Implications of Faint Heart Sounds After Acute Myocardial Infarction

Master cardiologists have recognized for many years that heart sounds, particularly the first heart sound may be faint or weakened shortly after acute myocardial infarction (AMI).[1,2] This, of course, assumes that other causes of decreased heart sounds have been excluded. The intensity of both the first and second heart sounds has been measured in patients hospitalized for myocardial infarction and both were shown to be lower intensity than in normal subjects.[3] It was obvious to cardiologists that in some unexplained way the faint heart sounds resulted from impaired ventricular function. The mechanism became clear when it was shown that the aortic valve, and by implication the mitral valve, close silently, and after coaptation vibrate.[4] These vibrations initiate vibrations elsewhere in the cardiovascular system, and are responsible for heart sounds.[4−6] The amplitude of the aortic component of the second heart sound is directly proportional to the speed at which the closed aortic valve distends toward the left ventricle as vibration is initiated.[5] In a comparable way, the amplitude of the mitral component of the first heart sound is directly proportional to the speed at which the closed mitral valve distends toward the left atrium. The driving force for the speed at which the mitral valve distends toward the left atrium after closure is the rate at which a pressure drop develops across the closed mitral valve. This is directly proportional to left ventricular isovolumic rate of change of pressure (dp/dt) at the instant of closure. The amplitude of the mitral component of the first heart sound has been shown to be directly proportional to peak left ventricular dp/dt,[7] which approximates left ventricular dp/dt at the instant of closure. The decreased amplitude of the mitral component of the first heart sound after myocardial infarction, therefore, results from a diminished left ventricular dp/dt, a well-established index of contractility. This is concordant with the presumption that a faint mitral component of the first heart sound after myocardial infarction reflects poor left ventricular performance, and requires no further discussion. The implication of a low intensity aortic component of the second heart sound, however, requires further consideration.

In the case of the aortic valve, the rate of the pressure drop across the closed aortic valve at the instant of valve closure closely corresponds to the rate of pressure fall in the left ventricular, negative dp/dt, at the instant of valve closure.[8] Therefore, the decreased intensity of the aortic component of the second heart sound in patients shortly after a myocardial infarction relates to impaired isovolumic relaxation.[8] What causes the impaired isovolumic relaxation? What is its significance?

Before considering the significance of impaired isovolumic relaxation, it is useful to identify other factors that can affect the intensity of heart sounds or the transmission of heart sounds. Factors that can affect vibration of the closed cusps and consequently the intensity of heart sounds are valve stiffening as in calcific stenosis and valve insufficiency.[9,10] The viscosity of blood, which is affected by the hematocrit, affects the speed at which the closed semilunar valves distend, and presumably the speed at which the closed mitral and tricuspid valves distend.[11] Transmission of heart sounds can be impaired by obesity or pericardial effusion. Other cardiac diseases, such as nonischemic cardiomyopathy, can result in impaired isovolumic relaxation and impaired isovolumic contraction. Although low intensity heart sounds in the proper setting may hint that AMI occurred, the goal of this communication is not to use low intensity sounds for diagnosis, but to explain the pathophysiological significance the low intensity heart sounds, assuming that AMI occurred.

Stiffening of the left ventricle would not explain impaired isovolumic relaxation shortly after an acute infarction when decreased heard sounds were described clinically,[1,2] and measured.[3] Stiffening of the left ventricle perhaps occurs with scarring and collagen deposition, but this is a late process. Impaired isovolumic relaxation occurs instantly with brief occlusion of a coronary artery.[12]

There has been considerable evidence since as long ago as 1930, that self-enlargement of the left ventricle, termed diastolic suction, causes early filling of the left ventricle.[13] During early diastole, left ventricular diameter increases in dogs whereas left ventricular pressure continues to fall.[14] This is not compatible with passive filling. Rather it is an active process. Cardiac muscle relaxation is dependent on reducing the concentration of free calcium ions and subsequent inhibition of myofibrillar adenosine triphosphatase.[15]

Myocardial ischemia induced by brief balloon occlusion of a coronary artery results in abnormal myocardial diastolic relaxation.[12,16] Impaired isovolumic relaxation and diastolic dysfunction occur together and appear to be part of the same process.[12] Slowing of left ventricular relaxation and loss of restoring forces reduce the rate of left ventricular isovolumic relaxation.[17] Decreased left ventricular restoring forces deprive the left ventricle of its early diastolic suction mechanism.[17]

Putting these diverse observations together, the direct cause of the low intensity mitral component of the first heart sound shortly after AMI is low left ventricular dp/dt, and the direct cause of the low intensity aortic component of the second heart sound is low left ventricular negative dp/dt (impaired isovolumic relaxation). A pathophysiological interpretation of the low intensity mitral component of the first heart sound is impaired ventricular contraction, and the pathophysiological interpretation of the low intensity aortic component of the second heart sound is impaired diastolic suction. Simple auscultation, therefore, can identify impaired systolic and diastolic function, which otherwise require imaging modalities which have their own strengths and weaknesses.[17]

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