Mechanism of Atrial Sounds in Atrial Fibrillation
Phonoechocardiographic Correlation. Report of a Case

Virinderjit S. Bamrah, M.D., Charles V. Hughes, M.D.,
and Felix E. Tristani, M.D.

SUMMARY Audible atrial sounds were noted in a patient with congestive cardiomyopathy and atrial fibrillation with slow ventricular rate. Oscillatory motion of similar periodicity as fibrillation waves in the ECG and the sounds in the phonocardiogram was demonstrated in aortic and left ventricular walls, mitral, tricuspid and aortic leaflets by echocardiography. This suggests that the fibrillation motion of the atria is forceful enough to cause vibration of the cardiohemic system resulting in the audible sounds.

AUDIBLE ATRIAL SOUNDS have been occasionally recorded in atrial flutter,1-6 but are extremely rare in atrial fibrillation. To date, only three case reports describing four patients have been published.10-12 It is the purpose of this presentation to discuss a patient with atrial fibrillation in whom atrial sounds were audible and to report, for the first time, a correlation of the phonocardiographic and echocardiographic findings.

Case Report

A 62-year-old Caucasian male was referred to the Veterans Administration Center, Wood, Wisconsin, for management of cardiac arrhythmia and heart failure. Dyspnea had been present for several months but, in October 1974, it increased in severity and he developed peripheral edema. The electrocardiogram showed atrial fibrillation with a slow ventricular rate and multiple ventricular extrasystoles. Diuretics were administered and he was referred to this Center for further medical management. There was no history of angina, myocardial infarction or hypertension. In 1942, a routine medical examination showed an irregular cardiac rhythm and an enlarged heart. In January of 1974, he developed pneumonia from which he had a slow recovery. There was no history of cigarette smoking, alcohol intake, or family history of heart disease. Physical examination revealed an irregular pulse of 50 beats per minute, blood pressure 120/70 mm Hg, and normal jugular venous pressure. The apex beat was of heaving quality and located in the sixth left interspace in the anterior axillary line. S1 was of variable intensity and S2 was narrowly split. A soft S3 was audible over the apex. A grade 2/6 soft early middystolic murmur was present over the apex. Multiple clicking sounds were audible at the apex and left lower sternal border, both during systole and diastole. There was no edema or hepatomegaly and the lungs were clear. Chest X-ray revealed moderate generalized cardiomegaly, minimal congestion of the lungs, and a small left-sided pleural effusion. Admission electrocardiogram showed coarse atrial fibrillation with a ventricular rate of 40 beats/min, ventricular extrasystoles, and nonspecific ST-T changes. Routine laboratory values were normal except for a BUN which was 31 mg%. A clinical diagnosis of congestive cardiomyopathy of uncertain etiology was considered, and the following additional studies were done.

Phonocardiogram

Multiple irregular sounds of variable amplitude were noted, best recorded from the apex and left lower sternal border in the medium frequency range (100-200 Hz) (fig. 1). These sounds roughly correlated with the fibrillation waves in the ECG. The sounds occurred at a rate of approximately 480/min, which was roughly identical to the rate of oscillatory motion of various cardiac structures, as shown by echocardiogram (figs. 2 and 3) and pressure tracings (fig. 4). They were present during both systole and diastole and were particularly prominent during the long cardiac cycles. In some cardiac cycles, these sounds became more frequent toward the latter portion of diastole. In addition, PCG showed early middysstolic murmur at the cardiac apex.

Echocardiogram

Examination revealed a large left ventricle and left atrium. The left ventricular posterior wall and ventricular septum were of normal thickness and had normal motion. Echocardiogram demonstrated an oscillatory motion of the anterior and posterior walls of the aorta, aortic leaflets during diastole (fig. 2), anterior mitral leaflet, mitral annulus and ventricular septum (fig. 3). This oscillatory motion had roughly the same periodicity as fibrillation waves in the ECG.

Cardiac Catheterization

Normal right and left heart pressures at rest were noted. The cardiac and stroke indices were diminished at 1.8 L/min/M² and 30 ml/beat/M², respectively, and arteriovenous oxygen difference was increased to six volumes percent. Mild supine exercise on a bicycle ergometer increased the left ventricular end-diastolic pressure abnormally. Stenotic lesions involving the right coronary artery (90%) and left anterior descending (60%) were present. A left ventriculogram showed grossly dilated cavity with generalized hypokinesia and minimal mitral regurgitation. The degree of left ventricular dysfunction was out of proportion to the

From the Cardiovascular Section, Medical Service, Veterans Administration Center, Wood (Milwaukee), Wisconsin, and the Department of Medicine, Medical College of Wisconsin, Milwaukee.

Address for reprints: Virinderjit S. Bamrah, M.D., Cardiac Catheterization Laboratory, Veterans Administration Center, Wood, Wisconsin 53183.

Received July 8, 1975; revision accepted for publication October 9, 1975.
extent and severity of angiographically-demonstrated coronary artery disease. Pressure pulse tracings from the right atrium, right ventricle, pulmonary artery, pulmonary artery wedge, and aorta demonstrated fibrillation waves during diastole. During cineangiography, the oscillatory motion of the atria was seen to be transmitted to the catheter in the aortic root and to the coronary arteries. This motion was most marked in the proximal portion of the circumflex vessel.

The patient was continued on digoxin, diuretics, and a permanent ventricular demand pacemaker was inserted. After improvement of his heart failure, the atrial sounds were no longer audible.

Discussion

Atrial sounds were described by Neporent and Da Silva in three patients, all having coarse atrial fibrillation and mitral regurgitation. Two of these patients had, in addition, aortic valve disease and the third had mitral stenosis. Our patient also demonstrated coarse atrial fibrillation and minimal mitral regurgitation, and his underlying heart disease was thought to be congestive cardiomyopathy of unknown etiology and coronary artery disease.

The available reports indicate that these sounds in atrial flutter or fibrillation are noted in the presence of congestive heart failure and high-grade atioventricular block resulting in slow ventricular rate with long diastolic pauses. They are usually of high frequency, clicking in quality, best heard over the base or midparasternal areas rather than at the apex. They are present during systole and diastole and tend to disappear with improvement of heart failure and increase of ventricular rate. Our patient demonstrated similar features, except that the sounds were best recorded from the apex and left lower sternal border. Two previous reports also describe flutter sounds from the same precordial location.

Gabor and Winsberg described coarse undulating motion of the mitral leaflets during diastole on echocardiography in atrial flutter and coarse fibrillation. In our patient, the coarse fluttering motion was recorded from the anterior and
posterior walls of the aorta, aortic leaflets, mitral annulus, and ventricular septum, in addition to the mitral and tricuspid leaflets. During cineangiography this oscillatory motion was also visualized in the atra, aortic walls, and coronary arteries. Pressure pulse recordings from the right and left heart chambers displayed coarse undulating fibrillation waves. The occurrence of these hemodynamic events, at a very rapid rate in our patient, show that sometimes in atrial fibrillation mechanical atrial activity may be quite vigorous. Prinzmetal et al., using cinematography, have described "L" waves in atrial fibrillation which occur at a rate of 400-600/min and may be of sufficient strength to accomplish systolic and diastolic changes in the atria.14

The mechanism of transmission of this fluttering motion from the atria, where it starts, to other cardiac structures, especially the aortic leaflets, is difficult to explain. It is possible that the fibrillatory contractions of the atria were strong enough to be transmitted to cardiac valves through the fibrous cardiac skeleton in which the valves are inserted. An alternate possibility is that during diastole, when the mitral valve opens, atrial contractions impart a fluttering motion to the mitral leaflets and, because of the anatomical continuity between the anterior mitral leaflet and aortic leaflets, this motion is transferred to the aortic leaflets. The echocardiogram revealed an increasingly coarse fluttering of the mitral leaflets associated with an increase in recorded atrial sounds in mid and late diastole. The probable explanation for this increase is that, as diastole proceeds and the atrioventricular pressure gradient diminishes in mid-diastole, the mitral leaflets move into a partly closed position and interfere with incoming bloodstream and flutter more coarsely as a result of atrial contractions. The relationships of the position of atrioventricular valves with the intensity of atrial sounds has also been discussed in earlier reports.3,5

A variety of mechanisms have been proposed to explain the production of atrial sounds in atrial flutter and fibrillation. Some of the earlier reports indicated that these were extracardiac sounds. Penny et al.7 compared these sounds to the opening snap in their patient with mitral stenosis and suggested that they were caused by rapid downward movement of the thickened mitral leaflets after each atrial contraction. The usual fourth heart sound in sinus rhythm is composed of two components on PCG, an early high frequency inaudible vibration occurring at the time of atrial contraction and a later low frequency audible component reflecting ventricular filling.15-18 Most of the published reports indicate that the audible flutter-fibrillation sounds represent the first high frequency component and are produced by changes in tension of atrial wall or atrioventricular valves at the time of atrial contractions.6-12 They are not considered to be of ventricular origin because they are high frequency sounds, occur both during systole and diastole, and are best heard over the base of the heart. The PCG from our patient shows that these sounds are present during diastole as well as systole when ventricular filling does not occur because of closed atrioventricular valves (fig. 1). Moreover, Massumi et al., using an intracavitary phonocatheter, recorded these sounds only from within the right atrium. There is only one report available which suggested that these sounds could be of ventricular origin because they were best recorded from the cardiac apex.6 Our patient reveals that, in the presence of coarse atrial fibrillation, atrial contraction may be forceful enough to be transmitted to various cardiac structures including the ventricles, the great vessels and the intracardiac valves. Probably the entire cardiohemic system vibrates as a result of these contractions, as demonstrated by echocardiography in our patient, resulting in audible sounds.

References
2. Lian C, Welti JJ: Flutter, ou mieux tre'mulation auriculaire et bradycarde par dissociation auriculo-ventriculaire complete; audition et enregistrement graphique des bruits auriculaires. Arch Mal Coeur 31: 518, 1938
3. Hecht HH, Myers GB: Auricular heart sounds in auricular flutter. Am J Cardiol 29: 610, 1945

FIGURE 4. Pressure pulse recordings from right ventricle (RV), pulmonary artery (PA), and pulmonary artery wedge (PAW). During diastole all the pressure pulse tracings show irregular fibrillation waves occurring at a rapid rate and roughly identical to the fibrillation waves of the electrocardiogram (ECG). Timelines are at an interval of 1.0 second.
Mechanism of atrial sounds in atrial fibrillation. Phonoechocardiographic correlation.

Report of a case.

V S Bamrah, C V Hughes and F E Tristani

Circulation. 1976;53:569-571
doi: 10.1161/01.CIR.53.3.569

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1976 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/53/3/569

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/