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Relation of physical, physiological and anatomic factors to auscultatory evaluation

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The intensity of the heart sound depends upon: 1) the distensibility of the aortic and pulmonary valves; 2) hemodynamic factors that cause the valves to distend and vibrate; 3) viscosity of the blood and its ability to inhibit diastolic valve motion; 4) the configuration of the aorta, pulmonary artery, and ventricle and the ability of the walls of the great vessels and ventricles to absorb or reflect sound energy; and 5) the capability of sound to be transmitted to the chest wall. Recognizing how these physical, physiological, and anatomic factors interact can help us to interpret auscultation of the intensity of the second heart sound.

Understanding the physiological, physical, and anatomic factors that produce the second heart sound is important to properly interpret its intensity during physical examination (1). An incomplete understanding of the mechanism which produces the second sound has resulted in failure to optimally interpret auscultation of this sound. We now know that it results from vibrations of the closed aortic and pulmonary valves (2). Silent coaptation of the semilunar valves is followed by vibrations of the closed cusps which result in transient pressure fluctuations. These vibrations are transmitted to the chest wall, where they produce compression and rarefaction of the air, which in turn produce sound.

To explain the mechanism of the second sound, Wiggers speculated that "the valve closes silently, and aftervibrations of the closed valves and column of blood produce the second sound" (3). Others have suggested that the vessel walls and infundibulum vibrate, as well as the column of blood and the semilunar cusps (4). The critical distinction is that the vibration of the column of blood and other structures, if they occur, are *secondary. The primary, initiating source of vibration is the semilunar valve itself.* Auscultation of the second sound can be fully interpreted only if the valves are considered the primary source of vibration.

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Silent Closure and Vibration of the Aortic Valve

In vitro, high-speed motion pictures of a normal porcine valve mounted in a hydraulic model of the cardiovascular system have shown that the second sound begins after the valve closes (2). This is compatible with the concept that the valve closes silently, and sound occurs with vibrations that follow closure. Initial motion of the closed cusps, as they begin to vibrate, is always characterized as distension toward the ventricle (5). In every case, the acoustic pressure signal, which we refer to as sound, when recorded in the left or right ventricle, appears as a compression signal. Simultaneously, the pressure transducer within the aorta or pulmonary artery shows the initial deflection on the acoustic pressure signal as a decompression signal (2,6). This signal indicates that the semilunar valves initially deflect toward the ventricle, causing a transient compression on an acoustic pressure sensor within the ventricle, and a transient decompression on a similar sensor within the aorta or pulmonary artery. With recoil of the semilunar cusps toward the aorta (or pulmonary artery), the opposite action occurs.

Pressure transients, which are detectable upon the ventricular and aortic (or pulmonary artery) pressure recordings (6), reflect the pressure changes within the ventricle and great vessels as the valve vibrates.

In fact, these pressure transients, when amplified and processed through a high pass filter, are represented as an acoustic pressure signal by an intraluminal micromanometer.

The most compelling evidence that valve vibration produces the second sound comes from its actual visualization on high-speed motion pictures in a hydraulic model of the cardiovascular system (5). High-speed (2000 frame/sec) motion pictures were obtained of a normal porcine valve mounted in a flow system which simulated aortic and ventricular pressures and aortic flow. Initially, the closed aortic cusp deflected toward the simulated left ventricle. Then, the cusp recoiled toward the aorta but did not reach the initial position that it attained at closure. Damped vibrations followed.

The velocities at which the valve cusps moved and recoiled were calculated from the curve that showed valve displacement as a function of time. The curve that describes the speed of distension and recoil of the cusps was strikingly similar to the simultaneously recorded acoustic pressure signal (5). This correlation was predictable from analogies of acoustic pressure produced by motion of a piston in an infinite baffle (7), in which the acoustic pressure is proportional to the velocity of the piston.

Factors Affecting Valve Vibration

Recognizing that the second sound is produced by pressure changes due to diastolic vibration of the closed semilunar cusps, we explored the factors that produce or modify these vibrations. We identified these factors by mathematically modeling the vibrations of the closed valves, assuming that they vibrate as a stretched circular membrane (7). These factors were investigated in vitro, in dogs, and in patients. As a result, we have been able to explain several auscultatory signs. The primary hemodynamic factor which determines the extent and rate of deflection of the cusps is the rate at which a pressure gradient develops between the aorta (or pulmonary artery) and the ventricle after valve closure. Anatomical factors also were identified by mathematical modeling of diastolic valve motion (7). Sound intensity was strongly dependent upon valve diameter, while the acoustic pressure signal varied both as the fourth power of the valve diameter and inversely as the square root of the stiffness and mass of the valve. We also found that the viscosity and the density of the blood affected the vibrations (7).

The important aspect of mathematical modeling is that it allowed us to identify previously unrecognized factors that affect the intensity of the second sound. Quantitative relations in the modeling depend upon assumptions in the model, and as more input data become available, these relationships may be refined.

Transient sounds

Transient sounds that followed the main component of the second sound occurred on intra-arterial sound recordings in patients during cardiac catheterization (2) as well as on phonocardiograms obtained at the chest wall. Studies in vitro also showed that transient sounds followed closure of a stent-mounted porcine valve. Coincident with these transients, we observed vibrations of the valve in vitro (5). It is apparent, therefore, that these sound transients are due to continuing vibration of the semilunar cusps.

Patients with poor ventricular performance

The primary hemodynamic factor that determines the extent of the distension and velocity of the cusp toward the ventricle (7) is the rate of development of a diastolic pressure gradient across the closed aortic (or pulmonary) valve. The amplitude of the acoustic pressure signal, therefore, depends upon this hemodynamic parameter. It has been shown that the rate of change relates to the amplitude of the second sound in dogs (8,9) and in patients (10).

We explored the way in which this hemodynamic factor causes the second sound to change in patients with impaired ventricular performance, particularly following myocardial infarction. In myocardial infarction, isovolumic relaxation is impaired (10). Relaxation may be considered a vital aspect of contractile performance (11). Because aortic diastolic pressure changes little in early diastole relative to the rapidly changing left ventricular pressure, the rate of change of the diastolic pressure gradient across the aortic valve depends largely upon left ventricular isovolumic relaxation (10). When isovolumic relaxation is impaired, as in myocardial infarction, the rate of change of the diastolic pressure gradient is impaired. Thus, the primary determinant of the velocity with which the closed aortic valve cusps will distend is also impaired, and the aortic component of the second sound is diminished. This relationship accounts for the tic-tac rhythm in myocardial infarction and the reduced second sound even in patients with normal arterial pressure (10).

Based on this information, we investigated whether changes of the heart sounds following myocardial infarction can be detected at the chest wall and whether they reflect impaired isovolumic relaxation (12). We used calibrated microphones, held in position by a suction cup, which permitted repetitive measurements of sound without significant changes of intensity. The aortic component of the second sound (A_2) in patients who suffered a recent myocardial infarction was significantly lower in intensity than A_2 in normal subjects. Others, too, have measured a significant difference of sound following infarction (13). The difference did not relate to blood pressure, since it did

not differ significantly between normal subjects and patients with infarction (12, 13). With noninvasive methods, by measuring the time of A_2 to the O-point of the apexcardiogram (the point at which the mitral valve opens), we could measure the isovolumic relaxation period (12). The ratio of diastolic blood pressure to the relaxation period measured the average rate of isovolumic relaxation. A prolonged average rate was shown to accompany the diminished A_2 in patients with myocardial infarction. Among the patients whose A_2 improved in intensity after the infarction, the relaxation rate increased. Thus measurable changes of A_2 were consistently recorded at the chest wall which varied according to changes in isovolumic relaxation (12) and which were in the range ordinarily detectable with normal hearing. Reductions of sound intensity often occurred even if a gallop rhythm or rales were absent. It therefore appeared to be a useful, complementary auscultatory sign.

In our study of heart sounds measured at the chest wall of patients following infarction, we also observed a diminished pulmonary component of the second sound (P_2) in comparison to normal subjects (12). The hemodynamic factor most directly related to the amplitude of P_2 is the rate of change of the pressure gradient across the pulmonary valve, which reflects right ventricular isovolumic relaxation (14). We believe that the reduced P_2 we observed after infarction represents impaired isovolumic relaxation of the right ventricle. Studies of both laboratory animals and patients indicate that occlusion of the right coronary artery affects right ventricular contractile performance (15, 16). This may be a method for identifying right ventricular infarction (16). Since, from our observations, such impaired performance seems to be frequent (12), measuring sound intensity at the chest wall may be a useful method for detecting it.

Calcific aortic stenosis

In calcific aortic stenosis, the intensity of A_2 is reduced, whereas in congenitally stenosed valves that have not yet undergone degenerative changes, the intensity of A_2 is normal (10). This apparent paradox is readily explained by the sound produced by diastolic vibration of the closed cusps. Because calcified stenotic valves are stiff and thickened, these characteristics would impair valve vibration (7). We mounted a heavily calcified stenotic human valve in a hydraulic model of the cardiovascular system to study its sound and vibration (10). High-speed motion pictures showed no vibration, and sound recorded near the valve was of very low amplitude. By contrast, a valve made stenotic by suturing the leaflet showed normal diastolic vibrations and produced normal sound. Clearly, calcified and deformed stenotic valves have a diminished ability to vibrate. Such valves are associated with an attenuated A_2.

It is likely that other factors contribute to the diminished A_2 in patients with calcific aortic stenosis. In a few patients in whom we were able to pass a catheter-tip micromanometer to the left ventricle, isovolumic relaxation was impaired. Consequently, the rate of development of the pressure gradient across such valves would be attenuated. This too would contribute to the diminished vibrations and the reduced sound.

Pulmonary hypertension

Recognizing that the physiology of the pulmonary component of the second sound is identical to that in A_2, we can explain characteristics of the intensity of P_2 that were paradoxical and previously unexplained. If the intensity of P_2 and A_2 depends upon the diastolic blood pressure, why does the intensity of P_2 exceed that of A_2 in pulmonary hypertension, since pulmonary artery diastolic pressure rarely exceeds aortic diastolic pressure? The explanation is that in patients with pulmonary hypertension, the rate of development of the diastolic pressure gradient across the closed pulmonary valve is higher than in patients with normal pulmonary arterial pressure (14). Consequently, P_2 increases in intensity because the pulmonary valve distends at a faster rate. However, the rate of change of the diastolic pressure gradient across the pulmonary valve, even in pulmonary hypertension, does not exceed the rate of change across the aortic valve in patients with normal aortic pressure (14). Therefore, other factors must permit P_2 to exceed A_2, including the cross-sectional area of the valve and its greater distensibility (7,14). Since the pulmonary valve is larger than the aortic valve, it is more distensible (14).

Consequently, a given distending force will cause the pulmonary valve to distend further and at higher speed. Even a lower rate of change of the diastolic pressure gradient across the pulmonary valve will cause the larger pulmonary cusps to vibrate with greater amplitude and a faster rate of deflection. The larger surface area of the pulmonary valve produces pressure changes of higher energy with any given vibration. For these reasons, the pulmonary component of the second sound exceeds the aortic component in pulmonary hypertension (14).

Accentuated heart sounds in anemia

Analysis of physical factors that affect vibration of a membrane in a liquid indicate that viscosity is a factor (17). Predictably, liquids with higher viscosity would diminish the speed and extent of deflection of the closed cusps with any given driving force. When we studied high-speed motion pictures of diastolic vibration of the cusps of a normal porcine valve (17), the extent and speed at which the closed cusps distended toward the ventricle were di-

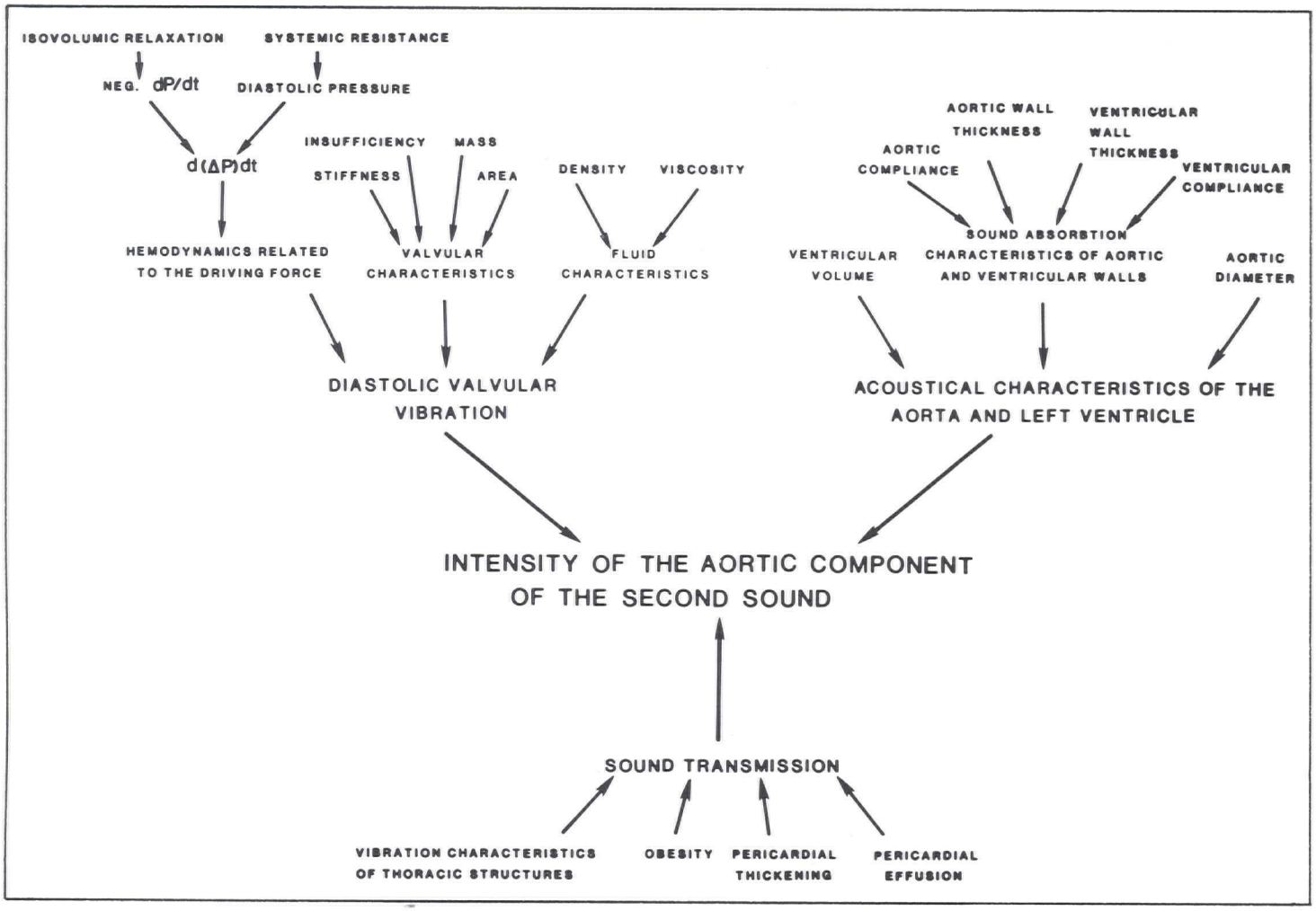

Fig. 1
Factors affecting intensity of the second heart sound.

minished in more viscous liquids. Conversely, vibrations were augmented in less viscous liquids. Thus, we predicted that heart sounds would be accentuated in anemic patients (17), since the viscosity of blood varies with the hematocrit (18). Using calibrated microphones uniformly affixed to the chest wall with a suction cup, we found that all heart sounds were accentuated in anemic patients (17). Since several factors affect both the viscosity of blood and the hematocrit (18), the heart sounds predictably would be affected in a comparable fashion.

According to our physical analysis, the density of blood would also affect the vibration (7), although variations of density are small. In vitro tests of valve vibration showed that wide variations of density are required for noticeable changes of sound. Such density variations exceeded the physiological range.

Other Factors Affecting the Second Heart Sound

The second heart sound may be affected by the stiffness of the aorta (4). The modeling suggests that the compliance, diameter, thickness, and sound absorption characteristics of the walls of the great vessels and the ventricles may also affect the second sound (7).

Factors that affect sound transmission, such as obesity, pericardial effusion, and emphysema (19), also affect interpretation of auscultation.

The intensity of the second heart sound, therefore, depends upon: 1) the distensibility of the aortic and pulmonary valves; 2) hemodynamic factors that cause the valve to distend and vibrate; 3) viscosity of the blood and its capacity to inhibit diastolic valve motion; 4) the general configuration of the aorta, pulmonary artery, and ventricle

and the ability of the walls to absorb or reflect sound energy; and 5) the capacity of sound to be transmitted to the chest wall. Numerous factors modify these variables (Fig. 1). Recognizing how these physical, physiological, and anatomic factors interact contributes to a meaningful interpretation of auscultation of the intensity of the second heart sound.

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