Pacemaker-Induced Change in Prosthetic Valvular Sounds*

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Variable intensity and timing of valvular sounds were noted in two patients with permanent pacemakers and prosthetic valves. In the first patient, who had a mitral and a tricuspid prosthesis, valvular opening sounds became widely separated with a change from nodal to ventricular paced rhythm. In a second patient, who had a mitral prosthesis in sinus rhythm with first-degree atrioventricular block, a very quiet closing sound increased markedly in intensity with the onset of ventricular pacing. The mechanism for these changes is described using a technique which at brief intervals throughout the cardiac cycle permitted documentation of simultaneously recorded phonocardiogram, electrocardiogram, and pulse tracings with a visible image of the prosthesis. In the usual patient with a prosthetic valve auscultatory changes should arouse a suspicion of malfunction; however, in the relatively uncommon patient with a prosthesis and an artificial pacemaker, it should be recognized that auscultatory changes may well have a physiologic explanation.

In patients with prosthetic valves, changes in the intensity or timing of the opening and closing sounds should alert the physician to the possibility of valvular malfunction;1 however, marked reduction in the intensity of the closing sound has been documented in a normally functioning mitral prosthesis as a consequence of presystolic closure in complete heart block,2 in first-degree atrioventricular block,3 after prolonged diastolic filling time in atrial fibrillation,4 or following premature ventricular contractions.5 The purpose of this report is to document marked alteration in prosthetic valvular sounds during artificial pacemaker-induced rhythms.

CASE REPORTS

CASE 1

A 43-year-old woman with rheumatic heart disease and intermittent heart block underwent replacement of mitral and tricuspid valves with Starr-Edwards prostheses. An epicardial pacing electrode was inserted on the right ventricle. After surgery, atrial fibrillation with complete heart block was present. Pacing from an external pulse generator produced marked changes in the auscultatory findings. With pacing, a loud single closing sound and prominent, very widely separated opening sounds were audible. With cessation of pacing, narrowly separated opening and closing sounds were audible. A permanent pacemaker proved necessary. When this pacemaker was activated or inactivated with the use of chest wall stimulation,6 the previously noted auscultatory changes could be produced at will.

CASE 2

A 49-year-old man with a Starr-Edwards mitral prosthesis was admitted with brief syncopal episodes. A very quiet first heart sound detected on admission suggested possible valvular malfunction; however, when an electrocardiogram revealed sinus rhythm with atrioventricular conduction prolonged at 280 msec, it was predicted from previous experience2,6 and later confirmed that the quiet closing sound was the result of presystolic valvular closure. When the patient subsequently developed intermittent second- and third-degree atrioventricular block, a permanent pacing electrode was placed in the right ventricle. With the onset of ventricular pacing, a loud mitral closing sound was audible. Suppression of the pacemaker with chest wall stimulation and with the patient in first-degree atrioventricular block again resulted in a very quiet first heart sound.

METHOD

The mechanisms involved in the change in auscultatory findings were documented with a previously described technique8 which permitted visual correlation of poppet position with the ECG and phonocardiogram at any time during the cardiac cycle. The demand pacemaker in each patient was inactivated temporarily by chest wall stimulation over the pulse generator using an external pulse generator.9 Recordings were obtained in spontaneous and in paced rhythms.

RESULTS

CASE 1

The patient’s phonocardiogram during ventricular pacing is shown in Fig 1. In strip A, the marked separation in time of tricuspid and mitral opening sounds is noted. These sounds are labeled in strip B. Following complex 1 of strip B, the external pacing...
FIGURE 1. Continuous recording of ECG and phonocardiogram in patient with mitral and tricuspid prosthetic valves during inhibition of implanted pacemaker by externally applied electrodes (case 1). A, Phonocardiogram during paced rhythm. B, Onset of external pacing resulting in suppression of pacemaker at beats 1 and 2 and onset of nodal rhythm with beat 3. C, Continuation of nodal rhythm and change in temporal sequence of mitral and tricuspid opening sounds. EXT PA, External pacemaker artifact; TOS, tricuspid opening sound; MOS, mitral opening sound; and ACS, aortic closing sound.

FIGURE 2A. Position of mitral and tricuspid poppets during one epicardially paced cardiac cycle with simultaneous recording of ECG and phonocardiogram (case 1). Letters refer to arrows in Figure 2B.

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artifact is visible and results in suppression of the implanted pacemaker with the appearance of nodal rhythm at beat 3. With the onset of nodal rhythm, the interval between tricuspid and mitral opening shortens and remains short throughout strip C (Fig 1), with nodal rhythm maintained.

Figure 2A shows the position of the mitral and tricuspid balls at different stages of the cardiac cycle during pacemaker rhythm. Since in Figure 2A the reproduction of the electrocardiographic and phonocardiographic detail from the video recording of the oscilloscope is not optimum, a photographic record of this same complex is reproduced in Figure 2B. The lettered arrows in Figure 2B indicate the times during the cardiac cycle at which the recordings in Figure 2A were obtained. The sequence reveals tricuspid closure (lower valve) just preceding mitral closure, and tricuspid opening preceding mitral opening by an abnormally long interval of 160 msec (Fig 2B). With suppression of pacing and the onset of nodal rhythm, the closing sequence is reversed (Fig 3A), that is, mitral followed by tricuspid, while the opening sequence is unchanged, namely, tricuspid followed by mitral, the former preceding the latter by approximately 40 msec (Fig 3B). Inspection of Figures 1 and 2B reveals that the
wide interval between tricuspid and mitral opening is predominantly a function of early movement of the tricuspid poppet, since the tricuspid opening sound precedes, rather than follows, aortic closure.

**Case 2**

Figure 4 demonstrates the apex phonocardiogram and ECG during suppression of pacemaker activity. First-degree atrioventricular block and a quiet early first heart sound are noted. The coincidence of this first sound with the upstroke of the QRS indicates that it precedes ventricular systole. In the lower tracing, obtained with the same recording intensity and filters, the onset of pacing is associated with a prominent first heart sound which coincides with the end of the QRS and is, thus, compatible with mitral closure following the onset of ventricular systole.

**Discussion**

While it would seem appropriate to discuss the auscultatory changes occurring in our patients with reference to physiologic events governing normal

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**Figure 2B.** Phonocardiogram and ECG during epicardial pacing (case 1). Arrows A through J indicate times during this particular cycle at which visual events A through J (Fig 2A) were recorded. PPA, Permanent pacemaker artifact; MCS, mitral closing sound; TOS, tricuspid opening sound; MOS, mitral opening sound; and ACS, aortic closing sound.

**Figure 3A.** Position of mitral and tricuspid poppets during one cardiac cycle (nodal rhythm) with simultaneous recording of ECG and phonocardiogram (case 1). Letters refer to arrows in Figure 3B.
atrioventricular valvular opening and closure, the grounds for comparison are limited by the different properties of normal and prosthetic valves. Unlike the prosthetic valve, opening of the normal valve is a silent event and is subject to considerably less inertia. Comparison is further limited by the fact that there is still considerable controversy regarding the relationship of normal mitral and tricuspid valvular closures to the production of the first heart sound. While recent echocardiographic studies have confirmed that mitral closure precedes tricuspid closure, the contribution of the latter to sound production remains debatable.

In this study the most clear-cut similarity between a normal auscultatory event and a prosthetic sound is the quiet closing sound in our second patient, who had a normally functioning mitral prosthesis and first-degree atrioventricular block. This finding can be compared with the anticipated quiet first heart sound in first-degree atrioventricular block in a patient with a normal atrioventricular valve. In both instances the soft sound is a consequence of presystolic valvular closure, and this mechanism has been demonstrated previously in patients with a mitral prosthesis. In our second patient the ability to initiate ventricular systole at will with ventricular pacing demonstrates that it is closure of the valve as a consequence of ventricular systole that is responsible for a normally prominent closing sound in the patient with a mitral prosthesis.

We are unaware of studies of the normal temporal sequence of events in patients with both mitral and...
tricuspid prostheses. Unfortunately, we cannot provide information on this topic in our first patient, since the rhythm was nodal and not sinus. This precluded the normal sequence of organized atrial contribution to ventricular filling; however, during nodal rhythm in this patient, the sequence of left and right ventricular depolarization would be physiologic. It is noted that the sequence of valvular closure and opening is that anticipated in a normal situation, namely, mitral closure, tricuspid closure, tricuspid opening, and mitral opening. The marked change in prosthetic sounds that occurred with pacing are those to be anticipated when right ventricular depolarization precedes left, namely, tricuspid closure and opening preceding those of the mitral prosthesis.

The event most markedly affected by the onset of pacing in this patient was the opening of the tricuspid prosthesis. This occurred approximately 100 msec earlier than during spontaneous nodal rhythm and actually preceded aortic valvular closure (Fig 1 and 2B). Since the opening sound is a consequence of right atrial pressure exceeding right ventricular pressure, it would appear probable that the premature tricuspid opening is a consequence of more rapid and effective right ventricular emptying under the influence of right ventricular pacing. The very wide separation of tricuspid and mitral opening sounds during pacing could also be attributed to delayed left ventricular activation and slower-than-normal decline in left ventricular pressure; however, there is no way to assess this factor from the data available.

Additional information will be required to further define the normal temporal sequence of events in patients with mitral and tricuspid prostheses, and it is hoped that in time the opportunity will arise to study such patients in sinus rhythm with normal intraventricular conduction and right and left bundle-branch block. We believe that the methods demonstrated here will prove useful in such studies; however, our major purpose has been to emphasize that in a patient with a demand pacemaker, marked changes in prosthetic valvular sounds may have a physiologic explanation and do not necessarily imply valvular malfunction.

References
6 Waider W, Craige E: First heart sound and ejection sounds: Echocardiographic and phonocardiographic correlation with valvular events. Am J Cardiol 35:346-356, 1975