Frequency of the First Heart Sound in the Assessment of Stiffening of Mitral Bioprosthetic Valves

PAUL D. STEIN, M.D., HANI N. SABBAH, B.S., JEFFREY B. LAKIER, M.D., DONALD J. MAGILLGAN, JR., M.D., AND SIDNEY GOLSTEIN, M.D.

SUMMARY The frequency spectrum of the first heart sound (S₁) was measured noninvasively in 54 patients with porcine bioprosthetic valves inserted in the mitral position. Phonocardiograms were recorded on magnetic tape on line with a signal processor with which the frequency spectrum and peak frequency of S₁ were determined. In 19 patients with normal natural mitral valves, the apparent peak frequency within the range of measured frequencies of S₁ was 46 ± 2 Hz (mean ± SEM). In 11 patients with porcine bioprosthetic valves implanted in the mitral position for ≤ 1½ years, the apparent peak frequency of S₁ was 43 ± 3 Hz, which was not significantly different from S₁ in patients with normal mitral valves. However, in 33 patients with porcine bioprosthetic valves in place 5–7 years, the apparent peak frequency of S₁ was higher, 67 ± 2 Hz (p < 0.001). In patients in whom the porcine bioprosthetic valve was implanted 5 years or longer, the frequency spectrum of S₁ showed a greater proportion of sound energy at frequencies between 50–200 Hz compared with patients in whom the prosthetic valve was implanted 1½ years or less. Changes in the frequency of S₁ in these patients may be a manifestation of stiffening of the valve as a result of early degenerative changes.

DEGENERATION of porcine bioprosthetic valves after 5 years of implantation is recognized.¹⁻³ Therefore, methods of detecting the degenerative process before it becomes clinically obvious should be assessed. Among the methods that appear to be of value for detecting early degenerative changes of bioprosthetic valves in the aortic position is frequency analysis of the sound related to valve closure.³ We demonstrated that the frequency of the second sound increases with a longer duration of implantation of these valves in the aortic position, and this appears to reflect stiffening of the valve.⁴ The second sound, which results from closure of the aortic bioprosthetic valve, is initiated by vibratory motion of the closed leaflets of the bioprosthesis valve in a fashion identical to the cause of the second sound produced by the natural aortic valve.⁵ The frequency of vibration of the valve, as with any vibrating structure, increases with stiffness of the vibrating membrane, in this case the valve leaflets.⁶ Therefore, changes that lead to stiffening of the leaflets would increase the frequency of the second sound. For the same reason, an increased frequency of the second sound has been observed in patients with calcific aortic stenosis.⁷

The cause of the first sound (S₁) has not been established, although there is evidence to suggest a valvular origin.⁷, ⁸ The first sound of bioprosthetic valves in the mitral position reasonably can be assumed to be initiated by vibration of the closed leaflets, in a fashion analogous to the second sound. If this is so, one may speculate that the frequency of the S₁ would be affected by stiffening of the leaflets of the bioprosthetic valve. The purpose of this study was to assess the frequency of the S₁ as it relates to the duration of implantation of bioprosthetic valves in the mitral position. Presumably, an increased frequency would suggest a method for detecting subclinical stiffening of the leaflets.

Methods

Phonocardiograms were recorded in 54 patients (15 males and 39 females) ages 14–72 years, with glutaraldehyde-stabilized porcine bioprosthetic valves (Hancock Laboratories, Inc.) implanted in the mitral position. The tissue annulus diameter of the porcine xenografts ranged from 27–35 mm. Phonocardiograms were also recorded in 19 control subjects ages 14–46 years. The control subjects were hospitalized because of noncardiac illnesses. None had cardiac murmurs or any other physical abnormalities on cardiac examination, and none had a history of heart disease. All patients with bioprosthetic valves were studied without knowledge of the duration of implantation.

The duration of bioprosthetic valve implantation in the 54 patients with such valves ranged from a few days to longer than 7 years (88 months). One patient had porcine valve degeneration and underwent replacement of her prosthetic valve shortly after the studies of sound were obtained. Her valve had been implanted 73 months and was removed because of severe prosthetic valve regurgitation. The leaflets of the resected valve were shown to be thickened and stiff, and one of the leaflets was totally incompetent and behaved as a flail leaflet (fig. 1). Scanning electron microscopy of the valve showed endothelial cell loss, focal deposits of platelets, leukocytes and erythrocytes.

Patients with bioprosthetic valves were excluded if they had a history of myocardial infarction, because the frequency of the first sound may be altered in such patients.⁹, ¹⁸ Patients were also excluded if they had aortic regurgitation, because vibration of the
bioprosthetic valve could be modified by the regurgitant flow, although in five patients with severe aortic insufficiency studied by others, the frequency spectrum of S₁ was normal. Patients were also excluded if the hemoglobin was markedly reduced (<11 g/100 ml) because the second heart sound, and presumably the first sound that results from the bioprosthetic valve, is predicted by vibration analysis to be affected by the viscosity of blood.

Heart sounds were recorded during quiet respiration with the patient lying on his left side. The microphone (Irex Medical Systems #120-131 Heart Sound microphone) was placed over the apex of the heart at the site of the maximum palpable impulse. It was held in this position by a suction cup. This permitted a uniform and consistent technique for the application of the microphone. The same microphone was used for all patients.

In each patient the phonocardiogram, carotid pulse tracing, lead II of the ECG and respiration were recorded on a VR-6 photographic recorder (Electronics for Medicine). The phases of respiration were measured with a nostril thermistor. These signals were also recorded simultaneously on an eight-track magnetic tape recorder (Hewlett-Packard Model 8868A). The frequency response of the sound amplifier and microphone combination was flat within 1 db from 80-300 Hz. In the lower frequency range, at 40 Hz there was a 6-db attenuation and at 20 Hz there was a 14-db attenuation. In the higher frequency range, at 600 Hz there was a 6-db attenuation and at 1200 Hz a 20-db attenuation.

The tape recorder was operated at a speed of 3.75 inches/sec and had a passband frequency of 0-1250 Hz. The frequency response over the passband was ±1.0 db, referenced to 10% of the upper band-edge frequency. The signal-to-noise ratio, measured with carrier deviation of ±40% at 10% of the upper passband and without flutter compensation, was 46 dB. The level of flutter at a speed of 3.75 inches/sec and a passband of 0.2-625 Hz was 0.40% peak to peak.

The frequency of the entire first sound was analyzed with a digital signal processor (Spectral Dynamics, SD360) from the data recorded on the magnetic tape recorder. After a transient capture of the portion of the cardiac cycle that contained the S₁, all other sounds were deleted. A fast Fourier transform was performed on the sound signal and displayed on linear coordinates over a frequency of 0-500 Hz. The peak frequency of the first sound was defined as the frequency with the highest magnitude shown in the frequency spectrum. Because of filtering in the system, the magnitude of frequencies below 50 Hz was attenuated. Therefore, frequencies below 50 Hz may have had higher energies that were not apparent because of the filtering in this system. In each patient two cycles were analyzed at random. The average difference between both analyses in terms of the peak frequency was less than 10%.

An average frequency spectrum of S₁ was computed from individual frequency spectra of normal patients, patients with porcine valves inserted less than 1½ years and patients whose valves were inserted 5-7 years. This was accomplished by digitizing the individual frequency spectra of S₁ from 50-250 Hz in each of the patients within each group and computing an average value. The intensities of various frequencies of S₁ were shown relative to the frequency at 50 Hz. The individual frequency spectra were digitized at 1-mm intervals, which was equivalent to a frequency interval of 1.3 Hz. Frequencies below 50 Hz were excluded because there was attenuation of the lower frequencies due to filtering. Frequencies above 250 Hz were excluded because attenuation of the sound energy frequently exceeded 40 db, which may result in a high signal-to-noise ratio.

Results

The apparent peak frequency of the S₁ in patients with normal mitral valves was 46 ± 2 Hz (mean ± SEM) (figs. 2 and 3). In 11 patients with porcine bioprosthetic valves implanted in the mitral position 1½ years or less, the apparent peak frequency of the S₁ was 43 ± 3 Hz, which was not significantly different from the apparent peak frequency of S₁ produced by

FIGURE 1. Degenerated bioprosthetic valve 73 months after insertion in the mitral position. The leaflets were thickened and stiff, and one of them was incompetent. The dominant frequency of the first sound recorded in this patient was 79 Hz.
the normal mitral valve. In 33 patients in whom the bioprosthesis valve was inserted 5 to 7 years, the dominant frequency was higher, 67 ± 2 Hz (p < 0.001, unpaired t test).

In patients in whom the porcine bioprosthetic valve was implanted 5 years or longer, the frequency spectrum of $S_1$ showed a greater proportion of sound energy at frequencies between 50–200 Hz compared with patients in whom the prosthetic valve was implanted 1½ years or less (fig. 4). The proportion of sound energy in the $S_1$ above 50 Hz in patients with prosthetic valves inserted 1½ or less years did not differ significantly from the sound energy of the $S_1$ in patients with a normal mitral valve.

**Discussion**

An increased stiffness of the valve would result in an increased frequency of vibration in accordance with the following equation, which was derived on the basis of factors that affect the frequency of vibration of the closed aortic valve. This has been shown in patients with calcified stenotic aortic valves. Bioprosthetic valves in the aortic position were also shown to have a higher frequency with a longer duration of implantation. Presumably, an aortic bioprosthetic valve implanted in the mitral position also shows a frequency described by this equation:

$$\omega = \sqrt{K/m (1 - D^2/4Km)}$$

where $\omega = \text{frequency of vibration}$, $m = \text{effective mass of the vibrating system (including the blood and valve leaflets)}$, $D = \text{damping force coefficient}$, and $K = \text{stiffness factor}$.

The observed increased frequency of $S_1$ that occurred with the longer duration of implantation implies that, at least with bioprosthetic valves, the first sound is of valvular origin. A nonvalvular origin of the $S_1$, however, has also been postulated. According to this theory, the initial audible components of the $S_1$ begin with abrupt tension of the closed mitral valve, which decelerates the moving blood. These vibrations, which occur during isovolumic contraction, are thought to result from overstretch of the mitral valve and recoil back toward the ventricles. In this case, the cardiohemic vibrating system consists primarily of the ventricular cavities completely enclosed by the valves and contracting myocardium. The frequency content of the $S_1$ during the isovolumic phase of contraction, therefore, according to Adolph et al., should depend on the relative contributions of mass and elas-
ticity associated with the oscillating left ventricle. The observation of a change of frequency of $S_1$ resulting from myocardial infarction is also compatible with a valvular origin of $S_0$, if one speculates that $S_1$ is modified by resonant vibrations of the left ventricular wall or by the characteristics of the wall as sound is transmitted through the ventricular wall.

Our results did not seem to reflect fibrotic changes that could have occurred in the left ventricular wall during the years after insertion of the valve. None of our patients suffered a myocardial infarction. All but one (the patient with valve degeneration) were symptomatically improved after insertion of the prosthetic valve. Presumably, therefore, the fibrosis or hypertrophy of the left ventricular wall in these patients would have occurred before insertion of the prosthetic valve. There is no evidence that hypertrophy or fibrosis increased after insertion of the valve.

The range of frequencies contained in the $S_1$ have been reported by several investigators. The apparent peak frequency of $S_1$ that we observed in control subjects and patients with bioprosthetic valves implanted 1½ years or less was comparable to the frequency of $S_1$ previously observed in normal subjects. Although with prosthetic stenosis, the mass of the valve increases, and this would tend to reduce the frequency as shown in the equation, the actual mass of the valve is only a small portion of the effective mass of the vibrating system. The effective mass includes the blood in the vicinity of the valve. The mass of the blood is considerably greater than the mass of the valve, so increments of valve mass would have only a small effect.

The fact that the frequency of the $S_1$ in patients with bioprosthetic valves implanted 5–7 years was higher than the dominant frequency in patients in whom such a valve was implanted 1½ years or less suggests that the analysis of the frequency of the $S_1$ might be useful in identifying patients with stiffening of the valve. Such stiffening may result from subclinical degenerative changes. An earlier diagnosis may be achieved with the use of frequency analysis, perhaps in combination with other noninvasive studies, such as echocardiography.

References

3. Ferrans VJ, Spray TL, Billingham ME, Roberts WC: Structural changes in glutaraldehyde-treated porcine heterografts used as substitute cardiac valves. Am J Cardiol 41: 1159, 1978
Frequency of the first heart sound in the assessment of stiffening of mitral bioprosthetic valves.

P D Stein, H N Sabbah, J B Lakier, D J Magilligan, Jr and D Goldstein

Circulation. 1981;63:200-203
doi: 10.1161/01.CIR.63.1.200

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1981 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/63/1/200