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PREFACE

A STUDY OF THE VALUE OF

PHONOCARDIOGRAPHY

IN CLINICAL MEDICINE

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P R E F A C E

The work presented here was originally undertaken with three main objects in view - firstly, to master the technique of phonocardiography; secondly, to decide whether accurate visual records of the heart sounds could be made with reasonable ease; and, finally, to decide what actual value such recordings might have for the practising physician.

It is already clear that, in the past, phonocardiographic analysis of the various heart sounds and murmurs occurring in health and disease has been of great interest to the physiologist and the enquiring physician. As a research instrument the phonocardiograph is of proved value; as an adjunct to the teaching of cardiology its value is also obvious; but whether or not it can take its place beside the electrocardiograph as an apparatus of practical value to the clinician is still undecided.

During the years 1948-52 the author of this thesis was working as senior medical registrar to Dr. R.W.D. Turner at the Western General Hospital. It was during this time that the Cardiac Department was being developed, and among other pieces of equipment a heart-sound recording apparatus was loaned to the Department by Dr. W.E. Evans of the London Hospital. The author was asked to familiarise himself with this machine, and he thereby gained some experience in the technique of phonocardiography. Later, more efficient and more accurate recording equipment was made available

at the hospital, and the work continued and developed in its scope until, before he left the unit, the author had personally made 213 recordings of the heart sounds from 151 patients. Recordings were all made either in the wards or, more often, in the Cardiac Department during the course of the normal daily hospital routine.

Experience was sought in the study of all varieties of cardiovascular disease and the work was not confined to one particular type of sound, murmur or condition. In this way recordings were made week by week, at first of a fairly simple nature; and thus, in the early months there was a tendency for recordings to be made of gallop rhythms rather than cardiac murmurs, the reason for this being that the low frequency of the heart sounds make them relatively easy to record when compared with the high frequency of heart murmurs. Later, as techniques improved and as more satisfactory phonocardiograph machines became available, all types of heart sounds and murmurs were recorded as they were encountered in the daily routine. The only factors that influenced the selection of cases were, therefore, firstly, the fitness of patients and their ability to co-operate without undue fatigue or distress, and, secondly, the desire by the clinician to obtain graphic information of a doubtful auscultatory sign which was felt to be of diagnostic importance or of medical interest.

In this way, third heart sounds were recorded in normal hearts and abnormal auscultatory phenomena such as gallop rhythms accompanying hypertension, congestive cardiac failure,

congenital heart disease, myocardial infarction and bundle-branch block and other conditions were studied. The systolic and diastolic murmurs occurring in apparently normal hearts and in the presence of aortic and mitral valvular disease were also recorded as they were encountered and when precise diagnosis was in doubt. In the case of mitral stenosis a particularly extensive study was made as surgical operations for relief of mitral stenosis were being developed at this time. A wide variety of recordings was also made in other conditions such as congenital heart disease, pericarditis, and other cardiovascular abnormalities. In particular, specific information was sought on the following points by studying the literature and making a series of recordings from normal subjects and from patients with cardiovascular disease.

- 1) The mechanism of the normal and abnormal heart sounds and murmurs, their physical and auscultatory characteristics, and their appearance when recorded graphically.
- 2) Whether or not any additional diagnostic information with regard to these sounds and murmurs could be obtained from such recordings.
- 3) Whether the auscultatory findings could always be correlated with the appearance of the phonocardiographic tracing.
- 4) The frequency of occurrence and significance of splitting of the first and second heart sounds.
- 5) The frequency of occurrence and significance of the physiological auricular and rapid filling sounds, and their relationship to the extra sounds responsible for clinical gallop rhythm.

- 6) The prognostic significance of rapid filling, auricular, indeterminate, and systolic gallop rhythms, their correct classification and relation to the different types of heart disease.
- 7) The recognition and differentiation between right- and left-sided gallop as originally described by Potain, and since, by others.
- 8) The heart sounds in bundle-branch block and the possibility of diagnosing right- and left bundle-branch block by auscultation and palpation.
- 9) The differentiation between innocent or unexplained systolic murmur and those due to clearcut organic disease.
- 10) The analysis of the auscultatory signs of mitral stenosis and incompetence and their value in the assessment of the severity and degree of the mitral lesion present. Also the separate identification and importance of splitting of the second heart sound, the opening snap of mitral stenosis, and the rapid filling gallop, and their relation to other events in the cardiac cycle.
- 11) The particular significance of the opening snap as a sign of mitral stenosis and its occurrence in mitral incompetence.
- 12) The practical value of recording auscultatory phenomena in miscellaneous cardiovascular abnormalities, either for diagnostic or teaching purposes.

All the 151 patients reported here were examined, carefully auscultated, phonocardiograms made, and the photographic recordings processed and analysed by the author himself.

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Only in the section dealing with mitral disease has information been used which was not obtained from the author's own recordings. In this section some of the final analytical data includes information obtained from a further series of recordings which were subsequently made, after instruction, by the senior electrocardiograph technician. The 66 recordings of 46 patients with mitral disease which are presented in this section, however, were all taken from the author's own series.

The clinical and phonocardiographic observations are presented in Volume I of this thesis and are set out in separate sections. Each section deals with a particular aspect of the problem and is preceded by a survey of the literature and concluded by a sectional summary of the observations. Finally, there is a summary of all the observations presented and the conclusions drawn therefrom. The phonocardiographic tracings which illustrate this thesis are presented together in Volume II.

I should like to express my grateful thanks to Dr.R.W.D.Turner, without whose help and encouragement this work could never have been undertaken nor the thesis written. I am also indebted to the physicians of the Northern Group of Hospitals, and in particular to Dr. E.B.French, Dr. J.G.Macleod, and Dr. J.A.Strong, all of whom were good enough to send their cases for phonocardiographic study. Dr. D.Walker very kindly provided the statistical analysis given in the Appendix. My sincere thanks are also recorded to Miss Marjorie Duesbury for typing the thesis so efficiently and for much help in its preparation.

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INTRODUCTION

When one reviews the literature describing the history of graphic heart sound recording one is impressed with the fact that an accurate and almost complete knowledge of the heart sounds, murmurs and their probable causes was well known to the clinicians before the first phonocardiographic tracing was ever made. Although recording instruments of great precision and complexity have been devised and used over the last twenty years they have, in the main, only served to confirm facts that were previously well known, but graphic proof of which was lacking. The only really new observations which the modern phonocardiograph has made possible have concerned relatively minor details of the form, timing and origin of the heart sounds and murmurs in health and disease. Nevertheless phonocardiography has of course played a part in helping the experimental physiologist to analyse the components of the heart sounds and the precise mechanism of the various murmurs, and it is in this sphere that the most original work has been done.

It is worth while emphasising, however, that by the year 1900, clinicians were familiar with the nature and origin of the first and second heart sounds. The auricular sound and the rapid filling gallop due to an accentuated pathological third heart sound were also described. In the year 1900 Potain had in fact introduced a full classification of the types of gallop rhythm, and this we still believe to be essentially correct. The murmurs of mitral and aortic disease were fully understood; the opening snap of mitral stenosis was well recognised and the association between the pre-systolic murmur and auricular systole understood.

By careful clinical observation combined with pathological studies the art of auscultation had reached so high a level that the heart sounds and murmurs in acquired and congenital heart disease were well recognised, and in most cases their mechanism was understood. The few gaps in knowledge that existed were filled when the Mackenzie polygraph came into use, and although modern phonocardiography has proved to be of considerable interest as a research tool it appears to have added little of practical value to our knowledge of auscultatory phenomena.

More recently the phonocardiograph has been utilised by the clinician as a diagnostic aid, and the various conclusions as to its value will be discussed subsequently.

Auscultation of the heart sounds

Dr. Douglas Guthrie, in his "History of Medicine" (1945), refers to the Ebers Papyrus which was written in approximately 1500 B.C. In this early medical work the pulse is noted as being palpable in various parts of the body and as being synchronous with the heart-beat, but before Harvey described the circulation of the blood it is not surprising that little, if any, attention was paid to the heart sounds. When in 1628 Harvey produced his "De Motu Cordis" he made specific reference to the audible heart sounds and associated them with the circulation of the blood.

However, the science of auscultation did not develop for another 200 years, and it was not until 1819 that the French physician, Laënnec, produced his remarkable treatise on the auscultation of the heart. This accurate account of the heart sounds and murmurs was followed by the equally remarkable and more extensive studies of Potain (1856, 1866, 1875 and 1900). The work of these two great French physicians led to the development of the art of auscultation which was to reach an extra-ordinarily high standard by the end of the 19th century.

Recording of the heart sounds

In 1894 Einthoven and Geluk made the first graphic record of the heart sounds, using a carbon microphone and capillary electrometer, and this arrangement was considerably improved upon when in 1907 Einthoven substituted a string galvanometer for the electrometer. Frank, in 1904, introduced the segment capsule, and it was the combination of these techniques that enabled a larger number of workers to begin their experiments on the recording of heart sounds and led over the subsequent thirty years to the establishment of the modern technique of phonocardiography. Since Einthoven's pioneer experiments much has been written about the theory and practice of heart sound recording. Most of the earlier work was done by investigators on the Continent, but additions to the literature on the subject came later from North and South America, Scandinavia and Great Britain.

On the Continent the work of Einthoven and his colleagues was followed by papers from Holowinski (1896), Hürthle (1904), Frank (1904), Weiss (1909), Weber (1912), Fahr (1912) and Ohm (1914), and a useful summary of the investigations up till 1916 can be found in a paper by Battaerd published in that year. In France the subject was studied extensively by Duchosal (1929), Lian and Minot (1933), Lian and Racine (1933), and up till this time it is probable that the greatest contribution to phonocardiography had come from Lian and his co-workers.

From South America, in 1939, came the classical work of Orias and Braun-Menendez, and in that year they published their monograph entitled "The Heart Sounds in Normal and Pathological Conditions". This still remains one of the most comprehensive studies of the subject, and surprisingly little information of real

added since this publication. It also contains an admirable historical summary of the art of auscultation and the theory and practice of heart sound recording.

In North America an enormous amount of work has been carried out over the last thirty years, originated largely by the studies of Wiggers and Dean (1916, 1917, 1918). Since then, contributions have been made by a variety of workers, notably Rappaport and Sprague (1941 and 1942), Luisiada and his co-workers (1948, 1949 and 1950), Wolferth and Margolies (1930, 1933, 1935 and 1940) and also by J.K.Lewis (1934 and 1938), Dock (1933 and 1945), Arenberg (1940), Eckstein (1937), Boyer (1940 and 1942), Thompson and Levine (1935 and 1936), and Levine (1933, 1937 and 1948). Together with Harvey, Levine produced a comprehensive monograph on the subject of the auscultation of the heart, profusely illustrated with phonocardiograms, in 1949.

Several admirable papers on the subject and some original work - especially in the field of calibrated phonocardiography - have come from Scandinavia in papers written by Frost (1949) and Mannheimer (1940, 1941 and 1942) and Carlgren (1946).

In Great Britain, although one of the first papers on clinical phonocardiography appeared in the Edinburgh Medical Journal in 1913 by Watson, Wemyss and Gunn, most of the early studies on heart sound recording were made by Sir Thomas Lewis, and, with this most notable exception, little attempt at heart sound recording was made until well after the Second World War. In the last ten years, however, a considerable amount of work has been carried out, and papers have originated from the London Hospital by Evans (1943, 1947, 1949, 1951), by Leatham (1949, 1951, 1954), and by Mounsey (1953, 1954, 1955); from St. Bartholomew's Hospital by Wells (1952 and 1954); and from St. Thomas's by Miles (1951). Work has also been published from Glasgow by

Sloan and his colleagues (1952 and 1953) and from Cambridge by Cowen and Parnum (1949) and by Donovan (1948).

Only a general and superficial survey of the literature has been given here, but the various papers referred to above and many more in addition are discussed later in more detail in the appropriate sections which follow.

The physical characteristics of the heart sounds and murmurs, the stethoscope, the human auditory apparatus, and the phonocardiograph

For the recording and interpretation of heart sounds a detailed knowledge of acoustics and electrophysics is not necessary, but a few elementary facts concerning the qualities of the heart sounds, the characteristics of the human ear, and the apparatus used should be clearly understood.

i. The heart sounds and murmurs

A combination of one or more of the following factors gives rise to the various heart sounds and murmurs: firstly, the movement of the valves; secondly, the contraction and relaxation of cardiac muscle; and, thirdly, the blood flow within the chambers of the heart and the aorta. It is possible, though unlikely, that the movements of the heart itself in relation to surrounding structures may also play a part in the production of the third heart sound.

The entire frequency range of heart sounds and murmurs lies somewhere between 25 cycles per second and 1,000 cycles per second. The sounds lie in the region 25 - 400 c/s. Low-pitched murmurs are in the same low-frequency bands, medium-pitched murmurs lie between 240 and 400 c/s., and high-pitched murmurs between 240 and 660 c/s.

Occasional high-pitched musical murmurs may be encountered between 660 and 1,000 c/s. The systolic and diastolic murmurs commonly encountered are probably of frequencies between 160 and 660 c/s. and may occasionally reach 1,000 c/s. as in the early diastolic murmur of aortic incompetence. Pre-systolic murmurs of mitral stenosis are mostly below 140 c/s. but may go as high as 400 c/s. (Rappaport and Sprague, 1941 and 1942; Cabot and Dodge, 1925; Williams and Dodge, 1926; Mannheim, 1940 and 1941).

As the heart sound vibrations are conducted to the surface of the chest they become modified by the various structures such as lung, bone, subcutaneous tissue, skin and fat through which they pass.

ii. The stethoscope

From the chest wall the sounds are again modified by the stethoscope depending on the size and type of chest-piece used and the length and diameter of the stethoscope tubing. (Rappaport and Sprague, 1941). Once the sounds reach the human ear they are again modified considerably, and here again the modifications will vary between different individuals, depending on the degree of training in auscultation and the particular characteristics of the listening ear.

Modification by transmission from the heart to the chest wall cannot be controlled or accurately estimated.

Modification by the stethoscope tends to attenuate the very low frequencies to a lesser extent with the open bell chest-piece of wide diameter than with the diaphragm chest-piece which leads to greater attenuation of the lower frequency sounds and therefore accentuates and brings out the higher frequencies.

A simple illustration of the part played by the stethoscope in the modification of heart sounds as they are conducted through it is provided by the experimental work of Rappaport and Sprague (1941 and 1942). They showed by accurate measurement that the bin-aural stethoscope was ten times more efficient for sound ranges between 60 and 400 c/s. than the mon-aural. Only between 850 and 1,000 c/s. was the mon-aural more efficient than the bin-aural.

iii. The human auditory apparatus

Modification of the sounds and murmurs by the human ear are considerable. Broadly speaking the low-frequency sounds are considerably attenuated as the human ear is relatively insensitive to low-frequency vibrations and it probably hears nothing at all below 20 - 30 c/s. The ear is, however, very sensitive to higher frequencies, having its maximum sensitivity around 2,000 c/s.

The sensitivity of the ear to changes in intensity of a sound as opposed to changes in frequency or pitch is, however, very different, and at high frequencies the intensity sensitivity is good. In other words, changes in the loudness of a sound will be better appreciated with low-frequency sounds than with those of higher frequency.

The practical implication of all this is that although the heart sounds arrive at the chest wall somewhat modified, the low-pitched sounds are many thousand times louder than the relatively high-pitched murmurs. A "true" recording of these sounds would therefore record all the vibrations present with an amplitude proportional to their actual intensity. This is referred to as a "linear" response, and a graphic recording under such circum-

stances over the frequency range 20 - 1,000 c/s. would show low-pitched sounds of many times the size of the higher pitched sounds. Such a graphic recording would be impracticable as the heart sound vibrations would be several feet in amplitude and the murmurs only a few millimetres if they were both recorded on the same scale. Passage through the stethoscope attenuates the low-frequency components still further, and, therefore, sounds and murmurs are approximated in their intensity by the time they reach the human ear. This process of attenuation of the low-frequency sounds is continued by the human ear as the low-frequency sounds are still a good deal louder than the high-frequency murmurs. The physical properties of the human ear are such that its sensitivity to the loudness of sounds falls off or decreases in a "logarithmic" manner from 1,000 c/s. right down to 20 c/s.

In this way the intensity of low-frequency sounds is attenuated considerably and they are heard by the ear to be only slightly louder than the higher frequency murmurs, hence the volume of the first does not drown the second. Different human ears differ in their precise capacity to attenuate the heart sounds just as they do to interpret them.

The characteristics of individual human hearing can be measured and an audiogram made. This is a graphic representation of the threshold of audibility at different frequencies and shows the intensity of a sound required to stimulate the ear at any frequency. Different listeners will have different audiograms, depending on natural auditory ability, inclinations and training.

(Fig. 1).

A sound may also be "masked", in that if it follows a high intensity sound, a low intensity sound may be inaudible. The human ear also has the faculty of "tiring" to a varying degree. That is to say, there may be a short refractory period following a loud sound during which the ear is insensitive, and this refractory or recovery period varies considerably between individuals.

We have, therefore, three stages of modification and attenuation of the heart sounds during the process of auscultation. Firstly, the vibration as represented at the chest wall. Secondly, the vibrations as presented to the ear by the stethoscope. Thirdly, the sounds as modified and interpreted by the human ear.

iv. The apparatus for recording heart sounds - the phonocardiograph

The remaining consideration is the apparatus to be used in converting the heart sounds into a graphic tracing which can be read and compared reasonably accurately with the sounds heard by the ear. The simplest and most direct method is to allow the sound vibrations to travel by air conduction from the chest wall along a narrow tube and to impinge on a membrane fastened across the end, in the same way as the sounds are conducted along the tube of a stethoscope and impinge on the tympanic membrane of the ear. To this membrane is fastened a small mirror which reflects a beam of light, and as the membrane vibrates with the sound waves the beam of light records the vibrations on a moving photographic plate. This was the method used, with various modifications and refinements by Frank, Wiggers and Dean, Orias and Braun-Menéndez, and many of the early workers. It was, however, rather awkward and cumbersome, and the sensitivity of the various membranes employed was not standard.

In more recent years electrical methods of sound recording have been introduced. With these techniques the sounds and murmurs are picked up by a microphone placed on the chest wall. The carbon microphone was used originally, but has since been replaced by the more highly sensitive crystal microphone which is in fact a highly modified form of electrical condenser designed to convert the minute pressure changes caused by sound vibrations into electric current. The current is taken through a series of amplifiers and filters and fed into a mirror galvanometer which transforms the electrical current back into vibrations - these being recorded by a light beam from the galvanometer mirror shining onto a moving roll of photographic paper.

The frequency response of the microphone, the amplifiers and the galvanometer must be as flat as possible over the range between 10 and 1,000 c/s. so that the sounds will be faithfully transmitted and recorded. Modifications and the necessary attenuation are carried out by a series of filters and amplifiers which can be put into the circuit at will. By means of these electrical methods of recording, amplifying and filtering, graphic tracings can be made which approximate to the three "physiological" stages of heart sounds encountered in auscultation and already discussed.

- (1) The vibrations as they exist on the surface of the chest wall, consisting of very large low-frequency waves only, recorded without further distortion or modification. This is known as the apical or linear phonocardiogram.
- (2) The vibrations as they are modified by the average stethoscope alone, that is to say, with the low frequencies moderately cut.

This is known as a stethoscopic phonocardiogram.

(3) The vibrations as they are modified by the stethoscope and the human ear. In other words, the graphic representation of what should be heard on auscultation. In these recordings the lower frequencies are even more severely cut, and the record is known as a logarithmic phonocardiogram.

In practice it is found that electrical recordings of the low frequency components, that is to say the heart sounds, are relatively easy and accurate. The phonocardiograph machine can be adjusted to amplify the lower frequencies in a way that the human ear cannot, although this will entail the masking of higher frequency murmurs. As the machine can respond to frequencies and intensities lower than those audible to the ear, the third heart sound and the auricular or fourth heart sound may be clearly demonstrated when they are inaudible to the human ear.

Conversely, the higher frequency murmurs can sometimes be appreciated by the ear, but the phonocardiograph may not be able to record them because of the inability of the galvanometer to respond to such frequencies at their relatively low intensity. Finer differences in timing can, however, always be shown on the phonocardiograph tracing, and measurement of the intervals between the various components of heart sounds and murmurs can be accurately made to a degree that is impossible for the ear. The phonocardiograph is also immune to some of the auditory defects of the ear such as the masking of fainter sounds by loud sounds and the "refractory" period which may occur and during which time the ear is insensitive.

Reference tracings

Apart from an accurate time marker it is essential to have a simultaneous physiological tracing which will provide "landmarks" in the cardiac cycle and also provide a reference by which events occurring in the phonocardiographic tracing can be accurately timed and correctly placed in the cardiac cycle.

The electrocardiogram may be used for this purpose and has the great advantage of being easy to record accurately and of providing clearcut easily-measured points of reference for auricular and ventricular systole. It is, however, singularly unhelpful in diastole.

The apical pulse - or 'linear phonocardiogram' as has been described - gives a record of the vibrations existing on the chest surface and records the onset of ventricular systole, the second and the third heart sounds. One of its disadvantages is that it cannot be recorded at the same time as stethoscopic or logarithmic phonocardiograms from the apex.

The jugular pulse gives several useful landmarks - apart from the "a", "c" and "v" waves, the beginning of the second sound and the position of the third sound may be seen. Its disadvantages are, firstly, the presence of a considerable time-lag due to the delay resulting from the slow venous pulse transmission from the heart to the jugular vein, and, secondly, it is sometimes extremely difficult to record satisfactorily, particularly in the absence of cervical venous distension, and in the presence of tachycardia.

A second phonocardiograph tracing is often of great help as a reference. For instance, a recording at the pulmonary area will usually show a clearcut second sound when this may be indistinct in the apical tracing, and it may be invaluable in differentiating between a split second sound, the opening snap of mitral stenosis, and the third sound at the apex.

Calibrated phonocardiography

Mannheimer and Carlgren (1940 and 1941) have described a technique for recording the heart sounds and murmurs and measuring their relative intensity and frequencies. In this way comparison may be made between different patients and the same patient on different occasions, and it is claimed that differentiation may be made between organic and functional murmurs by means of calibrated recordings.

Clearly there are some advantages in such a system of calibration and standardisation even if it only helps in the accurate comparison of recordings made at different times and in providing an objective series of recordings by which intensity of murmurs may be compared. By its means also, the subjective element of individual observer variation may be avoided.

Mannheimer and Carlgren employed a system using a calibrated microphone and high and low frequency pass filters in such a way that six channels each of a different frequency and capable of independent amplification could be recorded simultaneously. Their apparatus covered frequencies between 50 c/s. and 1,000 c/s., and calibration of intensity was obtained by feeding tones of known pitch and amplitude into the machine and comparing them to the sounds and murmurs recorded. An accurate quantitative measurement of the intensity of heart sounds and murmurs could be made in dynes/cm.² as well as the frequency of the fundamental tones and overtones which go to make them up.

Actually this method and the apparatus required are so extremely complicated that it seems doubtful whether the practical advantage to be gained from such a detailed study will ever be of interest to any but the occasional research worker. Without some

such system, however, no accurate comparison can be made between one recording and the next, and this limitation was occasionally regretted during the course of the studies reported here.

DESCRIPTION OF THE INSTRUMENTS USED

Three different electrocardiograph machines have been used during the course of these studies. The first two were discarded when the third and most efficient became available.

(i) Initially the only apparatus available was a British amplifier with two mirror galvanometers and a motor-driven camera which was directly operated from the amplifier. One galvanometer in this system provided a good tracing of the wave of excitation through the lead. A cathode ray oscilloscope was used. No technical details of the circuit and performance of this machine were obtainable, and although valuable experience was gained while using it for many recordings, there were a number of serious drawbacks associated with its use. One of the main difficulties revolved in its construction in the fact that it was difficult to calibrate and fine measurement of the events occurring in the cardiac cycle could not, therefore, always be made.

(ii) Trials were also made with a string galvanometer and amplifier designed for use with the standard Cambridge electrocardiograph instrument, but none of these recordings was used in this series as a better multi-channel instrument became available at about the same time.

OBSERVATIONS

DESCRIPTION OF THE APPARATUS USED

Three different phonocardiograph machines have been used during the course of these studies. The first two were discarded when the third and most efficient became available.

- (i) Initially the only apparatus available was a Boulitte amplifier with two mirror galvanometers and a motor-driven camera which was entirely separate from the amplifier. One galvanometer in this system recorded a sound tracing and the other an electrocardiograph lead. A Rothermel crystal microphone was used. No technical details of the circuit and performance of this machine were obtainable, and although valuable experience was gained while using it and many recordings made, there were a number of serious drawbacks associated with its use. One of the main difficulties resulted from inaccuracies in the time marker which was difficult to calibrate and fine measurement of the events occurring in the cardiac cycle could not, therefore, always be made.
- (ii) Trials were also made with a string galvanometer and amplifier designed for use with the standard Cambridge electrocardiograph instrument, but none of these recordings was used in this series as a better multi-channel instrument became available at about the same time.

(iii) The vast majority of the records, including all those used for accurate timing measurements, have been made with a specially modified Elmquist Triplex electrocardiograph with three additional amplifiers for recording phonocardiographs. The electrocardiograph itself was a battery-operated multi-channel instrument, employing three separate three-stage capacity coupled amplifiers, the output of each amplifier being fed to the coil of an oil-damped mirror galvanometer. A fourth galvanometer was included to record the difference in potential between the first and second galvanometer and was therefore only used normally in the recording of the three limb leads simultaneously. However, the machine used in this work had been specially modified so that all four galvanometers could be used quite independently for recording either electrocardiograph leads or they could be connected directly to external amplifiers and heart sound tracings recorded.

Three different phonocardiograph amplifiers have been used, each in conjunction with a rubber-shrouded Rochel Salt crystal microphone which had a frequency response that was linear up to about 10,000 cycles per second.

The first (Type I) consisted of a four-stage high gain amplifier with a five-bank resistance-capacity filter incorporated between the second and third amplifying stages so that suitable frequency coverage could be selected at will. A volume control in the last stage of the amplifier could be adjusted to obtain a reasonable phonocardiographic amplitude. The output from this heard sound amplifier was connected directly to a galvanometer in the electrocardiograph unit.

The second amplifier (Type II) was of similar principle to the first, employing the same type crystal microphone, with a three-bank resistance-capacity filter incorporated between the first and second stage. The three filter positions were designed to correspond to the linear, logarithmic and stethoscopic frequency responses. The amplifier itself had an overall higher gain, and the valve types employed were such that they had a very much lower electronic noise level, when compared with the first and original amplifier.

The third amplifier (Type III) also employed the same type of crystal microphone which was connected to the input of a four-stage low noise amplifier; a three-position inductance capacity filter being connected between the second and third stage of the amplifier. By means of this filter the frequency components of heart sounds could be selected at will. A volume control connected between the third and fourth stage of the amplifier could be adjusted to amplify the heart sounds to a suitable level for recording. The output from the phonocardiograph amplifier could be connected directly to one of the galvanometers in the Elmquist electrocardiograph unit.

The mirror galvanometers employed had a frequency response which was linear to 450 c/s., and a beam of light reflected by the mirror focused onto photographic paper, the paper being driven through the camera by a clockwork motor at either 4 cm. or 10 cm. per second, and time-marking lines were automatically exposed on the paper indicating 1/10th and 1/50th second intervals. The same motor drove the camera and time-marker, and there was a switch enabling the motor and time-marker to be driven at either a low or a high speed. For most work, low speed was found to be perfectly satisfactory.

In some of the records the time-marker registers only $1/10$ th second and in a few only $1/5$ th, but measurements can still be made up to $1/50$ th second with the aid of a transparent celluloid millimetre rule.

The accuracy of the time-marker was checked from time to time against the frequency of the A.C. mains current after determining the exact A.C. frequency for the particular hour of the day. (Fig. ii).

A special phlebogram attachment was also used for recording the jugular pulse as a reference tracing. This could be connected up to any one of the galvanometers and consisted of a light metal spring pulse receptor attached to a piezo-electric crystal and mounted on a stand. The crystal and pulse receptor were adjusted by means of a fine screw adjustment so that the tip of the spring rested on the pulsating vessel. This attachment was found to be of limited value and would only record venous pulsation of fairly considerable amplitude, hence in the majority of patients a phlebogram could not be obtained.

As a routine the following synchronous recordings were made: either a jugular venous pulse wave, an electrocardiograph lead and two phonocardiographs from different areas of the praecordium, or an electrocardiogram and three different phonocardiograms usually from the aortic area, the 4th left space just lateral to the sternal edge and the apex. Different variations of these recordings were used from time to time, depending on the object of the recording.

FIG. 1

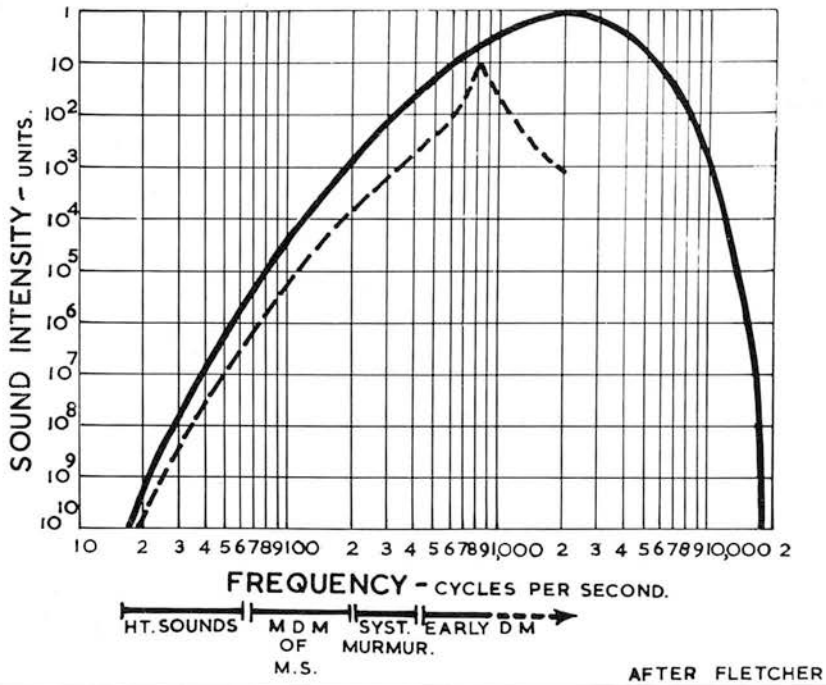


Fig. i. A NORMAL AUDIOGRAM

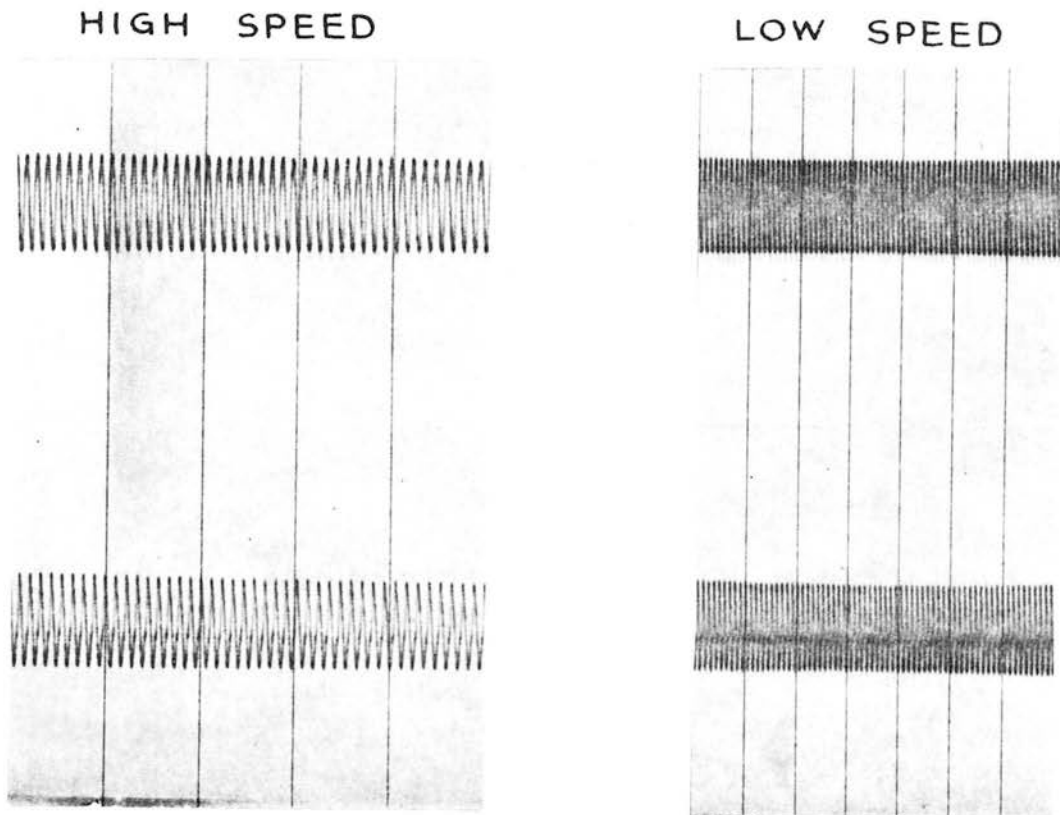
The continuous black line represents a curve, showing the threshold of audibility at different frequencies. (The dotted line should be ignored). Arbitrary units of sound intensity have been chosen, one unit being the lowest intensity which can be heard at 2,000 cycles per second, this being the frequency at which the ear is most sensitive. The approximate frequencies of heart sounds, mid-diastolic murmur (M.D.M.) of mitral stenosis, systolic murmur and aortic early diastolic murmur are also shown. (Reproduced through the courtesy of Dr. Aubrey Leatham from the Postgraduate Medical Journal, 1949, 25, 568)

FIG. II

Fig. ii. TIME MARKER CHECK.

By deliberate inducement of A/C mains interference in the galvanometer the paper speed and time marker are checked for accuracy with the motor running at both high and low speeds.

TIME MARKER registers $1/5$ th second in these records
A/C mains vibrations at 50 cycles per second.



Recording and processing

Ilford B.P. I recording paper 100 m.m. guage was used with the Elmquist camera, and special Boullitte recording paper 60 m.m. guage for the Boullitte camera. The records were processed using Ilford I.D.13 developer at a temperature of 60° F.

Frequency response of the amplifier and galvanometer

The high frequency response of the three amplifiers and the crystal microphone was well over 1,000 cycles / second and, therefore, all sounds up to this level, which includes all the heart sounds and murmurs, were faithfully conducted without distortion and only modified or amplified according to the filter employed and the volume setting of the amplifier. Unfortunately, however, the frequency response of the actual galvanometer was a limiting factor. The galvanometers used had a response which was linear from zero up to 450 c/s. and did not begin to fall off seriously until about 700 c/s. In fact, this allows for a fairly accurate recording of the majority of sounds and murmurs, and only some of the relatively high frequency sounds and overtones would fail to be recorded.

Fig. iii, iv, and v show the frequency response of the three amplifiers used with the Elmquist instrument. Fig.iii gives the response of the amplifier Type I but does not include the galvanometer response; this would have the effect of cutting off the amplitude above 700 cycles as shown in the response of amplifier Type II in Fig. iv which includes the galvanometer response. Fig. v shows the response of amplifier Type III, and the dotted line indicates the overall response of the amplifier plus the galvanometer above 450 c/s. The response curves of

FIG. III

FREQUENCY RESPONSE OF HEART SOUND AMP. TYPE 1.

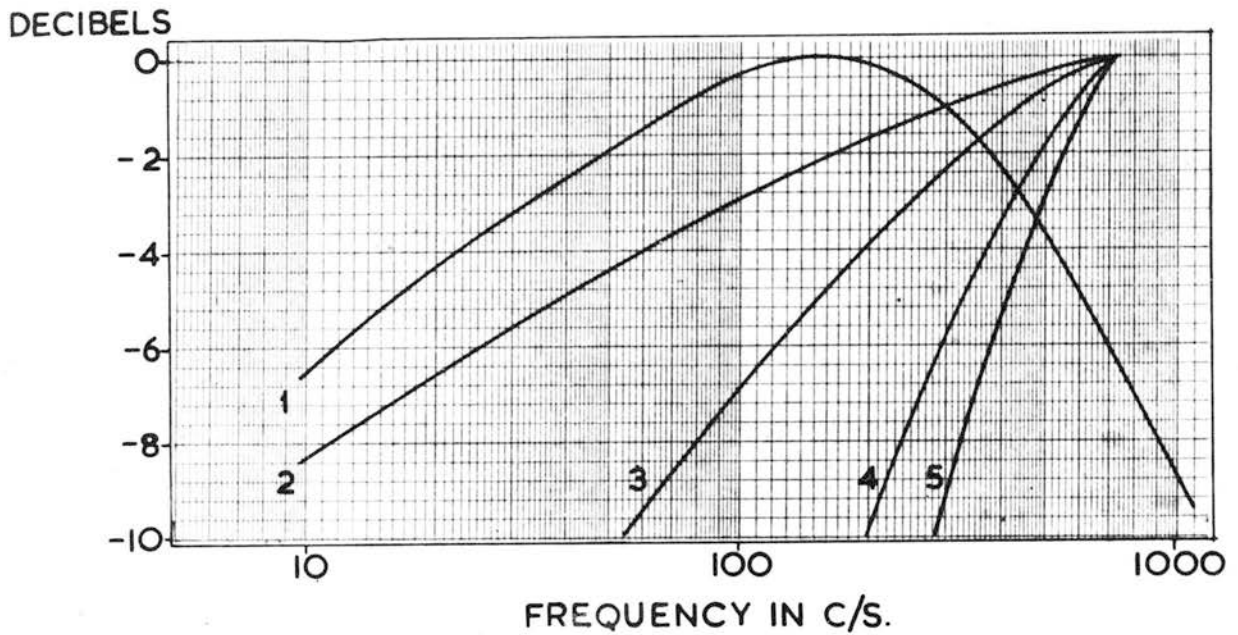
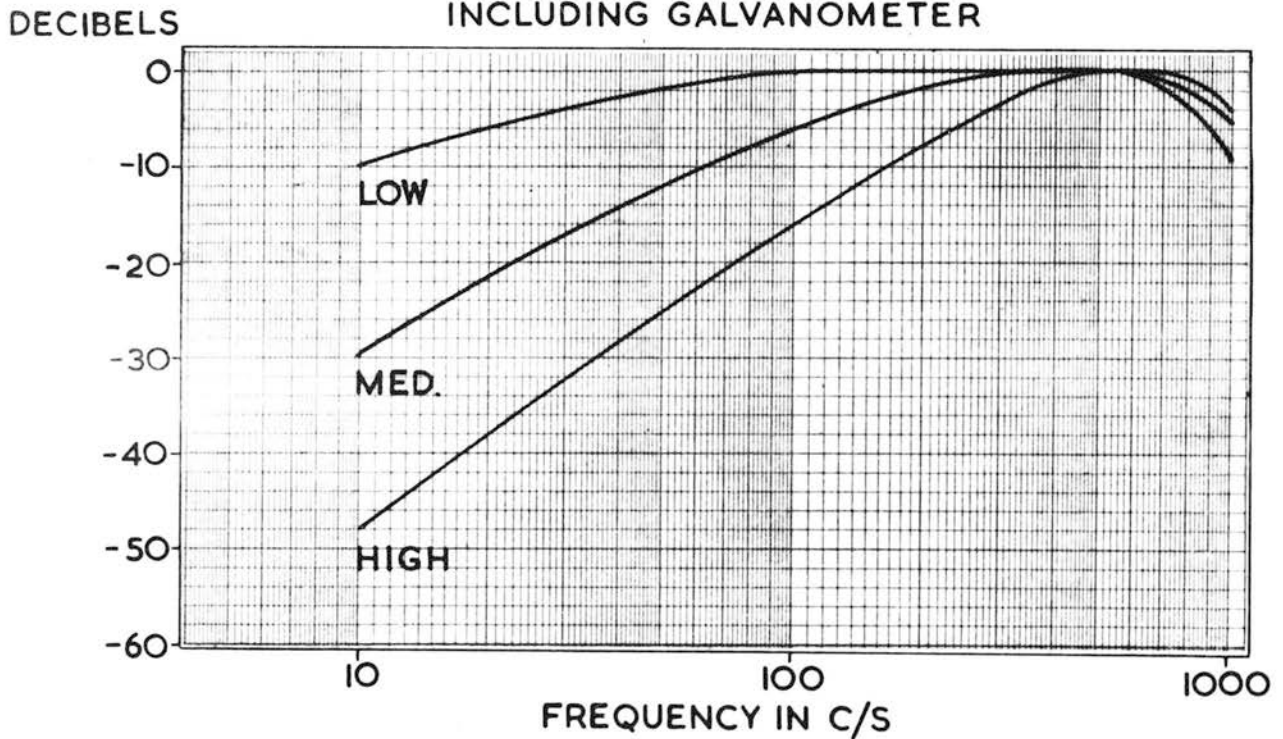
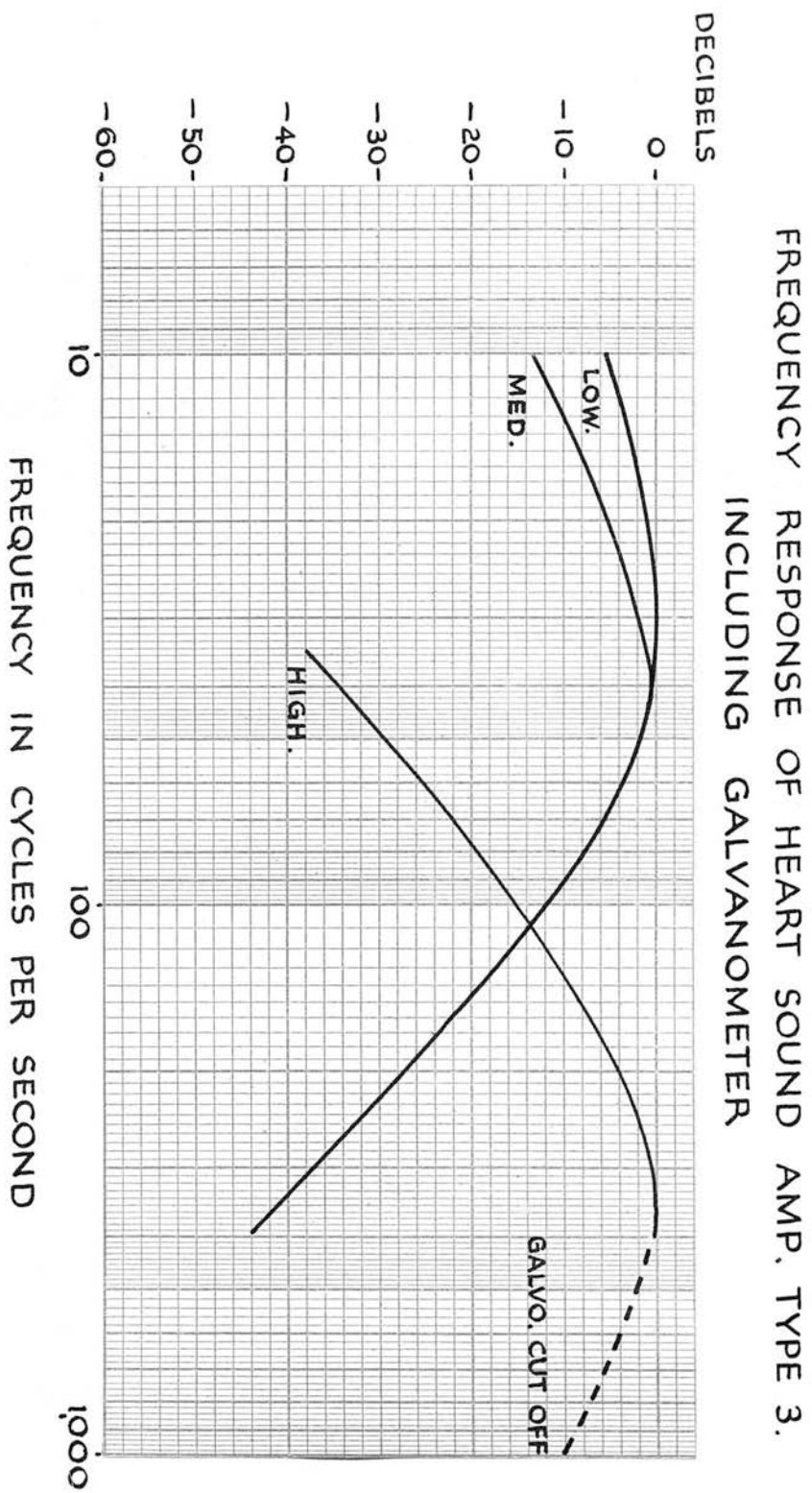


FIG. IV

FREQUENCY RESPONSE OF HEART SOUND AMP. TYPE 2.
INCLUDING GALVANOMETER



the various high and low frequency bands that could be obtained by selecting the different filter positions controlled by a switch on each amplifier can also be seen.

TECHNIQUE OF RECORDING HEARD SOUNDS AND MURMURS

As with electrocardiography it is essential to obtain the full confidence and co-operation of the patient in order to obtain a really good phonocardiograph tracing. The room in which recordings are made must be quiet and the patient warm, comfortable, relaxed, and lying supine. Under such circumstances good records may be taken while the patient is breathing quietly and normally, but records may also be taken with the breath held in any phase of respiration. Under certain circumstances the particular phase of respiration may influence the character of the heart sounds, as for instance when recording a split second sound, which may be present in one phase of respiration and not in another. In very ill or dyspnoeic patients breath sounds and other extraneous noises arising from movement of the patient may be unavoidable, and a satisfactory "base-line" to the tracing may not be obtainable. The microphones used are encased in rubber to deaden outside sounds, and the soft rubber rim helps to make an airtight junction with the skin, this being essential for a good recording. In some very thin and bony patients a completely airtight junction was obtained by interposing a ring of soft plasticine between the rubber microphone casing and the skin. Microphones are best held on by a broad rubber band encircling the entire chest. Bier's bandage doubled longitudinally was found to be most satisfactory for this purpose.

The ends of this can be held with a pair of artery forceps and the position of the microphone easily adjusted.

Usually sound tracings were taken from at least two or three different areas of the chest wall, one of which was used as a reference tracing to locate the position of the first and second heart sounds in the cardiac cycle. Initially, jugular pulse tracings were always attempted, but a good deal of difficulty was encountered in recording the venous pulse, and a lead I or II electrocardiograph and low frequency phonocardiograph were ultimately often used as routine reference tracings.

When actually making recordings the heart sounds could be monitored through a stethoscope attachment connected directly to the amplifier being used, but, with practice, visual control was also found to be helpful. While both listening to the heart sounds and watching the deflection of the galvanometer light spot the required frequency band was selected and then the amplifier sensitivity was slowly increased until the largest heart sound deflection consistent with the appearance of a steady base-line at a silent phase of the cardiac cycle could be seen. Records were also taken with a slightly greater and a slightly lesser amplitude, and one or more of the three was usually found to be satisfactory. In cases in which the cardiac murmurs were such that no silent period existed the procedure had to be modified and a number of records with varying frequency settings and at different amplitudes usually had to be taken and the optimal setting discovered by a process of trial and error.

THE NORMAL HEART SOUNDS

Two heart sounds are normally audible in health, but under certain circumstances a third and a fourth may also be heard. Of these latter sounds, the third heart sound, or protodiastolic sound, is sometimes heard in health, and the fourth heart sound, audible auricular sound or presystolic sound, is rarely if ever heard in health.

THE FIRST AND SECOND HEART SOUNDS

Mechanism

It is generally agreed that closure of the A-V valves is the main factor in the production of the first sound, which occurs at the moment of ventricular contraction (Wiggers and Dean, 1917). The part played in addition by audible muscular contraction of the auricle and ventricle and by the flow of blood has been much debated.

In 1851, Halford, in a letter to the Lancet, described an experiment indicating that the heart sounds were largely valvular in origin. He auscultated a dog's heart after exposure, then clamped the superior vena cava, the pulmonary vein and the inferior vena cava. Although the heart continued to contract, the sound became inaudible, reappearing when blood was allowed to flow again. Rouanet (1832), in his M.D. thesis, and Hope (1841) are both quoted by Dock (1945) as having made similar observations and having come to the same conclusion regarding the valvular origin of the first heart sound.

That muscle contraction does itself produce an audible sound has been known to clinicians for many years. In 1858, Clarke, Ellis and Shaw gave a bizarre description of how they listened to the sound produced by the contracting auricle for ninety minutes after the death of an executed criminal. Audible auricular contractions in complete heart block were also described by Robinson in 1908 and Wardrop Griffith in 1911.

Eckstein, in 1937, found experimentally that when skeletal or cardiac muscle contracts a sound is produced. Smith, Gilson and Kountz (1941) and Smith (1944), unlike Halford, were able to record the first heart sound persisting when the heart valves were prevented from moving and all blood flow through the heart stopped. Under these experimental conditions the sounds were changed in character, and the authors believed that they were produced by muscle contraction alone and were quite independent of valve movement or blood flow. They concluded that at least a part of the first sound was due to audible muscle contraction. Dock (1933 and 1945) and Lewis and Dock (1938), on the other hand, were convinced that both the first and second heart sounds are produced almost entirely by valve movement, and their experimental findings were at variance with those of ~~Smith et al.~~ Smith et al. They also point out that ectopic beats are silent if they occur when the A-V valves are closed and there can therefore be no valve movement. They also discuss the variation in intensity of the first sound which accompanies heart block and which is probably due to varying position of the A-V valve at the moment of systole.

From a practical point of view it does seem most likely that valve closure is the main factor in producing an audible first heart sound, and the muscular element, although present, is of less importance. The frequency range of sounds arising from contracting muscle must be at the lower range of audibility and anyway would probably be masked by the much higher frequency sounds made by valve closure.

There is general agreement that the second heart sound is due to sudden closure of the semilunar valves as the intraventricular pressure falls below the pressure in the aorta and the pulmonary artery (Billing, 1831; Rouanet, 1832; Schutz, 1933).

All the various frequency ranges can, however, be shown on the phonocardiograph tracing, and careful analysis of both first and second heart sounds have been made by ^{Schutz (1933),} Orias, Braun-Menéndez (1935), Rappaport and Sprague (1942), Mannheimer (1940 and 1941) and Luisada and Mendoza (1949).

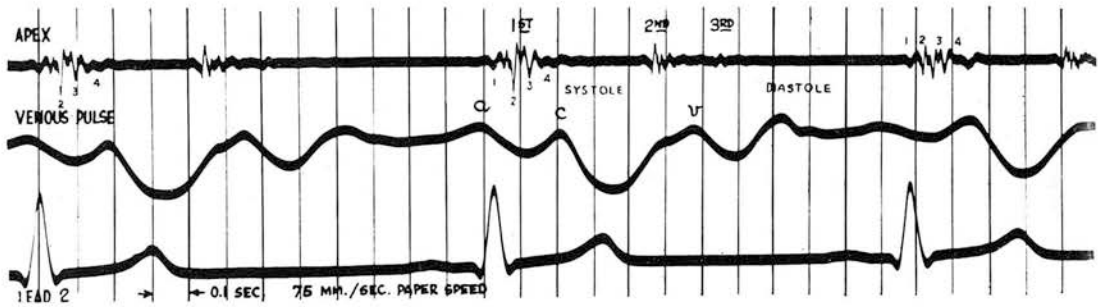
The analysis of Rappaport and Sprague is the most complete, and the graphic complexes are broken down to their valvular, muscular and vascular components. This is well illustrated by the accompanying table and illustration taken from their original article. (Table I and Fig.vi).

TABLE I

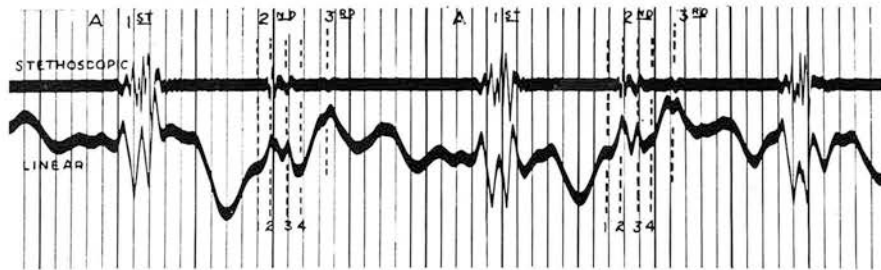
* ANALYSIS OF HEART SOUNDS
<p><u>First Sound</u></p> <ol style="list-style-type: none"> 1. An initial low-frequency auricular vibration which is almost always inaudible, but may be seen on the phonocardiogram. 2. The closure of the mitral and tricuspid valves. 3. The opening of the aortic and pulmonary valves. 4. The noise made by the acceleration of blood in the great vessels during the ventricular systolic ejection phase. <p>Of these, probably only component 2 is audible to the human ear.</p>
<p><u>Second sound</u></p> <ol style="list-style-type: none"> 1. Ventricular relaxation 2. Closure of aortic and pulmonary valves. 3. Arterial wall and blood column vibrations. 4. Opening of mitral and tricuspid valves. <p>Of these, probably only component 2 is audible to the human ear.</p>

+ after Rappaport and Sprague, 1942.

FIG. VI



—Simultaneous stethoscopic phonocardiogram, venous pulse, and electrocardiogram, to illustrate the relationship of the four components of the first heart sound.



—Simultaneous stethoscopic (upper) and linear (lower) phonocardiogram at the apex of a normal person. The four components of the second heart sound are present in the stethoscopic phonocardiogram, and their phase relationship to the apex beat is shown. A slight auricular vibration and the first and third heart sounds are distinctly registered.

Fig. vi. ANALYSIS OF THE HEART SOUNDS ACCORDING TO
RAPPAPORT & SPRAGUE
(Reproduced from the American Heart Journal,
1942, 23, 604)

Wiggers (1949), although agreeing that these various components of the heart sounds are indeed present and may possibly be identified on a phonocardiograph tracing, has also pointed out that it is most improbable that the vibrations of different structure maintain their separate identity to such an extent that they may be easily identified one from the other in a sound tracing. He concludes that they will almost certainly be heard as one sound by the human ear.

There seems, therefore, to be little practical advantage in this careful separation of the heart sounds into various components, but such a concept is of interest and of value in the study of the special phenomena referred to below. These are (1) the splitting of heart sounds which involves only the separation of one valvular component from another; (2) the separation of the auricular systolic component from the main body of the first sound as may occur under certain circumstances; and (3) the accentuation of the fourth part of the second sound (Rappaport and Sprague) in mitral stenosis to produce an audible "opening snap" of the mitral valve.

Intensity of the heart sounds

Since the original pioneer work of Dean (1916) in his demonstration of the mode of opening and closing of the A-V valve in the perfused cat's heart, variations in intensity of the first heart sound have been discussed at length by Dock (1933), Margolies and Wolferth (1930), Cossio *et al.* (1947), Levine (1948), and others.

As it is now generally agreed that the first heart sound is largely due to the sudden tensing and closure of previously slack A-V valves, one of the factors determining the loudness of the sound produced will be the position and state of tension of the valve at the moment of ventricular systole and the rate of rise of intra-ventricular pressure.

This theory is further amplified and illustrated by Cossio et al. who, following the work of Dock, analysed the intensity of the first sound in normal sinus beats and in both auricular and ventricular extra-systole. Cossio explained why the extra-systolic first sound was usually louder than the normal first sound. This he suggested depended on the position of the A-V valve at the onset of systole. In the case of a normal sinus beat the valves are optimum position for closure at the onset of ventricular systole, but in the case of an auricular extra-systole the valves are wide open at the onset of ventricular systole and not only close with a louder noise but also take longer to close as they have further to travel. This delay in closure corresponds with a prolonged ~~QRS~~ time interval between the onset of the QRS complex of the electrocardiogram and the loud extra-systolic first sound.

Usually the vigour of ventricular contraction and the rate and degree of the rise in intraventricular pressure will also influence the loudness of the first sound. Hence, in the normal heart, exercise will produce an increase in loudness of the first sound. Wiggers (1949) has, however, pointed out that this is not invariably the case as increased systolic output may occasionally be associated with a decrease of the amplitude of heart sounds.

Frequency of the first and second heart sounds

These are very difficult to measure accurately as the heart sounds consist of a mixture of TONES and NOISES, many of the latter being irregular and therefore non-periodic and impossible to measure. The TONES have a fixed, periodic and measurable frequency and they are present in a wide range and can be divided into fundamental tones and overtones. The fundamental tones make up the basis of the heart sounds, and in the case of the first and second sounds they lie mostly between 30 and 150 cycles per second. There are, however, overtones up to 500 c/s. but they are far less in amplitude than the fundamental tones and have been picturesquely described as resembling the high-pitched echoes from a shot fired in a cave. It is, however, these overtones of higher frequency which make up the audible character of the first and second heart sounds. The properties of hearing being such that sounds of 500 c/s. are very much better heard, even if their amplitude is much less, than the lower tones of 50 c/s. and thereabouts. (Einthoven, 1907; Williams and Dodge, 1926; Orias and Braun-Menéndez, 1939; Wiggers, 1949; Mannheimer, 1940 and 1941).

Splitting of the normal heart sounds

It is well recognised that the normal heart sounds are frequently split. Potain, in 1866, observed splitting of the second sound in healthy individuals. McKee (1938), recording the heart sounds of 100 normal schoolchildren, demonstrated splitting of the first heart sound in 23 per cent., splitting of the second in 38 per cent., and splitting of both in 14 per cent.

Leatham and Towers (1951) demonstrated simple splitting of the second sound in every one of 40 unselected healthy schoolchildren.

Splitting in health has also been recorded by Orias and Braun-Menéndez (1939), Boyer, Eckstein and Wiggers (1940), Mannheimer (1940), and others. According to Sansom (1881) and Cossio and Fongi (1936), splitting of the first sound may be due to separation of the auricular part from the remainder of the sound. Lian (1933-4) thought it might be due to a separation of the muscular and valvular parts of the first sound, but in the case of the second sound he considered splitting to be due to asynchronous closure of the pulmonary and aortic valves. Asynchronous valve closure seems to be the most likely explanation, and in 1925 Katz showed that asynchronous contraction of the ventricle may occur in normal animals.

In 1935 Wolferth and Margolies, by means of kymography, measured the movement of aortic and pulmonary systolic pulsation in normals and found that subjects with a split first sound showed asynchronous aortic and pulmonary systole but that either element might be the first. Following this, Leatham (1954) produced convincing evidence to prove that simple splitting of both first and second heart sounds is due to asynchronous closure of the semilunar valves in the case of the second sound and of the mitral and tricuspid valves in the case of the first.

According to Leatham, in the case of physiological splitting of the first sound the time interval between the first and second components of the split usually measure 0.02 to 0.03 second. The first part of the normally split sound is due to mitral closure and the second to tricuspid. This was clearly

demonstrated in ten normal subjects in the following way. Simultaneous phonocardiograms were recorded from the mitral and tricuspid areas, and the relationship of the first and second parts of the split first sound to left ventricular systole was established by timing with a carotid pulse tracing. In this way it was found that the first part of the split, which was loudest at the mitral area, coincided with the rise of pressure in the carotid artery and thus with left ventricular systole, while the softer second part of the split at the mitral area occurred after ventricular systole and must therefore be due to tricuspid closure.

In the case of physiological splitting of the second sound the time interval between the first and second part of the split varies considerably with respiration. Usually this interval is about 0.02 to 0.03 second during expiration, but may increase to as much as 0.1 second during deep inspiration.

Splitting of the second sound was shown by Leatham to be due to asynchronous closure of the pulmonary and aortic valves and the first element of the split proved in the following way to be due normally to aortic closure. Phonocardiograms from the pulmonary area and the aortic area were timed with a carotid pulse tracing. The first element was found to coincide with the incisura of the carotid pulse wave, to be loudest at the aortic area and to be due, therefore, to aortic closure. In this way the two components seemed clearly identified. Wolferth and Margolies, however, using a similar method, maintain that although the aortic element usually preceded the pulmonary, this relationship is not invariable (1935).

The two components of the split sound are of roughly the same high frequency. Split sounds commence at the same place as the normal undivided sound, and with the exception of the split second sound in deep inspiration the total duration of the sound does not considerably exceed that of the normal undivided sound (Frost, 1949).

Splitting of the sound is so common in health that its possible value as a sign of disease is bound to be very limited. It has also been found that the audible impression of a split sound has not always been confirmed by the presence of a clear splitting of the phonocardiograph tracing. Conversely, an apparent splitting of the phonocardiograph tracing has not always been observed clinically.

Bearing in mind the small time intervals involved, the auditory limitations of the human ear, and the possible masking or fatiguing effect on the ear that a loud first element of the split sound might have on a fainter second part, it is perhaps not surprising that all split sounds are not clearly heard as such by all observers.

Duration of normal heart sounds

The duration of normal heart sounds have been measured by various workers and some of their results are set out in Table 2. It will be seen that there is considerable variation in these results. This is understandable in view of the different techniques employed and in view of the absence of any adequate system of calibration in any of the methods other than that of Mannheimer. The total duration of vibrations will depend partly on the amplitude and

frequency of the tracing. Initial and terminal vibrations are of smaller magnitude than the main vibrations, and yet they are all part of the heart sound. If the phonocardiograph amplifier is set at maximum, all the vibrations will become visible and also in all probability the base-line between the heart sounds will be irregular. If, however, the amplification is decreased, initial and terminal heart sounds vibrations will not be seen, and the duration of the sound will appear significantly shorter.

TABLE 2

NORMAL VALUES FOR FIRST AND SECOND HEART SOUND DURATION			
Authority	Duration in seconds		
	First sound	Second sound	
Boyer et al.(1942) 50 medical students	0.06 - 0.11	0.04 - 0.06 ⁺	
Arenberg (1942) 200 healthy subjects & hospital patients	0.06 - 0.11	0.04 - 0.06	
Luisada & Mendoza (1949) 185 normal subjects	0.07 - 0.149	0.05 - 0.12	
Wiggers (1949) 151 young male students	0.06 - 0.16	0.04 - 0.11	
Rappaport & Sprague (1942) 33 normal subjects	0.105 - 0.165 0.08 - 0.13	0.085 - 0.145 0.08 - 0.110	Stethoscopic Logarithmic
Mannheimer (1940) 135 healthy children 0 - 14 years	0.05 - 0.15	0.03 - 0.12	

+ split second sound may occasionally have a total duration as long as 0.11

Relation of the normal first and second heart sounds
to the reference tracing

It is generally agreed that the first heart sound tracing begins with low frequency vibration and it is frequently stated that if the phonocardiograph is registered simultaneously with the electrocardiograph these vibrations never appear before the Q-wave.

Lewis (1912) stated that the first sound commences 0.002 to 0.26 second after the beginning of R. Wiggers and Dean (1916) found that the first sound commenced 0.01 to 0.02 second after the beginning of the ascending limb of R, and the main vibrations of the first sound do not appear until after the peak of R. Certainly any vibrations before the Q wave are not ventricular in origin. Frequently in fact, however, as Rappaport and Sprague have pointed out, a few small low frequency vibrations are seen in the PQ interval; these are almost certainly due to auricular contraction and will be discussed later. They are never seen when the rhythm is auricular fibrillation (Frost,), and in health they are not audible. The second heart sound is, as has been seen, shorter in duration than the first and occurs at a variable position just before or after the summit of the T-wave. All workers are agreed that it has no exact relationship to the T-wave and, therefore, the E.C.G. is of little value in measuring heart-sound phenomena occurring in relation to the second sound or the early and mid-diastolic period.

When compared with the jugular phlebogram the first heart sound coincides with the ascending limb of the peak of the "C" wave but rarely, if ever, extends beyond the peak of "C" (Rappaport and Sprague, 1942). The second sound coincides with the

beginning of the ascending limb of the "V" wave, which is often notched at the point of semilunar valve closure, and no vibrations of the second sound should extend beyond the peak of the "V" wave, which is coincident with the opening of the mitral and tricuspid valve. This latter phenomenon may be represented by a small final vibration on the normal P.C.G. but only becomes sufficiently loud as to be audible in mitral stenosis. When the second sound is split the latter component of the split occurs at the time of the ascending limb of the "V" wave. (Figs. vi and vii).

THE PHYSIOLOGICAL THIRD HEART SOUND

It is generally admitted that a faint low-pitched third heart sound may be heard following shortly after the second sound in a proportion of healthy young adults. This sound is usually heard most easily at or internal to the apex with the patient recumbent or on the left side. It may vary in loudness with the phases of respiration and is best heard with a bell-shaped chest-piece attached to the stethoscope. It has been referred to in the literature not only as the third heart sound, but also as the protodiastolic and the rapid ventricular filling sound.

Although extra sounds occurring in the cardiac cycle and giving rise to a triple or gallop rhythm were well recognised up to the end of the last century, these were always noted in

association with

FIG. VII

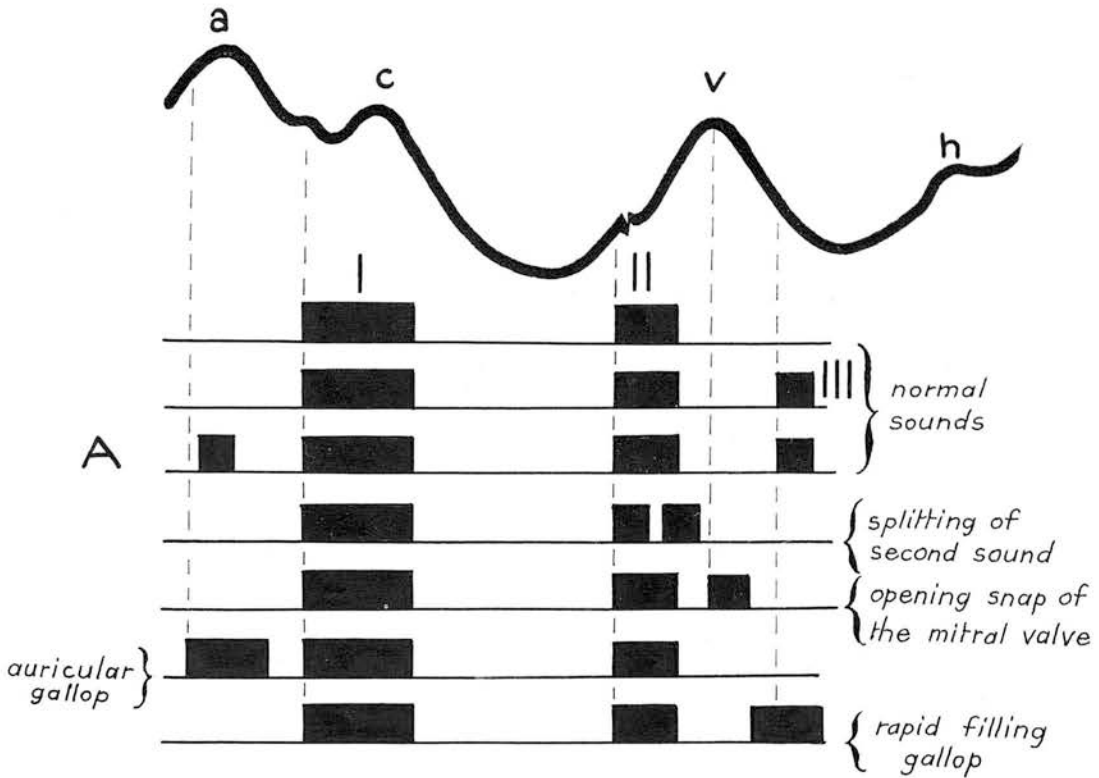


Fig. vii DIAGRAM OF THE HEART SOUNDS AND
THE VENOUS PULSE

The second component of the duplicated second sound coincides with the ascending line of the *v* wave. The opening snap of the mitral valve occurs simultaneously with the top of the *v* wave. The third sound appears in the descending line of the *v* wave.

I = the first sound, II = the second sound,
III the third sound. A = the auricular sound.

disease. The occurrence of a physiological triple rhythm was not noted until 1893 when, during the course of a discussion on triple rhythm in *La Semaine Medicale*, Barie described such a phenomenon due to a third heart sound occurring in diastole. He maintained that such a sound could often be heard in normal hearts but he does not differentiate in the text between the physiological third and splitting of the second sound, and he does not comment in any detail on the subject.

Obrastsov, in 1905, mentions the existence of a faint diastolic sound present in normal subjects when discussing pathological triple rhythm, but he also fails to give any details of the significance or actual nature of the sound. In 1907, however, Gibson in England, Hirschfelder in America, and Einthoven on the Continent, all working independently, noted the existence of a third heart sound in healthy young adults.

Gibson, when recording jugular phlebograms on a young patient with bradycardia, noted a positive venous pulse wave, which he called the "B" wave, following the "V" wave of each cycle and preceding the "A" wave of the subsequent cycle. Coincident with the start of the "B" wave Gibson heard a faint third heart sound, but he did not attempt to record it. This sound he thought was due either to rapid expansion of the ventricular wall or to closure of the semi-lunar valves, and he approached Einthoven, who had already heard the sound and was now able to record it on his string galvanometer and show its relationship to the "V" and B waves of the phlebogram. At the same time, Hirschfelder noted the extra venous pulse wave which he called the "H" wave and heard the third heart sound occurring at about the same time.

In 1908 and 1909 Thayer further elucidated the problem of the physiological third heart sound. He claimed that in 65 % of healthy people under 40-years of age he could demonstrate graphically a third heart sound following shortly after the second. He distinguished this third sound from the opening snap of mitral stenosis and from physiological splitting of the second sound, and he recorded apical cardiograms (that is to say, linear phonocardiograms) and phlebograms from 231 healthy people.

In his records the third sound coincided with either the descending limb of the jugular "V" wave, ~~at~~ the foot of "V", or ~~on~~ the ascending limb of the "H" or "B" wave following it. His mechanical recording methods were rather crude and he himself admitted that accurate timing was difficult. Thayer also noted that in his recordings the physiological third sound appeared at the same place in the cardiac cycle as the extra pathological gallop sound he had recorded in cases of ischaemic heart disease, aortic incompetence and adherent pericarditis.

He thought that the physiological third sound was probably due to sudden tensing of the A-V valve as a result of rapid blood flow from auricle to ventricle in early diastole, but he considered the pathological third sound to be due to a sudden rush of an abnormally large quantity of blood into the dilated ventricle of lowered muscle tonus.

Bridgeman, in 1915, reported hearing a third heart sound in 13 of 16 normal boys and was able to demonstrate the sound graphically in all 16. He believed that rapid ventricular filling was responsible for the production of this sound. Battaerd, in the same year, published a full account of the history of phonocardiography and showed several tracings of the physiological third sound.

Ohm (1913) was able to show that in all cases the physiological third heart sound, when demonstrable, coincided exactly with a deep negative venous pulse wave and, therefore, with the phase of rapid ventricular filling. He maintained that as a result of the sudden rush of blood from auricle to ventricle vibrations were set up in the ventricular wall which gave rise to a low-pitched sound. Orias and Braun-Menendez (1939) and Smith (1944) also produced convincing evidence of the muscular origin of the physiological third heart sound. Smith prevented the A-v valves from moving by splitting them with a metal plate in an experimental animal, but was still able to hear the third sound although its character was slightly altered. He also maintained that cutting off the blood supply to the heart did not abolish the third sound as long as the heart was contracting vigorously, and he concluded that the third sound was due entirely to muscle vibration and was independent of actual blood flow.

Lewis and Dock (1938), Dock (1945), and Brady and Taubman (1950) have all produced arguments and evidence in favour of the physiological third sound being due to sudden movement of the A-v valves.

Boyer, Eckstein and Wiggers suggested in 1940 that the sound might originate from the ventricle relaxing in early systole and striking the thoracic wall, but, two years later, Boyer himself disproved this by a complicated series of animal experiments. In these he isolated a dog's heart in a "cardiometer" in such a manner that all question of contact or thoracic vibration was excluded, but the third heart sound was still audible and its intensity

increased by intravenous infusion of saline, thereby increasing the velocity of blood flowing into the ventricle during the rapid inflow phase.

In humans Sloan and Wishart (1953) showed that the rate of filling of the heart could influence the physiological third heart sound. They decreased the venous return in healthy subjects by applying sphygmometer cuffs to the arms and legs, and the third heart sound either disappeared or became much fainter. This phenomenon was constant in 16 healthy subjects and the changes in intensity of the third sound were graphically recorded.

This theory, that the third heart sound in health arises as a result of the rush of blood into the ventricle and the sudden distension of ventricular walls which sets up a short series of faint low-frequency vibrations, seems to be the most likely. It is also subscribed to by many other authorities, including Wiggers (1949), Lian (1933), Wolferth and Margolies (1933), Mannheim (1940), Rappaport and Sprague (1943), Carlgren (1946), Luisada et al. (1948), and Frost (1949).

Characteristics of the physiological third heart sound

The physiological third heart sound itself when recorded consists of one to three simple low-frequency vibrations. Its actual measured frequency range will almost always lie in the region between 25 to 50 cycles per second. Occasionally frequencies of up to 100 c/s. may be recorded. However, there is an almost complete absence of relatively high-frequency overtones of

lesser amplitude which have been shown to give the first and second sounds their audible characteristics. (Rappaport and Sprague, 1942; Mannheimer, 1940 and 1941).

The fundamental tones of low frequency which make up almost all of the third heart sounds are at the lower range of human audibility and, therefore, it is easy to understand why the sound cannot always be heard and why its audibility will depend so much on the training and physical properties of the individual human ear. (See Fig. 1.).

Being made up of a few simple vibrations, the duration of the third sound and its exact time relationship to and the events in the cardiac cycle can be fairly accurately measured.

The length of duration of the sound does not seem to be of great importance, but as can be seen from Table 3 various observers have found it to last for .02 to .10 second, usually .03 to .06. Its relationship, however, to the second sound is much more important; it bears a relatively constant relationship to the beginning of the second sound (Frost, 1949), and this measurement helps to differentiate it from other events in diastole such as the opening snap of mitral stenosis, the pre-systolic or auricular sound, and splitting of the second sound. It can readily be distinguished from the mid-diastolic murmur because of its much shorter length and its obviously much lower frequency, apart from by other means.

Table 3 also shows the remarkably constant figures which various workers have found when measuring ^{the} time interval between the beginning of the second sound and the beginning of the third sound.

TABLE 3

THE PHYSIOLOGICAL THIRD HEART SOUND

Authority	Type and No. of subjects	Time in seconds between beginning of second and beginning of third sound	Duration of third sound in seconds
Boyer et al. 1940	50 medical students	0.10 - 0.14	0.03 - 0.06
Bridgeman, 1915	16 normal boys	0.13 - 0.18	-
Einthoven, 1907	-	0.11 - 0.15	0.02 - 0.03
Frost, 1949	16 patients with normal hearts	0.14 - 0.19	-
Luisada and Mendoza, 1949	185 normal subjects	0.12 - 0.19 ‡	0.04 - 0.06
Mannheimer, 1942	135 normal children, aged 0 - 14 yr.	0.11 - 0.18	0.02 - 0.08
Orias and Braun-Menendez, 1939	100 healthy students, aged 20-25 yr.	0.11 - 0.14	0.07 - 0.10
Rappaport and Sprague, 1942	33 normal subjects •x•	0.16 - 0.24 S 0.16 - 0.18 L	0.03 - 0.08 0.015 - 0.05
Wiggers, 1949	151 young med. students	0.11 - 0.14	-
Wolferth and Margolies, 1933 ++	10 healthy subjects	0.12 - 0.20	-

‡ Measured from the main oscillation of second sound to the beginning of third.

•x• Measured from the beginning of the second sound up to the middle of the third sound. L = logarithmic S = stethoscopic

++ Does not differentiate between 10 normals and 60 patients with heart disease; states that all fall within the same range (See Table 8)

There has been a considerable difference of opinion as to the frequency with which the physiological third heart sound may be heard in health, as can be seen from Table 4 and Table 5 and the claims of various observers fall between as wide a range as from 6% to 95%.

Frost (1949), in reporting a series of 6,000 adults of all ages and suffering from a wide variety of medical conditions, found a physiological third sound could be detected by clinical auscultation in less than 1%.

KcKee (1938), in a carefully-studied series of 105 healthy schoolchildren, heard a third sound in only six. On the other hand, Thayer's original paper in 1908, written at a time when he was dealing with an entirely new subject, made the original claim that the author heard a third heart sound in 24 normal people during the first four months after his attention had been drawn to the sign. Twenty of these were under 30-years of age, but the remaining four were between the ages of 30 and 44. In the following year he analysed a series of 231 healthy individuals in which he demonstrated a third sound in 65% of those under 40-years of age. In the under-20 group a third sound was present in 75%, and in the over-20 group, in 39%. In this series he also found two patients out of 21 who were over 40-years of age in whom a third sound was demonstrable. (Table 5).

Bramwell, in 1943, heard a third heart sound (which he describes as a "duplicated second", but identifies the extra sound with the third heart sound as described by Thayer and others) in 19% of 835 National Service recruits. In the under-20 group a third sound was heard in 43% and in the over-20's, in 10%. In his series, a third heart sound was heard in one patient over 40 (Table 5). Bramwell considers that the third heart sound occurs in

"over-active" hearts as a result of increased flow of blood through a normal mitral valve.

O'Meara (1947) claimed to have heard a third sound in 40% of a series of 1,360 healthy naval recruits. 53% of the recruits under 20 had audible third sounds, and in the over-20 group the percentage of third sounds was 19%. O'Meara claimed that in twenty patients over 40 a third heart sound was heard. (Table 5).

According to Leatham (1949) a third heart sound may be heard in the normal heart of children or adults under 40. Evans (1951 and 1943) states categorically that triple rhythm from the addition of a physiological third sound is common under 20, uncommon after 30, and never heard after 40.

In infants the third heart sound was recorded but not heard by Mannheim in six of 17 children under 1 year.

It is, therefore, quite clear that in at least a proportion of healthy children and young adults an audible third sound may be clearly heard by even an inexperienced auscultator, and with careful training in auscultation there is equally no doubt that a much higher proportion of third sounds can be appreciated. The importance of training and auscultatory experience is clearly brought out by the papers of Sloan, Campbell and Henderson (1952) and of O'Meara (1947).

Provided linear or stethoscopic techniques are used so as not to cut out too many of the low-frequency vibrations, and if sufficient care is taken, a third heart sound can also be demonstrated by phonocardiogram in the vast majority of young subjects, whether or not it can be heard on auscultation (See Table 4).

TABLE 4

THE INCIDENCE OF THIRD HEART SOUNDS EITHER HEARD OR RECORDED IN HEALTHY SUBJECTS					
AUTHORITY	NO. OF SUBJECTS INVESTIGATED	AUTHOR'S DESCRIPTION OF AGE GROUP OR TYPE OF SUBJECT	% OF THIRD HEART SOUND HEARD IN AUSCULTATION	% OF THIRD HEART SOUND DEMONSTRATED BY P.C.G.	
Thayer, 1909	231	Healthy subjects aged 1 to 60 yrs.	65	-	
Bridgeman, 1915	16	Healthy schoolboys aged 12 - 15 yrs.	80	100	
Steinberg, 1926	100	Healthy subjects aged 4 - 14 yrs.	95 +	-	
Gubergritz, 1926	600	?	93.7	-	
Leonhardt, 1932	41	Healthy subjects aged 3 - 14 yrs.	-	83	
Caeiro & Orias, 1937	20	Healthy medical students	-	65	
Orias & Braun-Mendez, 1939	100	Students, aged 20 - 25 yrs.	-	60	
McKee, 1938	105	Healthy schoolchildren	6	100	
Mannheimer, 1940	135	Healthy children, aged 0 to 14 yrs.	0.7	69.6	
Master & Friedman, 1942	100	Normal controls of all ages	-	12	
Rappaport & Sprague, 1942	33	Normal male students	-	85 stethoscopic 30 logarithmic	
Bramwell, 1943	835	National Service recruits	19	-	
Carlgren, 1946	150	Healthy children aged 3 - 17 yrs.	12	78	
O'Meara, 1947	1,360	Naval recruits	40	-	
Wiggers, 1949	151	Young male students	-	4 sitting 26 recumbent	
Sloan et al., 1952	123	Healthy subjects aged 17 - 32	9.8	40 logarithmic 100 stethoscopic	

+ direct auscultation without stethoscope

TABLE 5

AUTHORITY	AGE IN YEARS						
	1-10	10-20	20-30	30-40	40-50	50-60	60-67
Thayer	Total no. of subjects	39	90	55	26	14	7
	% with audible third sound	58.9	84.4	50.9	42.3	14	0
Bramwell	Total no. of subjects	228	315	300	Age over 39 69		
	% with third sound	43	14	9	Approx. 1		
	Actual no. of patients with third sound	96	33	27	1		
O'Meara	Total no. of subjects	745	255	174	103	70	13
	% with audible third sound	53	34.9	25.3	15.5	5.7	0
	Actual no. of subjects with audible third sound	399	89	44	16	4	0

Detailed analysis of the incidence of third heart sounds occurring in all age groups.

From the studies reported by THAYER (1909), BRAMWELL (1943), and O'MEARA (1949).

The physiological fourth heart sound

In addition to the first, second and third sounds, a fourth heart sound has been demonstrated occurring just before the first sound in healthy hearts. This has also been referred to in the literature as the auricular sound and the pre-systolic sound, and there is general agreement that it arises as a result of auricular systole. Orias and Braun-Menendez, in their monograph on the heart sounds (1939) quote "The tenth conclusion of the Report of the London Committee" which was published in 1840 and in which there was a description of audible auricular sounds. We have already seen that under different pathological and experimental conditions auricular contractions may be audible. (Clarke, Ellis and Shaw, 1858; Potain, 1875; Robinson, 1908; Eckstein, 1937), and in cases of auriculo-ventricular dissociation auricular contractions have been recorded by Lewis (1913), Bramwell (1935^a & b), Macleod, Wilson and Barker (1931) and others.

All these instances have, however, occurred under abnormal circumstances, and at the moment we are concerned only with the possibility of an auricular sound occurring in health. In a paper entitled, "Notes on the Normal Pre-systolic Sound", Bridgeman (1916) demonstrated an auricular sound visible on an apical cardiogram (or linear phonocardiograph) in 11 out of 16 healthy schoolboys. He noted that the sound occurred between the beginning of the auricular wave and the main vibration of the first sound in the apical cardiogram. Bridgeman considered that it was produced by tension in the auricular walls during their systolic muscular contraction and to be of a frequency below the limits of audibility.



The sound has been graphically recorded by a large number of observers and all are agreed that, because of its proximity to the loud first sound and its low frequency and amplitude, it is hardly ever heard in health unless the P-R interval is prolonged (Bramwell, 1935; McKee, 1938; Orias and Braun-Menendez, 1939); Mannheimer, 1940; Master and Friedman, 1942; Rappaport and Sprague, 1942; Evans, 1943; Carlgren, (1946; Levine, Harvey and Wiggers, 1949; and Weitzman, 1953). Evans, in 1943, mentioned 14 cases of latent heart block in which the auricular sound was either audible or recorded, and of these 14 cases, six were described as healthy young adults with a "physiologically prolonged P.R." Weitzman (1953) failed to hear the sound in a series of 100 normal subjects.

Characteristics of the physiological auricular sound

The physiological fourth or auricular sound, when visible on the normal phonocardiograph tracing, consists of one to three small low-frequency vibrations which may be separated from the main vibrations of the first sound, but usually become continuous with them.

The frequency of the vibrations of this sound lies in the same range^{as}, or even lower than, that of the third sound, that is to say, almost entirely under 100 cycles per second and usually between 25 and 50 c/s. (Wiggers, 1949; Mannheimer, 1940 and 1941), and there is almost complete absence of overtones, which explains the inaudibility of this sound to the human ear. When recorded with the E.C.G. as a reference tracing, the auricular

sound is seen to occur between the summit of the P wave and the onset of the first sound and R wave. It bears a constant relationship to the P wave, and in 5% of a large series of phonocardiograms Frost (1949) was able to make out initial vibrations of the first sound which occurred just before the Q wave and must, therefore, be due to auricular contraction .

If a jugular pulse tracing is used for comparison, the extra sound commences at or shortly after the peak of the A wave (Wiggers, 1949; Bridgeman, 1914).

There can be little doubt that this sound arises as a direct result of auricular systole for the following reasons:

- (i) it appears coincidentally with the A wave of the phlebogram and immediately after the P wave of the E.C.G.
- (ii) it occurs before the Q wave of the E.C.G. and must therefore arise before ventricular systole.
- (iii) it has never been recorded in the presence of auricular fibrillation (Evans, 1943; Mannheimer, 1940).
- (iv) the small group of low-frequency vibrations recorded during auricular systole in A.V. dissociation have a very similar appearance to the sounds under discussion. (Evans, 1943; Weitzman, 1953).

These various points will all be demonstrated in the subsequent recordings.

The actual cause of the sound under discussion may presumably be either due to muscle vibration set up by auricular muscle contraction, vibrations set up by rapid expulsion of blood into the auricle, or to movement of the A.V. valve as a result of auricular contraction and the passage of blood from auricle to ventricle.

A muscular origin for the sound is suggested by the work of Macleod, Wilson and Barker (1931), who made P.C.G. recordings of patients with auriculo-ventricular dissociation. They showed that even when the A-V valves were closed and there was no blood flow, auricular contraction still produced a sound.

It seems most likely, therefore, that vibrations set up in the auricles by muscular contraction are the main factor in the production of the actual sound, although there may be additional vibrations from the resultant blood flow.

Cossio and Fongi (1936) and Orias and Braun-Menendez (1939) believe that they can separate the physiological auricular sound into separate recognisable valvular and auricular components. They also believe, as was originally suggested by Sansom in 1881 that an early audible auricular sound may be responsible for one type of split first sound.

However, Wiggers (1949) and Mannheim (1946) consider that the vibrations of the auricular sound, consisting as they do of one, two or occasionally three small oscillations, are too small to be split up into separate components by present phonocardiographic methods.

Table 6 shows the frequency with which physiological auricular sounds have been demonstrated by various workers, the approximate age-group involved, and the main character of the sound as regards its duration and relationship to the P wave of the E.C.G.

TABLE 6

THE PHYSIOLOGICAL AURICULAR SOUND. ITS DURATION AND RELATIONSHIP TO THE P-WAVE OF THE ELECTROCARDIOGRAM

Authority	No. of subjects investigated	Author's description of age group and type of subject	Duration of sound (seconds)	Time interval between beginning of P-wave and auricular sound	% of auricular sounds demonstrated by P.C.G.
Bridgeman, 1914	16	Normal schoolboys	0.04	-	69%
Caeiro & Orias, 1937	20	Healthy medical students	0.1	0.034 to 0.046 ⁺	85%
McKee, 1938	105	Healthy schoolchildren	-	-	94%
Orias & Braun-Menéndez, 1939	100	Students aged 20-25 yrs.	-	0.09	-
Mannheimer, 1940	135	Healthy children aged 0 - 14 yrs.	0.01 to 0.07 Average 0.04	0.08 to 0.16 Mean 0.12	54%
Master and Friedman, 1942	100	Normal controls of all ages	-	-	38%
Rappaport and Sprague, 1942	33	Normal male students	-	-	58% stethoscopic 21% logarithmic
Carlgren, 1946	150	Healthy children aged 3 - 17 years	-	-	50%
Wiggers, 1949	151	Young male students	0.04 to 0.06	-	27% recumbent 22% sitting

⁺ Measured from the peak of the P-wave

RECORDINGS OF NORMAL HEART SOUNDS

Recordings were made and are presented from a selection of 13 normal controls. None of these patients had clinical, electrocardiographic or radiological evidence of heart disease, and in all of them the heart sounds on auscultation were within the limits of normality.

No attempt has been made to isolate and identify the muscular, valvular and vascular components of the sounds as it was felt that little could be gained from such a detailed and intricate study. A consideration of all the evidence available leads to the conclusion that the valvular components are most important in the production of the first and second heart sounds. The duration of sounds was not studied, although a rough measurement has been made. (Table). Without an efficient method of calibration, results from such a study may be quite misleading. The heart murmurs and pathological splitting of the sounds, and other abnormalities, will be dealt with in full in a later section.

CASE No.1 was a male patient, aged 27, who had no clinical cardiac abnormality. The heart sounds were clear and not split; neither were there any murmurs. Various tracings made with the Elmquist machine are shown from the mitral area and the aortic area, together with Lead II electrocardiogram and jugular venous pulse, apical, and radial pulse tracings. Tracings A, B, C and D show the jugular pulse and the E.C.G. together with apical P.C.G. recordings with four different frequency bands. (Type I amplifier, Bands 1, 2, 3 and 4). The various time relationships between

the venous pulse, the E.C.G. and the P.C.G. can be studied and the different types of tracing obtained with the differing frequency modifications seen. Band 1 brings out the lower frequency components of the heart sounds and Band 4 the higher. In each case the sensitivity of the amplifier has been increased during the recording, and hence, the intensity or loudness of the sounds increased. As the sensitivity increases, the base-line becomes ragged and irregular and the tracing imperfect due to artefact and the undesirable recording of extraneous noise. It can be clearly seen that measurement of intensity and duration of sounds may be quite misleading if recordings taken with different frequency settings or degrees of amplification are compared. In the case of Band 4 maximum intensity is needed all the time even to record the sounds as relatively small vibrations. This is because the phonocardiograph is much less sensitive to the high frequency than the human ear, and all the loud and easily recorded components of the heart sounds have been cut out by the filter. This particular frequency band (No.4) is of little practical value in most recordings. Bands 1, 2 and 3 approximate to what have been referred to in the introduction as linear, stethoscopic and logarithmic phonocardiograms. Strictly speaking, however, a linear phonocardiogram is a direct apical tracing such as is seen in the record G. The amplifier Type II which is used for many of the subsequent tracings is also so constructed that the three frequency bands - low, medium and high - correspond roughly with the linear, stethoscopic and logarithmic phonocardiograms. Tracing E shows a record taken with Band 3, ~~taken~~ from the aortic area. Here the second sound is seen to be

relatively louder than the first, and as the sensitivity of the amplifier is increased a systolic murmur appears which was not appreciated by the ear; its significance is very doubtful, but its very presence once again acts as a reminder that such observations must be interpreted with caution and always in the light of the clinical findings. Tracings F, G and H show apical and aortic phonocardiograms combined with the jugular pulse, apical pulsation (true linear phonocardiogram) and radial pulse tracings. In the tracing H very small coarse vibrations representing an inaudible physiological third heart sound can be seen, and by reference to the jugular pulse on tracing F this can be seen to occur coincidentally with the falling limb of the "V" wave, and the sound occurs about 0.16 second after the beginning of the second sound.

CASE No.2 was a patient, aged 48, with gastric ulcer. Recordings were taken from the apical and pulmonary areas and, simultaneously, the lead II E.C.G. and a jugular phlebogram. On auscultation a clear splitting of the second sound was heard over the pulmonary area. The tracings taken with medium and low frequency bands (A) do not show this splitting, but when the lower frequencies are cut out, then this physiological splitting can be demonstrated at the pulmonary area (B). An apical early systolic murmur is also seen on the tracing but this was not appreciated by the ear. One small low frequency vibration can be seen at the beginning of the first sound - and this occurs just before the Q-wave of the E.C.G. and coincident with the "a" wave of the phlebogram. This is presumably a physiological fourth or auricular systolic sound of an intensity below the auditory threshold.

Incidentally these tracings also show how variations in the frequency bands employed can produce marked differences in the appearance, apparent intensity, and duration of the same heart sounds recorded at the same area on the one occasion.

CASE No.3 was a healthy medical student, aged 21, with normal heart sounds and an occasionally audible physiological third sound. A recording of the jugular pulse, lead II E.C.G., and apical phonocardiogram show first, second, and very small vibrations of the third heart sounds. The third heart sound can be seen to coincide with the descending limb of the "v" wave on the phlebogram and occurs 0.18 to 0.20 second after the beginning of the second sound. Small auricular vibrations are also seen preceding the Q-wave of the E.C.G. and, therefore, are presumably due to auricular systole.

CASE No.4, another healthy medical student, aged 23, had apical recordings made with the Boulitte machine, and a small representation strip is shown. These tracings are very clear and give fine definition, but as has been mentioned before, some difficulty was encountered in making accurate measurements of the timing of events in the cardiac cycle with this particular piece of apparatus. The illustrative tracing does not have any unusual feature.

CASE No.5 was a healthy female, aged 26, with no cardiac abnormality, but an audible splitting of the first sound, maximum at the left sternal edge, in the third and fourth space. The tracing

shows a lead II E.C.G., a P.C.G. recording from the second left space and from the apex. The splitting of the first sound can be seen quite clearly, although this particular tracing is not perfect as the amplification of the tracing is too high and a clear base-line has not been obtained. The amplifier used for this recording from the second left space was the one described as 'Type III' in the introduction, and used with, but not designed for, the Elmquist apparatus. Three frequency channels or bands were available - low, medium and high. In record A the low frequency has been used, and in record B the medium frequency band. In both records the apical tracing has been recorded with amplifier Type I, using the frequency band 1.

CASE No. 6 was a healthy old man, aged 75, with no cardiac abnormality. His records are perfectly normal.

CASE No. 7, a male aged 48, similarly had no abnormality on clinical, radiological or electrocardiographic examination of the heart, although he had a previous history of angina of effort, with transient E.C.G. changes. Four years after the recordings were taken he was alive and symptom-free. His records also show no abnormality.

CASE No. 8, another male, aged 62, was admitted with retention of urine. His heart was clinically normal, B.P. slightly raised to $\frac{180}{100}$ on admission but falling to $\frac{160}{85}$ after rest in bed. He was ultimately subjected to prostatectomy and was alive and well without symptoms two years later. At the time of recording his only

auscultatory abnormality was a split first sound, and the record shows no definite abnormality. No splitting of the first sound can be seen, but on the apical tracing the first vibrations coincide almost exactly with the Q-wave of the E.C.G. and these may well represent the auricular component of the first sound.

Case No. 9 was a normal female child of 12 years, with no clinical abnormality of the heart. No third heart sound was audible, but splitting of the second sound, varying with respiration, was detected. Apical recording with the Boulitte (A), and the subject breathing freely, shows first, second and (inaudible) third heart sounds. Recordings, also from the apex (B) but with the breath held in expiration, show a definite splitting of the second sound. A low frequency, low intensity fourth or auricular sound is also seen occurring with and just before the Q-wave.

Unfortunately the Boulitte apparatus has an amplifier with fixed frequency. Intensity of sound recording only can be varied. Neither can a jugular pulse or other reference tracing than the E.C.G. be employed.

Case No. 10 was a male, aged 51. He also had a clinically normal heart, but splitting of the first heart sound had been heard. At the time of recording no such splitting was detected. The records from the aortic area and apex are perfectly normal, but once again a probable auricular component is seen occurring just before the Q-wave of the E.C.G. On the apical tracing an innocent systolic murmur can be seen.

CASE No.11 was a 25-year-old pregnant housewife. Her heart sounds were normal but she had an audible third heart sound, presumably due to the increased blood volume of pregnancy; the second sound was split, and the degree of splitting varied with respiration. Apical recordings show the normal first, second and third sounds, with the split second sound visible during inspiration. Timing is difficult to measure accurately, but the second component of the second sound begins approximately 0.04 second after the second sound itself. The third sound begins approximately 0.14 to 0.16 second after the beginning of the second sound. The records show a very clear differentiation between the split second and the third sound.

Tracings of the heart sounds were also made from two further normal patients - Case No.12, a female aged 42, and Case No.13, a male aged 27. These recordings showed no abnormality.

Third heart sounds which were physiological in nature were also demonstrated in three young people with minor degrees of congenital heart disease - Case Nos. 84, 86 and 88; also in three young pregnant women with mitral stenosis - Case Nos. 101, 111, and 112, and in two girls aged 17 and 13 years respectively, who had third heart sounds - Case Nos. 122 and 123. The first of these two latter cases had fairly severe mitral disease and the second had rheumatic chorea, and, therefore, in both cases the extra sound may in fact have been related to the disease rather than the age of the patient and come into the category of rapid filling gallop rather than physiological third sound. These recordings are all

dealt with fully in subsequent sections, as also are numerous examples of pathological rapid filling gallop from a variety of causes.

SUMMARY AND CONCLUSIONS

1. The nature, origin and probable mechanism of the normal first, second, third and fourth heart sounds have been discussed.
2. Thirteen recordings have been made from patients of both sexes and of varying ages (12 - 75 years). All had normal heart sounds and no cardiac abnormality at the time of recording.
3. It is clear from the literature reviewed that the physiological heart sounds have already been studied in some detail. A survey of this literature has been made and the published criteria for these sounds and their relation to each other are presented.
4. Illustrations are given, taken from records made with the various phonocardiograph machines and amplifiers used in this study. Some of the technical difficulties encountered have been mentioned. The effects of varying intensity and frequency bands are shown and the various forms of reference tracing used are also demonstrated.
5. Although the literature is reviewed, no attempt to make a comprehensive study of the normal heart sounds in health has been made here. The duration of the individual heart sounds has not been /

been studied in detail as it would appear that such measurements are unreliable and of little, if any, practical value. However, figures for the duration of both first and second sounds, as given in the literature, are quoted and tabulated, and the approximate heart sound durations from the cases reported here have been measured. (Table 7). In these cases the total measurement of the sounds has been given, and in the case of the first sound this includes any auricular vibrations preceding the Q-wave of the E.C.G. The average duration of the first sound was found to be 0.14 second, and of the second sound, 0.06 second.

6. The physiological third heart sound may be heard frequently in young healthy people. Its characteristics and frequency of occurrence are discussed. It seems that the proportion of subjects in whom it may be heard varies according to the experience of the observer. It is unusual to hear this sound in subjects over 40 years of age, but some observers claim that they have heard it in a small proportion of subjects over 40.
7. The physiological third sound may more often be demonstrated on the phonocardiograph tracing than heard with the stethoscope.
8. In this small series the third sound was heard in two patients but was visible on the tracing in four. One of these patients was a young pregnant woman. It is presumed that the increased blood volume of pregnancy and resultant increase of cardiac blood flow are responsible for this fairly common physical sign of pregnancy. (Further examples of this phenomenon are referred to briefly but dealt with fully in later sections).

TABLE 7

APPROXIMATE DURATION OF FIRST AND SECOND HEART SOUNDS
IN 11 NORMAL CASES

Case No.	Duration in seconds	
	First sound	Second Sound
1	.12	.06
2	.20	.10
3	.15	.08
4	? +	?
5	.15	.06
6	.15	.05
7	.16	.06
8	.16	.05
9	.16	.06
10	.16	.06
11	.14	.06
Average	<u>.14</u>	<u>.06</u>

The first sound has been measured from the beginning of the first vibration seen on the P.C.G. to the end of the last vibration and therefore includes the auricular sound when present.

+ Boulitte recording with no time-marker.

Cases Nos. 12 and 13 also had no time-marker and are, therefore, excluded from the Table.

9. The physiological third sound occurs at the time of rapid ventricular filling and is probably due to the sudden distension of the ventricular walls setting up a faint sound of low frequency.
10. In this small series the time interval between the beginning of the second sound and the beginning of the third fell between 0.12 second and 0.20 second, with an average of 0.16 second. It can be seen from the Table that this falls well within the average normal range as given by other workers.
11. The physiological fourth or auricular systolic sound has also been reviewed and its characteristics discussed. All workers are agreed that it is not normally audible in health.
12. It is, however, frequently visible on the phonocardiograph and is represented by a few low frequency vibrations occurring just before the commencement of ventricular systole. When recorded with the E.C.G. and jugular phlebogram, it is seen to occur between the summit of the P-wave and the Q-R segment of the E.C.G. and shortly after the peak of the "A" wave of the jugular pulse.
13. Vibrations believed to represent auricular systole are visible on five of the thirteen normal tracings presented here. An auricular sound was not audible in any of these cases.
14. Physiological splitting of both first and second sounds has been reviewed and their characteristics discussed.
15. Splitting of the first sound is demonstrated in two recordings and of the second sound in three, and the effect of respiration on splitting of the second sound is shown.

16. The interval between the commencement of the first or second component of the split second sound is usually from 0.02 to 0.03 second but may be more during deep inspiration.
17. A split second sound would appear to be due to slight asynchrony between closure of the aortic (which normally closes first) and pulmonary valves.
18. A split first sound is probably due in most cases to similar asynchrony between the mitral (which normally closes first) and the tricuspid valves. In two cases described here (aged 51 and 62 respectively), a split first sound was heard on auscultation. Phonocardiographic tracings showed an early component of the first sound which might have been due to auricular systole rather than valvular asynchrony.
19. A study of the split sound and the third and fourth heart sounds occurring in disease follows in subsequent sections.
20. Although no murmurs were heard in these cases, an early systolic murmur is seen on the apical phonocardiogram in four instances.

SPLITTING OF THE HEART SOUNDS IN DISEASE

Splitting of the first and second heart sounds due to disease was described in 1881 by both Sansom and Balfour, and was also discussed by Ringers and Phear in 1894 and Boyd in 1896. Sansom maintained that splitting of the first sound was usually due to separation of the auricular and ventricular components of the sound, but was occasionally due to asynchronous contraction of the ventricles and closure of the mitral and tricuspid valves. All agreed that in mitral stenosis the second sound may be widely split, but only Sansom made the clear differentiation that in this condition, apart from a tone duplication of the sound a distinctive unusually wide "splitting" might occur, the second component of which he thought was due to the sudden tensing of the mitral valve.

This extra sound, occurring only in mitral stenosis, was of course the 'opening snap' which had been described earlier by Bouillard (1835), Duroziez (1862), and others, and which will be dealt with in detail in a later section of this thesis.

The causes of pathological splitting of the heart sounds have also been investigated and discussed more recently by several workers, including Lian (1934), Orias and Braun-Menendez (1939), Frost (1946), and Wiggers (1949). In particular, Katz (1925), Wolferth and Margolies (1935), and Leatham (1954) have explained the mechanism which gives rise to splitting of the heart sounds.

The first sound is due largely to the closure of mitral and tricuspid valves, and if there is slight asynchronism of right and left ventricular contraction, then splitting of the sound may occur. According to Leatham the mitral valve usually closes slightly before the tricuspid.

Pathological splitting of the first sound may therefore occur more frequently in the presence of defects of conduction such as bundle branch block.

Splitting of the second sound is probably due to asynchronous closure of the pulmonary and aortic valves, and once again, Leatham believes that left ventricular events occur slightly before right ventricular and, therefore, under normal circumstances he attributes the first element of a split to aortic valve closure and the second element to the pulmonary valve.

Pathological splitting of the second heart sound may be due to any condition which involves electrical or mechanical delay in the closure of either the aortic or the pulmonary valve and may occur in the following conditions:

- bundle branch block
- pulmonary hypertension
- atrial septal defect
- pulmonary stenosis

In bundle branch block the delay will be in conduction, in pulmonary hypertension it will be mechanical, and in atrial septal defect there will be both an electrical conduction defect and a mechanical delay as a result of increased right-sided blood flow. In pulmonary stenosis also, prolonged right ventricular systole and mechanical delay in closure of the abnormal pulmonary valve will tend to cause splitting of the second sound.

It should also be possible to decide which component of either sound is due to right-sided activity and which to left, by comparing the intensity of the first and second parts of the split first sound at the mitral and tricuspid areas and of the split second sound at the pulmonary and aortic areas. Also, if aortic valve closure precedes pulmonary closure, then, as deep inspiration increases, the right ventricular filling rate and hence prolongs right ventricular systole, it will delay closure of the pulmonary valve and increase the degree of splitting. If the pulmonary valve is closing before the aortic, then deep inspiration will tend to decrease the interval between them and therefore abolish splitting of the second sound. This latter state of affairs is referred to as paradoxical splitting and most commonly occurs due to electrical delay in closure of the aortic valve, as occurs in left bundle branch block (Leatham, 1954).

With the exception of this observation on paradoxical splitting, which Leatham believes to be pathological, the characteristics of the split sound heard or recorded in disease do not vary from those of the physiological split sound. The time interval between the first and second components of the split sound, whether pathological or physiological, is the same, and so is their relationship to the E.C.G. or phlebogram reference tracing. According to Wolferth and Margolies (1933) the interval between the two elements of the split sound never exceeds 0.07 second, but Leatham has recorded an interval of as long as 0.1 second during deep inspiration.

DESCRIPTION OF CHARACTERISTICS AND DIFFERENTIAL DIAGNOSIS
OF SPLIT SOUNDS

The split sound consists of two usually high pitched elements of the same quality and separated by the smallest perceptible gap. On auscultation it gives the definite impression of being essentially a single sound with a double peak of intensity, and this is also its appearance on the phonocardiogram. The rhythm is always basically a dual rhythm and it does not have the cadence of a triple or gallop rhythm where three entirely separate sounds can be heard. The splitting is never palpable; the split first sound is heard best at the mitral area, and the split second, at the base. The degree of splitting of the second sound usually varies considerably with respiration.

Differential diagnosis

The opening snap of mitral stenosis gives the initial impression of being a very wide "splitting" of the second sound, but has clearcut differentiating characteristics. Typically it is best heard at the lower left sternal edge, and the snap itself, which is the second element of the wide "splitting", has a very distinct high-pitched snapping quality quite different from any other sound heard on auscultation and quite different from the second sound itself. The second heart sound and the snap are much further apart than the two elements of the split second sound, and the rhythm or cadence produced is quite different. If looked for, other signs of mitral stenosis will always be present when the snap is heard. Splitting of the second sound and the opening snap may frequently be present

in the same patient. The triple rhythm resulting from the addition of either a third or fourth heart sound is again quite distinct; the extra sound is of a much lower pitch than either the opening snap or the components of a split sound, and when audible, the triple rhythm produced gives the impression of three clearly separate sounds. The extra sound can often be palpated on the chest wall over the point of maximum intensity. There is usually no difficulty in distinguishing either the presystolic murmur of mitral stenosis or the early diastolic murmur of aortic incompetence from splitting of the first or second sounds. Occasionally, however, if the first sound is very loud or if the early diastolic murmur is very short, there may be some doubt. Again, the presence or absence of other characteristic physical signs will usually help to make the diagnosis, and the value of P.C.G. tracings in these circumstances will be discussed later.

Leatham (1954) has also drawn attention to another phenomenon which is sometimes confused with splitting of the first heart sound due to asynchronous closure of the A-V. valves. This is the pulmonary early systolic sound which is high-pitched and clicking in character and is heard only at the base immediately following the first sound; it may in fact be louder than the first sound. He calls it the 'pulmonary early systolic click' and considers it to be due to the ejection of blood into a dilated pulmonary artery. This physical sign usually occurs in pulmonary hypertension from any cause, pulmonary stenosis, or idiopathic pulmonary dilatation. More rarely, Leatham considers that an aortic systolic ejection sound may occur due to dilatation of the ascending aorta from coarctation, hypertension, aortic valvular disease, or other causes.

Although splitting of the heart sounds, and particularly paradoxical splitting, may occasionally be of value as a confirmatory sign in the presence of pulmonary hypertension or other conditions in which asynchronous valve closure is due to pathological change, it is usually of no clinical significance. Levine and Harvey (1949) state that splitting of either first or second heart sound is more frequently found in the absence of heart disease than in association with it.

For this reason, therefore, it is of the utmost importance that split sounds should be clearly differentiated from the opening snap of mitral stenosis, gallop rhythm, and the cardiac murmurs referred to above. Differentiation between these auscultatory phenomena may well be aided by the phonocardiograph, but it seems unlikely that the split sound occurring in health will have different phonocardiographic characteristics to that occurring in disease, as their mechanism will in many cases be similar. Examples of phonocardiograms taken from cases of physiological splitting have already been presented.

RECORDINGS OF SPLIT SOUNDS OCCURRING IN THE PRESENCE OF DISEASE

CASE No.14, a 44-year-old married woman, had several recordings made in an effort to help in the diagnosis. She had a long history of moderate hypertension (B.P. $\frac{200}{110}$, but fluctuating), and a history suggestive of angina pectoris. The clinical picture was confused by a considerable functional overlay, but the diagnosis of ischaemic heart disease was confirmed by a positive anoxaemic test (Turner and Morton, 1952). There was no evidence of renal impairment, optic fundi showed early hypertensive changes, and there were no signs of congestive cardiac failure, although clinical, radiological and electrocardiographic examination all showed evidence of slight left ventricular hypertrophy. On auscultation there was a very clear splitting of the first sound to be heard all over the praecordium. It was felt important to differentiate this from a presystolic gallop rhythm, particularly in view of the mixed clinical picture of organic and functional disease.

Recordings were made from the pulmonary area, from the aortic area, and from the apex. Unfortunately the apical record is technically poor. The pulmonary and aortic tracings, however, show a clearcut splitting of the first sound, commencing well after the onset of systole as judged by the QRS complex. There is, therefore, a true splitting and not a gallop rhythm, but the aetiology is not obvious and the P.C.G. does not help to differentiate between pathological and physiological causes. An early systolic murmur is also seen at both pulmonary and aortic areas.

The irregular base-line in the apical tracing is due to artefact.

Four years later this patient was alive and well, although still extremely neurotic, and the split first sound was still clearly audible. The anoxaemic test for myocardial insufficiency was repeated and found to give a normal response. In retrospect it would appear most likely that the splitting of the first sound in this case was not in any way relative to disease, but it is clearly not possible to be certain, and the phonocardiograph did not help in this differentiation. It did, however, exclude the presence of a gallop rhythm with its more serious prognostic significance.

Case No.15 was a 50-year-old housewife with moderate essential hypertension, who presented a problem similar to the previous patient. A clear splitting of the first sound was heard and a presystolic gallop queried.

A phonocardiogram from the apex confirms the presence of two main components of the first sound, both occurring after the R-wave of the E.C.G. and probably accounting for the audible split. Unfortunately no follow-up is available, and therefore, no further conclusion can be drawn. The records are not reproduced.

Case No.16 was a 62-year-old woman with suspected angina pectoris. The only detectable cardiovascular abnormality was, once again, a splitting of the first sound, thought by some to be a presystolic triple rhythm. This, however, varied with respiration, and the P.C.G. tracings made with the Boulitte machine show a distinct splitting of the main components of the first sound noticeable in full expiration. The entire sound appears well after

the Q-R. segment of the E.C.G. One month later this patient had a diseased gallbladder removed and lost all her chest pain. Four years later she was alive and well, without symptoms. Once again, the P.C.G. seems to have been of some diagnostic value.

Many of the recordings from the section on mitral stenosis and incompetence show splitting of the heart sounds. One particular example will be discussed here because of its relevance to the problem of split sounds in disease. This case - No. 127 - was a young pregnant woman aged 23. On auscultation, at the apex, a presystolic murmur, a loud first sound, a normal second sound followed by a clear opening snap, and a mid-diastolic murmur were all heard. At the base there was a split second sound and an early diastolic murmur. This woman was thought to have pure mitral stenosis and also aortic incompetence of mild degree.

Recordings (A) from the apex show the presystolic and mid-diastolic murmurs and the loud first sound. The opening snap is clearly seen, and simultaneous records from the third left intercostal space also show a split second sound. There is also a diastolic murmur which appears to start with the second sound. Different records (B), taken from the apex with a lower frequency band, probably show in addition a low-pitched third heart sound which by its timing can be seen to occur later than the opening snap.

These records, therefore, illustrate a split second sound, which may well be physiological, an opening snap of mitral stenosis, and a third heart sound probably associated with the pregnancy.

Several other recordings in the mitral series demonstrate splitting of the second sound, including Cases Nos. 95, 112, 115, and 122. Case No. 114 in the mitral series also demonstrates the relationship between the split second sound, the opening snap, and the jugular pulse tracing. Clearcut splitting of the second sound was also heard and demonstrated in Case No. 22, a male with ischaemic heart disease and rapid filling gallop; also in Cases Nos. 86 and 89, both suffering from congenital heart disease, and case No. 78, who had an innocent systolic murmur. This latter case is described in the section on cardiac murmurs. Patients Nos. 63, 65, 67, 74, and 70 all had split sounds and bundle-branch block, in which section they are dealt with. Case No. 76 also had a splitting of the second sound in addition to a systolic click.

SUMMARY AND CONCLUSIONS

1. The occurrence of splitting of the first and second sounds in the presence of disease has been discussed and the literature on the subject reviewed. Much of the relevant information on splitting of the sounds has been obtained during the studies of various heart lesions reported in other sections of this thesis, but, regardless of possible overlapping, the main conclusions and findings will be summarised here.
2. In the present study the auscultatory and phonocardiographic features of true splitting of the heart sounds have been found to be indistinguishable, whether physiological or accompanying cardiovascular abnormality.
3. Paradoxical splitting of the second heart sound has been reported in the literature as occurring in left bundle-branch block and aortic stenosis; it has not been detected in this series, but was not specifically sought.
4. Splitting of the sounds is a common physiological phenomenon often encountered in healthy subjects due to the delay in contraction of the right ventricle which occurs during inspiration. In disease, delay in contraction may effect right or left ventricles. It may be due either to electrical factors such as conducting system defect in bundle-branch block or to mechanical factors due to a discrepancy between stroke volumes of the right and left ventricles as may occur in pulmonary hypertension from any cause, atrial septal defect, or mitral and aortic valvular disease.

5. A split heart sound is seldom of direct diagnostic value by itself, but taken in conjunction with other findings it may be of assistance in arriving at a correct diagnosis.
6. It is important that splitting of the first sound be clearly differentiated from pre-systolic murmurs and auricular gallop rhythm, and that splitting of the second sound be differentiated from the opening snap of mitral stenosis, the physiological third sound, rapid filling gallop rhythm, and an early diastolic murmur. Systolic click may also be confused with splitting of the sound.
7. This is where the value of the phonocardiograph should be most apparent. Recordings are shown and case histories discussed in this and other sections where they have been either of diagnostic value or of specific interest; but a detailed study of this particular aspect of phonocardiography has not been attempted.
8. Recordings have been of assistance in differentiating between split sounds and auricular gallop rhythm. Cases have also been referred to and discussed in the appropriate section, where splitting of the second sound has been recorded in the presence of atrial septal defect, patent ductus arteriosus, mitral and aortic valvular disease and bundle-branch block.
9. As will be seen later, the exaggerated splitting of the second sound due to delayed pulmonary valve closure which is reported to occur in right bundle-branch block has not been observed. A number of cases with clear splitting of

the second sound occurring in left bundle-branch block have, however, been recorded and discussed in the section of this thesis which deals with bundle-branch block.

10. The relationship between the split second sound, the opening snap and the jugular venous pulse has been demonstrated.
11. Considering the evidence available from these limited studies and recordings it is concluded that the phonocardiogram does have a definite but restricted value in the study and differential diagnosis of splitting of the heart sounds. It has not been found to be as informative in the investigation of asynchronous closure of aortic and pulmonary valves and their clinical significance as has been suggested by some other workers.

GALLOP RHYTHM

In the presence of organic heart disease the development of an audible third heart sound, giving rise to a triple rhythm, may have an entirely different significance to the presence of a similar phenomenon in young healthy subjects. It is customary to refer to the latter as a 'physiological triple rhythm'. The term 'gallop rhythm' is reserved in this thesis for the rhythm produced when in addition to the normal first and second sounds an extra audible heart sound appears in diastole, and which is associated with heart disease. It will be seen that the gallop rhythm may be of three types, according to the position in the cardiac cycle at which the extra sound appears, and the following descriptive terms will be used: (See Fig. vii)

- (1) AURICULAR gallop - which is due to the addition of an audible auricular sound to the normal first and second sounds, the extra sound being dependent on auricular systole (Fourth sound gallop; pre-systolic gallop; or Evans type II B triple rhythm, 1943).
- (2) RAPID FILLING gallop - which is due to the addition of an audible sound at the same position as the physiological third sound in the cardiac cycle and occurs during the phase of rapid ventricular filling (Third sound gallop; proto-diastolic gallop; or Evans type I B triple rhythm).
- (3) SUMMATION gallop - when diastole is so shortened by tachycardia that it is either impossible to be certain which of the above-mentioned sounds the gallop is due to, or when, for a similar reason, one is superimposed upon the other to give a single sound (Mesodiastolic gallop or Indeterminate gallop).

Gallop rhythm was first recognised as a sign of heart disease and described by Charcelay de Tours in 1838, but the term "Bruit de Galop" was, according to Potain, probably introduced by Bouillard in 1847 when describing the cadence or rhythm of an auricular gallop. A full and accurate classification and description of gallop rhythm was made by Potain in 1900, but the same author had previously referred to the "Triple bruit du coeur" when describing a case of constrictive pericarditis in 1856 and "bruit de galop" occurring in cases of hypertension and "interstitial nephritis" when writing in 1875. Potain's classification needs little amendment today; it is the basis of the one given above and used in this thesis. Potain coined the three terms, pre-systolic gallop, proto-diastolic gallop, and mesodiastolic gallop. Potain also listed the types of pathological condition which gave rise to gallop rhythm, and discussed the possible mechanism of origin. He believed the extra gallop sound to be a pathological accentuation of a normal physiological phenomenon. He also classified gallop rhythm according to the area over which it was most easily heard. According to Potain, right-sided gallop was heard loudest to the right of the sternum, was rare, but found occasionally in hepatic and gastro-intestinal disturbance. Left-sided gallop was heard loudest over the apex or just internal to it, was much the commoner of the two, and occurred most frequently in hypertensive failure. Barie wrote a very clear account of the subject in *La Semaine Médicale* in the year 1893, but added little to the fundamental ideas of Potain.

The investigations of Hirschfelder, Gibson (1907), Thayer (1908), and Bridgeman (1914 and 1915) into the origin of the physiological third and auricular heart sounds have already been discussed. The knowledge gained by these workers paved the way to an understanding of the relationship between physiological triple rhythm and pathological gallop. Thayer and MacCallum (1906) had already noted that when the exposed heart of an experimental animal was accidentally rotated during auscultation a gallop rhythm developed. They also noted that the extra gallop sound coincided with an active dilatation and sudden filling of the left ventricle.

Robinson published in 1908 a full account of gallop rhythm illustrated by apical polygrams taken with a Mackenzie Polygraph. He showed how the auricular gallop sound coincided with the "a" wave of a jugular phlebogram and, therefore, auricular systole. He also thought that the rapid filling gallop sound coincided with the sudden flow of blood from auricle to ventricle in early diastole. In a clinical study without phonocardiograms, Holt (1927) reviewed the literature and discussed the occurrence of gallop rhythm. She queried the existence of right-sided gallop as had been suggested by Potain. She felt that although far too little was known about the aetiology of gallop rhythm, it should probably be regarded as a sign of bad prognosis. In the following year, Paul White analysed 100 cases of gallop rhythm all occurring in ill patients; no phonocardiograms were taken, but clinical assessment suggested that most of them fell into the category of rapid filling or summation gallop. 45% of these patients were

dead within two years. He concluded that gallop rhythm was most commonly found in young people with acute nephritis or mitral disease and in older patients with hypertensive, ischaemic or syphilitic heart disease, and it was particularly common in association with aortic lesions. Tachycardia and congestive cardiac failure both commonly accompanied gallop rhythm, and White frequently noted that when these were relieved by digitalis the gallop disappeared. In 64 of his cases an electrocardiogram was obtained and was normal in only two. Robinson's observations on the relationship of the auricular gallop sound to auricular systole were confirmed phonocardiographically by Mond and Oppenheimer in 1929, who studied a series of hypertensive patients with gallop rhythm, and similar conclusions were arrived at in 1931 by Macleod, Wilson and Barker. Both groups of workers demonstrated the persistent relationship between auricular sounds and auricular contraction, not only in gallop rhythm, but also in partial and complete heart block.

Duchosal in 1932 and Wolferth and Margolies in 1943 published exhaustive studies on gallop rhythm, together with phonocardiographic illustrations. In twenty cases of hypertension Duchosal thought that the relationship between the position of the auricular gallop sound and the P-wave might have prognostic value. He thought that in mild hypertension the gallop sound might just appear before the R-wave without making a distinct gallop or auricular sound and that the interval between the summit of the P-wave and the gallop sound was well separated from the first sound, forming a distinct audible and palpable gallop; the P-wave/gallop interval was, therefore, short and the prognosis

worse. Duchosal's figures for the duration of the auricular gallop sound in a series of 33 cases were 0.04 - 0.15 second and for the interval between the peak of the P-wave and beginning of gallop sound 0.02 - 0.14 second. He also made the interesting observation that the auricular sound in complete heart block has a double peak, whereas the sound in auricular gallop is single. Duchosal recognises that too much importance must not be placed on the significance of a gallop rhythm in view of the many unknown factors involved, and he admits that he has seen occasional cases where the gallop has persisted for years without apparent deterioration in health. In 60 cases of gallop Wolferth and Margolies concluded from a study of the phonocardiographs that there was no apparent difference in appearance and timing between the physiological third sound and the rapid filling gallop sound, and that the only criterion of difference between them depended on the patient's age and the state of cardiac function. Clinically, however, the pathological gallop rhythm was frequently palpable as well as being audible, low-pitched, and maximum at or near the apex or lower left sternal edge. Table 8 shows figures that have been quoted by various workers for the duration of the rapid filling gallop sound and its time relationship to the second sound. Comparison of these figures with the corresponding measurements for the physiological third heart sound in Table 3 show their close similarity. Wolferth and Margolies did encounter two patients with the condition of right-sided gallop described by Potain, but not recognised by most other workers since. They suggested the use of the word 'summation' to describe Potain's mesodiastolic gallop,

TABLE 8

THE RAPID FILLING GALLOP SOUND

Authority	Type and No. of subjects	Time in seconds between beginning of second sound & beginning of gallop sound	Duration of gallop sound in seconds
Duchosal, 1932	-	0.11 - 0.18	-
Frost, 1949	29 cases with heart disease	0.10 - 0.20	-
Lian and Racine, 1933	-	0.12 - 0.14	0.04 - 0.08
Mannheimer, 1942	135 children with C.D.H.	0.11 - 0.18	0.02 - 0.08
Wolferth and Margolies, 1933 † †	60 patients with heart disease	0.12 - 0.20	-

† † Does not differentiate between 10 normals and 60 patients with heart disease, stating that all fall within the same range. See Table 3.

and they demonstrated how the auricular and rapid filling sound could become superimposed in tachycardia or in complete heart block to produce an audible sound even when the two individual sounds were themselves separately inaudible. They confirmed the fact that true gallop rhythm only occurs in the presence of cardiac damage, and they discussed the differential diagnosis between gallop sounds, murmurs, split sounds, and the opening snap of mitral stenosis.

Lian and Racine (1933) and Lian (1934) analysed the subject of gallop rhythm at some length, considering it to be a sign of left ventricular insufficiency and overloading, and to have serious significance. They also describe two other phenomena similar to gallop rhythm: firstly, the "claquement mesosystolique pleuro-pericardique" - an innocent systolic clicking which they believe to be associated with pleuro-pericardial adhesion and which will be discussed later; and, secondly, the "vibration pericardique protodiastolique", which is an unusually loud high-pitched rapid filling gallop heard in cases of calcified pericarditis.

Bramwell in 1935 and Thompson and Levine in 1936 analysed the occurrence and significance of gallop rhythm from a clinical standpoint, but without phonocardiography, as did Master, Dack and Jaffe in the following year. All agreed that the appearance of a gallop rhythm was an event of serious prognostic significance. It was thought to be of particularly serious significance if it was associated with any of the following conditions: tachycardia, normal rhythm and a failing heart, rheumatic heart disease, myocardial infarction, or in patients

under forty years of age. In Thompson and Levine's series of 192 cases with gallop rhythm, 89 patients died, with an average survival time of ten months and twenty days after the appearance of the gallop.

Orias and Braun-Menéndez, in their monograph (1939), dealt comprehensively with the subject of gallop rhythm from both the historical and clinical aspects. Arenberg (1940) and Mannheimer (1940 and 1942) made further contributions to this study. The latter did not hear a gallop rhythm in any of 135 children all suffering from congenital heart disease. However, by means of calibrated phonocardiography he reported auricular and third sound vibrations on his records which he considered to be outside the normal limits for duration and size in 11 cases. In 1942 Mannheimer stated that in his experience, summation gallop has a much more serious prognostic significance than the other types, and in the same year Master and Friedman investigated 78 patients immediately after myocardial infarction and 100 normals of the same age group. They noted a gallop sound when congestive failure developed and associated with this there was a decrease in intensity of the first sound. (Auricular gallop developed in 33% of the infarction cases, although an auricular sound was visible on the phonocardiogram of 83%. Rapid filling gallop developed in 9%, although vibrations were seen on the P.C.G. of 47%. Summation gallop was seen in 6%. In the normal series gallop was never heard, but auricular vibrations were seen on the P.C.G. of 38% and third sound vibrations seen in 12%).

Garvin (1943), in a clinical and statistical study, but without phonocardiography, found that 25% (199) of a series of 790 adult autopsied patients dying of heart disease, had a gallop rhythm. The commonest cause of this was hypertensive and ischaemic heart disease, and Table 9 is taken from his paper. He also found that patients with these conditions and also other cardiac patients who developed a gallop rhythm tended to die younger than those who did not. Of his series, the average age at death of the patients with gallop rhythm was 49.5 years and of those without gallop rhythm was 58.8 years.

TABLE 9

‡ The Frequency Distribution of Gallop Rhythm in the Various Types of Heart Disease			
Type of Heart Disease	No. of Cases.	No. with gallop rhythm	%
Hypertensive Heart disease	264	93	35.2
Coronary heart disease	177	55	31.1
Rheumatic heart disease	119	12	10.1
Syphilitic heart disease	67	7	10.4
Cor pulmonale	54	10	18.5
Subacute bacterial endocarditis	31	2	6.4
Acute bacterial endocarditis	13	1	7.7
Thyroid heart disease	9	1	11.1
Calcified aortic stenosis	9	3	33.3
Obliterative pericarditis	7	0	0.0
Tuberculous pericarditis	7	4	57.1
Miscellaneous	14	3	21.4
Undiagnosed	19	8	42.1
	790	199	25.2

‡ (From GARVIN, C.F., Amer. J. med. Sci., 1943, 205, 814)

Evans reviewed the entire subject in 1943 and re-classified triple rhythm to include physiological sounds and pathological gallop as a result of a study of 270 subjects. He found a physiological third heart sound in 125 healthy people under 40 and a similar pathological gallop sound in 80 cases with heart disease, all of these having right ventricular failure. He also heard an auricular sound by means of a cardiophonograph in 14 cases with delayed A-V. conducting mechanism, and in 46 cases a similar auricular sound could be heard as a clinical gallop rhythm, these latter patients all having left ventricular failure.

From Scandinavia came two important contributions - those of Carlgren in 1946 and Frost in 1949. Carlgren produced a gallop associated with cardiac dilatation experimentally in 12 out of 30 rabbits after myocardial damage had been produced. In a study of 5,000 children in hospital he found a true gallop as opposed to a physiological sound in 104, and he concluded that although some evidence of heart disease, past or present, could be found in the majority of these, some seemed perfectly normal, and, therefore, a true gallop may rarely occur in normal children. Using Mannheim's calibration technique he found that pathological gallop sounds were of a significantly higher frequency and the amplitude (measured in dynes per second) was also greater than the corresponding physiological sounds. He maintained that a system of calibrated recording is essential if all gallop sounds are to be diagnosed and distinguished phonocardiographically from the physiological third or auricular sounds.

Frost, without special calibration technique, was unable to demonstrate any phonocardiographic difference between these sounds, and he concluded that the physical characteristics of the physiological third and the rapid filling gallop sounds were identical, as also were the auricular sound and the auricular gallop sound. In either case the extra sound bore the same relationship to the second sound or the P-wave regardless of heart-rate. He did not attempt to measure the amplitude of the sounds or to measure the frequency with any degree of accuracy. (See Table 3, Table 6 and Table 8). Unlike Evans, he did not find a frequent relationship between the presence of an auricular gallop and a prolonged P.R. interval, and unlike Duchosal he was unable to demonstrate any correlation between the P-wave/auricular gallop interval and the severity of the illness. In a later publication of the same year Frost analysed the number of gallop rhythms encountered in a series of 6,000 patients. Myocardial infarction accounted for 44% of the cases of gallop rhythm, ischaemic heart disease and hypertension for 17%, and affection of the aorta for 15%. He concluded that the mortality was higher in these cases because the gallop sound seemed to be a sign of low functional capacity being associated with raised blood pressure, cardiomegaly and electrocardiographic abnormalities. He rarely found a gallop with mitral stenosis, anaemia or thyrotoxic heart disease, but it occurred in several cases of constrictive pericarditis. He found no case of right-sided gallop, and auricular gallop was slightly more common than rapid filling gallop (although not all of his cases were confirmed phonocardiographically).

TABLE 10

THE AURICULAR GALLOP SOUND OCCURRING
IN PATIENTS WITH HEART DISEASE

Authority	No. of subjects investigated	Duration of sound. Seconds	Time interval between P-wave and beginning of gallop sound
Duchosal, 1932	33	0.04-0.15	Beginning of gallop sound 0.02 - 0.14 after <u>peak</u> of P-wave
Frost, 1949	75	-	0.084 - 0.190 after <u>onset</u> of P-wave
Lian et al.	-	0.06- .008	0.04 - 0.08 after <u>end</u> of P-wave
Mannheimer, 1940	135	-	0.08 - 0.144 after <u>onset</u> of P-wave
Weitzman, 1953	100	-	0.12 - 0.17 after <u>onset</u> of P-wave
Wolferth & Margolies, 1933.	60	-	0.08 - 0.14 after <u>onset</u> of P-wave

Leatham (1949) emphasised the importance of distinguishing between gallop rhythm and physiological splitting of the heart sounds, if necessary by varying the frequency bands of the phonocardiograph - splitting being better appreciated by the higher frequency bands and gallop rhythm by the lower.

Miles (1951), in a study of 100 unselected hypertensives, heard a gallop rhythm in 37. Like Evans, he found a definite tendency for the rapid filling gallop to be associated with right-sided failure. He concluded that although gallop tended to occur more frequently in the severe cases, it was not necessarily of serious prognostic importance and was probably of about the same significance as other factors in clinical assessment, such as heart size, presence of failure, and E.C.G. abnormality. He coined the word "indeterminate" gallop in preference to summation.

The probable mechanism of the physiological third and auricular sounds has been discussed in a previous section, and Kuo, Hildreth and Kay (1951) have produced convincing evidence to suggest that the gallop sounds are produced in a similar way. With the aid of an electrokymograph they were able to record a consistently abnormal upward swing in the left ventricular border coincidental with the gallop sound in four patients with hypertensive failure. If the failure was relieved with digitalis both the gallop and the abnormal ventricular wave disappeared, only to return when treatment was stopped and failure allowed to recur. Such an abnormal ventricular movement was demonstrated with both rapid filling and auricular gallop, and it was also shown that in both types of gallop the sound always occurred just before the peak of the ventricular filling wave at a time when the A-V. valves must be open.

It seems unlikely, therefore, that the extra sound could be due to movement of the valves themselves as suggested by Brady and Taubman (1950). Kuo et al. concluded that in both rapid ventricular filling and auricular gallop there is underlying congestive failure, with a resultant high venous pressure. Rapid ventricular filling results; and the gallop sound is due to the initial impact and sudden stretching effect of the rushing column of blood on the ventricular wall. In the case of auricular gallop they believe that an audible auricular contraction sound may be an added factor in producing the sound. These views as to the probable origin of the diastolic gallop sounds are shared by Wolferth and Margolies (1933), Orias and Braun-Menendez (1939), Mannheim (1942), and other authorities.

Weitzman in 1953 discussed the mechanism, significance, and characteristics of the auricular gallop sound. He suggested that the sound arose as a result of blood entering the ventricles during auricular systole when the ventricular wall was in a state of abnormality either due to hypertrophy or as a result of myocardial infarction, or when the auricular myocardium was itself hypertrophied and abnormal. In Weitzman's series of 100 patients the auricular gallop sound occurred 0.12 to 0.17 second after the onset of P and 0.05 to 0.09 second after the onset of auricular systole. This series of patients consisted of twelve with either pulmonary hypertension or congenital pulmonary stenosis, 46 with hypertension, 29 with myocardial infarction, four aortic valve lesions, and nine with

heart block. A further 205 cases suffering from similar diseases were carefully auscultated for the auricular gallop, and it was not heard. In another 100 normal control subjects no auricular sound could be heard. In the cases of complete heart block the auricular sound appeared split, the first part presumably due to auricular contraction and the second to sudden filling of the ventricle, this latter part being the audible portion of the sound. Weitzman concluded that a fourth sound gallop was never present without clinical, electrocardiographic or X-ray evidence of cardiac abnormality, and that it gave little additional prognostic help. Following myocardial infarction the prognosis was slightly worse if auricular gallop persisted, but he noted that such a gallop might sometimes persist for years without apparent significance.

Brigden and Leatham (1953) have drawn attention to the occurrence of an accentuated rapid filling sound giving rise to a gallop in mitral incompetence, and Besterman (1956) has pointed out the importance of differentiating this sound from a mid-diastolic murmur.

Both Turner (1949) and Evans (1949 and 1951) have emphasised the importance of a proper understanding and clinical appreciation of innocent triple rhythm and the gallop rhythm which occur in heart disease, and the latter has demonstrated the connection between the development of a rapid filling gallop and the onset of heart failure in myocardial infarction.

PRESENTATION OF CASES WITH GALLOP RHYTHM

There have been few comprehensive studies of the phonocardiographic aspects of gallop rhythm in this country, despite the fact that apparatus in current use usually records heart sounds with greater facility than the murmurs. In this thesis particular attention has been paid to the study of gallop rhythm, and a fairly comprehensive and representative series of cases has been recorded.

Recordings from 46 patients with gallop rhythm are presented, and a further 38 of the cases described in other sections have been referred to.

For convenience the section will be divided into three parts. Firstly, cases with rapid filling gallop will be presented; secondly, auricular gallop; and thirdly, summation or indeterminate gallop. The sub-division and classification have been made entirely on the grounds of clinical diagnosis by auscultation and not on the actual phonocardiographic analysis. As a result of this, a certain amount of overlap between the three sections has inevitably occurred.

Those patients thought clinically to have Rapid Filling Gallop

The physiological third heart sound has already been described and its probable mechanism discussed. Unless a specialised technique for calibrating phonocardiographic tracings is employed it appears that a physiological third and a pathological

rapid filling gallop sound cannot be differentiated either from the sound or graphic appearance alone. The pathological gallop is, however, always accompanied by other signs of heart disease or by evidence of systemic disease which, as a result of mechanical or toxic factors, has led to cardiac embarrassment. According to the published literature the pathological third sound of the rapid filling gallop, like the physiological third sound, commences somewhere between 0.10 and 0.20 second after the beginning of the second sound (see Table 8). It also coincides with the sudden fall of the "v" wave in the jugular pulse, the opening of the A-V. valves, and the sudden filling of the ventricles. It cannot, unfortunately, be accurately timed against the E.C.G. tracing because of its position in diastole. The sound may frequently be well heard by placing the ear directly against the chest wall, and may even be felt with the hand. A total of 13 cases of rapid filling gallop are presented here; their essential clinical details and phonocardiographic recordings are discussed. A further 27 cases are referred to, but described in detail in other sections.

The first two patients had classical acute rheumatic fever; a third had chorea with associated rheumatic carditis. All three recordings are presented here.

Case No. 17, the first patient with acute rheumatic fever, was a girl of 5 years. Apart from the joint symptoms and systemic upset she developed a marked tachycardia, an apical systolic murmur, and an audible third sound. After a rapid response to salicylates she made a slow but satisfactory recovery, and when last seen one year later there was no clinical evidence of rheumatic heart disease.

The recordings show a jugular pulse tracing which is unsatisfactory (no proper pulsation could be recorded), a Lead II electrocardiogram (P.R. interval = 0.14'), and an apical sound tracing taken with the Elmquist galvanometer Type I with frequency bands 1, 2 and 3 (Records A, B and C). An early systolic murmur and a rapid filling gallop sound are clearly seen. The extra sound begins about 0.12 second after the beginning of the second sound and it occurs just before the P-wave of the subsequent cardiac cycle. It must, therefore, be independent of auricular contraction. Its relation to the jugular pulse cannot be stated. It appears to be of similar intensity, but of lower frequency, than the second sound. From the tracing alone, or for that matter from the auscultatory findings, this gallop sound could not be differentiated from the physiological third sound. Mannheimer (1940) and Carlgren (1946), by means of their calibrated technique and the criteria they have set up, would probably claim that this differentiation could be made from measurement of the P.C.G. tracing. Without calibration, however, the diagnosis of pathological gallop as opposed to physiological third sound rests on the evidence of accompanying cardiac disease or abnormality.

Case No. 18, a 43-year-old Army sergeant, also had rheumatic fever with active carditis. On auscultation there was a clearly audible systolic murmur at the apex and a rapid filling gallop. At the time of recording the tachycardia and other symptoms had been controlled with salicylate. He was afebrile, but his B.S.R. was 60. There was no evidence of congestive cardiac failure. An electrocardiogram shows a degree of latent heart block (P.R. 0.22').

The phonocardiograph high-frequency recording (A) reveals the loud pan-systolic murmur. Lower frequency bands show the gallop sound occurring about 0.18 second after the beginning of the second sound (B). Its intensity can be seen on the long tracing to vary with respiration (C). An auricular sound can also be seen on the tracing, occurring between the P-wave and the Q.R. complex of the E.C.G. This was not audible and hence does not come into the category of a gallop sound. It is presumably the sound due to auricular systole associated with a prolonged P.R. interval (Evans, 1943). The pan-systolic murmur in this case was presumably due to mitral valve involvement, with resultant incompetence, and is similar to the organic murmur of acute rheumatism described by Besterman (1955). The early systolic murmur in the previous case (No. 17), however, had a slightly delayed onset and is of the type which Besterman and others describe as being benign.

Case No. 19, a male aged 21, had rheumatic chorea, and on physical examination the only positive finding apart from the choreiform movements were an apical systolic murmur and a loud rapid filling gallop. The second sound was split at the pulmonary area and there were no other auscultatory abnormalities. The gallop was maximum at the apex. Although the B.S.R. was normal, there was other evidence of carditis; the pulse-rate was 100 per minute, and there was a latent heart block, the P.R. interval being 0.24'.

Phonocardiograph recordings were taken on two occasions. Firstly (A), when the tachycardia was fairly marked, recordings from the apex showed a clear gallop with the extra sound occurring

about 0.12 second after the beginning of the second sound, but because of the latent heart block the gallop sound is also occurring well after the P-wave. Although the rhythm sounded like a rapid filling gallop, this P.C.G. could not distinguish it from a summation or auricular gallop. Later recordings (C) with the pulse slightly slower and using a higher frequency band, show that there is in fact a quadruple rhythm with both rapid filling and auricular sounds present. No quadruple rhythm was heard, so that presumably the two sounds were heard as one by the ear. A further record (C) shows the same effect shown on the one continuous tracing, by carotid sinus stimulation; as the pulse slows the single ^{gallop} sound becomes split into two. Presumably both of the gallop sounds are due to the rheumatic myocarditis, although a third heart sound could be physiological at this age and the auricular sound may also be related to the presence of latent heart block.

Case No. 20, a male aged 44, had acute nephritis. During the course of the acute phase of his illness a fourth sound gallop was heard, but unfortunately no recordings were made at this time. When recordings were made he had a clear rapid filling gallop which is visible on the tracing, and a splitting of the first sound at the apex. There is also a fairly well-marked auricular component of the first sound occurring well before the QR segment of the E.C.G. P.R. interval is 0.16 second, and the distance between the beginning of the second and the gallop sound is 0.14 to 0.16 sec. An apical systolic murmur is also seen. Tracings were taken on three separate occasions. Record A was taken at the time when the nephritis was active and a gallop rhythm present. The second was

taken three weeks later when the patient was much improved and the gallop and the systolic murmur had disappeared. Record C is shown because its timing is more accurate and it can be conveniently used for measuring time intervals, the time marker measuring ~~an~~ intervals of 0.1 and 0.02 of a second. This patient made an excellent recovery from his illness.

Several patients developed rapid filling gallop following acute myocardial infarction.

Case No. 21, a male aged 56, developed an anterior myocardial infarction, with no previous history of ischaemic pain. During the acute phase of his illness he developed a single paroxysm of auricular flutter, but no congestive failure. His heart rhythm returned to normal spontaneously and he made an excellent recovery. Following the infarction a rapid filling gallop was heard and he had P.C.G. recordings taken on three occasions, all from the apex.

The first record (A) was taken a week after the infarction and shows the gallop sound occurring 0.16' after the beginning of the second sound. Record B, taken using a different microphone and a lower amplitude, shows more clearly an auricular sound in addition to the rapid filling sound. One observer did actually claim that he heard a quadruple rhythm at this time, but his observation was not confirmed by others. (P.R. of E.C.G. = 0.18'). Record C was taken after discharge from hospital two months after the infarction, but a gallop rhythm was still present. This is clearly shown on the P.C.G. and is still due to the rapid filling or pathological third sound. At this stage the patient was free of symptoms and

had no other sign of cardiac failure. The base-line of the tracing is not clear throughout diastole, but no definite auricular sound is seen and no further conclusion can be drawn. Record D was taken four months after Record C, when the patient was back at work as a crane-driver and admitting to no symptoms. On examination he still had a marked triple rhythm, B.P. $\frac{190}{110}$, no evidence of congestive cardiac failure, a moderate degree of left ventricular hypertrophy, and hilar congestion on X-ray examination. This record, taken with the Elmquist machine, shows the rapid filling sound as before; there is also a curious sharp single sound visible between the second and third sound, but this was not heard and its nature is uncertain. There was no clinical, X-ray or other evidence to suggest associated mitral disease. In these latter records the interval between the beginning of the second and the extra sound varies between 0.16 and 0.18 second. Unfortunately no further followup of this patient is available.

Case No. 22, a 40-year-old male, suffered an anterior myocardial infarction and developed a loud gallop rhythm, loudest at the fourth intercostal space near the left sternal edge. There was no sign of congestive cardiac failure. This patient made a very good recovery, but angina of effort persisted, and when he was last seen four years later a gallop rhythm was still present and he was receiving long-term oral anti-coagulant treatment in an effort to prevent further infarction.

Phonocardiograms taken from the left parasternal region at the fourth intercostal space (Record A) shows the gallop sound

occurring 0.14 to 0.16 second after the beginning of the second sound. There is also a clear splitting of the second sound at the pulmonary area (Record B), and an auricular sound was visible on the tracing, but not heard.

Case No. 23, a 48-year-old male, had an anterior myocardial infarction and developed mild congestive cardiac failure, auricular fibrillation, and a partial left bundle branch block. B.P. was $\frac{150}{100}$, and the Q.R.S. interval of the E.C.G. measured 0.10 to 0.12 second. On auscultation a clear gallop rhythm was audible, maximal at the apex. This man made a good recovery, but three months later he was re-admitted with a further myocardial infarction and cardiac failure. He died suddenly with ventricular fibrillation. Post-mortem showed evidence of extensive myocardial infarction, both old and new, and signs of chronic venous congestion in several organs.

Phonocardiograms from the apex showed a rapid filling gallop sound occurring 0.12 to 0.14 after the second sound, clearly seen in the longer diastolic intervals.

Case 24, a 62-year-old housewife, had a posterior myocardial infarction and developed a gallop rhythm. She made a good recovery, and two years later she was alive and well, but still having occasional attacks of angina of effort. At this time she was fibrillating, but there was no gallop to be heard.

Phonocardiograms (which are not shown) confirmed the presence of a rapid filling gallop sound occurring 0.10 to 0.12 second after the onset of the second sound and also an apical systolic murmur.

Cases No. 25 and No. 26 both developed a rapid filling gallop as a result of chronic ischaemic heart disease without the occurrence of acute myocardial infarction. Both had congestive failure.

The first of these two, Case No. 25, was a 70-year-old male with a twelve-month history of congestive cardiac failure. On auscultation the gallop was heard all over the praecordium, but maximal at the apex. He had a left bundle branch block and his blood pressure was $\frac{150}{80}$. (E.C.G. showed Q.R.S. interval to be 0.14'). Conventional treatment relieved the cardiac failure and the gallop disappeared, but one year later he relapsed and died. Post mortem showed gross coronary disease with widespread myocardial fibrosis and considerable cardiomegaly.

Records taken on the first occasion, when the gallop was audible and failure present, show a broad first heart sound with initial low-pitched component coincidental with the Q.R. segment of the E.C.G. and a rapid filling gallop sound occurring 0.2 second after the second sound. (Records A and B). Records taken ten days later (C), when the cardiac failure had been relieved and gallop rhythm could no longer be heard, fail to show the gallop sound. This is despite the fact that the intensity of the first and second sounds and the frequency band used are approximately the same as those in tracing B, where the gallop sound is clearly seen.

The second of these two cases, No. 26, was a 57-year-old housewife. She had no history of angina pectoris and the blood pressure was $\frac{180}{120}$. X-ray screening showed a moderate degree of

of cardiomegaly with some left ventricular enlargement. There was no E.C.G. evidence of left ventricular hypertrophy and no hypertensive change in the optic fundi. Rest in bed, diuretics and digitalis led to considerable improvement and disappearance of the congestive failure. B.P. fell during hospitalisation to readings that were consistently below $\frac{160}{90}$. The patient died at home about a year later, but no further details are available and no post-mortem examination was carried out. Throughout admission a gallop rhythm was heard maximum at the apex.

Apical phonocardiograms (A and B) taken with slow and rapid camera speeds show a clear rapid filling gallop sound coincident with the fall in venous pressure following the "v" wave of the jugular pulse and occurring 0.14 to 0.16 second after the onset of the second sound. The tracing^{is} of the 'congestive' type and does not show a very clearcut pattern, but is clear enough to show that the extra sound is occurring before the "a - c" wave of auricular and ventricular systole. Without this venous pulse tracing it would have been difficult to dissociate the gallop sound from either a summation or auricular gallop because of the apparent coincidence with the P-wave of the E.C.G.

One case with a rapid filling gallop had gross hypertension and atherosclerotic changes with congestive failure. This was Case No. 27, a 59-year-old male who had a four-year history of recurrent episodes of congestive cardiac failure. Four years previously his blood pressure had been $\frac{220}{160}$.

On admission the signs of congestive cardiac failure were present, he had gross cardiomegaly with left ventricular hypertrophy, auricular fibrillation, and a blood pressure of $\frac{160}{110}$. On auscultation at the apex, a systolic murmur and rapid filling gallop were clearly heard. He died four months after admission, having failed to respond to orthodox therapy. At post-mortem, gross left and right ventricular hypertrophy were found, also extreme atherosclerosis of the aorta, and coronary, cerebral, and renal arteries. The myocardium showed evidence of old and recent areas of infarction.

Phonocardiograms confirmed the presence of a rapid filling gallop, and the extra sound was found to follow approximately 0.16 to 0.2 second after the beginning of the second sound. These records have not been mounted.

Case No. 28, a 65-year-old night-watchman, had a posterior myocardial infarction with no previous history of cardiac disease. He was admitted with congestive cardiac failure, but after treatment with conventional methods, including anticoagulants, he made a good recovery. On auscultation no gallop rhythm was heard at any time. Five years later he was still alive, but at the time of enquiry he was in hospital recovering from a further myocardial infarction.

Phonocardiograms show a rapid filling gallop in the apical tracing, although it was not audible. This cannot be classed as a true gallop as the sound was not heard, presumably because it was just below the limits of audibility. The gallop sound occurs between 0.16 and 0.18 second after the onset of the second sound. These records have not been mounted.

Case No. 29 was a 62-year-old housewife with active rheumatoid arthritis. Seventeen years previously she had been treated with radium for thyrotoxicosis, and seven years later had had a sub-total thyroidectomy. Her heart rhythm was known to have been auricular fibrillation for 17 years, but since thyroidectomy she had had no symptoms of thyrotoxicosis. On admission her blood pressure was $\frac{210}{130}$, the left ventricle was moderately enlarged, heart rhythm was auricular fibrillation, rate 65/minute. On auscultation a loud late mitral systolic murmur was heard and there was a clear rapid filling gallop maximum at the apex.

Phonocardiograms taken on two separate occasions confirmed the gallop (Record A), but were of no further assistance in diagnosis. (The gallop sound occurs 0.20 second after the beginning of the second sound). The late systolic murmur can also be seen on Record B.

After a long stay in hospital the rheumatoid went into remission, and during the time she was under observation no evidence of active heart disease or thyrotoxicosis were discovered. The gallop rhythm persisted. Six years later she was alive and well, but with active arthritis. Unfortunately she died shortly afterwards, but although post-mortem was performed the records are very inadequate. Death was certified as being due to acute bronchitis, right-sided cardiac failure and arteriosclerosis. Considerable dilatation of both auricles, dilatation and slight hypertrophy of the right ventricle, and considerable hypertrophy of the left ventricle were found. There was also a functional aortic incompetence, but no valvular lesion.

The coronary vessels were narrowed and calcified. Presumably the gallop rhythm was in fact associated with incipient cardiac failure secondary to either hypertension or ischaemic heart disease. The picture is not entirely clear; although she had auricular fibrillation and a marked hypertension, there were no signs or symptoms of cardiac failure when the recordings were made.

Rapid filling gallop sounds were also either heard or recorded in a number of cases as an incidental finding. These have been discussed in detail in other sections, but will be referred to here. (See Tables 12, 13).

The following young patients with either congenital or rheumatic lesions had a third or rapid filling sound which might have been either physiological or due to their underlying cardiac condition; none of them had evidence of congestive cardiac failure, and the extra sound was not always audible although clearly recorded. Cases Nos. 84, 86 and 88 all had mild congenital heart lesions. Case No. 93 was a young man with subacute bacterial endocarditis and an aortic valvular lesion. Cases Nos. 101, 112 and 127 were young pregnant women with mitral stenosis but only minimal incompetence, and their gallop may in fact have been a physiological third sound occurring as a result of the pregnancy and age.

In addition, a number of patients had a definite rapid filling sound as an incidental finding to a clearcut primary cardiac lesion, usually with associated congestive failure. Detailed descriptions of these cases will be found in the appropriate section. Such were Cases Nos. 87 and 89, with severe congenital heart disease. In both of these cases the gallop sound was recorded but not heard.

Cases Nos. 115, 117, 119, 122 had advanced mitral incompetence, and Case No. 123 had, in addition, active rheumatism. Cases Nos. 101, 103 and 104 developed a transient rapid filling gallop which was visible on the phonocardiogram, but not heard, following mitral valvotomy. Cases No. 77 and 82 are both described in the section dealing with systolic murmurs; in neither case was the gallop audible. Two patients with constrictive pericarditis had a loud audible gallop rhythm and these are dealt with in a special section of the thesis (Cases No. 61 and 62). Cases No. 64 and 73 had bundle-branch block in addition to the gallop rhythm, which was audible only in the latter case. Case No. 92 had severe aortic stenosis and incompetence with congestive cardiac failure and a loud gallop rhythm.

Obviously, all or most of the cases with summation or indeterminate gallop rhythm had a rapid filling sound, but these are described fully in that specific section.

Patients thought clinically to have an Auricular Gallop

It has already been seen that an inaudible vibration may often occur on the phonocardiograph of normal subjects, preceding the Q.R.S. complex of the electrocardiogram and presumably, therefore, due to auricular systole. According to the various published reports referred to earlier, these vibrations may be seen in phonocardiograms recorded from up to 94 % of healthy subjects (See Table 6). In the recordings presented so far in this study, five of the thirteen normal control subjects had such auricular vibrations visible on their phonocardiographic tracings.

By definition, the true auricular gallop sound must be audible and is always accompanied by other signs of heart disease. Unlike the rapid filling gallop which is easily distinguished both clinically and graphically from a split second sound, the auricular gallop may sometimes be close enough to the first sound to be confused with a splitting of the first sound. It is, however, always of a lower pitch and, in contrast to the split sound, may often be palpable as well as audible and heard by direct auscultation with the ear on the chest. When careful auscultation is employed the sound may always be recognised as a separate sound occurring in a rhythm or cadence of three distinct sounds. On the phonocardiograph the auricular sound is separated from the first sound by a short interval, it has a constant relationship to the P-wave of the E.C.G. and the "a" wave of the phlebogram, and it must commence before the Q-wave of the E.C.G.

In the series now under consideration an auricular gallop was heard in 21 patients. The first eight were all suffering from myocardial infarction.

Case No. 30 was a 65-year-old retired policeman. When first seen he had a four-month history of angina pectoris, and eight days before admission he had suffered a posterior myocardial infarction, confirmed by E.C.G. which showed the presence of a latent heart block (P.R. = 0.24'). He made a good recovery but developed a second infarction four months later, and at this time a clearcut gallop rhythm due to an audible auricular sound was heard.

Blood pressure was $\frac{195}{120}$, but there was no congestive cardiac failure. P.C.G. recordings from the apex and aortic area (Record A) demonstrated an auricular sound, loudest at the apex and occurring just at the end of the P-wave of the E.C.G. and well before the Q-wave. In the compensatory pause that follows the occasional ventricular extra-systole another gallop sound (Record B) can be seen. As the rate normally runs about 100/minute, it may well be that there is in fact a summation here between a rapid filling and auricular sound, but the main component is clearly seen to be the auricular sound. The rapid filling sound occurs 0.14 second after the onset of the second sound. It is worth commenting that on auscultation the auricular sound was only well heard at the apex, and even here was not as loud as either the first or second. The P.C.G. taken with low-frequency band (Elmqvist amplifier Type I, band 2) makes the auricular sound to be actually louder than the first sound. This, of course, is because the higher frequencies are better heard by the human ear, and although the low frequency sounds are in fact louder, they are not interpreted as such by the ear, but their correct proportionate intensities are shown on the P.C.G. Four months after these recordings, the gallop was still present, as were the symptoms of angina pectoris. Three years later, despite recurrent attacks of haematuria from a troublesome prostatic hypertrophy, the patient was alive, with no triple rhythm, but having developed auricular fibrillation and with severe limitation of exertion by chest pain and dyspnoea.

Case No. 31, a housewife of 69, suffered a myocardial infarction and developed congestive cardiac failure and a gallop rhythm, the auricular sound being both audible and palpable. Records from the apex on the day of admission were not very satisfactory as the patient was extremely ill, but they show a definite sound occurring before ventricular systole and the Q-wave of the E.C.G. (P.R. = 0.14'). The patient made a good recovery, and two years later was alive and well, still with a gallop rhythm. These records have not been mounted.

Case No. 32, a housewife aged 64, had a history of angina pectoris followed by a definite myocardial infarction. Clinically she had a clear gallop rhythm maximum at the apex due to an extra sound in pre-systole, although she was not in congestive failure. B.P. $\frac{240}{110}$. P.R. = 0.16'. The phonocardiogram (Record A) recorded with the lower frequency band shows a wide first sound with a single coarse vibration, occurring before the E.C.G. Q-wave. The sounds all vary in intensity with respiration. A higher frequency recording (Record B) corresponds to the logarithmic type of tracing and, therefore, the auricular sound appears separated from the first sound as it does to the human ear. There is also a systolic murmur which was not audible. Four years later this patient was alive and well, symptom-free, with no gallop rhythm.

Case No. 33 was a 63-year-old antique dealer who sustained a myocardial infarction and had a previous anginal history of one month's duration. He developed a gallop rhythm with an audible auricular sound maximal at the apex and early congestive cardiac failure, but he made a good recovery with orthodox treatment. When seen three years later he was symptom-free, his E.C.G. was normal, and no gallop was heard. The apical P.C.G. shows a clearcut auricular sound, but unfortunately the Lead II E.C.G. is of very low voltage and poor quality and is not very easy to use as a reference tracing. However, the auricular sound is situated just at the end of the P-wave and well before the Q of the E.C.G. P.R. interval measured on a Cambridge tracing on the same date was 0.18 second.

Case No. 34, a female aged 70, had a three-year history of angina pectoris and a myocardial infarction, with subsequent left ventricular failure. The gallop rhythm was clearly heard maximum at the apex, and the P.C.G. shows an auricular sound occurring in the P.Q. interval of the E.C.G. This sound is of a slightly higher frequency than most of the auricular sounds recorded. (P.R. = 0.16'). B.P. $\frac{175}{110}$. This patient had a further myocardial infarction six weeks later and died in congestive cardiac failure. No post-mortem was obtained.

Cases No. 35 and 36 both had anterior myocardial infarction with left ventricular failure and subsequent development of gallop rhythm. Records are shown of one, No. 35, a 71-year-old woman who was extremely ill at the time of recording; her heart sounds were faint and she could not co-operate to the full. The gallop was loudest at the apex but also well heard at the left sternal edge. Her blood pressure was $\frac{165}{70}$. As a result of the patient's inability to co-operate fully the recordings are not first-class, but careful scrutiny of the tracings recorded at the pulmonary area and the lower left sternal edge reveal a clearcut auricular sound visible on both high and low-frequency tracings and occurring in the P.Q. interval of the E.C.G. (P.R. = 0.14'). She was seen again six months later and was alive and well, but no further followup is available.

Case No. 36 had a loud auricular sound, but by the time he was able to have recordings taken the sound had disappeared and the records were equivocal. He had no evidence of cardiac failure, the P.R. interval on his E.C.G. was prolonged to 0.20 second, and his blood pressure was $\frac{140}{90}$. He made an uninterrupted recovery, and the P.R. interval shortened to 0.14 second. Four years later he was alive and well, B.P. $\frac{220}{110}$, with no further ischaemic symptoms.

Case No. 37, a male aged 61, had a posterior myocardial infarction twelve months before and again one week before admission. He developed what was thought by some to be a gallop rhythm and by others to be a splitting of the first sound. The sound was localised to the apex, easily heard with the ear by direct auscultation; there was an associated mitral systolic murmur and a splitting of the second sound at the pulmonary area. There was no evidence of

congestive cardiac failure at this time, and the blood pressure was $\frac{140}{86}$. He made an uninterrupted recovery and was well and symptom-free on discharge from hospital. His gallop rhythm could no longer be detected on auscultation. Unfortunately he died suddenly at home four months later and no post-mortem examination was made. The phonocardiogram shows no clear gallop rhythm, neither does it show an obvious splitting of the first sound. There is a small low-pitched element of the first sound occurring just before the Q-wave of the E.C.G. and, therefore, an auricular component must be present, but this is no more than may be seen on many normal phonocardiographic tracings. The P.R. interval is 0.14 second.

The next nine patients were all suffering from severe degrees of essential hypertension. The first had malignant hypertension and was aged 45. This was Case No. 38, and he gave a short history of headache and blurring of vision for four months. Phonocardiograms were recorded on three occasions during the course of his illness. On examination initially his resting blood pressure was $\frac{220}{130}$, he had left ventricular hypertrophy confirmed radiologically and on the E.C.G. He had bilateral papilloedema with retinal haemorrhage and exudate, and albuminuria with diminished renal function. The auscultatory findings were as follows. He had a loud gallop rhythm due to an audible auricular sound, heard maximum at the apex, but also all over the praecordium; the second sound was split at the base and the gallop could be heard well by direct auscultation with the ear on the chest.

Phonocardiograms at this time (A) show a clearcut auricular sound preceding the main part of the first sound, but actually occurring coincidental with the Q-wave of the E.C.G. It is demonstrated at the apex and the aortic area, and there is slight splitting of the second sound seen at the pulmonary area, where the recording is somewhat better (B). (P.R. = 0.16'). Treatment with methonium was disappointing and the patient was submitted to a bilateral sympathectomy. Four months later phonocardiograms were again recorded. At this time his blood pressure was lowered and his general condition somewhat improved. The gallop rhythm had now changed to dual rhythm and there was general agreement amongst all observers that the first sound was clearly split, the splitting being maximum at the apex. The P.C.G. (Record C) shows no change in the picture, the extra sound is well demonstrated at the base, but, for technical reasons, not seen at the apex; if anything, the gallop sound is occurring slightly earlier than before and there seems to be no reason why it should not be heard as clearly as it was formerly. The patient's condition improved thereafter and he continued with regular injections of hexamethonium, to which drug he was proving more sensitive after the sympathectomy. Four months after the last recording he still had normal heart sounds on auscultation and his blood pressure was varying around $\frac{160}{100}$. No gallop rhythm was to be heard. A P.C.G. (Record D) recording was made, and it should be noted that the time-marker is different on these later records. Recordings A and B had time marker recording intervals of 0.2 second. C has time marker recording 0.1 and 0.02 of a second, and on these latter tracings no definite auricular sound can be seen. There is a small

vibration coincident with the Q-wave of the E.C.G. but it is within normal limits and quite different from the distinctly separate vibration on Record A. There is no pre-systolic sound in the aortic tracing, as there was before. Three years later this patient was alive and relatively fit, his blood pressure was fairly well controlled, and there had been a definite regression in the hypertensive changes in his fundi, and the left ventricular hypertrophy was much less marked. He had no gallop rhythm. It is not clear why several independent observers should have agreed that initial sounds were those of a gallop and later those of a split first, when the P.C.G. on both occasions shows a similar gallop pattern. It could be that perhaps the intensity of the sound or other non-recordable characteristics were altering and that these characteristics had changed completely by the time the last recordings were made and the gallop no longer present on the tracing.

Case No. 39, a male aged 50, had severe hypertension, with a twelve-month history of recurrent cerebro-vascular accidents and other changes suggestive of the onset of a pre-malignant phase. His blood pressure was $\frac{240}{140}$ at rest, he had marked left ventricular hypertrophy, but good renal function, and only very early arteriolar changes in his fundi. There was no congestive failure and a gallop rhythm was heard. The extra sound was clearly pre-systolic in timing, maximum at the apex, but not palpable. Apical P.C.G. shows the gallop sound to coincide with the "a" wave of the

jugular pulse, and recordings are shown which were taken with the low-frequency band at different intensities and different recording speeds (A and B). (P.R. = 0.16'). Ten months later this patient had yet another cerebro-vascular accident and was not seen again at hospital, but he died shortly afterwards and no post-mortem was obtained.

Case No. 40, a woman aged 48, had severe hypertension with gross left ventricular hypertrophy, B.P. $\frac{260}{160}$, but no renal or retinal changes or congestive failure. Clinically she had a loud gallop rhythm maximum at the apex, but heard all over the praecordium. Recordings show the extra sound occurring in the interval between the P-wave and the Q-wave of the E.C.G. (P.R. = 0.18). She was later submitted to bilateral lumbar sympathectomy with definite benefit, and four years later she was symptom-free, although still hypertensive, and her gallop rhythm had disappeared.

Case No. 41, a woman of 62, suffered from severe hypertension and developed a cerebral thrombosis. She had gross left ventricular hypertrophy and marked retinal but no renal changes. Her blood pressure on admission was $\frac{210}{140}$. On auscultation a loud gallop rhythm was heard maximum down the left sternal edge, but also loud at the apex where there was a systolic murmur. The gallop sound was not palpable. Phonocardiograms were recorded on two occasions. The first (A) is a low-frequency band recording which demonstrates an auricular sound not obviously very distinct from the first sound but quite easily seen and occurring just

before the Q-wave of the E.C.G. and coincident with the "a" wave of the jugular pulse. The P.R. interval is 0.16 second. One month later, with orthodox treatment and bed rest, the patient was much improved and her gallop rhythm had disappeared. At this time further phonocardiograms were taken, and Recording B from a point just internal to the apex does not show a gallop sound. Unfortunately no followup information for this patient is available.

Case No. 42, a male aged 69, had gross left ventricular failure, with a gallop rhythm heard best at the fourth left intercostal space near the sternal edge. After admission to hospital his blood pressure was $\frac{180}{110}$, but fell to $\frac{140}{90}$. E.C.G. showed a latent heart block (P.R. = 0.3), but later he developed paroxysms of auricular flutter. His heart failure was thought to be secondary to hypertensive and ischaemic heart disease. With orthodox treatment he made a good recovery and on discharge the gallop rhythm was no longer audible. Two years later he was re-admitted to hospital with a recurrence of congestive failure and a blood pressure of $\frac{210}{120}$. He died several days later, and post-mortem confirmed the presence of gross cardiomegaly, left ventricular hypertrophy, and myocardial fibrosis with coronary atheroma. Phonocardiograms taken during his first illness show a clearcut auricular gallop sound coinciding with the "a" wave of the phlebogram and the P-wave of the E.C.G. These records have not been mounted.

Cases No. 43 and No. 44 were both recorded early in this series on the Boullitte machine and are not technically very satisfactory; both show auricular gallop sounds, but only one has been mounted.

The first of these two cases, No. 43, a 65-year-old woman, was admitted to hospital with haematuria, uraemia, and an attack of acute glomerulonephritis. She also had a raised blood pressure and evidence of previous hypertension. On examination she had oedema, raised jugular venous pressure, and cyanosis. Her blood pressure was $\frac{230}{130}$. On auscultation there was a clearcut auricular gallop sound and a soft systolic murmur heard at the apex. The aortic second sound was accentuated. She made a good recovery from the nephritis, but on discharge from hospital her blood pressure was still elevated ($\frac{190}{120}$) and she had evidence of impaired renal function and considerable left ventricular enlargement. Phonocardiogram was recorded at a time when she was very ill, but the presence of an auricular gallop is confirmed, the extra sound occurring in the P.Q. interval (P.R. = 0.2'). Unfortunately no followup is available as this patient left the district shortly after discharge from hospital. She was thought to have hypertensive heart disease secondary to chronic glomerulonephritis, with a recent acute exacerbation.

The second of these two cases was No. 44, a man aged 57, who had hypertensive heart disease with a four-year history of recurrent episodes of congestive cardiac failure. More recently he had suffered from disabling intermittent claudication. On examination he was found to have signs of congestive cardiac failure, his blood pressure was $\frac{180}{100}$, and he had gross left ventricular enlargement. There were no peripheral pulses to be felt in the lower limbs. On auscultation an apical systolic murmur and auricular gallop were heard. The gallop sound was loudest at the apex

and left sternal border, but also well heard and palpable high up in the epigastrium. The patient did not respond very well to treatment and died suddenly during an exacerbation of the failure three months later. At post-mortem gross atherosclerosis with occlusive thrombosis of the leg arteries and lower aorta were found. There was also considerable cardiac enlargement with both left and right ventricular hypertrophy, and patches of myocardial fibrosis. A moderate degree of aortic valve sclerosis was also present, and some emphysema. P.C.G. tracings showed a broad first sound with an auricular gallop sound occurring before the Q-wave of the E.C.G. (P.R. = 0.16'). This record has not been mounted; it is very similar to that of Case No. 43.

Case No. 45, a woman aged 48, had hypertensive heart disease with congestive cardiac failure. Clinically she had a loud auricular gallop maximum at the fourth space near the left sternal edge, but also well heard at the apex. The auricular sound was palpable as well as audible. At the aortic area the first sound appeared split and the gallop rhythm was not heard. At the pulmonary area the second sound was split and there was an apical systolic murmur. Apical P.C.G. recorded with the Boulitte shows a clear auricular sound just separated from the main part of the first sound and occurring in the P.Q. interval on the E.C.G. (P.R. = 0.16'). Unfortunately this patient left the district shortly after the recordings were made and no long-term followup is available.

Case No. 46 was a 65-year-old woman with rheumatoid arthritis and left ventricular failure. There was no evidence of peripheral venous congestion, but there were signs of bilateral pulmonary congestion. Clinically and radiologically there was definite left ventricular hypertrophy, but the E.C.G. was normal and the P.R. interval was 0.16 second. B.P. $\frac{170}{100}$, settled with rest and treatment to $\frac{130}{70}$. On auscultation there was a tachycardia, an apical systolic murmur, and an audible gallop rhythm maximal at the fourth left intercostal space near the sternal edge. She improved considerably with treatment, but her joint disability was such that return to full activity was impossible. She died seven years later, but no post mortem was obtained. Phonocardiograms were made on two occasions and show the gallop sound mid-way between the P and Q deflections of the E.C.G. The records are very similar to those of Case No. 45 and have not been mounted.

The remaining four patients fit only into a miscellaneous category, but all had gallop rhythm due to the addition of an auricular sound.

Case No. 47, a woman aged 68, had an audible auricular gallop for no apparent reason. Clinically she had bronchiectasis and no other disease. Her blood pressure was $\frac{110}{60}$ and her heart was normal clinically and radiologically. E.C.G. was normal. There were no signs of heart failure or a history suggestive of heart disease. The auricular sound was loud at the apex, but maximal at the left sternal edge, and could be heard all over the praecordium. It was not palpable. There was also an apical systolic murmur.

Seven years later she was alive and well. P.C.G. recordings were made on two occasions, and the auricular sound is clearly to be seen on both. A representative record is shown. The extra sound occurs just after the end of the P-wave and well before the Q-wave of the E.C.G. and can be seen in tracings taken during both inspiration and expiration. P.R. interval is at the upper limit of normal, being 0.2 second.

Case No. 48, a male aged 26, had acute glomerulonephritis and was a similar case in many ways to Case No. 20. He developed generalised oedema, hypertension, pulmonary basal congestion, albuminuria and haematuria, and a definite gallop rhythm. His E.C.G. at this time also showed changes indicative of myocarditis with inversion of the T-wave in Lead I. The P.R. interval was 0.14 second. The gallop was heard by several observers and was loudest at the apex. He made an excellent recovery, and two weeks later the gallop could no longer be heard, but the same observers all confirmed the presence of a split first sound. P.C.G. recordings were taken on two occasions - firstly, during the acute illness when the gallop was present, and, secondly, after recovery (Records A and B). They do not show the least suspicion of a gallop sound. Both recordings are more or less identical and show a loud first sound with a single low-pitched element occurring with the R-wave, but definitely after Q, much the same type of appearance as is frequently seen in normals. There is also a systolic murmur, but nothing else of note. It may be that the pathological gallop sound in this case has an unusual frequency and must be searched for more arduously with various frequency bands. It was certainly detected on auscultation.

Case No. 49 was a female, aged 47. She presented a difficult clinical problem as she had a long and complicated history of obesity, neurasthenia, spastic colon, and various other psychosomatic disorders. She did, however, complain of exertional dyspnoea and was known to have had at least one attack of paroxysmal tachycardia. As regards the cardiovascular system, there was no evidence of congestive failure, blood pressure was $\frac{150}{90}$, there was a moderate degree of generalised cardiomegaly with a greatly enlarged, vigorously-pulsating right auricle which could be easily seen on the X-ray screen. E.C.G. showed a sinus rhythm with latent heart block, P.R. interval being 0.28 second. Large notched P-waves suggested some auricular hypertrophy.

On auscultation a clear auricular gallop rhythm was heard, maximum at the third and fourth intercostal spaces near the left sternal edge. A doubtful pre-systolic murmur over this same area was also queried.

Phonocardiograms were taken, which require some explanation, as they were made with a demonstration machine which was on loan for a short period. Four tracings are seen on the record. They are, from the top: a jugular pulse wave, a Lead II E.C.G., sound recording from the pulmonary area and from the fourth left parasternal region. The jugular pulse shows giant "a" waves, very small "c", and fairly prominent notched "v" waves. P.C.G. tracings show an extra sound which occurs just at the descending limb of the P-wave and recorded best at the left sternal edge (Record A). It also coincides with the "a" wave of the jugular pulse, but in tracing B it seems almost to precede the "a" wave and to occur at the trough of the pulse tracing between the

"v" wave and the subsequent "a" wave. In view of these findings and the presence of latent heart block, the sound could possibly be a rapid filling gallop or a summation of both rapid filling and auricular gallop. Similar records (C) taken with a lower frequency band with a fast camera speed show the gallop to extend from the middle of the P-wave right on until well after the peak of "a", and it seems to have a much more precise relationship to the events of auricular systole than to the preceding cardiac cycle. Finally, Record D taken with a higher frequency band shows splitting of the first sound, and high frequency vibrations suggestive of a murmur in the period of auricular systole. This does not have the crescendo appearance of the pre-systolic murmur of mitral stenosis, but definitely accompanies auricular systole. The jugular pulse tracing is strongly suggestive of tricuspid stenosis, in the absence of evidence of pulmonary hypertension (Wood, 1950 and 1954). The P.C.G. confirms a gallop rhythm that is probably due to summation of the auricular and rapid filling sound and likely to be associated with auricular hypertrophy, a delayed A-V. conduction time, and an over-active right auricle. There may also be an atypical pre-systolic murmur recorded at the pulmonary area and down the left sternal edge.

Five years after the phonocardiograms were made this patient was still alive and well, having occasional bouts of paroxysmal tachycardia. No more specific details are available, and the precise anatomical diagnosis cannot yet be established.

The last patient with auricular gallop, Case No. 50, was a 45-year-old man with a ten-month history of exertional dyspnoea and a three-week history of paroxysmal attacks of nocturnal dyspnoea. On examination he had no evidence of right-sided failure but he had obvious mitral stenosis, also an auricular gallop rhythm. The E.C.G. showed a right bundle branch block and shortened P.R. interval (Q.R.S. = 0.12'. P.R. = 0.12'. Wolff-Parkinson White syndrome). On auscultation at the apex an auricular gallop was heard, which might at first have been confused with a pre-systolic murmur, but it gave the typical three-tone cadence of a triple rhythm. It was also well heard high up the epigastrium just inferior to the xiphoid process, where it was also clearly palpable. There was a mid-diastolic and a soft systolic murmur to be heard at the apex. No opening snap or splitting of the second sound was heard.

Because of increasing and dangerous attacks of paroxysmal dyspnoea this patient was subjected to mitral valvotomy, and a moderate degree of associated mitral incompetence and a calcified valve were discovered. However, mitral stenosis predominated and a fair splitting of the stenosed valve was obtained, with considerable subsequent benefit. Three years later he was very well indeed, but the gallop rhythm and E.C.G. appearances were unchanged.

Phonocardiograms made before valvotomy were recorded from the apex, the pulmonary area, and the epigastrium; also a jugular venous pulse tracing and the limb lead electrocardiogram.

The recordings show an auricular gallop sound occurring between the P-wave and the Q-wave of the E.C.G. and coincident with the "a" wave of the venous pulse. This is best seen with the lower frequency band recordings (A and B), whereas with the higher frequency band (C) the appearance of the apical tracing is more like that of a pre-systolic murmur, although the vibration of the auricular sound is still visible. The mid-diastolic murmur can be seen, but is not really well-demonstrated, and there is no opening snap.

The gallop sound must be due to auricular systole as it occurs before the Q-wave of the E.C.G. which indicates the start of ventricular systole, and 0.3 second after the preceding second sound. It cannot be a simple audible auricular systolic sound such as occurs in latent heart block because the A-V. conduction time is in fact shortened in this case and not lengthened. The presence of a calcified stenotic mitral valve makes the normal mechanism of auricular gallop most unlikely as the sudden rush of blood from auricle to ventricle would be impossible. A suggested explanation of the sequence of events is as follows. As a result of the degree of mitral stenosis and incompetence there is delay in left auricular systole due to the increased back pressure on the left side of the heart. This is also suggested by the broad, bifid P-waves (Lead I of the E.C.G.) which have a duration of at least 0.12 second,^{and} which, according to Wood (1950) are diagnostic of mitral stenosis and delay in left atrial activity. The right auricle therefore contracts before the left, but because of the intraventricular conduction defect there is delay in contraction of the right ventricle. The

sequence of events would then be - right auricular systole, left auricular systole, left ventricular systole, and, finally, right ventricular systole. In this way the right auricular systolic sound might become visible, as it would precede both right and left ventricular systole, by an abnormally long period of time. Left auricular systole would only result in a pre-systolic murmur because of the mitral valve abnormality, and so a gallop sound would be produced by the right auricle and a pre-systolic murmur by the left. These can both be detected by the different frequency bands recorded from the apex in tracings A and B. It is perhaps not stretching imagination even further to suggest that in the jugular venous pulse tracing there are two ventricular systolic waves - one representing the "c" wave of carotid (i.e., left ventricular systole) pulse and one the systolic pulse wave from the right ventricle conducted up into the neck vein. The duration of both first and second sounds is also greater than normal, although there is no clearcut splitting of either sound. The approximate duration of the first sound is 0.2 second and of the second, 0.1 second.

This case will also be considered again later in both the Bundle-Branch Block and Mitral Valvular Disease sections.

Auricular gallop sounds have also been heard or recorded in a number of cases which are discussed in other sections, as the auricular sound was either not heard at all or was only an incidental finding to the primary abnormality. In six of the 13 patients with rapid filling gallop there was also an auricular

sound to be seen on the tracing; it was inaudible in four (Cases No. 18, 19, 22 and 25), but was probably heard in two (Cases No. 20 and 21). (See Table 15).

In case No. 81 described in the section on cardiac murmurs there was an audible auricular gallop in addition to a loud systolic murmur. In the same section, Case No. 82 had a similar gallop sound seen on the phonocardiograph but not heard on auscultation; also in case No. 89, the gallop was seen, but not heard.

Several patients with bundle-branch-block had an auricular gallop and they are described in that section. In Cases No. 67, 68, and 69 the sound was seen on the phonocardiograph but not heard, but in Cases 63, 64, 65, 66, 70, 71, 72 and 75 the gallop sound was clearly heard on auscultation and seen on the P.C.G. tracing. (See Tables 18 and 19).

Obviously most of the cases with summation or indeterminate gallop had an auricular sound, but these are described fully in that particular section of the thesis.

Patients thought clinically to have a Summation or Indeterminate Gallop

The clinical diagnosis of a summation, or as Miles (1951) prefers to call it, an indeterminate, gallop is a purely arbitrary one. The term "indeterminate gallop" is really preferable because it may be used when, because of tachycardia or other reasons, the observer cannot localise the precise position of the gallop sound in diastole. It may in fact be a rapid filling sound accompanied by such a degree of tachycardia and shortening of diastole that the gallop sound loses its place in the protodiastolic period and becomes mid-diastolic, or even pre-systolic. It may also be due to an auricular sound either associated with tachycardia, or with such a prolonged P.R. interval, in the presence of latent heart block, that the auricular sound loses its place in presystole. Finally, it may be due to a true summation of the two extra sounds, usually as a result of tachycardia, with or without a degree of auriculo-ventricular conduction defect.

With suitable training an experienced listener can often identify the type of gallop rhythm accurately, even in the presence of a moderate degree of tachycardia, but in the cases reported here there was doubt as to the exact nature of the rhythm heard, and, therefore, the description of indeterminate gallop was used. Phonocardiograms always made the differentiation and analysis of these sounds much easier, as accurate measurement of timing with relation to other events in the cardiac cycle could be made. Also, the pulse could often be slowed by vagal stimulation.

It may be that the accurate classification of gallop sounds is important from the prognostic and diagnostic viewpoint and, therefore, phonocardiography can be of practical value. On the other hand, such differentiation may be of academic interest only. Master and Friedman (1942), in an enquiry into the problem of gallop rhythm, found that 6% of their cases with acute myocardial infarction developed a summation gallop. They concluded, as did Mannheim, writing in the same year, and numerous others, both before and since, that summation gallop has a more serious prognostic significance than other forms of gallop rhythm.

A study of nine cases of summation or indeterminate gallop is presented here. A tenth case is included in the section for convenience, as this patient had both rapid filling and auricular gallop at different stages of his illness and his records illustrate important features of this study. A further four cases presented in other sections are referred to here.

The first two cases had Cor Pulmonale from chronic respiratory disease.

Case No. 51 was a 55-years-old male with a long history of recurrent respiratory infection and exertional dyspnoea. He was admitted in gross congestive cardiac failure, the fourth such admission in 14 months. He had finger-clubbing and all the signs of chronic bronchitis with acute exacerbation, severe emphysema, and pulmonary fibrosis. His pulse was regular at 112/minute and his blood pressure was $\frac{110}{70}$. On auscultation there was a gallop rhythm. The gallop was best heard and felt

high up in the epigastrium, also well heard at the apex and all over the praecordium, but not to the right of the sternum. E.C.G. confirmed right ventricular hypertrophy. Despite digitalis, mersalyl, antibiotics and other treatment, the patient died within a few days of admission, and at autopsy the presence of bronchiectasis, emphysema and pulmonary fibrosis with much chronic inflammation was established. The presence of gross Cor Pulmonale with considerable right-sided hypertrophy and dilatation was also confirmed.

The records show a jugular pulse tracing, E.C.G. Lead II, and P.C.G. recording from the apex and the epigastrium. Apical recordings are of poor quality and no gallop is seen, but they are useful for localising the position of the second sound, which is not so clear on the epigastric tracing. The extra sound can be well seen on this latter recording, although the tachycardia makes it difficult to localise (Record A). When the pulse was slowed by vagal stimulation and the diastolic interval increased, the true nature of the extra sound is seen (Record B). It occurs 0.14 second after the second sound and has a constant relationship to the P-waves of the E.C.G. (P.R. = 0.16'). The gallop sound also occurs at the end of the 'v' wave and is not related to the 'a' wave of the jugular pulse.

In this case with Cor Pulmonale and congestive failure the gallop rhythm is in reality a rapid filling gallop. There may, however, be an auricular component making a secondary contribution to the gallop sound, but this is not obvious on the tracings.

Case No. 52, a male aged 57 years, had a two-year history of increasing dyspnoea and productive cough. He was admitted during an acute exacerbation of his chronic chest infection and was found to have congestive cardiac failure with Cor Pulmonale and all its associated clinical, radiological and electrocardiographic signs. The electrocardiogram also showed the presence of a latent heart block (P.R. = 0.26').

A loud gallop rhythm was heard maximal at the left sternal border in the third, fourth and fifth spaces, but also heard at the apex and high up in the epigastrium. In addition there was a loud high-pitched apical systolic murmur. Despite energetic treatment this patient died two months later and at post-mortem the diagnosis of Cor-Pulmonale, chronic bronchitis, and emphysema with pulmonary arteriosclerotic changes was confirmed. The right ventricle was grossly hypertrophied and both right and left auricles dilated.

Phonocardiograms were made on two occasions. Three of the tracings are mounted, recorded with different frequency bands and both camera recording speeds. The gallop sound is seen to be occurring in mid-diastole 0.20 second after the beginning of the second sound, but because of the prolonged P.R. interval it is also situated just after the P-wave of the E.C.G. (Records A and B). Unfortunately the jugular pulse tracing is poor, but it does give the impression that the sound occurs at about the time of the fall of the 'v' wave. Record C taken from the cardiac apex with a higher frequency band shows the systolic murmur. This gallop should clearly be classified as indeterminate as it may in fact be due to either auricular, or rapid filling sounds or to a summation of both.

Four patients had hypertensive heart disease with an indeterminate gallop rhythm.

Case No. 53 was a 59-year-old male who had been hypertensive for at least 12 years. Shortly before admission to hospital he had a single attack of paroxysmal nocturnal dyspnoea. On examination he had a slight pulmonary congestion (visible as hilar shadow on the X-ray screen) but otherwise no evidence of congestive failure; gross left ventricular hypertrophy (confirmed by E.C.G.), and early hypertensive changes in the retinal vessels. There was no evidence of renal damage and the resting B.P. was $\frac{290}{140}$.

On auscultation a gallop rhythm was heard at the apex. Recordings were made on two occasions and a clearcut gallop was seen on the first record (A). Unfortunately, because of the tachycardia, the exact nature of the gallop could not be shown. Later recordings were made when the heart-rate had slowed (B), and recordings were also made during carotid sinus stimulation (C and D). At the slower rates the gallop sound can be recognised as a rapid filling or pathological third heart sound, occurring 0.2 second after the second sound. The P.R. interval is 0.16 second. A curious change in the shape of the P-wave is also noted during carotid sinus pressure and the inaudible but visible auricular component of the first sound also seems to disappear. Actually the early auricular vibrations of the first sound are unusually clear and occur well before the Q-wave of the E.C.G. Nevertheless, they were not audible, did not constitute an auricular gallop, and did not apparently play any part in forming a true summation. The gallop rhythm disappeared with oral hypotensive therapy and the blood pressure fell.

Four years after these recordings were made the patient was still being treated with ganglion blocking agents, still had gross left ventricular hypertrophy, but no gallop rhythm, and his blood pressure was being maintained at about $\frac{170}{90}$ for most of the day.

Case No. 54, a housewife aged 49, was admitted in congestive cardiac failure. She had albuminuria, retinal changes, and gross cardiac enlargement, with malignant hypertension (B.P. $\frac{240}{130}$). On clinical examination a definite gallop rhythm was heard maximum at the apex. It was impossible to time accurately because of tachycardia, and it was, therefore, classified as an indeterminate gallop. Medical treatment brought about an improvement, but the patient relapsed on several occasions and ultimately became uraemic and died three years later. At post-mortem gross bilateral pyelonephritis was found, with considerable cardiomegaly.

Recordings of the gallop rhythm were made on two occasions. A representative record is shown and the loud gallop sound can be seen, apparently consisting of two complexes - the first 0.16 second after the beginning of the second sound and coincident with the peak of the P-wave (P.R. = .20'), and the second occurring in the P.Q. interval. Clearly, a true summation gallop and probably the two elements that can be seen are in fact the rapid filling and auricular sounds.

Case No. 55, a woman aged 43, also had severe essential hypertension in the pre-malignant phase, with a history of paroxysmal nocturnal dyspnoea, angina of effort, and congestive cardiac failure. She had considerable left ventricular hypertrophy (confirmed by E.C.G.) and early hypertensive retinopathy, but no albuminuria (B.P. $\frac{230}{140}$).

Clinically there was a loud gallop rhythm heard loudest at the apex - thought by some to be a rapid filling sound, but tachycardia made it impossible to be certain as to its precise nature. There was also a basal systolic murmur. Despite vigorous hypotensive therapy this patient died of a cerebral haemorrhage eighteen months later. At post-mortem the presence of cerebral haemorrhage was confirmed, and gross left ventricular hypertrophy, myocardial fibrosis, hypertensive vascular changes, and evidence of old pyelonephritis were all found. The gallop rhythm persisted up to the time of death.

Phonocardiograms were taken from the apex and aortic areas; probable auricular and rapid filling sounds were both seen in the recordings but were not very clear (Record A). During carotid sinus stimulation, however, the heart-rate was slowed sufficiently to separate the two components quite distinctly, and as the rate quickened after stimulation the two sounds merge into summation once again (Record B). The rapid filling component occurs 0.14 second to 0.16 second after the beginning of the second sound, and the auricular component is seen in the P.Q. interval of the E.C.G. (P.R. = 0.16').

Case No. 56, a male aged 47, had severe essential hypertension, with a history of headache and exertional dyspnoea, but no chest pain. He had gross left ventricular hypertrophy, signs of early left ventricular failure, but no frank congestive failure. His blood pressure was $\frac{240}{130}$, and there were signs of considerable impairment of renal function. He was known to have had a raised blood pressure for at least two years. A loud gallop rhythm was audible maximum at and internal to the apex, not palpable, but very clearly heard, and thought by some to be a rapid filling gallop.

There was also an apical systolic murmur. He was submitted to bilateral lumbar sympathectomy and the operation was successfully carried out on the left side. Ten days later, during the right-sided sympathectomy, cardiac arrest suddenly occurred, and although the heart was started again by cardiac massage, irreversible damage must have been done, because the patient died several hours later without recovering consciousness. At autopsy gross left ventricular hypertrophy and dilatation were found. There was also severe coronary, cerebral and aortic atheroma, the coronary vessel lumen being reduced to pin-point size at several points. There was also evidence of multiple small myocardial infarctions, both old and new.

Phonocardiograms were taken on two separate occasions and various recordings are shown. In the first recordings there is a fairly marked tachycardia, and the gallop sound is very clearly seen occurring at the same time as the P-wave of the E.C.G. (P.R. = 0.16'), but commencing actually just before it. The jugular pulse is of the type often associated with tachycardia, and the 'a' and 'v' waves are partially fused. Hence, there is no clear 'v' wave, but the gallop sound does in fact occur at the point where there is a small negative deflection in the phlebogram between the 'v' and 'a' waves. The gallop sound commences about 0.16 second after the preceding second sound. After one week the gallop rhythm was much less marked and could only be heard with difficulty at the apex when it sounded like an auricular gallop. This was confirmed phonocardiographically in records C and D, corresponding in frequency ranges to A and B. There is now a clearcut auricular sound coinciding with the P-wave of the E.C.G. and 'a' wave of the jugular pulse, but much closer to the first sound than the gallop sound in earlier records.

There is also a rapid filling gallop sound to be seen as well, occurring about 0.16 second after the second sound and coincident with the flattening out of the 'v' wave in the phlebogram. This suggests that the gallop was originally due to summation of the two sounds, but with rest in bed, diminution of the heart-rate and relief of the left ventricular failure, the auricular and rapid filling sounds have separated and only the former remains audible. Finally, a record (E) was taken during carotid sinus stimulation just after record D was taken. A period of complete asystole has occurred which lasted for several seconds; then there is a single auricular systole, followed by a gradual picking up of the sinus beats. The auricular systole gives a very atypical E.C.G. complex, but the P.C.G. vibrations look very much like an auricular sound and the first half corresponds closely with the auricular part of the subsequent heart sounds. There are no visible rapid filling sounds, but unfortunately the intensity of the amplifier is really too low to show very much. The auricular gallop sound, however, remains clearly seen.

In this case, therefore, a patient with hypertension and early left ventricular failure has a definite summation gallop which disappeared as the failure improved and the heart-rate fell. The components of the gallop became separated, but the auricular component probably remained to give a presystolic gallop rhythm.

The next two patients both had ischaemic heart disease. The first, Case No. 57, a male aged 60, had a recent posterior myocardial infarction and a four-year history of angina pectoris.

On admission following the infarction he had signs of early congestive cardiac failure, his blood pressure was $\frac{170}{110}$, and an indeterminate gallop rhythm was heard, loudest at the apex. The patient made a rapid recovery, and three weeks later all signs of cardiac failure were gone and the gallop rhythm was no longer audible. Eighteen months later, however, he died of a further presumed myocardial infarction, but no post-mortem was carried out.

Recordings were made on two occasions. Shortly after admission Record A shows a definite rapid filling gallop, the extra sound occurring 0.18 second after the beginning of the second sound and well before the P-wave of the E.C.G. (P.R. = 0.14 to 0.16'). There are also a few low-pitched vibrations immediately preceding the first sound and coincident with the Q-wave of the E.C.G., but these do not have the appearance of a distinct sound and are within the limits of normal. Records taken a month later when the patient was recovered do not show any gallop sound and neither was a gallop rhythm heard at this stage (Record B). The small immediate pre-systolic vibrations are relatively unchanged.

The second patient, No. 58, was a male aged 66. He had a twelve-month history of increasing breathlessness on exertion, paroxysmal nocturnal dyspnoea, and discomfort in the chest related to exertion. He was known to have been hypertensive in the past. On examination he had congestive cardiac failure, tachycardia, an enlarged left ventricle, and a B.P. of $\frac{130}{90}$. His fundi were normal and he had no albuminuria. X-ray of the chest showed an unfolded aorta and enlarged left ventricle.

E.C.G. showed a latent heart block (P.R. = 0.22') and a minor degree of intraventricular conduction defect. A loud gallop rhythm was heard all over the praecardium but maximum at the apex. There were no murmurs. The precise nature of the gallop was indeterminate; some observers thought it to be due to an added rapid filling sound. The patient made an excellent recovery with digitalis and mersalyl, but the gallop rhythm persisted. He was seen again six months later and was relatively well, with no failure and no further attacks of paroxysmal nocturnal dyspnoea. The gallop rhythm was still present. He died at home eighteen months after the recordings were made and no further details are available.

The recordings show a gallop sound following constantly 0.16 second after the beginning of the second sound and varying slightly in its relation to the P-wave in different records, but nevertheless always occurring just after the beginning of P., presumably because of the latent heart block. This may, therefore, be a true summation, but it seems more likely to be a rapid filling sound, which persisted for eighteen months and right up to the time of death.

The next patient also had myocardial ischaemia, but of a rather different nature and origin. He was Case No. 59, a 50-year-old male, who had a two-year history of angina pectoris and shortly before admission developed chest pain at rest. On examination he was in congestive cardiac failure, his B.P. was $\frac{104}{90}$, and he had a low volume pulse and tachycardia, but was in normal rhythm. There was gross left ventricular hypertrophy and a loud gallop rhythm maximum at and internal to the cardiac

apex. The aortic second sound was faint. There were both apical and basal systolic murmurs, but no thrills, and the basal systolic murmur was not conducted into the neck. E.C.G. showed both the changes of acute ischaemia and of gross left ventricular hypertrophy (P.R. = 0.16'). The patient initially made a slight improvement with diuretics and digitalis, but died a few days later. At post-mortem he was found to have an extreme degree of rheumatic calcified aortic stenosis, with only a pin-hole opening remaining in the deformed aortic valve. There was also considerable left ventricular hypertrophy, but the myocardium, the coronary vessels, and the other valves were normal.

Apical low-frequency P.C.G. recorded just after admission when the gallop was clear, shows a first sound and an auricular sound occurring in the P-Q interval of the E.C.G. The second sound is very indistinct, but can be seen occurring just at the end of the T wave of the E.C.G. Unfortunately no recordings from the pulmonary area which might have shown up the pulmonary second sound are available. There was clearly no aortic component to the second sound. When a ventricular extra-systole occurs there is no gallop sound to be seen in the auricular systolic period, but a rapid filling gallop sound follows the extra-systolic beat, occurring well before the next P wave and about 0.1 second after the preceding second sound. The gallop sound is, therefore, mostly due to an audible auricular sound, but may in fact be a summation of the auricular sound and the rapid filling sound.

The last patient, a male aged 64 (Case No. 60), does not really fall into the category of indeterminate gallop, but he had both auricular and rapid filling gallop recorded at various times, and will be discussed here.

He had been hypertensive for many years, and before admission he had several attacks of paroxysmal nocturnal dyspnoea. His B.P. was $\frac{200}{80}$, and he was in normal sinus rhythm, he had gross aortic atheroma with calcification and well-marked left ventricular hypertrophy, confirmed by X-ray and E.C.G. (P.R. = 0.18 second). He was also thought to have an aortic valvular lesion, but no definite early diastolic murmur was ever heard, although he had a systolic murmur at both apex and aortic area. No thrills were felt. A loud gallop was heard and thought to be due to an added auricular sound. P.C.G. recordings were taken at this time and on two subsequent occasions. The first recordings (A) were made on the original Boulitte machine when no time-marker was available, and the tracing is taken from the pulmonary area. A small, but definite auricular sound can be seen occurring in the P-Q interval. There is a systolic, but no evidence of a diastolic murmur. Six months later he went into cardiac failure with the onset of auricular fibrillation. However, some observers still thought that a pre-systolic sound could be heard and also palpated. Others thought the first sound to be split. The systolic murmur was loudest^{er} than before and was heard at both apex and aortic area. There was no early diastolic murmur and no audible third heart sound.

Recordings were taken at this time from the left sternal edge, the pulmonary area, and the apex, but naturally show no auricular sound. The recordings from the left sternal edge over the fourth intercostal space (Record B) do show a small low-frequency vibration immediately following the Q wave of the E.C.G. and which must be due to ventricular systole.

This may have given rise to the auditory impression of splitting of the sound. Following the second sound there are a few irregular vibrations of doubtful origin, possibly an early diastolic murmur, but they appear to be of much too low frequency for this. At the same time, apical tracings (C) show a rapid filling sound occurring approximately 0.16 second after the second sound. This man made a good recovery, but twelve months later congestive cardiac failure developed once again and he was re-admitted. At this time fibrillation was still present, but a definite rapid filling gallop was heard, and a splitting of the second sound at the pulmonary area. No auricular sound or splitting of the first sound was heard.

Recordings made with the Elmquist machine, which had by now replaced the Boulitte, confirm the presence of clear cut splitting of the second sound, most marked at the pulmonary area (Record D), and also the rapid filling gallop sound (Record E). No other definite abnormalities were seen, but the recordings are not of a high standard. For this reason no very definite conclusion can be drawn from these records. The disappearance of an auricular sound with the onset of auricular fibrillation is clearly demonstrated, but there was still some confusion in the interpretation of clinical signs. This was aided by the recordings, and with the onset of cardiac failure a rapid filling gallop developed which persisted for some time. Unfortunately there is no initial recording from the apex or lower left sternal edge, and a rapid filling gallop sound might well have been demonstrated then if looked for. It was certainly not heard. Two years later this patient was still alive, but he then left the district and no further followup is available.

Indeterminate gallop has already been discussed briefly in connection with Cases No. 30 and No. 50, where marked tachycardia due to severe illness, or the presence of latent heart block complicated the picture. In both of these cases the gallop sound was ultimately correctly identified by auscultation as an auricular sound, and the cases are, therefore, reported in that section. Indeterminate gallop is also referred to in Cases Nos. 98 and 124 in a later section which deals with mitral valve disease. (See Tables 13, 14, 16, 19,).
21 and 22

TABLE 11

Patients with Audible Rapid Filling Gallop described in this section

Case No.	Sex	Age	Time Interval between beginning of 2nd and 3rd sounds (in seconds)	P.R. Interval of E.C.G. (seconds)	Blood Pressure	Period of Followup	Diagnosis and Remarks
17	F.	5	0.12	0.14	110/70	1 Year. Alive	Acute Rheumatic Fever. No C.C.F.
18	M.	43	0.18	0.22	120/75	-	Acute Rheumatic Fever. No C.C.F. ++
19	M.	21	0.12	0.24	120/60	-	Rheumatic chorea. No C.C.F. ++
20	M.	44	0.14 - 0.16	0.16	110/70	-	Acute glomerulo-nephritis. No C.C.F. ++
21	M.	56	0.16	0.18	190/110	6 mths. Alive	Myocardial infarction. No C.C.F. ++
22	M.	40	0.14 - 0.16	0.18	?	4 yrs. Alive	Myocardial infarction. No C.C.F. ++
23	M.	48	0.12 - 0.14	-	150/100	3 mths. Died	Myocardial infarction. Bundle-branch block. Auricular fibrillation. C.C.F.
24	F.	62	0.10 - 0.12	0.18	?	2 yrs. Alive	Myocardial infarction. No C.C.F. Two years later had auricular fibrillation and gallop still.
25	M.	70	0.2	0.18	150/80	1 Year. Died	Chronic ischaemic heart disease. C.C.F. Bundle-branch block. ++
26	F.	57	0.14 - 0.16	0.20	180/120	1 Year. Died	Chronic ischaemic heart disease. C.C.F. B.P. fell to 160/90 with rest in bed.
27	M.	59	0.16 - 0.20	-	220/160	4 mths. Died.	Ischaemic and hypertensive heart disease. C.C.F. Auricular fibrillation. B.P. fell to 160/110.
29	F.	62	0.20	-	210/130	6 yrs. Died	Rheumatoid arthritis and previous thyrotoxicosis. Auricular fibrillation. No C.C.F.

++ Auricular gallop also seen on P.C.G. and possibly heard as well in Cases 20 and 21.
 C.C.F. = Congestive cardiac failure.

TABLE 12

Patients with Audible Rapid Filling Gallop described in other sections

Case No.	Sex	Age	Time Interval between beginning of 2nd and 3rd sounds	P.R. Interval of E.C.G.	Blood Pressure	Period of Followup	Diagnosis and Remarks
61	F.	46	(second) +0.14 - 0.16	(second) -	120/78	1 year. Alive	Constrictive pericarditis. Auricular fibrillation. C.C.F.
62	F.	19	0.10 - 0.12	0.28	115/80	?	Constrictive pericarditis. C.C.F.
73	M.	69	0.10 - 0.12	0.22	?	?	Ischaemic heart disease. C.C.F. ϕ
86	F.	19	0.16	0.16	110/70	4 yrs. Alive	Pulmonary stenosis. No C.C.F.
88	F.	6	0.10	0.16	?	4 yrs. Alive	Probable aortic stenosis. No C.C.F.
92	M.	53	0.16	-	105/85	5 weeks Died	Aortic stenosis and incompetence. C.C.F.
112	F.	29	0.14 - 0.16	0.16	125/80	?	Mitral stenosis (mild). No C.C.F. Pregnant.
113	M.	52	0.16	0.18	?	4 yrs. Alive	Mitral stenosis. Myocardial infarction. C.C.F.
117	F.	54	0.10 - 0.14	-	?	1 year Died	Mitral stenosis and incompetence. C.C.F. Auricular fibrillation.
119	F.	63	0.14 - 0.16	-	?	? Died	Mitral stenosis and incompetence. C.C.F. Auricular fibrillation.
123	F.	13	0.14	0.16	?	?	Rheumatic chorea. Early mitral stenosis. No C.C.F.

+ After pericardectomy, gallop sound was 0.16 - 0.18 after the second sound. ~~See also Case 60 in Table 16.~~

ϕ Also referred to in Table 19.
See also Case 60 in Table 16.

TABLE 13

Patients with Rapid Filling Gallop Sounds Recorded on P.C.G.
but not heard

Case No.	Sex	Age	Time interval between beginning of 2nd and 3rd sound (in seconds)	P.R. interval on E.C.G. (seconds)	Blood Pressure	Period of Followup	Diagnosis and Remarks
28	M.	65	0.16 - 0.18	-	?	5 yrs. Alive	Myocardial infarction. Auricular fibrillation. Congestive cardiac failure.
x30	M.	65	0.14	0.24	195/120	4 yrs. Alive	Myocardial infarction. Hypertension. No Congestive cardiac failure.
*64	F.	54	0.20	0.14	210/130	5 yrs. Alive	Myocardial infarction. Hypertension. Bundle-branch block. Left ventricular failure.
77	F.	49	0.15	0.20	?	3 yrs. Alive	Rheumatoid arthritis. Cardiac neurosis. No Congestive cardiac failure.
82	M.	54	0.10	0.16	?	3 yrs. Alive	Myocardial infarction. Congestive cardiac failure.
84	F.	7	0.14 - 0.16	0.16	130/40	4 yrs. Alive	Patent ductus arteriosus. No Congestive cardiac failure.
87	F.	61	0.22	-	?	?	Ventricular septal defect. Bundle-branch block. Congestive cardiac failure. Auricular fibrillation.
89	F.	62	Not measurable.	0.20	?	5 yrs. Alive	Atrial septal defect. Early congestive cardiac failure. Also had visible auricular gallop on P.C.G.
93	M.	27	-do-	Not measurable	?	5 yrs. Alive	Subacute bacterial endocarditis. No congestive cardiac failure.
101	F.	27	0.12	0.16	?	17 mths. Alive.	Mitral stenosis. Left ventricular failure. ++
103	M.	20	0.18	0.18	?	25 mths. Alive.	Mitral stenosis, with slight incompetence. ++
104	F.	30	0.16	0.18	?	15 mths. Alive.	Mitral stenosis. Left ventricular failure. ++
115	M.	50	0.14	-	-	4 mths. Died.	Mitral stenosis and incompetence. Auricular fibrillation. Cardiac congestive failure.
122	F.	17	0.14 - 0.16	0.16	?	2 yrs. Died.	Mitral stenosis and incompetence. Early Congestive cardiac failure.
127	F.	23	0.14	-	?	?	Mitral stenosis and aortic incompetence. No Congestive cardiac failure. Pregnant.

x Also included in Table ___.

++ All these records were made shortly after a successful valvotomy.

* Also included in Table ___.

TABLE 14

Patients with an Audible Auricular Gallop
described in this section

Case No.	Sex	Age	P.R. interval (seconds)	Blood Pressure	Period of Followup	Diagnosis and Remarks
*30	M.	65	0.24	195/120	4 yrs.	Myocardial infarction. Hypertension. No C.C.F.
31	F.	69	0.14	?	2 yrs. Alive	Myocardial infarction. C.C.F. Palpable gallop.
32	F.	64	0.16	240/110	4 yrs. Alive	Myocardial infarction. Hypertension. No C.C.F.
33	M.	63	0.18	?	3 yrs. Alive	Myocardial infarction. C.C.F.
34	F.	70	0.16	175/110	6 wks. Died	Myocardial infarction. C.C.F.
35	F.	71	0.14	165/90	6 mths. Alive	Myocardial infarction. L.V.F.
x36	M.	?	0.20	140/90	4 yrs. Alive	Myocardial infarction. No C.C.F.
x37	M.	61	0.14	140/80	4 mths. Alive	Myocardial infarction. No C.C.F. ? split 1st sound
38	M.	45	0.16	220/130	3 yrs. Alive	Malignant hypertension. No C.C.F. Gallop disappeared with treatment.
39	M.	50	0.16	240/140	12 mths. Died	Hypertension. No C.C.F.
40	F.	48	0.18	260/160	4 yrs. Alive	Hypertension. No C.C.F. Sympathectomy.
41	F.	62	0.16	210/140	6 mths. Alive	Hypertension. No C.C.F.
42	M.	69	0.3	210/120	2 yrs. Died	Hypertension. C.C.F.
43	F.	65	0.2	230/130		Hypertension. Nephritis. C.C.F.
44	M.	57	0.16	180/105	3 mths. Died	Hypertension. C.C.F. Palpable gallop.
45	F.	48	0.16	?	-	Hypertension. C.C.F. Palpable gallop.
46	F.	65	0.16	170/100	7 yrs. Died	Hypertension. L.V.F. Palpable gallop.
47	F.	68	0.20	110/60	7 yrs. Alive	Bronchiectasis. No C.C.F.
x48	M.	26	0.14	120/90	-	Acute nephritis. No C.C.F.
49	F.	47	0.28	150/90		? Tricuspid stenosis. No C.C.F.

* Also included in Table 13.

x Gallop heard but not demonstrated on P.C.G.

C.C.F. = Congestive cardiac failure

L.V.F. = Left ventricular failure

TABLE 15

Additional Cases With Auricular Gallop Sound
either heard or recorded on P.C.G.

Case No.	Sex	Age	P.R. interval (seconds)	Blood Pressure	Period of Followup	Diagnosis and Remarks
*18	M.	43	0.22	120/75	-	Acute rheumatic fever. No C.C.F.
*19	M.	21	0.24	120/60	-	Rheumatic chorea. No C.C.F.
*20	M.	44	0.16	110/70	-	Acute nephritis. No.C.C.F.
*21	M.	56	0.18	190/110	6 months Alive	Myocardial infarction. No C.C.F.
*22	M.	40	0.18	?	4 years Alive	Myocardial infarction. No C.C.F.
*25	M.	70	0.18	150/80	1 year Died	Ischaemic heart disease. C.C.F.
81	M.	76	0.16	?	2 weeks Died	Carcinoma of Lung with cardiac displacement. No C.C.F. Audible gallop.
^x 82	M.	54	0.16	?	3 years Alive	Myocardial infarction. C.C.F.
x89	F.	62	0.20	?	5 years Alive	Atrial septal defect. Early C.C.F.

See also Tables 18 and 19 for further cases of auricular gallop with bundle-branch block.

* Also included in Table 11 - auricular gallop probably not heard in Cases 18, 19, 22 and 25, but possibly heard in Cases 20 and 21.

x Also included in Table 13 - gallop sound not heard, but seen on P.C.G.

C.C.F. = Congestive cardiac failure.

TABLE 16

Cases of Indeterminate Gallop

Case No.	Sex	Age	Time Interval between beginning of second sound & gallop (in seconds)	P.R. Interval (seconds)	Blood Pressure	Period of Followup	Diagnosis and Remarks
51	M.	55	0.14	0.16	110/70	3 days. Died.	Cor pulmonale. C.C.F. ++
52	M.	57	0.20	0.26	-	2 mths. Died.	Cor pulmonale. C.C.F. ++
53	M.	59	0.20	0.16	290/140	4 yrs. Alive	Hypertension. Left ventricular failure. Gallop disappeared with therapy.
54	F.	49	0.16	0.20	240/130	3 yrs. Died	Malignant hypertension. C.C.F.
55	F.	43	0.14 - 0.16	0.16	230/140	18 mths. Died.	Hypertension. C.C.F.
56	M.	47	0.16	0.16	240/130	1 month. Died	Hypertension. Left ventricular failure. Died during sympathectomy.
57	M.	60	0.18	0.16	170/110	18 mths. Died	Myocardial infarction. C.C.F.
58	M.	60	0.16	0.22	130/90	18 mths. Died.	Ischaemic heart disease. C.C.F.
59	M.	50	Approx. 0.10	0.16	104/90	3 days. Died	Aortic stenosis. C.C.F.
124	M.	68	0.16	0.36	170/90	? six mths. Died	Ischaemic heart disease. C.C.F. Aortic and probable Mitral valvular disease.
60	M.	64	0.16	0.18	200/80	2 yrs. Alive	Hypertension. C.C.F. Not really an indeterminate gallop. Both auricular and rapid filling sounds present at different times. See text.

++ Gallop sound palpable in the epigastrium.

C.C.F. = Congestive cardiac failure.

TABLE 17

THE AETIOLOGY OF AUDIBLE GALLOP RHYTHMS
ENCOUNTERED IN THIS SERIES

TYPE OF HEART DISEASE	No. of Cases with Rapid Filling Gallop	No. of Cases with Auricular Gallop	No. of Cases with Indeterminate Gallop	TOTAL
Predominantly ischaemic heart disease	7	14	3	24
Predominantly hypertensive heart disease	2	15 †	4	21
Rheumatic heart disease	10 *	-	1	11
Cor pulmonale	-	-	2	2
Constrictive pericarditis	2	-	-	2
Thyrotoxicosis, acute nephritis, congenital heart disease, miscellaneous and undiagnosed	3	2	-	5

† Four other patients also had unequivocal evidence of ischaemic heart disease in addition to established hypertension

* One of these patients may have had a physiological third heart sound and not a gallop

SUMMARY AND CONCLUSIONS

A gallop rhythm may frequently develop as a result of the appearance of an audible sound in diastole. This may be an auricular systolic sound, a rapid ventricular filling sound or a summation of the two. The literature, probable mechanism and clinical significance of these gallop sounds has been reviewed and discussed.

The clinical details of 46 patients with gallop rhythm have been described in this section and a selection of the phonocardiographic recordings taken from them are presented. A further 38 cases and phonocardiograms from them are also referred to. In many cases serial records are shown and details of followup are given whenever available. The salient features of all the cases with gallop rhythm encountered in this study, including those whose clinical details are described in other sections are set out in the accompanying tables and summarised here. Tables 11 and 12 show the cases with rapid filling gallop, Tables 14 and 15 show those with auricular gallop. Other examples of auricular gallop occurring in the presence of bundle-branch block appear in Tables 18 and 19. Table 16 shows the essential features of cases with indeterminate or summation gallop. As some patients had both auricular and rapid filling gallop sounds recorded, there is a certain amount of overlap between these Tables, but whenever a case appears in more than one table this fact is indicated and the cross-reference provided. Although gallop rhythm is a purely clinical diagnosis and all such sounds are, therefore, audible on auscultation, those cases with heart disease and recordable gallop

rhythm which was not heard are also shown in separate tables and considered in the text and final analysis. A clear distinction has been made, however, between them and audible gallop rhythm.

As a result of this study it has been possible to make a number of observations on the occurrence and significance of gallop rhythm.

1. The view that the auricular and rapid filling gallop sounds have approximately the same physical characteristics as the corresponding physiological sounds and occupy the same position in the cardiac cycle is confirmed.

With the techniques employed in this study, the phonocardiographic appearance and character of the physiological third sound and the rapid filling gallop sound are identical. So is their relationship to the second heart sound and other events in the cardiac cycle such as the deflection of the jugular pulse tracing. Where an adequate phlebogram is available and the venous pulse-waves not obscured or altered by tachycardia or venous congestion the gallop sound appears at or about the time of rapid ventricular filling, coincident with the negative deflection at the end of the "v" wave. According to the literature the physiological third sound commences between 0.10 ' and 0.20 ' after the beginning of the second sound (Table 3). The corresponding time interval for the rapid filling gallop sound is reported as being the same (Table 8). In this study similar measurements for the interval between the beginning of the second and physiological

third sounds were found to be from 0.12 ' to 0.20 ' and the corresponding interval for the rapid filling gallop varied from 0.10 ' to 0.22 ', usually from 0.12 ' to 0.18 ' (Tables 11,12 & 13).

The direct relationship between the auricular gallop sound and auricular systole has also been confirmed graphically. In several cases the sound has disappeared either with the onset of auricular fibrillation or during ventricular extrasystolic beats. The auricular gallop sound has also been shown to coincide with the "a" wave of the phlebogram and the P wave of the electrocardiogram. The only apparent difference between the auricular gallop and the physiological inaudible auricular sound which has been demonstrated in several normal tracings appears to be in the intensity of the sound.

2. Both the physiological and pathological auricular and rapid filling sounds have been recorded when of insufficient intensity to be audible. In a number of cases gallop sounds which were heard on auscultation have been shown to decrease in intensity, becoming inaudible while remaining on the sound tracing, and ultimately have become unrecordable as the patient's condition has improved. Patients with both rapid filling and auricular gallop sounds which have been recorded and have later disappeared demonstrate this point. In particular, two severely-hypertensive patients - one treated by sympathectomy and the other by ganglion blocking agents - and several patients with congestive cardiac failure lost their gallop rhythm as the hypertension or congestive failure was successfully controlled.

In a similar way, a summation or indeterminate gallop may only be audible when the two components are close enough to make the total intensity of sound reach the level of audibility. The separation of rapid filling and auricular components and disappearance of the audible gallop sound has also been demonstrated.

3. The clinical diagnosis of all the various cases with gallop rhythm discussed and referred to in this study can be seen in Tables 11, 12, 13, 14, 15, 16, 17, 18 & 19.

Twentyfour patients had an audible rapid filling gallop and 15 had a similar gallop sound demonstrated on the phonocardiogram, but not heard. Of these 39 patients, clinical evidence of cardiac failure was present in 22, and eight were known to be hypertensive (resting diastolic pressure persistently 90 or more or systolic of 200 or more). In eight, the P.R. interval of the E.C.G. was 0.20 second or more. Mortality can be seen from Tables 11, 12 & 13. These figures are not suitable for statistical analysis because of the varying period of followup. Nine patients with an audible rapid filling gallop were alive after one year and five after four years. Only six were known to have died within one year. Twelve patients with an inaudible but recordable gallop were known to be alive one year and six four years later. Only one was known to have died within one year. This gives the impression that there is a greater mortality rate among those with an audible rapid filling gallop than among those with a similar sound which is below the threshold of audibility.

Thirtyone patients had an audible auricular gallop, some being described in this section and some in others, particularly in that section which deals with bundle-branch block. Eleven had a similar gallop which was demonstrated on the phonocardiogram but not heard.

(In two cases the sound was thought to be heard by some observers). Of these 42 cases, there was clinical evidence of cardiac failure in 19, and 20 had a resting diastolic pressure of 90 or more, although the systolic was 140 or less in two of these. Sixteen had a P.R. interval of 0.20 second or greater. Of these patients with an audible auricular gallop, 17 were known to be alive after one year and nine after four years. Six died within twelve months. Of the remainder with auricular gallop recordable on the phonocardiogram but not audible, three were known to be alive after one year and two after four years, while two were known to have died in the first year. Again, statistical analysis is not possible, but the figures (Tables 14 and 15) suggest that the development of an auricular gallop is not of grave prognostic significance. A study of the case details, however, confirms Weitzman's observation that the persistence of an auricular gallop following a myocardial infarction is a bad prognostic sign.

More hypertensive patients occur in this group with auricular gallop than among those with rapid filling gallop, but latent heart block seems to be as frequently associated with one group as with the other.

Ten patients had an audible indeterminate gallop. Of these, there was clinical evidence of cardiac failure in all. Eight had a diastolic pressure of 90 or more, although the systolic was normal in two. Four had a P.R. interval of 0.20 second or more (Table 16). Of these patients, six were known to be alive after one year and all but one were dead within four years.

Actually, eight were dead within eighteen months. These figures suggest strongly that the development of an indeterminate gallop is a sign of much worse prognostic significance than either auricular or rapid filling gallop, as has been suggested by both Mannheimer, and Master and Friedman. It will also be seen from the Table that the incidence of gallop rhythm in various types of cardiovascular disease encountered in this study is similar to that found by Garvin (Table 9) and many other observers. Clinical evidence of congestive cardiac failure was present in 39 cases out of 65 examples of various types of audible gallop rhythm.

4. In the 24 examples of audible rapid filling gallop there were 12 cases with congestive cardiac failure, predominantly right-sided, and no examples of isolated left-sided failure. In the 31 instances of clearly audible auricular gallop 13 had predominantly right-sided congestive failure, and in four, isolated left ventricular failure was diagnosed. Amongst the ten cases of indeterminate gallop there were eight cases of predominantly right-sided cardiac failure and two of isolated left ventricular failure (Fig. iii). The most frequent underlying aetiology can be seen from Table 17. In this series, therefore, rapid filling gallop tends to be associated with right-sided failure, particularly when due to ischaemic heart disease. Auricular gallop also is associated commonly with right-sided failure, but also occurs in the presence of isolated left ventricular failure, due either to ischaemic or hypertensive heart disease - more frequently, the latter.

Fig. VIII shows the incidence of congestive cardiac failure, left ventricular failure and electrocardiographic abnormality in 65 cases of audible gallop rhythm

Normal E.C.G. (Electrocardiogram)

Abnormal E.C.G.

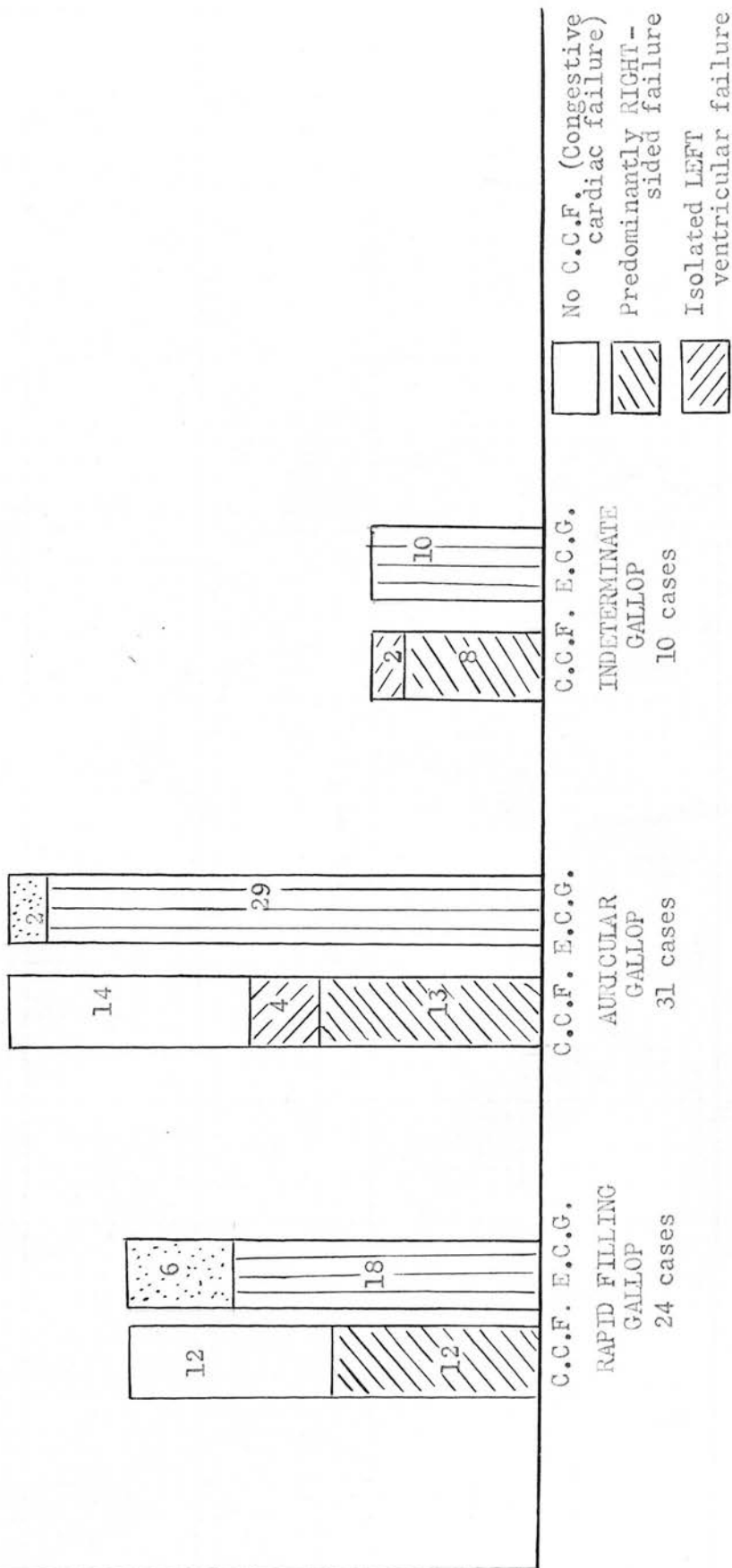
40

30

20

10

NUMBER OF PATIENTS



No C.C.F. (Congestive cardiac failure)
 Predominantly RIGHT-sided failure
 Isolated LEFT ventricular failure

As might be imagined, a true summation gallop may be associated with either primary left or right-sided failure. These conclusions coincide in general with those of Miles (1951) and Evans (1943), who consider that rapid filling gallop most commonly accompanies right, and auricular gallop left, ventricular failure. In this series, however, the correlation between left and right-sided failure and the variation of gallop rhythm is much less specific than has been suggested by the latter writer. The persistence of a gallop sound does not of necessity have a bad prognosis, and several patients had a persistent gallop rhythm over periods exceeding two years without obvious cardiac failure. In two patients a gallop without any very obvious cause lasted for over seven years (Cases Nos. 47 and 29).

5. The view that gallop rhythm is always accompanied by raised venous pressure and tachycardia has not been altogether confirmed in this study. The average heart-rate per minute in patients with rapid filling gallop was 88, in patients with auricular gallop 85, and in those with indeterminate gallop it was 110. Many of these patients, however, had been receiving digitalis in full doses.

6. The gallop sound was not palpable in any case with rapid filling gallop, but was easily felt in six cases with auricular gallop and in two with indeterminate gallop (these last two both having a cuneate *cor pulmonale*).

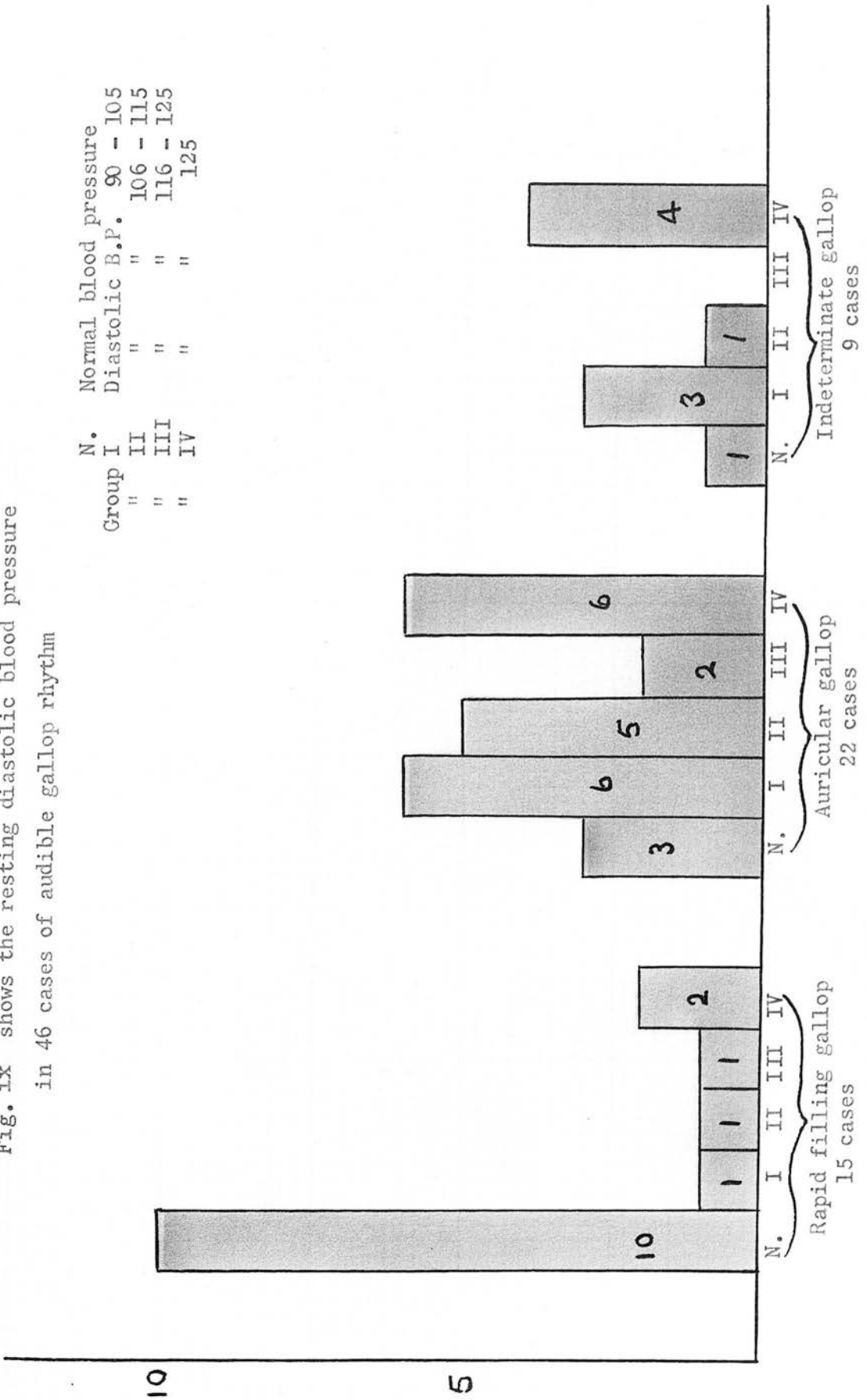
7. The gallop sound was almost always best heard at the apex or near the lower left sternal edge, or somewhere on a line joining these two points. A true right-sided gallop as described by Potain was never encountered, but in the two cases with *cor pulmonale* and in ~~two~~^{three} others (one also with severe chronic respiratory

- disease) an auricular or indeterminate gallop was best heard in the upper epigastrium over the right ventricle.
8. In two cases a quadruple rhythm was heard by some observers, and in many both rapid filling and auricular gallop sounds were recorded on the phonocardiogram. In one case, both types of gallop were heard at different times (Case No. 60).
 9. On at least two occasions the phonocardiograph was unable to record gallop sounds which had been heard on auscultation by several independent observers. More frequently it was able to give more exact information, as when it confirmed the presence of an auricular gallop when splitting of the first sound had been suspected clinically.
 10. It was not possible to confirm the observations of Duchosal, who claimed that prognosis varied with the time interval between the peak of the P-wave and the beginning of an auricular gallop sound, and that as a patient improved this P-G interval inversed until the gallop merged with the first sound.
 11. Slowing of the heart-rate by carotid sinus pressure was found to be extremely useful in several cases of tachycardia where an indeterminate gallop was diagnosed. In four of these cases the rapid filling and auricular sounds could be separated out on the sound tracing by means of this vagal stimulation and resultant bradycardia. These cases were, therefore, clear examples of true summation. In another three cases the precise constitution of the gallop could not be demonstrated, but there was good evidence to suppose they were examples of true summation. In the remaining three cases the indeterminate gallop was shown to be due

largely if not entirely to a rapid filling sound associated with tachycardia. The study of ventricular extra-systolic beats, when they occurred, also often gave additional information as to the true origin of the gallop, particularly in the presence of tachycardia, as the compensatory pause which follows these sometimes revealed a typical rapid filling sound. Carotid sinus stimulation was also found to cause a transient change in the configuration of the P-wave.

12. The association between the various degrees of hypertension, the presence or absence of right or left-sided heart failure, the presence of electrocardiographic abnormality, and the three types of gallop rhythm can be seen from Figs. viii and ix. The closest correlation appears to be between the presence of a gallop rhythm and abnormality of the E.C.G. tracing. The commonest abnormalities included left ventricular hypertrophy, latent heart block, bundle-branch block, auricular fibrillation, and the S.T. changes recognised as usually accompanying myocardial infarction. The next closest correlation was between the presence of hypertension and the gallop rhythm.

Fig. ix shows the resting diastolic blood pressure in 46 cases of audible gallop rhythm



GALLOP RHYTHM IN CONSTRICTIVE PERICARDITIS

That a gallop rhythm was sometimes associated with constrictive calcified pericarditis has been known since the time of Potain (1856), but it has been variously described as a split second sound, a rapid filling gallop, or an extra diastolic sound peculiar to constrictive pericarditis alone. Thayer (1908) refers to it, but it was first clearly described and analysed in two cases of calcified pericarditis in 1933 by Lian, Marchal and Pautrat, who called the extra sound "La vibration pericardique protodiastolique". These authors stated that the sound was often louder than the second sound, that it was heard best at the xiphoid, but was frequently heard all over the praecordium. Lian distinguished it clearly from a splitting of the second sound and from the rapid filling gallop or third sound by virtue of its greater intensity and "eclat". The gap between the end of the second sound and the beginning of the extra sound he gives as 0.04 to 0.02 second!

Garvin, writing in 1943 on the incidence of gallop rhythm, quotes 14 cases of "tuberculous or obliterative" pericarditis, of which four had a gallop rhythm, but he gives no further clinical details. Frost (1949) and Evans (1951) describe such a gallop as being commonly present in constrictive pericarditis. Evans and Jackson (1952) found either a broadly split second sound or a rapid filling gallop or third heart sound in 27 of their 30 cases. Later, Evans (1954) expressed the view that this extra sound is usually due to the sudden opening of the tricuspid valve. Mounsey, in 1955, reports on the sound occurring in 18 or 22 patients with constrictive pericarditis.

The gallop sound has, however, been shown to coincide with the sharp fall of venous pressure in early diastole, and in recordings of the right ventricular pressure Eliasch (1950) and Hansen and his co-workers (1951) showed that it was coincident with the moment of cessation of rapid filling of the ventricle.

With the aid of an electrokymograph MacCusick (1952) demonstrated that the sound coincides with the sudden cessation of outward movement of the ventricular wall in just the same way as Kuo et al. had demonstrated the relationship of the rapid filling gallop to sudden abnormal outward movement of the left ventricular border occurring in early diastole in the presence of congestive cardiac failure.

It is concluded, therefore, that the gallop sound of constrictive pericarditis and the rapid filling gallop have the same basic origin as they are both due to the sudden rush of blood into the ventricle early in diastole. In both cases the venous pressure is raised. In congestive failure the ventricular wall may be dilated and of poor tone, and the sound is accentuated, but in constrictive pericarditis the sound is louder still and has a different character because the constricting and often calcified pericardium limits the degree of dilatation and elasticity of the ventricle to such an extent that the sudden inrush of blood is halted abruptly and produces a louder clearer sound. For purely mechanical reasons also, if the movements of the ventricle are restricted, then the sound may occur slightly earlier than the rapid filling sound in cardiac failure or the physiological third sound (Mounsey, 1955).

PRESENTATION OF RECORDS

Two cases of constrictive pericarditis are described and their records shown.

CASE No. 61, a housewife aged 46, had a three-year history of progressive disability due to dyspnoea and swelling of the abdomen and legs. She had gross bronchiectasis and fibrosis of the left lung and also calcified constrictive pericarditis. On examination she was found to be in congestive cardiac failure with auricular fibrillation, and was extremely ill. (B.P. $\frac{120}{78}$). On auscultation a clear gallop rhythm was heard due to an added sound in the proto-diastolic period. This gallop was maximal at the pulmonary area and down the left border of the sternum, but heard all over the praecordium. The second sound was relatively quiet, and at the apex there was a loud systolic murmur but no diastolic murmur. The gallop sound had a curious quality and did not seem to have the usual characteristics of the audible rapid filling sound. The neck vein pulsation was very marked. In due course this patient had a left lobectomy and pericardectomy, with very great subsequent improvement. The heart sounds five months later were changed; there was a loud pan-systolic apical murmur with a loud first and normal second sound. The gallop rhythm was no longer heard. Thereafter she was free of cardiac failure and her general condition much improved. Recordings were taken on two occasions, before and after operation.

CASE No. 62, a girl aged 19, had been operated on for constrictive pericarditis seven years previously. For several weeks before admission she had been getting progressively more dyspnoeic and tired and had complained of chest pain. On examination she had neck vein congestion and hepatomegaly, but no oedema. (B.P. $\frac{115}{80}$). Pulsus paradoxus was discernable and there was an apical systolic murmur with a loud gallop rhythm. Despite the tachycardia the extra sound was recognised as being protodiastolic in time and was heard all over the praecordium, but maximum at the apex. E.C.G. showed normal rhythm with A-V block (P.R. = 0.28 second). Phonocardiograms were taken on one occasion.

Recordings are shown of these two cases, with simultaneous tracings of the E.C.G. and jugular phlebogram. The relationship between the gallop sound and the point of maximum fall of jugular pressure is clearly seen. In both, the time interval between the beginning of the second sound and the gallop sound of constrictive pericarditis - 0.14 to 0.16 second in the first case and 0.1 to 0.12 in the second - is seen to be much the same as that between the second and the rapid filling gallop sound heard in congestive failure and also the physiological third sound, although the time intervals in the second case (No. 62) are at the lower limit of the accepted range.

In both cases the gallop sound was louder than the second heart sound on auscultation and heard all over the praecordium, maximal at the pulmonary area in one and all down the left sternal edge in the other, where there was considerable mediastinal displacement to the left following previous partial pericardectomy.

In the first case, No. 61, record A shows the gallop sound from the pulmonary area. Record B shows the apical pansystolic murmur. Following pericardectomy the gallop sound is seen to be much softer (there is of course no accurate calibration), but still present although occurring slightly later in diastole (0.16 to 0.18 second after beginning of second sound). (Record C). This might well be expected after the pericardial constriction and the cardiac embarrassment were relieved. The gallop sound still occurs at the same point of maximum fall in jugular pressure. Clinically the gallop rhythm had disappeared after operation. On auscultation at the time the post-operative recordings were made, all that could be heard was the normal first sound, a loud harsh systolic murmur which had been noted before, and a slightly split pulmonary second sound which could not possibly have been confused with a rapid filling gallop sound. The extra sound still remaining on the tracing was presumably below the limits of audibility.

In the second case a similar gallop sound is seen, also coinciding with the trough in the phlebogram representing the sudden early diastolic filling of the ventricles. Unfortunately there is no single tracing with P.C.G., E.C.G. and jugular pulse tracing, but record A showing E.C.G., pulmonary and apical sound tracings, can be compared with record B showing venous pulse and P.C.G. tracing. In this case the time interval between the beginning of the second sound and the beginning of the gallop is 0.1 to 0.12 second. Here, the presence of a tachycardia and the latent heart block (P.R. = 0.28 second) make the records a little difficult to follow. The gallop sound might at first ^{sight} be thought to be an

auricular or summation gallop were it not for the occurrence of occasional dropped beats (Record C). At these times the gallop sound is still present after the preceding extra systolic sound followed by a long interval before the next, delayed, auricular systole. The gallop sound, therefore, is quite independent of auricular systole and must be due to rapid ventricular filling.

SUMMARY

1. In conclusion, the gallop rhythm so frequently heard in cases of constrictive pericarditis is discussed and recordings taken from two cases of calcified pericarditis are shown. One case has been recorded both before and after pericardectomy.
2. It seems most likely that this extra gallop sound arises by the same mechanism as the rapid filling gallop sound and the physiological third sound. It is due to the sudden halting of the inrush of blood into the ventricles which are prevented from further dilatation and expansion by the rigid calcified pericardial framework. Pericardectomy either abolishes or alters this sound considerably.

THE HEART SOUNDS IN
BUNDLE - BRANCH BLOCK

There has been considerable controversy in the past as to whether or not bundle-branch block produces auscultatory signs which are diagnostic of the condition. It has been stated for instance that the abnormal degree of asynchronous contraction between the right and left ventricles will produce a recognisable splitting of the heart sounds with a double apical impulse.

Katz (1925) demonstrated that under normal physiological conditions the ventricles contract at slightly different times. This is now generally agreed to be the cause of physiological splitting of the heart sounds. It has already been seen that this physiological splitting is so common in health that it is of little value in the diagnosis of heart disease.

Cowan and Bramwell (1925) and Hill (1930) agreed that it was impossible to diagnose bundle-branch block by clinical examination alone. Macleod, Wilson and Barker (1931) showed that in three cases of bundle-branch block in which an auricular gallop could be demonstrated, the extra gallop sound occurred before the R-wave, and was therefore due to auricular systole and could not have been due to asynchronous ventricular contraction. On the other hand, Campbell and Suzman (1932) reported a case of transient bundle-branch block in which an auricular gallop was present as long as the conduction defect remained, but disappeared once normal intraventricular conduction returned. The patient was hypertensive and was having attacks of paroxysmal nocturnal dyspnoea. It seems possible, therefore, that both the transient E.C.G. abnormalities and

gallop rhythm in this case may have been due to a silent myocardial infarction.

King and McEachern (1932) believe that bundle-branch block can nearly always be diagnosed clinically. By means of a simple apparatus for recording apex cardiograms they demonstrated visible apical re-duplication in 84 per cent. of 50 cases. The re-duplication was palpable in 80 per cent. of the cases, but on auscultation a re-duplicated first sound was heard in only 56 per cent. They claimed that it was also possible to differentiate this double systolic apical pulsation of bundle-branch block (which always commenced after the R-wave of the E.C.G. and was, therefore, due entirely to ventricular systole) from the pre-systolic deflection of auricular gallop. King and McEachern were also able to distinguish the normal monophasic apical pulsation and biphasic pulsation of the physiological split first sound, by means of apical cardiograms. These authors concluded that re-duplication of the first heart sounds in bundle-branch block was due to ventricular asynchrony and that this condition could be diagnosed clinically by means of a visible and palpable re-duplication of the apical thrust which was entirely within systole. They also maintained that asynchronous systolic murmurs could be detected in 12 per cent. of their cases, but gave no details as to how these were demonstrated. Most workers agree that splitting of the first and second sounds may occur in bundle-branch block but do not consider this gives rise to a diagnostic double apical thrust which is characteristic only of this condition. In 1934, J.K.Lewis, after studying 23 cases with bundle-branch block, found only one case with a characteristic

re-duplicated apical thrust and was unable to substantiate all of King and McEachern's claims. He concluded that the physical signs of intraventricular conduction defect were not usually diagnostic.

Bramwell in 1935, Wolferth and Margolies in the same year, and Orias and Braun-Menendez in 1939, came to the same conclusion as Lewis. Wolferth and Margolies also discussed the part played by asynchronism of ventricular contraction in producing splitting of the first heart sound and were able to demonstrate the correct relationship between the E.C.G. pattern and both right and left bundle-branch block and corresponding delay in mechanical events occurring in either the right or left ventricle. Evans (1943) describes 30 patients with bundle-branch block, of whom six had an auricular gallop with an associated A-V conduction defect and seven an auricular gallop associated with left ventricular failure. In others the first sound was obscured by a rough systolic murmur, but in only two was a clearcut dual rhythm heard. Evans and also Wiggers in the year 1949 and Wood in 1950 emphasised the fact that bundle-branch block may cause wide splitting of the first or second sounds, and in 1954 Leatham showed that although splitting of the sounds is common in health, a split first sound is most marked in lesions of the right branch due to the delay in right ventricular excitation and tricuspid closure. The tricuspid valve normally closes after the mitral, but closes even later in this condition because of the delay in electrical conduction to the right ventricle. Similarly, delay in closure of the pulmonary valve results in wide splitting of the second sound.

A conduction defect in the left branch will cause delay in aortic closure and paradoxical splitting as the normal order of

closure of pulmonary and aortic valves is reversed and the width of splitting decreases during the inspiratory phase.

Leatham distinguishes clearly between splitting of the first sound in bundle-branch block due to separation of the left and right ventricular complexes and gallop rhythm due to separation of the audible auricular sound from the ventricular complexes. Evans, however, in addition to describing the split due to separation of the two ventricular complexes, states that early occurrence of the auricular sound associated with lengthening of the P.R. interval may also be responsible for splitting of the first sound when it occurs in bundle-branch block. It is well recognised that in the presence of an auriculo-ventricular conduction defect such as occurs in latent heart block the auricular component may occur before the ventricular component of the first sound and give rise to an auricular gallop, but this is usually quite independent of a bundle-branch block unless the two abnormalities of conduction happen to be present together.

In this study, a diagnosis of bundle-branch block has been accepted if the Q.R.S. complex in any of the standard E.C.G. leads has measured 0.12 second or more in duration. (Nomenclature and Criteria for Diagnosis of Diseases of the Heart, 1946). Any vibrations in the phonocardiograph tracing which occur, certainly before the Q-wave and probably before the peak of the R wave, are accepted as being due to auricular and not to ventricular systole, (Lewis, 1912; Wiggers and Dean, 1916; Orias and Braun-Menendez, 1939; Rappaport and Sprague, 1942; and Wood, 1950). The clinical criteria

for distinguishing between a split and a gallop sound have already been discussed and are clearly open to individual observer error, but the P.C.G. criteria have been kept simple and based strictly on a consideration of the position of the P.C.G. vibrations in relation to the Q.R.S. complex of the E.C.G.

PRESENTATION OF CASES

Phonocardiograms were recorded from 16 cases of bundle-branch block. Each one has been subjected to full electrocardiographic study, that is to say, the standard limb leads I, II and III, unipolar limb leads VR, VL, and VF, and unipolar chest leads V 1 to 7 inclusive.

Ten of the cases had left and six right bundle-branch block. These are discussed and a selection of representative records are presented in this section. In some of the recordings with the Boulitte instrument the E.C.G. tracing is not always of good quality and the time-marker is not always accurate. When precise measurements such as the P.R. and Q.R.S. intervals cannot be made from E.C.G. tracings accompanying these phonocardiographs, limb lead complexes recorded with the standard Cambridge portable electrocardiograph have been mounted as well.

Patients with left bundle-branch block will be described first and those with right bundle-branch block later.

Patients with Left Bundle-Branch Block

The first four with conduction defects of the left bundle also had an audible ~~auricular~~ gallop rhythm which was interpreted clinically as being an auricular gallop. All had ischaemic heart disease and some were hypertensive also. One had a severe secondary anaemia.

The first case, No. 63, was a 60-year-old male with paroxysmal ventricular tachycardia and resultant congestive cardiac failure arising during the course of a severe chest infection. The infection and cardiac failure were successfully treated and normal rhythm restored with quinidine. The patient gave no previous history of angina pectoris or other relevant disability. When normal rhythm was re-established, an electrocardiogram showed left bundle-branch block (QRS = 0.14', PR = 0.20'), and on auscultation a loud gallop rhythm was heard all over the praecordium but was maximum at the apex where there was also a systolic murmur. This gallop persisted long after the congestive failure and tachycardia had been controlled. Six years later this man was still alive and well. Phonocardiograms were recorded on four occasions. The first set of records (A and B) were taken from the apex when the patient was still extremely ill and in congestive failure. They are, therefore, not very clear. However, the auricular sound is quite definite, occurring before the Q wave of the E.C.G., and in one tracing can be seen to be coincident with the 'a' wave of the jugular pulse. This auricular sound is absent before the extra-systolic beat, but in the compensatory pause following the extra-systole a rapid filling gallop can just be seen. The systolic murmur is also visible. Two months later the patient was completely

recovered, with no residual evidence of congestive failure, and at this time the auricular gallop sound was still audible and more easily demonstrated on the apical phonocardiogram. There is also a variable degree of splitting of the second sound (Records C and D). Two weeks later still, the tracings which were recorded from a point just internal to the apex are even clearer and show both auricular and split second sound. (Record E). In the final recordings where Lead I of the E.C.G. has been used instead of Lead II ventricular extra-systoles (one of them interpolated) can be seen (Record F). In all these records the gallop sound occurs before the Q wave of the E.C.G. and therefore before the initiation of ventricular systole. It cannot, therefore, be a direct result of the conduction defect and must be associated with auricular systole. Unlike the rapid filling gallop sound which disappears as the cardiac failure is controlled, the auricular gallop remains and therefore cannot be due to frank congestive failure. However, a degree of left ventricular failure probably persisted, and the bundle-branch block and history of paroxysmal tachycardia were clear evidence of the presence of ischaemic heart disease. Splitting of the second sound may often be associated with ^{right} bundle-branch block, but cannot be distinguished from normal splitting, which is common. In this case the time interval between the beginning of the second sound and the second part of the split varies between nil and 0.06 second. The approximate duration of the first and second sounds are 0.16 and 0.05 to 0.10 respectively, or approximately 0.2 second if the auricular gallop is included in the measurement of duration of the first sound.

Case No. 64, a woman aged 54, had severe benign essential hypertension but without congestive failure, renal damage, or retinal changes. Her pre-operative blood pressure was $\frac{210}{130}$ after two weeks rest in bed. She was subjected to bilateral lumbar sympathectomy, and on the fourteenth day following the second operation she suffered a myocardial infarction. At this time she had a loud gallop rhythm due to a palpable and audible auricular sound, loudest at the apex, but heard all over the praecordium. There was also a systolic apical murmur and the second sound was not obviously split. There were signs of left ventricular failure, the blood pressure was $\frac{130}{100}$, and her pulse-rate was 180/minute. E.C.G. showed left bundle-branch block (Q.R.S. = 0.14'. P.R. = 0.14'). She made an excellent recovery, but developed a rapid sinus tachycardia on the least exertion or emotional stress, the gallop rhythm persisted, and her B.P. returned to its earlier level of around $\frac{210}{130}$. 5 Years after the operation she was alive and well, leading, as her own doctor puts it, "a very pleasant, useful and uncomplaining life".

A P.C.G. was taken shortly after the myocardial infarction and on two subsequent occasions. The first record shows a systolic murmur, but fails to reveal a gallop sound, although this was clearly heard (Record A). The recording was, therefore, repeated four days later and an auricular sound is now clearly seen occurring just before the Q wave of the E.C.G. Lead II (Record B). At this time there was no obvious cardiac failure, but further recordings (C) with P.C.G. amplitude increased bring out another sound

occurring just 0.20' after the beginning of the preceding second sound and having a variable relationship to the P waves. This, therefore, must be a rapid filling gallop sound, presumably due to latent left ventricular failure and the hypertensive ischaemic heart disease. It was not heard on auscultation, but may well have been superimposed on the auricular sound to form a summation gallop. Alternatively, it may be of a too low frequency to be detected by the ear. The auricular gallop persisted, and seven weeks later, the recording (D) shows it still beginning just before the Q wave, and the low-pitched rapid filling gallop sound can also still be seen. There is a very clear splitting of the second sound, with the last element of the split being loudest. As with Case 61, the gallop sound here seems to be related to auricular systole and is presumably due not specifically to the bundle-branch block, but to the heart failure and primary heart disease. The total duration of the first sound including the auricular part, is approximately 0.20 second, and excluding the auricular component, approximately 0.18 second. The second sound duration is 0.10 to 0.12 with the interval between the beginning of the second sound and the beginning of the split approximately 0.04 to 0.06 second.

The next two patients both had hypertension, with cerebral atherosclerosis and also ^{probable} myocardial ischaemia.

Case No. 65 was a 76-year-old female with no history of angina pectoris or congestive cardiac failure. Her B.P. was $\frac{210}{110}$. She had left ventricular hypertrophy and a hypertensive retinopathy. E.C.G. showed left bundle-branch block (Q.R.S. = 0.12'. P.R. = 0.14'). There was no evidence of cardiac failure

on clinical examination. On auscultation an auricular gallop was heard, loudest at the apex. At this time, P.C.G. recorded from the apical area shows an auricular sound starting just before the Q wave of the E.C.G., a systolic murmur, and a splitting of the second sound (Record A). Another tracing (B) recorded from a point just lateral to the apex also shows the auricular gallop sound. This patient had a cerebral thrombosis several weeks later and died. No post-mortem was available. Time intervals on these phonocardiograms cannot be measured even approximately, as the time-marker setting was not known, but the electrocardiogram measurements can be made from the strip of Lead II which has been mounted separately.

Case No. 66 was a 70-year-old woman from whom no proper history could be obtained as she was suffering from advanced senile cerebral arteriosclerosis. She was hypertensive, with left ventricular hypertrophy, and had a left bundle-branch block (Q.R.S. = 0.12'. P.R. = 0.2'), but no evidence of congestive cardiac failure. Unfortunately, details of this woman's subsequent history are not available. On auscultation an auricular gallop was heard with some difficulty as both it and the first sound were relatively faint. No splitting of the second sound or other abnormalities were heard.

A P.C.G. taken from the apex shows a faint extra sound occurring coincidentally with the Q.R. wave, but before the R wave. The records are similar to those of Case No. 65 and have not been mounted. Duration of first sound is approximately 0.14 second.

The next three patients also had a gallop rhythm which was thought clinically to be due to a rapid filling sound. The first, Case No. 67, a post-menopausal woman with a severe secondary anaemic of the hypochromic nutritional type, also had left bundle-branch block. (Q.R.S. = 0.16 ' . P.R. = 0.18 second). Her haemoglobin was 40%, her blood pressure varied between $\frac{140}{90}$ and $\frac{170}{100}$, and on auscultation following admission a gallop rhythm and split second sound were heard. In addition to the rapid filling gallop, an auricular gallop was claimed to be heard by one observer. The anaemia was corrected with iron therapy, and twelve months later she was seen again with normal haemoglobin but still with definite rapid filling gallop heard best mid-way between the apex and the left sternal edge. There was also an apical systolic murmur and a splitting of the second sound maximal at the pulmonary area. At this time she was found to have a raised blood pressure ($\frac{200}{120}$), which persisted until she was last seen, alive and well, three years later, with the gallop rhythm still clearly heard and the blood pressure recorded at $\frac{200}{110}$. The anaemia had not recurred.

P.C.G. recordings were taken on two occasions - when she was first seen and the anaemia severe, and twelve months later when it had been corrected. The first recording from the apex (Record A) is of low amplitude, but does show a tiny auricular sound occurring before the Q wave of the E.C.G. There is no rapid filling gallop sound to be seen and no splitting of the sounds or other abnormality. Record B, taken twelve months later, shows the auricular gallop more clearly and also a split second sound recorded from the pulmonary area (Record C). Despite the tachycardia, it can be shown that the gallop sound is in fact an auricular sound

and not a rapid filling gallop. It has a fixed relationship to the 'a' wave of the jugular pulse and also to the P wave of the E.C.G. It occurs as long as 0.26 second after the preceding second sound. Mannheimer gives the maximum time interval between the beginning of the second and the beginning of a rapid filling sound as 0.18 second. All other observers consider this maximum time interval to be less than 0.20 second (See Table 7). Even the most experienced auscultator considered this to be a rapid filling gallop, and the error must presumably have been due to the tachycardia. For this reason also the jugular pulse tracing is not very helpful as there is fusion of the 'v' and 'a' waves. However, there is a slight dip to be seen in some cardiac cycles where the 'v' wave runs into the subsequent 'a' wave; this point occurs about 0.2 second after the beginning of the second sound and there is an occasional, though very small, vibration on the P.C.G. at this point which could represent the rapid filling sound. The split second sound recorded at the aortic area in Record C shows the loudest, therefore presumably aortic, component to be the last. This frequently occurs in left bundle-branch block where the aortic component of the second sound is delayed so that instead of forming the first part of a split second sound as it normally does it forms the latter part. The duration of the first sound was approximately 0.16 to 0.20 second and of the second sound, 0.08 second. The interval between the commencement of the first and second parts of the split second sound was approximately 0.04 second.

Both the next two cases developed rapid filling gallop as a result of cardiac failure due to ischaemic heart disease and both are described in the section on rapid filling gallop, although they also had left bundle-branch block.

The first, Case No. 23, had a myocardial infarction and auricular fibrillation. He died of a further infarct three years later. Phonocardiograms show the gallop sound occurring in the protodiastolic period; there is no splitting of the first or second sounds. Because of the fibrillation and despite the presence of ventricular asynchronous contraction, there are no vibrations on the phonocardiogram occurring before the S wave of the E.C.G.

The second, Case No. 25, had chronic ischaemic myocardial degeneration with congestive cardiac failure and bundle-branch block. In this case the gallop disappeared as the failure was successfully treated. One year later the patient died, and at post-mortem the presence of gross coronary atheroma was confirmed. Phonocardiograms showed that in addition to the rapid filling gallop sound, a small auricular vibration may be seen coincident with the Q.R. segment of the E.C.G. and most evident when the congestive failure was present. The Q.R.S. interval was 0.14 second and the P.R. interval 0.18 second. The duration of the first sound measured approximately 0.16 to 0.21 second, and of the second 0.05 to 0.10 second.

Three more patients had left bundle-branch block, and in all an auricular sound was demonstrated on the P.C.G. Clinically the first was thought to have a split first sound, the second dual rhythm, and the third only was thought to have an

auricular gallop on auscultation. The second and third patients also had an auriculo-ventricular conduction defect.

In case No. 68, aged 82, there were no signs or symptoms of cardiovascular disease other than the E.C.G. evidence of bundle-branch block (Q.R.S. = 0.18'. P.R. = 0.20'). On auscultation the first heart sound was thought to be split. He died several months later with multiple secondaries from a carcinoma of the larynx, but no post-mortem was available. P.C.G. shows a definite auricular sound beginning just before the Q wave of the E.C.G. and quite separate from the first sound, which is not obviously split on the recording. The heart-rate is varying slightly with an apparent sinus arrhythmia, and the auricular sound is intermittent, possibly varying with respiration. It seems to have no definite relation to the cycle length, and the sound was not heard clinically. The time-marker on this tracing is inaccurate, but standard E.C.G. leads recorded on the same day with the Cambridge instrument are mounted.

Similar records are those of Case No. 69, an 80-year-old male, but in this patient there was a two-month history of congestive cardiac failure due to ischaemic heart disease. On auscultation a dual rhythm was heard, with no gallop or splitting of the sounds. He had considerable cardiomegaly, with a B.P. of $\frac{110}{60}$. E.C.G. showed in addition to the bundle-branch block a latent heart block (Q.R.S. = 0.18'. P.R. = 0.24'). P.C.G. showed a definite auricular sound occurring before the Q wave of the E.C.G.

The first heart sound had a duration of approximately 0.20' and the second sound, approximately 0.10 second. In this case the auricular sound which was seen on the P.C.G. but not heard may have been due either to the latent heart block or to the heart failure, but it is not clear why a similar sound was present on the tracing of case 68 unless incipient cardiac failure was present but was not of a degree sufficient to be detected clinically. The bundle-branch block could not have accounted for the clearcut auricular sound in either case as the sound occurs well before the Q wave of the E.C.G. and, therefore, the commencement of ventricular systole.

Case No. 70 was a male aged 64 who gave a history strongly suggestive of previous myocardial infarction. On examination there was no evidence of congestive cardiac failure. The B.P. was $\frac{110}{70}$. The cardiac apex was not displaced, and an E.C.G. confirmed the presence of latent heart block and left bundle-branch block (P.R. = 0.32'. Q.R.S. = 0.14'). On auscultation there was a faint first sound and an audible auricular gallop rhythm. There was also a soft systolic apical murmur and a loud second sound.

The P.C.G. showed a very faint first sound with an auricular sound which can be seen to occur between the P and R waves of the E.C.G. The main part of the first sound also appears to begin just before the peak of R, but after the Q wave. There is also a systolic murmur maximal in mid-systole and the second sound is not split. These records are not mounted. Six years later this man was alive and well, but his cardiac state was not known.

It is clear from these three cases, two of them octogenarians, that the presence of a visible auricular sound on the P.C.G. tracing may not have any very serious significance, particularly if the P.R. interval is prolonged.

Patients with Right Bundle-Branch Block

Six patients were found to have a right bundle-branch block and their heart sounds were recorded. Four were thought to have a definite gallop rhythm clinically (three auricular and one rapid filling), and one either an auricular gallop or a split first sound. Five had hypertensive and ischaemic heart disease, and the sixth had mitral stenosis with a conduction defect.

The first patient with a clear auricular gallop clinically was Case No. 71, a male aged 56. He had a nine-month anginal history culminating in a myocardial infarction and admission to hospital. On examination he had marked left ventricular hypertrophy (B.P. = $\frac{180}{110}$), but there was no clinical evidence of congestive cardiac failure. E.C.G. showed right bundle-branch block (Q.R.S. = 0.12'. P.R. = 0.16'), and on auscultation there was a loud unequivocal auricular gallop heard all over the praecordium. It was maximal at the sternal edge in the fourth left space, but well heard and also palpable high up in the epigastrium. At the apex there was a systolic murmur and the first and second sounds were normal. This patient recovered from the acute episode and remained well until he died suddenly at home three months later.

Shortly before death he had been examined and the gallop rhythm found to be still audible. No post-mortem was obtained.

P.C.G. records taken on two occasions show no distinct auricular sound occurring before the Q wave of the E.C.G. The first sound is not obviously split, but lasts for approximately 0.20 second - this being well above the usual limit of normal. The initial low-frequency vibration of the first sound begins just before the peak of R wave and in appearance resembles the auricular sounds previously seen in cases of gallop rhythm. It is not distinct from the remainder of the first sound on the tracing, but when a ventricular extra-systole is recorded, this initial low-frequency component is missing. It seems reasonable to presume, therefore, that this sound, which had the auditory characteristics of an auricular gallop and occurred at the very point of initiation of ventricular systole, is due to auricular contraction. Phonocardiogram recordings A and B are from the apex, using different amplitude settings, and tracing B shows the extra-systole. Record C is taken from a point in the epigastrium immediately inferior to the xiphisternum. Repeat records did not show any change in the pattern, and the gallop rhythm was audible and palpable right up to the time of death. The duration of the first sound is approximately 0.20 second and of the second, approximately 0.1 second.

Case No. 72, a male aged 67, had both hypertensive heart disease with left ventricular hypertrophy and also chronic bronchitis and emphysema. On examination he was found to be in a state of congestive cardiac failure, his blood pressure at this time was $\frac{190}{110}$, and his E.C.G. showed a Q.R.S. interval of 0.14 second and P.R. interval of 0.20 second. Auscultation at the apex revealed a clearcut auricular gallop. This was even more clearly heard high up in the epigastrium. The gallop sound was not palpable. Treatment with rest, digitalis and mersalyl led to a fairly rapid recovery, but the gallop persisted and was still present when the patient was last seen eighteen months later.

A P.C.G. was recorded when the patient was in congestive failure. It shows a clear auricular gallop sound occurring between the P and the Q wave of the E.C.G. This extra sound must be due to auricular contraction and ^{auricular} systolic blood flow and is independent of the intraventricular conduction defect, because it occurs in its entirety before the onset of ventricular systole. The tracing which has been mounted was recorded from a point high up in the epigastrium. In contrast to Case No. 71, the P.C.G. here gives clearcut confirmation of the presence of an auricular gallop, Unfortunately the record is badly over-developed, but the essential details can be clearly seen. Total duration of the first sound is more than 0.20 second.

The third patient with a clinical auricular gallop presents a complicated picture and is referred to in detail at the end of the section on auricular gallop and also in the section on mitral stenosis.

This was Case No. 50. In addition to a right bundle-branch block he had a shortened P.R. interval and mitral stenosis (Q.R.S. = 0.12', P.R. = 0.12'). Despite the presence of calcification in the mitral valve cusps he was treated successfully by valvotomy. An auricular gallop was heard pre-operatively which might have been confused with a pre-systolic murmur; however, the gallop was quite definite clinically, well heard at the apex, and also in the upper epigastrium. It was still present three years after the operation. The clinical details and P.C.G. records are discussed elsewhere, and the auricular sound is clearly seen on the tracings.

The gallop sound must be due to auricular systole as it occurs before the Q wave; it cannot be a simple audible auricular sound due to delayed A-V conduction, because the P.R. interval is in fact shortened. The presence of a calcified narrowed mitral valve makes the normal mechanism of auricular gallop most unlikely as the sudden rush of blood from auricle to ventricle would be prevented at least on the left side of the heart. Presumably, therefore, this gallop must be purely right-sided in origin and this would also explain the fact that it was best heard in the epigastrium, that is to say over the right ventricle. The duration of the first sound is approximately 0.2 second and of the second, approximately 0.1 second.

Case No. 73, a 69-year-old male, was thought clinically to have a rapid filling gallop. His E.C.G. showed Q.R.S. interval to be 0.16 second and the P.R. interval 0.22 second. Phonocardiogram recordings were difficult as the patient was unco-operative, but a gallop sound can be seen on the apical tracing and occurs just about 0.10' to 0.12' after the beginning of the second sound. The

first and second sounds appear relatively normal, but the recordings are of poor quality, and have not been mounted. Approximate duration of the first sound is 0.16 second and of the second, 0.05 second.

Case No. 74, a male diabetic patient aged 69, had auricular fibrillation. On examination there was evidence of congestive cardiac failure and left ventricular hypertrophy from hypertension (Q.R.S. = 0.12'). On auscultation, a dual rhythm with split mitral first sound and a high-pitched apical systolic murmur were heard. There was no gallop.

Phonocardiogram Records A and B show a definite splitting of the first sound. There was, of course, no auricular component and the first sound commences just before the S wave, but after the R wave of the E.C.G. This splitting may well be due to genuine ventricular asynchrony, although the second sound is not obviously split. The duration of the first sound is approximately 0.16 to 0.18 second and of the second, 0.04 to 0.08 second. This man was alive and relatively well eight years later, despite the fact that he developed diabetic gangrene and had both feet amputated.

Finally, Case No. 75, was a 59-year-old male. On examination he was found to be hypertensive with gross left ventricular hypertrophy. He also had the history and physical signs suggestive of chronic bronchitis and emphysema. He was in congestive cardiac failure, and on auscultation some debate arose

as to whether a definite auricular gallop or a broad splitting of the first sound was present. Electrocardiogram showed the presence of right bundle-branch block. (Q.R.S. interval measured 0.12' and P.R. interval 0.20'). Following treatment with rest in bed, digitalis, and mersalyl, he made a good recovery, and a year later he was alive and well.

The phonocardiogram tracing taken from the apex is poor in quality, but just shows an auricular sound situated between the P and the Q waves of the E.C.G. The first sound is not obviously split, neither is the second. The records have not been mounted. Duration of the first sound is approximately 0.10 second and the second, 0.04 to 0.06 second.

TABLE 18

CASES OF LEFT BUNDLE-BRANCH BLOCK

Case No.	Sex	Age	Diagnosis of Type of Gallop		E.C.G. measurement in seconds		Approx. P.C.G. measurement in seconds		Blood Pressure	Period of follow up	Diagnosis and Remarks
			BY auscultation	BY P.C.G.	QRS	PR	1st sound	2nd sound			
63	M.	60	A	A	0.14	0.20	0.16	0.05 - 0.10 split	-	6 yrs. Alive.	Ischaemic heart disease. C.C.F. Rapid filling gallop also seen on P.C.G.
64	F.	54	A*	A	0.14	0.14	0.18	0.10 split	$\frac{210}{130}$	5 yrs. Alive.	Hypertension, Myocardial infarction. L.V.F. Rapid filling gallop also seen on P.C.G.
65	M.	76	A	A	0.12	0.14	-	split	$\frac{210}{110}$	3 mths. Died.	Hypertension. Ischaemic heart disease. No C.C.F.
66	F.	70	A	A	0.12	0.20	0.14	-	-	?	Hypertension and ischaemic heart disease. No C.C.F.
67	F.	-	R.F.	A ? R.F.	0.16	0.18	0.16 to 0.20	0.08 split	\emptyset	3 yrs. Alive.	Hypertension. Anaemia. Ischaemic heart disease. No C.C.F. Possible rapid filling gallop on P.C.G. also.
68	M.	82	-	A	0.18	0.20	split	-	-	6 mths. Died.	Ischaemic heart disease. No C.C.F. Clinically thought to have split 1st sound.
69	M.	80	-	A	0.18	0.24	0.20	0.10	$\frac{110}{60}$?	Ischaemic heart disease. L.H.B. C.C.F.
70	M.	64	A	A	0.14	0.32	-	-	$\frac{110}{70}$	6 yrs. Alive.	Ischaemic heart disease. L.H.B. No C.C.F.
23	M.	48	R.F.	R.F.	0.12	-	0.10	0.04	$\frac{150}{100}$	3 mths. Died.	Ischaemic heart disease. C.C.F. Auricular fibrillation.
25	M.	-	R.F.	R.F.	0.14	0.18	0.16 to 0.20	0.05 to 0.10	$\frac{150}{80}$	1 yr. Died.	Ischaemic heart disease. C.C.F.

* A = Auricular gallop. R.F. = Rapid filling gallop.

‡ The auricular gallop sound was also palpable in this case.

∅ Blood pressure originally 140/90, but rose subsequently to 200/120.

L.V.F. Left ventricular failure.

C.C.F. Congestive cardiac failure.

L.H.B. Latent heart block.

TABLE 19

CASES OF RIGHT BUNDLE-BRANCH BLOCK

Case No.	Sex	Age	Diagnosis of * Type of Gallop		E.C.G. measurement in seconds		Approx. P.C.G. measurement in seconds		Blood Pressure	Period of follow up	Diagnosis and Remarks
			By auscultation	By P.C.G.	QRS	PR	1st sound	2nd sound			
71	M.	56	A*	? A	0.12	0.16	0.20	0.10	$\frac{180}{110}$	3 mths. Died.	Ischaemic heart disease and hypertension. No frank congestive failure. See Text for comments on nature of gallop sound.
72	M.	67	A	A	0.14	0.20	0.20	-	$\frac{190}{110}$	18 mths. Alive.	Hypertension. Chronic Bronchitis & emphysema. Congestive cardiac failure. Gallop heard loudest in epigastrium.
73	M.	69	R.F.	R.F.	0.16	0.22	0.16	0.05	-	?	Ischaemic heart disease, Congestive cardiac failure. Latent heart block.
74	M.	69	-	-	0.12	-	0.18 split	0.04 - 0.08	-	8 yrs. Alive.	Ischaemic Heart Dis. & Diabetes. Congestive Cardiac Failure. Auricular Fibrillation. Marked splitting of first sound seen on P.C.G.
75	M.	59	? A ? split 1st sd.	A	0.12	0.20	0.10	0.04 -	-	?	Ischaemic heart disease, chronic bronchitis & emphysema. Hypertension Congestive Cardiac failure. See text for comments on gallop sound.
50	M.	45	A	A	0.12	0.12	0.2	0.1	-	3 yrs. Alive.	Mitral stenosis. Left ventricular failure. Subjected to successful valvotomy

* A = Auricular gallop. R.F. = Rapid filling gallop.

* Auricular gallop best heard and also palpable in epigastrium.

SUMMARY AND CONCLUSIONS

1. The literature concerning the heart sound mechanisms in Bundle-Branch Block and their phonocardiographic characteristics is reviewed and discussed.
2. Case histories and a selection of recordings from sixteen patients are presented and analysed. The view shared by many authorities that Bundle-Branch Block cannot usually be detected by auscultation, palpation or phonocardiogram is confirmed.
3. The re-duplicated palpable apical impulse described by King and McEachern in 1932 was not noted in any of these cases. Such a physical sign should be easily detected also in apical phonocardiograms recorded with a low-frequency band and would appear as a broad splitting of the sound into two components each consisting of fairly coarse vibrations. In this study a pronounced first sound splitting of this type was only encountered once. This was in Case No. 74, where such a split first sound was both heard on auscultation and recorded on the phonocardiogram.
4. A palpable auricular gallop which might be confused with a double apical impulse was, however, encountered in two cases. Graphic sound recordings proved this to be a true gallop rhythm and not a double ventricular impulse.
5. Analysis of the main clinical and phonocardiographic features of this series is set out in Table 18 and Table 19. In all, eight of the ten cases of left Bundle-Branch Block were thought

to have a gallop rhythm on auscultation (rapid filling gallop in three and auricular gallop in five); one was thought to have a split first sound, and one normal dual rhythm. Of the six cases with right Bundle-Branch Block, four had a definite gallop rhythm on auscultation (auricular in three and rapid filling in one); a clearcut splitting of the first sound was heard in one, and in the sixth case there was some doubt as to whether an auricular gallop or a split first sound was present. In the twelve cases with an audible gallop the phonocardiogram was confirmatory in eleven; in the twelfth (Case No. 67) a rapid filling gallop was diagnosed on auscultation, but the phonocardiogram showed a definite loud auricular gallop sound with a possible faint rapid filling gallop visible on the tracing as well. It seems unlikely that this latter sound could in fact have been audible. Of the remaining four cases, two showed an auricular sound on the phonocardiograph tracing when it had not been audible (in one of these a split first sound had been clinically diagnosed). In one case, broad splitting of the first sound was heard and demonstrated on the sound tracing, and in the last case there was some doubt as to whether a split first sound or auricular gallop was being heard, but the phonocardiograph showed an auricular gallop.

6. The auricular gallop sound commenced before the Q wave of the E.C.G. in all cases but one, in which it commenced just after the Q wave but before the peak of R, and in this case the gallop was palpable as well as audible and its phonocardiographic appearance and auscultatory character was

typical of the auricular gallop. In those cases where a jugular phlebogram is available the auricular gallop coincides with the 'a' wave. Two cases had auricular fibrillation, and in these, no phonocardiographic vibrations were seen before the R wave of the E.C.G. One of these two cases had a rapid filling gallop. This gallop sound cannot, therefore, be related to asynchronous ventricular contraction, but must be associated with auricular systole. Ventricular extrasystoles were seen on the tracings of three cases with auricular gallop, and in these the gallop sound did not occur in association with the ectopic beat.

7. In all the cases in this series where a gallop sound was heard or demonstrated on the P.C.G. recording there was also accompanying evidence of cardiovascular disease in addition to the presence of bundle-branch block. The bundle-branch block and the gallop rhythm were presumably both secondary to primary cardiovascular disease. At least five patients had hypertensive heart disease, eight had either right or left-sided cardiac failure or both, one had chronic bronchitis and emphysema in addition to hypertension, one had a severe anaemia but also developed hypertension subsequently, and one patient had mitral stenosis and the Wolff-Parkinson-White syndrome. An unequivocal latent heart block was present in three cases (P.R. interval being 0.22 second or more), and the P.R. interval was 0.20 second in another five cases. In these latter five cases an auricular gallop was heard in three, queried in one, but visible on the P.C.G. in

all five. In the three patients with unequivocal latent heart block an auricular gallop was heard only in one. It was visible on the P.C.G. in this case and one other in which it had not been detected clinically. The third case had an audible rapid filling gallop confirmed by P.C.G., but no auricular sound. An auricular gallop was, therefore, more commonly due to hypertensive or ischaemic heart disease associated with frank or incipient congestive cardiac failure than to prolonged auriculo-ventricular conduction time associated with Bundle-Branch Block.

8. Three patients had a loud auricular gallop well heard on auscultation over the epigastrium near the Xiphoid process. These may presumably have been examples of Potain's right-sided gallop. One had hypertension and a recent myocardial infarction with no obvious congestive cardiac failure, but possible early left ventricular failure (Case No. 71). The second had hypertension and also chronic bronchitis and emphysema with congestive cardiac failure. The third had mitral stenosis, Bundle-Branch Block and the Wolff-Parkinson-White syndrome (Case 50). However, the gallop was not well heard to the right of the sternum in any of the three.
9. In three cases an inaudible rapid filling sound was demonstrated on the phonocardiogram in addition to an auricular gallop.
10. Systolic murmurs were encountered and recorded in a number of cases, but no diastolic murmurs with the possible exception of Case No. 50, in which there was the unusual combination of mitral stenosis and a pre-systolic gallop rhythm as well.

11. The approximate duration of the first and second heart sounds has also been measured and is included in Table 18 and Table 19. As has already been indicated, this measurement cannot be made accurately in the absence of a calibration system, but even if allowances for approximation are made it can be seen that both the first and second heart sounds in Bundle-Branch Block tend to be longer in duration than the normal sounds (see Table 2 and Table 7). In those cases with auricular gallop the beginning of the first sound has been measured from the commencement of the first set of vibrations occurring after the Q wave of the E.C.G. In no case was this increased duration detected clinically. The average duration of the first sound in this series of sixteen cases with Bundle-Branch Block was approximately 0.17 second and of the second sound, approximately 0.08 second.
12. Definite splitting of the second sound was seen on the P.C.G. tracings in only four patients, all with left Bundle-Branch Block. It was heard in one further patient, but not demonstrated on the P.C.G. Paradoxical splitting of the second sound was not encountered, but it was not specifically sought. Abnormally broad splitting of the degree which might be expected to occur in right Branch-Bundle Block (Leatham, 1954) was not seen at all. It is possible, however, that this failure to demonstrate more split second sounds may have been partly due to failure of technique. This is suggested by the fact that the duration of ~~most~~ the second sound was ^{often} ~~probably~~ prolonged even though a clear splitting was not demonstrable.

Apart from this increased duration, the appearance of the split sounds that were recorded did not differ from those encountered previously in normal hearts.

13. In only one case (No. 67) was there a significant discrepancy between clinical and phonocardiographic findings, and the explanation for this is not clear. In several cases, however, gallop sounds were seen on the P.C.G. tracings when they were not heard on auscultation.
14. Of the sixteen patients, no followup was available in four. Of the remaining twelve, six were alive three or more years after they were first seen, three being alive after five or more years. Special mention should be made of Case No. 64, who was alive and well five years after bilateral lumbar sympathectomy for severe benign essential hypertension. During the whole of this period an auricular gallop was audible and there was no evidence of an auriculo-ventricular conduction defect. Two other patients with ischaemic heart disease had an audible auricular gallop which persisted for at least six years, but one had a classical latent heart block. Five of the sixteen patients died within twelve months of the gallop being first detected and recorded. Post-mortem followup was available in two of these, and in both, evidence of ischaemic heart disease was present. One other patient died of a cerebral thrombosis and one of multiple carcinomatosis, but no autopsy was performed in either case. Although numbers are not sufficient to draw

any statistically significant conclusion, it can be seen from Table 18 and Table 19, that the presence of an audible auricular or rapid filling gallop did not appear to have any influence on the prognosis in most of these cases with Bundle-Branch Block. The presence of an auricular gallop did not seem to be necessarily dependent on the existence of congestive cardiac failure. As far as this series is concerned, it is clear that an auricular gallop rhythm may persist long after the signs of frank congestive failure have disappeared and even after the patient has returned to full activity. In one case with a rapid filling gallop the extra sound disappeared as the congestive failure was successfully relieved, but in two others the patient died inside twelve months. All had congestive cardiac failure.

THE SYSTOLIC CLICK

A sound heard usually in late systole, but sometimes in mid or early systole, has been frequently reported and included by some as one of the variety of triple rhythm. It is, however, a high-pitched "clicking" sound and does not have the normal cadence so characteristic of physiological triple rhythm or of gallop rhythm.

This phenomenon was first described by Cuffer and Barbillon in 1887 as a meso-systolic gallop, and later, in 1900, by Potain, who called it a systolic gallop rhythm, and by Gallavardin, in 1913, who called it a mid-systolic click. In 1927, Holt queried its existence as a clinical entity and felt certain that it had no relation to gallop rhythm. In 1928, Paul White described four cases of systolic gallop out of 100 seriously-ill patients all having one form or other of heart disease with gallop rhythm. His cases were not proved by phonocardiography, but a clinical diagnosis of systolic gallop was made.

Lian and Deparis carefully analysed the subject in 1933 and considered it to be an innocent phenomenon. They described the sound as "La claquement meso-systolique pleuro-pericardique" and personally observed fifty cases. Phonocardiography showed the sound to be sometimes in early, sometimes in mid, but usually in late systole, and often followed by a late systolic murmur. It was usually heard loudest at the apex, it sometimes varied with respiration and posture, and it was described as a sharp, dry sound. According to Lian, patients with this systolic click have no evidence of heart disease, but may have a history of previous pleurisy or pericarditis, and in a series of four autopsies he

was able to demonstrate pleuro-pericardial adhesion, but no other cardiovascular abnormality. Many of these patients have a cardiac neurosis and complain of chest pain of a stabbing infra-mammary type. Lian believes that this is usually a doctor-induced cardiac neurosis as a result of the auscultatory abnormality being misdiagnosed as a gallop rhythm.

Thompson and Levine (1935), without phonocardiographic proof, diagnosed 35 cases aged from 11 to 73 and seen over a period of eleven years. They comprised 16% of all "gallop rhythms" seen in this period. The authors agreed that the sound varied with posture and might be transient. Two-thirds of their cases had no evidence of heart disease but had a cardiac neurosis; ~~but~~ the remainder had a mild degree of ischaemic or hypertensive heart disease. There were no deaths during the period of observation. Johnstone (1938) saw 21 cases in five years and proved them phonocardiographically. Most were psychoneurotic and in the younger age-group, and none had organic heart disease. He agreed with Lian as to the significance of the probable aetiology of the condition, but he was unable to demonstrate any pleural or pericardial abnormality from radiological or other investigation, and none of his patients died.

Orias, Braun-Menéndez (1939) found no cases of systolic gallop, but confirmed the existence of an innocent systolic click. Wolferth and Margolies (1940), on the other hand, thought that both conditions could be found. They agreed that the innocent systolic click is much commoner, but they describe two cases of a systolic gallop. Both of these patients

had well-developed aortic incompetence, and these workers believed that the gallop sound was probably due to a sudden checking of the movement of distension of the aorta or by its impact against surrounding structure or possibly to the impact of the cardiac apex against the chest wall. They are doubtful as to the significance of this phenomenon and consider it to be very rare.

Evans (1943) includes systolic triple rhythm in his classification, but thinks it to be rare and of no pathological significance. Frost (1949) observed systolic "clicking", but thought the extra sound to be unlike the low-pitched gallop sound. Frost felt that the systolic gallop described by Thompson and Levine (1935) as occurring in 16% of their gallop rhythm series could not have been due to the same sound phenomenon in every case, especially as there was no phonocardiographic proof and a third of the patients had heart disease. It seems probable that these figures were based on a different definition of gallop sounds. Frost is in agreement with Johnstone (1938), Gallavardin (1931), and Lian (1933) that the majority, if not all, of the so-called systolic gallop rhythms are due to an innocent systolic click occurring in healthy hearts.

Leatham and Turner, writing independently in 1949, mention this phenomenon as being an uncommon, almost always innocent occurrence. In 1954, Leatham, when discussing the causes of splitting of the first and second heart sounds, describes broad splitting of the basal first sound as being sometimes due to a "pulmonary early systolic click". This he ascribes to the vibrations set up by early systolic ejection of blood into a dilated

pulmonary artery. This may also occur in the aorta under similar circumstances and may well be a similar phenomenon to that described by Wolferth and Margolies (1940). In these cases it is clearly pathological.

PRESENTATION OF CASES

One typical example of what is thought to be the systolic click has been encountered and a phonocardiograph recording made.

This was patient No. 76, a housewife aged 48. She was admitted to hospital for treatment of a gastric ulcer and also had chronic rheumatoid arthritis. On auscultation a curious sound could be heard just before the second sound and giving rise to an atypical "triple rhythm". The first and second heart sounds were normal and there were no murmurs. Just before the second sound, but quite distinct from it, there was a loud metallic clicking sound, and the accent was on this extra systolic sound. It did not vary with respiration or posture and was heard loudest at the apex, but fairly widely over the praecordium as well at all areas but the aortic. In every other way the heart appeared normal clinically, radiologically and electrocardiographically. Two years after the recording, this patient had a partial gastrectomy for the recurrent gastric ulcer, and three years later she was alive and well and symptom-free.

The P.C.G. (A) shows an extra sound occurring just before the second sound, and tracings taken with higher frequency band (B) also show an early systolic murmur which was not heard clinically.

It is presumed that this extra sound is an example of the systolic click or the "claquement meso-systolique pleuro-pericardique" described by Lian and his colleagues and thought by him to be associated with pleuro-pericardial adhesion, but to be without serious significance. In this present case there was no evidence of adhesion or other cardiac abnormality.

The normal physiological splitting of the second sound can be seen in the tracing taken from the pulmonary area, and can be seen to vary, presumably with respiration. The systolic click should not be confused with this splitting. The second part of a split always commences after the beginning of the second sound, as can clearly be seen in the recordings from the pulmonary area, where it is usually best heard and recorded. The systolic click is maximum at the apex and in this case begins 0.06 second before the second sound, to which it bears a fixed relationship. Cases Nos. 77 and 78, in the systolic murmur section of the thesis, had similar "systolic clicks" visible on their phonocardiograph tracings.

SUMMARY AND CONCLUSIONS

1. Systolic clicks are not unduly rare, may be associated with pleuro-pericardial adhesion, but are not a sign of heart disease. They often accompany psychoneurosis.
2. For this reason and because they do not in fact sound like the classical gallop rhythm which is almost always associated with heart disease, and because they may, unlike the physiological third heart sound, be heard at all ages, systolic clicks should be classified separately and not referred to as "systolic gallop".
3. One such case is described, in which a late systolic click occurred in a patient with a normal heart. Other similar cases are referred to. In none of these cases was there any definite evidence of heart disease.

THE CARDIAC MURMURS

CLASSIFICATION

Classification and descriptions of the various heart murmurs and their mechanism can be found in many standard textbooks on clinical heart disease and in particular the works of Paul White (1937), Levine (1937), Sir Thomas Lewis (1942), Wiggers (1949), and Paul Wood (1950).

Orias and Braun-Menéndez (1939) and Levine and Hervey (1949) have both published monographs including complete surveys of the murmurs to be heard on auscultation, together with phonocardiographic illustrations.

Murmurs may be classified as organic, that is to say being due to structural heart disease, acquired, congenital or functional. The term 'functional' is most unsatisfactory as it embraces a wide variety of conditions known and unknown, varying from truly innocent murmurs to the murmurs occurring in severe anaemias and due to undiagnosed organic heart disease. The use of the term 'unexplained murmur' in place of 'functional murmur' is advocated in the report published by the New York Heart Association in 1946 on the nomenclature and criteria for diagnosis of diseases of the heart.

Murmurs may also be classified according to the time of their occurrence in the cardiac cycle as being systolic, early diastolic, mid-diastolic, presystolic, and continuous. Evans (1947) divided systolic murmurs into pan-systolic, early, mid, and late systolic.

Thayer and MacCallum (1906) produced a variety of valvular lesions in the experimental animal and by direct auscultation were able to reproduce and recognise the traditional murmurs of acquired and congenital heart disease. They made the important observation that a pulmonary systolic murmur could be produced very easily by even the slightest pressure on the pulmonary artery or manipulation of the pulmonary conus area. A systolic murmur was also produced while a saline infusion was given and the characteristics of the normal blood flow thereby altered.

RECORDING OF MURMURS

In 1912 Sir Thomas Lewis produced a beautifully clear and concise account of the phonocardiography of murmurs found in mitral stenosis, with illustrative recordings. Later, Battaerd (1915) gave a full account of the history of phonocardiography, but was not himself able to reproduce very satisfactory recordings. Further recordings and reports on the general subject of heart murmurs have been published by, among others, Wiggers (1918), Lian and Racine (1933), Arenberg (1940) and Leatham (1949).

THE FREQUENCY RANGE OF MURMURS

The approximate frequency range of heart sounds and murmurs has already been described, and it has been seen that the sounds, although of much greater intensity, occupy a relatively lower frequency band than the murmurs. Heart sounds occupy the range of approximately 25 to 400 cycles per second, with the fundamental frequency mostly below 100 c/s. Low-pitched murmurs are

mostly in the range 50 - 400 c/s., medium-pitched 240 - 400 c/s., and high-pitched 240 - 660 c/s., with some of the early diastolic murmurs of aortic incompetence occasionally reaching frequencies in the 660 - 1,000 c/s. range. (Cabot and Dodge, 1925; Williams and Dodge, 1926; Lockhart and McKee, 1938).

To recapitulate briefly on earlier discussions: the ear is relatively insensitive to lower frequencies, but sensitive to the higher, and so the low-pitched sounds such as the third and fourth heart sounds may be difficult to hear, but high-pitched murmurs relatively easy. On the other hand, the phonocardiograph, particularly because of the inability of its galvanometer to respond to higher frequencies, records the low-pitched sounds better than the higher-pitched murmurs. However, the apparatus used in this present investigation did in fact respond fairly well to most of the murmurs encountered, including the early diastolic murmur of aortic insufficiency.

Phonocardiographic studies of the murmurs associated with specific valvular and other cardiac lesions have been made by numerous workers. These will be referred to in later sections of the thesis in which the auscultatory signs of mitral stenosis and incompetence, aortic stenosis and incompetence, congenital heart disease, and other miscellaneous cardiovascular lesions are all considered independently. In a separate category, however, come the cases with systolic murmurs of doubtful aetiology, functional or "unexplained" murmurs where rheumatic or congenital heart disease was not thought, at least initially, to be present. It is worth while firstly to discuss the subject of these systolic murmurs, their significance and the frequency with which they occur, before studying the appropriate recordings.

SYSTOLIC MURMURS

In his article entitled, "The Systolic Murmur. Its clinical significance", Levine (1933) emphasised the importance of neither under- nor over-estimating the significance of a systolic murmur. He classified these murmurs into six grades of intensity, but, being an auscultatory classification, this is inevitably unscientific because of the subjective factors involved and the variation in auditory characteristics of individual observers. Levine points out that systolic murmurs may be due to mitral incompetence resulting from primary disease of the valve itself or secondary to generalised cardiac dilatation in the presence of a normal valve. They may be due to hypertensive heart disease, anaemia, fever, thyrotoxicosis and a number of other conditions. He points out that 90% of normal people develop a transient apical systolic murmur after brisk effort. In a survey of 1,000 children and adults considered initially to be "normal", 17 were found to have a grade III ("moderately loud") murmur and all had obvious heart disease when examined carefully. 196 had a grade I or II systolic murmur ("faintest possible" murmur that can just be heard or murmur classed as being "slight"), and of these only 45 were found to be completely free of signs, symptoms or history of heart disease on examination. Levine concluded, therefore, that in his series only 4.5 % of patients had an unexplained systolic murmur with no detectable evidence of heart disease.

The phonocardiographic aspect of these systolic murmurs has received special attention from several workers. McKee, using the Lockhart stethograph in 1938, found that in 90% of 105 normal children between 5 and 17 years of age there was a systolic murmur which could be demonstrated graphically, whereas in only 5% could the murmur be heard clinically. This systolic murmur was often very short and of low intensity. In 119 children of the same age group, but all suffering from rheumatic heart disease, a systolic murmur was frequently heard and recorded and was often indistinguishable in any way from that heard or seen in healthy children. McKee reported, however, that the systolic murmur in the group of children with organic heart disease tended to be both louder and of higher pitch than the murmur in the healthy group. Rappaport and Sprague (1943), recording a series of 33 normals, found a systolic murmur in 58% of stethoscopic and 85% of logarithmic phonocardiograms.

Mannheimer, in 1940, although reviewing the entire subject most thoroughly, confined his investigation to 135 healthy children and 135 with congenital heart disease. In three-quarters of the normal children a systolic murmur was registered, usually of a fairly low frequency. (in 75% of the cases the murmur was of a frequency between 50 and 175 c/s.) and low amplitude. Mannheimer emphasised the fact that these faint systolic murmurs are very common in normal healthy children and must not be mistaken for the murmur of organic heart disease. In the 135 cases of congenital heart disease a variety of systolic murmurs were encountered; the

systolic murmur was registered in more than 90% of the cases and was both louder and occupied a higher frequency range than the normal. In over 90% the murmur occupied the frequency range 50 to 500 c/s. and in many extended up to the 500 to 1,000 c/s. range as well. Continuous murmurs were demonstrated in 10 cases of this series and diastolic murmurs in 2.

William Evans (1947) has discussed the subject in some detail and analysed the characteristics of heart murmurs, illustrating many of his points with phonocardiograms recorded by Leatham. He uses a line drawn through the end of the the S-wave of the E.C.G. as a landmark in differentiating between auricular and ventricular systole. In a series of 330 patients with innocent systolic murmurs he found that the murmur was always mid or late systolic in timing and commenced well after the S line. In 74 cases with mitral stenosis he found that the systolic murmur, when present, was always early or pan-systolic and started with or before the S line. Forty cases of aortic stenosis, 43 cases of hypertension, and a smaller number of cases with congenital heart disease, heart block and anaemia were also analysed. With the exception of the murmurs of heart block, hypertension and some cases of aortic valvular disease, Evans concluded that the systolic murmur of organic heart disease always commences with or before the S line and the murmur of functional heart disease always starts well after the S line.

Cowen and Parnum (1949) were unable to substantiate several of Evans' claims. They found that in a series of 84 cases with systolic murmur, both organic and functional (unexplained), no distinction could be made between the two types of murmur by

timing the onset of the murmur with the S line of the E.C.G. Unlike Evans, Cowen and Parnum were unable to obtain any additional evidence over and above that obtainable as a result of clinical study in 30 cases of mitral stenosis and in 20 cases of aortic incompetence. Finally, they were also unable to substantiate Evans' claim that an inaudible aortic early diastolic murmur could be demonstrated with the aid of a phonocardiogram in a significant proportion of hypertensives.

Evans has since repeated his claims (1951) and still believes that organic and functional systolic murmurs and the auscultatory signs of rheumatic and congenital heart disease can only be accurately assessed by means of phonocardiography.

There is sometimes difficulty, when analysing the records of a systolic murmur, to decide where the first heart sound finishes and the murmur commences. Frequently it may be necessary to decide whether certain vibrations in early systole are due to a prolonged first heart sound or a short early systolic murmur. This may be done in a variety of ways. The total duration of the first sound is rarely more than 0.16 second (stethoscopic) or 0.13 second (logarithmic), and so an idea of the point at which the sound ends and the murmur commences may be obtained by simple measurement. The change in frequency may also be of help because, as Rappaport and Sprague (1941 and 1942) have pointed out, the terminal vibrations of the first sound are always coarse and of a low frequency whereas the vibrations of a systolic murmur are always considerably higher. A jugular phlebogram, if available, will be of assistance because the end of the first sound always occurs before or, at the latest, on the peak of the "c" wave.

Making allowance for the difference in duration of the sound recorded from different areas and also when recording with different frequency bands, the position of the first sound on one tracing can also be checked by comparing it with a second synchronous recording from the pulmonary or aortic areas or with a high frequency recording in which the vibrations due to the murmur will have been largely cut out.

Systolic murmurs, thought to be innocent and unassociated with heart disease, have been demonstrated on the recordings of five of the normal subjects illustrated in earlier sections. These were cases No. 1, 2, 5, 10 and 14.

A further seven cases are described here. The first three were thought to have normal hearts with "innocent" late systolic murmurs. The fourth had thyrotoxicosis with an early systolic murmur, and the last three all had serious heart lesions with loud pan-systolic murmurs.

PRESENTATION OF CASES WITH SYSTOLIC MURMUR

CASE No. 77, a woman of 49, suffered from rheumatoid arthritis and also a cardiac neurosis. There was a history of possible rheumatic fever at the age of 20, at which time she was told that her heart was diseased. Following this, she developed left mammary pain and had two years of comparative invalidism. She subsequently married and bore four children without difficulty. There was no history of congestive failure, but she complained of palpitation and chest pain.

Physical, electrocardiographical and X-ray examination revealed no cardiac abnormality other than a systolic murmur. The heart was not enlarged and B.P. was normal. On auscultation the murmur was maximum at the apex, but heard well down the left sternal edge, it was classed as moderate to loud in intensity (grade III or IV, according to Levine), and was loudest in late systole. The murmur could also be detected at the pulmonary and aortic areas and was just heard at the root of the neck. The first and second heart sounds were normal, although the second sound at the apex was partly obscured by the murmur. When last seen, three years after the recordings were made, she was alive and well, but the rheumatoid arthritis was still troublesome and the physical signs unchanged.

Phonocardiograms show the systolic murmur quite clearly, recorded from both apical and pulmonary areas. It is maximum in, but not confined to, late systole. It occurs well after the S line of the E.C.G. Records taken at different recording paper speeds with high frequency band (A) and (B) are shown. There is a clear accentuation of the early part of the apical systolic murmur which occurs in mid-systole and might well represent the systolic click referred to earlier. It was, however, not audible as a separate sound distinct from the systolic murmur. Record B shows a ventricular extra-systole followed by a compensatory pause. In a third record (not mounted) a tiny rapid filling sound occurring 0.15 second after the second sound is seen, but was not audible.

Without further followup the presence of a valvular lesion such as mitral incompetence cannot definitely be ruled out here, but the murmur has all the characteristics of a "functional" late systolic murmur, and there was no evidence of left ventricular enlargement or systolic expansion on X-ray screening. It is worth noting that several of the cases of pure mitral incompetence described by Brigden and Leatham (1953) had murmurs confined to late systole, and it is clear that such murmurs are not always innocent.

A case similar to No. 77 was Case No. 78, another female, aged 57, who was also thought to have a cardiac neurosis. She complained of dyspnoea on exertion and palpitation of six months' duration, but she had no history of rheumatic fever or other cardiovascular symptomatology. On examination there was no evidence of congestive failure and the B.P. was $\frac{130}{85}$. The apex beat showed a slight systolic retraction between the fourth and fifth ribs. On auscultation there was a loud first heart sound with slight to moderate systolic murmur (grade II to III) maximum at the apex but heard all over the praecordium and not conducted up into the neck. The murmur was loudest in late systole and at the apex obscured the second sound, which was loud and clearly split at the pulmonary area.

X-ray screening revealed slight left ventricular enlargement, but no systolic expansion or other abnormality. E.C.G. was normal. Six years later she was alive and well, but there was some increase in the size of the left ventricle.

Phonocardiograms taken from the apex and pulmonary area with both low and high frequency bands and slow and fast motor speeds are shown. The systolic murmur does appear to be maximum in late systole, but probably starts immediately after the first sound and well after the S wave of the E.C.G. The second sound is seen to be clearly split into aortic and pulmonary components in all the tracings and, as in case No. 77, there is an accentuation of the systolic murmur which looks like a systolic click occurring in early to mid-systole. This was not audible as a separate sound, but is clearly seen, particularly in the higher frequency tracing C. A few low-frequency vibrations in early diastole can be seen on the apical tracing (Record A). It is most unlikely that these do in fact constitute a true early diastolic murmur, because they are only seen at the apex and there is no sign of such a murmur on the high-frequency tracing from the base. The murmur of aortic or pulmonary incompetence always shows up best in the high-frequency ranges.

This case has a pan-systolic murmur, a split second sound, and a possible systolic click, with no clearcut evidence of cardiac pathology. Final followup must, however, be awaited before any more definite conclusion could be drawn.

CASE No. 79, a male aged 50, also had a late systolic murmur which was described as a "typical late innocent systolic murmur". On clinical, electrocardiographic and radiological examination no other cardiovascular abnormality was discovered, although he was suffering from a definite cardiac neurosis as a result of ill-advised previous medical attention. On careful auscultation a loud high-pitched systolic murmur (grade III to IV) was heard maximal at the apex and loudest in late systole.

Seven years later he was alive and well, with no change on clinical or radiological examination.

Apical phonocardiographic recordings show the murmur clearly maximum in late systole and starting well after the first sound; unfortunately there is no tracing from the pulmonary or aortic areas and the second sound cannot therefore be properly studied. Judging from these records and the jugular venous pulse tracing, the main vibrations at the end of the murmur are probably due to aortic closure and represent the first part of the second sound; the small following vibration may be the pulmonary component of the second sound.

CASE No. 80, a female aged 46, had thyrotoxicosis with typical physical signs. She had no cardiovascular abnormality other than a faint systolic murmur heard at the apex (grade I) and a somewhat louder systolic murmur at the pulmonary area (grade II). The first and second sounds were normal. Phonocardiograms taken from the pulmonary area and apex show the murmur to be clearly maximal in early systole, ending before the second sound which is not split.

CASE No. 81, a male aged 76, proved to be a most interesting case. He was admitted to hospital complaining of weakness, dyspnoea and cough with haemoptysis. On physical examination there were no signs of congestive cardiac failure, but there was a tachycardia with heaving apical pulsation in the fifth intercostal space just in the mid-clavicular line. On auscultation a very loud (grade V) harsh systolic murmur was to be heard, which seemed to be accentuated in late systole.

This murmur was loud at the apex, but maximal in the third left space. The first and second sounds were thought to be probably normal, but much obscured by the murmur. An auricular gallop or broadly split first sound was also queried by one observer. The T-wave in the E.C.G. leads V 1,2,3,4 was inverted and the initial diagnosis was one of ventricular septal defect with a probable unconnected pulmonary lesion. The patient died two weeks after admission, and at autopsy the heart was found to be compressed from behind forward by a carcinoma mass arising from the left lower lobe bronchus and almost as big as a football. This tumour was an oat-cell bronchogenic carcinoma and had invaded the pericardium and the wall of the left auricle, but otherwise the heart itself was normal and there was no valvular lesion or septal defect.

Phonocardiograms are shown, taken from the apex, using two different frequency bands (Records A and B), and from the third left space, using the same frequency bands (Records C and D). The murmur is best shown in the high-frequency tracings (B and D); it is pan-systolic and largely obscures the heart sounds; there is a definite auricular systolic sound seen best in the low-frequency tracing taken from the third left space (C) and occurring during the P-R interval.

The systolic murmur in this case must have arisen as a result of the cardiac deformity resulting from the compressing tumour mass. It may well have had a similar mechanism to the systolic murmur produced experimentally by Thayer and McCallum in 1906 by manipulation of the pulmonary conus in the normal dog's heart. The auricular gallop was also presumably due to early failure resulting from the mechanical cardiac embarrassment and also the direct involvement of the left auricle.

CASE No. 82, a male aged 54, also presented some very interesting features. This man had been perfectly healthy until one month before admission, when he developed classical symptoms of angina pectoris, with two particularly severe episodes. He also had paroxysmal nocturnal dyspnoea. On examination after admission there were signs of early congestive failure to be found, the apex beat was displaced to 1" outside the mid-clavicular line, and there was a very loud (grade V) apical systolic murmur and thrill. This murmur was also heard all over the praecordium, but was not propagated up into the neck. The pulmonary second sound was accentuated. The only E.C.G. abnormalities were inverted T-waves in unipolar leads recorded from the second and third intercostal spaces and the T-wave in Lead V 4 was of unusually high voltage.

The patient made an excellent recovery, no further symptoms developed, and the signs of cardiac failure all disappeared. There was no evidence of mitral insufficiency or other lesion on X-ray screening of the heart. It was concluded that the patient had probably suffered a small myocardial infarction and as a result of this had ruptured one of the papillary muscles in the left ventricle. This man was last seen three years later, when he was quite symptom-free and able to undertake all normal physical activities. He had no symptoms of angina pectoris, and on auscultation the loud grade V systolic murmur and thrill were still present. E.C.G. at this time was normal.

Phonocardiograms A, B and C, taken from the apex and pulmonary artery with different frequency bands, show this loud pan-systolic murmur occupying the whole of the space between the first sound and the pulmonary component of the second sound. According to Leatham, the systolic murmur of aortic stenosis finishes before the pulmonary second sound. Several of these features are of interest. Firstly, there is an auricular vibration seen best in the low-frequency tracing from the apex (A), and in this record also there is a probable rapid filling sound also seen in the apical tracing and occurring just over 0.1" after the beginning of the second sound. This gallop sound was not audible, but might well have been present due to the cardiac failure. It is also possible that an early diastolic murmur is present in tracing B and in tracing C, but this may be confused with the gallop and the fact that the end of the second sound is not quite clear on these records. Certainly no other signs of aortic valvular disease were present.

A female, aged 63 (Case No. 83), was thought to have mitral incompetence. She gave a four-year history of exertional dyspnoea and chest pain related to exertion. Recently she had suffered repeated attacks of paroxysmal nocturnal dyspnoea. There was no relevant previous history. On examination there was no evidence of congestive cardiac failure. Blood pressure was $\frac{170}{100}$. The apex was displaced to the left, and on auscultation there was a moderate grade III systolic murmur maximal at the apex, but also heard at the aortic area. The apical first sound was normal, but the aortic second sound was inaudible. No diastolic murmur or other abnormalities were heard. There was no thrill.

Considerable left ventricular hypertrophy was evident from E.C.G. and X-ray screening. The patient made a good recovery without the use of drugs. On discharge she was symptom-free, the auscultatory signs were unchanged, and the B.P. was $\frac{130}{80}$. She died seventeen months later, and at autopsy gross calcified aortic stenosis was found to be present.

Phonocardiograms taken from the apical (A) and aortic area (B and C) show the typical murmur of diamond shape which occurs in aortic stenosis beginning just after the first sound and finishing before the second and maximum in mid-systole. There is no visible early diastolic murmur, and on the apical tracing splitting of the second sound is visible. The clinical diagnosis here had been one of mitral incompetence; the P.C.G., however, was quite typical of aortic stenosis (Levine and Harvey, 1949; Leatham, 1951), and this diagnosis was ultimately confirmed at autopsy.

SUMMARY AND CONCLUSIONS

1. The classification, frequency ranges, and literature describing cardiac murmurs have been briefly referred to.
2. Systolic murmurs of unknown aetiology or associated with certain types of cardiovascular disease have been discussed in more detail and the frequency with which they occur and their characteristics outlined.
3. Five cases of innocent systolic murmur not thought to be associated with heart disease are referred to, their detailed histories and recordings being presented in the section dealing with normal heart sounds and physiological splitting of the sounds.
4. A further seven cases are described in detail. The first three are classified as having murmurs maximal, but not confined to late systole. In all three no definite evidence of heart disease was present. In two of them there was an accentuation of the systolic murmur seen on the P.C.G. occurring in mid-systole which had the appearance of the systolic click, but was not heard as such on auscultation. One case had thyrotoxicosis and an early systolic murmur. Three cases had loud pansystolic murmurs; in the first this was apparently due to distortion of the heart as a result of a tumour mass occupying the retro-cardiac space and compressing the heart. In the next, the most likely diagnosis was one of a ruptured papillary muscle; and in the last, a clinical diagnosis of probable mitral incompetence was correctly changed to one of aortic stenosis following a study of phonocardiographic tracings. This diagnosis was confirmed at autopsy.

5. In two cases a rapid filling sound was seen on the P.C.G. but not heard. In two cases an auricular sound was recorded; in one of these two such a gallop sound had been suspected clinically.
6. All the systolic murmurs in this small series commenced after the 'S' line of the E.C.G., regardless of whether they were felt to be organic or "innocent". The suggested value of this S line as an aid to differentiation between organic and innocent murmurs has, therefore, not been confirmed.
7. Whereas pan-systolic murmurs appear to be organic in origin, a study of the systolic murmurs recorded in this and other sections reveals that innocent or "unexplained" murmurs not thought to be due to organic heart disease may be early or late systolic in timing. Organic murmurs also have been demonstrated which are confined to either early or late systole.
8. A phonocardiographic study of systolic murmurs alone seems to be of limited practical value, but when all parts of the tracing are examined, including the heart sounds and diastolic period, information of diagnostic importance may often be obtained.

CONGENITAL HEART DISEASE

Few extensive studies of heart sound recordings in congenital heart disease have been made, and although Scandinavian workers have studied fairly large groups of children with congenital heart lesions, this has usually been with some specific and fairly limited purpose in mind. For instance, Carlgren (1946), when investigating the occurrence of gallop rhythm in children, confined his interest to the gallop sounds and did not analyse the other sounds and murmurs in the 306 congenital hearts he studied. Mannheimer (1940) made phonocardiograms from 90 children with congenital heart disease during the course of his study of calibrated phonocardiography and the electrocardiographs of normal children and children with heart disease. He found that the average amplitude of the heart sounds in those children with congenital heart disease was greater than in the normals and that the systolic murmurs were louder and of higher pitch. Although gallop rhythm was never heard in his series, gallop sounds were occasionally demonstrated in the tracings.

In adults with congenital heart disease a gallop rhythm is not a common feature of the auscultatory signs, although a rapid filling gallop is frequently heard in the presence of atrial septal defect and of course may appear with the onset of congestive failure whatever the aetiology.

Various fairly limited aspects of the phonocardiography of congenital heart disease have been considered by Orias and Braun-Menéndez (1939), Evans (1943 and 1947), Leatham (1949), Cowan and Parnum (1949), Levine and Harvey (1949, and Wells(1954).

Heart sound recordings in this series have been made from a small entirely unselected group of cases of congenital heart disease. These are presented here, but no systematic attempt has been made to study this particular subject as in fact only a relatively few cases of congenital heart disease were encountered during the years when the recordings were being made.

The diagnosis is a clinical one in every case and has been made in the light of several years' observation and after full clinical, radiographic, and electrocardiographic investigation, and, in some cases, cardiac catheterisation. Brief resumes of the case histories and clinical findings are given in each case.

PRESENTATION OF CASES

Records were made from two cases of patent ductus arteriosus, both before and after ligation of the ductus, and from one case of pulmonary stenosis, one of ventricular septal defect, one of congenital aortic stenosis, and from one patient with an atrial septal defect with a possible Lutembacher's syndrome.

The two cases of patent ductus occurred in young, otherwise healthy schoolgirls and were quite typical as far as physical signs and history were concerned. Both had only very slight limitation of exertion. In each case auscultation revealed a Gibson continuous murmur, maximum at the second and third left intercostal space in the parasternal line. The phonocardiographic appearance of the murmur was similar to that pattern described

originally by Sir Thomas Lewis in 1913 and later by Leatham (1949), Wells (1952), and Bonham-Carter and Walker (1955). It is continuous throughout the cardiac cycle, but maximum in late systole and early diastole. A third heart sound was demonstrated in one case and was thought to be physiological. It was not heard.

Following ligation of the ductus - in neither case was it divided - the continuous murmur disappeared, but a pulmonary systolic murmur persisted in each case.

Followup over the next four years showed no sign of clinical deterioration in either case, but in both, the continuous murmur recurred. As these children both remained extremely well, continued to develop normally, and showed no evidence of cardiac enlargement or deterioration of the cardiac state, it is difficult to be dogmatic with regard to the aetiology of these recurring murmurs. It does, however, seem probable that recanalisation of the ductus may be occurring.

The first of these two cases, No. 84, was a girl aged 11 years. The cardiac abnormality had been discerned when she was 5 years old, but apart from a very slight degree of limitation of exertion by dyspnoea, she was a perfectly healthy girl. On examination there was no cyanosis, no finger-clubbing, and no sign of cardiac abnormality other than the presence of a loud Gibson murmur maximum at the second left space 1" from the sternal edge, with a palpable systolic thrill. The mitral first and second sounds were loud and clear, the pulmonary second sound was audible, and there was no cardiomegaly. B.P. $\frac{115}{60}$. Femoral and radial pulses were normal.

After the ductus had been tied, only an apical systolic murmur and accentuated pulmonary second sound were noted. Eighteen months later, however, the continuous Gibson murmur had returned, but was not so loud as it had been formerly and there was no palpable thrill. Four recordings were made, one before and three after the operation. A and B were recorded from the pulmonary and mitral areas respectively before operation and show the continuous Gibson murmur maximum at the pulmonary area. C and D were recorded two weeks after operation and show almost complete disappearance of the murmur and the unmasking of a third heart sound seen on the apical tracing. E and F were taken two months later, when the murmur has recurred, and G eighteen months after operation. This last record was made with the Elmquist machine and is, therefore, not strictly comparable with the earlier Boulitte recordings, but shows the Gibson murmur at the pulmonary area, ~~and~~ the apical systolic and third heart sound.

The second case, No. 85, a girl aged 9 years, gave a precisely similar history. Only very slight limitation of severe exertion by dyspnoea was admitted and the child appeared perfectly normal apart from the auscultatory findings. A typical Gibson murmur was heard maximum in the second and third left intercostal space near the sternal edge, with a palpable systolic thrill. At the apex there was a systolic murmur only, the first sound was split, and at the pulmonary area the second sound was accentuated. B.P. $\frac{130}{80}$. No cardiomegaly. When seen two weeks after operation, the continuous murmur had vanished, but three months later a soft continuous Gibson murmur was again audible at the pulmonary area; there was a clear splitting of the first sound at the apex.

Eighteen months after operation the first sound was still clearly split at the apex, where there was a short systolic murmur. The pulmonary murmur was definitely louder, but not nearly as loud as it had been before operation, and there was no thrill. Recordings were made on five occasions - once before and four times after operation. Record A shows the pre-operative tracing from the pulmonary area. B and C were recorded from the pulmonary and apical areas respectively two weeks after operation, and D eighteen months after. This last record, made with the Elmquist machine, shows the clear splitting of the first sound at the apex, the early coarser component occurring before the S wave of the E.C.G., but after Q, and the continuous pulmonary murmur is seen to be maximum in late systole and early diastole. There is some variation in intensity of the murmur with respiration.

Case No. 86 was a girl of 19 who had a mild, uncomplicated congenital pulmonary stenosis. She had no disability and no symptoms. On examination there was no evidence of cardiac enlargement. On auscultation, a loud systolic murmur maximum in the third left space was heard, and there was a markedly accentuated pulmonary second sound and audible third sound. The systolic murmur was accompanied by a palpable thrill. On later occasions the pulmonary second sound was heard to be split, but this observation was not made on the day when recordings were being taken. Phonocardiography in this case showed three things in particular.

Firstly, with low-frequency recording, a third heart sound is clearly recorded from the apex (Record A). Secondly, the pulmonary systolic murmur recorded with a high-frequency band is seen to be high-pitched and to have the diamond shape characteristic

of 'ejection' murmurs (Record B). Thirdly, the pulmonary second sound is seen to be accentuated and finely split when high-speed recordings are made (Record C.)

The pulmonary systolic murmur is pan-systolic, but commences after the S line of the electrocardiogram, it is maximum in mid-systole, but extends right through to the second sound. Evans (1947) maintains that the systolic murmur of pulmonary stenosis, and in fact all organic systolic murmurs, commence at or before the S line of the E.C.G. Cowan and Parnum (1949) could not confirm this observation and found that in 66% of cases with organic heart disease the systolic murmur commenced after the S line of a Lead II electrocardiogram.

It has also been stated that if the pulmonary and aortic components of the second sound are both visible, then the systolic murmur extends as far as, or even beyond, the aortic component, but stops short of the pulmonary component. (Leatham, 1954). This particular relationship is not clear in these recordings.

The patient was alive and well, symptom-free, and with no change in the physical signs four years later, She would appear to fall into the category of mild valvular stenosis described by Wood in 1950, and which differs in many ways from the more severe cases.

Case No. 87 was an adult female thought to have a ventricular septal defect. On examination she was found to have a grossly dilated heart, congestive failure, hypertension, bundle branch block, and auricular fibrillation. A loud systolic murmur and thrill were maximal at the fourth left space near the sternal edge.

On the phonocardiogram the murmur is seen to commence with the first sound and in fact obscures it, becomes maximal in early systole, and finishes well before the second sound. A rapid filling gallop sound, presumably due to the cardiac failure, is clearly seen, but was not heard. The second sound is split. Unfortunately this patient died at home about three years after the records were taken and no post mortem was obtained. The clinical and radiological diagnosis was one of undoubted ventricular septal defect.

Evans (1951) and Wells (1954) maintain that the murmur of ventricular septal defect is truly pan-systolic and fills the whole of systole, reaching right up to the second sound. In case No. 87 the murmur finishes well before the second sound, but without autopsy proof of the diagnosis no further comments can be made.

Case No. 88, a small girl aged 7, was believed to have congenital aortic stenosis. She was completely symptom-free, but a loud systolic murmur was to be heard all over the praecordium. It was maximal just internal to the apex and there was a systolic thrill. The first and second sounds were faint, but a third heart sound could sometimes be heard.

The recording shows a diamond-shaped systolic murmur maximum in mid-systole and clearly finishing before the second sound. The characteristic murmur (Leatham, Levine and Harvey, 1949) can best be seen in the recording from the third left space, using a medium frequency band, and the heart sounds are best seen in the apical low-frequency tracing recorded synchronously. There is no obvious splitting of the second heart sound and the systolic click or ejection sound is not clearly seen, neither was it heard on auscultation.

Case No. 89 was a 62-year-old woman with an atrial septal defect. On examination a loud systolic murmur and thrill were found to be maximum at the third and fourth left intercostal spaces in the para-sternal line, the apical first sound was accentuated, and the pulmonary second sound was not thought to be split. On various occasions both a mitral pre-systolic and an early diastolic down the left sternal border had been queried. The diagnosis of atrial septal defect was confirmed by X-ray screening which revealed a hilar dance and gross cardiomegaly. Electrocardiogram showed a partial right bundle branch block.

Phonocardiograms were made on two occasions at an interval of six months. Records from the third left intercostal space near the sternal edge show a loud murmur, maximum in early and mid-systole, and a split second sound is clearly seen, although it was not heard. Records A and B were recorded on one occasion, and record C six months later, and all show the same essential features. No early or mid-diastolic murmur are demonstrated, but there is a series of low-frequency vibrations coinciding with auricular systole and most marked on the apical recording. (D). They occur between the R-wave and the Q-wave of the E.C.G. (P.R. = 0.20'). On one record (C) there is a definite rapid filling sound, although this was not audible.

The auricular vibrations might possibly represent a pre-systolic murmur and suggest the possibility of a Lutembacher syndrome, but it seems much more likely that they do in fact represent an inaudible auricular systolic sound, associated with auricular dilatation and hypertrophy and early congestive failure.

When last seen five years later this patient was relatively well, but on the verge of congestive failure. No final diagnosis or pathological followup is yet available.

According to Evans (1947) and Leatham (1949), the systolic murmur of atrial septal defect is maximal in early systole and normally finishes before the second sound which may often be split. The murmur in Case No. 89 fits this description, and the second sound is clearly split, although, surprisingly enough, this splitting was not audible. No early diastolic murmur from pulmonary incompetence was observed, but a rapid filling gallop sound is clearly seen.

SUMMARY AND CONCLUSIONS

1. The literature which describes the phonocardiographic recording of various auscultatory abnormalities which occur in congenital heart disease is briefly discussed.
2. Recordings taken from six cases are presented. In each, the diagnosis was clinical, and only in two was it positively confirmed.
3. Two young girls with patent ductus arteriosus are described and their recordings, made both before and after operation, are shown. In both cases the continuous Gibson murmur was demonstrated; it disappeared after ligation, but not section of the ductus, but within a matter of months it had returned. Over the followup period both children developed normally and had no symptoms. The murmur did not return to the same degree of intensity as it had been before operation in either case. In one of these patients a third heart sound was demonstrated after operation.
4. Recordings are shown from a young woman with mild congenital pulmonary stenosis. The systolic murmur was pan-systolic, but, unlike the murmur of pulmonary stenosis described by Evans, it commenced after the S line of the electrocardiogram. A splitting of the second sound and a third heart sound were also demonstrated.
5. One patient with a ventricular and one with an auricular septal defect are also described and their recordings shown. In neither case was the murmur truly pan-systolic and in both a splitting of the second sound and a rapid filling gallop were also demonstrated. In the latter case, definite auricular systolic vibrations were also visible and a possible Lutembacher syndrome was queried. Unfortunately, final followup was not available in either case.
6. In one case of congenital aortic stenosis the typical 'ejection' systolic murmur with a diamond-shape was recorded.

AORTIC VALVULAR DISEASE

For the sake of convenience the phonocardiography of aortic stenosis and incompetence is dealt with in a separate section from that of mitral valve lesions. In many cases where the aetiology is rheumatic, affections of both valves co-exist, but the examples given here are from cases with predominant aortic disease with little or no mitral lesions.

From the auscultatory findings alone it is usually impossible to decide whether an aortic lesion is rheumatic, syphilitic, arteriosclerotic, or congenital. Despite some claims, it is now generally agreed that these same limitations apply to the phonocardiographic tracings, although if recordings provide evidence of associated mitral stenosis, then the rheumatic aetiology will be confirmed.

The murmurs of aortic valvular disease have been recorded by Lewis (1913), Lian (1933), Orias and Braun-Menéndez (1939), and others. Bellet, Gourley, Nichols and McMillan (1939) described the loud musical diastolic murmur due to retroversion or eversion of the valve leaflets and they illustrate these descriptions with some rather poor-quality tracings. McKee (1938) and Arenberg (1941) both commented on the technical difficulties in recording the high-pitched basal diastolic murmurs with standard apparatus where sufficient attenuation of the lower frequencies cannot be obtained. Evans (1947) claimed that in the majority of cases of aortic disease due to rheumatic fever

he was able to record evidence of associated mitral murmurs even when the latter were not audible. He also claimed that inaudible aortic diastolic murmurs could be recorded in at least 25% of cases of hypertension. Neither of these claims was substantiated by Cowan and Parnum in 1949. These workers reported that they had gained no additional aetiological information to that obtained clinically after P.C.G. examination of 21 cases of aortic incompetence and also that they found no early diastolic murmurs in recordings from 20 cases of hypertension. Evans also claimed that in all cases of aortic stenosis an early diastolic murmur from associated incompetence could be demonstrated, but Levine and Harvey (1949) and others have failed to confirm this claim.

Wells, Rappaport and Sprague (1949) made the first complete study of the basal diastolic murmurs, and by using logarithmic frequency response phonocardiograms they were able to record faithfully the high-frequency low-amplitude murmurs without excessive deflection from the louder low-frequency vibrations of the heart sounds. They were able to record several early diastolic murmurs of aortic incompetence which were inaudible to the human ear, and they pointed out that the murmur may follow either part of a split second sound or there may be a slight gap between the end of the second sound and the beginning of the murmur. It is occasionally a purely decrescendo murmur after the second sound, but more commonly has a crescendo-decrescendo quality and is usually maximum at or just after the apex of the "v" wave of the jugular phlebogram.

Leatham, in the same year, made many similar observations regarding the early diastolic murmurs of aortic incompetence, and he also described the typical diamond shape maximum in mid-systole of the murmur of aortic stenosis. The same observations were made independently by Levine and Harvey. Leatham further pointed out in 1951 that these systolic murmurs usually started a little after the first sound and finished to leave a characteristic gap just before the second sound. The systolic murmur of aortic stenosis always retains this very characteristic shape and appearance.

In differentiating between the conducted apical diastolic murmur of aortic incompetence described by Austin Flint in 1862 and the mitral diastolic murmur of mitral stenosis, Luisada and Montez (1950) did not find the phonocardiograph of much value, but Wells (1954) points out that this characteristic decrescendo or crescendo-decrescendo shape of the aortic diastolic murmur should lead to its easy differentiation from a mitral mid-diastolic. He also points out that the associated feature of mitral stenosis such as the 'opening snap' will usually be present to make the phonocardiograph of definite value in differential diagnosis.

PRESENTATION OF CASES

Recordings were taken from seven patients with aortic valvular disease, and many of the findings of Wells et al. and Leatham were confirmed.

Of these seven cases, the first two have been mentioned earlier. Case No. 83, a 63-year-old woman, is included in the section dealing with systolic murmurs. Clinically, she was at first thought

to have mitral incompetence. She had paroxysmal nocturnal dyspnoea, cardiomegaly, and a loud apical systolic murmur. Phonocardiograms showed this murmur to have the typical diamond-shaped configuration of aortic stenosis, and the clinical diagnosis was amended accordingly. Sixteen months later she died, and necropsy confirmed the presence of gross calcific aortic stenosis. No early diastolic murmur was heard or demonstrated on the tracings.

Case No. 59, a man aged 50, who had gross aortic stenosis confirmed at autopsy, has been included in the gallop rhythm section as he had a loud gallop sound secondary to left ventricular failure. Apart from this, his heart sounds and recordings were not typical of aortic stenosis presumably because of the fact that he was in terminal congestive failure with consequent diminished cardiac blood flow.

Case No. 90, a male aged 43, and Case No. 91, also a male, aged 52, both had predominant aortic stenosis and a history of rheumatic fever.

The former probably had associated mitral stenosis, with a short mitral mid-diastolic murmur, not always audible, and a loud aortic systolic but no early diastolic murmur and no congestive failure.

The latter had congestive cardiac failure, auricular fibrillation and anginal chest pain. On clinical examination there was an apical systolic thrill maximum at the second and third intercostal spaces near the sternal edge. Over the same area there was a loud musical systolic murmur which was conducted up into the neck. An early diastolic murmur was also

heard at the aortic pulmonary and mitral areas, but was loudest down the left sternal edge. The first heart sound was accentuated.

Phonocardiograms are shown from both cases, and in each case the diamond-shaped systolic "ejection" murmur is demonstrated best over the aortic area, but also well seen at the apex. The murmur begins just after the first sound and finishes well before the second. There is also an associated early diastolic murmur commencing with the second sound in each case.

In Case No. 90, record A shows the low-frequency recording from the aortic and apical areas. Record B shows high-frequency recordings from the aortic area and medium-frequency from the fourth left para-sternal space where the early diastolic murmur is best seen. No mid-diastolic or pre-systolic vibrations can be seen. This man died five years later, and at autopsy the presence of both mitral and aortic stenosis was confirmed.

In Case No. 91 recordings were also made from the aortic area with the medium-frequency channel and fourth left para-sternal space, using a moderately high frequency (A and B). The systolic murmur with characteristic diamond-shape is again clearly seen, and also the characteristic crescendo-decrescendo shape of the early diastolic murmur as described by Wells et al. (1949). The systolic murmur is obvious on both tracings A and B, but in the latter the amplitude has been increased to bring out the typical features of the diastolic murmur which is maximum down the left sternal edge. This patient suffered repeated episodes of congestive failure and died 12 months later. No post-mortem examination was made.

Case No. 92 was a male patient, aged 53. He had a twelve-month history of increasing disability due to paroxysmal nocturnal dyspnoea and angina of effort, recently progressing to

angina decubitus. On admission he was fibrillating, the signs of congestive cardiac failure were present, and on auscultation there was evidence of aortic stenosis. The first heart sound was clear and followed^{by} a loud harsh systolic murmur and thrill. The murmur was well heard at the aortic area, but maximum at the apex. The second sound was faint and there was an audible third sound gallop. No diastolic murmurs were heard. Blood Pressure was $\frac{105}{85}$. E.C.G. confirmed the auricular fibrillation and also showed a left bundle branch block. X-ray screening showed marked left ventricular enlargement.

Despite adequate treatment, this patient failed to improve, his congestive failure became worse, and he died five weeks after admission.

Autopsy confirmed the presence of gross calcified aortic stenosis with a pin-hole aperture. There was also considerable left ventricular hypertrophy and widespread atherosclerosis, although the coronary arteries were relatively healthy. There was a terminal pulmonary embolism.

Phonocardiograms taken from the aortic area and apex demonstrate the systolic murmur and the third heart sound. The former is best seen in the high-frequency recordings in tracing A and the latter, in the lower frequency apical recordings seen in tracing B. The murmur starts just after the first sound and finishes before the second. A faint early diastolic murmur can also be seen occasionally in the apical tracing of record B, although this was not heard.

Case No. 93, also a male, aged 27, had subacute bacterial endocarditis, normal heart sounds, a soft apical systolic and an aortic early diastolic murmur beginning immediately after the second sound, but not really well shown on the tracing recorded from the apex.

A third heart sound is visible, which could be accounted for by the patient's age or as a sign of active carditis. It was not, in fact, heard. He made a successful recovery, but was left with a moderate degree of aortic incompetence. Five years later he was very well, fully compensated, but with some left ventricular hypertrophy and a persisting aortic incompetence.

Case No. 94 was a 35-year-old male. He had a history of four or five attacks of rheumatic fever, but no symptoms of cardiac pain or decompensation, and he had always been able to undertake a reasonable amount of exertion. He was admitted to hospital on account of a haematemesis from a gastric ulcer, but was found to have a double aortic lesion with a marked degree of incompetence. On examination there was no evidence of cardiac failure; capillary pulsation was visible. Corrigan-type radial pulse was palpable and Blood pressure was $\frac{200}{70}$. The apex beat was displaced and forcible. There was a loud aortic systolic murmur accompanied by a thrill, maximum at the aortic area and conducted up into the neck. There was also a loud early diastolic murmur which persisted right into mid-diastole and was maximum down the left sternal edge, but heard all over the praecordium. First and second heart sounds were normal and no pre-systolic murmur was heard, although the presence of a mid-diastolic murmur independent of the aortic early diastolic was queried, by some. Otherwise there were no clear-cut signs of mitral stenosis. X-ray of chest and E.C.G. confirmed the presence of left ventricular hypertrophy, and the latter also demonstrated the presence of latent heart block (P.R. = 0.24').

Six years later the signs were those of gross aortic incompetence with early congestive cardiac failure.

Phonocardiograms were taken on two occasions during the patient's first admission to hospital with a haematemesis and are of particular interest. They show, in addition to the early diastolic and systolic murmurs at the base, the presence of a pre-systolic murmur at the apex which might be due to associated mitral stenosis or might be an Austin Flint murmur associated with the aortic lesion. However, it so happens that bouts of nodal rhythm occur, and with this consequent delay in auricular systole the pre-systolic murmur disappears. The presence of associated mitral stenosis is thus confirmed by the phonocardiogram, and the pre-systolic murmur is clearly dependent on auricular systole. Record A shows the apical low-frequency tracing on the first occasion. B shows high-frequency recordings from the aortic area where the "to and fro" murmur of a double aortic lesion can be seen. The early diastolic murmur begins with the second sound and is decrescendo in shape. It persists into mid-diastole. Jugular phlebogram is partially obscured by the superimposed 'water-hammer' carotid arterial pulsations of free aortic incompetence. In records C and D taken three months later the development of an apical pre-systolic murmur can be seen. In the latter of these two tracings the rapid paper speed enables the vibrations to be very clearly seen. Recordings E and F, and G. and H, demonstrate the frequency band 1 and 2 recorded at the apex in the presence both of normal rhythm and of nodal rhythm, the pre-systolic vibrations being present in the former and absent in the latter. The systolic and early diastolic murmurs persist in all records, but no apical mid-diastolic murmur can be demonstrated. The first heart sound is

accentuated in nodal and decreased in normal rhythm, when the auricles are contracting before the ventricles.

It should be noted that the pre-systolic vibrations associated with auricular systole occur late in the P.R. period. During nodal rhythm all phonocardiographic vibrations before the R-wave of the electrocardiogram disappear.

SUMMARY AND CONCLUSIONS

1. Seven cases of predominant aortic stenosis or incompetence have been presented and the literature reviewed.
2. There are insufficient cases here to draw any definite conclusions, but the early diastolic and systolic murmurs of aortic valvular disease recorded conform with the criteria laid down by Wells and by Leatham.
3. The systolic murmurs were characteristically diamond-shaped and maximum in mid-systole.
4. Two of the cases in which pure aortic stenosis was diagnosed clinically were shown to have an early diastolic murmur visible on the tracing. This murmur had not been audible.
5. The majority of the early diastolic murmurs were decrescendo in shape; one was clearly crescendo-decrescendo. All began with the second sound and all finished well before the following first sound. When phlebogram tracings were available this murmur was seen to coincide with the "v" wave.
6. Four patients came to autopsy, and the diagnosis was confirmed.
7. Gallop rhythm was heard and recorded in two cases in which congestive failure was present. In a third case it was recorded at the apex but not heard.
8. Additional evidence was provided by the sound tracings in at least four of these cases, although it was never of great clinical importance. On one occasion the tracing proved the

existence of an associated mitral lesion, and twice an early diastolic murmur of aortic incompetence was demonstrated in cases with predominant aortic stenosis when no diastolic murmur could be heard. On one further occasion a patient thought clinically to have mitral incompetence was shown by the P.C.G. to have aortic stenosis, and this was confirmed at autopsy.

9. In the patient shown to have an associated mitral stenosis (Case No. 94) bouts of nodal rhythm alternating with normal sinus rhythm showed a pre-systolic murmur present only during sinus rhythm. The vibrations dependent on auricular systole all occur before the R-wave and the ventricular systolic vibrations all occur after the R-wave.
10. Splitting of the second sound was not found to be a common feature in the cases of aortic valvular disease reported here. A split second sound can be seen on the tracing of Case No. 90. In others it may possibly be obscured by the systolic and early diastolic murmurs.

MITRAL VALVULAR DISEASE

The auscultatory signs of stenosis and incompetence of the mitral valve are familiar to all. The presystolic murmur, accentuated first heart sound, variation in the pulmonary component of the second sound, the opening snap, and the rumbling apical mid-diastolic murmur are all signs of pure mitral stenosis which have been recognised for many years. It is only more recently, however, that the pan-systolic murmur, the rapid filling gallop, the absence of any opening snap, and the presence of a first heart sound of normal or even diminished intensity have been accepted as being characteristic auscultatory signs of mitral incompetence. This latter advance has been due largely to the stimulus resulting from recent advances in cardiac surgery. The successful development of operative procedures on the mitral valve itself has made exact pre-operative diagnosis imperative and has also created opportunities at the time of operation for precise confirmation of the relative degrees of stenosis and incompetence of the valve itself.

In previous sections the heart sounds, splitting of those sounds in health or disease, and the systolic murmur have been described and discussed in detail.

The way in which the classical studies of the great clinical observers of the 19th and early 20th centuries, and especially Laennec, Potain, Mackenzie and Lewis, paved the way for the phonocardiographic studies of the last two or three decades has also been outlined.

Much work has been carried out in the last 50 years in order to elucidate the precise nature and mechanism of the heart sounds and murmurs in mitral disease. In 1909 Cohn studied the pre-systolic murmur in a case of mitral stenosis with 2 : 1 heart block and showed with the aid of sphygmogram and phlebogram that it was dependent on auricular contraction. Lewis (1912) made similar studies with phonocardiographic tracings and was able to confirm Mackenzie's original clinical observation that the pre-systolic murmur disappeared with the onset of auricular fibrillation. Independent publications on the nature and origin of the pre-systolic murmur and also the mitral first sound came later from Sewall, Reid, Bramwell, Nylin and others.

The significance of the opening snap of mitral stenosis was first recognised by Bouillard in 1835, then by Duroziez (1862), and later by Rouches, who, in his M.D. thesis dated 1888, described the sound as "La claquement d'ouverture de la Mitral". Its significance and importance were further emphasised by Guttman (1872) and Sansom (1881), who both attributed its origin to movement of the mitral valve itself; also by Potain (1875), Gallavardin (1905), and several others. Boyd in 1896, writing in the Transactions of the Medico-Chirurgical Society of Edinburgh, gives a fascinating account of this particular physical sign of mitral stenosis. Thayer in 1908 coined the English term "opening snap" - a descriptive name which has never been superseded. The relations between the opening snap and the various forms of triple heart rhythm were described and illustrated phonocardiographically by Lian and his colleagues in 1933.

The most comprehensive account of the characteristics of the snap came, however, from Margolies and Wolferth in 1932, and little has really been added to their account of this physical sign since. They believed the opening snap to be due to sudden restriction of the opening movement of the mitral valve itself.

By taking Kymographic, phonocardiographic, electrocardiographic, and jugular pulse tracings from sixty cases they analysed the time relationship between the snap, the second heart sound, the mid-diastolic murmur, the 'v' wave of the phlebogram, and other events in the cardiac cycle. They differentiated the opening snap quite clearly from the third heart sound and splitting of the second. The snap was found to bear a most constant relationship to the first (aortic) part of the second sound, and the interval between the first part of the second sound and the beginning of the snap usually fell within the range of 0.06 to 0.11 second, the shortest they recorded being 0.03 second and the longest 0.19 second. They also showed that the duration of this time interval varied directly with the duration of the previous cardiac cycle, a finding which was later confirmed in more detail by Messer et al. (1951), who associated it with the pressure gradient across the mitral orifice.

Later studies by Mannheimer, Levine and Harvey, Leatham, Sellors et al., Mounsey, and others further elucidated and emphasised the value of the opening snap as a diagnostic aid. Mounsey (1953) recorded 33 patients with mitral stenosis and was able to hear or show on the phonocardiographic tracing an opening snap in every case but one. In his series the interval between the beginning of the second sound and the snap varied between 0.03 and 0.14

second, the average being 0.07. He also described the various features which differentiate the opening snap from splitting of the second sound and the third heart sound.

The diastolic murmurs of mitral stenosis have recently been studied in detail by Evans (1947 and 1951), Leatham (1949), Cowen and Parnum (1949), Wells (1952) and Wynne (1952). Luisada et al. (1950 and 1955) made a special study of apical diastolic murmurs which might simulate mitral stenosis, and concluded that they could be differentiated on the phonocardiogram. They believed that these murmurs were often produced by a "relative" mitral stenosis in such conditions as acute rheumatic fever, hypertensive and coronary heart failure, and severe anaemia. Similar studies on apical diastolic murmurs simulating mitral stenosis were made by Bramwell in 1943 and by Alimurung et al. in 1949.

By measurement of the time intervals between various events in the cardiac cycle Wells (1954) claimed that an assessment could be made of the severity of mitral stenosis. He made a phonocardiographic and electrocardiographic study of thirty patients all of whom were subjected to valvotomy. Basing his theories on those of Messer et al. he claimed that the size and nature of the mitral orifice could be estimated by a calculation based upon the measurement of cardiac cycle length, the distance between the Q-wave of E.C.G. and the first sound, and the distance between the second heart sound and the opening snap.

The systolic murmurs of mitral stenosis and incompetence were also studied in great detail by Brigden and Leatham in 1953 and Mounsey and Brigden in 1954. After detailed clinical, radiological and phonocardiographic study of thirty patients with pure organic mitral incompetence, Brigden and Leatham came to the following conclusion regarding the auscultatory signs. The loud apical murmur is usually pan-systolic and maximum in late systole (unlike the murmur of aortic stenosis, which is maximum in mid-systole), and splitting of the second sound at the pulmonary area may be wider than usual. In contrast to mitral stenosis, the accentuated mitral first sound and the opening snap are usually absent and the third heart sound is frequently present in mitral incompetence. The frequent occurrence of pan-systolic murmurs in mitral incompetence confirmed at operation was also observed by Mounsey and Brigden, but these authors also noted that an early systolic apical murmur may sometimes be associated with a minor degree of mitral incompetence, and they did not observe accentuation of the murmur in late systole.

Some of the changes in the heart sounds of children with early mitral disease and acute rheumatic fever were studied with the aid of phonocardiograms by McKee (1938), Taquini *et al.* (1940) and, later, by Besterman (1955) who emphasised the significance of an accentuated third heart sound appearing in children who have developed mitral incompetence associated with acute rheumatic carditis.

More recently the whole subject of clinical signs and the diagnosis of mitral stenosis and incompetence has been reviewed by Bramwell, and as a result of combined medical and surgical studies detailed analyses have been made by Logan and Turner (1953) and by Wood (1954). Logan and Turner describe detailed clinical and operative findings and the subsequent followup of 100 patients with predominant mitral stenosis subjected to valvotomy and sixteen cases with predominant incompetence treated with a different type of operation. Wood analysed results and observations on 150 patients with mitral valve disease submitted to operation and a further 150 who were considered unsuitable for operation. The effect of valvotomy on auscultatory signs was also carefully studied. In both these series great emphasis is placed on the importance of a careful analysis of the physical signs, and in particular the auscultatory findings, in assessing the relative degree of stenosis and incompetence of the mitral valve. There is general agreement on the value of a pre-systolic mitral murmur, a loud mitral first sound, absence of systolic murmur, and presence of mitral mid-diastolic murmur and opening snap being strong evidence for the presence of predominant mitral stenosis and a lesion which is likely to be amenable to surgical splitting of the valve. The signs of mitral incompetence are also fairly closely agreed upon, but agreement is not so close on the value of the accentuated second sound at the pulmonary area as a sign of pulmonary hypertension. Wood considers that the accentuated pulmonary component of the second sound is an important, but not infallible, guide

to the presence of pulmonary hypertension. Turner concluded that although a loud pulmonary second sound was usually associated with severe pulmonary hypertension, there was sometimes considerable discrepancy and indeed variation of intensity of the sound occurring in the same patient. (A Graham-Steele murmur was noted only in a few cases with severe pulmonary hypertension).

PRESENTATION OF CASES

During the course of the studies reported here, phonocardiograms were made on a total of 66 separate occasions from 46 different patients, all of whom were thought to have mitral stenosis. Of these cases, twelve were thought also to have associated mitral incompetence of greater or lesser degree, and seventeen had, in addition, an aortic valvular lesion. In all of these seventeen cases, however, the aortic lesion was relatively unimportant and of little or no haemodynamic significance.

Of the 46 patients, sixteen thought to have mitral stenosis were submitted to the operation of mitral valvotomy, and phonocardiograms were made both before and after the operation in 10. Two other patients with predominant mitral incompetence were operated on in an attempt to relieve the incompetence, by passing a sling made from a strip of pericardium across the incompetent valve cusps. One of these patients had phonocardiograms made both before and after operation. All the surgically treated patients have been reported in the literature by Logan and Turner (1952 and 1953).

Phonocardiograms were made in some cases as a matter of routine because the signs were typical of mitral stenosis or incompetence, and others were recorded because their auscultatory findings were either atypical or difficult to interpret and it was felt that graphic sound tracings might help to identify the precise sounds and murmurs and establish the exact diagnosis. Followup and post-mortem findings when available have been included in the case histories.

This is, however, a study of the heart sounds and murmurs in mitral stenosis and not an enquiry into the value of mitral valvotomy or the natural history of mitral valve disease, so extensive followup information has not in fact been sought or included here. Brief clinical histories and the relevant details are set out in the following pages, and a number of representative phonocardiographic tracings are presented.

The first eighteen cases, comprising the operative series, are considered first. Eleven were recorded both before and after operation, three only in the post-operative and four in the pre-operative period.

The first two cases had mitral stenosis associated with a significant degree of incompetence; the remaining sixteen had pure or very largely predominant mitral stenosis, and three of them were young pregnant women. Altogether there were four males and 14 females in the series.

Case No. 95, a 32-year-old housewife, had moderately severe mitral stenosis and incompetence with a history of exertional dyspnoea and haemoptysis. She was in normal rhythm and had no congestive cardiac failure and no aortic valve lesion. On auscultation at the apex the first sound was accentuated, there was a loud systolic murmur, a split second sound, and a mid-diastolic murmur. No opening snap could be heard.

At operation the presence of gross mitral incompetence was discovered, and a pericardial strap was threaded across the valve orifice (Logan and Turner, 1952). After operation the systolic murmur was thought to be diminished in intensity; the mid-diastolic murmur persisted, as did the split second sound which varied with respiration. This patient considered herself to be improved, and ten months later was alive, and no deterioration of her condition had occurred.

Phonocardiograms taken three weeks after operation show medium and low frequency recordings from the pulmonary and apical areas. The records mounted show the split pulmonary second sound very clearly, also the apical mid-diastolic murmur, but not the systolic.

The split second sound is distinguished from an opening snap because of its characteristic appearance and because the interval between the commencement of the first and second components of the split is only 0.04 to 0.06 second (See Table 20). T-wave abnormalities seen in the electrocardiogram are presumably due to the operative handling of the heart and pericardium.

Case No. 96, a 37-year-old housewife, had mitral stenosis and incompetence, aortic incompetence, and auricular fibrillation. She gave a history of exertional and paroxysmal dyspnoea and a cerebral embolism with transient hemiplegia, from which she had made a good recovery. On auscultation at the apex there was a loud first sound, a systolic murmur, an accentuated pulmonary second sound, and an opening snap. There were also mid-diastolic and early diastolic murmurs.

At operation a fairly severe degree of both incompetence and stenosis of the mitral valve was encountered; valvotomy was performed, and in addition, a pericardial sling was also placed across the valve. Ten months later she was well and considered herself to be definitely improved.

Phonocardiograms were made both before and after operation. Records A and B show recordings from the fourth intercostal space in the left parasternal line and from the apex. They demonstrate a ^{split} second sound, an opening snap, and also the early diastolic, mid-diastolic and systolic murmurs. Both the splitting of the second sound and the opening snap vary with the varying cycle length associated with auricular fibrillation. Records C and D show similar tracings after operation. Clinically the signs were largely unchanged, although the early diastolic murmur was no longer heard. As there is no accurate calibration, the recordings are not strictly comparable, but splitting of the second sound, opening snap, systolic and diastolic murmurs are still seen. These records are very important because they demonstrate the presence of both splitting of the second sound and the opening snap of mitral stenosis on the same tracing, proving that they are in fact

quite distinct entities. It also shows that an opening snap may still be present despite a significant degree of mitral incompetence. The apical systolic murmur is maximum in early systole and seems to finish just before the second sound.

The interval between the first and second components of the split second sound is approximately 0.04 second, and between the beginning of the second sound and the opening snap, approximately 0.10 second.

Case No. 97, a 30-year-old housewife, had predominant mitral stenosis and also moderate aortic incompetence, with an 11-year history of exertional dyspnoea, some ankle swelling, haemoptysis and palpitations.

Successful valvotomy was performed and the mitral stenosis was both confirmed and relieved. She had little or no disability from the aortic lesion and was still much improved eighteen months later.

Phonocardiograms were made before the operation only and show the early and mid-diastolic, but not the pre-systolic, murmurs. The opening snap is also seen despite the fact that it is partially masked by the early diastolic murmur which seems to start with the second sound and carries right on to merge with the mid-diastolic murmur. There is also an early systolic murmur. The interval between the beginning of the second sound and the snap is approximately 0.06 second.

Case No. 98, a girl aged 16, had a two-year history of dyspnoea on exertion, palpitation, and attacks of acute pulmonary oedema. All the classical signs of pure mitral stenosis were present, and no evidence of an aortic lesion.

Valvotomy was performed successfully, and 25 months later, considerable improvement had been maintained.

Phonocardiograms (A and B) were made before the operation. These show the loud apical first and pulmonary second sounds, the pre-systolic and mid-diastolic murmurs, and the opening snap. Fine splitting of the accentuated second sound at the pulmonary area is seen in the higher frequency recordings (B); this was not audible. There is also a coarse vibration which coincides with the P-wave of the electrocardiogram and which may represent auricular systole. It occurs 0.25 second after the beginning of the second sound and cannot, therefore, be a rapid filling sound. It was not audible. The opening snap occurs 0.10 second after the beginning of the second sound.

Case No. 99 was a 28-year-old pregnant woman with a two-year history of slight limitation of exertion by dyspnoea. She had, on auscultation, pre-systolic, early diastolic, and mid-diastolic murmurs, and there was some debate as to whether the second sound was split or whether an opening snap was present. On auscultation at the apex and left sternal edge with the patient erect, the impression was of a split sound, but with the patient lying supine it was more like a single second sound followed by an opening snap.

Phonocardiogram recorded before operation revealed the typical configuration of split second sound only and no snap. The distance between the first and second components of the second sound was approximately 0.06. At operation, pure mitral stenosis was found and a good operative result obtained.

The next two cases had recordings made after valvotomy only.

Case No. 100 was a 27-year-old woman with severe mitral stenosis and slight aortic incompetence, a 13-year history of gradually increasing exertional dyspnoea, and recent haemoptysis. Typical physical signs were present on auscultation, including the opening snap. At operation a tight stenosis was encountered and a good splitting of the valve obtained. Three years later considerable improvement was maintained.

Phonocardiograms were made after operation, when the apical pre-systolic, systolic and early diastolic murmurs, also opening snap and accentuated first and pulmonary second sounds were audible.

At this time no mid-diastolic murmur could be heard. The tracings show the sounds and murmurs as they were heard, confirm the presence of an opening snap following successful valvotomy, and also show a mid-diastolic murmur. These records are not mounted. The opening snap occurs 0.08 to 0.10 second after the second sound.

Case No. 101 was a 27-year-old pregnant woman with mild thyrotoxicosis. During the first four months of pregnancy she had signs of early congestive failure and paroxysmal dyspnoea. The typical signs of pure mitral stenosis were present. At valvotomy a tight mitral stenosis was encountered, and this was successfully relieved with considerable subsequent improvement. She later gave birth to a healthy baby and seventeen months later was well and symptom-free.

Phonocardiograms were made shortly after the operation. At this time she had a loud apical pre-systolic murmur, no systolic murmur, but a mid-diastolic murmur and an opening snap, and also a probable third heart sound. Apart from the murmurs, the tracings show the accentuated first sound, split second sound at the pulmonary area, also an opening snap, and a third heart sound, despite considerable tachycardia. This record is particularly interesting as it shows on the same tracing a split second sound, an opening snap, and a third sound, all persisting after successful valvotomy. The third sound forms the initial vibration of the mid-diastolic murmur and is presumably associated with the patient's age and pregnancy, or is possibly due to the recent operation. Tachycardia and a time marker of 0.2' makes accurate timing difficult in these records.

The next ten cases all had both pre- and post-operative recordings made.

Case No. 102, a 37-year-old male, had a three-year history of mild congestive cardiac failure, and on examination he was found to have pure mitral stenosis and auricular fibrillation, with typical physical signs. Experienced observers were satisfied that an opening snap could be heard, but others were in some doubt over this point. A successful valvotomy was performed; at operation a slight degree of incompetence was encountered, but the patient was much improved thereafter and was well two years later. After operation the auscultatory signs were unchanged.

Phonocardiograms taken before (A and B) and after (C and D) operation are shown. These confirm the presence of an opening snap on both occasions, more obvious in the latter recordings, and best heard and recorded from the left sternal edge (Records B and C). It should be noted that in records A and B the time marker registered 0.1 second, but in C and D it registered 0.2 second. The opening snap occurs from 0.08 second to 0.10 second after the beginning of the second sound.

Case No. 103 was a 20-year-old male, with a history of recurrent haemoptysis and dyspnoea on exertion. On examination he was found to have all the classical signs of mitral stenosis, with a clear opening snap and also an apical systolic murmur, but no evidence of aortic lesion. Successful valvotomy was performed, and at operation a very slight degree of mitral incompetence was found. After operation the opening snap was no longer audible, but within three months it had returned. Two years later this patient's improvement was maintained.

Pre-operative records from the apex were made with a string galvanometer designed for use with the standard portable Cambridge electrocardiogram and show the pre-systolic and mid-diastolic murmurs and the opening snap, but no systolic murmur. Post-operative records made with the Elmquist multi-channel instrument are not strictly comparable. They do not show the murmurs so clearly and they do not demonstrate an opening snap, but a third heart or rapid filling sound can be seen - possibly associated with the operation. In the earlier records the snap occurs 0.08 to 0.10 second after the beginning of the second sound and the comparable measurement for the third sound in the later record is 0.18 second.

Case No. 104 was a 30-year-old woman with a three-month history of exertional dyspnoea and attacks of paroxysmal dyspnoea. She had typical signs of mitral stenosis, confirmed at valvotomy. Considerable improvement followed valvotomy, and this was still maintained when she was seen fifteen months later. Pre-operatively an opening snap was both heard and recorded, the snap occurring between 0.08 and 0.10 second after the beginning of the second sound. Post-operatively, however, a split second sound was heard and recorded (occurring 0.02 to 0.04 second after the beginning of the second sound), but the opening snap was no longer present. A clearcut rapid filling sound was also recorded post-operatively, occurring 0.16 second after the second sound. These records are not mounted.

Cases Nos. 105 and 106 were both housewives, aged 35 and 47 years respectively. They had mitral stenosis, a mild degree of aortic incompetence, and auricular fibrillation. Typical auscultatory findings were present in both, and recordings were made before and after successful valvotomy. These patients were alive and their improvement was maintained when last seen two years later.

Phonocardiograms showed the murmurs and also the opening snap which varied in its position relative to the second sound as the cycle length varied. There was no significant difference between the pre- and post-operative records, and they have not been mounted.

Case No. 107, a housewife aged 36, had mitral stenosis with possible aortic incompetence diagnosed clinically. She had a three-year history of mild exertional dyspnoea. All the auscultatory signs of pure mitral stenosis were present, including the opening snap, and a basal early diastolic murmur was queried. Valvotomy was successful, and at operation the presence of pure mitral stenosis was confirmed. Nineteen months later, improvement was maintained.

Three sets of phonocardiographic recordings were made. The first record (A), taken with the Boulitte instrument, from the apex, shows the mitral diastolic murmur and the opening snap. Record B, recorded two years later with the Elmquist, confirms these various auscultatory signs, the opening snap and diastolic murmurs being particularly well seen. There is also a visible auricular sound. Finally, record C was made after valvotomy and, although not easily comparable, it does not reveal any definite changes. There is probably an early diastolic murmur recorded from the base, but not the apex. Before valvotomy it appears to start after the opening snap, but after valvotomy it can be seen to follow immediately upon the second sound. The opening snap occurs from 0.08 to 0.1 second after the beginning of the second sound.

Case No. 108, a 40-year-old male, had a six-year history of exertional dyspnoea, with recent haemoptysis and paroxysmal nocturnal dyspnoea. Heart rhythm was normal. On auscultation there was an apical pre-systolic murmur, a loud first sound, no systolic murmur, a split pulmonary second sound, a mid-diastolic murmur, and a loud opening snap. At valvotomy a good result was obtained, but a very slight degree of mitral incompetence was found.

Two years later, considerable improvement was being maintained. Clinically the auscultatory findings were almost identical before and after the operation, but the phonocardiograms do not show quite identical patterns. There is, however, no marked change, and in view of the lack of calibration, no strict comparison between the two sets of records can be made. The mid-diastolic and pre-systolic murmurs are present both before and after the operation; before operation there is a split second sound and an opening snap seen at the apex (Records A and B). In the post-operative apical tracings the snap is not so well demonstrated in the Elmquist tracings (C and D). However, the snap is more clearly seen in a tracing made with the portable Cambridge instrument (Record E), and the split second sound can just be seen on tracing D recorded from the third left space at the sternal edge. The second half of the split second sound begins 0.04 to 0.06 second after the first part, and the opening snap occurs approximately 0.10 second after the beginning of the second sound in the pre-operative and 0.08 second in the post-operative tracing.

Case No. 109, a 38-year-old housewife, had a three-year history of exertional dyspnoea. The classical auscultatory signs of pure mitral stenosis were present. The lesion was confirmed at operation and a successful valvotomy performed. Sixteen months later the patient's improvement was maintained. After operation the signs were relatively unchanged, but the opening snap had a slightly different character and sounded rather more metallic in quality.

The phonocardiograph shows only two minor differences: firstly, the snap seems to be slightly closer to the second sound in the post-operative records and, secondly, there is a small vibration very like a third heart sound or rapid filling gallop in the low-frequency post-operative recording from the apex. This was not heard. The records are not of a good quality and the murmurs are not well shown. The opening snap occurs at an interval of from 0.08 to 0.10 second after the beginning of the second sound and the gallop sound 0.14 to 0.16 second.

Case No. 110, a 29-year-old woman, had interesting records. She had considerable disability from her mitral lesion and a history of subacute bacterial endocarditis. There was also clinical evidence of aortic incompetence. On auscultation, apical, pre-systolic, systolic and mid-diastolic murmurs were heard. The first sound at the apex and second sound at the base were accentuated. There was no opening snap and at the base systolic and early diastolic murmurs were heard. At operation a moderate degree of mitral incompetence was found, together with a calcified valve. Only a moderately successful splitting of the mitral valve was obtained, and following operation a bout of auricular fibrillation occurred. The operative result was poor, and two years later the patient was still fairly badly disabled. The pre-systolic murmur disappeared during the bout of auricular fibrillation, otherwise there was little change following the valvotomy. The opening snap was never heard.

Phonocardiograms were taken on four occasions over the course of two years. The first record (A), made with the Boulitte instrument two years before operation, shows the mid-diastolic and pre-systolic murmurs at the apex. Record B was made eighteen months later, during a bout of fibrillation; the systolic murmur is clearly ^{seen} maximal at the pulmonary area, but there is no apical pre-systolic murmur. Three days later, normal rhythm was re-established, and record C shows the return of an apical pre-systolic murmur recorded at both base and apex. It should be noted that the lowest sound tracing (B) is from the pulmonary area, the upper sound tracing from the apex, whereas in record C the lowest is from the apex and the upper from the pulmonary area. Finally, records D and E were made three months later, after valvotomy, and shows no change from C. The pre-systolic apical murmur and pulmonary systolic are still recorded. No early diastolic murmur can be seen in any of the tracings and the systolic murmur, although visible on the apical tracing, still appears most marked at the pulmonary area and is maximal in early systole.

Presumably as a result of the calcified valve and the significant degree of mitral incompetence, the opening snap was not loud enough to be heard. It is just seen on one tracing (Record B), but none of the others. Its absence on auscultation was taken to be a sign of mitral incompetence and, therefore, its phonocardiographic demonstration was not of any diagnostic assistance. When seen, it occurs approximately 0.08 to 0.10 second after the beginning of the second sound and has the graphic characteristic of a snap rather than of a split second or gallop sound.

Case No. 111, a pregnant housewife aged 26, had a five-year history of exertional dyspnoea and recent attacks of paroxysmal dyspnoea. Clinically she had pure mitral stenosis and a probable aortic stenosis. On auscultation the signs of mitral and aortic stenosis were present; there was a split second sound at the pulmonary area, but no opening snap and no early diastolic murmur. Some observers thought that a rapid filling or third sound could be heard. At operation the presence of mitral stenosis was confirmed. A satisfactory result was obtained and she was still improved seventeen months later. After operation the auscultatory signs changed considerably in this patient. The pre-systolic murmur disappeared, the systolic murmur became louder at base and apex, and the splitting of second sound was not so obvious. Mid-diastolic murmur and possible rapid filling gallop were as before.

Phonocardiographs before operation (Records A and B) showed a loud basal diamond-shaped systolic murmur, typical of the ejection murmur of aortic stenosis, and a split second sound at the pulmonary area. Mid-diastolic, but no real pre-systolic murmur, vibrations were seen, although the mitral first sound was clearly accentuated. No definite gallop sound was seen, but the coarse initial vibrations of the mid-diastolic murmur might conceivably represent such a sound. After operation the recording still showed the aortic systolic murmur and split second sound at the base. The pre-systolic murmur is still not visible, and the mid-diastolic (not well shown) still has a few coarse vibrations at its commencement. No early diastolic murmur was recorded at any time. It seems

likely that paradoxical splitting of the second sound has occurred, although this cannot be proved without further records. The first component of the split second sound is very much louder than the second component at the pulmonary area. The pulmonary component might be expected to be loudest here; also, the presence of pulmonary hypertension and aortic stenosis would lead to the same supposition.

Case No. 50, a 45-year-old male, has been discussed in both the sections on gallop rhythm and on bundle-branch block. He gave a ten-month history of exertional dyspnoea and recent paroxysmal nocturnal dyspnoea. On auscultation there was a loud pre-systolic gallop rhythm to be heard at the apex which was at first thought to be a pre-systolic murmur. However, it gave the typical three-time cadence of gallop rhythm and was heard and also palpable well up in the epigastrium. An apical mid-diastolic murmur and systolic murmurs were audible. The Wolff-Parkinson-White syndrome was also diagnosed, but because of increasing attacks of paroxysmal dyspnoea he was subjected to valvotomy, when a moderate degree of mitral incompetence was discovered and also some calcification of the valve. However, some splitting was obtained, with considerable subsequent clinical improvement. Three years later this was being maintained, although the conduction defect and gallop rhythm were still present.

A phonocardiogram was made before operation only. This shows both a pre-systolic murmur and a gallop sound occurring before the R-wave. A typical auricular gallop sound with coarse vibrations is seen in the low-frequency tracing (A), but with the higher frequency tracing the typical features of a pre-systolic murmur are revealed.

The mid-diastolic murmur is not well seen and there is no opening snap. The section on gallop rhythm should be consulted for further details and discussions.

The remainder of the patients to be discussed were not subjected to mitral valvotomy; all had mitral stenosis, and some of them also had associated mitral incompetence or aortic incompetence. There were eight males and twenty females.

Case No. 112 was a 29-year-old ^{pregnant} housewife. The typical signs of mitral stenosis were present, but at the pulmonary area the second sound appeared to be split when the patient was auscultated in the supine position, but when the patient was erect the sound had more of the auditory characteristics of a single second sound with a following opening snap. A rapid filling or third sound was clearly heard at the apex.

Phonocardiograms were made on two occasions. The apical tracing recorded with a relatively low-frequency band shows the pre-systolic and mid-diastolic murmurs, but in particular it brings out the extra diastolic sound (Record A). Recordings from the pulmonary area confirm the auscultatory findings. In the supine position an apparent split second sound is seen, best in the high-frequency recording. With the patient erect there is a wider gap between the two sounds and the appearance is much more like that of an opening snap. In both positions, records were taken with three different frequency bands (Records B, C and D, recorded in the supine position; E, F. and G in the erect position). An auricular systolic sound can also be seen on the lower-frequency tracings.

The time interval between the beginning of the second and the apparent rapid filling or third sounds is 0.14 to 0.16 second. This may in reality, however, be only a coarse component of the mid-diastolic murmur. The interval between the second sound and the opening snap or second component of a split sound is very slightly less in the supine than in the erect position, but seems to be between 0.06 and 0.08 second and is probably in fact an opening snap, its character and appearance changing slightly with the altered blood flow consequent upon a change of posture.

Case No. 113 was a 52-year-old male, with established mitral stenosis, who sustained a myocardial infarction and was admitted to hospital in congestive cardiac failure. The typical murmurs and sounds of mitral stenosis, including the opening snap, were heard on auscultation, and also a rapid filling gallop which disappeared as the failure was relieved.

Phonocardiographic recordings with different frequency bands (Records A, B and C) show pre-systolic, early systolic, and mid-diastolic murmurs and also an accentuated first sound, opening snap, and a rapid filling gallop sound. In all records the initial vibrations of the mid-diastolic murmur seem to form a clear sound, particularly in the low-frequency recordings, and this is interpreted as being the rapid filling sound.

The relationship between these various events in the cardiac cycle and the phlebogram is perfectly clear on the tracings, but their interpretation is not quite so straightforward. The phlebogram is of the type described by Paul White as "congestive". It occurs with tachycardia and congestive cardiac failure. Because of this there is no clearcut separate "v" wave or indication of the moment of opening of the A.V. valves. The opening snap and gallop sound cannot, therefore, be correlated with the rise and fall of the jugular "v" wave, and the jugular pressure remains

high throughout diastole. The opening snap occurs 0.08 second after the beginning of the second sound, and the gallop sound 0.16' after. This gallop sound appears to "fire off" the mid-diastolic murmur, and its auditory characteristics, its time relation to the second sound, and its position in the cardiac cycle all tend to confirm its identity. Unfortunately there is no falling limb of the "v" wave (the "y" wave") with which to establish its precise timing. Its presence is explained by the existence of a fairly severe degree of congestive failure associated with myocardial infarction and a mild-to-moderate mitral stenosis and possibly incompetence. This patient made an excellent recovery from the infarction but, three years later, congestive cardiac failure recurred coincident with the onset of auricular fibrillation, and a series of pulmonary emboli. However, he again made a good recovery and, one year later was alive and well.

Case No. 114. This 64-year-old woman had rheumatoid arthritis, mitral stenosis, and a mild degree of aortic incompetence. On auscultation at the apex a loud pre-systolic murmur and first sound were heard, a soft late systolic and a normal second sound. An opening snap was clearly heard, also a mid-diastolic murmur, and, on some occasions only, an early diastolic murmur at the base. At the pulmonary area the second sound was clearly split.

C

Phonocardiographic recordings (A, B, and D) show the pre-systolic murmur, the loud first sound, the late systolic murmur, the opening snap, and a probable early diastolic murmur. All the events cannot be clearly demonstrated on one single recording and there is no adequate record of the opening snap with simultaneous phlebogram tracing. One small section of the record taken over the apex (B) does, however, show the second part of a split second

sound situated on the ascending limb of the "v" wave of the phlebogram and the snap near the peak of the "v" wave. As the opening snap occurs at the peak of the "v" wave in the phlebogram, it seems reasonable to suppose (in the absence of marked asynchrony of ventricular contraction and splitting of the sounds) that it is related to the opening mitral valve cusps, the mitral and tricuspid valves normally opening at about the same time. Unfortunately, no adequate followup of this patient is available.

Case No. 115, a male aged 50 years, had mitral stenosis with probable incompetence, auricular fibrillation and congestive cardiac failure. On auscultation at the apex, a loud first sound and a moderately loud pan-systolic murmur were heard. There was also a faint, atypical opening snap,^{and}/_a mid-diastolic murmur, and an early diastolic murmur was heard at both the base and the apex. This man failed to respond to treatment and died four months later. At autopsy, gross mitral valve thickening and distortion with both stenosis and incompetence was demonstrated. The aortic and other valves were normal, but there was a considerable degree of left ventricular hypertrophy and signs of pulmonary and systemic chronic venous congestion.

Phonocardiograms were taken from the fourth space, two inches from the left sternal edge - this being the optimum position for hearing all the signs. Simultaneous jugular phlebogram and electrocardiographic tracings were also taken. The sound tracings reveal a loud first sound, no pre-systolic murmur, a systolic murmur, a split second sound, and a definite third heart sound. Mid-diastolic and early diastolic murmurs are not well seen on the tracing, but are probably present.

The systolic murmur is in fact pan-systolic, but maximum in early systole, and is not like the systolic murmur of aortic stenosis. A split second sound is seen, with the second element of the split occurring on the ascending limb of the "v" wave and about 0.04 to 0.05 second after the first part of the second sound. A rapid filling gallop sound is also seen occurring on the descending limb of the "v" wave and 0.14 second after the beginning of the second sound. There is no opening snap.

In this case the phonocardiogram showed the faint atypical opening snap heard on auscultation to be in fact a split second sound. This would suggest that a diagnosis of predominant mitral stenosis was unlikely. The rapid filling gallop sound also is in favour of mitral incompetence and, of course, was partly associated with the presence of congestive failure. The early diastolic murmur must have been due either to cardiac dilatation and functional aortic or pulmonary incompetence as it may possibly have been a Graham-Steele murmur. Analysis of this phonocardiogram gave more accurate information than was available from simple clinical auscultation.

Case No. 116 was a 44-year-old housewife with mitral stenosis, auricular fibrillation and congestive failure. An opening snap and mid-diastolic murmur were well heard, but no early diastolic murmur. Phonocardiogram shows the accentuated first sound, systolic and mid-diastolic murmurs, the opening snap, and a probable decrescendo early diastolic murmur, seen at both apex and base, which is continuous with the mid-diastolic murmur. It seems likely, therefore, that this patient had associated aortic incompetence, but unfortunately no followup was available as she had a stroke and died at home not long after the records were made.

The next two cases both had severe mitral stenosis and incompetence, and both came ultimately to autopsy one year after recordings were made and the presence of gross mitral valve thickening with a significant degree of incompetence was confirmed. In the first case there was considerable calcification of the valve and in the second, an active subacute bacterial endocarditis.

Case No. 117 was a 54-year-old housewife with auricular fibrillation and congestive failure. Apical systolic and mid-diastolic murmurs were heard and an extra sound in diastole, thought by most observers to be a rapid filling gallop, but by some to be an opening snap.

Phonocardiograms showed this sound to be quite distinct and occurring from 0.10 to 0.12 second after the beginning of the second sound. This might, from appearance, have been either a gallop or opening snap, and no phlebogram tracing was available for timing events in diastole. In view of its relationship to the second sound (0.10 to 0.12 second^{is} over the upper limit of the usual timing of opening snap with second sound), the presence of mitral incompetence and congestive failure, and its auditory characteristics, a diagnosis of rapid filling gallop seemed most likely. Certainly no 'snapping' movement of the valve cusps could have been produced in this case.

The second case - No. 118 - a 44-year-old male, had little disability. Systolic and mid-diastolic apical murmurs were heard, but no pre-systolic, although rhythm was normal sinus in type. A split second sound was audible over the pulmonary area and no opening snap or gallop.

Phonocardiograms confirmed these findings, and the lack of a pre-systolic murmur may possibly be associated with the very small P-waves of the electrocardiogram (Record A). The absence of an opening snap in this case provided confirmation of the gross degree of mitral incompetence as opposed to stenosis, and the presence of a split second sound is clearly seen (Record B).

The next three patients all had auricular fibrillation and congestive cardiac failure.

Case No. 119 was a 63-year-old woman with mitral stenosis and incompetence in whom auscultation also revealed an aortic early diastolic murmur, a definite opening snap, and a rapid filling gallop. She died at home shortly after, and no post-mortem was obtained.

Case No. 120, a 40-year-old male, had long-standing pure mitral stenosis and suffered a myocardial infarction in addition. On auscultation the typical signs, together with an opening snap, were heard at the apex. There was no gallop. This man made a good recovery and was alive and well two years later.

Case No. 121 was a 55-year-old woman with isolated mitral stenosis and cardiac failure. The typical physical signs and opening snap were clearly heard.

Phonocardiograms were made from these three cases on several occasions, and representative tracings are shown from Cases 119 and 120. The opening snap was recorded in each case, and as with all cases of auricular fibrillation, the time interval between the beginning of the second sound and the snap varies with the length of the

preceding cardiac cycle (from 0.06 to 0.10 second in Case 119 and from 0.08 to 0.10 second in both Cases 120 and 121). The rapid filling gallop sound is also seen in Record A from No. 119 and occurs from 0.14 to 0.16 second after the second sound. In none of these cases was it found possible to demonstrate the murmurs really well, although systolic, mid-diastolic and early diastolic murmurs can be made out on Record B from Case No. 119.

Case No. 122, a young girl aged 17 years, had severe rheumatic heart disease with a history of recurrent attacks of rheumatic fever. She had early congestive cardiac failure, but was in normal rhythm. On auscultation at the apex, pre-systolic, systolic and mid-diastolic murmurs were heard; there was an accentuated first sound and either a split second or an audible opening snap. At the base there was a loud pulmonary systolic, probably an early diastolic, murmur, and the second sound was clearly split. Partly due to tachycardia, these sounds were a little difficult to analyse in detail, but it was clear that the patient had severe heart disease with mitral stenosis and probable incompetence. A gallop rhythm, presumably due to early cardiac failure or possibly due to age alone, was also audible. Whether she had an opening snap due to predominant mitral stenosis or a splitting of the second sound was not certain clinically. She was thought also to have an aortic lesion. Two years later this patient died with acute heart failure, and at autopsy the presence of gross mitral stenosis and incompetence was confirmed. There was also a slight degree of tricuspid valve thickening, but the aortic valves were normal. Considerable cardiac enlargement and the signs of chronic venous congestion in various viscera were present.

Phonocardiograms were made on two occasions, but they are not easy to interpret because of the slow time marker which registers 0.20 second. However, on careful examination, the pansystolic and mid-diastolic murmurs can be seen, but there is too much tachycardia to distinguish a separate early diastolic murmur. A rapid filling gallop is seen best on the apical tracing, and on the basal recordings there are clearcut vibrations from both splitting of the second sound and an opening snap. It is difficult to be dogmatic over these details without a jugular pulse tracing and in the presence of tachycardia. The interval between the two portions of the split second sound is approximately 0.02 second, between the beginning of the second sound and the opening snap, 0.06 to 0.08 second, and the gallop sound, 0.14 to 0.16 second.

The phonocardiogram in this case, therefore, was of some practical value in helping to analyse the signs of mitral stenosis and incompetence. The basal early diastolic murmur was presumably due to cardiac dilatation and a relative pulmonary incompetence.

Case No. 123 was a 13-year-old girl with rheumatic chorea and early mitral stenosis. There was a previous history of recurrent joint pains. On auscultation a normal first heart sound, soft systolic and mid-diastolic murmurs, and a third heart sound could be heard at the apex. She also had a marked sinus arrhythmia. Unfortunately no followup was available.

The phonocardiogram shows the systolic murmur and the third heart sound, but no mid-diastolic murmur. The extra sound in diastole occurs 0.14 second after the beginning of the second sound.

Case No. 124, a 68-year-old male, presented a difficult diagnostic problem. He was admitted to hospital with congestive cardiac failure and an acute chest infection. He had a 20-year history of exertional dyspnoea, but no effort pain. His blood pressure was 170/90, rhythm normal, but he had auscultatory evidence of aortic sclerosis with some stenosis and incompetence and also a probable mitral stenosis. The signs of the former lesion obscured the latter. Electrocardiogram showed left ventricular hypertrophy and a latent heart block (P.R. = 0.36 second). X-ray screening confirmed the presence of both left ventricular and auricular enlargement. On auscultation there was no evidence of a pre-systolic murmur; the first sound at the apex was normal and there were loud systolic and loud early diastolic murmurs heard all over the praecordium, but maximum at the aortic area. At the mitral area a loud gallop rhythm could be heard, with the added sound occurring in the middle of diastole and obscuring any definite mid-diastolic murmur. This man made a good recovery, but unfortunately no followup was obtainable.

Phonocardiogram confirms the presence of aortic systolic and early diastolic murmurs. They also show a clearcut gallop sound occurring 0.16 second after the beginning of the second sound and just after the P-wave of the E.C.G. It seems to coincide also with a mid-diastolic murmur, and the presence of mitral stenosis is confirmed. Presumably, therefore, this is an audible auricular sound which is associated with latent heart block and possibly also with the congestive cardiac failure. There may be an element of summation between the auricular sound and the mid-diastolic murmur, or in the presence of mitral incompetence and cardiac failure, between the

auricular sound and a rapid filling gallop sound. The phonocardiogram in this case was of some value in establishing the presence of mitral stenosis and in the precise identification of the sounds and murmurs.

Case No. 125 was a 25-year-old male with a double mitral lesion of moderate severity and mild aortic incompetence. The auscultatory signs were typical, but most observers thought that a rapid filling gallop or a third heart sound could be heard. There was no evidence of frank congestive cardiac failure. Eleven months after the recordings were made this man died at home after a cerebral embolism. No post-mortem was carried out.

Phonocardiograms show the systolic and diastolic murmurs, best recorded at the apex (Record A). The extra sound in diastole is seen in all the tracings, but most clearly in the one taken from the third left intercostal space (Record B); it occurs 0.10 to 0.12 second after the beginning of the second sound and seems to occur at the beginning of the mid-diastolic murmur. Once again, a jugular pulse tracing would have been of considerable value in helping to identify it as a gallop sound or opening snap. The cardiac rhythm is normal and regular, and the apparent slight variation in the length of the interval between the second and the extra sound is probably not real, but rather is it due to the variation in intensity of the sound in different tracings. This is probably a rapid filling gallop sound associated with the double valvular lesion and possibly early congestive failure. The patient is also in the age-group where a physiological third heart sound might well be heard, but it is not possible to be dogmatic in this case and the extra sound may in fact be either an opening snap or a gallop.

Case No. 126, a pregnant woman aged 29, had relatively mild mitral stenosis and aortic incompetence, with all the typical auscultatory signs. Phonocardiograms were not of good quality and are not mounted. They did, however, show the pre-systolic and mid-diastolic murmurs and an opening snap occurring 0.08 second after the beginning of the second sound.

Case No. 127, aged 23, was pregnant. She had mitral and aortic disease but showed no evidence of cardiac failure. Phonocardiograms were made on three occasions and show pre-systolic and mid-diastolic murmurs, also splitting of the second sound, an opening snap, and a diastolic sound which, in view of the patient's age and the pregnancy, is probably a physiological third heart sound. The early diastolic murmur was not clearly demonstrated. These records and the patient's history are discussed in the section on splitting of the heart sounds.

Case No. 128 was a 48-year-old woman with severe thyrotoxicosis, but was also thought to have mitral stenosis. As a result of tachycardia there was some doubt as to the precise nature of the physical signs, and pre-systolic and mid-diastolic murmurs were suspected but indefinite. The first sound was accentuated and there was no opening snap to be heard. Three years later this patient was still troubled with symptoms referable to thyrotoxicosis and by this time she was fibrillating.

Phonocardiograms were made when she was first seen and demonstrate a definite pre-systolic murmur, which starts well before the Q.R. segment of the electrocardiogram, and there is a fine splitting of the second sound, the second component following 0.04 second after the beginning of the first part of the sound. Although no definite opening snap or mid-diastolic murmur was demonstrable,

the pre-systolic murmur serves to confirm the presence of mitral stenosis.

Case No. 129, a 73-year-old male, had auricular fibrillation and an acute respiratory infection. There was an anginal history and his left leg had been amputated nine months previously following a popliteal embolism. He recovered from the chest infection, and during routine X-ray screening of the chest it was noted that his left auricle was unduly prominent. The heart was carefully examined and auscultated again, and this re-examination revealed a probable apical mid-diastolic murmur, although this was not absolutely clearcut.

Phonocardiograms taken with the patient lying on his left side with breath held demonstrated a mid-diastolic murmur, and the presence of mitral stenosis was confirmed. One year later this man was alive and well, but there has been no followup since.

Eight patients remain - six with a mitral lesion only and two with both mitral and aortic disease. They will be briefly described and representative recordings included.

Cases No. 130 and 131 had both mitral and aortic lesions. No. 130, a 19-year-old girl, had severe rheumatic heart disease, with a history of repeated episodes of congestive cardiac failure. On examination she was found to have auricular fibrillation, and on auscultation at the apex there was a loud first sound, a very loud systolic murmur, and both early diastolic and mid-diastolic murmurs. An extra sound, early in diastole and maximal at the left sternal edge was also consistently heard. There was some

difference of opinion between observers as to whether this was an opening snap or a third heart sound, and the early diastolic murmur was not always audible.

Phonocardiograms show the loud first sound and apical pan-systolic murmur, also the early diastolic running into the mid-diastolic murmur (Record A). No opening snap or third heart sound can be seen, but a split second sound is recorded at the left sternal edge in the fourth intercostal space (Record B). The latter part of the split commences 0.02 to 0.04 second after the beginning of the second sound.

Almost exactly two years after these recordings were made this patient died and at post-mortem gross mitral and tri-cuspid stenosis and incompetence were discovered. Considerable cardiomegaly with right ventricular hypertrophy and left auricular dilatation were both present, but the aortic and pulmonary valves were normal. The early diastolic murmur must have been due to functional aortic or pulmonary incompetence, and it is conceivable that the audible gallop sound may be masked on the tracing (A) by the early diastolic and mid-diastolic murmurs. On this particular record the tracing taken from the fourth intercostal space in the left parasternal line shows a possible early diastolic sound which could be due to a rapid filling gallop, but it is inconstant. The record is of poor quality also and serves mainly as a reference tracing by showing the position of the second sound.

Case No. 131, a man aged 25, had no history of disability or previous rheumatic fever. The presence of aortic incompetence was confirmed by an easily audible early diastolic murmur, but there was no definite pre-systolic and only a doubtful mid-diastolic

murmur. There was, however, a loud mitral first sound and clearcut opening snap. Unfortunately no followup was available for this patient and the phonocardiograms were not technically good. They do, however, show a probable opening snap occurring 0.08 second after the beginning of the second sound, and this may well be taken as confirmatory evidence of mitral stenosis. The prolonged diastolic murmur might otherwise have been taken for an Austin Flint murmur associated with mitral incompetence.

Cases Nos. 132, 133, 134 and 135 were all women in their forties, with normal rhythm, no congestive failure, and pure mitral stenosis of mild degree. The typical auscultatory signs, including a clearly audible opening snap, were manifest. (The interval between the beginning of the second sound and the opening snap was 0.07, 0.06, 0.09 and 0.10 second respectively). The only followup available was on patient No. 133, who was alive and well three years later.

Phonocardiograms from two of these cases are presented here as examples.

Case No. 134 had apical recordings made with the Boulitte machine, and these show the pre-systolic murmur, the loud first sound, opening snap and mid-diastolic murmur.

Case No. 135 has an apical recording mounted, which shows only faint vibrations of a pre-systolic murmur, a loud first sound and a clear opening snap.

The remaining four patients all had mitral stenosis with auricular fibrillation.

Case No. 136, a 55-year-old woman, had on auscultation an apical mid-diastolic murmur and a definite opening snap, heard all over the praecordium, but maximum down the lower left sternal edge. There were no other auscultatory abnormalities. Phonocardiograms from the apex (Record A) showed a loud first heart sound, a mid-diastolic murmur and a clearcut opening snap varying in its precise position with the irregular cycle length of auricular fibrillation, but occurring from approximately 0.06 to 0.08 second after the beginning of the second sound. Recordings from the pulmonary area (B) show the opening snap particularly clearly; they were made with the patient's breath held in full expiration, at which time the snap was heard loudest.

Case No. 137 was a 42-year-old housewife, with a five-year history of mild exertional dyspnoea, but no frank cardiac failure. On auscultation at the apex the first sound was accentuated; there was no systolic murmur, an opening snap was clearly audible, as was a long rumbling mid-diastolic murmur. The snap was maximum down the left sternal edge but heard all over the praecordium. Shortly after this patient was first seen the rhythm returned to normal, but unfortunately only one opportunity occurred when recordings could be made, and this was during the episode of auricular fibrillation. Four years later the patient was alive and well, but fibrillation had become permanently re-established.

Phonocardiograms from the apex (Records A and B), taken with different camera speeds, show the loud first sound, the opening snap, and the mid-diastolic murmur. In recordings from the pulmonary area (C) the second sound is loudest and the snap is clearly seen. The distance between the beginning of the second sound is from 0.06 to 0.10 second and varies considerably with the irregular cycle length.

Case No. 138 was a 46-year-old housewife, who had a series of pulmonary emboli associated with the onset of cardiac failure and auricular fibrillation. Fortunately she made a good recovery after full digitalisation, but the fibrillation remained permanent. On auscultation the signs of mitral stenosis were typical, but the mid-diastolic murmur was not easily heard. It was best appreciated with the patient in the left lateral position. Three years later this woman was alive and relatively well, but her activities were fairly severely restricted by exertional dyspnoea.

Phonocardiograms failed, even after repeated attempts, to demonstrate the mid-diastolic murmur, but the opening snap was well recorded from all areas, especially from the pulmonary area. The snap occurs from 0.10 to 0.12 second after the beginning of the second sound.

Case No. 139, A 58-year-old housewife, had a five-year history of increasing exertional dyspnoea. She was in cardiac failure and had all the signs of pure mitral stenosis of fairly severe degree. An opening snap was heard over all the praecordium. This woman died suddenly at home two months later and no post-mortem was carried out. The immediate cause of death was not clear.

Phonocardiograms showed the mid-diastolic murmur and opening snap occurring 0.10 to 0.12 second after the second sound.

TABLE 20

RELATIONSHIP BETWEEN
THE SECOND SOUND, THE OPENING SNAP, AND THE RAPID FILLING GALLOP

AUTHORITY	OPENING SNAP Interval in seconds between beginning of second sound and beginning of snap	SPLITTING OF SECOND SOUND Interval in seconds between beginning of each component of second sound	RAPID FILLING GALLOP Interval in seconds between beginning of second and gallop sounds
Margolies & Wolfarth (1932)	Min. 0.03 Max. 0.19 Majority 0.06 to 0.11	Less than 0.06	Min. 0.12 Max. 0.20
Mounsey (1953)	Min. 0.03 Max. 0.14 Average 0.07	Min. 0.01 Max. 0.05 Average 0.03	Min. 0.16 Max. 0.24 *
Present series (1955)	Min. 0.04 Max. 0.14 Majority 0.06 to 0.10	Min. 0.02 Max. 0.06 Average 0.04	Min. 0.10 Max. 0.22 Average 0.15

* In this case the measurement was from the beginning of the second sound to the mid-point of the gallop sound, and the figures are quoted from Orias and Braun-Mendez (1939) and Rappaport and Sprague (1942).

TABLE 21

ANALYSIS OF HEART SOUNDS IN THE OPERATIVE
SERIES OF PATIENTS WITH MITRAL LESION

Case No.	Sex	Age	Time interval between opening snap and second sound	Time interval between rapid filling gallop & second sound	Diagnosis confirmed at operation
95	F	32	-	-	Mitral stenosis and incompetence
96	F	37	0.10	-	-do-
97	F	30	0.06	-	Mitral stenosis
98	F	16	0.10	-	-do-
99	F+	28	? See text	-	-do-
100	F	27	0.08 to 0.10	-	-do-
101	F+	27	0.06	0.10 *	-do-
102	M	37	0.08 to 0.10	-	Mitral stenosis and slight incompetence
103	M	20	0.08 to 0.10	0.18 *	-do-
104	F	30	0.08 to 0.10	0.16 *	Mitral stenosis
105	F	35	0.08 to 0.10	-	-do-
106	F	47	0.06 to 0.10	-	-do-
107	F	37	0.08 to 0.10	-	-do-
108	M	40	0.08 to 0.10	-	Mitral stenosis with slight incompetence
109	F	38	0.08 to 0.10	0.14 to 0.16 *	Mitral stenosis
110	F	29	0.08 to 0.10 [∅]	-	Mitral stenosis with slight incompetence
111	F+	26	-	? See text	Mitral stenosis
50	M	45	-	-	Mitral stenosis with slight incompetence

+ Pregnant

∅ Opening snap seen on sound tracing, but not heard.

* Rapid filling gallop occurred in post-operative period.

Measurements in seconds from the beginning of the second sound to the beginning of the opening snap or rapid filling gallop.

In cases of auricular fibrillation average figures for the time intervals have been given.

TABLE 22

ANALYSIS OF HEART SOUNDS IN THE NON-OPERATIVE
SERIES OF PATIENTS WITH MITRAL LESION

Case No.	Sex	Age	Time interval between opening snap and second sound	Time interval between rapid filling gallop & second sound	Clinical diagnosis
112	F	29	0.06 to 0.08	0.14 to 0.16	Mitral stenosis. Pregnant.
113	M	52	0.08	0.16	Mitral stenosis. Myocardial infarction.
114	F	64	0.08	-	Mitral stenosis and aortic incompetence.
115	M	50	-	0.14	Mitral stenosis and incompetence. [⊗]
116	F	44	0.08 to 0.10	-	Mitral stenosis. [⊗]
117	F	54	-	0.10 to 0.12	Mitral stenosis and incompetence. [⊗]
118	M	44	-	-	-do-
119	F	63	0.06 to 0.10	0.14 to 0.16	Mitral stenosis and incompetence. Aortic incompetence.
120	M	40	0.08 to 0.10	-	Mitral stenosis and myocardial infarction.
121	F	55	0.08 to 0.10	-	Mitral stenosis. [⊗]
122	F	17	0.04 to 0.06	0.14 to 0.16	Mitral stenosis and incompetence. [⊗]
123	F	13	-	0.14	Mitral stenosis and chorea.
124	M	68	-	-	Mitral stenosis. Aortic sclerosis. [⊗] Latent heart block.
125	M	25	-	0.10 to 0.12	Mitral stenosis and incompetence. Aortic incompetence.
126	F	29	0.08	-	Mitral stenosis and aortic incompetence. Pregnant.
127	F	23	0.09	0.14	-do-
128	F	48	-	-	Mitral stenosis. Thyrotoxicosis.
129	M	73	-	-	Mitral stenosis.
130	F	19	-	-	Mitral stenosis and incompetence. [⊗] Tricuspid stenosis.

TABLE Contd./

TABLE 22 (Contd.)

131	M	25	0.08	-	Mitral Stenosis and aortic incompetence.
132	F	42	0.07	-	Mitral stenosis.
133	F	40	0.06	-	-do-
134	F	43	0.09	-	-do-
135	F	46	0.10	-	-do-
136	F	55	0.06 to 0.08	-	-do-
137	F	42	0.06 to 0.10	-	-do-
138	F	46	0.10 to 0.12	-	-do-
139	F	58	0.10 to 0.12	-	-do- *

* Congestive cardiac failure also.

SUMMARY AND CONCLUSIONS

A study has been made of sixty-six recordings from forty-six patients with mitral valve disease, mostly cases with predominant or pure mitral stenosis. Eighteen of these patients were submitted to surgical operation and eleven had phonocardiographic recordings made both before and after operation.

The occurrence of an opening snap and a rapid filling gallop sound and their relationship to the precise anatomical lesion, together with the timing of these events in the cardiac cycle, have been analysed and set out in Table 21 and Table 22.

The following observations and conclusions are drawn as a result of a study of these patients and their heart sound recordings.

1. The phonocardiograph recordings were almost always of considerable interest, but usually served only to confirm the clinical auscultatory findings. On a few occasions, however, additional information of real practical value was obtained from these tracings. The actual recording and analysis of the sounds and murmurs was of the greatest value in training the clinician in the art of careful auscultation and in analysing the individual components of the various sounds and murmurs.
2. It was found as a general rule that heart sounds, both normal and pathological, were much more easily recorded than murmurs. The first and second heart sounds, splitting of these sounds, and the presence of additional gallop sounds or the opening snap were almost always better detected by the phonocardiogram

than by the human ear. In view of the lack of an accurate method of calibration, no accurate measurements or comparisons of sound intensity could be made, although frequently, in fact, certain phenomena such as the increased intensity of the first sound in mitral stenosis was quite obvious from the records. The murmurs, on the other hand, were usually better identified by the ear than by the phonocardiographic recording. This, however, was not always the case, and on several occasions confirmation of the presence of doubtful murmurs was possible.

3. On a number of occasions, as in Cases Nos. 99, 102, 111, 117, 124, 122 and 130 , there was some difference of opinion between individual observers as to the precise nature of the auscultatory signs in a particular case. Analysis of the phonocardiograms almost always solved this particular difficulty, and in this way a group of observers ultimately came to fairly close agreement on the precise nature of certain sounds and the criteria for their diagnosis. The differentiation between splitting of the second sound, a rapid filling gallop, and the opening snap of mitral stenosis was the most frequent practical problem, which was solved by phonocardiography in this series.
4. A careful study has been made of these three phenomena, and the time intervals between the beginning of the second heart sound, the second component of a split second sound, the opening snap and the rapid filling gallop have been measured in a large series.

With some of the tracings, accurate measurement of small time intervals is difficult owing to absence or irregularity of marker, notably with the Boullitte machine. However, with the vast majority of tracings made with the Elmquist instrument, measurements of up to 0.02 second could be made with a high degree of accuracy. Arbitrary criteria for the time relationship between the second heart sound, the second component of a split sound, the opening snap and the rapid filling gallop have been established as a result of studies by several workers.

Using these criteria as a basis (see Table 20), but also taking into account the various clinical, radiological, electrocardiographic and other features of the cases, a series of 218 recordings from 159 patients with mitral disease has been analysed. The opening snap was in fact recorded from 112 patients, 39 of them both before and after valvotomy.

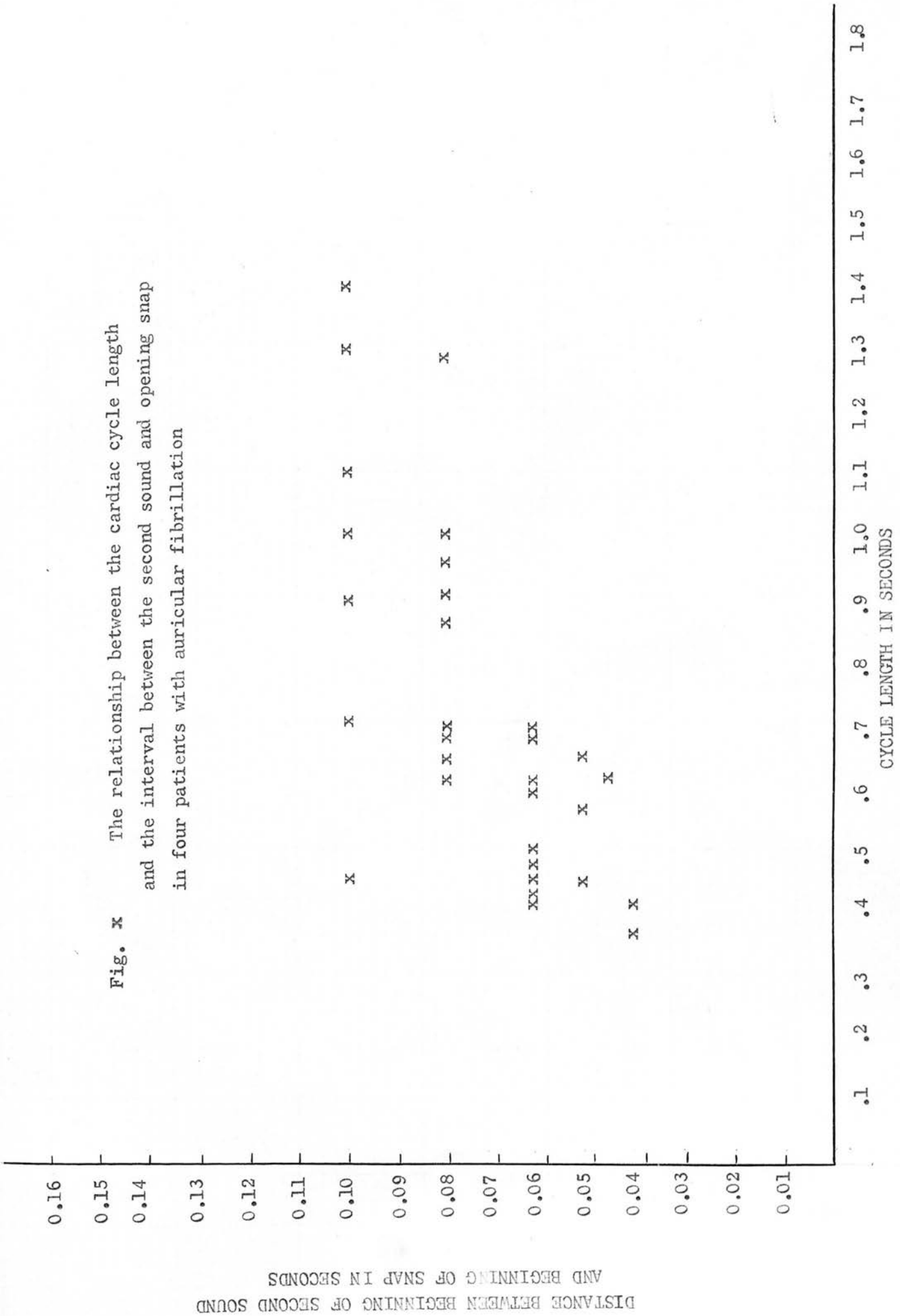
In order to obtain this grand total of recordings and cases a series of recordings made by Mr. R. Alford of the Cardiac Department, Western General Hospital has been included for the purpose of measurements only, in addition to the 66 recordings presented in this thesis. Only those records in which accurate measurement of the relevant time intervals could be made have been included.

From these observations, the interval between the beginning of the second sound and the beginning of the opening snap appears to be remarkably constant in patients with mitral stenosis, the longest being 0.14 second and the shortest 0.04 second; average 0.082 second in patients with normal rhythms and 0.096 second with auricular fibrillation.

Several observers have noted minor variations of this interval in the presence of auricular fibrillation, whereas in normal rhythm it is relatively constant. It has been claimed by Margolies and Wolferth and by Messer and others that this measurement between the second sound and the snap varies directly with the duration of the previous cardiac cycle. This observation has been confirmed by the present study, as can be seen by reference to Fig. x, where the R-R interval of the electrocardiogram has been charted against the time interval in question. A statistical analysis has been made of records of four patients with mitral stenosis and auricular vibrillation. (See Appendix). After a very short cardiac cycle the opening snap may not appear on the tracing and is presumably either absent altogether or masked by the second sound.

The time relationship between the second heart sound and the third sound and also the distance between the two components of a split second sound has already been discussed in previous sections of the thesis. In order to facilitate contrast and comparison, Table 20 has been constructed. This compares the time intervals under discussion, as found by Margolies and Wolferth (1932) and by Mounsey (1953), and in the study here presented. There is a small amount of overlap, as can be seen, but in the majority of cases the opening snap occurs at a time when split second sounds and rapid filling gallop do not occur. Only in twelve cases out of the total of 112 in which a snap was recorded did the sound fall outside the range of 0.06 to 0.10 second after the second sound. Wolferth and Margolies also claim that the human ear can be trained to detect sound intervals

Fig. x The relationship between the cardiac cycle length and the interval between the second sound and opening snap in four patients with auricular fibrillation



up to 0.12 second with an accuracy of + or - 0.02 second. It is, therefore, reasonable to suppose that these differences under discussion could be recognised by the trained listener.

5. During these recordings it was noted that the opening snap was heard equally well at the apex, the pulmonary area, and the fourth intercostal space near the left sternal edge in seventy cases. It was heard loudest at the pulmonary area in ten cases, at the apex in five, and equally well at the pulmonary area and left sternal edge in thirteen; at the apex and left sternal edge in six and at the apex and pulmonary area in two. It was usually best recorded at the area where it was best heard, but for technical reasons this was not always the case, and in view of the variety of amplifiers used and the absence of calibration, no definite conclusion can be drawn from the observation^{of} loudness and ease of recording from various sites on the chest wall. The snap was usually equally well heard with the patient either lying or sitting. In two cases, Nos. 99 and 112, there was a change related to posture. The former was thought to have an opening snap when auscultated in the supine position, but when sitting erect the impression was of a split second sound. Phonocardiograms confirmed the presence of a split second sound, and at valvotomy the presence of pure mitral stenosis was confirmed. In the latter case a split second sound was heard and recorded when the patient was supine, but an opening snap was heard and recorded in the erect position.

6. The opening snap occurs just as frequently in auricular fibrillation as in normal rhythm. It also may be heard with the ear and seen on the phonocardiogram in the presence of aortic incompetence, although a loud early diastolic murmur tends to obscure it. On several occasions the opening snap has been demonstrated on the same tracings as splitting of the second sound and a rapid filling gallop.
7. A loud clear opening snap of characteristic auditory quality (most aptly described by the word "snap") was usually associated with pure mitral stenosis, although occasionally patients with isolated stenosis diagnosed clinically and confirmed at operation did not exhibit this particular sign. Out of 32 cases with pure mitral stenosis only six did not have a snap heard or recorded, and complicating factors were present in four of these six. Occasionally, also, despite the presence of a significant degree of associated mitral incompetence confirmed either at autopsy or operation, an opening snap, often faint or slightly atypical, was present. In seven out of fourteen cases with mixed mitral stenosis and incompetence an opening snap was detected, but at least four of these had only a mild degree of incompetence. Usually a clear typical opening snap was an auditory and phonocardiographic sign of some value in establishing the diagnosis of pure mitral stenosis. In several patients gross calcification of the mitral valve was found at autopsy or at operation. In only one of these (Case No. 110) was a possible opening snap recorded. This was never heard on

auscultation, but can just be seen on one of several tracings. In two others of the operative series slight calcification of the cusps was encountered, but the valves themselves were moderately supple. In both cases a snap was heard and recorded.

8. A split second sound was heard and recorded in fourteen of the 46 cases. It did not seem to have any specific features which distinguished it from the physiological split second sound.
9. A rapid filling gallop was sometimes heard and demonstrated in patients with mitral stenosis, but never in a case of uncomplicated stenosis of significant degree (See Table 21 and Table 22). It was encountered in young patients who were either suffering from a mild degree of stenosis or were pregnant, when the gallop may in fact have been due to a physiological third sound. Otherwise it was only found in patients with a significant degree of associated mitral incompetence, tricuspid or aortic disease in the presence of congestive cardiac failure. It was also rarely heard, but more often recorded during the immediate post-operative period following valvotomy. This was presumably related to the operative handling of the myocardium and pericardium or to the establishment of a significant degree of mitral incompetence as a result of the valvotomy.

Occasionally the early vibrations of a mid-diastolic murmur may take on the appearance of a gallop sound. The phonocardiographic tracing shows this as a single or double

low-frequency vibration as in Case No. 113 and it is impossible to differentiate on the record from a true rapid filling gallop sound. In some cases, where other factors such as pregnancy, associated mitral incompetence or congestive failure, are present, it may in fact be a gallop sound superimposed on the murmur as it certainly occurs at the appropriate point in diastole.

10. The pre-systolic murmur of mitral stenosis was not always easy to demonstrate on the phonocardiogram even though the recordings seemed to be technically perfect in every other way. It is suggested that this murmur may be at least in part an auditory illusion associated with a very loud mitral first sound. Nevertheless, a pre-systolic murmur has often been recorded in this series, and the direct association between auricular systole and the pre-systolic murmur has been demonstrated in Case No. 110 and also in Case No. 94 in the previous section. In both these cases the heart sounds were recorded during normal rhythm and auricular fibrillation. Pre-systolic murmurs were also clearly demonstrated occurring during the P.Q.R. period in several cases, including Nos. 101, 107, 108, 113, 127 and 134. In all the cases with auricular fibrillation presented in this section no vibrations are visible on the sound tracing before the R wave and, in most cases, the S wave of the electrocardiogram (for example, Cases Nos. 96, 102, 115, 117, 118 and 119.) Any sounds occurring before the R.S. segment of the E.C.G. are presumably, therefore, resulting from auricular systole.

11. Unlike the published experience of other workers, some difficulty was often encountered in recording low-pitched mid-diastolic murmurs, and on only one occasion was such a murmur clearly demonstrated when not heard by the ear.
12. Two cases had predominant mitral incompetence, and a further ten cases had predominant mitral stenosis with an associated mild degree of incompetence. Six of these had recordable mitral systolic murmurs, but in none of them was the murmur maximal in late systole as suggested by Brigden and Leatham (1953). In most, the murmur was pan-systolic, but maximum in early systole. Even in four cases with a fairly gross degree of mitral incompetence confirmed at either operation or post-mortem (Cases Nos. 96, 115, 122 and 130) the murmur was maximum in early systole or else filled the whole of systole without localised accentuation of intensity. Its configuration was, however, quite unlike the diamond-shaped systolic murmur of aortic stenosis which is maximum in mid-systole.

Clear splitting of the second sound was also encountered in these cases, and here the findings are in agreement with those of Brigden and Leatham.

13. Two patients with a marked degree of mitral incompetence underwent surgical operation in an attempt to carry out a plastic repair of the lesion. The second of these two had both a split second sound and an opening snap demonstrated on the phonocardiograph tracing both before and after operation. Both patients survived the operation and were relatively well ten months later, with auscultatory signs unchanged.
14. Sixteen patients with pure or predominant mitral stenosis and a relatively minor degree of incompetence had a conventional mitral valvotomy and all but one were classed as having been successful. With this one exception, all maintained their improvement during a followup period of from one to three years.
15. Certain features of the operative series are worth special mention.

Case No. 50 - a 45-year-old male with right bundle-branch block, a shortened P.R. interval, and established mitral stenosis, has already been considered as he had an audible auricular gallop. Operation confirmed the presence of mitral stenosis with a moderate degree of associated incompetence and some valve calcification. No opening snap or pre-systolic murmurs were audible, and presumably the atypical signs were in this case due to the conduction defect and accompanying mitral valve lesion.

Ten patients had phonocardiograms made both before and after valvotomy; all showed the usual physical signs of mitral stenosis, and in some cases the auscultatory signs were changed following the operation, but in others no difference

was noticed. No definite conclusions can be drawn from these graphic changes because records taken at different times cannot be accurately compared. Some comments, however, can be made. In Case No. 102 the mid-diastolic murmur and opening snap seem to be more marked following the operation. Pre-operative tracings here had been of considerable value as they confirmed the presence of a rather doubtful snap, and this evidence combined with other features helped to confirm the diagnosis of predominant stenosis. Operation was therefore attempted and considerable benefit achieved.

In Cases 103 and 104 the opening snap was heard and recorded pre-operatively, but disappeared after valvotomy. In the former case it recurred three months later. In both, a transient rapid filling sound appeared in the tracing in the post-operative phase. In several patients the opening snap persisted following a successful valvotomy.

Case No. 111, a 26-year-old woman, had a moderate degree of aortic stenosis as well as mitral stenosis, but she had no opening snap. Despite the associated mitral lesion the phonocardiograph tracing made from the aortic area showed typical configuration of the ejection murmur and hence was of some value in helping to rule out the existence of mitral incompetence as a cause for the systolic murmur.

16. In this operative series, none of the eighteen cases have died up to the time of followup (one to three years). In the remaining 28 cases, nine are known to have died during the followup period. Five of these died in hospital and post-mortem confirmation of diagnosis was available.

17. In an effort to confirm Wells's observation (1954) measurements were made of the distance between the Q-wave of the electrocardiogram and the beginning of the first heart sound in both pre- and post-operative recordings. This was carried out with Cases Nos. 102, 103, 107, 108, 109, 110 and 111. Where an opening snap was present, similar measurements were made between the beginning of the second sound and the opening snap. In each case the average reading was taken from the measurement of several cycles. It was found that in the majority of cases, successful valvotomy led to a diminution of the first of these two measurements, but there was no significant difference between the pre- and post-operative measurements of the interval between the second sound and the opening snap (Table 23). Any differences in either case were extremely small and these measurements were found to be difficult to make, particularly as it is so often difficult to be precise about the exact position of commencement of the first or second sound.

It is felt that little, if any, significance can be attached to these results. Precise comparison cannot be made without some calibration system so that the pre- and post-operative records can be made under exactly similar circumstances with the same frequency recordings, the same intensity, and, of course, the same location of the microphone on the chest.

TABLE 23

Measurement of time interval
between Q wave and first heart sound, and the second
heart sound and opening snap, in both
pre- and post-operative phonocardiogram

Case No.	Q - I interval in seconds		II - O.S. interval in seconds	
	Pre-operative	Post-operative	Pre-operative	Post-operative
102	0.07	0.04	0.1	0.1
103	0.1	0.06	0.1	=
107	0.06	0.06	0.09	0.09
108	0.06	0.05	0.1	0.08
109	0.06	0.05	0.1	0.08
110 ³⁶	0.08	0.08	=	=
111	0.08	0.06	=	=

In each case the measurement given is the average of several cycles.

* Poor operative result.

MISCELLANEOUS RECORDINGS

A number of recordings were taken of the heart sounds and murmurs from a variety of less common, but interesting conditions and also from problem cases in which it was hoped that a phonocardiographic record might assist in diagnosis.

Complete Heart Block

Two female patients and one male had complete heart block with audible auricular sounds, and recordings were made from all of these cases.

The first example, Case No. 140, a single woman aged 62, was admitted in congestive failure, and at this time the auricular sounds were quite clearly heard. By the time she was well enough for recordings to be made these sounds were no longer audible, but can still be seen on the tracings. She also had an apical systolic murmur, and an E.C.G. revealed the presence of a right bundle branch block. This patient made a temporary recovery from the cardiac failure, but despite continued nursing care and treatment, she died three months later. Autopsy revealed a gross degree of generalised atherosclerosis with considerable myocardial fibrosis. Recordings were made on two occasions, and the phonocardiogram shows the auricular sounds coinciding with the P-waves of the E.C.G. Jugular venous pulse tracing is of poor quality and does not show the "a" waves of auricular systole well. The first sound is broadly split but commences well after the R-wave of the E.C.G. and varies with the proximity of auricular systole. A curious late systolic sound is seen, which may in fact represent broad splitting of the second sound, although neither splitting of this sound or any late systolic sound was audible.

The second case, No. 141, aged 54, is of particular interest as a good jugular pulse tracing was also obtained. She had a previous history of angina pectoris and was admitted to hospital with a hemiplegia. Her pulse-rate was found to be regular at 45/minute, blood pressure was $\frac{244}{98}$, and the heart was not clinically enlarged; neither was there any sign of cardiac failure. When this patient was examined carefully in the supine position it was noted that, in addition to the regular bradycardia of complete heart block, neck vein pulsations could be seen of a rather unusual type. These consisted of small frequent auricular waves at a rate of approximately 75/minute, and a less frequent wave at a much slower rate - approximately 50/minute - but much greater amplitude. This wave of greater amplitude was found, on palpation, to be a venous wave, and not a carotid pulsation. The carotid pulse could be palpated only with difficulty, and was running at a steady 45/minute. It was also noted clinically at this time that, on auscultation, an auricular sound could occasionally be heard, and the phenomena of variation in intensity of the first heart sound was very obvious. For these reasons, a simultaneous recording was made of the E.C.G. (Lead II), the neck vein phlebogram, and the heart sounds recorded with a low-frequency band at the apex. These recordings demonstrate clearly the independent rhythm of the auricles and ventricles, the variation in intensity of the first sound, and the occurrence of a giant "a" wave on the phlebogram whenever it chanced that the auricle contracted during the phase of ventricular systole, i.e., when the tricuspid valves were closed. At all other times, when the tricuspid valves were open in ventricular diastole, the "a" wave on the phlebogram was

of the normal size. Also well seen in the tracing was the coincidence of the P-wave in the E.C.G., the "a" wave in the phlebogram, and the auricular sound in the P.C.G. Similarly, the intensity of the first sound varies with the relationship of auricular systole to ventricular systole and must depend on the position of the tricuspid and mitral valves at the moment of ventricular systole (Griffith, 1911).

The third case, No. 142, was similar in almost every respect to case No. 141. His recordings were used for a demonstration in the Department of Medicine at the University of Edinburgh.

Continuous Vascular Murmurs

Case No. 143 was a young lactating woman, aged 28. On auscultation a continuous vascular hum was heard, occupying the whole of systole and diastole and being loudest over the third and fourth left space in the parasternal line. This murmur was similar in its auscultatory characteristics to the Gibson murmur of patent ductus arteriosus, it had not been noted before the pregnancy, and it disappeared during the followup period after lactation had ceased. No other clinical or radiological abnormalities were found. Recordings were made from the third left space, using two different frequency bands (Records A and B). The murmur is seen to be maximal in systole, particularly mid-systole, and there is a slight accentuation in pre-systole. There is also a marked respiratory variation. The murmur does not have the appearance described by Bonham-Carter (1955) and others, nor is it like a purely venous hum as it was not obliterated by local

pressure. It was presumably due to a small arterio-venous aneurysm, perhaps involving the internal mammary vessels, which has assumed increased dynamic significance as a result of the increased local blood flow associated with lactation. Alternatively, it may have been due simply to increased vascularity of the mammary tissues and a localised innocent vascular abnormality.

Case No. 144, a woman of 73, had gross portal cirrhosis with ascites, and a continuous venous hum was heard over the epigastrium. Venous hums to be heard on auscultation over the epigastrium in cases of portal cirrhosis were described more than a hundred years ago by Pégot (1833) and by Cruveilhier (1835), and early in this century, by Baumgarten (1908). Hanganutz (1922) collected six cases all of which presented a small cirrhotic liver, portal hypertension, a venous anastomosis in the falciform ligament and abdominal wall connecting the portal and the epigastric veins and a bruit or hum audible over the epigastrium and lower end of the sternum. He believed this syndrome to be a separate clinical and pathological entity and applied to it the name Cruveilhier-Baumgarten cirrhosis. Armstrong et al. (1942) reviewed the cases published to that date and divided them into two groups. The first group consisted of young people in whom there were atrophy of the parenchyma of the liver, but little or no fibrosis, and a large patent vein which joined the left portal vein to the abdominal parietal veins by way of the free margin of the falciform ligament. Armstrong and his colleagues believed that this was the umbilical vein and that its congenital patency was the primary abnormality in these cases, the changes in the liver being secondary to the

deprivation of portal blood. They considered these cases to be examples of Cruveilhier-Baumgarten cirrhosis. In the second and much larger group, which they referred to as the Cruveilhier-Baumgarten syndrome, no congenital anomaly was present. The primary condition was hepatic cirrhosis with associated portal hypertension. Partial recanalisation of the dilated umbilical vein occurred in some cases and in others enlargement of the para-umbilical veins and of the connections between them and the epigastric veins. The patient No. 144 was thought to be an example of the second group of cases described by Armstrong et al., that is to say, a case of the Cruveilhier-Baumgarten syndrome, in which cirrhosis and portal epigastric anastomosis were associated with gross ascites. On auscultation the murmur was heard over the epigastrium, the medial third of both costal margins, and over the sternum as far up as the third costal cartilage. It was maximal on either side of the xiphoid cartilage, was heard in all phases of respiration and did not vary with posture. It resembled the murmur of a patent ductus arteriosus in that it was continuous throughout systole and diastole, and varied slightly in intensity and pitch with the cardiac cycle. In character the murmur was a soft high-pitched hum not unlike the noise of wind blowing through telegraph wires, and conformed to the original description of a venous hum given by Laennec (1819), who likened it to the sound of the "sea or that produced by the application of a large shell to the ear". There was no palpable thrill. Although no large subcutaneous veins were visible above the umbilicus the murmur disappeared when light digital pressure was applied across the epigastrium in the mid-line and reappeared immediately pressure was released.

The phonocardiogram shows a continuous venous hum recorded from a point just over the lower end of the sternum and which disappears when light digital pressure is applied with the edge of the hand across the epigastrium, whereas the normal heart sounds persist.

Necropsy ultimately confirmed the presence of gross hepatic cirrhosis, and the portal-epigastric anastomosis was demonstrated after death by injecting into the superior mesenteric vein a solution of methylene blue and 12% potassium iodide. The methylene blue dye aided the dissection, and a drawing of this is shown in Fig. 11.^{*} The potassium iodide was intended as a radio-opaque medium, and lateral and antero-posterior X-rays were in fact taken, but were not very successful. They did, however, show that the radio-opaque substance had traversed the portal epigastric anastomosis and had entered the internal mammary veins.

It is not possible to determine exactly the site of origin of the murmur in this case. According to Lutembacher (1936) the murmur arises where a small vein enters a dilated one, for example at the junctions of the para-umbilical veins with the dilated and tortuous epigastric veins. The disappearance of the sound on light pressure over the epigastrium would be compatible with this explanation. On the other hand, the maximum intensity of the bruit in the region of the xiphisternum suggests that it may originate in turbulence occurring at the junction of the left portal and umbilical veins. The disappearance of the sound on pressure over the epigastrium could then be accounted for by the cessation of flow into the patent end of the umbilical vein.

Observations made by McFadzean and Gray (1953) provide some support for this view. In five out of nine cases of hepatic cirrhosis they reported that a venous hum was not audible over the epigastrium but was detected on direct auscultation of the liver and became progressively louder as the porta hepatitis was approached. Large veins were demonstrated in the falciform ligament in three of these cases. In another case which they reported in detail they concluded that the murmur originated in arterio-venous communications within the liver and was conveyed directly to the abdominal wall. That this was not the mechanism of its production in the present case is suggested by the fact that the murmur disappeared when light pressure was applied to the epigastrium. This case was reported in detail by the author and Mr. A.I.S. Macpherson (Brit. Heart Journal, 1955, 17, 105).

Foetal Heart Sounds

At the request of Dr. (now Professor) A.S. Duncan, foetal heart sounds from healthy pregnant women on four occasions. (Cases No. 145, 146, 147 and 148). It was felt that in cases of foetal distress a simple recording technique might enable the obstetrician to assess the foetal state from hour to hour, even when the heart sounds could not be heard clinically. No difficulty was experienced in recording these sounds under normal conditions, the microphone being placed on the mother's abdominal wall over the point where the sounds were loudest, and a maternal apical phonocardiogram was recorded synchronously as a reference tracing. One example of these recordings is shown, taken from Case No. 145.

MISCELLANEOUS DIAGNOSTIC PROBLEMSUnexplained tachycardia

Case No. 149, an extremely nervous and emotional spinster, aged 36, had a persistent and unexplained tachycardia, the rate being normally about 120/minute and never falling below 90/minute even during sleep. She had a deep-rooted cardiac neurosis based on a number of factors, including an unsatisfactory domestic background, constitutional inadequacy and previous medical mismanagement. There were no other clinical abnormalities such as thyrotoxicosis or signs of other disease. X-ray screening of the heart and lungs were normal, and electrocardiogram revealed a sinus tachycardia with a normal P.R. interval (0.16') but large P-waves.

Clinically the first sound at the apex was accentuated and there was a grade III apical systolic murmur and a split second sound. No diastolic murmurs were heard. The tachycardia was such that further analysis of the heart sounds was difficult, but it was thought by some that a third heart sound was present. A possible mitral stenosis was queried, and in an effort to aid diagnosis a phonocardiogram was recorded.

The recording made on two occasions confirmed the presence of a loud first heart sound and a pan-systolic murmur at the apex. No diastolic murmurs were seen, but an extra sound coincident with the P-wave was recorded (Records A and B), and in the pulmonary area the second sound was split (Record C). Electrocardiographs suggested auricular hypertrophy and the extra sound can be seen to have a fixed relationship to the P-wave and to begin approximately 0.20' after the beginning of the previous second sound.

Although the picture was not entirely clear, the tachycardia was thought to be secondary to an anxiety state and cardiac neurosis. Mitral stenosis was excluded on clinical, radiological and phonocardiographic evidence, and the gallop rhythm was presumably a summation gallop associated in some way with auricular hypertrophy and tachycardia. A course of thiouracil was in fact tried at a later date, without benefit, and at no time was there any definite evidence of thyrotoxicosis.

Hysterectomy was performed uneventfully four years later and the patient was last seen six years later, when she was found to be definitely improved. The sinus tachycardia had persisted, but was less marked than before, being only 90/minute; the grade III apical systolic murmur was still present, but no further abnormalities were discerned. Cardiac X-ray was normal and E.C.G. unchanged.

The cause of the large P-waves and the gallop sound remains obscure, but may have been due in some way to the persistent tachycardia. The aetiology of the pan-systolic murmur also is not clear, but it was felt here that the P.C.G. was of real value at least in excluding the presence of mitral stenosis as an aetiological factor.

Myocardial Infarction with Associated Mitral Stenosis

Case No. 150, a housewife aged 57, gave a clearcut history of angina pectoris for two months, with recent onset of pain at rest. She was admitted in congestive failure, with electrocardiographic evidence of recent posterior myocardial infarction. There was a previous history of rheumatic fever. The congestive failure improved with digitalisation and diuretics, but she died suddenly one month after admission.

On auscultation following admission a mid-diastolic murmur and what was thought to be a third heart sound were heard at the apex. These disappeared as the failure improved, and they were considered to be due to a relative mitral stenosis secondary to acute cardiac dilatation. A phonocardiogram, however, showed a definite mid-diastolic murmur even when this could no longer be heard and also the opening snap characteristic of mitral stenosis. The opening snap was situated approximately 0.08' after the beginning of the previous second sound and was not, therefore, a third heart sound. Unfortunately the time marker on these records taken with the Boulitte machine is not very accurate, but the figure 0.08' is well within the appropriate range for the opening snap.

The phonocardiographic tracings were taken to provide definite confirmation of the presence of organic rheumatic mitral stenosis, and at autopsy this was confirmed as well as the presence of extensive coronary artery disease, recent myocardial infarction, and terminal pulmonary emboli.

This was one of the cases in which the phonocardiograph influenced the clinical diagnosis and in fact was found to give more accurate information than was provided by clinical auscultation.

Rheumatic Valvular Disease Simulating Patent Ductus Arteriosus

Case No. 151, a small girl of 11 years, was asymptomatic but gave a previous history of rheumatic fever and was noted to have the following auscultatory signs. At the apex there was a normal first sound with a grade III systolic murmur and normal second sound, followed by a soft early diastolic murmur. At the aortic and pulmonary areas the systolic murmur was still heard, the early diastolic murmur was louder, and at the first left intercostal space a murmur suggestive of the continuous Gibson machinery murmur was heard, being made up of the systolic and early diastolic components. A third heart sound was present at the apex. There was no other clinical abnormality. X-ray screening showed slight left auricular enlargement and a dynamic wide aorta. Electrocardiogram was normal.

A phonocardiogram was made on two occasions, and the records show an apical systolic murmur starting with the first heart sound, maximum in early systole and finishing just before the second sound (Record A). A third heart sound is also seen in the tracing with lower frequency band (Record B). In the aortic and pulmonary areas the tracings show an early diastolic decrescendo murmur with the typical configuration of aortic incompetence (Record C). There is no visible mid-diastolic murmur or opening snap of mitral stenosis. Even at the first left space, where the murmur had been suggestive of a patent ductus on auscultation, the P.C.G. tracing does not have the diamond shape maximum in late systole and early diastole as described by Bonham-Carter and Walker (1955) (Records D and E).

On the basis of the full clinical evidence and the phonocardiograph tracings it was felt that, despite the patient's age and excellent general condition, the diagnosis was one of rheumatic aortic incompetence and mitral incompetence rather than patent ductus arteriosus.

This diagnosis has been maintained over the subsequent six years of followup. There has been one further attack of rheumatic fever, and when the patient was last seen the murmurs were as before, but slight cardiomegaly had developed with a mild degree of left ventricular hypertrophy, enlargement of the pulmonary conus and left auricle, and a wide dynamic aorta as before. Absolute confirmation of diagnosis is not yet possible.

SUMMARY AND CONCLUSIONS

A further twelve miscellaneous cases not fitting into any of the previously-mentioned categories had recordings made, either because of their particular interest or in an effort to assist in diagnosis.

1. Three cases of complete heart block were recorded, and both the auricular sound and the varying intensity of the first heart sound were demonstrated on the phonocardiograph. Giant "a" waves were also recorded on the phlebogram, and the mechanism of these phenomena together with their relation to the independent auricular and ventricular rhythms have been discussed.
2. In two cases, continuous vascular murmurs have been recorded. The first occurred in a lactating woman and was thought to be due to a small arterio-venous communication probably involving the internal mammary vessels. The second was a continuous venous hum heard over the epigastrium in an elderly woman with the Cruveilhier-Baumgarten syndrome and it was abolished by local digital pressure. This case is discussed in some detail and special examination of the vessels involved was made at autopsy. Neither of these conditions appears to have been recorded before.
3. Foetal heart sounds were recorded without difficulty in four cases, and it is concluded that such recordings may be of value in the clinical assessment of foetal distress.

4. In three cases where diagnosis was in doubt, phonocardiographic recordings were made. In the first case, mitral stenosis was excluded, partly on phonocardiographic evidence. In the second case, the presence of an unsuspected mitral stenosis was confirmed in a patient with a myocardial infarction. In the third case, the presence of a patent ductus arteriosus was suspected clinically, but phonocardiogram showed that this was unlikely, and a diagnosis of a rheumatic aortic and mitral lesion was ultimately decided upon.

In all these three cases the sound records were felt to be of some practical value in establishing a correct diagnosis. In the first and third, no absolute confirmation of diagnosis is yet available, but in the second, an autopsy subsequently showed the phonocardiographic diagnosis to be correct.

SUMMARY OF THE OBSERVATIONS PRESENTED
IN THE THESIS AND THE CONCLUSIONS DRAWN THEREFROM

1. The development of the art of auscultation and the graphic recording of heart sounds and murmurs during the last century has been described and the relevant literature reviewed. The way in which the sounds and murmurs are modified in passage from the heart to the human ear or to the phonocardiographic tracing is explained by a consideration of the physical characteristics of the human ear and the stethoscope or the various components of an electrical recording system. As a result of these physical characteristics and resultant modification of sounds and murmurs, the heart sounds, which are of low frequency, become greatly attenuated by the stethoscope and human ear and are not heard so easily as the higher frequency murmurs, which suffer less attenuation. Conversely, the characteristics of the phonocardiograph with its system of electrical as opposed to air conduction are subject to less modification of the lower, but more of the higher, frequencies than the ear. The low frequency sounds, therefore, are better recorded than the high frequency murmurs.

2. A full description has been given of the various phonocardiograph instruments, together with the recording apparatus and reference tracings used in this study. The actual techniques of heart sound recordings are outlined, and it has been found that the most satisfactory results have been obtained with a modified Elmquist Triplex electrocardiograph instrument used in conjunction with three amplifiers for sound recording.

The most useful reference tracings for timing events in the cardiac cycle were a limb lead electrocardiograph tracing, a second phonocardiograph tracing (in addition to the one being studied), and, particularly for events in diastole, a jugular pulse tracing. Some difficulty was experienced with the latter, and on numerous occasions when it would have been of value satisfactory tracings could not be made. Good recordings of the sounds and murmurs necessitate a comfortable relaxed patient, not distressed by dyspnoea, and a completely quiet environment free from extraneous sounds and also vibration. A reasonable series of recordings from each patient took on an average from $1\frac{1}{2}$ to 2 hours to complete, including the processing of the photographic record.

3. A limited number of recordings of normal heart sounds have been analysed and the literature reviewed.

The first and second heart sounds are due largely to valve closure, but muscle contraction also takes part, and the resultant sound is a complex mixture of high and low frequencies. Measurements of the duration of these sounds are of

no practical value as they will depend to a large extent on the sensitivity of the recording instrument. It has been demonstrated that variation in intensity and frequency of the phonocardiogram can alter the apparent duration of the sounds. Without calibration techniques an accurate comparison between one sound and another and between a sound in the same individual on different occasions is impossible.

A physiological third heart sound is frequently heard in the younger age group, but even more often recorded because of its low frequency. It occurs between 0.12 and 0.20 second after the beginning of the second sound.

The physiological fourth or auricular sound is never heard, but may frequently be recorded. It occurs at the time of auricular systole and its direct relationship to the P-wave of the electrocardiogram and the "a" wave of the jugular phlebogram has been demonstrated.

4. Splitting of either first or second sound is due to ventricular asynchrony and may occur in health or disease. Apart from paradoxical splitting of the second sound, which has been described in the literature, but which was not encountered in this series, there is no way of distinguishing splitting due to normal physiological causes from that which accompanies cardiovascular disease. On several occasions, however, phonocardiography has been of value in differentiating between splitting of the sounds and gallop rhythm.

In the case of the first sound the first component of the split always commences after the R-wave of the electrocardiogram whereas the auricular gallop sound occurs before the R-wave. When the second sound is split the second part of the split usually commences 0.02 to 0.06 second after the beginning of the first part.

5. Sixtyfive cases comprising a comprehensive series of audible gallop rhythms have been described and their aetiology and followup discussed, analysed and tabulated. A further series of cases were found to have gallop sounds visible on the phonocardiograph but not detectable on auscultation. This whole subject has also been reviewed.

The great importance of precise differentiation between split sounds, pre-systolic and early diastolic murmurs, the opening snap of mitral stenosis, and the various gallop sounds is emphasised, and the considerable value of the phonocardiograph in this context has been proved. It has been shown that auricular and rapid filling gallop occurs at the same time in the cardiac cycle as the physiological auricular and third sound. They are indistinguishable by ordinary routine phonocardiographic techniques, but may frequently differ in intensity. Their precise relationship to the deflection of the electrocardiogram and jugular phlebogram has been demonstrated.

It is considered that they have the same basic mechanism. The physiological third heart sound is probably due to sudden distension of the ventricular wall associated with rapid

ventricular filling, and the corresponding gallop sound which has the same time relationship to the second sound is always associated with either high venous pressure, myocardial disease, or both. The physiological fourth or auricular sound and the auricular gallop sound have been shown to be directly dependent upon auricular systole and are due to the sound made by contraction of the auricular muscle and possibly, also, to the resultant rush of blood from auricle to ventricle. It is always inaudible in health, though the auricular sound may be heard in complete or latent heart block and an auricular gallop when there is raised auricular pressure or a myocardial lesion. Summation gallop is due to the superimposition of auricular and rapid filling gallop upon each other. This fact has been clearly demonstrated during carotid pressure with consequent vagal stimulation and temporary bradycardia. As a result of this manoeuvre the two components of the gallop sound can be separated and each identified. On other occasions the two components have been shown to separate and the summation gallop disappear as the clinical state of the patient has improved and the pulse rate fall.

Analysis of the prognostic and diagnostic significance of the various forms of gallop rhythm showed that true summation gallop rhythm was of much more serious prognostic significance than either auricular or rapid filling gallop. It was always associated with frank congestive cardiac failure which might be predominantly either left or right-sided and was always accompanied by electrocardiographic abnormality, tachycardia and other signs of gross heart disease, hypertensive, ischaemic or cardiopulmonary.

Figures are not capable of statistical analysis, but it is suggested that auricular gallop is of less serious prognostic significance than rapid filling gallop. However, the presence of either of these two gallop sounds was probably of no greater help in arriving at an estimate of prognosis than a consideration of other aspects of the cardiac state, such as previous history and underlying cardiac lesion, congestive cardiac failure, blood pressure, or electrocardiographic assessment.

The presence of an audible auricular gallop has been shown to be compatible with a survival of from four to seven years or more.

A latent heart block was slightly more often associated with auricular gallop than with rapid filling gallop, but was by no means confined to the former group.

Auricular gallop was only associated with frank congestive cardiac failure in about half the cases, and this, when present, was commonly right-sided, but examples of left-sided failure also occurred. The underlying aetiology was either ischaemic or hypertensive heart disease - usually the latter.

Rapid filling gallop was only associated with frank congestive cardiac failure in about half of the cases, but when failure was present it was predominantly right-sided in type and usually due to myocardial infarction and ischaemic heart disease. Latent congestive failure was probably present in others, and several of the patients with this variety of gallop were suffering from carditis associated with such conditions as acute rheumatism or acute glomerulonephritis. In these latter cases the prognosis was, of course, relatively favourable.

No clearcut association between rapid filling gallop and right ventricular failure on the one hand and auricular gallop and left ventricular failure on the other was found in this series.

Rapid filling gallop associated with constrictive pericarditis has been demonstrated and its disappearance following pericardectomy observed. From studies of these recordings and after noting the relationship between the sound and the jugular pulse wave, it is clear that this sound has the same basic mechanism as the third heart and rapid filling gallop sounds. The extra sound in constrictive pericarditis is characteristically accentuated because of the sudden and slightly premature halting of the in-rushing blood into the ventricle by a rigid pericardial framework. For this reason also the sound may occur slightly earlier in diastole than the third heart sound in a normal heart or the rapid filling gallop sound in congestive failure due to other causes.

All the gallop rhythms encountered were predominantly left-sided. The classical right-sided gallop as described by Potain and heard best to the right of the sternum was never found in this series. However, a gallop sound maximum in the epigastrium was heard in five cases, three having cardio-pulmonary disease.

On one occasion it was thought that an auricular gallop originating entirely in the right side of the heart was demonstrated. The phonocardiogram always enabled gallop rhythms to be identified and classified even when there was difference of opinion over the auscultatory signs.

In the absence of an audible gallop rhythm, gallop sounds were detected on the phonocardiogram in a number of patients. These patients had a better prognosis than those with audible gallop.

Sometimes the gallop sound, whether auricular or rapid filling, became inaudible, but remained visible on the tracing as the clinical condition improved.

On the few occasions when a gallop sound was clearly heard on auscultation, but not recorded, there was always a technical reason, and recordings were of a poor quality.

6. It has not been found possible to diagnose bundle-branch block by auscultation, palpation, nor by phonocardiographic appearances alone.

The duration of both first and second heart sounds when measured on the phonocardiogram tends to be greater than normal, but this measurement is open to considerable error. Broad splitting of the sounds was not found in this series, but in four patients splitting was encountered which was in no way different from that seen in many other cases, including normal subjects.

The reduplicated apical impulse that has been described as characteristic of bundle-branch block has not been encountered in this series. Auricular and rapid filling gallop were frequently found in the presence of bundle-branch block but were not directly connected with the intraventricular conduction defect.

Both the gallop rhythm and the bundle-branch block occurred in these cases as a common result of the underlying ischaemic or hypertensive heart disease.

As with the other cases of gallop rhythm, rapid filling gallop was heard and recorded in association with cardiac failure, usually disappearing with clinical improvement. Auricular gallop was more frequently encountered in this group, sometimes, but not always, associated with latent heart block, and as far as the limits of this study are concerned, it seemed to be without particularly ominous prognostic significance.

7. The view that systolic clicks have an entirely different mechanism and significance to gallop rhythms and are not associated with heart disease has been confirmed.

8. Systolic murmurs may be organic, functional, or unexplained.

Some workers have claimed that functional murmurs not associated with heart disease may be distinguished from organic murmurs if a system of calibration and accurate measurement of frequencies is employed. This technique was not available for the studies presented in this thesis.

It has also been claimed that functional and organic systolic murmurs may be differentiated on the phonocardiograph by their position in early, mid or late systole, and in particular by their precise relationship to the S line of the electrocardiogram. These precise claims have not been borne out by this study. However, all of the pansystolic murmurs encountered here have been associated with organic disease.

In one case a loud pan-systolic murmur resulted from pressure on the heart mass from outside the pericardium without any valvular or other intracardiac lesion being present.

When a heart murmur of doubtful origin and significance is heard on auscultation a study of the phonocardiographic characteristics of the murmur and also of the other features of the tracing may be of value in establishing its true significance.

9. A series of recordings from patients with congenital heart disease has been made, but as the number of cases is small and as final confirmation of diagnosis in several of the cases is not yet available, it is unwise to draw conclusions.

In several cases, however, a study of the configuration of the tracings was of some assistance in making a diagnosis.

In two patients with patent ductus arteriosus the continuous Gibson murmur has been demonstrated and its absence after operative tying of the ductus confirmed. In each case, however, the murmur recurred after a period of months without any other clinical evidence of recanalisation. The true significance of this observation is uncertain as further followup is necessary.

10. The appearance of the characteristic diamond-shaped systolic murmurs of aortic or pulmonary stenosis and the de-crescendo or crescendo-decrescendo configuration of the early diastolic murmur of aortic incompetence have been described and illustrated.

Early diastolic murmurs were demonstrated on the phonocardiogram when inaudible to the human ear, and the tracing gave additional information in these and other cases of aortic valvular disease, several of which were confirmed at autopsy.

11. A detailed study has been made of the auscultatory signs and phonocardiographic appearances in mitral stenosis and incompetence.

The importance of the opening snap as a physical sign of mitral stenosis is established and its differentiation from the split second sound and rapid filling sound clearly illustrated. This differentiation was frequently aided by phonocardiography.

The time interval between the beginning of the second sound and opening snap for a large series has been found to be relatively constant; it usually falls between 0.06 and 0.10 second (extremes 0.04 and 0.14). In the presence of auricular fibrillation this measurement has been shown to vary directly with the previous cardiac cycle length.

The opening snap was usually loudest at the apex, the pulmonary area, or at a point on a line between the apex and the left sternal edge at the level of the fourth space. In only two cases where there was some confusion between an opening snap and a splitting of the second sound did the phonocardiogram not provide a clearcut answer.

A loud clear opening snap is characteristic of pure mitral stenosis. It was only very occasionally not heard or recorded in pure stenosis. It is not present in pure mitral incompetence or in the presence of gross valve calcification, but in a number of instances where a degree of both stenosis and incompetence were present a snap which was usually faint or atypical has been demonstrated.

In one case with predominant mitral incompetence, but also a significant degree of stenosis (both confirmed at operation), a snap was present and persisted after the valve had been dilated with the finger and partially split and a pericardial sling had been threaded across the valve in an attempt both to reduce the incompetence and to increase the mobility of the valve cusps.

A split second sound was often demonstrated in both mitral stenosis and incompetence, and a rapid filling gallop was encountered in some patients with incompetence or other associated heart lesion with or without cardiac failure. It was also recorded in the immediate post-operative period following valvotomy as a result either of the establishment of a significant degree of mitral incompetence or possibly from a transient traumatic myocarditis or pericarditis.

A mid-diastolic murmur was recorded on numerous occasions, but only once was it demonstrated by phonocardiogram when it was not heard on auscultation.

The association of the presystolic murmur with auricular systole has been confirmed.

The systolic murmur of mitral incompetence was always either pan or early systolic in timing, and its characteristics have been demonstrated and discussed. Its appearance is quite distinct from the diamond-shaped systolic murmur of aortic stenosis.

No accurate calibration was available and, therefore, no strict comparison could be made between recordings before and after the operation. No striking changes were, however, encountered despite the fact that in all cases of predominant stenosis a considerable degree of improvement resulted from the operation. In most, the

opening snap persisted after operation and the murmurs were not significantly changed. In some, the snap disappeared.

In most cases the time interval between the Q-wave of the E.C.G. and the beginning of the first sound diminished following valvotomy.

12. In a series of miscellaneous recordings a variety of individual problems and relatively unusual lesions were studied.

The various auscultatory findings in complete heart block were graphically demonstrated and their relationship to auricular and ventricular activity demonstrated.

Continuous vascular murmurs have been recorded for the first time. A case of the Cruveilhier-Baumgarten syndrome has been described together with a detailed account of the clinical, phonocardiographic and pathological findings.

The ease with which foetal heart sounds may be recorded has been shown and the possible value of this indicated.

CONCLUSION

In a number of instances phonocardiograph recordings have been shown to be of some practical value as an aid to correct diagnosis. Generally speaking, however, records have only served to confirm the auscultatory findings.

The heart sounds have proved much easier to record than the murmurs. Frequently, gallop sounds have been demonstrated on the tracing when not heard.

Phonocardiograms have been shown to be superior to auscultation for heart sound analysis, including the diagnosis of gallop rhythm with its differentiation from splitting of the sounds, the opening snap of mitral stenosis, and systolic clicks. This differentiation can be made by studying the appearance and noting the relationship of the sounds in question to the electrocardiogram and phlebogram reference tracing or by measuring the time interval between the extra sound and the second sound. Although there is a slight overlap, these latter measurements will differentiate in the vast majority of cases between the rapid filling gallop, splitting of the second sound, and the opening snap of mitral stenosis.

In the assessment of murmurs, however, the human ear has usually proved more sensitive than the phonocardiographic techniques used in this study, with the probable exception of early diastolic murmurs of aortic incompetence. Nevertheless, information on a variety of murmurs, such as the recognition of the characteristic appearance of the systolic murmur of aortic stenosis, may be obtained from sound recordings.

Apart from information about the origin and mechanism of heart sounds which has been obtained in the past from phonocardiography, it has also proved to be of permanent value in teaching the art of auscultation.

By a study of sound recordings the various heart sounds and murmurs can be well demonstrated, and a considerable increase in the accuracy of auscultation can be achieved.

Recordings, although not very difficult, are time-consuming and require a good deal of specialised experience and equipment. In this aspect the phonocardiograph contrasts with the electrocardiograph to a certain extent and will, therefore, never be as important a routine aid to cardiological diagnosis as the electrocardiogram.

Finally, it is concluded that the evidence presented in this thesis shows the phonocardiograph to be of great value for precise analysis of the heart sounds and murmurs. The additional information gained in this way is always of considerable academic or scientific interest. From the point of view of the clinician, however, it often serves only to provide confirmation of the auscultatory diagnosis.

The phonocardiograph, therefore, has a limited value in clinical cardiology as it only provides additional information of diagnostic or prognostic importance in a relatively small proportion of cases. In these few cases, however, vital information may be obtained that would not be available from any other diagnostic aid.

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APPENDIX

Analysis of readings from which Fig. 10 is derived (see p.293)

Number of readings (n) = 31

Cycle length, i.e., the measurement in seconds
between peak of two consecutive R waves of the E.C.G. = x

Interval between second heart sound and opening snap,
i.e., the measurement in seconds between the beginning
of the second heart sound and the beginning of the
opening snap of the phonocardiographic tracing = y

Standard deviation of x = s_x

Standard deviation of y = s_y

Coefficient of correlation (r) = $\frac{\text{Covariance } xy}{(s_x)(s_y)}$

$$r = \frac{\sum xy - n\bar{x}\bar{y}}{(n-1)} \times \frac{(n-1)}{\sqrt{(\sum x^2 - n\bar{x}^2)(\sum y^2 - n\bar{y}^2)}}$$

$$\sum x = 22.39$$

$$\sum y = 2.235$$

$$\bar{x} = 0.7223$$

$$\bar{y} = 0.072097$$

$$\sum x^2 = 18.6009$$

$$\sum y^2 = 0.1727$$

$$\sum xy = 1.7311$$

$$\underline{r = +0.6965}$$

Significance of difference between $r = 0.6965$ and $r = 0$

$$t = \frac{r \cdot \sqrt{n-2}}{\sqrt{1-r^2}} = 5.23$$

Degrees of freedom (n - 2) = 29

Entering "Student's" t-tables with 29 degrees of freedom
 $P < 0.01$

Therefore the correlation between the cycle length (x)
and the interval between the beginning of the second
heart sound and the opening snap of the phonocardiographic
tracing (y) is positive and highly significant.