S interval exceeds 0.50 sec, the mitral valve reopens in late diastole and a first heart sound of medium intensity is recorded.

Figure 3 demonstrates a simultaneous recording of the electrocardiogram, phonocardiogram and echocardiogram of the anterior tricuspid leaflet. The recorded apical diastolic murmur extends well beyond the point of tricuspid valve closure. This is evidence against a tricuspid origin of the diastolic murmur.

Discussion

Auscultatory phenomena produced by atrioventricular dissociation include variation in intensity of the first heart sound, atrial sounds occurring at various times during the cardiac cycle and diastolic murmurs.

Studies by Shah et al. and Burggraf and Craigie have demonstrated the temporal relationship between the timing of atrial systole and the intensity of the first heart sound. These investigators showed that atrial systole alone can bring about atrioventricular valve closure, and that the loudness of the first heart sound is related to the timing of atrioventricular valve closure in relation to ventricular systole. When the P-R interval exceeds 0.20 sec, the mitral valve is closed before the onset of ventricular systole and the first
Atrial sounds called "galop du bloc" by Gallavar- din in 1914 have been noted in as many as 50% of cases of heart block. An interesting finding occasionally noted in complete heart block is a split atrial sound. The first component, which occurs about 0.15 sec after the P wave, corresponds to the usual fourth heart sound and is most likely related to atrial contraction. The second component occurs 0.20-0.24 sec after the P wave and has been attributed by some investigators to closing of the atrioventricular valves. In the case reported here, the atrioventricular valves were still open in the time range of 0.20-0.24 sec after the P wave. Furthermore, at times after atrial systole, instead of a diastolic murmur, we recorded two distinct sounds similar to the split atrial sounds reported by others. Thus, it appears likely that the mechanisms involved in the production of split atrial sounds in complete heart block were the same as those for the diastolic murmur as discussed below.

The genesis of the diastolic murmur in complete heart block is unclear from the literature. In congenital heart block where a diastolic murmur is frequently recorded, it is attributed to the very large stroke volume that has to traverse the atrioventricular valves during diastole. The murmur has only occasionally been reported in elderly subjects. Rytand reported nine patients with complete heart block and an audible diastolic murmur. He postulated that the murmur is produced by modification of the movements of the mitral valve leaflets after atrial systole, and considered the process of aging of the valvular leaflets responsible for such a mechanism. However, the vast majority of elderly patients with complete heart block do not manifest a diastolic murmur following atrial systole. Fortuin and Craige, on the basis of their own echocardiographic studies and in conjunction with cineangiographic studies of Criley et al., have suggested a unitarian concept for the genesis of certain diastolic murmurs. These murmurs include the presystolic murmur of mitral stenosis, the Austin Flint murmur of aortic regurgitation and the mid-diastolic murmur of mitral regurgitation and large left-to-right shunts. In each of these conditions a common mechanism appears to be involved in the production of the diastolic murmur, namely, antegradation across a closing mitral orifice. Similarly, in our patient, the diastolic murmur starts with the rapid closing movement of the mitral valve leaflets and ends at the time of its closure.

The observation that diastolic murmurs occur infrequently in elderly patients with complete heart block merits further comment. The patient reported here had aortic stenosis with left ventricular hypertrophy. It is possible that the decreased compliance known to be present in patients with ventricular hypertrophy resulted in a rapid increase in left ventricular pressure during diastolic filling. This, in turn, produced a more rapid closing movement of the mitral valve leaflets, while antegradation across the valve was still in progress, resulting in the production of a diastolic murmur. In this respect, it is interesting to note that eight of nine patients reported by Rytand.
had conditions associated with increased left ventricular diastolic pressures (hypertensive heart disease in seven, ischemic heart disease in one). The only patient in this series without any apparent underlying heart disease had a ventricular rate of 27 beats/min. It is reasonable to assume that the marked increase in stroke volume in this patient produced a rapid increase in left ventricular pressure after atrial systole, resulting in exaggerated closing movement of the mitral valve, with production of a diastolic murmur in accordance with the mechanism proposed by Fortuin and Craige. This hypothesis also explains the diastolic murmur of congenital complete heart block in which the stroke volume is also significantly increased.

References
The genesis of the diastolic murmur of complete heart block. Phono-echocardiographic observations.
M Jelveh, M Berger and E Goldberg

Circulation. 1978;58:747-750
doi: 10.1161/01.CIR.58.4.747

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1978 American Heart Association, Inc. All rights reserved.
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:
http://circ.ahajournals.org/content/58/4/747

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally
published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the
Editorial Office. Once the online version of the published article for which permission is being requested is
located, click Request Permissions in the middle column of the Web page under Services. Further
information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/