On Cardiovascular Sound

Further Observations by Means of Spectral Phonocardiography

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Spectral phonocardiography, an adaptation of the method of sound spectrography devised at the Bell Telephone Laboratories, differs from conventional oscillographic phonocardiography principally in the fact that frequency spectrum is displayed as well as the dimensions of time and intensity. The display of frequency spectrum is responsible for three advantages of the method: (1) Quality, or timbre, is given physical definition, (2) resolution in the time dimension is improved, and (3) a better display of the wide dynamic range of cardiovascular sound is attained. This method can do, or can be made to do, all the ear can, and can probably emulate the performance of the ear because 1) it is not wed to a particular frequency-intensity response curve, 2) it suffers from no "psychoacoustic" impediments, 3) it provides better resolution in the time dimension, and 4) it produces permanent, quantifiable records. Discussed here are selected features of mitral valve disease, aortic valve disease, gallops, systolic clicks, and extracardiac sounds. In general, intensity and peak frequency (or frequency range) vary in a parallel manner in sound of cardiovascular origin. Artefacts due to noncardiovascular ambient noise and to electrical interference can be identified and disregarded in the heart sound analysis.

CONVENTIONAL phonocardiography, now about 60 years old,1,2 has contributed significantly to the interpretation of clinical stethoscopy of the cardiovascular system and to the understanding of cardiovascular physiology, normal and abnormal. Timing of auscultatory findings and temporal correlation with other physiologic events have been the principal types of usefulness in both the clinical and the physiologic domain. Particularly at the lower end of the frequency scale where the ear is much less sensitive the conventional phonocardiogram has provided valuable information. Inevitably the information about cardiovascular vibrations in the audible range is limited by the methods available to the investigator. Further advances in the study of heart sounds and murmurs require new methodological approaches. This communication presents observations made by one such new method, sound spectrography, which in its application to the study of cardiovascular sound may well be termed spectral phonocardiography. This report is a continuation of two early ones, one of which was largely technical3 and the other more particularly clinical.4

About 10 years ago the Bell Telephone Laboratories developed a method for three dimensional display and analysis of speech sound.5 It has been called sound spectrography (also "visible speech") because frequency spectrum is displayed in detail. It was early demonstrated that whereas the oscillogram of speech sound has no particularly distinctive characteristics—it might, in fact, represent a very abnormal phonocardiogram—the spectrogram of the same sound is sufficiently detailed and characteristic that one can not only learn to read what is being said but also discern minor individual peculiarities of speech such as regional accents. The application of this method to phonocardiography was further encouraged by verbal reports of success with its use in other connections: for example, when used on complex airplane engines it has

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been possible to detect and diagnose abnormality much as a good mechanic does in listening to an automobile motor.

Elsewhere we have described the modifications of the commercially available sound spectrograph which are necessary for the particularly demanding application of phonocardiography. These modifications are of three main types:

1. The filter system must be modified to make available the width of pass-band which provides optimum simultaneous resolution in the time and frequency dimensions.

2. Parallel recording of other physiologic events (for correlative purposes) has been instrumented.

3. Improved resolution of the intensity display has been obtained by development of a photographic recording system.

METHODS

Figure 1 presents a schematic representation of the methods employed in these studies. For pick-up from the patient, a condenser-type microphone (Altec 21BR150) has been employed. This is a calibrated transducer with linear characteristics. Recordings were made under as quiet ambient conditions as possible. The microphone-chest piece combination employed provides a maximum of 20 decibels attenuation of ambient noise above 200 cycles per second. The patient is essentially isolated from floor vibrations by an air mattress.

An Ampex two-channel, type 350, magnetic tape recorder has been employed. Recordings have been made at 7½ inches per second. To record the full dynamic range of the heart sounds, three degrees of low frequency compression have been available. With no compression the response characteristics of the entire system are flat above 30 cycles. At 20 cycles the response has dropped 5 to 10 decibels and at 15 cycles has dropped a maximum of 20 decibels below the response level above 30 cycles. Initial recording on magnetic tape has increased the flexibility of the method. At the convenience of the investigator, the tapes can be edited and sections selected for analysis. Recording at the bedside, in the operating theatre, or in the animal experimental laboratory has been feasible.

By means of frequency-modulated carriers, electrocardiogram, respiration mark and other physiologic events have been recorded simultaneously on the magnetic tape and then on the spectral phonocardiogram, as indicated in the sketch (fig. 1). The conventional phonocardiogram can, if desired, be included as one of these parallel recordings.

In effect the principle of the analyzer of the spectral phonocardiograph is this: the segment of sound for analysis is "played over" from the original tape to the magnetic margin of a disk mounted on the same axis as the kymograph drum on which the records are made. The analyzer itself is in essence a single pass-band filter the tuning of which is changed progressively as the segment of sound being analyzed is played back repeatedly through it. For example, during the first rotation of the magnetic disk and the coaxial drum, the information in the 15 to 20 cycle per second band is inscribed on the record, with the second rotation the information in the 20 to 25 cycle per second band and so on up the record from bottom to top until the entire frequency scale has been scanned.

Note that in the finished record three variables of the heart sounds are displayed. As usual, time is on the abscissa. Frequency spectrum is on the ordinate. Intensity is indicated by degree of blackness of any given portion of the record since the brilliance of the light in the photographic recording unit (above in fig. 1) is a function of the intensity in each frequency band and since there is also intensity-modulation of the spark of the direct-writing unit (below in fig. 1). Most of the recordings up to this time have been made on electro-sensitive paper, i.e., are of the direct-written type. Since the range of intensity displayed by this paper is limited, perhaps to 15 decibels, the photographic recording system has been devised to increase the resolution in the intensity dimension by several times. Although the dynamic range of the heart sounds may be as much as 100 decibels, the range in any one frequency band should be much less and easily encompassed by the capacities of the display unit. More of a problem is the limitation of intensity recording imposed by the two magnetic recording steps. A comparison of a direct-written and a photographic recording is provided in figure 2.

In the analyzing step the pass-band which has
provided optimum simultaneous resolution in frequency and time has been one with a width of about 23 cycles per second at 6 decibels below the intensity level at the center frequency. However, occasionally when the information desired was of a special variety other widths of pass-band might be employed, e.g., a wider one for particularly sharp time definition. A 750 cycles per second frequency range was adequate in the majority of cases although a 1500 cycles per second or higher scale was occasionally necessary to encompass the total frequency range encountered. In essence, then, since the drum rotates 425 times in the inscription of the analysis from 0 to 750 cycles per second, we have analyzed these sounds through the equivalent of approximately 425 overlapping filters, each with its center frequency about 1.8 cycles per second from the next.

The presently used spectrograph produces a record with a four-inch frequency scale (ordinate). A segment of sounds 6.6 seconds in duration is displayed on a record 12½ inches long.

Calibration of intensity has been accomplished by the introduction just beyond the microphone of a calibrating signal at 60 cycles. By means of the cathode ray oscillographic monitor, identical gain in the step of “playing over” into the analyzer is assured. We have adopted the practice of noting in connection with each finished record the oscillographic amplitude of the calibrating signal in millimeters. The microphone has been held to the chest in such a way that pressure with which it is applied is the weight of the microphone itself (fig. 3). This obviates the sound variations which result when a rubber strap or similar nonstandard device is employed. It is possible by these means to obtain recordings which, as far as the sound which arrives at the surface of the chest is concerned, are entirely comparable from time to time in the course of valvular heart disease, before and after operation, and so on.

It may appear that this method has sacrificed precise intensity definition for frequency detail. It should be noted, however, that intensity has most significance in relation to the frequency level at which it is located. Further detail as to intensity at specific points in time is provided by the intensity-frequency “sectioner” of the sound spectrograph. Such “sections” allow one to state, if such detailed information be of use in specific situations, the maximum intensity (in dynes per square centimeter of sound pressure or other unit) in a given frequency band at a given point in time.
**Fig. 4.** a–f. In "a" and "b" are presented successive frames in the closure of the pulmonary valve; in "c" to "f" are presented a comparable series in the closure of the tricuspid valve. See text for details.

**Some Aspects of Normal Heart Sounds**

The first heart sound is often a longer sound than the second heart sound. Figure 4 presents two sequential frames (made at the speed of 24 frames per second) in the closing of the pulmonic valve and four in the closure of the tricuspid valve in the same isolated beef heart under identical circumstances. It is of interest that the tricuspid valve took about twice as long to close. The fact that the tricuspid valve closed first at the middle is also of some interest.

Lamb\(^6\) states that “according to a general

\(^6\) From a film (PMF5162) made by the Armed Forces Institute of Pathology.
principle... the higher harmonics are excited in greater relative intensity the more abrupt the character of the originating disturbance.”

This principle may explain in part the higher frequency content of the second sound as compared with the first and also the high frequency content of systolic clicks, opening mitral snaps and the valve sounds in the fetus, children, tachycardia and small animals.

**Diseases of the Mitral Valve**

Few sounds commonly encountered in clinical practice are more complex in their genesis and acoustics than those associated with mitral stenosis. It is useful in this connection to refer to Duroziez’s onomatopoetic device: “fout-ta-ta-rou.”7 “Fout” refers to the presystolic murmur and snapping first mitral sound; “ta-ta”, to the second sound and the opening snap; “rou” to the so-called mid-diastolic component of the diastolic murmur, which may be introduced by a discernible third heart sound. (See fig. 5.)

There are two principal theories of the genesis of the opening snap: one holds that it is due to sudden checking of opening.8 The other holds that the opening snap of the mitral valve and perhaps the snapping first sound at the apex, the closing mitral snap if you will, are the result of a “wind-in-the-sail” mechanism. Obviously the theories are not mutually exclusive.

Shown in figure 6 is a sketch which serves to illustrate the second theory. It is generally held that the type of mitral stenosis likely to produce an opening mitral snap is that in which the valve leaflets are fused into a funnel-shaped curtain with ring-like thickening of the margin of the “fishmouth” and some increased rigidity of the valve curtain, yet sufficient flexibility to permit a snapping back and forth as pressure relationships on its opposite surfaces undergo cyclical variations. With fall in ventricular pressure below that of the left atrium the valve curtain billows toward the ventricle producing a snapping sound, “the opening snap.”9 With ventricular contraction and elevation of ventricular pressure above that in the atrium an abrupt bulging occurs in the opposite direction with production of a snapping sound which is probably responsible in part for the accentuated first apical sound. Holldack9 has aptly compared the opening snap to the snapping of a sail with shift in the wind. It was the opening snap which in combination with the first and second sounds produced at least one type of *bruit de rappel* described by Bouillaud10 who with others was also responsible for relating this type of heart involvement to acute inflammatory rheumatism (rheumatic fever). Rouches11 introduced the customarily used term *le claquement d'ouverture de la mitrale*, saying his mentor Potain used it in his lectures. William Sidney Thayer12 introduced the English analogue: “opening snap.” The study of Margolies and Wolferth13 published in 1932 remains one of the best. It seemingly received scant notice at the time it appeared, probably because of limited interest in, and restricted value of, the auscultatory minutiæ of valvular lesions. These investigators described the variation in the second sound-snap interval with heart rate, the not infrequent gap between the snap and

**Fig. 6.** The abrupt billowing of the mitral curtain toward the atrium with ventricular systole and toward the ventricle in diastole is a possible explanation for the snapping first sound and the mitral opening snap in mitral stenosis. Adapted from Holldack (9).

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**Fig. 5**

Duroziez’s onomatopoetic device.

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the beginning of the rumble and the area of maximum audibility in the left midprecordium.

The opening snap has, in most cases, persisted after operation. The "wind-in-the-sail" explanation is not entirely satisfactory in this instance; the postoperative opening snap may be a less specific type of flapping of the stiffened leaflets—like a housewife shaking a rug or a bed-sheet. Or the persistence of the opening snap may be evidence for the view that abrupt checking of opening is principally responsible for its genesis.

The opening snap is usually loudest in an area of the chest directly overlying the mitral orifice. The scatter map of the points of maximum audibility in the series of Margolies and Wolferth resembles the fluoroscopic image in a case of mitral stenosis with valvular calcification. The opening snap is often louder in the pulmonary area rather than at the apex. It is to be noted that although the second pulmonic sound may be genuinely split in mitral stenosis, more often what has in the past been called a split or reduplicated second pulmonic sound is in fact the second pulmonic sound proper plus the opening mitral "snap." Not infrequently the opening snap is louder in the aortic than in the pulmonary area. Wood found that the opening snap was absent in a case of predominant mitral stenosis with aortic regurgitation. He suggested that the regurgitation against the aortic leaflet of the mitral valve during isometric relaxation and early diastole might prevent the sudden billowing of the mitral sail responsible for the snap. Galloward in one case and Mozer and Duchosal in two likewise failed to detect an opening snap in the presence of aortic regurgitation. On the other hand, Margolies and Wolferth found five cases of opening snap in spite of the presence of aortic regurgitation.

Within limits the interval between the second sound and the opening snap tends to vary directly with the duration of the preceding diastolic period. Since this interval is a measure of isometric relaxation, the question of the basis for this variation resolves itself into the question of why isometric relaxation is shorter after shorter diastolic periods, and vice versa.

Opening snaps are identified as such by several means: (1) They tend to be 0.08 to 0.10 second after the second sound. (Split heart sounds, in the absence of bundle branch block or shunt with discrepancy in the stroke volumes of the two ventricles, seldom show a separation of the components of more than 0.06 second. A third heart sound gallop seldom is closer than 0.12 second to the second sound.) (2) In the spectral phonocardiogram in particular it is usually possible in some areas to demonstrate slight splitting of the second sound proper, thereby accounting for the closure of both sets of arterial valves. (If the usually employed filter setup is changed for one with wider pass-band characteristics the splitting can be better displayed.) (3) They are snapping or clicking in quality as evidenced in the spectrogram by relatively high frequency span and often either harmonic pattern or relatively pure frequency content.

Somewhat puzzling is the brief interval which is present in many cases between the opening snap and the onset of the murmur. (In others the rumble begins immediately with the opening snap.) Can it be that the pressure differential adequate to snap the mitral curtain toward the ventricle is not sufficient to spread the stenotic fishmouth and initiate passage of blood to the ventricle (and the murmur)? Is the anatomical difference between the cases with an interval and those without a qualitative or a quantitative one? In general it appears that the difference is quantitative, i.e., that the cases, in which the rumble begins immediately with the opening snap, have a higher grade of mitral obstruction.

It is also difficult to understand the origin of a third heart sound in this situation where filling of the ventricle is impeded. It seems likely that the third heart sound recorded in some cases of "tight" mitral stenosis is a third sound gallop arising in the right ventricle. With exercise and the left lateral decubitus position the third sound may be exaggerated and the opening "snap" diminished. If a third sound gallop appears to arise in the left ventricle, as, for instance, in cases of mitral stenosis without signs of right ventricular hypertrophy and dilatation and therefore without basis for a right ventricular origin of the gallop, insignifi-
cant mitral valve obstruction is likely to be present.

The genesis of the snapping first sound may be in part that indicated by the sketch in figure 6 and in part snapping of the shortened, stiffened chordae tendineae when they bring the attached mitral valve “up short” on contraction of the ventricle. Furthermore, the fibrotic change in the valve cusps may produce a ringing, accentuated second aortic sound. There is a close correlation between an accentuated ringing first apical sound and an opening mitral snap; both, furthermore, are indicative of predominant mitral stenosis.\(^{13}\)

As is discussed and demonstrated below (see figs. 13 and 15) the characteristic murmur of ejection stenosis (e.g., aortic or pulmonary stenosis) is diamond-shaped on conventional phonocardiography and like a Christmas tree in the spectrogram. These configurations are mainly due to a midsystolic peak in intensity and in frequency. In mitral stenosis one might anticipate that the presystolic murmur would have a similar configuration. Such is probably the case when the P-R interval (atrioventricular conduction) is sufficiently prolonged that the presystolic murmur “stands alone” and is not cut short by the first heart sound. The presystolic crescendo can be considered to represent half the “Christmas tree” of an ejection stenosis murmur.

Conventional phonocardiography has not always corroborated the auditory impression of a crescendo character of the presystolic murmur of mitral stenosis. The reason in many cases may have been that the instruments used were insufficiently sensitive to the higher frequencies (which impress the ear most) to demonstrate the intensity increment which the higher pitched components contribute to the presystolic crescendo.

Note in figure 7, a recording from the apical area (D. C., 687240), the close reproduction of the Duroziez device. In the pulmonary area (not displayed) both a split second sound and an opening mitral snap were evident. In some cycles a distinct third heart sound is seen. The half Christmas tree appearance of the presystolic murmur is well displayed.

The patient (S. O. N., 616116) from whose apical area the recordings in figure 8 were made at an interval of six months has a type of diastolic rumble which begins immediately with the opening snap. In the interval between the two recordings the patient developed atrial fibrillation. Note the disappearance of the presystolic element of the diastolic rumble and the variation in the interval between the second sound and opening snap. In the first recording the interval is brief since the heart

![Image](http://circ.ahajournals.org/)

FIG. 7. Apical area in patient with predominant mitral stenosis
rate is rapid with correspondingly short diastolic periods. With atrial fibrillation the interval is shorter after the shorter diastolic periods, e.g., the last cardiac cycle in the recording. Variation in the length of isometric relaxation is indicated by the variation in the position of opening snap in relation to the second sound. The second sound is relatively constant in its position relative to the QRS of the electrocardiogram. The delay of the first heart sound in the presence of mitral stenosis and atrial fibrillation is displayed by the recording. Usually in atrial fibrillation the first heart sound is accentuated after shorter diastolic intervals because the atrioventricular cusps come together from a more widely open position. In this case of mitral stenosis, an opposing factor, namely, poor ventricular filling with short diastoles, may dominate and result in a diminution of both the intensity and the frequency span of the first sound. (See the first sound in the last cycle of fig. 8).

Figure 9 presents the recordings from the pulmonary area and apex of a patient (H. L., 346674) with virtually all the hallmarks of mitral stenosis except an unequivocal and consistently present diastolic rumble. We have observed this situation previously and it is rather commonplace after mitral valvulotomy. In this pair of recordings note that the opening snap is much more striking at the pulmonary area than at the apex. Note, furthermore, the accentuated and ringing (viz., the harmonic pattern) first apical sound and the accentuated second pulmonic sound. That the extra sound is indeed an opening snap and not the second component of a widely split second sound is supported by the fact that slight splitting of the second sound is present accounting for both the aortic and the pulmonic closure sounds.

Figure 10 presents the sounds at the apex in a patient (A. B., 167680) who three years previously had mitral valvulotomy for relatively pure mitral stenosis. To be noted are (1)
the decrescendo type of systolic murmur of
the mitral regurgitation created at operation,
(2) the opening mitral “snap,” and (3) the
fact that the diastolic murmur is limited to
presystole. The last phenomenon has been
observed in other patients after operation.  

Some of the motion pictures which have
been made of in situ functioning A-V valves
demonstrate a low frequency flutter of the
valve margin when the atrium contracts. The
passive flow of blood into the ventricle in
early diastole produces little vibration of the
valve margins. The facts that the valve cusps
have floated up toward the closed position
when the atrium contracts and that increased
velocity is imparted to the atrioventricular
stream by atrial contraction may account for
this marginal flutter in the normal situation.
As for the postoperative state: these cases
have no evidence of persistent obstruction of
any significant degree. However, the anatomical
state of the mitral leaflets is far from nor-
mal. They are irregular and present abnormal
“tag ends” for vibration. They are stiffened to
the point that they vibrate more noisily with
atrial contraction. Atrial contraction may, in
the postoperative period, still be unusually
forceful as a result of the persisting hyper-
trophy of the left atrial wall. All of these
considerations are consistent with the observa-
tion that the presystolic portion of the diastolic
murmur is the first to appear and is the part
present in cases of trivial mitral stenosis. In
essence mitral valvulotomy tends to convert
the patient from one with severe stenosis to one
with mild stenosis and a varying amount
(from none up) of mitral regurgitation.

The following features of interest are dem-
onstrated by the patient (E. L., 227489) (with
mitral stenosis and regurgitation and atrial
fibrillation) from whose apical area the re-
cording presented in figure 11 was made: (1)
The low frequency content of the first apical
sound is striking. This is a dull sound. (2) The
systolic murmur is relatively high-pitched
and is crescendo in frequency and intensity
construction. This type of systolic murmur is
to be compared with that in figure 10. (3) A
third heart sound initiates the diastolic
rumble. (4) There is no presystolic component
to the diastolic murmur. (5) There may be a
faint opening snap. (6) The rumble is decres-
cendo in intensity and to some extent in peak
frequency. One would suspect predominant
regurgitation in this instance and the suspicion
is supported by ancillary clinical information
on this patient.

The systolic murmur of mitral regurgitation
is always holosystolic but it may be cresendo
(see fig. 7b of Reference 3), decrescendo (see
figure 10) or plateau in nature (see fig. 7 of
Reference 4).

More often than not a third-sound gallop is
present with mitral regurgitation. This is probably due to increased volume and rate of diastolic filling of the ventricle. Not only the usual quota of blood enters the ventricle during each diastole but also that volume regurgitated during previous systole. This same factor, increased volume of mitral flow, is responsible for the diastolic rumble being louder than would be the case with the same degree of obstruction and no mitral regurgitation—mitral regurgitation tends to create a relative mitral stenosis. In this sense mitral regurgitation should be listed among the conditions which may simulate mitral stenosis.

Relative mitral stenosis can occur in two situations (usually both factors are operating in a given case): (1) Torrential flow through the mitral orifice and (2) dilatation of the ventricle without proportional dilatation of the mitral ring. The first factor is probably the leading one in the genesis of the middiastolic rumble which occurs with patent ductus arteriosus and interventricular septal defect, although dilatation of the ventricle is probably also present. The second factor is principally responsible in the genesis of the diastolic rumble which occurs early in acute rheumatic carditis. However, here too there is probably usually increased mitral flow since there is usually mitral regurgitation. In chronic anemia, such as that of sickle cell disease, the apical diastolic rumble is probably about equally the result of the ventricular dilatation and the increased flow accompanying anemia.

In figure 12 is presented the recording at the apex after exercise in a patient (E. F., A58028) with active rheumatic carditis. Before exercise the systolic murmur and a much less conspicuous protodiastolic gallop were present. After exercise the gallop was exaggerated and was followed by a short rumble. During a course of cortisone in high dosage, the gallop and rumble became fixed even at rest. We have observed this same type of diastolic rumble, initiated by a third sound gallop, in teen-age patients after cardiac decortication for constrictive pericarditis. In these there has been no reason to suspect organic mitral stenosis. The left ventricle has at times been evidently dilated.

DISEASES OF THE AORTIC VALVE

The murmur of aortic stenosis has not only an intensity peak in midsystole as revealed by conventional phonocardiography (giving the familiar diamond configuration) but also a frequency peak in midsystole as revealed by spectral phonocardiography. This results in the characteristic Christmas tree shape shown in figure 13. The frequency peak occurs in this case at approximately the point midway between the first and second thirds of systole. It is clear from examination of this type of record why conventional phonocardiograms of the “high frequency” type usually demonstrate best the typical diamond configuration of ejection stenosis murmurs. (Fig. 15 presents the same “Christmas tree” pattern in the murmur of pure pulmonary stenosis of valvular type.)

Figure 13 presents the recording from the aortic area of a case of rheumatic heart disease with aortic stenosis and regurgitation. The gap between the end of the systolic murmur and the second sound and diastolic murmur is noteworthy. Poorly organized harmonics are also apparent in the systolic murmur. The simultaneous photographic and direct-written recordings presented in figure 2 for the sake of comparison were made from the aortic area of a patient with a “raspy” systolic murmur of calcific aortic stenosis. It is seen that the raspy quality is attributable to the presence of
harmonics and the particular frequency distribution of the overtones.

In hypertension the second sound in the aortic area becomes accentuated not only in intensity but also in peak frequency as indicated in figure 19. A "ringing" quality to the ear, manifested by the presence of harmonics in the spectrogram, is also acquired. Similar alterations in the second aortic sound may occur in the absence of hypertension if structural changes occur in the valve cusps altering their vibrational characteristics. A ringing, accentuated second aortic sound is characteristic of sclerosis of the aortic cusps. It has in the past been accepted as *prima facie* evidence of syphilitic aortitis. Since intimal changes of atherosclerotic nature occur at the level of the sinuses of Valsalva and on the aortic cusps themselves as a secondary effect of syphilitic aortitis, a ringing aortic second sound in syphilitic aortitis may have a basis in these changes. However, the sign would seem too nonspecific to be used as more than supporting evidence for the diagnosis.

Early in rheumatic aortic valvulitis, before or as aortic regurgitation develops, the second aortic sound may be accentuated in normotensive patients. A case in point is illustrated in figure 14. This 15 year old girl (N. B., 657032) has mild aortic regurgitation and normal blood pressure but a much accentuated second sound in the aortic area. This finding may be due to changes in the vibrational properties of the valve as a result of rheumatic fibrosis. Another contributing factor in the accentuated aortic second sound of rheumatic aortic valvulitis when aortic regurgitation is present may be the "systolic collapse" phenomenon of Wiggers. Because of the lower diastolic pressure against which the ventricle is ejecting, ejection occurs earlier in systole with a higher systolic pressure peak and a sharp collapse of the pressure curve after this peak. This rapid fall in pressure may result in accelerated aortic valve closure and an accentuated second heart sound.

There are, then, two mechanisms for ringing second aortic sound: (1) Increased force of closure as in diastolic hypertension and (2) stiffening of the valve cusps. Both may be factors in a given case.

**Gallops**

Figure 15 presents an example of atrial gallop which occurred in a patient with pure pulmonary stenosis of valvular type (J. B., 680181). Note the characteristic appearance of

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**Fig. 13.** Aortic area in aortic stenosis and regurgitation on rheumatic basis. Note Christmas tree configuration of systolic murmur, gap between murmur and second sound ("2"), diastolic murmur decreasing in both intensity and peak frequency.

**Fig. 14.** Aortic area in 15 year old girl with early aortic valvulitis on a rheumatic basis. Second aortic sound (A2) is very ringing in character with harmonic pattern and accentuation of both intensity and peak frequency. These changes may be due to valvular fibrosis. There is an early diastolic murmur.
this ejection stenosis murmur. Figure 16 presents the recordings in four areas of a patient (W. B., 593724) with severe arterial hypertension. Again there is a striking presystolic or atrial gallop loudest at the apex. In the aortic area there is a faint early systolic click of the type frequently seen in aortic atheromatosis, aortic dilatation, syphilitic aortitis or hypertension (see below). Of these two cases of presystolic gallop, the extra sound presumably arises in the right side of the heart in the case of the first and in the left side in the case of the second.

The patient (A. E., 299576) from whom the recording in figure 17 was made has very marked delay in atrioventricular conduction as indicated by the long P-R interval in the electrocardiogram. There is a striking atrial, or presystolic, gallop ("a") in this recording from apical area. The first sound is indistinct and merges indistinguishably with the systolic murmur. The second sound is likewise difficult to identify. The patient has aortic regurgitation and stenosis and mitral regurgitation. The early diastolic murmur may be transmitted from the aortic area. The fact that the atrial sound is followed by no murmur, comparable to the presystolic murmur of mitral stenosis with sinus rhythm and an anomalous P-R interval, suggests that mitral stenosis is not present. Nylin demonstrated such a murmur after the auricular sound in a patient with heart block and mitral stenosis. In brief, this is a case of atrial, or presystolic, gallop in which the gallop sound was rendered mesodiastolic in timing by the prolongation of atrioventricular conduction.

Figure 18 presents another mesodiastolic gallop recorded at the tricuspid area in a patient (C. C., 196902) with severe hypertension. This is probably a summation gallop since in many cycles slight splitting of the gallop sound

![Fig. 15. Presystolic gallop ("a") in association with pure pulmonary stenosis. Note Christmas tree configuration of this ejection stenosis murmur.](image1.png)

![Fig. 16. Recorded at areas indicated in patient with malignant hypertension. Note early systolic click at base and the presystolic gallop.](image2.png)
is discernible, the first component probably being protodiastolic and the second presystolic.

The patient whose heart sounds at the apex are pictured in figure 12 above had active rheumatic carditis with mitral regurgitation. The protodiastolic gallop (“G”) so frequently present and seemingly due to hyperdynamic ventricular filling is displayed. (See section on disease of the mitral valve.)

Systolic Clicks and Other Circumscribed Systolic Sounds

Systolic clicks and other circumscribed systolic sounds can be classified as to mechanism in the following manner:

I. Those due to arterial abnormalities.
   a. Dilatation of the pulmonary artery, as in mitral stenosis, interatrial septal defect, patent ductus arteriosus.
   b. Dilatation of the ascending aorta, as in atherosclerosis, hypertension and syphilitic aortitis.

II. Those arising by extracardiac mechanisms.
   a. Pleuropericardial adhesions.
   b. Plaques of calcification in the pericardium.
   c. Grating of xiphosternal, costochondral or chondrosternal joints by ventricular contraction.
   d. Uncoiling and dilatation of auricular appendage with mitral regurgitation.
   e. Systolic expansion of ventricular aneurysm.
   f. Mediastinal emphysema.
   g. Left pneumothorax.

III. Atrial sound in midsystole due to low nodal (“infranodal”) pacemaker with retrograde conduction.

Figure 19 is the spectral phonocardiogram from the aortic area in a patient (E. W., 782911) with severe arterial hypertension. The accentuated second sound is very striking. Attention is called to the early systolic click. The patient, 33 years old, has mild dilatation...
and elongation of the aorta with "kinked" innominate-subclavian-carotid axis. The early click in hypertension was described by Potain. A snapping of the aortic wall as it is distended by blood may be the mechanism. Leatham and Vogelpoel observed an early systolic click in three cases of coarctation of the aorta, three of aortic stenosis, two of aortic insufficiency and two of aortic sclerosis. All of these had a dilated ascending aorta by x-ray study. In Leatham's experience the systolic click was usually about 0.07 second after the beginning of the first heart sound. A similar early systolic click in the aortic area in malignant hypertension is seen in figure 16.

Figure 20 presents the precisely comparable situation in the pulmonary area of a patient (Z. O., 673149) with interatrial septal defect, pulmonary hypertension and dilated pulmonary artery. The first sound, as well as the second, is slightly split, a common finding in atrial septal defect, even in the absence of bundle branch block. The early systolic click has an intensity concentration at about 240 cycles. In conventional phonocardiograms it is sometimes difficult to distinguish early systolic clicks from split first sounds. The spectral phonocardiograph has less difficulty in this regard. The sound looks like a "click," has "purer" frequency content than heart sounds usually do, and often does not have its frequency "bottom" at zero as do heart sounds proper.

Leatham and Vogelpoel consider this variety of systolic click to represent "ejection vibrations" comparable to those recorded directly from the ascending aorta and pulmonary artery of dogs by Wiggers and Dean in 1917. They are presumed to result from sudden tensing of the great vessel or abrupt attainment of its maximum capacity. Is it possible that the fundamental (seemingly at 240 cycles per second in the click of figure 20) represents the natural frequency of the pulmonary arterial wall in this patient? They are usually inaudible but may become audible when the pulmonary artery or ascending aorta is nearer the anterior chest wall, if the
sound is delayed a bit, as in pulmonary or aortic stenosis, and if they are exaggerated by disease in the wall of the great vessel. The *sine qua non* appears to be dilatation of the great vessel. Leatham and Vogelpoel saw the early systolic sound in 44 cases of pulmonary hypertension with dilatation of the pulmonary artery and in five cases with only the dilated pulmonary artery. Lian and Walti described it in pulmonary stenosis with dilated pulmonary artery.

In figure 21 is presented the heart sounds in the pulmonary area in a patient (M. B., 673169) with marked ascites. These adventitious systolic sounds changed virtually not at all with respiration and were fairly well localized to the area stated. We suspect they are produced by movement in costochondral and/or chondrosternal joints and that the pronounced abdominal distension is an important factor in their genesis, by displacement of the heart. The close similarity to the typical xiphosternal crunch (fig. 22) is to be noted. Note again the elevation of the frequency bottom in the case of these clicks.

In figure 22 is demonstrated a typical xiphosternal crunch. As is frequently the case, confusion for pericardial friction rub occurred in this patient (O. F., 378868). The site of sound production in this situation appears to be the joints of the thoracic cage components in the region of the lower left sternal border. The occurrence of this sound predominantly or only when the subject is in the upright position is consonant with this view. It is doubtful in our minds that the xiphosternal joint often has anything to do with this sound; sternal, or chondrosternal, crunch might be a better name.

The late systolic click demonstrated in figure 23 was very loud over the entire precordium of M. G. (A15325), a 15 year old Negro girl who was observed in acute rheumatic fever a few months before this recording. During the peak of this disease, a pericardial friction rub was present. Subsequently the very loud systolic click developed. The chain of events provides circumstantial evidence for a pericardial origin of the click. Note the features that distinguish the click from a valve closure sound.

The striking systolic clicks displayed in figure 24 were recorded in a 58 year old woman...
(688712) who had had acute rheumatic fever in youth (with, however, no evident valvular residua) and had mild Trichterbrust. The basis may be either pericardial adhesions, or movement of sternocostochondral joints, or both. The evidence that systolic clicks may result from pericardial adhesions was provided by autoptical correlations and by the observation fluoroscopically that localized tugging on the diaphragm occurred synchronously with the systolic click. Note that the telesystolic click in the case illustrated in figure 24 does not have its frequency “bottom” at zero, a characteristic of systolic clicks.

Elsewhere we have presented the spectral phonocardiograms of typical cases of systolic click producing so-called systolic gallop rhythm. Our studies would support the impressions of those who doubt the existence of the entity called systolic gallop, at least this entity distinct from the systolic click. We have observed nothing except these short and high-pitched and therefore clicking sounds. Thompson and Levine stated that 16 per cent of all gallops they encountered were systolic; from our experience this appears to be an accurate approximation of the incidence of systolic clicks. We are in agreement with the French school which refers to these sounds as “claquements” and decries the term “systolic gallop” customarily used in the English cardiological literature.

Figure 25a presents the recording of an early systolic click heard in a well-localized area at the apex. The heart sounds themselves are not remarkable. As is frequently the case with systolic clicks, the adventitious sound has its lowest frequency components at 60 cycles or higher rather than reaching the bottom level as in the case of the heart sounds proper. In this instance there are actually two systolic clicks, the first of which is exceedingly faint. Six years previously the patient (E. H., 452300) had staphylococcal endocarditis on the mitral valve, previously damaged, it was thought, by rheumatic fever. The patient probably has a moderate degree of mitral regurgitation and presents on fluoroscopy and roentgenkymography a most dramatic picture. With each systole the left auricular appendage uncoils, rises out of its bed in the A-V groove and probably expands as does the posterior
aspect of the left atrium. It may be this movement of the left auricular appendage which is responsible for this peculiar sound, perhaps through impact on the pericardium. The roentgenkymograms of this patient demonstrating this finger-like rhythmic outpouching of the left auricular appendage have been published elsewhere. The mechanism of production of the systolic click in this case is comparable to that in ventricular aneurysm.

(Pericardial changes represent another mechanism for systolic clicks after myocardial infarction.)

The 50 year old patient (W. D., 684406) from whom the recording (at the lower left sternal border) in figure 25b was made has electrocardiographic evidence of an old anterolateral myocardial infarction and clear clinical and radiologic evidence of a ventricular aneurysm at the lower left heart border. The sound tracing reveals an early systolic click. This early systolic click is probably the result of expansion of the aneurysm in early systole.

Pericardial adhesions are a less likely basis for the sound. In spite of the loud second sound the patient was normotensive. As was discussed above, this finding suggests sclerosis of the aortic cusps.

Closely related to systolic clicks both as to probable mode of genesis and as to prognostic significance are the circumscribed midsystolic
murmurs which are often introduced by a systolic click. There seems to be general agreement that these are extracardiac in origin, again being related probably to pericardial adhesions and/or roughening. Elsewhere we have published an example of a systolic murmur which probably can be identified as innocent because it is circumscribed, is mid-systolic in timing and is initiated by a systolic click. Figure 26 very forcefully illustrates the usefulness of the information proved by this type of display. The patient (A. H., A81746), 11 years old, had severe rheumatic fever two years previously. A persistent apical systolic murmur was occasion for concern, although no other signs of cardiac abnormality were present. The circumscribed character of the murmur with abrupt onset identifies the sound as extracardiac in origin. Obsolete pericarditis is the probable basis.

The identification of the type of systolic sounds under discussion in this section has usefulness in connection with another not infrequent problem in cardiological practice: the differentiation of chest pain due to pericarditis from that due to coronary artery disease. Even though a pericardial friction rub is not present the presence of systolic clicks or of circum-

**Fig. 27.** In the midst of the normal inspiratory breath sounds there is a circumscribed sound which occurs at about the same point in inspiration (at the beginning) and in systole (early) on each occasion. Recorded in aortic area of patient with advanced pulmonary tuberculosis.

**Fig. 28.** Recording in patient with acute tuberculous pericarditis.
scribed systolic sounds favors the diagnosis of previous pericarditis.

Other Sounds of Extracardiac Origin

In figure 27 is a recording from the aortic area in a patient with advanced pulmonary tuberculosis. With inspiration an early to midsystolic scratchy sound developed. Three of these are marked with arrows. In the cardiac cycle immediately following the first two arrowed “scratches” a slight “scratch” is visible at the same point in systole. The sounds running through the heart sounds in the region of the scratches are typical inspiratory breath sounds. Note the clarity with which the “scratch” and the respiratory sounds can be distinguished. At times pleural and pericardial rubs may be difficult to distinguish clinically since the beating of the heart may generate sound in diseased pleura and pericardial frictions are prone to show pronounced variations with respiration. In this particular case there is no evidence of pericarditis: the scratch was confined to the second and third right intercostal spaces and there was no enlargement of the cardiovascular silhouette.

Fig. 29. Apex in patient with marked ascites. A decrescendo systolic murmur occurs in expiration.

Fig. 30. Apex in patient with very flat chest and mild cardiomegaly due to hypertension. A crescendo murmur occurs in inspiration.
The recording in figure 28 was made from a patient with acute tuberculous pericarditis. There is a loud pericardial friction rub. It has an intensity-frequency-time pattern which is quite distinct from that of murmurs but which is more easily displayed than described. There are two areas of silence in the rub: (1) after atrial contraction but before ventricular contraction and (2) just after the second heart sound during isometric relaxation. There are three main periods to the rub: systolic, protodiastolic, and presystolic.

In figure 29 is shown the recording from the pulmonary area in a patient (W. B., 674663) with pronounced ascites due to Laennec's cirrhosis. There was tachypnea and tachycardia. With expiration an early decrescendo systolic murmur developed. Autopsy revealed no abnormality in the heart and surrounding tissues. In figure 30 is shown the apical recording in a patient (P. R., 364990) with a very flat chest and mild cardiomegaly due to renal hypertension. The contrast with the case in figure 29 is of great interest. In this case the extra sound is late systolic and crescendo and occurs in inspiration. These cardiorespiratory murmurs probably find ready explanation in pulmonary compression by the beating heart or possibly in the rubbing of normal serosal surfaces.36

SUMMARY AND CONCLUSIONS

The conclusions from these studies are of two types: (1) those concerning the usefulness, realized or potential, of spectral phonocardiography, and (2) those concerning cardiovascular sound as revealed by this method. Although few of these observations on cardiovascular sound are original to these studies, several are demonstrated in more convincing fashion than has previously been possible. The following observations on cardiovascular sound have been made: (Some of these have been discussed very little here but are covered in more detail in other publications from this laboratory.)

1. In general intensity and peak frequency (or frequency range) vary in a parallel fashion in given heart sounds or murmurs. The louder a sound or murmur, or a portion thereof, the higher is its frequency span. For example, the murmur of aortic stenosis, described as diamond-shaped because of its intensity peak in mid-systole as displayed by classical phonocardiograms, also has a frequency peak in mid-systole. Similarly murmurs which are so described are crescendo or decrescendo both as to intensity and as to peak frequency. (It is of interest that DeCastro37 came to the same conclusion on the basis of stethoscopic estimation of pitch.) Accentuated heart sounds, such as the first sound at the apex in mitral stenosis and the second sound at the base in hypertension of the greater or lesser circulation, are accentuated in both intensity and in peak frequency. Other cases in point are the accentuated first sounds after shorter diastolic periods in atrial fibrillation and the cyclical variations in the continuous murmur of patent ductus arteriosus, Blalock-Taussig anastomosis and thyroid bruit.

2. In general, the more abrupt the physiological process producing the sound, the higher the frequency representation in the sound. Cases in point include the normal second heart sound, systolic clicks, opening mitral snaps, fetal heart sounds, and the sounds in children, small animals, and tachycardia. Dull heart sounds, sounds of so-called “poor quality,” usually lack components of higher frequency, due in part to slower, less “snappy” closure of the valves.

3. Since they are total and detailed representations of cardiovascular sound, spectral phonocardiograms display timbre (quality) and permit its description in physical terms. The following are specific points in this connection:

a. “Ringing,” “tambour,” “pistol-shot” have their physical basis in a short duration and in high frequency content (wide frequency span) with conspicuous harmonics.
b. Clicks and snaps are brief sounds with moderately high peak frequency and uniform intensity distribution over the frequency range, i.e., no harmonic banding. Frequently clicks and snaps do not have their frequency bottom at or near “zero” as do the valve sounds.
c. Musical murmurs owe their acoustical properties to the presence of more or less well-
organized harmonic systems. The configuration of these systems as to timing, intensity and frequency determines whether "rasping," "shriek," "honking," "piping," "croaking" best describes the particular murmur. Slopes in, or curvature of, harmonics in musical murmurs can be related to hemodynamic variations at the generator.

d. The low frequency content is the main characteristic responsible for the rumbling quality of the apical diastolic murmur in mitral stenosis.

4. At least partial explanation is provided for the frequent failure of conventional phonocardiography to demonstrate the typical diamond-shape of "ejection stenosis" murmurs, the crescendo character of the presystolic murmur of mitral stenosis or the decrescendo of aortic regurgitation: the contribution of the higher frequencies is not recorded because of lack of sensitivity in that range.

5. Since artefacts such as borborygmi, breath sounds, hiccoughs, electrical interference, speech sound can be identified in the spectral phonocardiogram, they are less likely to occasion confusion.

The usefulness of spectral phonocardiography in clinical and physiological investigation is indicated by the above. In general, the spectral phonocardiogram resembles closely the mental image of heart sounds and murmurs. As much information can be displayed in the spectral phonocardiogram as can be made out by the auditory mechanism; still more can be displayed since the spectral phonocardiogram suffers from no physiologic masking, no fatigue phenomena, and no peculiarities of intensity response at various frequency levels, and since it has a time resolution superior to that of the ear. The resolution of this method in the time dimension is put to test in the display of minute splitting of heart sounds. The improved resolution of time results from the spreading out of the frequency spectrum; even if splitting is not discernible at points of higher intensity, it is likely to be evident in other areas of lower intensity. The spreading out of the frequency spectrum permits a display of the full dynamic range of cardiovascular sound; for example, a very faint diastolic murmur can be accurately displayed in its true proportions in the presence of very loud systolic murmur.

Since it emulates the ear, spectral phonocardiography is ideal for teaching clinical stethoscopy, for detailed presentation of these sounds on the printed page and for precise, quantifiable recording of the course of certain forms of heart disease and the effects of some types of cardiovascular surgery.

Summario in Interlingua

Phonocardiographia spectral es un adaptatio del metodo phonospectrographic disveloppate al Laboratorios Telephonie Bell. Illo differre ab phonocardiographia oscillographic conventional principalmente in le facto que illo monstra non solo le dimensiones de tempore e intensitate sed etiam spectros de frequentia. Iste ultime facto explica tres advantages del metodo: (1) Illo provide un definition physieal del qualitate o timbro del sono. (2) Illo perimtte un meliorate resolution del dimension temporal. (3) Le extendite scala dynamic del sonos cardiovascular es plus claramente demonstrabile in illo.—Iste metodo es capace o pote esser disveloppate a devenir capace a facet toto lo que le aure es capace a facet. Illo probablemente pote equalar le attingimentos del aure proque (1) illo non es restrignet a un specific curva de responsas de frequentia e intensitate, (2) illo non sufre de ule impedimento "psycho-acoustic," (3) illo provide un melior resolution del dimension temporal, e (4) illo produce permanente e quantificabile registrationes. In le presente reporto es discutite seligite aspectos de morbo del valvula mitral, de morbo del valvula aortic, de galpo, del clic systolic, e de sonos extracardiac. In general, intensitate e frequentia maximal varia parallelmente in sonos de origine cardiovascular. Artefactos debite a sonos del ambiente noncardiovascular e a interferentia electric es identificabile e pote esser disconsiderate in le analyse del sono cardiac.

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On Cardiovascular Sound: Further Observations by Means of Spectral Phonocardiography

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