NONINVASIVE TECHNICS *in* CARDIOLOGY

The Phonocardiogram, Apexcardiogram, and Systolic Time Intervals

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Preface

THE PURPOSE of this book is to acquaint the practicing cardiologist and internist with noninvasive diagnostic technics: specifically, the apexcardiogram, the recording of heart sounds and murmurs, and the systolic time intervals. There are, of course, a number of other noninvasive methods, but most are still in the research stage. Since this text is directed to the clinician seeing patients, elaborate details on mechanisms, theories, and controversial subjects have been deliberately avoided. The author felt that the reader would benefit more by illustrative case histories and how these noninvasive methods helped establish an objective diagnosis and rational treatment without the aid of cardiac catheterization.

Noninvasive diagnostic technics are not new in cardiology. However, not until the advent of coronary cinearteriography, ventriculography, and proper instrumentation could the data being recorded by these methods be confirmed and correlated with the patient's disease. Traditionally, these technics have been used for the graphic recording of events of the cardiac cycle, for teaching, and to have a permanent record of one's auscultatory findings. Thus, in the past, emphasis was placed on using these methods to confirm what one heard on auscultation. Now, the converse is true: auscultation should confirm the sound abnormalities and the hemodynamic findings recorded with these noninvasive technics. One has to work with these methods only briefly before realizing that the amount of information one can obtain in a very few minutes dwarfs the kind of knowledge conventionally obtained on the routine examination of a patient. Indeed, most clinicians would have to admit that, except for obvious murmurs, examination of the asymptomatic patient with early or even moderately advanced heart disease is not very rewarding. Our mistaken assumption has been that, because palpation, auscultation, electrocardiography and the chest x-ray have been essential for the diagnosis and treatment of advanced disease, the same must hold true for the detection of early or moderate but asymptomatic disease. The high mortality from heart disease despite the medical attention of a physician, and the fact that half of all deaths occur in patients without prior knowledge of disease, would suggest otherwise.

At last we are beginning to appreciate our limitations in the early detection of heart disease. No choice remains, then, but to go to more sensitive methods of examination and use them on a routine day-to-day basis in the examination of a patient rather than only in rare instances. Noninvasive technics will satisfy these purposes admirably. Moreover, we can now be assured they can be used clinically as well as in research. For this reason, this text has been written. It describes the clinical application of these methods and how they may be used in the early diagnosis of disease. After all, if you had heart disease you would like it diagnosed as early as possible so that you could do something about it. The patient deserves the same.

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acquisition of patients and catheterization data were responsible for much of the information on the pages that follow. I would also like to thank my technicians, Mrs. Susanne Willits and Mrs. Gayla Tuthill, for their great help in making recordings on patients and in compiling data, as well as my secretary, Mrs. Nanette Myers, for her efficiency and patience beyond the call of duty, in typing this text.

HOWARD H. WAYNE, M.D.

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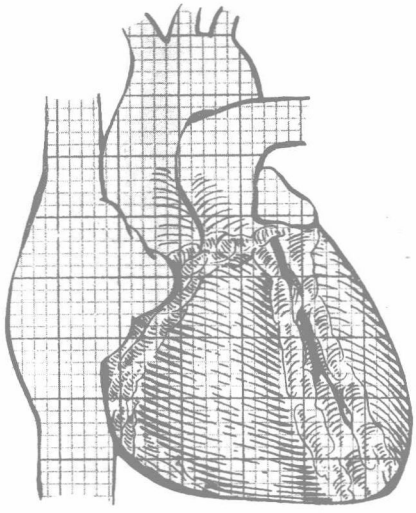

SECTION I

Sounds and Murmurs

CHAPTER I

Need for New Testing Methods

GENERATIONS OF PHYSICIANS have grown dependent upon the stethoscope, the electrocardiogram, the chest x-ray, and more recently palpation as their primary tools in cardiac diagnosis. In the hands of the expert, these methods can provide considerable information. However, too few clinicians have the required expertise to translate their findings properly. Unfortunately, too, many physicians have a near-obsession for the need to document objectively the presence of disease, particularly ischemic heart disease, before instituting treatment.

Although most physicians, when confronted with classic angina pectoris, feel reasonably secure in their diagnosis even without additional clinical support, they are less certain when pain or other symptoms are atypical. Indeed, ischemic heart disease may exist in advanced form without any clinical manifestations.

While the usual tools used in the examination of the patient, i.e., the stethoscope and electrocardiogram, are far too insensitive to uncover early and often even moderately advanced heart disease, the sad fact is that this insensitivity is not generally appreciated. Sir Thomas Addison made a remark about the stethoscope 125 years ago that is perhaps even more applicable to the electrocardiogram today. He said: "They look upon the instrument as all-sufficient; they neglect or disdain to make those careful and minute inquiries that no sound and sensible physician ever fails to do, thereby converting an invaluable tool into what in their hands, at least, proves an imperfect and treacherous substitute."[1] It is therefore not surprising that half of all deaths from acute myocardial infarction are reported to occur in persons with no prior clinical heart disease,[2] and 25% are "silent" or atypical.[2, 3]

Unwarranted dependence is often placed on the electrocardiogram. Although the presence of angina pectoris usually indicates the presence of disease of more than one vessel, 50-83% of such patients have normal resting electrocardiograms.[4, 5] In a recent study from the Cleveland Clinic, 49% of 723 patients under the age of 40 with atypical chest pain had significant coronary artery disease, but only 15% had an abnormal electrocardiogram.[6]

Electrocardiographic testing of patients during exercise tends to identify ischemic heart disease more accurately, but unwarranted dependence has been placed upon it as well. While a positive test is reliable evidence of ischemic heart disease, the converse is not nearly so true. Thus, Goldschlager *et al.*[7] found that nearly 40% of angina patients with one- and two-vessel disease had normal electrocardiograms during the treadmill test and 22% of symptomatic patients with three-vessel disease had normal treadmill tests. Other workers have reported similar results.[8, 9] The Masters two-step test is even less reliable.[10–12] Indeed, it has been suggested that the test is too inaccurate for general use and should be discarded.[13]

Exercise testing had been used not only to diagnose ischemic heart disease but to estimate the degree of left-ventricular dysfunction and coronary artery disease. The extent of the S-T segment depression does correlate somewhat with the extent of coronary artery disease. Unfortunately, one cannot reliably relate exercise-induced electrocardiographic changes to either the severity of disease or left-ventricular performance. Blomquist[14] has recently reviewed the use of exercise-testing extensively in the diagnostic and functional evaluation of patients with

ischemic heart disease. Studies correlating the electrocardiographic response to exercise, with findings at coronary cinearteriography, have demonstrated an abnormal tracing in zero to 30% of patients with no demonstrable arterial lesions. The number of patients with significant coronary artery disease and negative electrocardiographic responses tended to be even higher. Physical work capacity in patients with angina pectoris correlated poorly with the severity of left-ventricular dysfunction present at rest as judged from the end-diastolic pressure, ventricular volume, and wall motion. Moreover, only a weak correlation existed between the extent of arterial disease determined from cinearteriography and physical work capacity.

While modern monitoring technics have significantly reduced the risk of death after a patient has entered the hospital, routine methods have achieved little success in predicting an impending infarction.[15] Of extreme interest are the recent findings that most patients with an approaching myocardial infarction have warning symptoms for 1 to 2 months in advance.[16, 17] Many have consulted their physicians as recently as 1 to 2 weeks before the actual attack.[18] Both patient and physician alike become falsely reassured by the pantomime of the doctor placing his stethoscope upon a patient's chest and, not hearing any abnormalities, recording an electrocardiogram, which is also frequently normal. The absence of audible abnormalities does not indicate that no abnormalities exist, any more than a normal resting and exercise electrocardiogram automatically excludes disease.

These considerations are undoubtedly prime reasons why the early death rate from ischemic heart disease is so high; 60–70% of all deaths occur before the patients have reached a hospital.[19–21] Conceivably, if ischemic heart disease or an approaching infarction could be detected earlier, corrective measures, prophylactic medications, or surgery would reduce the present high mortality.

More accurate and definitive tests do, of course, exist in the form of coronary cinearteriography. Although such technics have advanced our knowledge enormously, they are not yet generally available. They require highly trained personnel and expensive equipment, are invasive, and cannot be performed practically on a serial basis in the same patient. Furthermore, such tests are not cheap, nor are they 100% accurate. Significant discrepancies often exist between the clinical picture, exercise testing, and the coronary cinearteriography findings.[6, 7] Opinion, too, differs on when such tests should be performed.

Lack of sensitivity with standard methods of examination is not the sole criticism of the electrocardiogram and the stethoscope as diagnostic tools. In all patients but those with far-advanced disease, essentially no correlation exists between the findings and actual function. For example, the electrocardiogram of an elderly patient and a professional athlete may both be considered normal. Even more incriminating is the lack of correlation with survival in the average infarction patient. The tracings of two patients in an intensive care unit may look similar after an acute infarction; yet one cannot predict who will live or die. After the patient has recovered, one cannot, except in the patient with evident advanced disease, determine from the average examination whether he will be disabled, be able to return to work, or be able to run several miles a day.

Many clinicians hope that the computer will solve some of the previously discussed problems. It is highly doubtful whether computerization of insensitive and/or obsolete tests for the detection of *early* disease will enhance their diagnostic accuracy significantly. What is urgently needed are new tests in the early diagnosis of heart disease and impending myocardial infarction.

For these reasons, increasing attention is being directed toward the so-called noninvasive tests in the study of early heart disease. Actually, these tests are not new. They include phonocardiography, jugular and carotid pulse recordings, and the apexcardiogram, as well as other tests. The graphic registration of physiologic events by these external recording methods has been used for many years in research, for teaching, and for timing the various sounds and murmurs in the cardiac cycle. Over the years, correlation with the direct findings during cardiac catheterization has made clinicians realize that these external recording methods can, in themselves, provide a great deal of information. That is to say, once the disease and its physiologic and pathologic consequences had been studied with indirect technics and the findings correlated with direct hemodynamic data, it became possible to obtain hemodynamic information from indirect or noninvasive methods alone and thus to assess cardiac function qualitatively and sometimes quantitatively. Noninvasive tests possessed additional advantages.

They caused no complications, were inexpensive, and could be done repeatedly in the same patient. Some of these methods can now provide information that is not usually obtainable even with cardiac catheterization unless very special and complicated procedures are used.

This text will review many of our findings with the use of the noninvasive techniques in well over 1,000 patients during the past 5 years. We have become convinced that the routine use of these external recording methods offers great potential to the clinician. Indeed, any physician who wishes to, can acquire diagnostic skills which will enable him to diagnose heart disease much earlier in its clinical course than he probably ever thought possible.

The ability to discover early abnormalities in the asymptomatic patient has distinct benefits. First, it provides incentive for the "well patient" to change his living habits. For example, most of us are aware that regular physical exercise is desirable, but few of us are sufficiently motivated to act on this knowledge. Thus many, if not most, patients are without symptoms because their ordinary physical activities are sedentary. Even moderate exertion for short periods may be comfortably tolerated. Mild dyspnea, fatigue, or palpitations, if present, are minimized or explained as "being out of condition" or "due to age."

Subjecting the heart of such a potential cardiac victim to moderate prolonged work loads on a regular basis should uncover early symptoms due to progressive narrowing of a major vessel. Then, prophylactic antiarrythmic drugs, coronary vasodilators, anticoagulants, and other treatment can be started in the hope of averting a catastrophic event. At the very least, constant and regular exercise may stimulate the growth of collateral vessels in the myocardium and hopefully elicit "medical revascularization" of potentially ischemic areas. Since changes such as these require time, any method that uncovers the earliest possible evidence of disease or left-ventricular dysfunction gives the patient and physician a head start in instituting a prophylactic program.

Similar logic applies to motivation of asymptomatic patients to discontinue smoking and to reduce their intake of cholesterol, fats, and calories. The corollary with reference to the physician also holds; that is, the knowledge that a patient does have heart disease, even though symptoms are lacking, will make both the patient and physician alert for early symptoms, even when atypical.

CHAPTER 2

The First and Second Heart Sounds

THE FIELD OF PHONOCARDIOGRAPHY is represented by an extensive body of literature. In fact, a recent publication by the American Heart Association[22] contains a bibliography spanning a full century. Since this is not a text on phonocardiography per se, the entire field cannot be reviewed in this discussion. Rather, examples of the newer and the most frequent clinical applications of routine heart-sound recordings will be presented.

Heart sounds should be routinely recorded in all patients, particularly those who return for follow-up care at regular intervals, in the absence of detectable auscultatory findings. We deceive ourselves if we believe that we can accurately compare the pitch, intensity, and quality of the various heart sounds each time the patient returns. We might as well ask ourselves to compare the performances of a symphony given many weeks or months apart. Although we would recognize the music, most of us would be unable to compare, except in the crudest fashion, the loudness and quality of the music. How, then, can we expect to compare the sounds and murmurs of a patient's heart at wide intervals of time?

Second, the heart sounds represent a complex of vibrations exhibiting different characteristics at different frequencies. Abnormal sounds such as a fourth sound or an ejection click often cannot be separated anymore than we can identify fine details in the countryside while traveling at high speed in an automobile. The fact that they can be heard at all is not attributable to our acoustic ability but to the advanced degree of heart disease that causes increasing separation and a higher frequency of these sounds. To identify such abnormal sounds in the presence of early disease is beyond human capability, even for the experienced cardiologist.

Third, some sounds, such as the third or fourth sounds, are of low frequency and soft in character in the presence of early or even moderate disease. Often they are below the audible frequency range (25 Hz) for most listeners. Under absolutely ideal conditions, such as in a quiet room, with the bell of the stethoscope placed exactly over the apex impulse under just the right amount of pressure, one may detect these sounds. Unfortunately, such ideal circumstances, as well as adequate time, are not always available.

How much simpler it is to avoid such problems by routinely recording calibrated sounds, placing the microphone directly over the apex impulse, with the patient in the left lateral position, and at the same phase of respiration. If one has the proper instrumentation, such a recording can be obtained in 2 to 3 minutes, and one has a permanent record.

Instrumentation

A number of different microphones and recorders are available. The crudest method is to convert a single-channel electrocardiogram machine into a sound recorder with appropriate transducers and a microphone. However, those who use such methods are often unaware that the heated stylus is simply incapable of recording higher-frequency sounds. In addition to the naturally low-frequency response due to inertia and friction of the system, the stylus must be in con-

tact with the paper long enough to cause an inscription. At higher frequencies, not enough time exists to record such sounds with fidelity. Lower-frequency sounds, such as a fourth sound, can, however, be recorded accurately. The single-channel instrument does not permit one to time the heart sounds with other events of the cardiac cycle. For example, an ejection click can usually be identified accurately only when the heart sounds are recorded with a carotid pulse. The click can then be seen as a higher-frequency sound occurring simultaneously with or immediately following the carotid upstroke. Similarly, it may not be possible to identify a fourth sound without a simultaneously recorded electrocardiogram. When this is done, the fourth sound is identified as occurring just before the QRS complex.

Multichannel instruments, using a heated stylus, are now being advertised as a satisfactory solution to the problems of timing with a single-channel instrument. The manufacturers of these instruments recommend the envelope system as a way of recording higher frequencies with a heated stylus. While the envelope system can accurately time a sound and measure its amplitude, it cannot record its frequency. More importantly, however, the envelope system lacks sufficient resolution to separate sounds. This deficiency may prompt one, for instance, to erroneously call an ejection click the last component of the first sound complex, or misinterpret a split first or second heart sound.

Two basic types of recorders are available with proper frequency response. The first type are photographic recorders which use bromide paper (Hewlett Packard, Electronics for Medicine, Cambridge), but they have the disadvantage of requiring one to develop the paper before reading the phonocardiogram. A second type, based upon a jet ink spray under pressure, has an adequate frequency response (650 Hz) for recording sounds and murmurs and can be read directly after recording (Elema Schonander Mingograf).

Recording Technic

The patient is placed in the left lateral position and the apex impulse is carefully palpated and marked. A strap is placed around the patient's chest just tightly enough to hold the microphone firmly in place. Care must be taken not to fasten the strap too tightly or damping will occur. If a dynamic or bell microphone is used, one must avoid even minute air leaks or the sounds will lose significant amplitude. With the strap in place, a microphone is placed over the exact center of the apex impulse. One must make certain that the marked location of the impulse does not move away from its true location because of skin movement or pull created by the strap. One can then record the heart sounds in select band-pass frequency filter ranges. Generally, the sounds are reproduced in filters peaking at 25, 50, 100, 200, and 400 Hz in commercial instruments. Some also contain a broad band from 70 to 600 Hz which approximates the range of human auditory perception. Depending upon the number of available channels, one may record all these frequency ranges simultaneously, or two to three at once. It is advisable to record these sounds simultaneously with a carotid pulse and an electrocardiogram lead. Ideally, then, one should use at least a three-channel instrument, but two can record all necessary information. Since the heart sounds are also recorded with various band-pass filters, all at different peak frequencies, it is obvious that the greater the number of channels available, the more quickly one can record the necessary information. Cost and available time are usually the deciding factors in selection.

It is advisable to calibrate the phonocardiograph channels so that 1 millivolt standardization provides about 10 millimeters of deflection, to ascertain that all frequency ranges are recorded with equal gain. Also, the heart sounds are best recorded during quiet expiration. Thus, if one always places the microphone in exactly the same location, any significant change in amplitude or timing of sounds would suggest alteration in one or more of the mechanisms responsible for their production.

The First Heart Sound

Mechanism

Considerable controversy exists concerning the origin and causes of the various components of the first sound. While a detailed discussion of this subject is not within the scope of this text, the following comments summarize some of the current thinking. It is generally agreed the first sound consists of four components. The initial first-component vibrations are low in frequency, begin after the onset of the QRS of the electrocardiogram, and tend to coincide with the initial ven-

tricular upstroke of the apexcardiogram. The sound appears to originate from vibrations caused by the onset of ventricular contraction and the acceleration of blood toward the atrioventricular valves. Low-frequency vibrations created by atrial contraction may be confused with the initial low-frequency vibrations of the first sound. Such confusion is more apt to arise when one records with a band-pass filter which peaks at 25 Hz. The two can generally be separated by noting the relation of the sounds to the QRS complex. Low-frequency fourth-sound vibrations occur before, while first-sound vibrations occur after the onset of QRS.

The second component of the first sound is believed by most workers in the field to be largely due to mitral valve closure.[23–25] Others maintain that this component is due to the abrupt tensing of the left-ventricular wall, papillary muscles, and chordae tendineae.[26] It is higher in frequency and amplitude than the first component and is best recorded at 50–200 Hz.

The third component of the first sound has about the same frequency but is usually of slightly lower amplitude than the second component when recorded over the apex. Over the lower left sternal border, however, it may equal or exceed the second component. Again, most investigators feel that this component arises from closure of the tricuspid valve, since this vibration is heard best along the lower left sternal border. Normally, tricuspid valve closure follows mitral valve closure because of asynchronous activation of both ventricles (left precedes right) as well as the pressure-volume differences in each ventricle. Luisada disputes tricuspid closure as playing a significant role in the genesis of the third component.[27, 28]

Piemme *et al.*[29] have suggested that it is the change in the rate of pressure rise in the left ventricle, just after the aortic valves open, which causes abrupt tensing of aortic structures. This does not adequately explain third components that are recorded well before opening of the aortic valves.

The fourth or final component is low in both frequency and amplitude in the normal individual and coincides with the ejection of blood into the great vessels. Because of the shorter isovolumic contraction time of the right ventricle, as well as the lower pulmonary diastolic pressure, pulmonary ejection begins before aortic ejection; however, normally it is impossible for one to separate these two components from each other.

Factors Controlling Amplitude of the First Heart Sound

EXTRACARDIAC FACTORS.—The most important single factor determining the recorded amplitude of the first sound (and all other sounds) is the position of the microphone pickup. While placement is not so critical as with the fourth heart sound, to be discussed later, amplitude decreases exponentially as the microphone moves away from the point of maximal intensity. Almost invariably this point is the center of the apex impulse, when the patient is lying in the left lateral position. It is worth mentioning again that, unless the microphone is strapped over the apex impulse snugly but not tightly, the loose underlying skin is likely to cause movement of the microphone after the strap is released following placement. As a rule, the amplitude of the first sound decreases as the recording microphone is moved from the apex impulse in the left lateral decubitus to the apex in the supine position. When the microphone is placed over the lower left sternal border, the second and third components are about equal in intensity. With the microphone at the base of the heart, all components of the first sound are reduced.

A second factor influencing the amplitude of the first as well as all other heart sounds is phase of respiration. Generally, sounds are heard best in end expiration. Forced expiration should be avoided, since the subject may unconsciously perform a mild Valsalva maneuver.

The third factor influencing sound amplitude is transmission. In the child and thin-chested adult, sounds are very loud and recorded amplitude is quite high. In women with large amounts of breast tissue, patients with emphysema, individuals with heavily muscled chest walls, or persons who are very obese, transmission of all sounds may be reduced considerably. In the presence of pericardial effusion, transmission may be decreased to some degree. However, equally if not more important is the position of the atrioventricular valves, to be discussed below.

CARDIAC FACTORS.—Cardiac factors affecting the first heart sound are listed in Table 2-1. Normally, the first sound amplitude is considerably greater than the second sound in the absence of systemic hypertension (i.e., 1.5–2.0 times). Before

TABLE 2-1.—CARDIAC FACTORS CONTROLLING AMPLITUDE OF FIRST HEART SOUND

FACTOR	MECHANISM	EXAMPLES
Integrity of mitral valve apparatus	Damage to valve leaflet	Rheumatic valvulitis
	Dilatation of ring	Congestive heart failure
	Ruptured chordae tendineae	Idiopathic
	Dysfunction of papillary muscle	Inferior wall ischemia
Rate of pressure rise in ventricle	Positive inotropic agents	Digitalis, catecholamines, thyroid hormones
	Negative inotropic agents	Propranolol, antiarrhythmic drugs
	Impaired wall motion: dyskinesis, akinesis, asynchrony	Acute myocardial infarction, cardiomyopathy, hypothyroidism, ventricular aneurysm, congestive failure
	Increased wall motion	Hyperdynamic heart, ventricular hypertrophy, tachycardia
	Diastolic fiber length (Frank-Starling mechanism)	
	Reduced	Shock, hypotension, tachycardia, PVC
	Increased	Elevation of left ventricular end diastolic pressure
	Variable	Atrial fibrillation, complete heart block
	Damage to aortic valve	Aortic stenosis and insufficiency
	Slowing of ventricular activation	Complete left bundle branch block
Factors affecting position of AV valves	P-R interval	First, second and third degree heart block
	Pressure gradient across AV valve	Mitral stenosis, atrial tumor
	Volume of blood flow across AV valve	Anemia, pregnancy, hyperthyroidism, left-to-right shunt
	Duration of diastolic filling period	Atrial fibrillation, PVCs, pericardial effusion

deciding that the first sound is reduced, one must exclude the extracardiac factors already mentioned. In these instances, however, the second sound is reduced as well.

Integrity of the mitral apparatus.—Since proper closure of the mitral valves is one of the most important determinants of the amplitude of the first sound, a normal sound cannot occur if any portion of the valve apparatus is significantly damaged. There may or may not be an associated murmur of mitral regurgitation. At times the murmur may be inaudible even with significant regurgitation.

The mechanism of improper valve closure can best be determined from the clinical setting. The most frequent cause is papillary muscle dysfunction, which will usually be evident from the patient's history, electrocardiogram, etc. Advanced congestive failure is usually obvious, while major damage to the valve itself is almost always accompanied by a loud regurgitant murmur.

Force of ventricular contraction[30–32].—A reflection of this force is the rate of pressure rise in the ventricle, also known as the left-ventricular dp/dt. A variety of factors influence dp/dt, including inotropic agents, the character of myocardial wall motion, the resting diastolic volume, the integrity of the aortic valves, and the rate of activation of the left ventricle.

Positive inotropic agents, such as digitalis, catecholamines, and thyroid hormone, increase the amplitude of the first sound. The effect of catecholamines and thyroid hormone is more evident because of the associated tachycardia. On the other hand, negative inotropic agents in adequate dosage clearly reduce the amplitude of the first

sound. The effect of antiarrhythmic agents is minimal at best.

Wall motion and contractility also influence the first heart sound. Diffuse or localized disease of the myocardium reduces left-ventricular dp/dt and the resulting intensity of the first heart sound. Acute myocardial infarction is a prime example.[33] During the first few days of the infarction, catecholamine secretion may be elevated with actual increase in the first sound. This is most likely to occur during the first 24 hours. As ischemia develops, wall motion is impaired, the force of ventricular contraction is reduced, and the first sound is diminished. Serial recordings during an acute infarction usually demonstrate gradual increase in the amplitude of the first sound.[34] Other conditions associated with impaired myocardial contractility, such as chronic ischemic heart disease, cardiomyopathy, ventricular aneurysm, and heart failure, also reduce first-sound amplitude. In the case of ischemic heart disease, marked reduction of both sounds constitutes strong evidence for diffuse disease.

Myocardial wall motion may be increased under certain circumstances and in such instances may be associated with more forceful ventricular contractions. This occurs most frequently in systemic hypertension. Increased amplitude of the first sound is often evident. In the hyperdynamic heart syndrome, a marked increase in amplitude of the sound is almost invariably present and may be the first clue that this entity is present. Reduction usually takes place after treatment with propranolol. Tachycardia from any cause increases the amplitude of the first sound. Although this is partly due to enhanced contractility from calcium release, a more important factor is the more widely open atrioventricular valves due to the short diastolic filling period (see below).

Another factor is the diastolic volume (preload). The reader will recall the Frank-Starling mechanism, in which increasing diastolic fiber length is associated with increased cardiac output. As diastolic volume increases, the force of contraction and therefore the first heart sound, also increase. Reduced filling pressure, as seen in shock or hypotension, is commonly associated with soft and low amplitude sounds. In tachycardia and in the beat following a premature contraction, diastolic filling time is shortened with a decrease in ventricular dp/dt. In these instances, however, the reduced force of contraction may be partly counteracted by the position of the atrioventricular valves.

When left-ventricular end-diastolic pressure is elevated, although diastolic fiber length is increased, the underlying impaired myocardial contractility tends to reduce the first sound. In addition, the elevation in pressure may induce partial closure of the valves.

In the presence of atrial fibrillation, first-sound amplitude is related primarily to the degree of diastolic filling. The greater the degree of filling, the more forceful the succeeding ventricular contraction. If associated mitral stenosis is present and first sound amplitude does not vary with preceding diastolic length, calcification and immobility of the valves should be suspected.

Complete heart block, although associated with a constant diastolic length, produces variable ventricular filling, depending upon the timing of atrial systole. If atrial systole occurs when the atrioventricular valves are open, enhanced ventricular filling occurs with an increased sound. Thus, a first sound of variable amplitude occurring at regular intervals should make one suspect complete heart block.

Miscellaneous factors may also reduce the force of ventricular contraction. In aortic stenosis and insufficiency, the ventricle is unable to raise its pressure as high as when the valve is intact. Consequently, the first sound is often reduced. In complete left-bundle-branch block, activation of the left ventricle is markedly reduced, with a slower rate of pressure rise. In such instances, the first sound is almost always markedly reduced.

Factors affecting position of atrioventricular valves.—Under normal circumstances, the timing of atrioventricular valve closure is determined by the P-R interval. When this interval is short (i.e., 0.12 seconds), atrioventricular valves are widely patent and will close with more force shortly after the onset of ventricular contraction. When the P-R interval is prolonged (i.e., 0.20 seconds), the atrioventricular valves have floated back to a nearly closed position, creating a soft first sound. If the P-R interval is markedly prolonged, as in first-degree block, the valves will separate again. In such instances, their force of closure depends upon their position.

When left-atrial pressure is elevated, as in mitral stenosis, a gradient exists between the left atrium and left ventricle. In mild stenosis this gradient is minimal late in diastole, so that the rate