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Abnormal diastolic mechanical vibration transmission characteristics of the left ventricle

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Summary

In 18 open chest canine experiments, the ability of the left ventricle to transmit a mechanical vibratory tone from base to apex has been studied. An artificial tone source of constant amplitude and frequency was applied to the base of the exposed left ventricle. A miniature vibration sensor was applied to the anterior epicardium near the ventricular apex. This permitted us to obtain a heart surface phonocardiogram, in order to detect the portion of the source vibration which was transmitted to the apex. In three of the dogs, the heart surface phonocardiogram from the apex was compared with simultaneous intraventricular phonocardiograms which showed the amplitude of the vibration which reached the intraventricular blood mass.

It was found that the normal ventricle transmits the tone from base to apex almost exclusively during systole, when the ventricle is contracted and stiff. In marked contrast, the normally relaxed and soft ventricle fails almost completely to transmit the tone to the apex.

In conditions of poor relaxation ability of the left ventricle due to global hypoxemia, manifested by a long " Time Constant T ", an abnormal diastolic transmission of the tone to the apex occurred during early diastole. We have defined this abnormal early diastolic " crescendo-decrescendo " type of transmissibility as " Type 1 ". The Type 1 transmission is related to impaired relaxation of the ventricle and is a manifestation of one or more abnormal ventricular muscle properties, such as myocardial stiffness, viscosity and density.

In conditions of left ventricular hemodynamic failure caused by global hypoxemia, a separate abnormal mid-to-late diastolic " crescendo " type of transmissibility was found, and is defined as " Type 2 ". This Type 2 transmission is associated with elevated end-diastolic pressure, perhaps in conjunction with abnormal diastolic myocardial properties.

Key words

Mechanical vibration Left ventricle Transmission Diastole Ischemia Phonocardiography Auscultation

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Introduction

In a previous report, we have described our experimental technique and initial findings concerning the transmission of a vibratory tone from the base to the apex of the left ventricle (LV) in the open chest canine[1]. The present report is a more detailed description of the types of diastolic abnormalities in LV base-to-apex transmissibility which we have observed during global hypoxemia caused by interruption of the mechanical respirator.

Methods

Eighteen dogs weighing from 20 to 30 kg were anesthetized with intravenous sodium pentobarbital, and placed on a mechanical respirator. The dogs were placed in the supine position and a median sternotomy was performed. The pericardium was opened and sewn to create a pericardial cradle. A miniature accelerometer was attached by cyano-acrylate glue to the anterior surface of the epicardium at the LV apex. This accelerometer has been described in previous reports from this laboratory[2,3]. The accelerometer and its associated plastic attachment plate have a mass of approximately 1 gm, and is ideally suited for studies of heart surface vibrations. In three dogs, a high fidelity micromanometer tipped catheter (Millar) was inserted into the LV chamber directly through the LV wall at the apex, and an intraventricular phonocardiogram was obtained. A vibration source of constant amplitude was placed in contact with the basal aspect of the left ventricle, near the aortic root. This arrangement is shown schematically in **Fig. 1**, in which the micro-manometer tip of the catheter is placed within the chamber near the base of the ventricle. In some instances, an additional micro-manometer tipped catheter was inserted from the right carotid artery and situated above the aortic valve, to obtain an intra-aortic phonocardiogram from the proximal aortic root. This allowed an assessment of the amplitude of the tone vibration reaching the blood of the proximal aorta. The frequency of the vibration tone source was selected within the the range of 40 to 120 cps., and was kept constant throughout the protocol. Experiments were done during the control condition and during the time of global ischemia and heart failure caused by interruption of the mechanical respirator.

Results

During the control condition, the heart surface phonocardiogram indicated that the left ventricle transmitted the vibration from base to apex almost exclusively during systole. This is shown in **Fig. 1**. The intraventricular phonocardiogram obtained simultaneously near the base of the ventricular chamber shows that the vibration within the blood near the base is essentially continuous. This indicates that the intermittent appearance of the tone at the apical heart surface is not caused by sporadic contact with the vibration source, and confirms that the diastolic absence of the apical surface vibration is a result of very poor diastolic base-to-apex transmissibility in the normal condition. As discussed in our earlier report[1], the same result is obtained at any frequency of the tone source which we have studied, within the range of 40 to 120 cps. The result is not, therefore, the result of a resonance phenomenon.

Fig. 2 shows the results obtained when the LV has impaired relaxation due to global ischemia but is not yet in hemodynamic failure. The left panel of figure is the control condition, in which the " Time Constant T "[4] is approximately 55 msec. The right panel of this figure shows the abnormal diastolic transmission pattern when the Time Constant T is approximately 120 msec, indicating impaired muscular relaxation. At this time, the early diastolic period, during isovolumic relaxation, is accompanied by a persistence of tone transmission exceeding that of systole, and decaying away exponentially as the LV pressure curve reduces to its diastolic level. We have defined this type of " crescendo-decrescendo " early diastolic transmission pattern as " Type 1 ".

Fig. 3 shows the results obtained when the global ischemia produces LV hemodynamic failure. Following the " Type 1 " pattern in

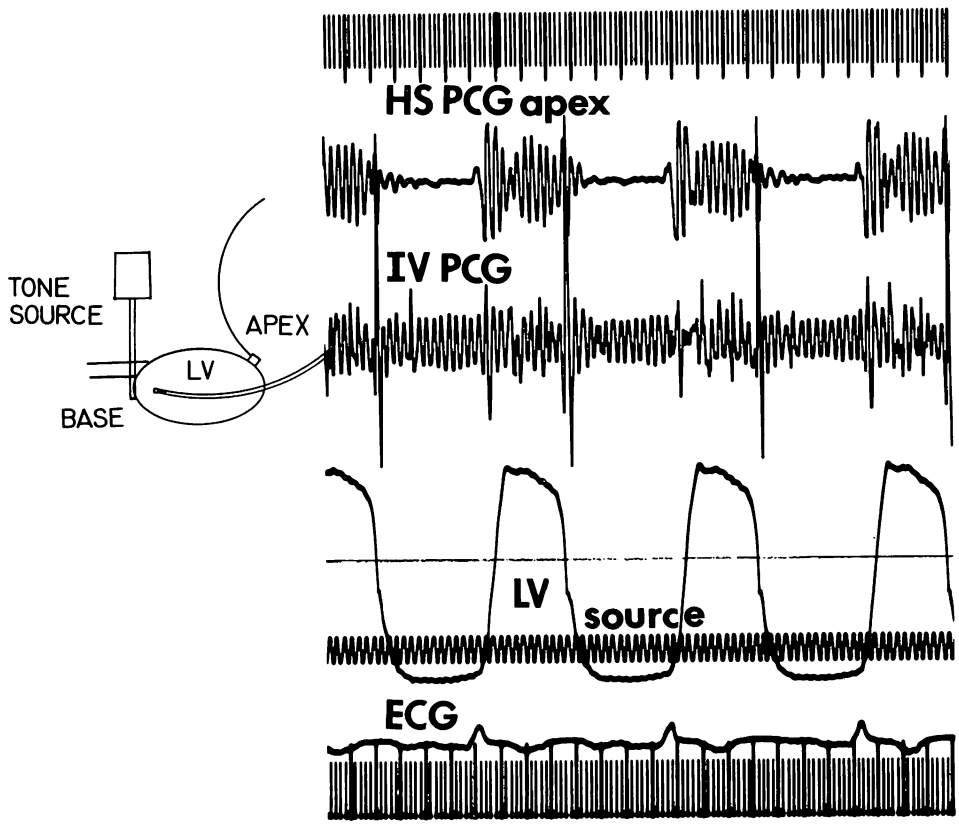

Fig. 1. Simultaneous heart surface phonocardiogram at the apex (HS PCG-apex) **and intraventricular phonocardiogram from the micro-manometer tipped catheter** (IV PCG).

The IV PCG is recorded at the base of the LV chamber. Also shown are LV pressure (LV) and a signal of the voltage supplied to the tone source (SOURCE). The electrocardiogram Lead II (ECG) is also shown. The HS PCG apex signal indicates transmission of the tone to the apex exclusively in systole, while the IV PCG signal shows that the intraventricular blood near the base of the LV chamber has essentially a constant vibration from the tone source.

early diastole, the transmission begins to grow in mid-diastole. This " crescendo " pattern has been defined as " Type 2 ". At moderate heart rates, the Type 2 pattern continues to grow until the next systole. At very low heart rates, with the resulting long diastolic phase, the Type 2 pattern plateaus to a constant amplitude, sometimes with an additional enhancement at the time of the next atrial contraction. In **Fig. 3**, however, we have noted an unusual pattern in the Type 2 transmission. The electrocardiogram (lead II) in this figure shows an unusual inflection in mid-diastole, as indicated by the arrow. This inflection may be a very late " U " wave. At the time of this diastolic electrical activity, a distinct alteration is seen in the transmission pattern. The accompanying intra-aortic phonocardiogram substantiates that this transition is not the result of a change in contact of the vibration to the heart, since the intra-aortic blood vibration is essentially constant in mid-to-late diastole. Thus, the transition in the heart surface phonocardiogram indicates an abrupt change in the

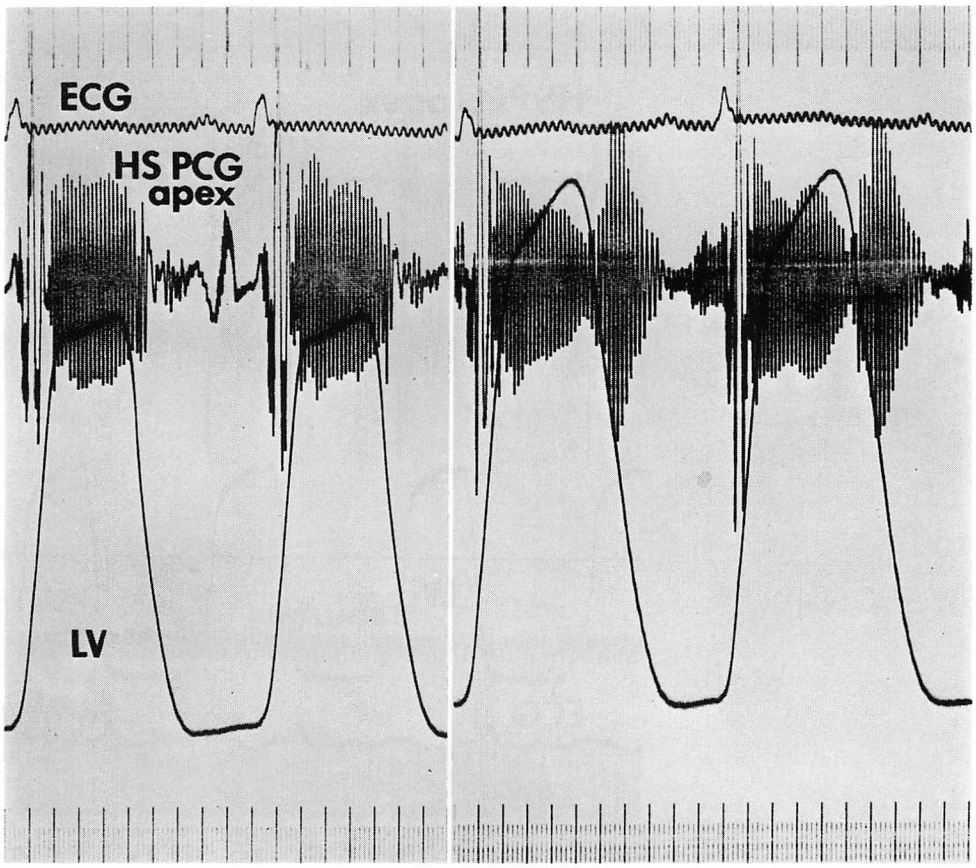

Fig. 2. Phonocardiograms demonstrating " Type 1 " diastolic transmission abnormality.
The left panel shows the control condition. The right panel shows the characteristic " Type 1 " diastolic transmission abnormality during isovolumic relaxation. Abbreviations as in Fig. 1.

physical properties which determine the base-to-apex transmissibility, associated with the electrical activity manifest as the inflection in the ECG.

Discussion

The normal ventricle usually displays a small and rapidly decaying transmission during isovolumic relaxation. The Type 1 abnormality is, then, the result of a very large exaggeration of the normally small isovolumic transmissibility. The association of the Type 1 pattern with abnormally long " Time Constant T " suggests that it is due to impaired myocardial relaxation. The pattern is, however, not simply the result of a continued systolic contraction, since the magnitude of the transmission is often larger than the transmission occurring at any time during systole, as indicated in **Fig. 2**. We are unable at present to determine which of the myocardial properties are altered at the time of this Type 1 transmission. We believe that myocardial stiffness has been altered during Type 1, but the myocardial viscosity and density may also be important in this phenomenon.

The Type 2 transmission phenomenon is associated with LV hemodynamic failure and appears to be directly associated with diastolic

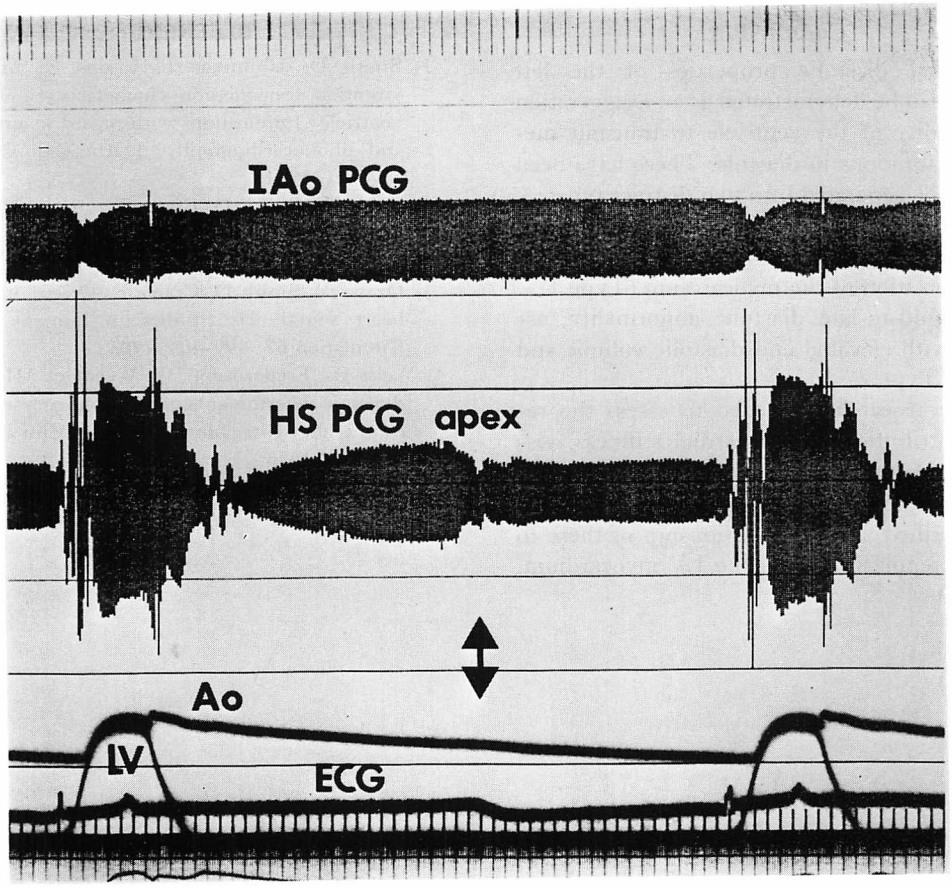

Fig. 3. Phonocardiogram demonstrating " Type 2 " diastolic transmission abnormality.

This tracing demonstrates the " Type 2 " diastolic transmission abnormality in mid-to-late diastole, in addition to the " Type 1 " abnormality during early diastole. An inflection in the ECG signal in mid-diastole is associated with a distinct alteration in the Type 2 transmission pattern (arrow). The intra-aortic phonocardiogram (IAo PCG), recorded at the proximal aortic root, indicates a constant amplitude of the source vibration, as well as constant mechanical coupling of the source to the heart, at the time of the alteration in the Type 2 transmission pattern (arrow). Other abbreviations as in Fig. 1.

engorgement of the LV chamber as the end-diastolic volume and pressure become abnormally elevated. We have noted that clamping of the inferior vena cava has a direct and profound effect on the presence and amplitude of the Type 2 diastolic transmission. When the LV end-diastolic volume and pressure are reduced due to the clamping of the inferior vena cava, the amplitude of the Type 2 transmission is greatly reduced.

We are particularly interested in the relationship of the mid-diastolic electrical activity and the alteration in the pattern of the Type 2 transmission shown in **Fig. 3**. It is suggested by this figure that electrical repolarization of the myocardium is directly associated with a transition in the diastolic physical properties of the myocardium.

Conclusion

Abnormal diastolic properties of the left ventricle can be demonstrated as an exaggeration of the ability of the ventricle to transmit mechanical vibrations in diastole. These have been shown to be separated into two distinct types:

1. An early diastolic abnormality, during isovolumic relaxation, in association with impaired relaxation of the myocardium (Type 1).
2. A mid-to-late diastolic abnormality, associated with elevated end-diastolic volume and pressure (Type 2).

Further research is needed to assess the relative contribution of myocardial stiffness, viscosity, density, and ventricular volume in producing the transmission patterns that we have described, and the relationship of these to electrical repolarization of the LV myocardium.

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