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EXPERIMENTAL STUDIES

Mechanical Vibration Transmission Characteristics of the Left Ventricle: Implications With Regard to Auscultation and Phonocardiography

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Systolic-diastolic phasic alteration of left ventricular mechanical vibration transmissibility was studied in an open chest canine preparation. A continuous vibratory tone was applied to the base of the heart, and a miniature heart surface vibration sensor applied to the epicardium near the ventricular apex. This allowed the detection of the percent of the vibration that was transmitted from source to sensor. These data were compared with those from intracardiac phonocardiograms obtained using a micromanometer-tipped catheter. It was found that in systole, the ventricle transmitted a vibratory tone from the cardiac base to the apex so that it was readily detected by the heart surface sensor. In marked contrast, during diastole the relaxed ventricle failed almost completely to transmit the vibration to the apical position. When the dog experienced heart failure during hypoxia, the ventricular diastolic vibration transmissibility was found to equal or exceed that of the systolic phase.

Clinicians have long been aware of the transmission of heart sounds and murmurs from their cardiac source to various locations on the chest wall. It is generally assumed that transmission of heart sounds and murmurs is through the blood mass within the heart and great vessels (1). However, some take exception to this concept and attribute a preferred transmissibility to bone (2). In experiments on open chest anesthetized dogs, we studied the ability of the heart to transmit an artificial mechanical vibration. This report documents our initial findings, which we feel have implications with regard to the transmission of intrinsic cardiac sounds and murmurs.

Methods

Experimental preparation. Eleven mongrel dogs ranging in weight from 20 to 30 kg were anesthetized with intravenous sodium pentobarbital. Respiration was controlled by a mechanical respirator. The dogs were placed in the supine position and a median sternotomy was performed. The pericardium was opened and sewn so as to create a cradle for the heart. A miniature accelerometer (Entran Devices, Inc.) was attached to the exposed anterior epicardial surface using cyanoacrylate glue. The accelerometer is of the bonded strain gauge type and has a frequency response from zero to several hundred cycles per second, and a mass of approximately 1 g. This instrument has been described in a previous report from this laboratory (3). A heart surface phonocardiogram was derived from this acceleration signal by processing through a high pass filter with a corner frequency of 55 cycles/s and a roll off of 12 dB/octave. The accelerometer was attached to the anterior aspect of the left ventricular apex. The phonocardiographic signal was recorded on a direct current input channel of a multichannel recorder (Electronics For Medicine, model VR12) with a frequency bandwidth of 250 cycles/s. A micromanometer-tipped catheter (Millar Instruments, Inc.) was inserted through the left ventricular wall at the apex, and a high fidelity pressure signal was obtained. The pressure signal was recorded on the multichannel recorder with a frequency bandwidth of 250 cycles/s. In three dogs, the first derivative of left ventricular pressure (dP/dt) signal was obtained from the pressure signal by an RC differentiation circuit with a time constant of less than 0.2 ms. An intraventricular phonocardiogram was obtained from the dP/dt signal by passage through a high pass filter with a corner frequency of 55 cycles/s and a roll off of 12 dB/octave. This signal was recorded on a DC input channel of the multichannel recorder

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JACC Vol. 4, No. 3
September 1984:517-21

with a frequency bandwidth of 250 cycles/s. The electrocardiogram was recorded as a timing reference. The vagus nerve was exposed in the neck and electrodes attached. This provided a method whereby the heart rate could be intermittently slowed.

Protocol. A vibration source of constant amplitude was applied to the epicardial surface near the base of the heart. The frequency of this tone was set between 40 and 120 cycles/s. The frequency was constant throughout the protocol. In some instances, a signal representative of the voltage supplied to the vibrator was recorded on the tracing. After control studies were made, the respirator was interrupted in eight dogs to study vibration transmissibility during hypoxemia.

Results

Transmissibility, definition and description of phasic changes. For the purposes of this report, "transmissibility" is defined as the percent of vibration transmitted from source to sensor. Since the source vibration was fixed at a constant amplitude, transmissibility is proportional to the magnitude of the vibration shown in the phonocardiogram. Figure 1 shows the results obtained from 1 dog, which were typical in all 11 dogs. The heart surface phonocardiogram indicates that the base to apex transmissibility of the ventricle closely follows the pressure signal such that the tone is essentially lost at the apex in diastole. This was true even when the diastolic phase was greatly prolonged by electrical vagal stimulation (Fig. 2).

The intensity of the vibratory tone within the heart was tracked, and the results are shown in Figure 3. The intraventricular phonocardiogram and apical heart surface phonocardiogram are shown simultaneously. In the left panel of Figure 3, the micromanometer tip of the catheter is placed within the left ventricular chamber near its base. The intraventricular phonocardiogram obtained from this position shows a relatively constant vibration of the blood at the base near the source of the vibration. The right panel of Figure 3 shows the intraventricular phonocardiogram obtained when the micromanometer is moved within the left ventricular chamber to a position near its apex. At this position, the blood vibration is attenuated during diastole, so that the signal closely resembles the heart surface phonocardiogram detected by the accelerometer placed on the epicardium near the apex.

Changes in transmission with hypoxemia. In all eight dogs in which the heart was subjected to hypoxemia from asphyxia, the phasic pattern was altered such that the diastolic transmissibility equaled or exceeded that during systole (Fig. 4).

Discussion

Historical review. In the 1960s, Faber (4) studied the propagation of heart sounds in dogs by attaching several microphones to the chest wall and noting the time of arrival of the vibrations of the mitral component of the first heart sound at these locations. From the velocity of transmission, calculated by the arrival time of the vibrations at these listening sites, Faber concluded that transmission could not be by way of the blood mass, but rather through the ventricular wall. Furthermore, Faber and Burton (5) found that the amplitude of sounds was augmented by registration over a distended chamber as compared with a flaccid one. In separate studies, Zalter et al. (6) and Feruglio (7) placed sound-generating catheters in the heart and studied the trans-

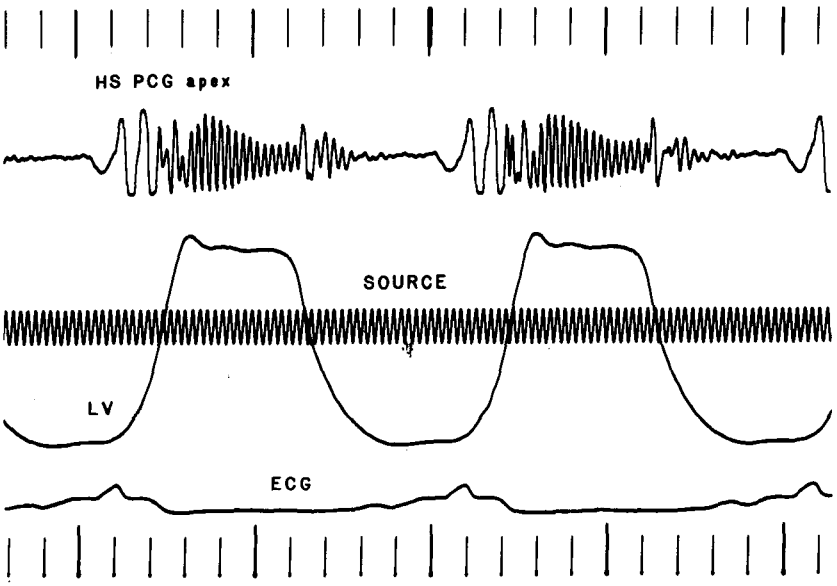

Figure 1. Recording of the heart surface phonocardiogram obtained on the anterior epicardial surface near the apex (HS PCG apex). Left ventricular (LV) pressure is shown with the electrocardiogram (ECG). A signal representative of the voltage supplied to the tone source is also shown (SOURCE).

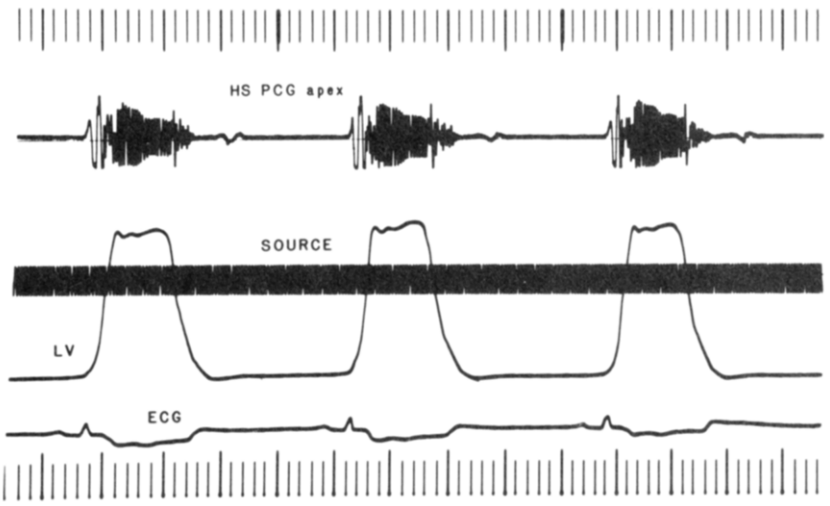

Figure 2. A recording with the same signals as in Figure 1, in which electrical vagus stimulation is used to prolong diastole. Abbreviations as in Figure 1.

mission of the resulting vibrations. No systolic-diastolic phasic variation was reported in these studies.

In 1963, Heintzen and Vietor (8) applied a vibratory tone to the anterior chest wall and, using a phonocatheter, detected the portion of this vibration that reached the intracardiac blood mass. Their results clearly indicated a phasic variation in the chest wall to intracardiac blood transmissibility.

Comparison of surface and intracardiac vibrations. During our studies, we took care to provide a constant mechanical coupling of the tone source to the base of the heart. Nevertheless, some degree of phasic fluctuation of the pressure of contact of the vibrator to the heart is inevitable. The ability of our experimental technique to provide essentially constant mechanical coupling of the tone source to the heart is shown in Figure 3. In this figure, the intraventricular phonocardiogram shows a diastolic intraventricular blood vibration near the base, which is comparable in magnitude to that during systole. As the catheter is pulled back toward the apex, the attenuation of the diastolic vibration is apparent, so that the intraventricular phonocardiogram near the apex of the chamber resembles that obtained on the epicardial surface near the apex.

Role of myocardial stiffness in transmission. It can be demonstrated through an engineering analysis that the stiffness of the myocardium plays a major role in the systolic-diastolic phasic alteration in myocardial vibration transmissibility. This is apparent in the observation that a stiff piece of wood will transmit a vibration much better than will a piece of soft foam rubber of the same density and size. It is, therefore, not surprising that a contracted and stiff muscle is capable of transmitting vibrations more efficiently than when it is relaxed and soft. This is quite easy to demonstrate

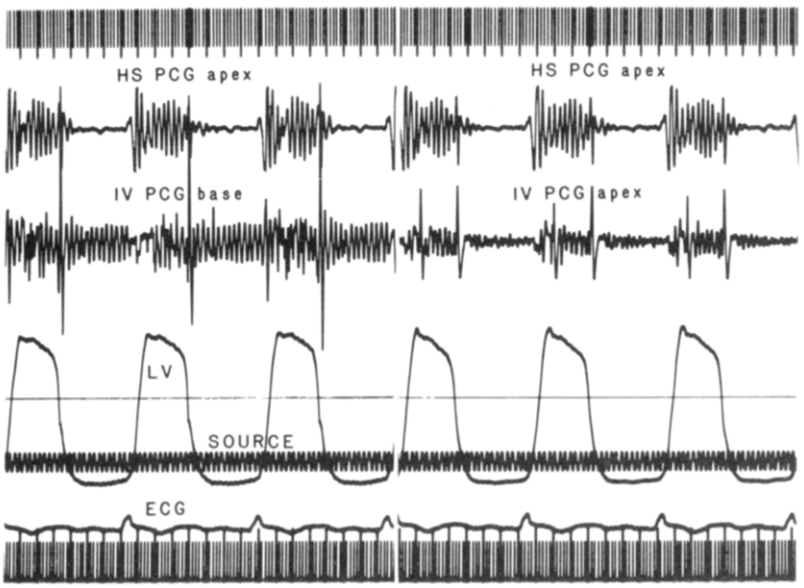

Figure 3. A simultaneous study of heart surface phonocardiography and intraventricular phonocardiography at two different positions within the left ventricular chamber. In the **left panel,** the intraventricular phonocardiogram (IV PCG) is recorded near the base of the heart, near the tone source. A diastolic vibration from the tone source is shown which essentially equals that of systole, indicating a constant coupling of the vibration source to the base of the heart. The **right panel** shows the intraventricular phonocardiogram near the ventricular apex. At this position, the diastolic tone vibration is much attenuated, such that the intraventricular phonocardiogram at this position resembles that at the apical epicardium (HS PCG). Other abbreviations as in Figure 1.

JACC Vol. 4, No. 3
September 1984:517–21

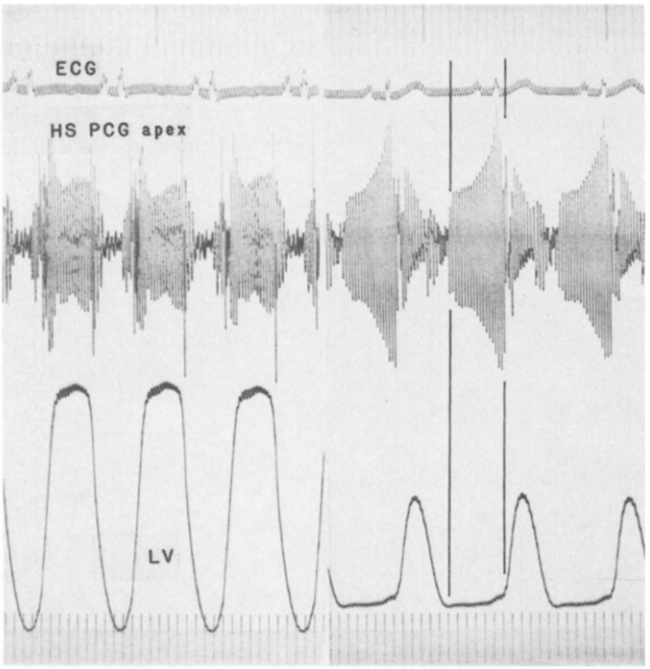

Figure 4. The **left panel** shows the systolic dominance of transmissibility that occurs in the control condition. The **right panel,** recorded during heart failure subsequent to hypoxemia, shows paradoxical enhancement of diastolic vibration transmissibility. Abbreviations as in Figure 1.

in the case of skeletal muscle by applying a vibration source to the skin overlying the muscles of the forearm and applying a vibration sensor (or a conventional stethoscope) to the skin of the forearm at some distance from the source. When the arm is flexed, the vibration is transmitted along the muscle much more efficiently as compared with the relaxed condition. Templeton et al. (9) demonstrated that the normal systolic ventricular chamber stiffness is at least 3 times greater than that of diastole. In view of this, it is to be expected that the systolic-diastolic phasic variation in ventricular transmissibility should be quite significant. Intrinsic heart sounds and murmurs cannot serve to demonstrate a phasic variation in transmissibility because they do not provide a constant source of vibration throughout the cardiac cycle. Through the use of an artifical tone source, however, a profound phasic alteration can be clearly shown.

Possible role of resonance of the left ventricle. To investigate the possibility that the transmission patterns were the result of resonance of the left ventricle, we performed the experiments at many different frequencies ranging from 40 to 120 cycles/s. The results were the same for each of these frequencies, suggesting that a resonance phenomenon is not responsible for the observation described.

Possible explanation for diastolic enhancement. We are unable to determine the relative roles of stiffness, viscosity and density in causing the patterns of transmissibility that we observed in this preliminary study. It is evident, however, that substantial phasic alterations in physical properties occur that have a profound influence on the ability of the heart to transmit mechanical vibrations. Furthermore, the phasic pattern of transmissibility can be significantly altered by certain pathologic conditions such that diastolic transmissibility is greatly enhanced.

The paradoxical enhancement of diastolic transmission that we observed is most probably caused by a change in the intrinsic material characteristics of the myocardium, perhaps as a result of impaired relaxation associated with global ischemia. In this regard, it is worth noting that when the tone source and sensor are left in place as the heart dies and becomes completely inert, the transmissibility is seen to diminish gradually to almost zero despite the fact that the heart remains distended and engorged with blood.

Role of wall stiffness in intracardiac blood transmissibility. When a vessel with stiffness characterized by Young's modulus holds a fluid with a stiffness characterized by its bulk modulus and the vessel stiffness is much less than that of the fluid, the effective stiffness of the vessel-fluid combination is limited and determined by the vessel stiffness. This is apparent in the observation that the velocity of pressure-pulse propagation along the aorta is between 3 and 8 m/s, as compared with the velocity of a compressional wave within the blood of about 1,500 m/s (10). The velocities of heart sounds in general indicate that they travel as transverse vibrations along the various structures, including the ventricular wall (10). This suggests that transmission of sound through the intracardiac blood in the frequency range pertinent to cardiac sounds and murmurs is also mediated by myocardial wall stiffness, which is indicated by the intracardiac phonocardiogram of Figure 3. It is not clear from our results whether the intracardiac vibration near the apex reaches this position primarily by transmission through the intracardiac blood mass or along the myocardium. However, regardless of the route of transmission, it is shown that at this distance from the tone source, a diastolic attenuation of the tone has occurred within the blood.

Clinical applications. We believe that our results have direct implications in the study of auscultation and phonocardiography. For instance, the distribution and intensity of systolic versus diastolic murmurs on the anterior chest surface should be significantly affected by the much greater transmissibility of the contracted ventricle. Furthermore, substantial differences in results are to be expected when intracardiac phonocardiograms are used to record cardiac sounds and murmurs as compared with phonocardiograms obtained on the anterior chest. This is demonstrated by Figure 3, in which the intracardiac phonocardiogram recorded near the base of the ventricular chamber is a very poor indicator of the vibratory pattern seen on the heart surface near the apex.

In earlier studies from this laboratory (11), the fourth heart sound was found in association with abnormally stiff

ventricular chambers in late diastole. In view of the results of the present study, it is possible that a paradoxical enhancement of diastolic transmissibility might play a role in the appearance of this vibration on the precordium.

Implications for future research. We believe that the study of ventricular vibration transmissibility can provide insight into the complexities of myocardial contraction and relaxation and alterations due to pathologic conditions in a functioning in vivo preparation. Thus, assessment of ventricular vibration transmissibility can potentially provide information beyond the scope of auscultation and phonocardiography.

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