Chest Wall Velocity and the Second Heart Sound
An Improved Sensor of S2 Splitting

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SUMMARY We report a new method of detection of the timing of the aortic and pulmonary valve closure that depends not on the registration of audible vibrations, but rather, on subtle but distinct movements of the chest wall, which are external manifestations of these events. We studied these phenomena in six open-chest dogs and in 69 human subjects. The dog studies show that the two distinct inward movements detected by a motion sensor applied to the epicardium in the vicinity of the right ventricular outflow tract correlate with the timing of the incisural notches of the pressure signals from the great vessels. In humans, these movements are transmitted to the skin surface and can be detected noninvasively. In 48 of the 69 human subjects (70%), these spikes provided a significantly better indication of the timing of semilunar valve closure than did the conventional phonocardiogram.

THE SECOND HEART SOUND (S2) has been recognized for over a century as a key feature of the physical examination.1 In 1866, Potain2 attributed its two components to closure of the aortic and pulmonary valves. Leatham3 stressed the significance of respiratory variations in the splitting interval that separate these components, and the association of abnormal splitting characteristics with certain disease states is well known.

The identification of the two components of S2 may be obvious by auscultation or by phonocardiography. Frequently, however, delineation is uncertain or impossible, particularly in the presence of chronic pulmonary disease or when a murmur interferes with the S2.

Because the time of closure of the pulmonary valve is only rarely seen with echocardiography, the identification of the closing time of this valve, and thus the splitting interval, often cannot be accurately determined in the noninvasive graphics laboratory.

Several authors have reported that low-frequency precordial vibration signals can be used to record S2 splitting.4-5 We report a technique that depends on the registration of small but reproducible movements of the compressed skin surface of the chest wall, which are external manifestations of the closure of the aortic and pulmonary valves.

Materials and Methods
This investigation is divided into canine studies in which the heart is exposed in an open-chest procedure and noninvasive studies in patients and normal volunteers in the cardiac graphics laboratory.

The instrument used to detect epicardial surface velocity in the canine studies and the compressed precordial skin surface velocity in the human studies is called a rigid reference frame surface velocity analyzer. The signal from this device is abbreviated as SVA, but is not to be confused with a velocity signal obtained by integration of the signal from a miniature accelerometer, which was also abbreviated as SVA in a previous article from this laboratory.6

Canine Studies
Six mongrel dogs that weighed 23–35 kg were anesthetized with i.v. sodium pentobarbital and ventilated by means of 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 pericardial cradle for the heart. The rigid reference frame surface velocity analyzer uses a rigid structural reference frame with a rigid adjustable support arm that holds a transducer, the sensing surface of which is a relatively rigid circular diaphragm 2 inches (5.1 cm) in diameter. The periphery of this diaphragm is fixed with respect to the transducer housing. The transducer itself can be of the piezoelectric, electromagnetic, or other type, with suitable electronic circuitry so as to allow it to provide a signal of the velocity of the central portion of the sensing diaphragm with respect to the structural reference frame. This device is designed principally for noninvasive studies in humans, but is equally suitable for canine experiments. It uses a flexible nonmetallic plate placed beneath the patient’s back as he lies on the examination bed. Attached to both ends of this back plate are metallic side pieces that allow a magnetic attachment of the reference frame, which arches over the thorax like an inverted letter U (fig. 1). In the canine experiments, the back plate was rigidly attached to the operating table beneath the dog. The sensing diaphragm of the transducer was applied directly to the epicardial surface of the right ventricular outflow tract, using light contact pressure so as not to distort heart function. No quantification of the amplitude of the signal was attempted in the canine or human study, so the zero baseline of velocity was not recorded on the tracings. The zero baseline can be approximated, however, by the level of the SVA signal in late diastole, when the velocity is near zero at moderate heart rates. An upward excursion of the SVA signal above the baseline corresponds to an upward (outward) movement of the epicardial surface toward the transducer. The frequency response curve of the device is essentially flat from 1 to 100 cps. This signal was recorded on a DC input...
channel of a multichannel recorder with a frequency response of 0–250 cps.

Micromanometer-tipped catheters (Millar Instruments) were placed directly through the wall of the proximal aorta and pulmonary artery to obtain pressure signals, recorded with a frequency response of 0–250 cps. In one dog, a micromanometer-tipped catheter was also placed through the wall of the left ventricle at the apex to record left ventricular (LV) pressures. The aortic and LV dP/dt signals were derived from the pressure signals by electronic differentiation. In another dog, an M-mode echocardiogram was simultaneously recorded with the echocardiograph transducer held directly on the heart surface in order to record semilunar valve movement. In another dog, flow signals from the aorta and pulmonary artery were obtained using an electromagnetic flowmeter (Carolina Medical Electronics), the cuff of which was placed around the artery immediately above the semilunar valve. Blood flow signals were recorded with a frequency response of 0–120 cps. The results of the canine studies were recorded on an Electronics for Medicine VR12 multichannel recorder except for the study using M-mode echocardiography, which was done with an Irex 101 multichannel recorder. Left and right ventricular pacing was used in all six dogs as a technique of altering the timing of semilunar valve closure. The pacing wires were sewn directly to the epicardial surface. The vagus nerve was exposed in the neck and electrodes were attached. Electrical vagus stimulation was used to intermittently depress the sinoatrial node so as to slow the heart rate and to facilitate studies of the S₂.

Human Studies

Noninvasive observations were made on 44 normal volunteers and 25 patients with a variety of pathologic conditions. Precordial motion was studied with the rigid reference frame surface velocity analyzer. The nonmetallic back plate of the instrument was placed under the subject’s back. The structural reference frame was magnetically attached to the metallic side pieces of the back plate and thus supported over the chest of the subject as he lay in a semireclining position on the examination bed. The adjustable support arm holds the transducer rigidly and firmly in contact with the skin of the chest wall over the right ventricle, usually in the third left intercostal space at the sternal border. The pressure applied through the transducer housing and sensing diaphragm compresses the skin and soft subcutaneous tissues and provides an efficient mechanical coupling of the transducer to the underlying cardiac movements, while simultaneously reducing the artifactual effects of surface vibrations that result from the movement of the skin. The SVA signal was recorded on a DC input channel of a multichannel recorder, with a frequency response of 0–2500 cps for the volunteers and from 0–100 cps for the patients. This difference was due to the different recorders used. Despite the rigid and unyielding nature of this device, it produced no discomfort and allowed practically normal respiration. An upward excursion of the signal above the baseline corresponds to an upward (outward) movement of the skin surface toward the transducer. The zero baseline of velocity is approximated by the level of the signal in late diastole, when the velocity is near zero at moderate heart rates.

A conventional acoustic microphone of the Leatham air-coupled type was placed in the second left intercostal space, the so-called pulmonary auscultation area, by means of a suction attachment, in close proximity to the sensing diaphragm of the rigid reference frame surface velocity analyzer. Heart sounds were obtained using the Leatham medium filter setting. The SVA signal was recorded simultaneously with the heart sounds (phonocardiogram), an ECG and, in 25 subjects, an M-mode echocardiogram using an Irex 101 or System Two multichannel recorder. The paper speed was 100 mm/sec. In only six subjects did the position of the device compete with that of the microphone for optimal registration of the components of S₂. In this situation, the SVA signal and heart sounds were recorded sequentially at this position rather than simultaneously, so as to eliminate any bias against one or the other system owing to improper positioning on the chest. The signals were recorded during normal respiration except during some portions of the echocardiographic examination.

Results

Canine Studies

The SVA signal obtained from the right ventricular epicardial surface revealed two inward (negative) spikes at the time of S₂. These spikes were synchronous with the aortic and pulmonary valve closure (AVC and PVC spikes, respectively) within the limits of resolution of echocardiography. The AVC spike is sharper (higher-frequency components) than the PVC spike. Figure 2 shows the close timing relationship of these spikes with the incisura of the pressure pulses obtained from the aorta and pulmonary artery and with
PVC as seen by echocardiography. This figure shows the correlations of these signals in normal sinus rhythm, LV pacing to simulate right bundle branch block, and right ventricular pacing to simulate left bundle branch block. In all six dogs, the spikes correlated with the incisural notches of the pressure signals. This was true in normal conduction as well as when the splitting interval was greatly widened or even reversed by pacing. A similar result is shown in figure 3, in which the pressure pulses are displayed along with the flow signals of the aortic and pulmonary artery and the SVA signal.

Figure 4 shows the aortic pressure, aortic dP/dt, the LV pressure, and the LV dP/dt along with the SVA signal in a dog. Beats 1 and 4 are controls. In beat 2, pressure is insufficient to cause a pressure pulse in the aorta. In the absence of this pressure pulse and the associated incisural notch, the AVC spike of the SVA signal is also absent, and only the PVC spike remains. The sharp negative spike of the LV dP/dt signal is also lost in this beat. Beat 3 results in a small aortic pressure pulse and a small negative spike in the left LV dP/dt signal along with an attenuated AVC spike of the SVA signal. The correlation between the sizes of the negative spike of the LV dP/dt, the positive spike of the aortic dP/dt, and the AVC spike is shown. This tracing was recorded during LV pacing to widen the splitting interval of the AVC and PVC components and with vagal stimulation to depress the sinoatrial node. This dog also had occasional beats that failed to produce a pressure pulse in both the aorta and pulmonary artery, in which case both the AVC and the PVC spikes were absent in the SVA tracing.

**Human Studies**

SVA signals from the compressed skin surface overlying the right ventricle showed two distinct negative (inward) velocity spikes that correlated with the vibrations of A2, and also with P2, when seen, in the conventional phonocardiogram (fig. 5). The morphology of these spikes was quite similar to that recorded on the epicardial surface of the dog, with the AVC spike usually sharper (higher-frequency components) than the PVC spike. These spikes tracked the A2 and P2 components of the phonocardiogram faithfully as the splitting interval changed with respiration (fig. 5).

The echocardiogram of AVC was recorded simulta-
neously with the SVA signal in 20 normal volunteers. In 19, the AVC spike was synchronous with the time of coaptation of the leaflets of the aortic valve with the limits of resolution afforded by the echocardiogram when recorded at a paper speed of 100 mm/sec (fig. 6). In one volunteer, the AVC spike of the SVA and \( A_2 \) of the phonocardiogram occurred 10 msec after coaptation of the aortic leaflets, as seen in the echocardiogram. The echocardiogram of pulmonary valve closure was recorded simultaneously with the SVA signal in six volunteers. The PVC spike occurred synchronously with the time of closure of the pulmonary valve, within the limits of resolution afforded by the echocardiogram. Furthermore, the PVC spike tracked the time of closure of the pulmonary valve as the splitting interval changed with respiration (fig. 7).

The SVA signal was also recorded simultaneously with echocardiograms of tricuspid valve opening in 11 volunteers and mitral valve opening in 16 volunteers. We verified that no valvular event, such as a tricuspid or mitral valve opening snap, caused the spike in the SVA signal attributed to PVC.

In all of the 44 volunteers the conventional phonocardiogram showed discernible \( A_2 \) vibrations. In contrast, \( P_2 \) was clearly discernible in the \( S_2 \) vibration complex in only 12 of the 44 subjects, with 32 considered to have ambiguous and indistinct \( P_2 \) vibrations that did not allow a confident assessment of the splitting interval of \( A_2P_2 \). In all 44 volunteers, however, the AVC and PVC spikes were clearly shown. Therefore, in 73% of the volunteers, the SVA signal provided a significantly better indication of the splitting interval than did the conventional phonocardiogram. A tracing typical of the results of the normal volunteer study is shown in figure 8, in which \( P_2 \) cannot be clearly delineated in the \( S_2 \) vibratory complex of the phonocardiogram but with the PVC spike of the SVA signal clearly shown.

In the 25 patients studied, all had clear \( A_2 \) vibrations in the phonocardiogram. In contrast, only nine had a clearly visible \( P_2 \) and 16 had indistinct and ambiguous \( P_2 \) vibrations that did not allow a confident assessment of the splitting interval of \( A_2P_2 \). The AVC and PVC spikes were clearly shown in all patients, including a patient with chronic obstructive pulmonary disease. Therefore, in 64% of the patients, the SVA signal provided a significantly better indication of the splitting interval than did the conventional phonocardiogram. An example is shown in figure 9, in which a late systolic murmur obscures paradoxical splitting of the \( S_2 \) in a patient with left bundle branch block. Figure 10 is another example, obtained from a patient in right ventricular failure manifest by distended neck veins, a hepatojugular reflex and a right ventricular heave on physical examination. The diagnosis was supported by echocardiographic evidence of an enlarged right ven-

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**Figure 4.** The surface velocity analyzer (SVA) signal obtained from the right ventricular epicardial surface of a dog, along with aortic (Ao) and left ventricular (LV) pressure signals and their derivatives (Ao \( dP/dt \) and LV \( dP/dt \)) and the ECG. Beat 2 fails to cause an aortic pressure pulse and is accompanied by a total loss of the aortic valve closure spike of the SVA signal. The tracing was recorded during LV pacing to widen the splitting interval between aortic and pulmonary valve closure and electrical vagal stimulation to depress the sinoatrial node. Time lines are 50 msec.

**Figure 5.** The surface velocity analyzer (SVA) signal recorded at the left sternal edge (LSE) with the phonocardiogram in the pulmonary auscultation area (PCG-PA) and the ECG. The aortic and pulmonary valve closure (avc and pvc) components track the \( A_2P_2 \) components of the phonocardiogram as the splitting interval widens due to normal respiratory variation. This tracing is from a normal volunteer with a particularly clear \( P_2 \) component in the phonocardiogram. Thin time lines are 10 msec.
indicative of right ventricular failure. \(^1\) A dropout of \(P_2\) in the phonocardiogram gave the erroneous impression of \(A_P\) fusion during expiration.

In one volunteer, we recorded a very rare end-expiratory merging of the AVC and PVC spikes to a large single inward spike, showing that the two movements can occur simultaneously and can be summed together in the SVA signal (fig. 11).

**Discussion**

Palpation of the precordium has long been used as an important feature of the physical examination of the heart. Graphic records of precordial movement have been available since the introduction of the sensing capsule by Marey in 1861. \(^7\) Considerable information of diagnostic value can be obtained from apexcardiography \(^8,9\) and kinetocardiography, \(^10\) which are methods of recording positional changes of the chest wall due to cardiac function. Several investigators, \(^3,5\) using various techniques, have reported that low-frequency precordial vibratory signals can be used to record \(S_2\) splitting. In 1961, Agress et al., \(^4\) using a capacitance microphone suspended from a horizontal bar, recorded wave forms similar to those of the SVA instrument, and noted that slight degrees of splitting could be detected with the device. Undoubtedly, M-mode echocardiography, which was unavailable at the time, would have greatly facilitated these pioneering research efforts.

In 1971, Ikegaya, \(^11\) a Japanese engineering professor interested in calibration of phonocardiographic microphones, reported a study that used an instrument similar to the rigid reference frame surface velocity analyzer. The signal from this device clearly showed features that we believe are AVC and PVC spikes. This engineering study was not concerned, however, with the exact association of the features of the wave form with particular cardiac events.
In our dog studies we have been critical in the understanding of the AVC and PVC movements, providing us with the opportunity to search invasively for their source and also allowing us to manipulate the timing of semilunar valve closure by ventricular pacing techniques so that we could verify the temporal association of these spikes with AVC and PVC. The cause and effect relationship of the semilunar valve closure to the AVC and PVC spikes was further demonstrated during our dog studies when the occasional loss of an aortic pressure pulse during a weak beat was accompanied by a total loss of the AVC spike with only the PVC spike remaining (fig. 4). In this same dog, the occasional loss of both aortic and pulmonary artery pressure pulses was also accompanied by the total loss of both the AVC and PVC spikes. The presence or absence of the aortic and pulmonary artery pressure pulses during these weak beats was determined by observation of the pressure signals from the micromanometer-tipped catheters. In figure 4, the pulmonary artery pressure signal was replaced with the LV dP/dt signal to illustrate the close association of the magnitude of the negative spike of the LV dP/dt signal with that of the AVC spike, such that the sharp negative spike of the LV dP/dt signal was absent when the AVC spike was absent. Figure 4 suggests a possible role of AVC in the formation of the sharp negative LV dP/dt spike, which may be important in the proper interpretation of this hemodynamic measurement. This is a subject of recent investigation in our laboratory. We believe that the AVC and PVC movements occur as a result of a transitory reverse blood flow in the proximal aorta and pulmonary artery in protodiastole. This flow is abruptly halted by closing and tensing of the semilunar valves. The origin of the AVC spike and its relationship to A2 are being studied in our laboratory. In adults, the compression of the skin and soft tissues, using a fixed transducer with a relatively rigid sensing diaphragm, is critical to the efficient detection of the PVC movement on the skin surface. Of the three measurements of motion of the compressed skin surface — positional change, velocity and acceleration — the velocity signal is by far the best wave form for studying semilunar valve closure. In the positional signal, these movements are almost imperceptible, while the acceleration signal begins to take on the characteristics of the conventional phonocardiogram with a complex of vibrational components tending to obscure the two events so clearly seen in the velocity signal.

Even when the A2P2 components of the conventional phonocardiogram “merge” to a single vibratory complex, we can routinely detect a persistent, measurable splitting interval of the AVC and PVC components of

Figure 9. The surface velocity analyzer (SVA) signal recorded at the left sternal edge along with the phonocardiogram in the pulmonary auscultation area (PCG-PA) and mitral auscultation area (PCG-MA) and the ECG. The patient has left bundle branch block. This recording shows a paradoxical splitting obscured by a late systolic murmur. The pulmonary valve closure (pvc) spike of the SVA signal comes before aortic valve closure (avc). Time lines are 40 msec.

Figure 10. The surface velocity analyzer (SVA) signal recorded at the left sternal edge with the phonocardiogram in the pulmonary auscultation area (PCG-PA) and the ECG in a patient with right ventricular failure. The end-expiratory beat (the first beat shown) appears to show a normal fusion of the components of the phonocardiogram, while the interval between aortic and pulmonary valve closure (AVC and PVC) is greatly fixed at about 50 msec. With inspiration (INSPIR.), the PVC spike contains higher-frequency components and the phonocardiogram shows a large P2. Time lines are 40 msec.
the signal from the rigid reference frame surface velocity analyzer owing to its greater resolution of narrow splitting. It therefore appears that true "merging" of $S_2$ to a singlet usually does not occur. In one normal child volunteer, however, we recorded a rare end-expiratory merging of the AVC and PVC spikes (fig. 11). The $S_2$ vibration of the accompanying phonocardiogram is larger in amplitude and different in morphology (more singular in appearance) during this beat than in the beats before and after it, and probably would have been difficult to interpret by auscultation.

The clinical value of this device is not limited to measuring the splitting interval when it is so narrow as to become a continuum in the conventional phonocardiogram. The identification of the AVC and PVC spikes does not depend on the detection of the high-frequency vibrations arising from semilunar valve closure, and they can be identified even when one of the phonocardiographic components is obscured by a late systolic murmur (fig. 9). We have also recorded AVC and PVC spikes in a patient with loud respiratory sounds that obscured the phonocardiogram.

In instances in which the vibrations associated with pulmonary valve closure are of insufficient amplitude and frequency to register on the conventional phonocardiogram, a clear PVC spike is still detected (fig. 10). The failure to record $P_2$ in the phonocardiogram can lead to the erroneous conclusion that narrow splitting or merging of the components of $S_2$ has occurred, even though the splitting interval of semilunar valve closure is wide, as indicated by the AVC and PVC spikes. This is of particular importance in detecting right ventricular failure (fig. 10) or atrial septal defect, because a persistent wide splitting is indicative of these conditions.

A comparison of the relative magnitudes and shapes of the AVC and PVC spikes might also provide useful clinical information. We have noted that in aortic incompetence, the AVC spike is markedly reduced in size relative to PVC, while in pulmonary hypertension the relative sharpness of the PVC spike is increased. These aspects of the two movements have not been fully studied.

Certain limitations are inherent in a comparison of the AVC and PVC spikes and the phonocardiogram, arising principally from the ambiguity of the $S_2$ phonocardiographic vibratory complex. Some authors take the onset of the first vibrations of $A_2$ in the phonocardiogram as the time of $A_2$, while others designate the time of $A_2$ as the time of its maximal amplitude. We have found that the initial $A_2$ vibrations in the phonocardiogram occur at the time of rapid downstroke of the AVC movement.

The size of the SVA signal is affected by the pressure of contact of the analyzer on the skin surface, and therefore, no quantification of the signal magnitude was attempted in this study. The timing of the AVC and PVC spikes is not measurably affected by changes in contact pressure if the tissues are sufficiently compressed so as to record a clear signal.

The signal is not free of the need for subjective interpretation. In the second and third beats shown in figure 10, the PVC movement has three inflections near its nadir, causing some confusion as to the time of the spike. In fact, we frequently see two or three inflections in the PVC downstroke movement. We have conjectured that these multiple inflections might be the result of asynchrony of leaflet closure of the pulmonary valve, although we have not studied this possibility effectively. We have defined the time of the AVC and PVC spikes as the time of maximum negative excursion of the AVC and PVC movements (fig. 10).

Another problem of interpretation is illustrated in figure 11, in which the summation singlet of AVC and PVC (arrow) is followed by a secondary low-frequency movement, which could be misinterpreted as the PVC spike. Inspection of the sequence of beats shows that this is not so, because a clear PVC spike is shown in the remaining beats, and it is obvious that the two spikes have merged together in the summation beat. If the splitting interval is persistently so narrow as to prevent individual identification of the AVC and PVC components of the signal, however, an error in interpretation could be made. This has never happened in our studies with the instrument.

In conclusion, we believe that the signal from the rigid reference frame surface velocity analyzer is a significant improvement in the noninvasive assessment of the splitting interval of semilunar valve closure, removing much of the ambiguity and subjectivity

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**FIGURE 11.** The surface velocity analyzer (SVA) signal at the left sternal edge (LSE) in a human along with the conventional phonocardiogram in the pulmonary auscultation area (PCG-PA) and the ECG. The first beat shows a narrow splitting of aortic and pulmonary valve closure (AVC and PVC), which continues to narrow at end expiration to a summation singlet (short arrow). The phonocardiogram shows an unusual $S_2$ during this beat, with a more singular vibration of larger magnitude than the beats before and after it. This summation beat is followed by a normal widening of the splitting interval with inspiration (INSP.). Time lines are 40 msec.
associated with interpretation of the $S_2$ vibratory complex in the conventional phonocardiogram. It provides clinically relevant information about the splitting interval at times when the phonocardiogram fails to do so, and is therefore offered as a supplement to phonocardiography.

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Influence of Age on Wall Thickness, Cavity Dimensions and Myocardial Contractility of the Left Ventricle in Simple Transposition of the Great Arteries

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with statistical analysis by Gilles Ducharme, M.Sc.

SUMMARY This study was carried out to establish a reference table of echocardiographic values for the left ventricle of simple d-transposition of the great arteries (d-TGA) and to determine at what age left ventricular dimensions in these patients become different from those of a normal population. Fifty-three patients with d-TGA and normal pulmonary pressure and 395 normal children ages 1 day to 10 years were studied by M-mode echocardiography. Results show that in d-TGA, left ventricular systolic and diastolic internal diameters are normal at birth. After 1 month, however, both diameters were below normal and despite a progressive increase with age, the mean values were always below normal. The mean posterior wall thickness of patients with d-TGA was also normal at birth but did not increase with age (2.3 mm in diastole and 4.3 mm in systole) and became significantly thinner than normal at 10 months of age in diastole and 7 months in systole. Septal thickness of patients with d-TGA did not differ from that of the control group. The shortening fraction and mean velocity of circumferential fiber shortening were significantly greater in d-TGA at all ages. Left ventricular measurements related to age are presented and should be of help in interpreting M-mode echocardiograms of patients with d-TGA.

MEASUREMENTS of anatomic specimens by echocardiography have shown that patients with d-transposition of the great arteries (d-TGA) have a thinner left ventricular (LV) wall than subjects with normally related great arteries. This is expected because in transposition, the left ventricle is connected to the low-pressure pulmonary circulation. However, the influence of age on LV characteristics has only been studied in anatomic specimens and never, to our knowledge, in living patients with d-TGA. This information is important for at least three reasons. First, a reference table based on echocardiographic measurements related to age from patients with uncomplicated d-TGA is needed to interpret echocardiograms of patients with d-TGA, because comparison with normal subjects is obviously erroneous. Second, studies of fixed anatomic specimens cannot provide information about the dynamic characteristics of the LV wall. Third, with the increasing popularity of anatomic correction of d-TGA, it has become important to establish the age at
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