# TRIPLE CARDIAC RHYTHM

A STUDY OF CARDIAC EXTRA SOUNDS BY PHONOCARDIOGRAPHY

BY

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" --- un triple bruit du coeur, constitué par l'addition aux deux temps normaux d'un troisième temps étranger à ceux-ci, qui n'est ni un souffle ni un frottement mais un bruit frappé, interposé entre les bruits normaux dans l'un ou l'autre silence."

Pierre-Carl Potain (1900)
Les bruits de galop.
Semaine Méd., vol. 20
p. 175

"The chief value of the recorded heart sound is the possibility of accurately timing its occurrence in relation to the events of the cardiac cycle."

Thomas Lewis (1915)
Lectures on the heart.
London: Shaw & Sons.
p. 54

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# PREFACE

This thesis is a record of the work which I have carried out during the last four years in cardiology. Much of it has already been published, including some investigations in which I was assisted by junior colleagues and, in one case, by the University Medical Officer: reprints of these articles are presented in Appendix E. I hope, with the consent of the Faculty of Medicine of Glasgow University, to publish in the near future my work on gallop rhythm in cardiac patients, which constitutes Part IV of the thesis.

All the phonocardiography has been carried out by myself and I have studied all the books and articles mentioned; at the expense of completeness of the bibliography I have rejected all references which I could not personally obtain and verify. An attempt has been made to strike a balance between literary and experimental research.

The immense clinical value of cardiac auscultation has been abundantly proved and it implies no disparagement of the method to say that this mode of investigation is inadequate to reveal the physical nature of the vibrations set up by the beating heart. An understanding of this had to await the development of graphic methods of record-

ing and analysing these vibrations. Phonocardiography is of value also in the precise localization of sound phenomena to particular phases of the cardiac cycle.

Ideally the phonocardiogram would show waves at times when the normal listener was conscious of sound phenomena and only at these times. This ideal can never be fully attained since 'normal' auditory mechanisms vary considerably and even the best phonocardiograph has a response corresponding only approximately to any selected auditory response. Some of the efforts made by previous and present workers to approach this ideal are described.

The phonocardiograph used in the current investigations has been built up over a period of years by myself, aided by other members of the staff of the Institute of Physiology of Glasgow University. The present model has evolved gradually from its predecessors and is still in process of evolution but, as described here, it performed very satisfactorily the sundry tasks it was set. During the course of these investigations minor alterations were made to the amplifiers and more efficient electrical screening was achieved in order to reduce mains interference and other sources of artefact. A new and very sensitive microphone was obtained for the study of pathological cases, and different time-marking motors were tried out at different stages of the work. Since the investigations

described in this thesis were completed I have made further modifications in the instrument; these are mentioned briefly in Chapter 4.

The first major investigation I attempted was of the incidence of the physiological third heart sound in healthy young adults. Articles in the literature had suggested that this sound would be audible in a majority of these individuals, which was contrary to my own experience and that of many of my clinical colleagues. The incidence on auscultation was studied by three observers, including myself, and compared with the incidence on phonocardiograms of the same subjects. Observations were made also on the fourth heart sound and on the systolic extra sound, which appeared on some phonocardiograms but were not heard in any of the subjects.

The study of triple cardiac rhythm in normal subjects led on to a study of it in patients with heart disease. Here the situation was more complicated and triple rhythm, where present, might be due to any one of a variety of extra sounds. In some cases too the heart sounds were obscured by murmurs. Since triple cardiac rhythm in heart disease is generally believed to be of bad prognostic significance, the opportunity was taken to seek

evidence for or against this belief by a follow-up of the cardiac patients investigated.

Experiments were performed both on human subjects and on dogs, with the object of elucidating the mechanism of production of cardiac extra sounds. In general, the experimental work on human subjects followed systematic observations on the normal human heart sounds, and experimental work on dogs followed and supplemented that on human subjects.

A variety of interesting murmurs was recorded in the patients with heart disease. These are not related to the main theme but, since they merit some comment, are described in Appendix A.

In view of their intrinsic interest all the phonocardiograms from patients with heart disease are displayed, but only representative examples of the many normal phonocardiograms and of the many experimental records are included.

Certain terminological criteria have been adopted for 'heart sounds', 'triple cardiac rhythm', and other phenomena. The current modern usage is employed, in so far as there is agreement on this, but an attempt is made in Chapter 2 to achieve precise definition.

The presentation of this thesis is not an indication that no further investigation of the heart sounds is contemplated, but rather that my investigations have now

reached a stage beyond which a general survey, such as is attempted here, would be unwieldy. I hope in the future to pursue the study of certain aspects of the subject, in particular the incidence and significance of triple cardiac rhythm in patients with arterial hypertension, and the recording of heart sounds with an intracardiac microphone in the dog.

# PART I

## HEART SOUNDS AND MURMURS

"Et quẽadmodum cernere licet, cum equus potat, & aquam deglutit, singulis gulae tractibus absorberi aquam, & in ventriculum demitti, qui motus sonitum facit & pulsum quendam & auscultatibus, & tãgentibus exhibet, ita dum istis cordis motibus fit portionis sanguinis è venis in arterias traductio, pulsum fieri, & exaudiri in pectore contingit."

William Harvey (1628)
Exercitatio anatomica de motú cordis et sanguinis in animalibus.
Frankfurt, Fitzeri.
P. 30.

## Chapter 1

## Historical review: auscultation of the heart

The history of the discovery and interpretation of the auscultatory phenomena produced by the beating heart is a lesson in what can be achieved by the acute observer with his unaided senses or with the minimum of apparatus. The literature in this field is so vast that no attempt at a comprehensive review of it can be undertaken here. A highly selective survey of certain outstanding discoveries is presented as a general introduction to the detailed study of triple cardiac rhythm, which forms the major part of this thesis, and to the notes on cardiac murmurs, which are appended.

It is strange that such an obvious vital phenomenon as the heart sounds should have passed without comment in medical literature until the seventeenth century. It is reasonable to assume that primitive man, laying his head on another's chest, must have been aware of the heart sounds; furthermore he may well have associated their cessation with death. Yet the first reference to these sounds in the literature is by Harvey (1628), who compared the sound produced by the heart pumping blood from veins to arteries with that produced in a horse's throat during swallowing.

Development of the stethoscope

After Harvey's time auscultation of the heart by placing the ear against the patient's chest wall was practised by some physicians, but it was not until the invention of the stethoscope (Laënnec, 1819) that the possibilities of auscultation began to be explored. From experiments with stethoscopes of various materials and various shapes Laënnec found that a hollow cylinder of wood was the best instrument for general use.

Piorry (1828, 1835) modified the Laënnec stethoscope by reducing the thickness of the stem and introducing the trumpet-shaped chest-piece.

Although the rigid monaural stethoscope was standard medical equipment for the greater part of the next century, alternative instruments gradually became popular. Comins (1829) devised a jointed ear-tube, which 'can be used in the highest ranks of society without offending fastidious delicacy'. He also suggested a pattern for a binaural instrument. To C.J.B. Williams is attributed the invention of the first binaural stethoscope in 1829 (C.T. Williams, 1907); this was a clumsy instrument with lead pipes as the flexible tubes and without ear-pieces.

The invention of the first stethoscope of modern type with chest-piece, rubber tubing and ear-pieces is attributed to G.P. Cammann in 1855 (D.M. Cammann, 1886).

The invention of a chest-piece with a rigid diaphragm is attributed to Bianchi in 1894, and the modern diaphragm type of chest-piece was patented by Bowles in 1901 (Rappaport & Sprague, 1941).

Lilienstein (1911) devised an electrical stethoscope with a carbon microphone, for application to the chest, and telephone ear-pieces. Jacobsohn (1923) used a microphone with a 3-valve amplifier and loud-speaker to make heart sounds audible to a number of observers; with this instrument the quality was so altered that sounds could not be distinguished from murmurs. The electrical stethoscope introduced by Frederick and Dodge (1924) included an electronic amplifier with electrical filters for selected frequencies of sound waves; it supplied several ear-pieces and gave much more satisfactory reception than Jacobsohn's instrument. A model described by Gamble and Replogle (1924) was designed to permit simultaneous auscultation by 75 individuals. Sell (1932) devised an instrument which gave a satisfactory rendering of the heart sounds by loud-speaker.

The acoustical properties of various types of stethoscope were investigated by Landes (1931), Johnston and Kline (1940), and Rappaport and Sprague (1941). For routine auscultation a binaural stethoscope with open bell

chest-piece was found satisfactory. For maximum efficiency the internal volume of the bell should be minimal. A large-diameter bell has the best response to low-frequency sounds; a Bowles diaphragm, on the other hand, attenuates low-frequency sounds without interfering with the transmission of higher frequencies.

Today the simple binaural stethoscope with open bell chest-piece still finds favour among experts as the instrument of choice for auscultation of the heart.

Observations on heart sounds and murmurs

Laënnec (1819) described two heart sounds corresponding to each pulse beat, a loud, dull sound attributable to ventricular systole and a clear, abrupt sound 'analogue au claquement de la soupape d'un soufflet', which he attributed to atrial systole. The clear, abrupt sound was what we now know as the second heart sound but Laënnec failed to appreciate its significance, although Harvey had already observed that the atria contract just before the ventricles. Laënnec also described certain abnormal sounds associated with heart disease. In patients with hypertrophy of the right ventricle he noted that the short sound (the second heart sound) might be double, triple or even quadruple. In some cases of gross enlargement of the heart, and in some cases where

a valve orifice was narrowed by disease, he noted that a sound resembling the sawing of wood was produced at each heart beat.

Heart sounds. Members of the British Association for the Advancement of Science (1835, 1836a,b, 1840) observed the exposed hearts of calves, asses, dogs and other domestic animals and confirmed Harvey's observations that the atria contract first, followed by the ventricles. They found Laënnec's rigid stethoscope inconvenient to apply to such an actively moving organ as the beating heart, so used a flexible ear-tube instead. They found that the first heart sound was loudest over the ventricles and occurred at the beginning of ventricular systole. It was attributed to the sudden increase in muscular tension of the ventricles when they begin to contract and, to a lesser extent, to forcible closure of the atrio-ventricular valves and to the rapid passage of blood over the irregular inner surface of the ventricles after the opening of the semilunar valves. The second heart sound was loudest at the aorta and pulmonary artery and occurred at the beginning of ventricular diastole. It was attributed to sudden closure of the semilunar valves by recoil of the columns of blood in aorta and pulmonary artery, impelled by the elastic tension of these vessels. An extra sound was sometimes heard, coinciding with atrial

systole, but this was difficult to detect because it merged with the following, much louder, ventricular sound. It is interesting to note that an explanation of the first and second heart sounds more than a century later (Luisada, 1948) does not differ in any important respect from that of these early investigators.

Some members of the British Association (1840) visited the London Zoo and listened to the heart sounds of eighteen species of wild animal, including the ostrich, leopard, seal, puma, elephant, antelope, lion and giraffe. All these animals were found to have two heart sounds, corresponding to the human sounds but with minor differences of quality and relative intensity. The members of the Association expressed their thanks to 'the distinguished veterinary surgeon of the establishment, without whose kind assistance it would have been out of our power even to have attempted anything in several instances'.

Charcelay (1838) noted triple rhythm in a patient with heart disease. Bouillaud (1841) stated that triple or quadruple rhythm of the heart is heard only in patients with organic valvular disease, and Skoda (1844) described doubling or tripling of the second heart sound in mitral stenosis. Drasche (1855) described splitting

of the first and second heart sounds, and Schäfer (1861), Potain (1866), and Marey (1881) observed that this could occur in people with normal hearts. Potain (1875) described 'bruit de galop', a term which he had learnt from his clinical teacher, Bouillaud; the extensive literature on gallop rhythm is reviewed in Chapter 7.

Rouchès (1888) described the 'claquement d'ouverture de la mitrale', characteristic of mitral stenosis.

Obrastzow (1905) described physiological and pathological reduplication of the first and second heart sounds. He mentioned also accessory heart sounds, including the third heart sound. The literature on the physiological third heart sound is reviewed in Chapter 5.

Benjamins (1914) passed the membrane-covered endpiece of a binaural stethoscope down the oesophagus of his subjects to behind the heart. In many subjects he could hear four heart sounds, one extra sound corresponding to atrial systole and the other (best heard with the endpiece of the stethoscope lying behind the ventricle) the third heart sound. With a similar device Taquini (1937) heard the atrial sound in every case, but never the third heart sound.

With the advent of phonocardiography the timing of heart sounds became more precise. Evans (1943) classified triple cardiac rhythm on the basis of the time of occurrence of the extra sound which, added to the normal first

and second heart sounds, produced triple rhythm. He found that the extra sound might be the third heart sound, occurring early in ventricular diastole, the fourth heart sound, late in ventricular diastole, or the systolic extra sound, usually late in systole just before the second heart sound.

Cardiac murmurs. Corrigan (1832) noted a systolic murmur at the base of the heart in 'permanent patency of the mouth of the aorta or inadequacy of the aortic valves'. Where the damage to the valves was severe the systolic murmur was accompanied by an early diastolic murmur. Hope (1839) described this diastolic murmur in more detail; he also recognised the systolic murmur of mitral incompetence. Fauvel (1843) defined the typical presystolic murmur of mitral stenosis, 'un bruit de râpe intense, précédant le premier bruit, finissant avec lui, ayant son maximum d'intensité à la pointe du coeur et à gauche'. Stokes (1854) described the systolic murmur of aortic stenosis, 'a most distinct musical tone' in the superficial arteries. Flint (1862) noted a presystolic murmur at the mitral area in some cases of aortic incompetence without mitral disease. Duroziez (1862) distinguished the 'grondement' of the mitral diastolic murmur from the 'souffle' of the aortic diastolic murmur. Roger (1879) noted the loud systolic murmur typical of interventricular

septal defect. Steell (1888) described the diastolic murmur of pulmonary regurgitation resulting from mitral stenosis.

Abbott's work on congenital heart disease (Abbott, 1927) placed on a firm foundation our knowledge of the cardiac murmurs in these conditions and of their significance. The clinical applications of the study were brought up to date by Taussig (1947).

Perhaps the most important recent advance in the practice of cardiac auscultation is Levine's classification of murmurs into six degrees of loudness (Levine, 1948). A first degree murmur is just audible, whereas a sixth degree one can be heard with the stethoscope held away from the chest wall: first and second degree murmurs may be physiological but louder ones indicate heart disease. Levine and his colleagues found good agreement between different observers as to the degree of loudness to be attributed to the murmurs in individual patients.

Recent work (aided by phonocardiography) includes comprehensive studies of mitral incompetence (Brigden & Leatham, 1953) and of mitral stenosis (Logan and Turner, 1953; Wood, 1954).

# Chapter 2

## Physical principles involved in auscultation and phonocardiography

### The physical nature of heart sounds and murmurs.

The study of the physical nature of the sound waves set up by the beating heart dates from the earliest days of phonocardiography. Einthoven and Geluk (1894) noted from analysis of their records that the heart sounds are 'noises' in the physical sense, the vibrations being so irregular that it is difficult to assign a definite frequency to them. These workers noted also that vibrations of the chest wall below audio-frequency tended to mask the 'sound' vibrations on the record unless some form of high-pass filter were introduced to eliminate the low-frequency components. In their apparatus the filter was a mechanical one; an open side-tube between chest-piece and microphone served to side-track the low-frequency waves. Weiss and Joachim (1908) pointed out that this mechanical filter failed to give true 'sound' records since some vibrations below audio-frequency were still recorded. They found that the 'rough' murmurs of mitral or aortic stenosis consisted of extremely irregular vibrations, clearly distinguishable from heart sounds, whereas the softer murmurs of valvular incompetence might

be distinguishable from heart sounds on a phonocardiogram only by the greater duration of the murmur.

The electrical stethoscope introduced by Frederick and Dodge (1924) could be adapted for phonocardiography by substituting a recording galvanometer for one of the output receivers. Analysis of the records (Cabot & Dodge, 1925) showed that the heart sounds consist of vibrations at frequencies of less than 150 cycles per second (c.p.s.), whereas murmurs fall into the frequency range, 120-660 c.p.s. Breath sounds, broadly speaking, are of higher pitch, with few components below 240 c.p.s. Cabot and Dodge found that practically all the sounds of interest in auscultation are at frequencies below 1000 c.p.s. Trendelenburg (1932) confirmed that murmurs are of higher pitch than heart sounds.

Williams and Dodge (1926) found that the energy of low-frequency praecordial vibrations (50-60 c.p.s.) is from 1000 to 10,000 times greater than that of the vibrations of a higher frequency range (190-200 c.p.s.). They noted also the considerable variation in intensity of heart sounds between different individuals; the loudest heart sounds are about 40 times more intense than the faintest ones.

As early as 1860 Heynsius concluded that the production of murmurs in the cardio-vascular system depends on the velocity of blood flow from a narrow to a wider part,

the murmur arising primarily in the circulating fluid. Bondi (1936) studied in models the vibrations produced by fluid flowing along tubes and explained cardiac murmurs on the basis of his findings. He concluded that murmurs are primarily due to turbulence of the blood stream, the heart and vessel walls being affected secondarily and, in turn, transmitting the vibrations to the surrounding tissues. He found a critical velocity of flow for each model, below which no murmurs were produced and a critical velocity, above which murmurs were always produced. Between these values murmurs were produced if the walls of the channel were rough or irregular. The nature of the fluid contained in the system was relatively unimportant.

Eckstein (1937) recorded sound vibrations from the exposed dog heart, from contracting frog skeletal muscle, and from active isolated portions of cat ventricular muscle. He found that the sounds produced depended on the vigour of the contraction and on the degree of shortening permitted. This supports the commonly held view that the first heart sound is due in part to muscular contraction.

Modern methods of phonocardiography (Mannheimer, 1940; Rappaport & Sprague, 1941) have permitted more accurate analysis of heart sound vibrations than was previously possible. In so far as it is possible to assign a 'frequency' to such an irregular series of

vibrations as those which constitute heart sounds it seems that the fundamental tone of the first heart sound is 30 - 40 c.p.s.; of the second sound, 40 - 50 c.p.s.; and of the third and fourth sounds, 25 - 30 c.p.s. The third and fourth heart sounds have very few overtones.

Cardiac murmurs as a rule have a fundamental frequency of more than 70 c.p.s. (often as much as 200 c.p.s.) and there are more overtones.

That the heart sounds are primarily produced in the heart and are not due to impact of the beating heart against the chest wall has been established by those investigators, including myself, who have listened to and recorded the heart sounds of animals both from the intact chest wall and thereafter from the surface of the exposed heart. The character of the sounds is similar in each case but, as would be expected, the sounds are louder at the heart than at the chest wall.

Physical principles involved in auscultation

The frequency of sound waves to which the human auditory mechanism is most sensitive is about 2,000 c.p.s. (Fletcher, 1948). As the frequency is reduced from this the sensitivity of the ear diminishes in a logarithmic manner. It follows from Fletcher's tables that for a third heart sound at a frequency of 25 c.p.s. to be audible it must be approximately $10^7$ times more intense than a faint aortic diastolic murmur at 500 c.p.s. Vibrations

below 20 c.p.s. are not normally audible.

For a sound to be audible the intensity, frequency, and duration must be adequate. The human ear can detect changes of frequency more readily than changes of intensity; under suitable conditions a change of frequency of 0.5% can be detected, whereas a change of intensity of about 25% is usually the least that can be appreciated (Holldack, 1949). At frequencies between 32 and 2,500 c.p.s. two cycles are sufficient for auditory perception (Orías & Braun-Menéndez, 1939).

Assuming a frequency within the 'range of audibility' and a duration of at least two cycles, a certain minimum pressure of sound is necessary to produce sensation. An arbitrary standard set as the 'threshold of audibility' is 0.0002 dynes per sq. cm. at a frequency of 500 c.p.s. (Counihan, Rappaport & Sprague, 1951).

In auscultation with a stethoscope the observer does not hear the cardiac vibrations as they exist at the surface of the heart. These are modified by three main factors (Rappaport & Sprague, 1942):

'a. The heart sounds are altered in their transmission from the source to the surface of the chest.

b. The heart sounds that reach the surface of the chest are additionally modified by the acoustic stethoscope and the type of chest piece employed.

c. The observer does not perceive the heart sound

vibrations as they are transmitted to the ear by the acoustic stethoscope.'

I have already indicated that transmission to the chest wall involves a reduction in intensity, without any obvious selective attenuation of particular frequencies detectable by auscultation or phonocardiography. I have mentioned also the selective response of the ear to particular frequency components of the heart sound vibrations. The second factor (modification by the acoustic stethoscope) merits more attention.

Any chest-piece applied to the chest wall functions as a diaphragm pick-up. In the case of an open bell the diaphragm is formed by the skin bounded by the lip of the bell and is damped by the underlying flesh. This skin diaphragm conforms to the usual physical laws of stretched membranes; the larger the diameter, the lower is its natural period. The natural period of even a wide chest-piece (5 cm. diam.) is at the upper limit of heart sound frequencies, so resonance effects are unlikely, but raising this natural frequency serves to reduce the low-frequency response. Hence a small chest-piece (2.5 cm. diam.) or increased pressure of application, or both, serves to reduce the loudness of the heart sounds as appreciated by the ear and may reveal a faint, high-pitched murmur by reducing masking. The rigid diaphragm of the Bowles chest-piece has the same effect. In any

type of chest-piece, for maximum efficiency the internal volume should be minimal.

Damping by conducting tubing of the usual calibre employed in the modern stethoscope (about 5 mm. bore) is another physical factor of some importance. As a result of physical tests Rappaport and Sprague (1941) concluded that the length of the tubing had no marked effect on the transmission of sound waves below 100 c.p.s.; on the other hand, from 100 to 1000 c.p.s., increasing the tubing length progressively attenuated the high-frequency components. It follows that, for the study of heart sounds, length of tubing is not a critical factor. In a later study Rappaport & Sprague (1951) found that tubing of 1/8 inch (3 mm.) calibre conducted sound waves more efficiently in the frequency-range 20 - 115 c.p.s. than did the 3/16 inch (5 mm.) tubing usually employed in stethoscopes.

Physical principles involved in phonocardiography

Orías and Braun-Menéndez (1939) outlined the essential features of a good phonocardiograph. It must be sensitive to the whole frequency range of heart sounds and murmurs and have a natural frequency sufficiently high not to distort the record of the vibrations; a natural frequency of at least 750 c.p.s. is required. The amplitude of the deflections of the recording apparatus should be directly proportional to the energy variations which

excite it, and the recording device should have minimal inertia and be fully damped.

Wiggers and Dean (1917a) demonstrated that the natural frequency of a Frank segment capsule is 150 - 250 c.p.s., and Lewis (1920) showed that the natural frequency of an Einthoven string galvanometer is 200 - 250 c.p.s. These devices are therefore unsuitable for phonocardiography. The modern, very sensitive, oil-damped mirror galvanometer is satisfactory but the ideal recording device is the cathode-ray oscillograph, which gives a deflection directly proportional to the voltage signal applied, has negligible inertia and is aperiodic.

For a phonocardiogram to be of value some reference tracing indicating the phases of the cardiac cycle must be recorded simultaneously on the record so that the deflections corresponding to sound phenomena may be related to the time in the cardiac cycle when they were produced. The various reference tracings which have been employed are described in Chapter 3.

Some indication of the duration of heart sounds and murmurs is also required. It is convenient to have a time markingon the record.

The beating heart produces vibrations of very low frequency at the front of the chest and Fredericq (1892)

demonstrated on dogs that these vibrations are similar to those recorded from inside the heart. These vibrations are of much greater amplitude than those of audio-frequency. The human ear does not respond to very low-frequency vibrations; in other words it acts as a high-pass filter. An undistorted phonocardiogram, however, shows only the low-frequency vibrations, which mask the heart sounds. In order to record heart sounds and murmurs some sort of high-pass filter must be introduced into the instrument to eliminate these low-frequency vibrations. Some of the methods adopted are described in Chapter 3.

A phonocardiograph, then, may be employed in different ways. It may be used to obtain an undistorted record of praecordial vibrations (apical cardiogram); it may be used to record the vibrations within selected frequency ranges; or an attempt may be made to introduce distortion similar to that introduced in auscultation by the human auditory mechanism.

Rappaport and Sprague (1942) recommended that three different types of phonocardiogram should be recorded from each subject: linear, stethoscopic and logarithmic. The linear phonocardiogram is an undistorted record of all praecordial vibrations picked up by a microphone applied to the chest (apical cardiogram). The stethoscopic record introduces the distortion normally produced

by a stethoscope. The logarithmic record introduces the distortion due in auscultation to the characteristics of the stethoscope and of the human ear. The logarithmic phonocardiogram is thus the only record in which the relative amplitude of the various deflections is comparable to the relative loudness of the sounds on auscultation.

Only the logarithmic phonocardiogram can be interpreted in the light of all the experience derived from auscultation. The other types of phonocardiogram may be found to show typical deflections in particular affections of the heart and may prove a valuable supplementary means of cardiac investigation, but the clinical background is still lacking (Leatham, 1952; Sloan, 1953).

There is little difference in the quality of heart sounds and murmurs as heard by direct auscultation with the ear against the chest and by 'mediate' auscultation with a stethoscope, apart from some attenuation of low-frequency vibrations by the stethoscope. In the study of heart sounds by phonocardiography I have found a correspondingly small difference in the appearance of linear and stethoscopic records, and I do not now believe that any advantage is to be gained by recording both. The linear record is usually preferable since it avoids the minor degree of distortion introduced by a stethoscope.

In experimental work on animals, however, when sounds are to be recorded from the exposed heart, it is more convenient to interpose a length of stethoscope tubing between end-piece and microphone than to attach a heavy microphone directly to the heart.

Ideally a phonocardiograph might be calibrated so that the amplitude of deflections on the record corresponds to a known energy of the sound waves picked up by the instrument. Calibration of the phonocardiograph has proved more difficult and less useful than the calibration of some other recording devices, e.g. the electrocardiograph: the advantages and limitations are discussed in Chapter 3.

Definitions

In the light of our present knowledge of the physical nature of heart sounds and murmurs and of the response of the human ear or the phonocardiograph to these phenomena it is convenient at this stage to attempt an arbitrary definition of some of the terms employed throughout this thesis.

By 'heart sounds' is meant sound vibrations originating in the heart, of audio-frequency and of sufficient duration and intensity to be audible or to be recorded on a logarithmic phonocardiogram. These vibrations have a fundamental frequency below 50 c.p.s. and each group of

vibrations corresponding to a heart sound normally lasts for not more than a certain period of time, viz.: 0.15 sec. in the case of the first heart sound, 0.10 sec. for the second sound, 0.06 sec. for the third sound (Luisada, Mendoza & Alimurung, 1949) and 0.10 sec. for the fourth sound (Caeiro & Orías, 1937). These arbitrary criteria serve to distinguish a sound from a murmur, the vibrations of which have usually a fundamental frequency of at least 70 c.p.s. and last for more than 0.15 sec. The distinction is a crude one but it will serve. When a murmur does not coincide in time with any heart sound, as is usually the case, it is easily identified.

By 'heart sound vibrations' is meant all the vibrations set up at the front of the chest by the beating heart, as recorded on a linear phonocardiogram. This corresponds to the palpable apex beat, but the phonocardiograph shows the vibrations in more detail than can be appreciated by palpation.

To distinguish deflections on a phonocardiogram due to extraneous sounds from those produced by the heart itself a further arbitrary criterion is adopted. A set of deflections is attributed to cardiac activity only if it occurs at the same phase of the cardiac cycle in at least three successive cycles.

The 'first heart sound' is that produced at the onset

of ventricular systole and the 'second heart sound' is that produced at the onset of ventricular diastole. Splitting of the first or second heart sounds must be distinguished from cardiac extra sounds. In general the two components of a split sound are of the same quality and loudness and are separated by only a short interval of time. More precise definition is possible by phonocardiography, as detailed in Chapter 4.

The 'third heart sound' is that produced shortly after the opening of the atrio-ventricular valves, at the time when the rate of filling of the ventricles is maximal. In view of its time of occurrence it is sometimes called the 'rapid-filling sound'. Following precedents in the literature I distinguish the physiological third heart sound of a healthy heart from the protodiastolic gallop sound of a diseased heart. Whether there is in fact any fundamental difference between these two phenomena is discussed in Chapter 7. The term, protodiastole, is taken in its usual sense as meaning approximately the first third of diastole rather than in the more restricted sense of Wiggers (1923, 1934), who limits it to the period between the beginning of ventricular relaxation and the closure of the semilunar valves.

The 'fourth heart sound' coincides in time with atrial systole but is not normally audible. Atrial systole normally contributes to the vibrations which

constitute the first heart sound: the criterion of a fourth heart sound is that the atrial systolic sound should be separated from the ventricular systolic sound by an appreciable interval. The appearance of a fourth heart sound in a patient with heart disease constitutes presystolic gallop rhythm.

In tachycardia the third and fourth heart sounds may be superimposed to produce a 'summation sound'. This can be diagnosed with certainty only if the two components can be separated by slowing the heart.

Another sound, which occasionally occurs, is the 'systolic extra sound' between the first and second heart sounds. The explanation of this phenomenon is still to seek, but it does not seem to be related in any way to heart disease.

An extra sound, which occurs in protodiastole and is believed to be diagnostic of mitral stenosis, is the 'opening snap of the mitral valve'. This has to be distinguished from the rapid-filling sound and from the second component of a split second sound, as described in Chapter 4.

The term 'cardiac extra sounds' is used to include the third, fourth, summation, and systolic extra sounds, the corresponding 'gallop' sounds of heart disease, and

the opening snap of mitral stenosis. By 'triple rhythm' is meant the addition of one of these extra sounds to the first and second heart sounds. The addition of more than one extra sound to the first and second heart sounds constitutes quadruple rhythm or some higher multiple.

Quadruple rhythm due to the simultaneous appearance of the third and fourth heart sounds has been called 'train wheel rhythm' (Luisada, 1952, 1953).

This arbitrary classification is obviously unsatisfactory: the same sound phenomenon is classified as a physiological third heart sound if there is no evidence of heart disease and as protodiastolic gallop rhythm if the patient is known to suffer from some cardiac affection. It is, however, current usage (Wood, 1950; White, 1951) and I adopt it here.

Murmurs are named in the conventional fashion according to the phase of ventricular systole or diastole in which they occur and, where appropriate, are further described as long or short, loud or faint, crescendo or diminuendo.

# PART II

## PHONOCARDIOGRAPHY

"La médicine d'aujourd'hui, a fortiori celle de demain, ne doivent pas, ne peuvent pas se passer de la phonocardiographie."

Camille Lian (1946)
Les applications pratiques de la phonocardiographie.
Algér. Méd., vol. 50
p. 496

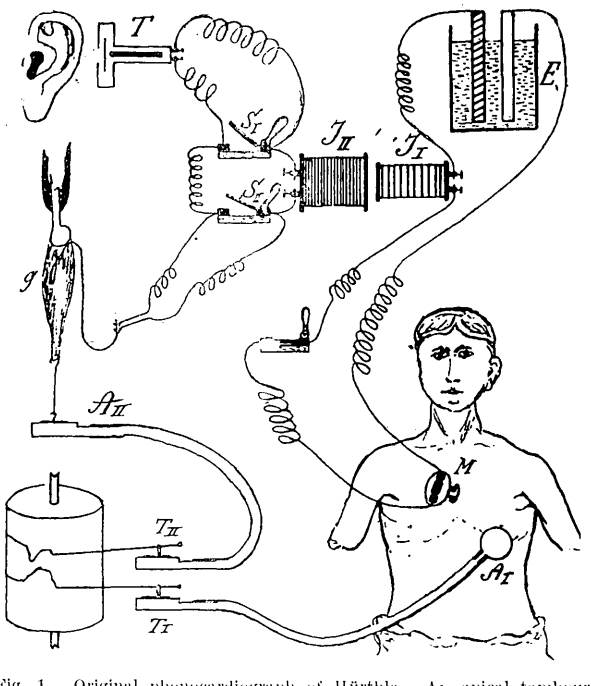


Fig. 1. Original phonocardiograph of Hürthle. $A_I$, apical tambour; $A_{II}$, tambour for frog muscle twitch; E, source of electric current; g, frog muscle-nerve preparation; $I_I$, $I_{II}$, induction coil; M, microphone; $S_I$, $S_{II}$, switches; T, telephone ear-piece; $T_I$, $T_{II}$, recording tambours. (Hürthle, (1893). *Dtsch. med. W'schr.* **19** : 80)

# Chapter 3

## Historical review: development of the phonocardiograph

### Early phonocardiography

Attempts to record the time of occurrence of the heart sounds in the cardiac cycle date from the second half of the nineteenth century. The apical cardiogram was recorded by means of a tambour applied to the praecordium and connected by rubber tubing to a recording tambour with a lever writing on a kymograph drum. The observer listened over the praecordium and made a mark on the corresponding point of the cardiogram every time he heard a heart sound.

Hürthle (1893) was the first to record objectively the time of occurrence of the heart sounds. The ingenuity of his apparatus cannot fail to arouse a physiologist's admiration and merits the inclusion here of his original diagram (Fig. 1). A carbon microphone was incorporated, along with a source of electrical current and a switch, in the primary circuit of an induction coil. By means of switches, either pin electrodes or a telephone earpiece could be brought into the secondary circuit. By applying the pin electrodes to the sciatic nerve of a frog muscle-nerve preparation a twitch was obtained corresponding to each heart sound: this was recorded on a

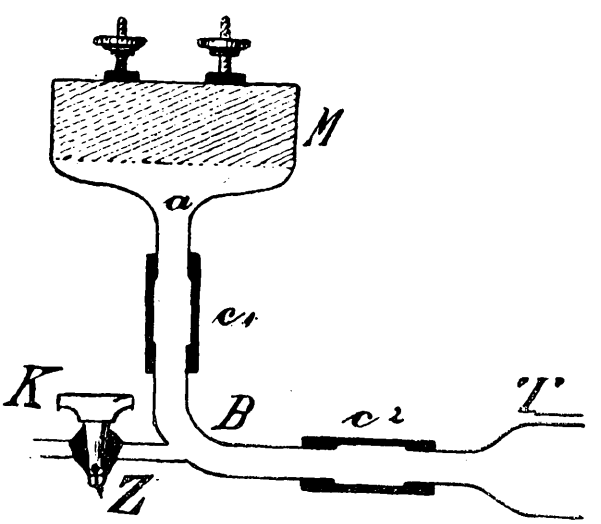


Fig. 2. Mechanical high-pass filter for phonocardiograph. a, funnel of microphone; B, metal tube; $c_1$, $c_2$, rubber tubing; K, tap; M, microphone; T, chest-piece; Z, side-tube. (Einthoven & Geluk (1894). *Pflüg. Arch. ges. Physiol.* **57**: 626)

kymograph drum. An apical cardiogram was recorded simultaneously as a reference tracing and a time trace was added. The delay incurred by the latent period of the frog preparation was found by tapping the praecordial tambour of the cardiogram with the bell of the microphone: the mean time-lag was of the order of 1/100 sec. and remained very constant in each preparation, provided the muscle was kept at a constant temperature.

Einthoven and Geluk (1894) replaced the frog preparation of Hürthle's original apparatus with a capillary electrometer. This instrument was sufficiently sensitive to respond to ordinary speech at a distance of a few metres, but inertia of the mercury column distorted the record of speech or heart sounds. Even in this primitive instrument the need for a high-pass filter was recognised (Fig. 2). A side-tap was introduced between chest-piece and microphone; when this tap was closed the cardiogram was recorded; when it was open the low-frequency vibrations were filtered off and sound waves were recorded. A reference tracing of the left carotid pulse was obtained by means of tambours.

The next year Hürthle (1895) modified his original apparatus. An ingenious tuning-fork microphone with a special resonance chamber was used to pick up the heart sounds and the vibrations were recorded by a tambour

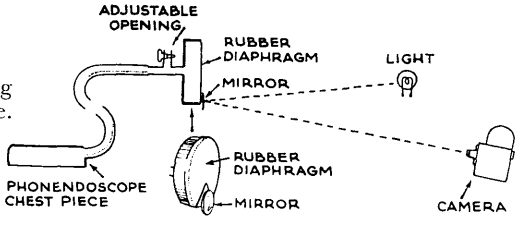


**Fig. 3.** Diagram of phonocardiograph employing Frank's segment capsule as the recording device.

carrying a small metal disc and activated by an electro-magnet.

Holowinski (1896) employed as a recording device for phonocardiography a telephone receiver with a slightly convex plate of glass attached to the diaphragm and separated by a very narrow air-space from a fixed, flat plate of glass. Spectral rings were formed, which expanded and contracted with each movement of the diaphragm, and these rings were photographed. Crehore and Meara (1911) described a similar device, which they called a 'micrograph'.

Frank (1904) connected a stethoscope chest-piece by rubber tubing to a tambour, bearing a thin rubber membrane. To the membrane was applied a small mirror, from which a beam of light could be reflected on to a moving strip of photographic material. By adjusting the distance of the camera from the mirror, a suitably magnified record of the movements of the membrane could be obtained. Later, as a result of detailed studies of the mechanics of vibrating membranes, Frank (1908) introduced the 'segment capsule', which has since played such an important part in physiological investigations. The principle of the 'direct' phonocardiograph, employing Frank's segment capsule as the recording device is illustrated in Fig. 3.

Marbe (1907) and Roos (1908) employed the 'manometric

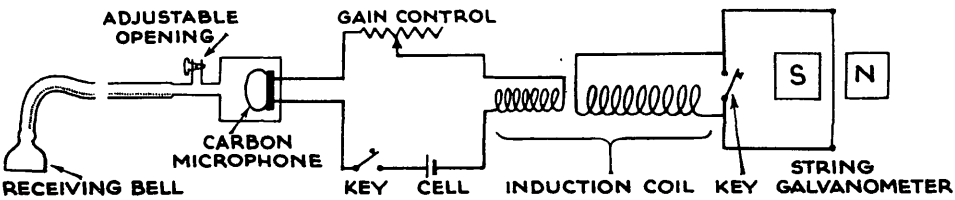


**Fig. 4.** Diagram of phonocardiograph employing Einthoven's string galvanometer as the recording device.

flame' as a recording device for phonocardiography. The chest-piece was a small capsule, bearing a thin rubber membrane, which was firmly applied to the praecordium. A steady stream of acetylene was passed through the capsule to a small burner, which played a smoky flame on a moving strip of paper. Vibrations of the praecordium caused elliptical figures to appear on the soot tracing.

Einthoven (1907a) replaced the capillary electrometer of his original apparatus with a string galvanometer, which gave more satisfactory records of sound waves (Fig. 4). Einthoven failed at this time to record a simultaneous reference tracing, as he had done with his previous, more cumbrous, apparatus.

Weiss and Joachim (1908) conducted sound waves from the praecordium to a soap-film membrane, on which rested a very fine lever of silvered glass. Movements of this lever were recorded photographically.

Gerhartz (1911) connected a phonendoscope to a collodion membrane bearing a fine plate of iron between the poles of a magnet and carrying a mirror for light-beam recording. The magnet damped the oscillations of the membrane.

Hess (1920) applied to the chest wall a flat metal cup, connected by rubber tubing to a tambour with a membrane of very thin rubber. Attached to the centre of

the membrane was a fine thread, which was thrown into vibration by vibrations of the membrane. Optical means were employed to throw an enlarged image of the shadow of the thread on to a moving strip of photographic film.

Reference tracings

I have already indicated in Chapter 2 the fundamental importance of a simultaneously recorded reference tracing for the interpretation of the phonocardiogram (P.C.G.). It is convenient to digress at this stage on a consideration of the various types of reference tracing which have been used and of their respective merits and demerits.

Apical cardiogram. The earliest reference tracing was the apical cardiogram (Hürthle, 1893). As the 'linear phonocardiogram' this has been recorded by a number of investigators, including Yoshioka (1932b), Smith, Edwards and Kountz (1941), Smith, Gilson and Kountz (1941), Rappaport and Sprague (1942), and myself. Luisada (1945, 1948) considers it the best reference tracing for phonocardiography. The apical cardiogram is easy to record and, by modern methods, can be obtained from the same chest-piece as that used for simultaneous recording of the stethoscopic or logarithmic P.C.G. (Aixala, 1947)., It indicates activity of the ventricle and can therefore be related directly to the sound components produced in that chamber.

In practice, however, it is less easy to interpret than the electrocardiogram (E.C.G.) or the venous pulse, and it does not give so much information as the latter about the different phases of ventricular diastole.

Arterial sphygmogram. The arterial pulse has been used as a reference tracing, notably by Einthoven and Geluk (1894) and Kahn (1910), who recorded it from the carotid artery; by Lian and Racine (1933a), who used the brachial artery; and by Lockhart (1938), who recorded the radial sphygmogram. The arterial sphygmogram has no advantage over the apical cardiogram and has the added disadvantage of occurring later, after a period of delay which varies in different individuals.

Electrocardiogram. Bull (1911) employed the E.C.G. as a reference tracing for phonocardiography. He recorded the E.C.G. by means of an Einthoven string galvonometer and the P.C.G. by means of a Weiss phonocardiograph, the glass lever of which interrupted the light-beam of the string galvanometer. Kahn (1911) took composite records of P.C.G. and E.C.G., recorded simultaneously by the same string, but these were difficult to interpret. Eyster (1911) recorded P.C.G. and venous pulse, followed by E.C.G. and venous pulse, so that the sounds could be related indirectly to the waves of the E.C.G. Fahr (1912) related the heart sounds to the E.C.G., using a single string

galvanometer. He took three records of each case; P.C.G. alone, E.C.G. alone, and P.C.G. and E.C.G. superimposed.

A great technical advance in the use of the E.C.G. as a reference tracing was the employment by Lewis (1913a,b) and Battaerd (1915) of two Einthoven string galvanometers, one to record the P.C.G. and the other the E.C.G. simultaneously on the same record.

The E.C.G. is easy to record and to calibrate. It has found favour as a reference tracing with many workers, including Lewis (1920), Asher (1932), Duchosal (1932), Routier and Van Heerswynghels (1935), Bramwell (1935a), Calò (1938), Mannheimer (1940), Levine (1945), Evans (1948), Evans and Lian (1948), Luisada and Roitman (1948), Cowen and Parnum (1949), Ernst (1952), and Caniggia (1953). Unfortunately it csnnot serve to distinguish the various sounds which may occur in early diastole. Furthermore the time relationship between the electrical changes indicated by the E.C.G. and the mechanical events of the cardiac cycle is inconstant (Ohm, 1913; Lewis, 1915; Luisada, 1941; Donovan, 1948; Levine & Harvey, 1949; Ernst, 1952): this inconstancy, however, is usually of minor degree and for practical purposes can be ignored.

Phlebogram. Phlebography (Friedreich, 1866) is a

much older method of investigation than is phonocardiography. Gottwalt (1881) described the waves of the jugular phlebogram, and these have since been studied in detail by many workers, notably by Mackenzie (1893, 1902), Edens (1910), Wiggers (1928), and Groedel (1945). Among those who have used the phlebogram as a reference tracing for phonocardiography are Eyster (1911), Weber and Wirth (1912), Ohm (1913), Bridgman (1915), Wiggers and Dean (1917b), Cossio (1938), Orías and Braun-Menéndez (1939), and Donovan (1948).

The venous pulse is easily recorded either by direct or by electronic methods. The pioneers were, of course, limited to direct methods of phlebography; the venous pulse waves were picked up by a funnel, shallow cup, or pelotte, applied to the neck and were transmitted along rubber tubing to a recording tambour (Marey, 1878) or, in the later models, to a segment-capsule (Frank, 1908). Fenning (1943) recorded the jugular phlebogram by means of a condensor microphone applied to the neck and connected to a string galvanometer. When electronic amplifiers were introduced it became possible to construct an extremely sensitive phlebograph, with an electro-magnetic or crystal microphone pick-up and a sensitive galvanometer or a cathode-ray tube for recording (Miller and White, 1941; Vannotti & Schmid, 1943).

The time taken in transmission of the pulse wave from the right atrium to the jugular veins is very nearly constant and, when the phlebogram is used as an empirical reference tracing, can be ignored. The phlebogram clearly indicates the times of atrial systole, ventricular systole, opening of the atrio-ventricular valves, and the 'rapid-filling' phase of early diastole. A theoretical objection is that the jugular phlebogram indicates activity of the right side of the heart, whereas the heart sounds are mainly due to activity of the left side. In cases of gross asynchrony of the ventricles, as for example in bundle-branch block, this can be a serious disadvantage (Wolferth & Margolies, 1935), but such gross asynchrony is rare.

Pneumogram. A reference tracing, which has achieved very limited popularity, is the 'pneumogram' or 'cardio-pneumogram' (Braun-Menéndez & Vedoya, 1937; Luisada, 1942). This is a record of pressure changes in the mouth or nostril with the glottis open or, in the case of experimental animals, of pressure changes recorded from a tracheal cannula. The waves of the pneumogram correspond to the main events of the cardiac cycle, viz.: atrial systole, the isometric and isotonic phases of ventricular systole, and the rapid-filling and isostatic phases of ventricular diastole. In the original human

technique the record was taken from the closed mouth with the nostrils occluded. The more modern method, employing electronic filters, permits recording during normal respiration, the slow respiratory waves being filtered off. The method requires very careful attention to technical detail, and interpretation of the record may be difficult.

Fontanellar sphygmogram. In phonocardiography of infants Segura (1936) recorded the fontanellar pulse as a reference tracing.

Multiple reference tracings. It is an advantage to be able to record simultaneously with the P.C.G. more than one reference tracing. With the modern multichannel phonocardiographs described below, this presents no difficulty and even such early investigators as Van Zwaluwenberg and Agnew (1912), using segment capsules, managed to record simultaneously along with the heart sounds any three of the following: right atrial pressure curve, oesophageal pressure curve, carotid sphygmogram, apical cardiogram, jugular phlebogram. Usually a pulse tracing and one E.C.G. are considered sufficient (Duchosal, 1929; Battro, Braun-Menéndez & Orías, 1936; Jochim, Gaddas & Marquis, 1937; Leatham, 1949), but some investigators have recorded along with the heart sounds more than two reference tracings. Barry (1925) and Oliveira

(1949) recorded simultaneously the P.C.G., E.C.G., phlebogram, and arteriogram. Lagerlöf and Werkö (1949) obtained simultaneous records of P.C.G., E.C.G., intracardiac pressure, arterial blood pressure, and respiratory movements.

Modern phonocardiography

Two of the older methods of phonocardiography have remained popular, the 'direct' method and recording by means of a string galvanometer. The direct method, using a Frank segment capsule, has been used with minor modifications by Pereira (1935), Orías and Braun-Menéndez (1939), and Boyer, Eckstein and Wiggers (1940). A double-string Einthoven galvanometer has been employed by Macleod, Wilson and Barker (1932), Evans (1948), and Cowen and Parnum (1949).

Other methods have developed along with modern advances in electronic engineering. Williams (1921) adapted an electro-magnetic microphone for phonocardiography: this was a great advance on the carbon or condensor microphones previously used. Frederick and Dodge (1924), Gamble and Replogle (1924) and Hoskin (1925) incorporated such a microphone in the electrical stethoscopes, with 3-stage valve amplification, which they employed for teaching, and Yoshioka (1932a) used one in his phonocardiograph.

Sacks and Marquis (1935) used a piezo-electric crystal microphone with a portable amplifier and loud-speaker for clinical teaching. They pointed out the advantage of this kind of microphone: it is compact, requires no special transformers or batteries, is free from background noise, is sensitive to vibrations of the frequency of heart sounds and murmurs, and has a natural frequency well above those encountered in cardiac auscultation. Miller and White (1941) used this type of microphone for phonocardiography.

The introduction of electronic amplifiers made possible the use of a variety of sensitive recording devices. Among these were the argon glow tube (Asher, 1932), which responded by variations in light intensity to signals of audio-frequency; the record was photographed along with the E.C.G. on the camera of a string galvanometer. Another device (Hollingsworth, Sorensen and Driessche, 1937) was a thin metal rod attached to the coil of a moving-coil loud-speaker and placed to cast its shadow beside that of the string of an Einthoven electrocardiograph. Lutembacher (1926), Leoper, Lemaire, Lefèvre and Bussard (1933), and Smith, Essex and Baldes (1950) recorded heart sounds on the sound track of cinematograph film.

Mirror galvanometer. Perhaps the most popular recording device today is the mirror galvanometer. Among those who have used this type of phonocardiograph are: Schütz (1929), Weber (1931), Kerr, Sampson, Lagen and Kellogg (1932), Wolferth and Margolies (1933), Clerc, Zadoc-Kahn and Tavecchi (1934), Bierring, Bone and Lockhart (1935), Schwarzschild and Feltenstein (1935), Taquini, Massell and Walsh (1940), Mannheimer (1940, 1941, 1946), Rappaport and Sprague (1941), Frost (1944), Carlgren (1946) and Ljunggren (1949).

A great advantage of mirror galvanometers for phonocardiography is the ease with which a number of simultaneous records can be obtained of heart sounds or of reference tracings. Mannheimer (1940, 1941) employed five channels to record simultaneously by mirror galvanometers four different frequency bands of heart sound vibrations and an E.C.G. Frost (1944) and Ljunggren (1949) found four channels sufficient. Leatham (1949) combined a two-stringed Einthoven phonocardiograph with two mirror galvanometers to obtain four simultaneous records. He now prefers an instrument with four mirror galvanometers (Leatham, 1953, personal communication). Counihan, Rappaport and Sprague (1951) used a standard commercial 3-channel phonocardiograph with d'Arsonval

mirror galvanometers, the 'Sanborn Tribeam'.

Cathode-ray oscillograph. Koczkás (1932, 1934), Boone (1940), and Kountz, Gilson and Smith (1940) devised phonocardiographs incorporating a cathode-ray tube. Donovan (1943) pointed out the advantages of the cathode-ray oscillograph (C.R.O.) over other devices used for recording heart sounds. He also described a phonocardiograph incorporating a double-beam C.R.O. (Donovan, 1944, 1945). Dawson and Morgan-Jones (1944) used an electronic switch to split the beam of a single-beam C.R.O. so that P.C.G. and E.C.G. could be recorded simultaneously. Campbell, Sloan and Andrew (1952) devised a phonocardiograph with a double-beam C.R.O. as the recording device; this instrument is described in Chapter 4. Dunn and Rahm (1952) described a multi-channel phonocardiograph with four single-beam cathode-ray tubes, and Besterman and Harrison (1953) described a model with six such tubes; a simultaneous photographic record of four tracings was obtained in each case.

Pen recorder. Pen recorders have been used for phonocardiography by Rushmer, Bark, and Ellis (1952), Rushmer, Sparkman, Polley, Bryan, Bruce, Welch, and Bridges (1952), and by Gunn and Wood (1953). This device admittedly cannot record audio-frequency vibrations but it can give a general impression of the intensity of sound phenomena

at different stages in the cardiac cycle.

Recording from a distance. Parker, Breakell, and Christopherson (1953) described a small, portable, radio transmitter, which could be worn by an actively-moving subject and used to transmit his heart sounds to a receiving set some distance away. The advantages of such an instrument for the study of heart sounds during various forms of active exercise are apparent.

Calibration of the phonocardiograph.

Calibration of the phonocardiograph presents practical difficulties. Firstly it is inconvenient to set the instrument to a constant sensitivity, as is usually done with the electrocardiograph, since the intensity of heart sounds and murmurs is so variable. Secondly it is technically difficult to measure the energy of sound waves applied to the ear or to the corresponding pick-up of a phonocardiograph.

The intensity of the heart sounds at the front of the chest is of very limited value as an indication of the state of the heart, since this intensity depends not only on the intensity of the sounds produced in the heart but also on the position of the heart, the state of the lungs, and the composition and thickness of the chest wall. Calibration of the record is, however, desirable in view of discrepancies which occur between auscultatory and phonocardiographic findings. When a patient has a loud

first and second heart sound and a faint diastolic murmur, the gain of the instrument must be so reduced to record the whole extent of the sound vibrations that those corresponding to the murmur are no longer visible. At the other extreme, when a patient's heart sounds are faint, an inaudible murmur may be recorded. This is an inevitable feature of phonocardiography but calibration of the record serves to indicate the degree of gain employed and is of help in correlating auditory with graphic findings.

Mannheimer (1940), by means of an ingenious 'pistonphone', succeeded in calibrating his phonocardiograph so that the output in millivolts from the microphone could be interpreted in terms of sound pressure on the microphone diaphragm, measured in dynes per square centimetre.

Wells, Rappaport and Sprague (1949) standardized their P.C.G.s by recording at the end of each tracing a signal corresponding to a pure tone of 500 c.p.s. at 80 decibels above a standard threshold of audibility (0.0002 dynes per sq. cm.). Counihan, Rappaport and Sprague (1951) recorded a signal at 90 decibels above this threshold.

Dunn and Rahm (1952, 1953a,b) used a pistonphone for primary calibration. Their secondary calibrator was a headphone receiver driven by an audio-oscillator

at 100 cycles per second and checked against the original calibration.

Besterman and Harrison (1953) used a 50-cycle signal to produce a measurable deflection on the P.C.G. They give no details of the intensity of signal required.

Summary

Early attempts at phonocardiography succeeded in indicating the time of occurrence of the heart sounds but failed to record the audible sound waves.

In order to determine the time of occurrence of heart sounds or murmurs in the cardiac cycle a simultaneously recorded reference tracing is essential. Those which have been used include: apical cardiogram, arterial sphygmogram, electrocardiogram, jugular phlebogram, pneumogram, and fontanellar sphygmogram. The most satisfactory is the jugular phlebogram, since it indicates clearly the timing of diastolic events. With modern multi-channel phonocardiographs more than one reference tracing can be recorded.

Two early methods of phonocardiography are still favoured by some investigators today. These are the 'direct' method, using a segment capsule as the recording device, and the string galvanometer.

With the development of modern electronic technique,

more sensitive and accurate instruments have become available. A piezo-electric crystal microphone is usually employed to pick up praecordial vibrations; appropriate amplifiers then augment the signals, which are displayed by mirror galvanometers or on a cathode-ray oscilloscope.

Calibration of a phonocardiograph presents practical difficulties but is of value when the record is to be compared with auditory findings. One method of calibration is to record at the end of the P.C.G. a standard electrical signal corresponding to a sound of known frequency and intensity applied to the microphone.

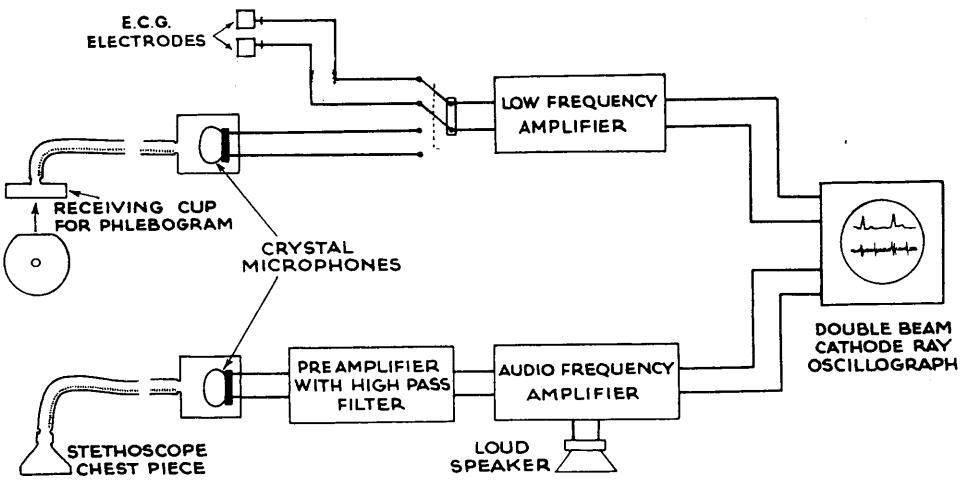


**Fig. 5.** Diagram of phonocardiograph employing a double-beam cathode-ray oscillograph as the recording device.

# Chapter 4

## A modern electronic phonocardiograph

The essential characteristics of a good phonocardiograph are considered in Chapter 2 and the attempts which have been made to achieve a satisfactory instrument are described in Chapter 3. The ideal recording device is the cathode-ray tube, since it has no inertia which could distort the vibrations to be recorded and the amplitude of the deflections is directly proportional to the movements which excite them.

The instrument described below was constructed for the investigations described in this thesis but it is adaptable and has proved a good general-purpose phonocardiograph. Minor alterations and improvements have been made during the four years that it has been in use, but the fundamental design as described by Sloan (1951) and Campbell, Sloan and Andrew (1952) has not been altered.

Fig. 5 is a block diagram to show the principle of this phonocardiograph and Plate 1 (Volume 2) shows the actual instrument. There are four main units: pre-amplifier, audio-frequency amplifier, low-frequency amplifier, and double-beam cathode-ray oscillograph (C.R.O.) display unit.

Sound channel

Chest-piece. Heart sound vibrations are picked up by a piezo-electric crystal microphone applied to the praecordium. For linear phonocardiography the conical chest-piece of the microphone is held against the praecordium by a rubber band round the chest or by suction. For stethoscopic phonocardiography a similar chest-piece is connected by a length of rubber tubing to the crystal microphone, which is suspended by rubber tubing from a bracket or held in a clamp.

The chest-piece of the microphone itself is of metal or, in the latest model, of bakelite. The separate chest-piece, of exactly the same dimensions, is of perspex. The shape in each case is a simple truncated cone, 5 mm. thick; the maximum internal diameter is 47 mm.; the minimum, 10 mm.; and the depth, 14 mm. This broad, shallow type of chest-piece was shown by Rappaport and Sprague (1941, 1942) to cause minimal attenuation of low-frequency phenomena, such as the cardiac extra sounds, which are the principal subject of these investigations. The stethoscope tubing, where used, is 46 cm. long; 5 mm. bore; with a wall 3.5 mm. thick. Wiggers and Dean (1917b) found neglible delay in the transmission of sound waves in a 70 cm. length of rubber tubing of this calibre.

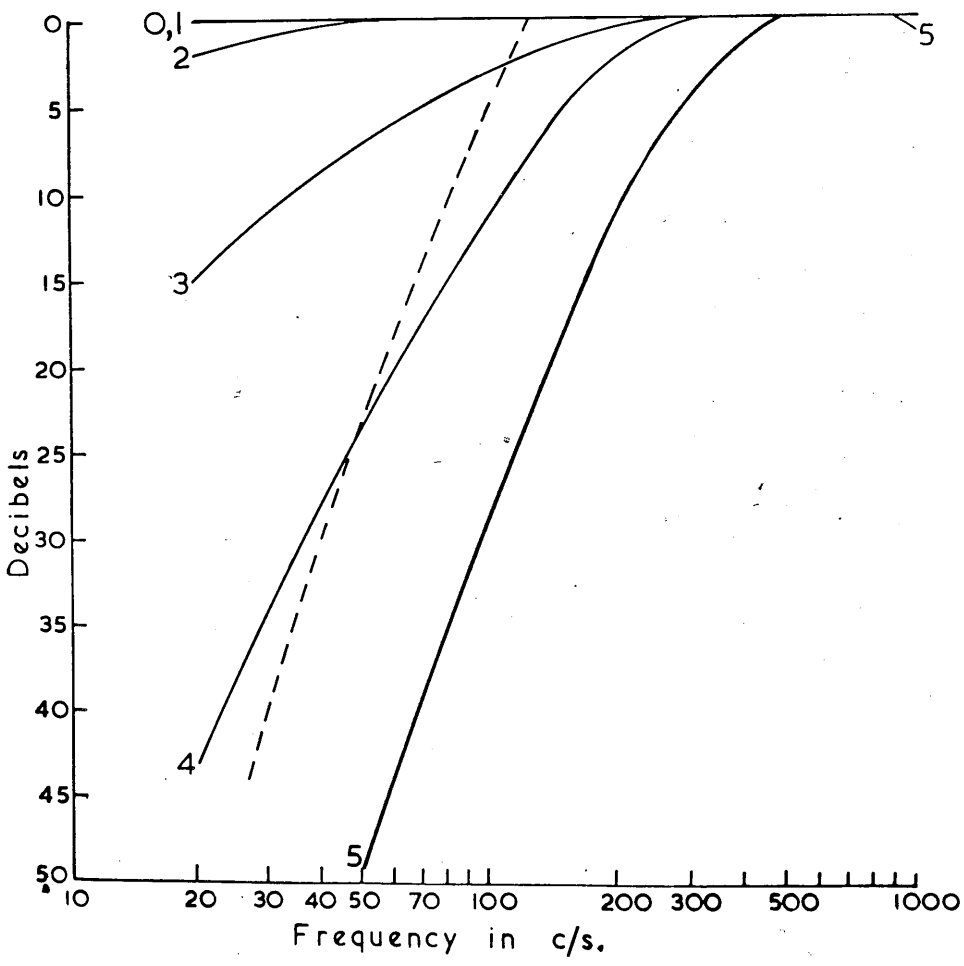


FIG. 6.—Frequency response curves of preamplifier compared with normal auditory response. The figures 0, 1, 2, 3, 4, and 5 refer to positions of filter switch on preamplifier. Interrupted line represents normal auditory response (Fletcher, 1948).

Preamplifier. Plate 2 (Volume 2) is the circuit diagram of the preamplifier. It is based on that described by Cowen and Parnum (1949) but modified for use with a C.R.O. instead of with the string galvanometer used by these authors.

Briefly, the output from the crystal microphone is applied to the grid of the first valve, which is connected as a conventional stage of amplification. A switch in the anode circuit gives a step control of gain, and a 2-megohm potentiometer gives continuous gain control. The second stage (half of the double-triode valve) is a cathode follower with low output impedance.

The filter network closely follows that of Cowen and Parnum (1949). At settings 1, 2, and 3, single-stage resistance-capacity filters are introduced, with time-constants of 0.0068, 0.0013, and 0.00034 sec. respectively. At settings 4 and 5, a two-stage and a three-stage filter respectively are introduced, the time-constant of each additional section being 0.00034 sec.

Routinely the filters were set at 0 (linear gain) for linear or stethoscopic phonocardiography and at 4 for logarithmic phonocardiography. Fig. 6 shows the frequency-response curve of the preamplifier at the different filter settings: position 4 gives the response

most nearly approaching that of the human ear.

The filter network is followed by a further stage of amplification and another cathode follower (second half of the double-triode).

The power supply is conventional with a voltage stabilizer in the H.T. supply of the first three stages. The supply to the final output valve is taken from another point in the smoothing circuit to decouple this stage and prevent oscillation.

Audio-frequency amplifier. The circuit of this component (Plate 3) is only slightly modified from that described by Williamson (1951). The original negative-feedback stabilization has been abolished to obtain greater gain and the output transformer has been wound with a 600-ohm winding in addition to the low-impedance winding for the speaker, enabling both cathode-ray tube and speaker to be supplied by the same amplifier.

The loud-speaker is a permanent-magnet moving-coil model with a 25 cm. cone.

The audio-frequency amplifier output is led to the lower beam of the double-beam C.R.O.

Reference channel

Either of two reference tracings is recorded simultaneously with the P.C.G. The venous pulse is picked up by a flat cup held against the side of the neck over the jugular bulb and leading, by a short length of rubber

tubing, to a piezo-electric crystal microphone. For the E.C.G., I employ routinely Lead II. By means of a double-pole double-throw switch either the leads from the microphone or the E.C.G. leads are connected to the input of the low-frequency amplifier.

Low-frequency amplifier. The low-frequency amplifier originally incorporated in the phonocardiograph was one designed many years ago for an entirely different investigation in the Institute of Physiology. It proved satisfactory for recording either the E.C.G. or the phlebogram but, since the investigations described here were completed, has been replaced by one of more modern design.

The circuit of the original low-frequency amplifier (Plate 4) consists of a stage of push-pull amplification followed by a Toennies compressor stage and two stages of single-sided amplification. The amplifier is resistance-capacity coupled throughout, with time-constants of 4 sec. A stepped gain control is provided between the second and third stages and a high-frequency filter can be switched in to reduce interference, including mains hum. In order to minimize this interference the first two stages are battery operated, both for low-tension and high-tension supplies. The two later stages are mains driven.

The output from the low-frequency amplifier is led to the upper beam of the cathode-ray tube of the display

unit.

Display unit

The recording device is a double-beam cathode-ray tube (Cossor, type 89J). The circuits of the C.R.O. unit and time base are shown in Plates 5 and 6. The display unit is of conventional pattern with voltage adjustments for brilliance and focus. As an aid to correct photographic exposure a 'brilliance meter' is incorporated on the panel. The time-base incorporates a thyratron valve and is adjustable over a wide range of frequency.

For visual observation the beams are made to traverse slowly; for recording, this is stopped and the recording paper is run past horizontally. Records are made with a camera with motor drive attachment (Cossor, type 1428) at a film speed of 1.2 inches (30 mm.) per second on recording paper (Ilford B.P.1).

An ophthalmoscope lamp, mounted on the camera just in front of the oscillograph screen, is activated by an electric motor-driven commutator to give timing marks on the record at 1/10 sec. or, in the later records, at 1/5 sec. intervals.

Phlebography in experimental animals

For the investigation of extra cardiac sounds in the dog (Chapter 11) the jugular phlebogram was required as

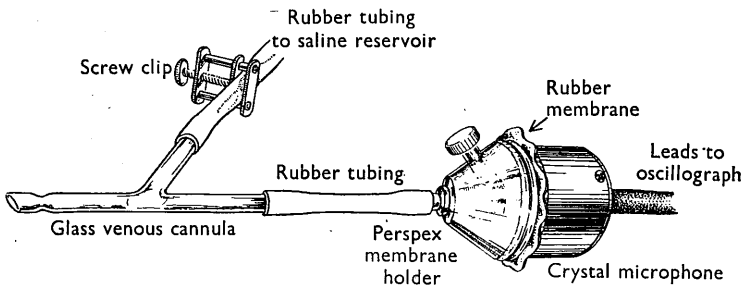


Fig. 7. Apparatus for recording the venous pulse.

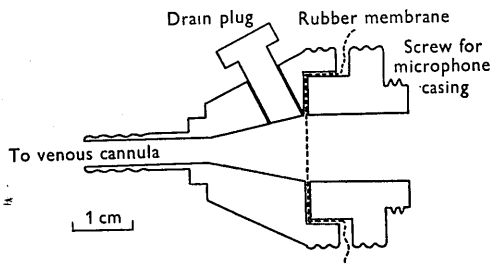


Fig. 8. Sagittal section of Perspex membrane-holder.

a reference tracing. In order to obtain an accurate record of the venous pulse without recording other pulsations from the neck it was decided to cannulate the external jugular vein and connect the cannula by some device to the piezo-electric crystal microphone already in use for phlebography. Since a venous cannula obstructs the normal flow of blood along the vein and allows its walls to come together, obliterating the venous pulse, a flow of saline into the vein is necessary to keep the vessel patent.

The device used for recording the jugular phlebogram of the dog is shown in Figs. 7 and 8 (Sloan & Wishart, 1953b).

A cannula introduced into the vein is connected to a chamber separated by a rubber membrane from the crystal microphone. A side-tube from the cannula leads to a reservoir of mammalian Ringer solution, from which intravenous infusion is maintained during the experiment. The membrane is of rubber dam, held in position by friction in a perspex membrane-holder, which permits direct observation of the fluid inside. A drainage plug allows flushing out of blood or air bubbles from the chamber. The microphone casing is attached to the membrane-holder by a screw thread.

The apparatus was tested to eliminate the possibility of distortion of the record by resonance effects at the frequency of the waves of the venous pulse. It was set up as for normal use but the cannula, instead of being introduced into a vein, was connected by rubber tubing to a piston device. This was activated by an electric motor to produce a regular to-and-fro surge of fluid in the system, of comparable amplitude to that produced in the dog's jugular vein by the beating heart.

The records obtained are shown in Plates 7 & 8 (Volume 2). Since no resonance effects were observed at frequencies of from 4 to 40 c.p.s. it was assumed that no distortion of the wave-form due to resonance would be produced at the frequency of the venous pulse waves, each of which lasts, on an average about 0.1 sec.

Technique of human phonocardiography

For routine phonocardiography of human subjects the individual is stripped to the waist and recumbent for about five minutes before the record is taken. During this time details of health are noted and auscultation of the heart performed. The site of the apex beat is identified and marked, as is any other site from which it is proposed to record heart sounds. Electrodes are applied to the subject's right wrist and to both ankles; those

from right wrist and left ankle are connected to the electrocardiograph leads, that from the right ankle to earth. This precaution, and sometimes also additional earthing of the screened leads from the electrodes, is necessary to reduce mains interference, one of the major complications of using such a sensitive instrument as this. The gain of the low-frequency amplifier is adjusted so that the waves of the E.C.G. are clearly shown but do not encroach on the lower half of the screen. Since the gain may be varied from one subject to the next, the amplitude of the deflections of the E.C.G. does not correspond to any standard voltage applied to the leads.

The stethoscope chest-piece or the microphone is then applied to the praecordium in the region of the cardiac apex and is held in position by a rubber band round the chest or, in some of the later investigations, by suction. The preamplifier is set at filter position 4 for logarithmic phonocardiography and the gain is adjusted to give a suitable amplitude of deflection on the record as detailed below. The loud-speaker is then switched on to confirm the auscultatory findings and reveal any extraneous sounds: a howl from the speaker at this stage, due to positive feed-back, indicates that the chest-piece is not air-tight against the chest and requires adjustment.

When any necessary adjustments have been made the speaker is switched off.

A serial number is marked on the recording paper. The subject is then asked to take a normal breath in and out and thereafter to hold the breath out for a few seconds while the record is taken. This is a usual practice to avoid confusion of the record by breath sounds (Leatham, 1949; Wells, Rappaport & Sprague, 1949), although it is not difficult to recognise and distinguish these on the record should they occur.

To record the jugular phlebogram as the reference tracing, the low-frequency amplifier is switched from the electrocardiograph leads to the phlebograph microphone and an assistant holds the receiving cup of the phlebograph against the subject's neck over the right jugular bulb. When the cup is in air-tight contact with the skin of the neck the waves of the phlebogram appear on the C.R.O. screen and the gain of the low-frequency amplifier is adjusted to show them clearly in the upper half of the screen. With the subject once more holding his breath in expiration a further P.C.G. is recorded.

Ideally, for a logarithmic P.C.G., the microphone should be separated from the chest by a length of stethoscope tubing since the preamplifier filter introduces only the distortion normally due to the human auditory mechanism,

without that due to the stethoscope which is usually employed. In practice I have found the distortion produced by a short length of rubber tubing is so small that I now prefer to apply the microphone, with its attached chest-piece, directly to the chest. This has been the practice of other investigators for some time (Rappaport & Sprague, 1941; Luisada, 1948; Leatham, 1949) and is technically much more convenient.

To record stethoscopic or linear P.C.G.s the procedure is essentially similar to that described above. The stethoscope chest-piece (stethoscopic P.C.G.) or the microphone (linear P.C.G.) is held against the praecordium by a rubber band or by suction, and the preamplifier is used without filters (filter position 0). For these records the gain of the preamplifier and the amplifier must be greatly reduced from that required for the logarithmic P.C.G.

The standard set of records I have obtained on each human subject or experimental animal has depended on the nature of the investigation in each case and is noted in connection with the several investigations.

Interpretation of the P.C.G.

For routine phonocardiography the E.C.G. serves to distinguish systolic from diastolic events but for the timing of events in diastole, as in the investigation of

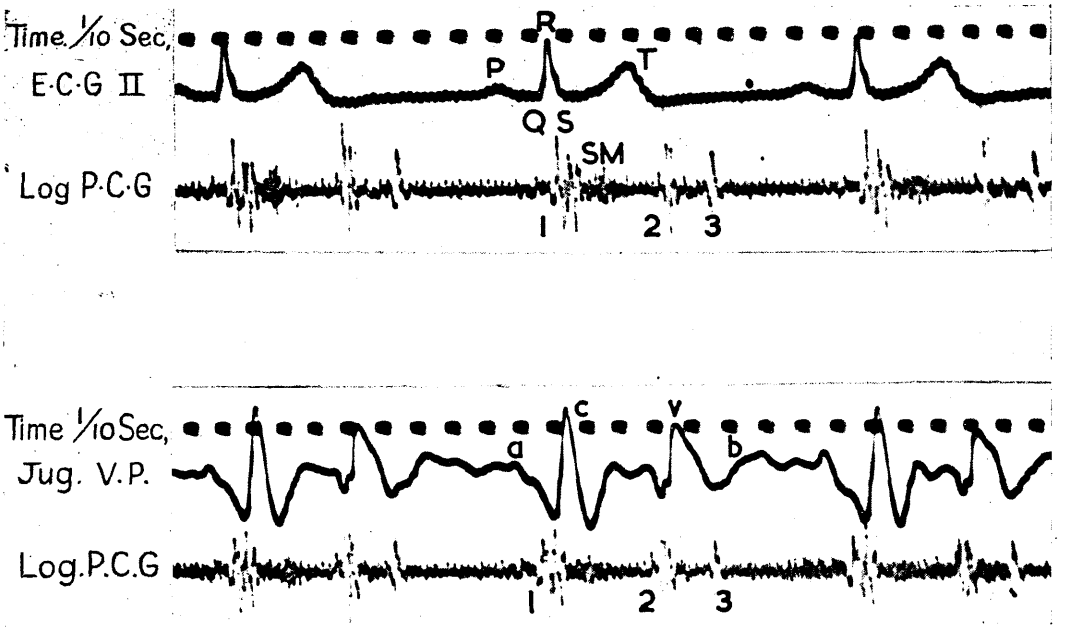


FIG. 9.—Logarithmic phonocardiograms from the mitral area of a healthy subject. Reference tracings: electrocardiogram, lead II (E.C.G. II) and jugular venous pulse (Jug. V.P.). 1, 2, 3, heart sounds; SM, systolic murmer.

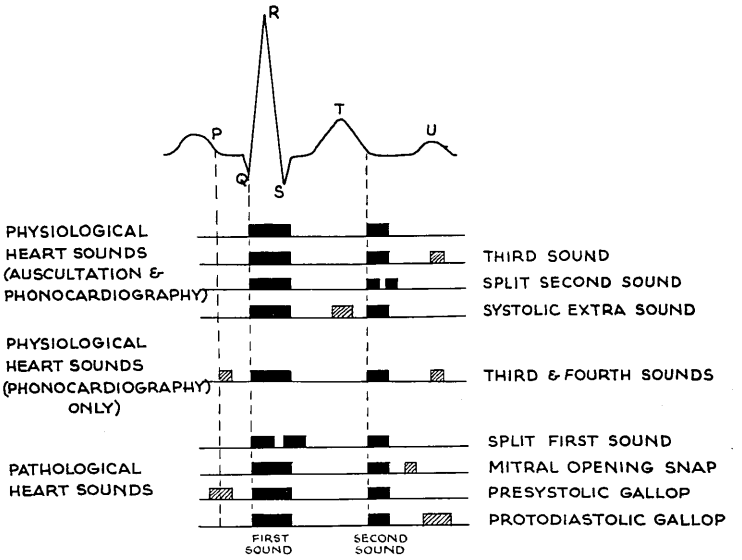


Fig. 10. RELATIONSHIP OF HEART SOUNDS TO WAVES OF ELECTROCARDIOGRAM.

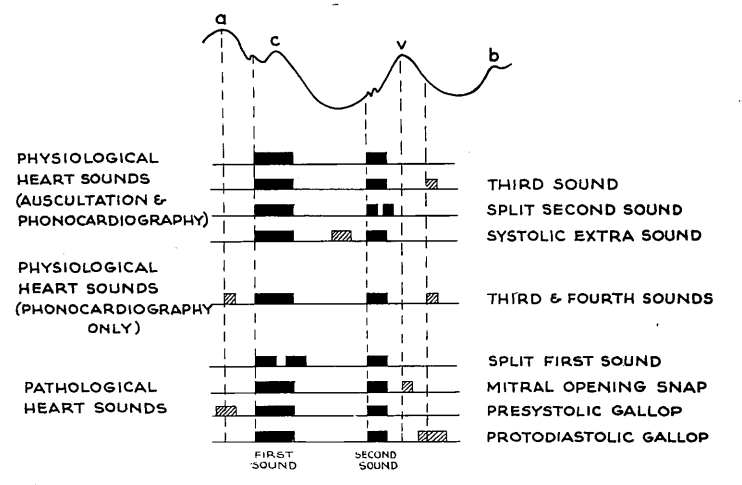


Fig. 11. RELATIONSHIP OF HEART SOUNDS TO WAVES OF JUGULAR PHLEBOGRAM, (After Cossio & Orías, 1935).

triple cardiac rhythm, the jugular phlebogram is required. I usually record both on consecutive records as in Fig. 9; the E.C.G. aids in identification of the waves of the venous pulse in the few cases where this proves difficult. The time-relationship of the heart sounds to the waves of the E.C.G. and of the phlebogram is shown in Figs. 10 and 11.

Using the E.C.G. as the reference tracing the Q wave immediately precedes ventricular contraction and hence the first heart sound. The second heart sound coincides with the end of the T wave or immediately follows it. The E.C.G. gives no exact timing of cardiac events in early diastole but, in late diastole, a distinct sound between the P and Q waves is identified as a fourth heart sound or as the corresponding presystolic gallop sound.

In the case of the jugular phlebogram the c wave coincides with the first heart sound. A notch on the upstroke of the v wave, clearly seen in records by a sensitive phlebograph, corresponds to the second heart sound. The third heart sound and protodiastolic gallop sound are distinguished from other protodiastolic sounds by their relation to the v wave. The second component of a split second sound occurs before the crest of the v wave, and the opening snap of the mitral valve starts at the summit of this wave, whereas a third heart sound

or protodiastolic gallop sound occurs on the downstroke or at the foot of the v wave. The fourth heart sound and presystolic gallop sound correspond to the summit of the a wave of the venous pulse.

Recent modifications

Since the completion of the investigations recorded in this thesis two main modifications have been introduced into the phonocardiograph as it is here described.

The first change is the replacement of the original low-frequency amplifier (Plate 4) by a model of more modern design, which is entirely battery-driven. This has abolished the mains interference, which sometimes appeared on the reference tracings.

The second modification is the addition to the pre-amplifier of a calibration device. With the assistance of Mr. J.R. Greer of the Department of Natural Philosophy I have incorporated in the amplifier a circuit which provides a standard 50-cycle a.c. signal of the same order of voltage as that normally induced by the crystal microphone during the recording of heart sounds. The microphone has been calibrated at the National Physical Laboratory so that the voltage output can be interpreted in terms of sound pressure on the microphone diaphragm. By turning a switch on the preamplifier panel the P.C.G. is interrupted and a 2.4 mV 50-cycle signal, corresponding to sound pressure on the microphone of 6 dynes per sq. cm.,

is fed into the sound channel at the same gain and filter setting and is recorded at the end of the P.C.G. This corresponds to a sound approximately 25 decibels above the threshold of audibility at this frequency. Plate 79 (Volume 2) shows calibrated P.C.G.s of a normal subject.

Further experiment is required to find whether this arbitrarily chosen signal is the most useful for general purposes and whether the present design of the calibrator is the most efficient for providing the signal.

Summary

A two-channel electronic phonocardiograph is described. The sound channel comprises a crystal microphone, preamplifier with high-pass filters, and audio-frequency amplifier leading to one beam of a double-beam cathode-ray oscillograph. The audio-frequency amplifier also supplies a loud-speaker. The reference channel comprises electrocardiograph electrodes and a crystal microphone, either of which may be connected to a low-frequency amplifier leading to the other beam of the oscillograph.

For phonocardiography the time-base of the cathode-ray oscillograph is switched off and the beams are photographed on a horizontally moving strip of recording paper. A lamp, mounted on the camera, flashes at 1/10 sec. or 1/5 sec. intervals to provide a time marking.

The jugular phlebogram in experimental animals is recorded from the lumen of the vein by means of a special

device, incorporating a crystal microphone, attached by rubber tubing to a cannula in the animal's external jugular vein.

Logarithmic, stethoscopic, or linear P.C.G.s may be recorded. Routinely two logarithmic P.C.G.s are obtained, one with the E.C.G. (Lead II) and the other with the jugular phlebogram as the reference tracing. Where appropriate, stethoscopic or linear P.C.G.s are recorded as well.

Cardiac extra sounds in diastole are distinguished by their temporal relationship to the waves of the jugular phlebogram.

The original amplifier in the reference channel has recently been replaced by one of improved design. A calibration device has been fitted to the sound channel and is at present under trial.

# PART III

## CARDIAC EXTRA SOUNDS IN HEALTHY HUMAN SUBJECTS

"La science ne s'établissant que par voie de comparaison, la connaissance de l'état pathologique ou anormal ne saurait être obtenue sans la conaissance de l'état normal."

Claude Bernard (1865)
Introduction à l'étude de
la médecine expérimentale.
Paris, Baillière et Fils.
p. 7

## Chapter 5

## Physiological cardiac extra sounds

The distinction between splitting of the first or second sound and the addition to these sounds of a cardiac extra sound has already been drawn in Chapters 2 and 4. As indicated in Chapter 2 the term, 'triple cardiac rhythm', is limited by current convention to the addition of an extra sound to the cardiac cycle and excludes the rhythm due to splitting of the first or second heart sound. The times in the cardiac cycle when physiological extra sounds most commonly occur are protodiastole (3rd heart sound), presystole (4th heart sound), and late systole (systolic extra sound).

In considering the normal occurrence of cardiac extra sounds it is important to distinguish phonocardiographic from auscultatory findings. The third heart sound is a normal finding by either technique but the fourth heart sound is not normally audible, although it may be seen on the P.C.G. of healthy individuals. The systolic extra sound is a rare phenomenon but, when present, may be audible as well as detectable by phonocardiography.

As mentioned in Chapter 2, the heart sounds are of low frequency (fundamental tone, 25-50 c.p.s.). Since the human ear is relatively insensitive to vibrations of

this frequency-range it follows that cardiac extra sounds are more readily detected by phonocardiography than by auscultation. Even in logarithmic phonocardiography, in which the frequency-response curve of the ear is simulated by the phonocardiograph, a higher incidence of extra sounds is usually found than by auscultation. In cases where triple cardiac rhythm is detected by auscultation it is usually possible to determine by phonocardiography the exact time of occurrence of the extra sound in the cardiac cycle, but an audible sound does not always appear on the logarithmic P.C.G.; this apparent anomaly has been noted in connection with calibration of the phonocardiograph (Chapter 3).

Since the third heart sound is by far the commonest physiological extra sound on auscultation it has been the main subject of this study. The occurrence of the fourth heart sound is of less interest, since it is inaudible in healthy subjects, and I have not detected a systolic extra sound by auscultation in any of my normal subjects.

Third heart sound

The physiological third heart sound was first described by Obrastzow (1905), although its discovery is generally attributed to Gibson (1907) and Hirschfelder (1907). In the light of Gibson's description, Einthoven

(1907b) re-examined some of his original P.C.G.s and detected a third heart sound on several of them: he expressed the opinion that it was probably a normal phenomenon but often masked on the P.C.G. by irregularity of the base-line.

The physiological third heart sound is best heard at the apex-beat (Gibson, 1907) or slightly medial to the apex-beat (Gallavardin, 1912) and is loudest at the end of expiration (Thayer, 1908). It is most obvious with the patient recumbent, especially lying on the left side (Thayer, 1908; Routier & van Bogaert, 1934b; Orías & Braun-Menéndez, 1939). In some healthy young subjects it is audible only for a few minutes after the subject lies down (Bridgman, 1915; Melik-Gülnasarian, 1932a; Routier & van Bogaert, 1934b).

Thayer (1909) and Lewis (1913a) detected the third heart sound relatively more frequently at slower heart rates. It is accentuated by pressure on the abdomen (Steinberg, 1926; Gubergritz, 1926) or by raising the limbs (Thayer, 1908, 1910; Orías & Braun-Menéndez, 1939). Increased vagal activity favours its occurrence (Luisada, 1948). Fazekas (1942a,b) heard the third heart sound more frequently in young people who took an active part in sport than in their less active coevals.

Bramwell (1943) found that a 'duplicated second heart sound', apparently identical with the physiological

Table 1. Incidence of the third heart sound in children without heart disease: auscultation

| Date | Author | No. of subjects | Age-group (years) | Incidence of 3rd sound (per cent) |
|---|---|---|---|---|
| 1909 | Thayer | 39 | 0 - 9 | 58.9 |
| " | " | 90 | 10 - 19 | 84.4 |
| 1915 | Bridgman | 16 | 12 - 15 | 81.2 |
| 1926 | Steinberg | 100 | 4 - 14 | 48.0 |
| 1938 | McKee | 105 | 5 - 17 | 5.7 |
| 1940 | Mannheimer | 135 | 0 - 14 | 0.7 |
| 1942 | Fazekas | 25 | 14 - 20 | 48.0 |
| 1946 | Carlgren | 150 | 3 - 17 | 12.0 |

Table 2. Incidence of the third heart sound in adults without heart disease: auscultation

| Date | Author | No. of subjects | Age-group (years) | Incidence of 3rd sound (per cent) |
|---|---|---|---|---|
| 1909 | Thayer | 55 | 20 - 29 | 50.9 |
| " | " | 26 | 30 - 39 | 42.3 |
| " | " | 14 | 40 - 49 | 14.0 |
| " | " | 7 | 50 - 59 | 0.0 |
| 1926 | Gubergritz | 600 | x | 93.7 |
| 1944 | Frost | 169 | x | 0.6 |
| 1947 | O'Meara | 745 | 17 - 20 | 53.6 |
| " | " | 255 | 21 - 30 | 34.9 |

x = not mentioned

third heart sound, was commoner in men under 20 years of age than in older individuals.

Auscultation. It is strange that a phenomenon, such as the third heart sound, which was ignored for centuries, should now be heard in so many cases by so many observers: as Goethe observed, 'Was man weisz, sieht man'. There is now general agreement that the physiological third heart sound is more commonly heard in young people. According to Evans (1948, 1951) it is rarely heard after the age of 25. Evans (1951, personal communication) had never heard a third heart sound in an individual over the age of 40, without some evidence of heart disease. Authorities differ widely in their views on the actual incidence of the third heart sound in various age-groups, as shown in Tables 1 and 2.

In any individual with a third heart sound the phenomenon tends to be variable (Thayer, 1909; Gallavardin, 1912; Wolferth & Margolies, 1933), the sound sometimes waxing and waning not only from day to day and from hour to hour but even from one cardiac cycle to the next. Furthermore, as the third heart sound is seldom much above the threshold of audibility of the observer, even a slight diminution of intensity may suffice to render it inaudible. In a subjective finding dependent on auscultation, individual opinion is notoriously un-

Table 3. Incidence of the third heart sound in children without heart disease: phonocardiography

| Date | Author | No. of subjects | Age-group (years) | Incidence of 3rd sound (per cent) |
|---|---|---|---|---|
| 1915 | Bridgman | 16 | 12 - 15 | 100.0 |
| 1932 | Leonhardt | 41 | 3 - 14 | 83.0 |
| 1936 | Segura | 120 | 0 - 2 | 0.0 |
| 1940 | Mannheimer | 135 | 0 - 14 | 69.6 |
| 1942 | Fazekas | 25 | 14 - 20 | 72.0 |
| 1946 | Carlgren | 150 | 3 - 17 | 78.0 |

Table 4. Incidence of the third heart sound in adults without heart disease: phonocardiography

| Date | Author | No. of subjects | Age-group (years) | Incidence of 3rd sound (per cent) |
|---|---|---|---|---|
| 1934 | Braun-Menéndez & Orías | 100 | 20 - 25 | 60.0 |
| 1934 | Clerc et al | 33 | 16 - 51 | 33.3 |
| 1935 | Pereira | 50 | 18 - 43 | 32.0 |
| 1937 | Caeiro & Orías | 20 | x | 35.0 |
| 1940 | Boyer et al. | 150 | x | 26.0 |
| 1944 | Frost | 169 | x | 10.7 |

x = not mentioned

reliable and the most expert auscultator is not necessarily he who claims to hear the sound in the greatest number of cases.

In view of all these factors close agreement between observers as to the incidence of the third heart sound, even in the same group of individuals, is not to be expected. The very great divergence of opinion as to the incidence of the third sound in the different groups shown in Tables 1 and 2 is no doubt due, in part, to the factors noted above. Yet even allowing for these, and for the fact that many observers have failed to standardize the conditions under which auscultation was performed, the differences are surprisingly great.

Phonocardiography. As with auscultation so with phonocardiography there is a marked difference of opinion as to the incidence of the third heart sound in different age-groups of healthy individuals. According to Lewis (1913a) the third heart sound is registrable in only a small proportion of normal hearts. Schütz (1933) recorded it commonly from children but rarely from healthy adults. Luisada (1943) registered both third and fourth heart sounds on most P.C.G.s from normal young individuals. The findings of those investigators who have expressed their results numerically are summarized in Tables 3 and 4.

Since many of these workers failed to standardize the conditions under which phonocardiography was undertaken, no satisfactory comparison of their findings is possible. An even greater diversity might have been expected since the performance of different phonocardiographs varies so greatly and many workers fail to indicate what distortion, if any, of the original chest-wall vibrations has been introduced. Since the third heart sound is a low-frequency phenomenon deflections corresponding to it will be recorded more frequently on linear or stethoscopic than on logarithmic P.C.G.s. In unfiltered (linear) P.C.G.s, McKee (1938a) found vibrations at the time of occurrence of the third sound in all her subjects, and Kountz, Gilson and Smith (1940) in most of theirs. Sloan, Campbell and Henderson (1952) compared logarithmic, stethoscopic and linear P.C.G.s of a group of healthy young adults, as described in Chapter 6.

Fourth heart sound

The fourth heart sound, due to atrial systole, was detected by members of the British Association (1840) on auscultation of the exposed heart of animals. It was heard in human subjects by Benjamins (1914) and recorded by Taquini and Braun-Menéndez (1935) by passing a stethoscope down the oesophagus to behind the atria. It is inaudible at the front of the chest in normal individuals,

Table 5. Incidence of the fourth heart sound in children without heart disease: phonocardiography

| Date | Author | No. of subjects | Age-group (years) | Incidence of 4th sound (per cent) |
|---|---|---|---|---|
| 1914 | Bridgman | 16 | 12 - 15 | 68.8 |
| 1936 | Segura | 120 | 0 - 2 | 38.0 |
| 1938 | McKee | 105 | 5 - 17 | 94.0 |
| 1940 | Mannheimer | 135 | 0 - 14 | 54.8 |
| 1946 | Carlgren | 150 | 3 - 17 | 50.0 |

Table 6. Incidence of the fourth heart sound in adults without heart disease: phonocardiography

| Date | Author | No. of subjects | Age-group (years) | Incidence of 4th sound (per cent) |
|---|---|---|---|---|
| 1934 | Braun-Menéndez & Orías | 100 | 20 - 25 | 15.0 |
| 1935 | Pereira | 50 | 18 - 43 | 16.0 |
| 1937 | Caeiro & Orías | 20 | x | 85.0 |
| 1940 | Boyer et al. | 150 | x | 27.0 |
| 1944 | Frost | 169 | x | 1.2 |

x = not mentioned

although the corresponding gallop sound is a common manifestation of heart disease. The fourth heart sound is recorded more frequently from the mesocardiac area than from other regions of the praecordium (Orías & Braun-Menéndez, 1939). The influence of posture and exercise do not appear to have been studied.

Phonocardiography. Although normally inaudible, the fourth heart sound has been recorded by phonocardiography in many individuals by a number of investigators, whose findings are summarized in Tables 5 and 6. The inaudibility of this commonly recorded event is probably attributable to two factors. Firstly the low fundamental frequency of the fourth sound vibrations (25-30 c.p.s.) approaches the lower frequency limit of human audibility, and there are few overtones. Secondly perception of the atrial vibrations may be masked by the closely-following, much louder, first heart sound.

Summation sound

At a rapid heart rate, such as that produced by active muscular exercise, a third heart sound might become louder due to an atrial systolic component being added to it. By phonocardiography it should be possible to demonstrate the fusion of the third and fourth sounds when tachycardia is induced in a normal heart but, in the ex-

periments described in Chapter 10, this did not occur.

I have found no reference in the literature to a summation sound occurring in a normal heart, but summation gallop rhythm has been described by a number of clinicians, whose findings are noted in Chapter 7.

Systolic extra sound

The systolic extra sound is a rare phenomenon, of no known significance but now believed to be innocent (Michaud & Fleisch, 1923; Macleod, Wilson & Barker, 1932; Thompson & Levine, 1935; Evans, 1943, 1948, 1951). It may be very loud (Thys & Brial, 1947). Very rarely there may be several extra sounds during systole.

Since the systolic extra sound has in the past been taken as a physical sign of heart disease it is referred to again in Chapter 7, along with the other extra sounds which occur in cardiac patients.

Fifth heart sound

Calò (1949, 1951) described a fifth heart sound, consisting of a small vibration occurring at a constant time interval after the third heart sound and attributed by him to elastic recoil of the ventricular wall after the rapid-filling phase. The fifth heart sound was never audible but was detected as a rare phenomenon on P.C.G.s both of normal subjects and of cardiac patients. The term, 'fifth heart sound', has not so far been accepted

by other cardiologists.

Summary.

The third heart sound can be heard and recorded in many healthy children and young adults but is not normally audible after about the age of thirty years.

The fourth heart sound is not normally audible but may appear on the P.C.G. of healthy subjects at any age.

A systolic extra sound is rare but may be heard and recorded in the absence of heart disease.

## Chapter 6

## The incidence of cardiac extra sounds in a series of healthy young adults

Although observers differ widely as to the incidence of the physiological third heart sound on auscultation in various age-groups of the population, the majority of those who have studied the problem find a high incidence in children and young adults (Tables 1 & 2). In my own experience, which is mainly confined to adults, I have heard the third sound in only a small proportion of healthy individuals. I have encountered much difference of opinion among physicians as to the presence or absence in particular cases of a third heart sound.

This investigation was planned to ascertain the incidence of the third heart sound in a group of healthy young adults and the degree of agreement between observers as to its presence in particular cases (Sloan, Campbell & Henderson, 1952).

Auscultation was performed independently by three observers, each of whom noted the presence or absence of a third heart sound in each subject. The observers listened also for any other audible extra sounds. Logarithmic, stethoscopic and linear P.C.G.s were then recorded from each subject. The occurrence of cardiac extra

sounds in the logarithmic P.C.G.s was compared with the auscultatory findings of each of the three observers, and the incidence of vibrations corresponding in time to cardiac extra sounds was compared in the three types of P.C.G.

Taking the group as a whole, the influence of age, sex and heart rate on the presence or absence of a third heart sound was assessed statistically. The incidence of the fourth heart sound on logarithmic, stethoscopic and linear phonocardiography was determined, and the relation of fourth sound to third sound on logarithmic P.C.G.s was assessed.

To summarize, the information which was sought regarding cardiac extra sounds in this group of subjects falls into the following categories:

(1) Auscultation.

(a) The incidence of the third heart sound.

(b) The degree of agreement between observers as to the presence or absence of a third heart sound.

(c) The relation of an audible third heart sound to the age, sex and heart rate of the subject.

(d) The incidence of any other cardiac extra sound.

(2) Phonocardiography.

(a) The incidence of the third heart sound on logarithmic phonocardiography.

(b) The relation of a third heart sound on the logarithmic P.C.G. to the age, sex and heart rate of the subject.

(c) The incidence of vibrations at the time of occurrence of the third heart sound on stethoscopic and linear P.C.G.s.

(d) The incidence of other cardiac extra sounds on logarithmic phonocardiography.

(e) The relation of the fourth heart sound to the third heart sound on logarithmic P.C.G.s.

(f) The incidence of vibrations at the time of occurrence of the fourth heart sound on stethoscopic and linear P.C.G.s.

(3) Combined auscultation and phonocardiography.

The degree of agreement between observers and the logarithmic P.C.G. as to the presence or absence of the third heart sound.

Observers

The three observers who listened to the heart sounds in this investigation had normal hearing as tested by routine audiometry but were of different degrees of experience in listening to heart sounds. Observer 1 had

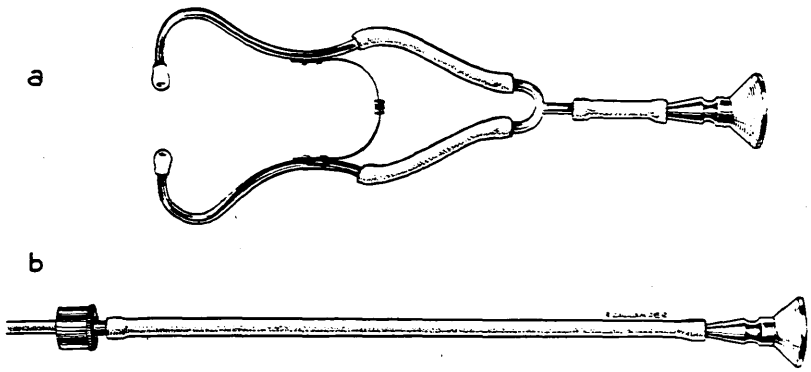


Fig. 12. STANDARD STETHOSCOPES:
a, FOR AUSCULTATION;
b, FOR PHONOCARDIOGRAPHY.

practised Medicine for 27 years, observer 2 for 10 years, and observer 3, since qualifying in Medicine 5 years previously, had been engaged mostly in ophthalmology.

Method

The stethoscopes used for auscultation were of a standard pattern shown in Fig. 12 (a). The chest-pieces were identical with that used for phonocardiography, already described in Chapter 4. For auscultation the chest-piece was joined by a single length of rubber stethoscope tubing to a Y-piece, whence two lengths of the tubing led to an ordinary binaural ear-piece. The length of tubing from chest-piece to ear was 46 cm., the shortest found to be compatible with convenience in auscultation; the bore of the tubing was 5 mm. and the wall was 3.5 mm. thick.

The phonocardiograph employed has been described in Chapter 4. In keeping with the recommendations of Rappaport and Sprague (1942) and Cowen and Parnum (1949), logarithmic, stethoscopic and linear P.C.G.s were recorded. The jugular phlebogram was used as a reference tracing in each case and an additional linear P.C.G. was recorded with the E.C.G. (Lead II) as the reference tracing, to aid in the identification of the waves of the venous pulse in difficult cases.

For the logarithmic P.C.G. a chest-piece, identical

with that used for auscultation, was applied over the cardiac apex and held in position by a rubber band round the chest. From the chest-piece a length of 46 cm. of rubber tubing, of the same calibre and thickness as that of the stethoscopes, connected the chest-piece to the crystal microphone (Fig. 12b). The distortion of sound waves produced by the chest-piece and stethoscope tubing was not analysed: the intention was primarily to equate the physical conditions for phonocardiography and for auscultation.

For the stethoscopic P.C.G. the chest-piece and stethoscope tubing were as for the logarithmic record but no filters were employed in the preamplifier (filter position 0). For the linear P.C.G. the stethoscope chest-piece and tubing were removed and a metal chest-piece of the same dimensions was screwed to the microphone casing and held directly against the chest by a rubber band: once again the preamplifier was used without filters.

Subjects

The subjects of this investigation were students at Glasgow University. At this University all students are encouraged to report annually to the Student Health Service for routine medical examination; during the session 1950-51, 30.9% of them did so. In order to obtain

as nearly as possible a representative cross-section of the student community, every fifth student who reported for medical examination during the Martinmas term (October - December), 1950, was asked to be a subject for this investigation. Out of a total of 125 students who were asked, 123 did offer themselves as subjects, so the results are reasonably representative of the students as a whole. Of the subjects, 96 were men and 27 were women. It so happened that none of them had any evidence of heart disease.

Criteria

Each subject was examined by each of the three observers independently, not necessarily on the same day. Auscultation was performed at the mitral area with one of the standard stethoscopes, the subject being supine and rested for approximately five minutes. An occasional extra sound was ignored but a regularly recurring one was noted as a positive finding.

For phonocardiography the subject was again supine and rested for approximately five minutes. Heart sounds were recorded at the mitral area with the breath held at the end of expiration in order to standardize the physical conditions and eliminate breath sounds.

In this investigation, as in that by Luisada and

Roitman (1948), the amplitude of the extra sound vibrations was measured against that of the first and second sounds. The gain of the amplifier system was adjusted so that the final photographic record of the first or second sound deflections, whichever was the greater, was not less than 1 cm. and not more than 2 cm. With this as a standard, any deflection occurring in the position of a cardiac extra sound in at least three successive cycles was taken as indicating the presence of the sound: otherwise the sound was judged to be absent.

Results

The detailed findings by auscultation and by logarithmic phonocardiography of each subject are given in Table 30 (Volume 2). Plates 9 and 10 (Volume 2) are examples of the records obtained.

Auscultation.

(a) The third heart sound was heard by a majority of the three observers in 12 cases (9.8%).

(b) It is evident from Table 30 that the agreement between observers is of a low order. The incidence of the third sound on auscultation is so small that the ordinary $\chi^2$ test of significance is inapplicable to assess the agreement between the observers. Using Fisher's exact method the agreement is found to be significant. The details are discussed below.

(c) By the classical $\chi^2$ method a negative correlation was found between the incidence of the third heart sound as heard by the observers and the age of the subject. No significant relation was found between the sex or heart rate and the presence of a third heart sound.

No other cardiac extra sound was heard in any of the subjects. In 22 cases an apical systolic murmur was audible.

Phonocardiography.

(a) In the logarithmic P.C.G.s a deflection in the position of the third heart sound was recorded in 48 cases (39.0%). In all of these it was found to commence either during the down-stroke or at the foot of the v wave of the venous pulse; in other words it was a true 'rapid-filling' sound.

(b) The incidence of the third heart sound, as recorded on the logarithmic P.C.G. was not found to bear any significant relationship to the age, sex or heart rate of the subject.

(c) A definite deflection was seen in the position of the third heart sound in the stethoscopic and linear records of every subject.

(d) In 34 cases (27.6%) regularly recurring deflections corresponding in time to atrial systole were recorded on the logarithmic P.C.G. These deflections

Table 7. Incidence of the third heart sound in 123 young, healthy adults

| Auscultation Heard by observers | Phonocardiography (logarithmic) | |
|---|---|---|
| | Recorded | Not recorded |
| None | 29 | 60 |
| Observer 1 only | 1 | 1 |
| Observer 2 only | 3 | 1 |
| Observer 3 only | 8 | 8 |
| Observers 1 and 2 | 2 | 0 |
| Observers 1 and 3 | 2 | 2 |
| Observers 2 and 3 | 1 | 2 |
| Observers 1, 2 and 3 | 2 | 1 |

coincided with or closely followed the P wave of the E.C.G. and began 0.02-0.04 sec. after the beginning of the a wave of the jugular phlebogram; they represent the fourth heart sound. None of these could be summation sounds since diastole was in no case short enough to permit superposition of third and fourth sound vibrations.

Systolic extra sound vibrations were seen on the logarithmic P.C.G.s of 4 subjects. In 31 cases a systolic murmur was recorded.

(e) The fourth heart sound was significantly commoner in cases in whom a third sound also was recorded ($P < 0.001$).

(f) Deflections corresponding in time to atrial systole were constantly present in the stethoscopic and linear P.C.G.s. Since these records show a continuous series of waves throughout the period of ventricular systole it is impossible to distinguish any vibrations corresponding to a definite systolic extra sound.

Combined auscultation and phonocardiography. The relation between the findings by auscultation and by phonocardiography regarding the presence or absence of a third heart sound in the subjects investigated is summarized in Table 7. In 60 subjects none of the observers heard the third sound and none was seen on the logarithmic

P.C.G. In 29 subjects the third sound appeared on the logarithmic record but was not heard by any of the three observers. Observer 1 reported a third sound in 11 subjects, his findings being confirmed by the logarithmic P.C.G. in 7 cases. Observer 2 heard a third sound in 12 subjects, this being confirmed by the logarithmic record in 8 cases. Observer 3 heard a third sound in 26 subjects but the logarithmic P.C.G. showed the sound in only 13 of these.

Discussion

Authorities differ widely as to the actual incidence of the third heart sound (Tables 1 - 4) but most agree that it is detectable on auscultation or by phonocardiography in only a proportion of normal subjects. There are two possible explanations of this. The 'sound' may be due to vibrations which are present in only a proportion of subjects; the third heart sound would then be due to a phenomenon of cardiac activity peculiar to certain individuals. Alternatively the vibrations may be present early in diastole in all subjects but in many cases be of such amplitude and frequency that they cannot be detected. On this latter interpretation detection of a third heart sound may depend largely on extraneous circumstances such as the experience of the observer, the level of background noise in the environment, and indeed

Table 8. Agreement between observers 1 and 2 as to the presence or absence of a third heart sound: auscultation

| | | Observer 1 | | Total |
|---|---|---|---|---|
| | | Heard | Not heard | |
| Observer 2 | Heard | 5 | 7 | 12 |
| | Not heard | 6 | 105 | 111 |
| Total | | 11 | 112 | 123 |

Table 9. Agreement between observers 1 and 3 as to the presence or absence of a third heart sound: auscultation

| | | Observer 1 | | Total |
|---|---|---|---|---|
| | | Heard | Not heard | |
| Observer 3 | Heard | 7 | 19 | 26 |
| | Not heard | 4 | 93 | 97 |
| Total | | 11 | 112 | 123 |

Table 10. Agreement between observers 2 and 3 as to the presence or absence of a third heart sound: auscultation

| | | Observer 2 | | Total |
|---|---|---|---|---|
| | | Heard | Not heard | |
| Observer 3 | Heard | 6 | 20 | 26 |
| | Not heard | 6 | 91 | 97 |
| Total | | 12 | 111 | 123 |

on many factors having nothing to do with the activity of the heart. The discrepancies between the findings of different observers favour the second alternative.

Auscultation. The incidence of the third heart sound on auscultation in our series of cases (9.8%) is lower than that of most previous observers. It is of interest to note that the greatest number of third heart sounds was noted by the least experienced observer, whose auscultatory findings showed the poorest correlation with the P.C.G.s.

The degree of agreement between observers as to the presence or absence of a third heart sound is found, using Fisher's exact method (Fisher, 1946) from three 2 x 2 tables (Tables 8, 9 & 10).

Analysing these tables we find that agreement between observers is significant in each case ($P < 0.05$).

That there is significant agreement, albeit of a low order, between observers as to the presence or absence of a third heart sound in particular cases suggests that the observation is worth making, although in any individual case the prospect of complete agreement between a number of auscultators may be remote.

In this investigation the ages of the subjects ranged from 17 to 32 years, with more than 90% under the age of 26. With each of the observers the incidence of the

third heart sound was lower in the higher age-groups. Taking the whole series there was a significantly higher incidence of the third heart sound in the younger subjects. Although this relationship has been noted by many observers no adequate explanation has been offered.

No significant sex difference in the incidence of the third heart sound was noted by the previous investigators whose results I have studied, and none was found in this investigation. In contrast to Thayer (1909) and Lewis (1913a), who detected the third sound more frequently at slower heart rates, there was no significant correlation in this series between the heart rate and the presence or absence of the sound.

Phonocardiography. Our findings of the incidence of the third heart sound on the logarithmic P.C.G.s of our series of healthy adult subjects show reasonable agreement with those of earlier workers (Table 4).

Deflections in the position of the third heart sound were recorded on the linear and stethoscopic P.C.G.s of all our subjects, indicating that vibrations were set up by the heart at this time in all of them. In the majority, however, these vibrations were of such low frequency as to be inaudible, and they were not seen on the logarithmic P.C.G.s.

Deflections corresponding to the fourth heart sound were seen on the logarithmic P.C.G. of 34 of the subjects (27.6%), which is rather higher than the incidence observed by most previous investigators (Table 6). The presence of the fourth heart sound in many logarithmic P.C.G.s, though it was never audible, may be attributed to two factors, as suggested in Chapter 5. Firstly, the fourth sound occurs at such a short interval of time before the succeeding first sound that it may be masked. Secondly the absolute low-frequency cut-off of the phonocardiograph may be below that of the human auditory mechanism, although the frequency-response curves are similar.

I have remarked that there is a highly significant positive correlation between the occurrence of third and fourth sounds on the P.C.G. This is not unexpected since both sounds occur at a time when the ventricles are filling rapidly and appear to be related in some way to this rapid filling. The aetiology of cardiac extra sounds is studied in Chapters 7 and 9.

As in the case of the third heart sound, so with the fourth sound, vibrations are constantly produced by atrial systole but, in most cases are not of sufficient frequency and amplitude to constitute a 'sound'.

Table 11. Agreement between observer 1 and logarithmic P.C.G. as to the presence or absence of a third heart sound: auscultation and phonocardiography

| | | Observer 1 | | Total |
|---|---|---|---|---|
| | | Heard | Not heard | |
| P.C.G. | Recorded | 7 | 41 | 48 |
| | Not recorded | 4 | 71 | 75 |
| Total | | 11 | 112 | 123 |

Table 12. Agreement between observer 2 and logarithmic P.C.G. as to the presence or absence of a third heart sound: auscultation and phonocardiography

| | | Observer 2 | | Total |
|---|---|---|---|---|
| | | Heard | Not heard | |
| P.C.G. | Recorded | 8 | 40 | 48 |
| | Not recorded | 4 | 71 | 75 |
| Total | | 12 | 111 | 123 |

Table 13. Agreement between observer 3 and logarithmic P.C.G. as to the presence or absence of a third heart sound: auscultation and phonocardiography

| | | Observer 3 | | Total |
|---|---|---|---|---|
| | | Heard | Not heard | |
| P.C.G. | Recorded | 13 | 35 | 48 |
| | Not recorded | 13 | 62 | 75 |
| Total | | 26 | 97 | 123 |

Combined auscultation and phonocardiography. It is evident from Table 7 that the agreement between auscultation and phonocardiography is of a low order. In only two cases was there unanimity as to the presence of the third heart sound, in 60 cases there was complete agreement as to the absence of the sound, and in the remaining 61 cases there was more or less disagreement. The results may be further analysed by comparing the findings of each observer with the P.C.G.s (Tables 11, 12 & 13). The probability of the agreement noted between observer and phonocardiogram occurring by chance is: for observer 1, $P > 0.05$; observer 2, $P < 0.05$; and observer 3, $P > 0.1$: agreement is significant only in the case of observer 2.

One might expect, if the logarithmic phonocardiograph acts as the 'ideal observer' that it would show a third heart sound on all records of subjects in whom a third heart sound was heard and also in some of the records from subjects in whom no third heart sound was heard. In fact approximately four times as many third sounds were recorded on the logarithmic P.C.G.s as were heard by a majority of the observers, but in five cases where a third sound was heard by a majority of the observers it was not recorded on the logarithmic P.C.G. This apparent fallacy is explained by the varying gain of the instrument required to obtain satisfactory records of sounds of

very different intensity. Williams and Dodge (1926) indicated that the heart sounds of some individuals are as much as 40 times louder than those of others. Consequently it is impracticable to record at constant gain on a narrow strip of photographic material the heart sounds of different subjects. The sensitivity of the ear probably does not vary much from day to day in an individual observer but that of the instrument must be varied considerably from subject to subject. It follows that a third heart sound on auscultation is, in a sense, an absolute phenomenon, whereas on a P.C.G. it is a relative phenomenon, relative to the loudness of the first or second heart sound. If the first or second sound is particularly loud the gain of the instrument must be reduced below the usual working level in order to record the deflections; in these circumstances any deflection due to the third heart sound is also reduced and may disappear.

The negative correlation between the audible third heart sound and the age of the subject did not occur with phonocardiography, which indicates that this negative correlation depends on the absolute intensity of the sound rather than on its intensity relative to other heart sounds; in other words there is a falling off in the

absolute intensity of all the heart sounds as age advances.

Summary

In a group of 123 University students without any evidence of heart disease a third heart sound was heard by a majority of three observers in 12 cases (9.8%).

The agreement between the observers as to the presence or absence of the third heart sound was of a low order but was significant.

A negative correlation was found between the incidence of the third heart sound, as heard by the observers, and the age of the subjects, but no correlation was found between the incidence and the sex or heart rate of the subjects.

No other cardiac extra sound was heard in any case.

Deflections occurred in the position of the third heart sound in all linear and stethoscopic P.C.G.s and in the logarithmic P.C.G. of 48 of the subjects (39.0%).

The incidence of the third heart sound, as shown on the logarithmic P.C.G., was not related to the age, sex or heart rate of the subject.

Vibrations were recorded at the time of the fourth heart sound in all linear and stethoscopic records and, in 34 subjects (27.6%), on the logarithmic P.C.G. In the logarithmic records they were significantly commoner in cases where a third sound also was recorded. A systolic extra sound was recorded on the logarithmic

P.C.G. in 4 cases.

There was poor agreement between the observers' finding of the presence or absence of a third heart sound by auscultation and the presence or absence of the corresponding deflections on the logarithmic P.C.G.

# PART IV

## CARDIAC EXTRA SOUNDS IN PATIENTS WITH HEART DISEASE

"Thus historically it is not incorrect to regard gallop rhythm and triple rhythm as synonymous; but there is an advantage in excluding certain types of triple rhythm from the cadences embraced by the bruit de galop. Thus it is preferable and customary to speak of presystolic (auricular), protodiastolic, systolic, and summation gallop on the one hand; and of systolic clicks, the normal third heart sound and the triple rhythm of mitral stenosis on the other."

Paul Wood (1950)
Diseases of the heart and circulation.
London: Eyre & Spottiswoode
p.166

"Der Galopprhythmus des Herzens ist doch ein Schrei des Herzens nach Hilfe, und trotzdem gibt es nur wenige Aertze, die ihn hören."

W.B. Obrastzow (1905)
Ueber die verdoppelten und akzessorischen Herztöne bei unmittelbarer Auskultation des Herzens.
Z. klin. Med., vol. 57
p.88

# Chapter 7

## Historical review: gallop rhythm and the mitral opening snap

### Definition

Much of the confusion which still exists today as to the significance of gallop rhythm is attributable to the lack of a clear definition of the term. Since Potain (1875) popularized the expression, 'bruit de galop', which he had learnt in Bouillaud's clinic, clinicians throughout the world have applied it to widely different phenomena. Although, in Potain's original description, gallop rhythm meant presystolic gallop (the addition to the first and second heart sounds of an extra sound coinciding with atrial systole) the term was variously applied by subsequent workers to this or to other forms of triple cardiac rhythm, or even to splitting of the first or second heart sound.

The equine analogy was reduced to absurdity by Fraentzel (1881, 1889) who identified cardiac rhythms corresponding to 'ordinary', 'school' or 'race' gallop.

Potain later widened his original concept of gallop rhythm, defining it as triple cardiac rhythm due to the addition to the two normal heart sounds of a third and different sound, which is not a murmur or a friction sound, but an abrupt sound interposed between the normal sounds

(Potain, 1900a). This definition excludes splitting of the first or second heart sounds, in which case the two components are of the same quality and intensity and separated from each other by a very short interval.

After the discovery some five years later of the physiological third heart sound the term 'gallop rhythm' came to be reserved by most workers in this field for pathological triple cardiac rhythm. As mentioned in Chapter 2 it is unfortunate that such a distinction should be made, namely that a criterion of gallop rhythm should be other evidence of heart disease in the patient, but the distinction is generally adopted and I employ it here. (see page 91

The precise localization of cardiac extra sounds in the cardiac cycle had to await the development of the phonocardiograph; only with this instrument could the different diastolic sounds be identified with any certainty.

Another problem is set by the systolic extra sound. Systolic gallop rhythm was described by a number of observers, who related it to particular types of disease. The systolic extra sound is now considered to be without pathological significance but this view may not be the ultimate one; in any case a historical review must include 'systolic gallop rhythm'.

It has never been made quite clear whether triple rhythm due to the opening snap of the mitral valve should or should not be classified under the general heading of gallop rhythm. As a rule the opening snap has been considered as an isolated phenomenon, distinct from other forms of triple cardiac rhythm.

A definition of gallop rhythm must be arbitrary, but some definition is essential. In an attempt to be up-to-date and yet consistent I suggest the following modification of Potain's definition: 'Gallop rhythm is triple rhythm of the heart due to the addition to the first and second heart sounds of the third heart sound, the fourth heart sound, or a summation sound, in a patient with heart disease'.

Early observations on gallop rhythm

The first case in the literature of what we should now call gallop rhythm is that described by Charcelay (1838). In this patient a diastolic extra sound was audible at the right sternal border at the level of the second rib; there were also systolic and diastolic murmurs. Autopsy showed an atrial septal defect with dilatation of the right atrium, mitral stenosis, and aortic incompetence. Charcelay attributed the extra sound to audible contraction of the enlarged right atrium.

Bouillaud (1841) described the characteristic triple

rhythm of mitral stenosis, 'bruit de rappel', which he attributed to splitting of the second heart sound. Splitting of the second heart sound in cases of mitral stenosis was noted also by Skoda (1844) and Duroziez (1862). Potain (1866) and Guttmann (1869, 1878) affirmed that splitting of the first or second heart sound is not necessarily associated with valvular disease but occurs also in individuals with perfectly normal hearts.

Potain (1875) described presystolic gallop rhythm, which he associated with cardiac hypertrophy in the absence of valvular disease; it was commonly a manifestation of kidney disease, especially of chronic interstitial nephritis. The sound was attributed to sudden distension of the ventricles at atrial systole. Potain mentioned other forms of triple cardiac rhythm, including those due to the reduplicated second heart sound of mitral disease and to the systolic extra sound. Exchaquet (1875), Johnson (1876), Lépine (1881), and Cuffer and Guinon (1886) supported Potain's views on the mechanism of production of the presystolic sound and on the relation between Bright's disease and presystolic gallop rhythm.

A different explanation of the presystolic extra sound was offered by Walshe (1873), Barr (1877), and Bindley (1878), who all ascribed it to asynchronous con-

traction of the two ventricles. Sansom (1881) and Fraentzel (1881) attributed the presystolic gallop sound to an audible atrial contraction, but Sansom thought it might sometimes be due to asynchronous contraction of the two ventricles; Peter (1883) believed that it was always due to this.

D'Espine (1882), Leyden (1886), and Bouveret and Chabalier (1889) considered that gallop rhythm is due to ventricular systole taking place in two stages when an enfeebled heart is acting against high arterial pressure; each phase of ventricular systole, according to this theory, causes sudden tension of the atrio-ventricular valves. The extra sound is produced in the left ventricle when the aortic pressure is raised, as in chronic interstitial nephritis, or in the right ventricle when there is some obstruction to the pulmonary circulation. François-Franck (1880) and Barié (1883, 1893b,c) considered that this obstruction might be due to reflex constriction of the pulmonary blood vessels resulting from some disturbance of the alimentary canal or of the liver. Cuffer and Barbillion (1887) agreed with D'Espine on the connection between left-sided or right-sided gallop rhythm and hypertension in the corresponding artery. They noted that the diastolic extra sound might occur so long before the first heart sound as to approach the preceding second

heart sound. They did not attempt to explain the diastolic extra sound but concluded that contraction of the ventricle in two stages would produce a systolic extra sound.

Kriege and Schmall (1891) and Huchard (1893) believed that the presystolic extra sound was due to sudden filling of the ventricle to its elastic limit; with arterial hypertension or myocardial hypertrophy this elastic limit was more readily attained.

The need to distinguish diastolic extra sounds from reduplication of the first or second heart sounds, was emphasised by François-Franck (1889), Barié (1893a), and Phear (1897).

Potain (1894), as was his wont, cleared up the confusion. He distinguished gallop rhythm due to a dull sound, palpable rather than audible, from reduplication of the first or second heart sounds and from the opening snap of the mitral valve. He also described presystolic, protodiastolic and systolic forms of gallop rhythm. A great advance was his identification of presystolic gallop rhythm, in which the time interval between the extra sound and the following first sound is constant, and of protodiastolic gallop rhythm, in which the extra sound follows at a constant interval after the preceding second heart sound, whatever the period of diastole in

which the extra sound actually occurs. For example a presystolic (atrial) gallop sound, occurring at a constant time before the first sound, might become mesodiastolic or even protodiastolic in time when tachycardia shortened the diastolic interval.

In a later paper, Potain (1900a) stressed again the distinction between gallop rhythm and reduplication of the first or second heart sound, the distinction between diastolic gallop rhythm and systolic gallop rhythm, which he associated with arteriosclerosis, and the distinction between left-sided diastolic gallop, attributable to cardiac hypertrophy, and right-sided diastolic gallop, which he associated with hepatic or gastro-intestinal disturbance. Left-sided gallop rhythm was best heard at the cardiac apex, whereas right-sided gallop rhythm was best heard over the lower part of the sternum.

A. Chauveau (1900) and H. Chauveau (1902) attributed the presystolic extra sound to a ventricular pressure wave caused by contraction of the papillary muscles during the interval between atrial and ventricular systole. Bard (1906, 1908) and Trautwein (1907) thought it was usually due to separation of the muscular and valvular components of the first heart sound, but Bard believed that a presystolic sound could also be produced by contraction of a hypertrophied atrium.

At the turn of the century the development of phonocardiography ushered in the modern era in the investigation of heart sounds, including those involved in gallop rhythm. Potain's acute clinical observations have not been superseded but it is now possible by phonocardiography to establish the precise time of occurrence of extra sounds in the cardiac cycle and to study the physical characters of these sounds. According to Lewis (1915): 'Timing by auscultation is often a difficult or impossible task if it is to be carried out with any pretension to accuracy'.

Presystolic gallop rhythm

As noted above, the classical presystolic gallop rhythm described by Potain has been variously attributed to audible contraction of the atria, sudden distension of the ventricles by the blood forced into them by atrial systole, asynchronous contraction of the ventricles resulting in asynchronous closure of the mitral and tricuspid valves, or two-stage ventricular systole. The last two of these mechanisms, whose occurrence is still matter for dispute, would cause splitting of the first sound rather than the cardiac extra sound which we now call the fourth heart sound or presystolic gallop sound.

Left-sided presystolic gallop, as noted by D'Espine (1882), Cuffer and Barbillion (1887), Barié (1893a,c), and Potain (1900a), is commoner than right-sided pre-

systolic gallop, is more constant when it does occur, and is the rhythm typically associated with the left ventricular hypertrophy which follows chronic interstitial nephritis.

Little interest has been shown since Potain's time in right-sided presystolic gallop, except by Lutembacher (1916), Giroux (1923), and Laubry, Routier and Largeau (1924), who all considered it an important sign of right-sided cardiac insufficiency.

Aetiology. Atrial systole normally produces a sound, audible from the oesophagus and recordable from there and, in many cases, from the praecordium, as already noted in Chapter 5. Clark (1858) reported an interesting case of a criminal executed by judicial hanging, in whom a regular single heart sound was audible at the front of the chest an hour and a half after execution. When the chest was opened the right atrium was found to be beating regularly but the ventricles were inert.

Chauveau (1885) described the first case of complete heart block in the literature: he noted that atrial systolic sounds were audible, especially when they occurred during ventricular systole when the flow of blood into the ventricle was blocked by the tense atrio-ventricular valves. Josué and Godlewski (1913), Gallavardin (1914), and Lewis (1915), confirmed the audibility of atrial sounds

in cases of complete heart block.

Lépine (1882) and Müller (1906) noted that the normal apical cardiogram (linear P.C.G.) showed a wave corresponding to atrial systole; when gallop rhythm was audible this wave was of greater amplitude and occurred earlier than the normal presystolic wave. Pawinski (1907) believed that exaggerated atrial activity caused early closure of the atrio-ventricular valves and, if atrio-ventricular conduction were impaired, the muscular component of the first heart sound would be delayed, further separating the two components. Robinson (1908) found on auscultation that the extra sound of presystolic gallop did not resemble the isolated atrial sound of complete heart block. Von Wyss (1911) believed that presystolic gallop was an exaggeration of the normal atrial systolic sound, which he demonstrated by phonocardiography. Taking the jugular phlebogram of patients with presystolic gallop rhythm he found that the a-c interval was usually at or above the upper limit of normal, indicating a prolonged interval between atrial and ventricular systole.

Potain's explanation of the atrial systolic sound as being due to sudden distension of the ventricles by the inflow of blood during atrial systole was supported by Gallavardin (1914) and Pezzi (1914). Lewis (1915), Reid (1921), and Duchosal (1932) found that the audible

atrial sound in cases of complete heart block was often double. In Lewis's opinion the first component of the double atrial sound was produced by contraction of the atria and tension of their wall; the second component occurred in early atrial diastole and was due to closure of the atrio-ventricular valves.

Gallavardin (1920), Laubry and Mougeot (1921), Giroux (1923), and Routier and van Bogaert (1934a) supported the view of Von Wyss (1911) that gallop rhythm is due to delayed atrio-ventricular conduction. Gallavardin found that ventricular systole also was prolonged in these cases. Bordet, Yacoël and Giroux (1923) described six patients with gallop rhythm in all of whom the E.C.G. showed prolongation of the P-R, Q-S, and Q-T, intervals. They noted that the atrial sound could occur at any time in diastole and they associated it with sclerosis, hypertrophy, or hypotonicity, of the ventricular muscle.

Holt (1927), reviewing the literature to date, associated presystolic gallop rhythm with atrial hypertrophy and, in some cases, delayed atrio-ventricular conduction. White (1928) and Frost (1944), on the other hand, rarely found any degree of heart block in patients with gallop rhythm. Holt explained the presystolic gallop sound as primarily due to vibration of the ventricular wall or of the atrio-ventricular valves, and affirm-

ed that it is usually impossible to distinguish left-sided from right-sided gallop.

Mond and Oppenheimer (1929) demonstrated the presystolic gallop sound by phonocardiography and related it to arterial hypertension. They believed that the vibrations produced by atrial systole are normally damped by the flaccid ventricular walls; if, however, the tone of the ventricular wall is high, this damping is reduced and an atrial sound becomes audible. This explanation is compatible with that of Chauveau (1885) on the first reported case of complete heart block. The contrary view was expressed by Gubergritz (1929), Mozer and Duchosal (1930), Duchosal (1932), Bramwell (1935b), and O'Farrell (1939), who attributed the presystolic gallop sound, as Potain had done, to sudden distension of a hypotonic ventricle. Mozer and Duchosal demonstrated by phonocardiography that with tachycardia and delayed atrioventricular conduction the gallop sound occurred in early diastole; with the onset of auricular fibrillation the gallop sound disappeared. Fogelson (1932) confirmed the disappearance of the presystolic gallop sound with the onset of auricular fibrillation.

King (1928) and King and McEachern (1932) believed that bundle branch block caused reduplication of the first heart sound, simulating gallop rhythm, but Campbell and

Suszman (1932) reported a case of true presystolic gallop rhythm with bundle branch block. Fogelson (1932), Macleod, Wilson and Barker (1932), and J.K. Lewis (1934) detected a high incidence of gallop rhythm in patients with bundle branch block. Fogelson believed that the gallop rhythm in these cases was caused by asynchronous contraction of the two ventricles, but Lewis considered that the gallop rhythm and the bundle branch block were independent manifestations of the same underlying cardiac lesion.

Cossio and Lascales (1936) agreed with the previous observations of Lewis that an isolated atrial sound is usually double and that the second component is due to stretching of the ventricular wall or of the atrio-ventricular valves by the rapid inflow of blood at atrial systole. Cossio, Berconsky and Trimani (1942) held that the first component is due to distension of the ventricles and the second component to elevation and tension of the atrio-ventricular valves.

Lewis and Dock (1938) gave as the probable causes of presystolic gallop rhythm either delayed atrio-ventricular conduction or myocardial failure. Lian and Welti (1938) considered arterial hypertension and left ventricular failure the most important aetiological factors. Lian and Hubert (1948a) restated this opinion. Evans (1943) distinguished the fourth heart sound which occurs with

delayed atrio-ventricular conduction from that due to left ventricular failure.

The opinion that presystolic gallop is due merely to exaggeration of the normal atrial systolic vibrations and is not qualitatively different from the fourth heart sound normally revealed by phonocardiography was held by Lewis and Dock (1938), Arenberg (1941), Frost (1944, 1949a), and Cossio and Korn (1947). On the other hand, Mannheimer (1940), Carlgren (1946), and Luisada and Roitman (1948), held that gallop sounds are distinguishable by phonocardiography from physiological extra sounds by their greater amplitude and higher frequency.

Pathological significance. In addition to Bright's disease and arterial hypertension, noted by Potain and many subsequent observers as aetiological factors in many cases of presystolic gallop rhythm, a number of other pathological processes have been incriminated. These include: pneumonia or typhoid fever (Fraentzel, 1881; Pawinski, 1907); diphtheria (Leyden, 1882; Vierordt, 1888; Fraentzel, 1889; Elliott, 1921); acute rheumatism or scarlatina (Fraentzel, 1889; Giroux, 1923); aortitis (Barié, 1893c; Laubry & Pezzi, 1926); mitral stenosis (Mannheimer, 1942); coronary artery disease (Garvin, 1943; Weber, 1944); and pericarditis or toxic myocarditis (Weber, 1944). Frost (1949b) found that

Table 14. Incidence of presystolic gallop rhythm in patients with heart disease: auscultation

| Date | Author | No. of subjects | Clinical condition | Incidence (per cent) |
|---|---|---|---|---|
| 1935 | Bramwell | 1353 | Heart disease | 4.7 |
| 1941 | Arenberg | 181 | Organic heart disease | 3.3 |
| 1947 | Cossio & Korn | 4052 | Heart disease | 4.5 |

Table 15. Incidence of presystolic gallop rhythm in patients with heart disease: phonocardiography

| Date | Author | No. of subjects | Clinical condition | Incidence (per cent) |
|---|---|---|---|---|
| 1932 | Duchosal | 33 | Heart disease | 69.7 |
| 1934 | Lewis | 23 | Bundle branch block | 39.1 |
| 1941 | Arenberg | 181 | Organic heart disease | 11.6 |
| 1945 | Friedländer | 83 | Acute rheumatism (children) | 6.0 |
| 1946 | Carlgren | 5000 (approx.) | Suspected heart disease (children) | 0.5 |

gallop rhythm was rare in cases of mitral stenosis, anaemia, or thyrotoxicosis. Contro and Luisada (1952) and Caniggia (1953) confirmed by phonocardiography the high incidence of presystolic gallop in patients with bundle branch block.

Incidence. The incidence of presystolic gallop rhythm, as noted by those observers who have expressed numerically their findings by auscultation, is shown in Table 14. The degree of agreement between different observers is remarkable.

The incidence as detected by phonocardiography is shown in Table 15. The wide discrepancies here may be due to the diverse nature of the groups studied as well as to the different characteristics of the phonocardiographs employed and to different interpretation of the records obtained.

Prognosis. Most clinicians, who have commented on this aspect of the matter, affirm that presystolic gallop rhythm is of grave prognostic significance. These include: Fraentzel (1881), Vierordt (1888), Schrumpf (1918), Elliott (1921), Giroux (1923), Freundlich (1931), Bramwell (1935b), O'Farrell (1939), Mannheimer (1942), Garvin (1943), Frost (1944, 1949b), and Friedländer (1945). But Guyard (1899), Bacaloglu (1900) and Mollard and Dumas (1914) found in patients with typhoid fever that gallop rhythm was of commoner occurrence in those

who eventually recovered than in those who did not.

Gubergritz (1914) held that the prognosis was bad only if the blood pressure was low, and Mollard and Dumas (1914) and Routier and van Bogaert (1934c) that gallop rhythm did not affect the prognosis. Mozer and Duchosal (1930), Duchosal (1932), and Routier and van Heerswynghels (1935) believed that the prognosis was worse the greater the time interval between the presystolic extra sound and the next first heart sound. Fogelson (1932) stated that the prognosis is bad when gallop rhythm occurs along with bundle branch block, but good when it is associated with delayed atrio-ventricular conduction. Bramwell (1935b), the only one of the above authors to cite evidence in support of his opinion, found that only 15 out of a series of 63 patients with presystolic gallop rhythm lived for more than 18 months after the gallop rhythm had been detected.

In patients with arterial hypertension, Miles (1951) found no evidence that the existence of gallop rhythm was of any prognostic significance.

Protodiastolic gallop rhythm.

Potain (1894) described protodiastolic gallop rhythm, in which the extra sound (now called the third heart sound) occurs at a constant time interval after the second heart sound.

Obrastzow (1905) observed that the third heart sound might occur in the absence of heart disease but, in this case, only when the patient was recumbent. The corresponding protodiastolic gallop sound in patients with heart disease was louder and did not disappear when the patient stood up.

Aetiology. Müller (1906) detected a wave corresponding to the extra sound in the apical cardiogram of patients with protodiastolic gallop rhythm. By recording the apical cardiogram simultaneously with the jugular phlebogram he ascertained that the wave coincided in time with the rapid-filling phase of ventricular diastole after the opening of the atrio-ventricular valves.

Most authorities agree that the physiological third heart sound and protodiastolic gallop sound are connected with rapid ventricular filling. Robinson (1908), Laubry and Mougeot (1921), Gubergritz (1929), and Routier and van Bogaert (1934b) believed that the sound depends on rapid filling of ventricles with poor myocardial tone. Bard (1922) thought it was produced by active ventricular relaxation.

Von Wyss (1911) and Holt (1927) confused the protodiastolic gallop sound with the opening snap of the mitral valve.

White (1928) and Carlgren (1946) found protodiastolic

gallop the commonest form of gallop rhythm. White and, later, Gubergritz (1929) noted by auscultation that, at a rapid heart rate, the protodiastolic gallop sound could become mesodiastolic or even presystolic in time.

Wolferth and Margolies (1933) employed phonocardiography in the investigation of protodiastolic gallop rhythm but, since their reference tracing was the E.C.G., they were unable to distinguish the rapid-filling sound from other diastolic sounds. Taquini, Massell and Walsh (1940), employing phonocardiography with the jugular phlebogram or the apical cardiogram as reference tracing, were able to identify protodiastolic gallop rhythm in some of their rheumatic patients.

Most writers on the subject opine that a protodiastolic gallop sound is either identical with the physiological third heart sound, or merely an exaggeration of the normal phenomenon. Among these are: Conner (1927), White (1928), Wolferth and Margolies (1931, 1933), Melik-Gülnasarian (1932a), Lian and Racine (1933b), Routier and Van Heerswynghels (1935), Arenberg (1941), and Frost (1944, 1949a). On the other hand, Mannheimer (1940), Carlgren (1946), and Luisada and Roitman (1948) believed that the protodiastolic gallop sound, like the presystolic gallop sound already described, differs from the

Table 16. Incidence of protodiastolic gallop rhythm in patients with heart disease: auscultation

| Date | Author | No. of subjects | Clinical condition | Incidence (per cent) |
|---|---|---|---|---|
| 1932 | Melik-Gülnasarian | 3000 | Cardio-vascular disease | 57.0 |
| 1936 | Battro et al. | 20 | Bundle branch block | 10.0 |
| 1940 | Taquini et al. | 15 | Early rheumatic heart disease (children) | 33.3 |
| 1941 | Arenberg | 181 | Organic heart disease | 23.2 |
| 1947 | Cossio & Korn | 4052 | Heart disease | 0.9 |
| 1953 | Brigden & Leatham | 30 | Mitral incompetence | 50.0 |
| 1953 | Mounsey | 33 | Mitral stenosis | 0.0 |

Table 17. Incidence of protodiastolic gallop rhythm in patients with heart disease: phonocardiography

| Date | Author | No. of subjects | Clinical condition | Incidence (per cent) |
|---|---|---|---|---|
| 1940 | Mannheimer | 135 | Congenital heart disease (children) | 43.0 (approx.) |
| 1941 | Arenberg | 181 | Organic heart disease | 20.4 |
| 1945 | Friedländer | 83 | Acute rheumatism (children) | 8.4 |
| 1946 | Carlgren | 5000 (approx.) | Suspected heart disease (children) | 1.6 |
| 1951 | Schoelmerich & Gehl | 35 | Mitral disease | 20.0 |
| 1953 | Brigden & Leatham | 30 | Mitral incompetence | 63.3 |
| 1953 | Mounsey | 33 | Mitral stenosis | 24.2 |

corresponding normal sound in pitch as well as in loudness.

Pathological significance. Thayer (1908) associated protodiastolic gallop rhythm with aortic insufficiency or adherent pericardium. Blacher (1914) found it a common occurrence in severe cases of diphtheria with cardiac decompensation. Of ten patients with protodiastolic gallop described by Conner (1927), eight had rheumatic heart disease, one had coronary thrombosis, and one had essential hypertension. Lian and Racine (1933b) and Frost (1944) considered protodiastolic gallop a sign of left ventricular failure, whereas Evans (1943) and Miles (1951) associated it with right ventricular failure. Rheumatic heart disease was stressed as an aetiological factor by Routier and Van Bogaert (1934b), McKee (1938b), Taquini, Massell and Walsh (1940), Friedländer (1945), and Carlgren (1946). Cossio and Korn (1947) thought that overactivity of the heart, disturbed atrio-ventricular conduction, auricular flutter, or mitral incompetence, could be responsible for protodiastolic gallop. Evans (1949) found that it was often a sign of impending myocardial infarction. Schölmerich and Kirberger (1953) related it to arterial hypertension.

Incidence. Tables 16 and 17 indicate the incidence of protodiastolic gallop rhythm in patients with heart disease, as detected by auscultation or by phonocardio-

graphy. As noted previously, the condition cannot be diagnosed with any certainty by auscultation.

Mannheimer (1940) observed that the incidence of the third heart sound in a group of 135 children with congenital heart disease (approximately 43%) was significantly lower than in a corresponding control group of normal children. Mannheimer believed, as noted above, that the pathological third heart sound differs in quality from the physiological sound.

Prognosis. Few opinions have been expressed in the literature as to the prognostic significance of protodiastolic gallop rhythm. Huchard (1893) believed that a gallop sound early in diastole indicates atrial dilatation without hypertrophy and carries a worse prognosis than does presystolic gallop rhythm.

Hamilton (1941), in a carefully investigated series of 32 patients with diastolic gallop rhythm, found a higher mortality in patients with protodiastolic gallop than in those with presystolic gallop. Mannheimer (1942), Friedländer (1945), and Cossio and Korn (1947) believed that protodiastolic gallop is not a danger sign, but White (1928) and Frost (1944) believed that it has the same ominous significance as presystolic gallop.

Summation gallop rhythm.

Obrastzow (1905) observed that, with tachycardia,

presystolic and early diastolic extra sounds might coincide. Macleod, Wilson and Barker (1932) described a case of summation gallop, identified by auscultation and by phonocardiography; when the heart rate was slowed, the third and fourth heart sounds were separated, giving rise to a quadruple cardiac rhythm. Summation gallop was described also by Wolferth and Margolies (1933) and by Routier and van Bogaert (1934a). Battro, Braun-Menéndez, and Orías (1934) described a series of 22 cases of audible gallop rhythm, 15 of which were identified by phonocardiography as summation gallop.

Other investigators who have described summation gallop include: Houssay (1936), Mannheimer (1942), Forman (1942), Evans (1943), Weber (1944), Friedländer (1945), and Simon (1946).

Tommaselli (1953) described a patient with intermittent 2: 1 heart block, in whom protodiastolic gallop rhythm appeared and disappeared with the heart block. She attributed the gallop rhythm to a summation sound produced by the coincidence of a third heart sound, itself inaudible, with the atrial sound of the following blocked beat.

Aetiology. Since summation gallop is due to coincidence of the third and fourth heart sounds, tachycardia

is usually the most obvious factor in its production; to this may be added, in some cases, delayed atrio-ventricular conduction. From the standpoint of auscultation, an audible summation sound may result from superposition of previously inaudible vibrations corresponding to the third and fourth heart sounds. The identification by phonocardiography of summation gallop cannot be achieved on a single record, although it may be suspected, but depends on separation of the two components of the extra sound when the heart rate is slowed, as it may be by appropriate stimulation of the vagus. This type of quadruple rhythm is 'train wheel rhythm' (Luisada, 1952, 1953).

Pathological significance. Laubry and Pezzi (1926) regarded summation gallop and the corresponding quadruple rhythm as signs of severe myocardial damage. Weber (1944) pointed out that the combination of sinus tachycardia with delayed atrio-ventricular conduction, which tends to produce summation gallop rhythm, occurs especially in toxic myocarditis.

Prognosis. No particular prognostic significance has been attached to summation gallop, except by Mannheimer (1942), who attributed to it the same grave prognosis as he ascribed to presystolic gallop rhythm.

Laubry and Routier (1925) found that the quadruple rhythm due to both third and fourth heart sounds occurring independently presaged death within a few days.

Systolic gallop rhythm.

Aetiology. Potain (1875) described systolic gallop rhythm. He attributed it to an extra sound produced in an arteriosclerotic aorta by the impact of blood ejected from the left ventricle during systole.(Potain, 1894). Cuffer and Barbillion (1887) observed that the systolic extra sound is an inconstant, dull sound, often coinciding with a visible apical impulse and uninfluenced by bodily or respiratory movements. They noted an anacrotic pulse wave on the arterial sphygmogram of these cases and interpreted this as due to contraction of the ventricles in two distinct stages.

Robinson (1908) excluded the systolic extra sound from his cateogries of gallop rhythm.

Amblard (1920) considered systolic gallop rhythm a sign of cardiac insufficiency. He found that in mild cases of cardiac insufficiency this rhythm sometimes appeared only after exercise.

Routier and Van Heerswynghels (1935) and Evans (1943) admitted that the aetiology of the systolic extra sound has still to be explained.

Pathological significance. Cuffer and Barbillion (1887) expressed the view that the systolic extra sound indicates a heart weakened by disease; they incriminated particularly typhoid fever. Potain, as noted above, associated it with arteriosclerosis. Amblard (1920) detected systolic gallop rhythm especially in patients with typhoid fever or diphtheria. Wolferth and Margolies (1931) and Lian and Racine (1933b) held that the systolic extra sound is of extra-cardiac origin, possibly due to pleuro-pericardial adhesions, but, later, Wolferth and Margolies (1940) concluded that systolic gallop rhythm might also be due to an aortic lesion or to arterial hypertension. Evans (1943, 1951), Weber (1944), and Levine (1948) believed the systolic extra sound to have no pathological significance.

Prognosis. Of the authors who attributed some pathological significance to the systolic extra sound, Laubry and Pezzi (1926) held that it is not necessarily a grave omen, and White (1928) believed that the prognosis is better with this type of gallop rhythm than with any type of diastolic gallop.

Opening snap of the mitral valve.

Since the opening snap of the mitral valve is different in character and significance from the other

cardiac extra sounds which have been described, it is not usually regarded as a type of gallop rhythm.

Bouillaud (1841), Skoda (1844), and Duroziez (1862) noted splitting of the second heart sound at the base of the heart in patients with mitral stenosis. Guttmann (1869) observed that the splitting was sometimes best heard at, or just medial to, the cardiac apex. Guttmann (1878) suggested that the extra sound might be due to vibration of the stenosed mitral valve itself, and Sansom (1881) also was of this opinion. Neukirch (1886) noted that the components of the split second sound of mitral stenosis were more widely separated than those of a physiologically split second sound. He attributed the second component to the stenosed valve's resistance to opening and he observed that a triple second sound occurred only in patients with mitral stenosis.

Rouchès (1888) published the first description of the 'claquement d'ouverture de la mitrale', a term which he had learnt from Potain. He found that this extra sound occurred just after the reduplicated second heart sound, when this was present. The opening snap was more abrupt than the extra sound of protodiastolic or presystolic gallop and was audible rather than palpable, but, like left-sided gallop sounds, it was best heard in the region

of the cardiac apex.

Von Wyss (1911) demonstrated the mitral opening snap by phonocardiography. Margolies and Wolferth (1932) showed by phonocardiography that the mitral opening snap is usually synchronous with the summit of the v wave of the jugular phlebogram, which indicates the moment of opening of the mitral valve. It is separated by a short interval from the characteristic mid-diastolic murmur, which usually follows it. Cossio and Orías (1935) and Battro and Braun-Menéndez (1937) confirmed the relationship of the opening snap to the summit of the v wave of the jugular phlebogram. They emphasised that a simultaneous phonocardiogram and jugular phlebogram are essential to distinguish the opening snap from the second part of a split second heart sound and from a third heart sound.

Margolies and Wolferth (1932), Lian (1946), and Mounsey (1953) tried to distinguish the mitral opening snap from other diastolic sounds by its time of occurrence after the first part of the preceding second heart sound, but Messer, Counihan, Rappaport and Sprague (1951) and Wynn (1953) found that this distinction by time alone was often impossible.

Cossio and Orías (1935) and Orías and Braun-Menéndez (1939) found that in cases of mitral stenosis with auri-

cular fibrillation the opening snap might be the only auscultatory sign of mitral stenosis.

The rarity of an opening snap in cases where the predominant mitral lesion is incompetence was noted by Brigden and Leatham (1953), Logan and Turner (1953), and Wood (1954).

Aetiology. Rouchès (1888) and Potain (1894) attributed the opening snap to sudden stretching of the cusps of a stenosed mitral valve when the high pressure difference between atria and ventricles fails to separate them normally in early diastole. This view has been upheld by subsequent investigators.

Wolferth and Margolies (1937) combined roentgenkymography with phonocardiography of a case of mitral stenosis with early calcification of the mitral valve. They confirmed that the mitral opening snap occurred when downward movement of the calcified cusps was checked in early diastole.

Luisada (1953) has sometimes recorded an apparently physiological opening sound of the mitral valve; this occurs earlier than the pathological opening snap and is never audible.

Pathological significance. Most authorities are agreed that the opening snap of the mitral valve is characteristic of mitral stenosis and occurs only in this

Table 18. Incidence of the opening snap of the mitral valve in patients with mitral stenosis

| Date | Author | No. of subjects | Mode of investigation | Incidence (per cent) |
|---|---|---|---|---|
| 1937 | Battro & Braun-Menéndez | 15 | Phonocardiography | 46.7 |
| 1953 | Logan & Turner | 100 | Auscultation | 87.0 |
| 1953 | Mounsey | 33 | Auscultation | 84.8 |
| " | " | " | Phonocardiography | 97.0 |
| 1953 | Wynn | 230 | Auscultation or phonocardiography | 73.0 |

condition. Rouchès (1888) stated that the opening snap indicates a moderate degree of mitral stenosis, with the cusps of the mitral valve still flexible, a view which still finds favour with the most recent investigators.

With the growing popularity of the operation of mitral valvotomy during the last few years, interest has been revived in the physical signs of mitral stenosis. Logan and Turner (1953) and Sellors, Bedford and Somerville (1953) believe that the opening snap is a useful indication of operability, since it is usually absent when the cusps of the mitral valve are very rigid or calcified and when the predominant defect of the mitral valve is incompetence. Wynn (1953) and Wood (1954) confirmed that the opening snap is rare where there is gross calcification of the mitral valve.

Incidence. Margolies and Wolferth (1932) detected an opening snap of the mitral valve by auscultation and by phonocardiography in most of their cases of mitral stenosis. As shown in Table 18, most modern observers have found a high incidence of the mitral opening snap in patients with a clinical diagnosis of mitral stenosis. The diagnosis was confirmed at operation in 15 of the 33 cases described by Mounsey, and in every case of Logan and Turner's series.

Prognosis. Until recent years the mitral opening snap was of little help in prognosis, apart from indicating the stage the disease had reached. Nowadays, however, with the advent of mitral valvotomy, the sign becomes of considerable importance as an indication of the operability of the case.

Summary

In view of the diversity of opinion on the nature and significance of the several types of gallop rhythm, an assessment of the balance of opinion in each case may not be out of place.

Presystolic gallop. In the view of the majority of observers presystolic gallop, due to the fourth heart sound, is the commonest form of gallop rhythm. The extra sound coincides with atrial systole and may be due to an audible contraction of the atria, to the impact against the ventricular wall of blood expelled from the atria, or, probably, to a combination of both these factors. It is probably an exaggeration of the physiological fourth heart sound, which can be recorded in many healthy people but is normally inaudible.

The left ventricle, in particular, appears to be the site of production of the presystolic gallop sound; and most observers associate presystolic gallop rhythm with left ventricular failure. It seems that sudden filling

of the ventricle to its elastic limit is particularly liable to produce the sound. The elasticity of the ventricular wall is reduced by hypertrophy, hypotonicity, or both. Potain and many subsequent observers associated presystolic gallop rhythm particularly with Bright's disease and arterial hypertension. In general it appears to be a sign of a failing heart and of grave prognostic significance.

Protodiastolic gallop. Protodiastolic gallop rhythm is due to the third heart sound occurring in a patient with heart disease. It occurs at the time, shortly after the opening of the atrio-ventricular valves, when the rate of filling of the ventricles is maximal. It has been attributed to the impact of the inflowing blood against the ventricular wall, or to the floating into apposition of the cusps of the atrio-ventricular valves.

Protodiastolic gallop rhythm is usually best heard over the left ventricle. The gallop sound is of the same nature as the physiological third heart sound, but it is more constant in its occurrence and is affected little if at all by respiration or by changes in posture.

Protodiastolic gallop, like presystolic gallop, has been attributed to alterations in the tone of the myocardium. It has been associated with a number of infectious or degenerative diseases, which are known to affect the heart, especially acute rheumatism. It may indicate

incipient or established heart failure but appears to be of less serious prognostic significance than presystolic gallop.

Summation gallop. At rapid heart rates diastole is shortened, bringing the third and fourth heart sounds closer together. When these sounds coincide the result is a summation gallop sound, which may be clearly audible even in cases where each of its components individually lies below the threshold of audibility. Another factor which would tend to produce summation gallop is delayed atrio-ventricular conduction, but this is seldom found in these cases.

The diagnosis of summation gallop depends on separation of the two components by slowing of the heart rate. The prognostic significance of summation gallop is that of its presystolic element.

Systolic gallop. Systolic gallop rhythm depends on the occurrence of a systolic extra sound in a patient with heart disease, but there is no adequate evidence that this extra sound is in any way related to the heart disease. Its aetiology is still obscure.

Opening snap of the mitral valve. The mitral opening snap coincides in time with the opening of the mitral valve early in ventricular diastole. It seems to be due to

sudden tension of the cusps of the stenosed mitral valve when they fail to be adequately separated by the blood flowing into the ventricle, even although the pressure difference between left atrium and left ventricle at this period is abnormally high in cases of mitral stenosis.

The opening snap of the mitral valve has been noted only in cases of mitral stenosis. It does not occur in very early or in very advanced cases. It is of favourable import in the assessment of a case for mitral valvotomy.

## Chapter 8

## Cardiac extra sounds in a series of patients with heart disease

In view of the diversity of opinion as to the incidence and significance of gallop rhythm in patients with heart disease I thought it would be of interest to investigate the phenomenon by phonocardiography. Facilities for this study were obtained through the cooperation of the Medical Superintendent and the Physicians of Stobhill General Hospital.

Phonocardiography was performed on patients with any diagnosis of heart disease in the Medical wards. As far as possible the cases were unselected but a few very ill or moribund patients were not investigated. Patients with arterial hypertension were included in the series only if there was some definite cardiac lesion or electrocardiographic evidence of 'strain'.

The hospital diagnosis based on clinical, electrocardiographic and, in some cases, other special investigations was accepted. In a few cases this conflicted with the findings by phonocardiography; these discrepancies are noted in the captions to the plates of P.C.G.s.

From the series two main diagnostic groups emerge, patients with disease of the mitral valve and patients with arterial hypertension. These groups are sufficient-

ly large to merit separate analysis.

No attempt was made to compare the auscultatory findings of different observers, but my own were noted in each case for comparison with the findings by phonocardiography. Logarithmic and linear P.C.G.s were recorded from each subject. No stethoscopic P.C.G.s were obtained because the observations on normal subjects (Chapter 6) had shown that these were unlikely to yield any information which could not be obtained from the linear records.

The widely held belief that gallop rhythm is of grave prognostic significance is based more on general impressions than on the critical analysis of series of patients. I felt that a series such as that reported here would offer a favourable opportunity for investigation of the subsequent fate of those patients with triple cardiac rhythm and of those without. An attempt was made to find out in every case, 6-9 months after phonocardiography, whether the patient's general health had improved or deteriorated, and to relate this to the presence or absence of gallop rhythm on the P.C.G. It is hoped to continue the follow-up for a period of at least two years but an assessment of even the short-term prognosis should be of value.

In most of the patients in this series, murmurs as well as sounds were heard and were recorded on the logarithmic P.C.G. Since these murmurs, though not directly

relevant to the study of triple cardiac rhythm, are of some clinical interest, they are described in the captions to the plates of P.C.G.s and discussed in Appendix A.

To summarize, the information sought in this group of patients was as follows:

(1) Auscultation.

(a) The incidence of each type of cardiac extra sound.

(b) The relation of each type of cardiac extra sound to any particular type of heart disease.

(2) Phonocardiography.

(a) The incidence of each type of cardiac extra sound on logarithmic phonocardiography.

(b) The relation of each type of cardiac extra sound, recorded on the logarithmic P.C.G., to any particular type of heart disease.

(c) The occurrence of each type of cardiac extra sound, recorded on the logarithmic P.C.G., in patients with disease of the mitral valve and in patients with arterial hypertension.

(d) The relation of cardiac extra sounds, recorded on the logarithmic P.C.G., to the age, sex, and heart rate of the patients.

(e) The incidence of vibrations at the time of occurrence of cardiac extra sounds on linear phonocardiography.

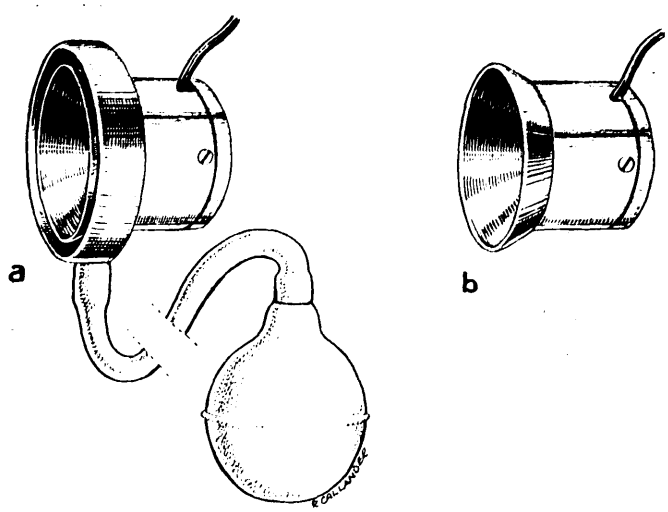


Fig. 13. MICROPHONES FOR APPLICATION TO CHEST: a, WITH SUCTION CHEST-PIECE; b, ALTERNATIVE MODEL FOR ATTACHMENT BY RUBBER BAND.

(f) The relation of cardiac extra sounds on the logarithmic P.C.G. to the corresponding vibrations on the linear P.C.G.

(3) Combined auscultation and phonocardiography.

The degree of agreement between the detection of triple cardiac rhythm by auscultation and by logarithmic phonocardiography.

(4) Follow-up after 6-9 months.

(a) The prognostic significance of gallop rhythm and of the mitral opening snap.

(b) The prognostic significance of cardiac extra sounds in patients with disease of the mitral valve and in patients with arterial hypertension.

Method

Ordinary binaural stethoscopes with simple conical chest-pieces were used for auscultation. A small chest-piece bell (24 mm. internal diameter and 11 mm. deep) was used routinely, and a larger bell (47 mm. internal diameter and 14 mm. deep) was used in a few instances when the heart sounds were faint.

For phonocardiography a crystal microphone, with vulcanite chest-piece, was applied directly to the patient's chest. The internal diameter of the chest-pieces was 36 mm. and the depth 21 mm. The one used routinely had an outer suction ring, as shown in Fig. 13a, for holding

it against the chest. When, for any reason, the suction chest-piece could not be satisfactorily attached to the patient, the alternative chest-piece (Fig. 13b) was applied and held in position by means of a rubber band round the chest.

Two logarithmic P.C.G.s and one linear P.C.G. were recorded from the region of the cardiac apex in each case. The first logarithmic P.C.G. was recorded with the E.C.G., and the second with the jugular phlebogram, as reference tracing. The reference tracing for the linear P.C.G. was the jugular phlebogram.

As in the investigation of physiological cardiac extra sounds (Chapter 6) the filter settings on the pre-amplifier were 4 for logarithmic phonocardiography and 0 for linear phonocardiography.

When the hospital diagnosis, or my own auscultatory findings, indicated the advisability of taking a set of records from some other praecordial area in addition to those from the apex, this was done. For the purpose of analysis, however, only the apical records are considered.

Subjects

The series reported here comprises one hundred in-patients at Stobhill General Hospital, each of whom had definite evidence of heart disease, either primary or secondary. As far as possible the cases were unselected

but, as mentioned above, a very few of the most seriously ill patients were not investigated. Of those on whom phonocardiography was performed, the heart sounds in a few cases were so faint that no satisfactory record could be obtained. When the maximum amplitude of neither the first nor the second heart sound deflections could be raised to 1 cm. on the record without oscillation of the amplifier circuits, the case was excluded from the series.

Details of investigation

Ambulant patients were examined on a bed, and very ill patients were examined on the trolley on which they were brought to the phonocardiograph. The apex-beat was identified and marked, as was any other region of the praecordium from which records were to be obtained.

Phonocardiography was performed with the patient recumbent and normally with the breath held at the end of expiration. A few dyspnoeic or confused patients were unable to hold their breath satisfactorily. In these cases breath sounds were recorded on the logarithmic P.C.G., but these were easily identified; they are labelled on the records where they occur.

As in the investigation of normal subjects, the gain of the amplifier system was so adjusted that, on the final photographic record, the deflections corresponding to the first or second heart sound, whichever was the greater, were not less than 1 cm. and not more than 2 cm. in amp-

Table 19. Diagnoses of 100 patients with heart disease

S = sinus rhythm

F = auricular fibrillation

| Diagnosis | Without cardiac failure | | With cardiac failure | | Total |
|---|---|---|---|---|---|
| | S | F | S | F | |
| Mitral valve disease only | 24 | 7 | 1 | 2 | 34 |
| Combined mitral and aortic valve disease | 11 | 1 | 1 | 1 | 14 |
| Aortic valve disease only | 2 | - | 2 | - | 4 |
| Congenital heart disease | 5 | - | - | - | 5 |
| Angina pectoris | 4 | - | - | - | 4 |
| Bundle branch block | 4a | - | 1 | - | 5a |
| Myocardial infarction | 5 | - | 3 | - | 8 |
| Cor pulmonale | 4b | - | - | - | 4b |
| Arterial hypertension | 19c | 1 | 10d | 1 | 31cd |
| Acute rheumatic carditis | 4e | - | - | - | 4e |
| Myocardial degeneration only | 1 | - | - | - | 1 |

a = Including 1 case of mitral and aortic valve disease and 1 case of angina pectoris
b = Including 1 case of bundle branch block
c = Including 1 case of aortic stenosis, 1 case of angina pectoris, 1 case of bundle branch block, and 1 case of cor pulmonale
d = Including 1 case of mitral stenosis and 3 cases of myocardial infarction
e = Including 2 cases of mitral valve disease and 1 case of mitral and aortic valve disease

litude. Cardiac extra sounds were distinguished from murmurs by the criteria detailed in Chapter 2. For a cardiac extra sound to be considered present, the appropriate deflections had to appear at the same time in the cardiac cycle in at least three successive cycles.

At the follow-up, 6-9 months after the P.C.G. had been recorded in each case, a letter was written to the patient's doctor to ask whether the patient was still alive and whether the general state of health was improved, unchanged, or worse since the date of recording. In the few cases in which no reply was received the patient or a near relative was visited and asked for the information.

Results

The diagnosis and the detailed findings by auscultation and by logarithmic phonocardiography of each subject are given in Table 31 (Volume 2). The records are shown in Plates 11-72 (Volume 2) and details of each case are given in the corresponding captions. The murmurs are discussed in Appendix A.

The diagnoses are given in Table 19, which shows also the incidence of auricular fibrillation and of congestive cardiac failure. Forty-eight of the patients had physical signs of mitral disease, 14 of these with a concomitant lesion of the aortic valve. In 29 patients mitral stenosis was the only valvular defect; 5 of these had

undergone mitral valvotomy but they are included in the group in view of the persistence of some of the classical signs of mitral stenosis.

In a total of 31 patients there was arterial hypertension; 11 of these showed signs of cardiac failure and 16 others showed electrocardiographic evidence of left heart strain.

(1) Auscultation.

(a) I heard cardiac extra sounds in only 5 patients; a third heart sound in 3 patients, a fourth heart sound in one, and an opening snap of the mitral valve in one.

(b) The three patients in whom I heard proto-diastolic gallop rhythm had mitral stenosis with auricular fibrillation; one of them had aortic incompetence as well and another showed signs of congestive cardiac failure. Their ages were 29, 31, and 32 years respectively, an age-group in which the physiological third sound is rarely heard,

The one patient with audible presystolic gallop rhythm was a man aged 60, with no signs of cardiac failure but a history of angina of effort during the preceeding 5 months. The E.C.G. showed evidence of left bundle branch block.

The one patient in whom I heard an opening snap of the mitral valve, a girl aged 14, had mitral stenosis,

and her E.C.G. showed evidence of myocardial disease.

(2) Phonocardiography.

(a) The incidence of each type of gallop rhythm on logarithmic phonocardiography was appreciably higher than that on auscultation. The logarithmic P.C.G. showed a third heart sound in 19 cases, 4 of whom had a fourth heart sound as well. Including these 4, a total of 14 patients had a fourth heart sound on the logarithmic P.C.G. A systolic extra sound was detected in 3 cases, in one of whom the mitral opening snap also was recorded. A total of 9 patients, including this one, had a mitral opening snap.

(b) Twelve of the 15 patients with protodiastolic gallop rhythm had disease of the mitral valve, one of the others was a case of cor pulmonale, and another had congenital pulmonary stenosis, all conditions which subject the right ventricle to undue strain. The remaining patient with protodiastolic gallop had arterial hypertension with electrocardiographic evidence of myocardial degeneration.

Five of the 10 patients with presystolic gallop rhythm had arterial hypertension complicated by myocardial disease or strain. Of the remaining 5, two had acute rheumatism (one with mitral stenosis and the other with mitral and aortic incompetence), one had an anterior myo-

cardial infarction and two had left bundle branch block. All these patients, therefore, had some myocardial damage or strain; in most cases the left ventricle particularly was affected. Only in the two patients with bundle branch block did the P-R interval on the E.C.G. exceed 0.2 sec.: in all the other patients with a fourth heart sound the P-R interval was within normal limits.

Train wheel rhythm, due to the occurrence of both third and fourth heart sounds, was noted only in four old men with left heart strain; in two cases this was the result of aortic valve disease and in the other two it was the result of arterial hypertension.

Triple rhythm due to a systolic extra sound was recorded from two patients. One of these was a case of arterial hypertension with left heart strain; in the other, as a result of bronchial asthma and pulmonary emphysema, the right side of the heart was subjected to strain.

An opening snap of the mitral valve was noted only in patients on whom a diagnosis of mitral stenosis had been made on other criteria. Two of these patients had undergone a clinically successful mitral valvotomy. One patient had quadruple rhythm due to a systolic extra sound and a mitral opening snap.

Taking all the patients with cardiac failure from whatever cause the incidence of gallop or train

Table 20. Incidence of cardiac extra sounds in patients with mitral valve disease and in patients with arterial hypertension: logarithmic phonocardiography

| Diagnosis | Cardiac extra sounds | | | | | | |
|---|---|---|---|---|---|---|---|
| | 3 only | 4 only | 3 + 4 | Sys. only | O.S. only | Sys. +O.S. | None |
| Mitral stenosis only | 4 | 1 | - | - | 7 | 1 | 16 |
| Mitral stenosis and aortic incompetence | 6 | - | 1a | - | 1 | - | 5b |
| Mitral incompetence without stenosis | 2 | 1c | - | - | - | - | 3 |
| Arterial hypertension | 2 | 5 | 2 | 1 | - | - | 21d |

Sys. = Systolic extra sound
O.S. = Opening snap of the mitral valve

a = With mitral incompetence
b = Including 1 case with mitral incompetence and 1 case with mitral incompetence and aortic stenosis
c = With aortic incompetence
d = Including 1 case with mitral stenosis

wheel rhythm was not significantly higher than in the patients without cardiac failure.

(c) The incidence of cardiac extra sounds in patients with mitral valve disease and in patients with arterial hypertension is shown in Table 20.

Of the 48 patients with disease of the mitral valve, 42 had predominant mitral stenosis; in 13 of these there was also a lesion of the aortic valve. Eight of the 29 patients with pure mitral stenosis had a mitral opening snap (one of them with a systolic extra sound as well), four had protodiastolic gallop, and one had presystolic gallop. Two of the patients with an opening snap and three others had previously undergone mitral valvotomy.

Only one of the 13 patients with both mitral stenosis and aortic incompetence had a mitral opening snap, but six had protodiastolic gallop and one had train wheel rhythm. This is a significantly higher incidence of the third heart sound than was found in pure mitral stenosis ($P < 0.05$).

Of the six patients with mitral incompetence unaccompanied by stenosis two had protodiastolic gallop rhythm and the only one with an associated aortic incompetence had presystolic gallop rhythm.

A mitral opening snap occurred in 6 out of a total of 31 cases of mitral stenosis with sinus rhythm

and in 3 out of 11 cases with auricular fibrillation; this is not a significant difference.

In the 31 patients with arterial hypertension, protodiastolic gallop occurred in two cases, presystolic gallop in five, train wheel rhythm in two, and a systolic extra sound in one. This is not a significantly higher incidence of the fourth heart sound than is found in the other cardiac patients. Neither gallop nor train wheel rhythm was related to cardiac failure in this group.

(d) Neither protodiastolic gallop nor presystolic gallop showed any significant correlation with the age, sex, or heart rate of the patients. The systolic extra sound occurred so infrequently that any attempt at statistical analysis would be valueless. The mitral opening snap was not significantly related to the age, sex, or heart rate of the patients in whom it was demonstrated. Eight of the 9 patients with a mitral opening snap were women but, since 31 out of the 42 patients with mitral stenosis were women, the incidence was not significantly higher in women with mitral stenosis than in men with the same condition.

(e) Vibrations were recorded at the time of the third heart sound on the linear P.C.G. of 75 patients, and at the time of the fourth heart sound in 52 patients, 45 of whom also had third heart sound vibrations. Vibrations

Table 21. Agreement between the findings by auscultation and by logarithmic phonocardiography as to the presence or absence of a cardiac extra sound in patients with heart disease

| | | Observer | | Total |
|---|---|---|---|---|
| | | Heard | Not heard | |
| P.C.G. | Recorded | 4 | 36 | 40 |
| | Not recorded | 1 | 59 | 60 |
| Total | | 5 | 95 | 100 |

at the time of the mitral opening snap were recorded on the linear P.C.G. of 9 patients; in 6 of these a mitral opening snap was recorded on the logarithmic P.C.G.

(f) The incidence of the third heart sound on the logarithmic P.C.G.s was significantly related to the incidence of the corresponding vibrations on the linear P.C.G.s ($P < 0.05$): in the case of the fourth heart sound and of the mitral opening snap this relation was highly significant ($P < 0.001$).

(3) Combined auscultation and phonocardiography

Table 21 shows the agreement between the detection of cardiac extra sounds by auscultation and by logarithmic phonocardiography. Since the number of cardiac extra sounds detected by auscultation was so small the group was not further subdivided for statistical analysis.

In 59 patients no cardiac extra sound was heard or recorded. In one patient a cardiac extra sound was heard but none was recorded. In 36 patients a cardiac extra sound, or sounds, was recorded but not heard. In the remaining four the same cardiac extra sound was both heard and recorded. Statistical analysis by Fisher's exact method (Fisher, 1946) shows that the correlation between the findings by auscultation and by logarithmic phonocardiography fails to reach significance.

Table 22. Follow-up of cardiac patients after 6-9 months

| General condition | Cardiac extra sounds | | | | | | |
|---|---|---|---|---|---|---|---|
| | 3 only | 4 only | 3 + 4 | Sys. only | O.S. only | Sys. +O.S. | None |
| Improved | 8 | 5 | 1 | 1 | 2 | 1 | 24 |
| Unchanged | 4 | 2 | - | 1 | 5 | - | 24 |
| Worse | 1 | 3 | 1 | - | - | - | 3 |
| Died | 2 | - | 2 | - | - | - | 8 |
| Untraced | - | - | - | - | 1 | - | 1 |

Sys. = Systolic extra sound
O.S. = Opening snap of the mitral valve

(4) Follow-up after 6 - 9 months.

(a) Ninety-eight patients were traced after a period of 6-9months had elapsed from the time their P.C.G. was recorded. Table 22 shows the result of the follow-up in the patients with the several cardiac extra sounds and in the patients whose logarithmic P.C.G. had shown no cardiac extra sound. The patients with gallop or train wheel rhythm were not significantly worse than the other patients at the end of the follow-up period.

Taking all the cases with a third heart sound, including those with a fourth sound as well, deterioration is not significant compared with the other patients. On the other hand, in the group of patients with a fourth heart sound (including those with a third sound as well), although less than half of the patients had deteriorated in 6-9 months, this was significantly worse than the progress of the other patients ($P < 0.05$). Of the patients with train wheel rhythm due to the presence of both third and fourth heart sounds, two out of the four had died in the period under review and the condition of one of the remaining two had deteriorated.

All the patients with a systolic extra sound and/or a mitral opening snap were either unchanged or improved at the follow-up.

Table 23. Follow-up of patients with mitral valve disease and of patients with arterial hypertension after 6 - 9 months

| Diagnosis | General condition | Cardiac extra sounds | | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | 3 only | 4 only | 3 + 4 | Sys. only | O.S. only | Sys. +O.S. | None |
| Mitral stenosis only | Improved | 3 | 1 | - | - | 2 | 1 | 7 |
| | Unchanged | 1 | - | - | - | 4 | - | 6 |
| | Worse | - | - | - | - | - | - | 1 |
| | Died | - | - | - | - | - | - | 2 |
| | Untraced | - | - | - | - | 1 | - | - |
| Mitral stenosis & aortic incompetence | Improved | 2 | - | - | - | - | - | 1 |
| | Unchanged | 1 | - | - | - | 1 | - | 3b |
| | Worse | 1 | - | - | - | - | - | - |
| | Died | 2 | - | 1a | - | - | - | - |
| | Untraced | - | - | - | - | - | - | 1 |
| Mitral incompetence without stenosis | Improved | 2 | 1c | - | - | - | - | 2 |
| | Unchanged | - | - | - | - | - | - | 1 |
| | Worse | - | - | - | - | - | - | - |
| | Died | - | - | - | - | - | - | - |
| Arterial hypertension | Improved | - | 2 | 1 | 1 | - | - | 5 |
| | Unchanged | 2 | 2 | - | - | - | - | 9d |
| | Worse | - | 1 | 1 | - | - | - | 2 |
| | Died | - | - | - | - | - | - | 5 |

Sys. = Systolic extra sound
O.S. = Opening snap of the mitral valve

a = With mitral incompetence
b = Including 1 case with mitral incompetence and 1 case with mitral incompetence and aortic stenosis
c= With aortic incompetence
d = Including 1 case with mitral stenosis

(b) An attempt was made to assess the prognostic significance of cardiac extra sounds in the patients with mitral valve disease and in those with arterial hypertension (Table 23).

Examining in more detail the fate of the patients with mitral stenosis as the only valvular lesion, most were found to have improved during the follow-up period, whether or not extra sounds had been present. Of the 12 patients submitted to mitral valvotomy (7 of them during the follow-up period) 8 were improved at the follow-up and the general condition of the remaining 4 was unchanged: an opening snap was noted in 5 of these patients and a third heart sound in two. One other patient (with no opening snap) died under anaesthesia before the operation was started. Of the remaining 15 patients who were traced, 6 were reported as improved; of these one had a third and one a fourth heart sound but none had an opening snap: 7 were unchanged; of these one had a third heart sound and two an opening snap: one was worse and one had died; neither of these had any cardiac extra sounds.

Of the three patients with mitral stenosis and aortic incompetence reported as improved at the follow-up, two had protodiastolic gallop: of the five unchanged, one had protodiastolic gallop and one an opening snap: one

who was worse and two who had died had protodiastolic gallop, and the remaining fatal case had train wheel rhythm. This shows, in the patients with combined mitral and aortic valve disease, a significantly worse prognosis in those with a third heart sound ($P < 0.05$).

Of the six patients with mitral incompetence not associated with stenosis, five were improved and one unchanged at the follow-up; two of those improved had protodiastolic gallop. The one patient with both mitral and aortic incompetence had presystolic gallop and was improved at the follow-up.

Of the 31 patients with arterial hypertension, 9 were improved at the follow-up, including two with presystolic gallop, one with train wheel rhythm, and one with a systolic extra sound; 13 were unchanged, including two with protodiastolic and two with presystolic gallop; four were worse, including one with presystolic gallop and one with train wheel rhythm; and the five who died had no cardiac extra sounds.

## Discussion

The series of patients investigated cannot be regarded as typical of heart disease as it occurs in the community, or even as typical of what one might expect to find in the wards of a general hospital. The high proportion of patients with mitral stenosis is attributable

to the policy of the hospital at this time of bringing in known cases of mitral stenosis for assessment of operability and for preoperative and postoperative treatment. The mitral valvotomies were performed elsewhere, most of them at Glasgow Western Infirmary.

The second largest group in the series was that of patients with arterial hypertension and cardiac disease or strain. This also may be ascribed, at least in part, to artificial selection, since some of the patients were brought into hospital for treatment with ganglion-blocking drugs.

Of the five patients with congenital heart disease, two were cases of patent ductus arteriosus admitted prior to operation.

Since the P.C.G.s analysed were recorded at the mitral area it can be assumed that the gallop sounds recorded correspond to 'left-sided gallop' as opposed to the 'right-sided gallop' of some of the older authors. It is probable, however, that at least some of these gallop sounds take origin in the right side of the heart.

(1) Auscultation.

(a) The incidence of audible protodiastolic gallop rhythm in the series (3%) is much lower than was found by most previous investigators (Table 16). The incidence of presystolic gallop rhythm also (1%) is lower

than that found by previous workers (Table 14). Since these findings depend on the notoriously unreliable sense of hearing and were not checked by other observers it would probably be unwise to read much significance into them, but they are considered in relation to the phonocardiographic findings described below.

(b) The three patients in whom protodiastolic gallop rhythm was heard were cases in which the right side of the heart was subjected to strain as a result of mitral stenosis. The only patient in whom a mitral opening snap was heard was another case of this condition. The only patient with audible presystolic gallop rhythm had bundle branch block, a condition which has been associated by several investigators with this type of gallop rhythm.

(2) Phonocardiography.

(a) The incidence of protodiastolic and of presystolic gallop rhythm on logarithmic phonocardiography in this series (15% and 10% respectively) is again lower than that found by most previous investigators (Tables 15 and 17), but the groups observed are not strictly comparable. Since the extra sounds responsible for gallop rhythm are of low frequency it is possible that earlier instruments permitted more low-frequency (inaudible) vibrations to appear on the record. It is possible also

that less stringent criteria were adopted for the identification of cardiac extra sounds.

(b) The fact that protodiastolic gallop rhythm occurred principally in association with lesions which throw an excessive load on the right side of the heart links up with the observations of Evans (1942) and Miles (1951), who regarded it as a sign of right ventricular failure but, in the present series, it was associated with strain rather than with congestive failure. Rheumatic heart disease has been noted as an aetiological factor in protodiastolic gallop by a number of authors: only one of the patients with protodiastolic gallop in this series had acute rheumatism at the time of investigation but the majority had lesions attributable to previous rheumatic involvement of the heart.

Only one of the patients with presystolic gallop rhythm had mitral stenosis and this patient had also an acute rheumatic carditis. Four of the other patients with presystolic gallop rhythm had arterial hypertension, and one had aortic stenosis, both conditions tending to strain the left side of the heart; this agrees with the findings of the majority of previous investigators. Of the five hypertensive patients with presystolic gallop rhythm, three showed evidence of chronic nephritis, which Potain and his school regarded as the commonest cause of

presystolic gallop.

Two of the patients with presystolic gallop rhythm had bundle branch block and another had myocardial infarction, both conditions which are associated in the literature with this type of gallop rhythm. The appearance of a fourth heart sound did not depend on prolongation of the P-R interval: this view is further supported by experimental evidence in Chapter 11.

Train wheel rhythm due to the third and fourth heart sounds has seldom been referred to in the literature. Luisada (1953) associates it with ventricular dilatation or strain. The four patients with train wheel rhythm in the present series were all elderly men suffering from conditions which would throw an excessive load on the left side of the heart; one had aortic stenosis, one aortic incompetence, and the other two showed electrocardiographic evidence of left heart strain.

No diagnosis of summation gallop was made. In 3 cases noted as having a fourth heart sound, summation gallop might have been suspected from the logarithmic P.C.G., but the linear P.C.G. showed a separate group of vibrations corresponding to the third heart sound. In only one case (Patient No. 29) is it felt that summation gallop might have been demonstrated if steps had been taken to slow the heart rate.

(c) From the series of cardiac patients investigated, two main diagnostic groups emerge, mitral disease and arterial hypertension. In the former group the mitral opening snap was found only in cases of mitral stenosis and was commonest when this was the only lesion: when there was an associated mitral incompetence the third heart sound was of commoner occurrence than the mitral opening snap. This supports the view of Logan and Turner (1953) and Sellors, Bedford, and Somerville (1953) that the opening snap is a favourable indication for mitral valvotomy. Although protodiastolic gallop rhythm was commoner in combined mitral and aortic valve disease than in pure mitral stenosis, it occurred also in two of the five patients with pure mitral incompetence: the suggestion that the pathological third heart sound is associated particularly with right ventricular strain is not discredited.

In patients with arterial hypertension the fourth heart sound was of common occurrence, either in presystolic gallop or in train wheel rhythm but it was not significantly commoner in this group than in the other cardiac patients. The fourth heart sound was not an indication of cardiac failure, either in patients with arterial hypertension or in the series of cardiac patients

as a whole. Gallop rhythm is seldom found in advanced cardiac failure; it seems to be a sign of strain rather than of decompensation.

I intend in the future to investigate a large series of patients with arterial hypertension, including those with no clinical or electrocardiographic evidence of cardiac strain, in order to ascertain the relation of gallop rhythm to cardiac strain and to cardiac failure in these cases and to note any appearance or disappearance of gallop rhythm with treatment or with spontaneous fluctuations in blood pressure.

(d) Previous investigators have found no significant association between gallop rhythm or the mitral opening snap and the age or sex of the patients concerned: my observations are in agreement with this. On the other hand, train wheel rhythm in this series occurred only in men. Unlike some previous investigators I did not find that tachycardia was at all essential to the production of gallop rhythm.

(e & f) As might be expected, the incidence of deflections corresponding in time to extra cardiac sounds was higher on linear than on logarithmic P.C.G.s, although there was a significant correlation between them. These deflections were not present in every case as they were on the stethoscopic and linear records of normal subjects

(Chapter 6). A greater range of intensity of these vibrations is found with heart disease than in normal subjects: at one extreme the fourth heart sound or mitral opening snap appears in the logarithmic P.C.G.; at the other extreme, deflections corresponding to the third or fourth heart sound are absent even from the linear P.C.G.

As noted in Chapter 6, there is normally no quiescent base-line during systole on the linear P.C.G., so no deflections during this period can be identified as corresponding to a definite systolic extra sound.

(3) Combined auscultation and phonocardiography.

The detection of cardiac extra sounds on auscultation showed no significant agreement with their detection by logarithmic phonocardiography; since these sounds were heard in only five patients such correlation could hardly have been expected. The phonocardiograph, as usual, proved a more sensitive means of detection than the human ear, in spite of the limitation of its sensitivity by the electrical filters in the preamplifier. The factors involved have already been discussed in Chapter 6.

(4) Follow-up after 6-9 months.

(a) The bad prognostic significance attributed in the past to presystolic gallop rhythm is borne out by the subsequent history of the patients investigated in

this series. Even after the short period of 6-9 months had elapsed from the time the P.C.G. was recorded, the condition of patients whose records had shown presystolic gallop rhythm had deteriorated significantly relative to that of the other patients. This supports the view that presystolic gallop rhythm is of grave prognostic significance. It will be of interest to ascertain, in the future, the condition of these patients two years after the original examination.

The same deterioration was not observed in the case of those patients with protodiastolic gallop rhythm. This supports the contention of Mannheimer (1942), Friedländer (1945), and Cossio and Korn (1947) that protodiastolic gallop is not an ominous sign, in contrast to the belief of Huchard (1893), Hamilton (1941), and White (1951) that it is of graver prognostic significance than is presystolic gallop. In the younger patients, of course, protodiastolic gallop may be quite physiological: in the presence of heart disease, triple rhythm due to the third heart sound is arbitrarily designated protodiastolic gallop.

Although two out of the four patients with quadruple (train wheel) rhythm had died during the follow-up period, this sign does not seem to be so grave as was suggested by Laubry and Routier (1925), who affirmed that

it presaged death in a few days. The two deaths which occurred in my series occurred the one 3 months and the other 7 months after the quadruple rhythm had been detected.

Systolic gallop rhythm appeared to have no ominous significance, a finding which agrees with that of most recent observers. It may be mere coincidence when a systolic extra sound occurs in a patient with heart disease.

The mitral opening snap confirms a diagnosis of mitral stenosis but its prognostic significance appears to be merely that of the concomitant mitral stenosis.

(b) All the patients with a mitral opening snap who were traced after 6-9 months were either unchanged or improved, as were most cases of mitral stenosis without an opening snap. The improvement in this series was not related to the operation of mitral valvotomy since most of the patients reported as improved had not been operated upon.

Although it was recorded only in patients with mitral stenosis, it is doubtful whether the mitral stenosis itself can be the cause of the opening snap, since two of the patients showing it had undergone apparently successful mitral valvotomy. In a larger series, Wood (1954) found that the opening snap persisted in most cases after

mitral valvotomy. Deformity of the valve orifice rather than actual narrowing may be the explanation of the opening snap; this deformity may even be increased by successful division of the commissures.

In the patients with mitral stenosis and aortic incompetence protodiastolic gallop rhythm was a bad prognostic sign: of the patients with pure mitral incompetence both those with protodiastolic gallop rhythm improved: in neither of these groups was there any case of presystolic gallop but the one patient with pure mitral and aortic incompetence showed this phenomenon. This is negative support for the view that presystolic gallop depends on left ventricular strain.

An interesting and unexpected finding in the patients with arterial hypertension was that in none of those who died during the follow-up period had cardiac extra sounds been detected. Four out of the five fatal cases showed signs of congestive cardiac failure at the time of the examination, and it is quite possible that gallop rhythm, previously present, may have disappeared with the onset of cardiac failure. The whole question of the incidence and significance of gallop rhythm in arterial hypertension is due for reinvestigation now that the facilities of modern phonocardiography are available.

Summary

In a series of 100 cardiac patients, protodiastolic gallop rhythm was heard in 3 cases, presystolic gallop rhythm in one, and an opening snap of the mitral valve in one. The patients with protodiastolic gallop had rheumatic heart disease and the patient with presystolic gallop had bundle branch block. The opening snap occurred in a patient with mitral stenosis.

On logarithmic phonocardiography, protodiastolic gallop rhythm was recorded in 15 patients and presystolic gallop in 10. Another 4 patients had train wheel rhythm, due to the presence of both third and fourth heart sounds. A systolic extra sound was detected in 3 cases and a mitral opening snap in 9, one of whom had also a systolic extra sound.

In general, protodiastolic gallop was associated with right heart strain, and presystolic gallop or train wheel rhythm with left heart strain: none of these phenomena was a sign of cardiac failure. The mitral opening snap was detected only in patients with mitral stenosis.

The mitral opening snap occurred more commonly in patients in whom mitral stenosis was the only valvular lesion; it was rarer when the mitral stenosis was accompanied by aortic incompetence and it did not occur at all

when the predominant mitral lesion was incompetence. Protodiastolic gallop rhythm was commoner in combined mitral and aortic valve disease and in mitral incompetence than in pure mitral stenosis.

Presystolic gallop rhythm was of common occurrence in patients with arterial hypertension and left ventricular strain: it was not a sign of cardiac failure in these patients.

Neither gallop rhythm nor the mitral opening snap was related to the age, sex, or heart rate of the patients.

Vibrations were recorded at the time of the third heart sound on the linear P.C.G. of 75 patients, and at the time of the fourth heart sound in 52 patients, 45 of whom had also third heart sound vibrations. Vibrations at the time of the mitral opening snap were recorded on the linear P.C.G. of 9 patients. The appearance of cardiac extra sounds on the logarithmic P.C.G. was significantly related to the presence of the corresponding vibrations on the linear P.C.G.

There was no significant agreement between the detection of cardiac extra sounds by auscultation and by logarithmic phonocardiography.

Presystolic gallop rhythm, as detected by logarithmic phonocardiography, was found to be of grave prognostic significance. Protodiastolic gallop rhythm did not, as a rule, have this ominous significance.

Most of the patients with mitral valve disease were either unchanged or improved after 6-9 months. The progress of those with pure mitral stenosis was not significantly related to the presence or absence of cardiac extra sounds at the original investigation or to the performance of mitral valvotomy. In the patients with combined mitral and aortic valve disease, protodiastolic gallop was a bad prognostic sign.

In the patients with arterial hypertension, neither gallop nor train wheel rhythm had any grave prognostic significance: none of those who died during the next 6-9 months had any cardiac extra sounds at the original investigation.

# PART V

## EXPERIMENTAL STUDY OF TRIPLE CARDIAC RHYTHM

"Our knowledge of the cause of the third sound is so meager as to make valueless any speculation with regard to it."

J.A.E. Eyster (1911)
The time relations of the venous pulse and the heart sounds.
J. exp. Med., vol. 14 p. 601

" --- but why think, - why not try the experiment?"

John Hunter (1775)
Letter to Edward Jenner

## Chapter 9

## The mechanism of production of cardiac extra sounds

Many views on the aetiology of cardiac extra sounds have been expressed (Chapter 7), but the number of authors hazarding an opinion as to the mechanism of production of these sounds is greatly in excess of the number of those who have sought any experimental verification of their ideas.

### Experiments on man

Experimental work on human subjects has necessarily been limited in scope. Most authors have been content to observe the incidence and nature of the extra sounds as detected by auscultation or by the various modes of phonocardiography. The influence of the rate of return of venous blood to the heart on cardiac extra sounds has, however, been studied by a few investigators. Gubergritz (1926) increased the venous return by pressure on the abdomen or slowed the heart by pressure on the eyeballs; either manoeuvre tended to reveal a third heart sound or to enhance one already present. Thayer (1910) and Bridgman (1915) made the third heart sound appear by raising the subject's limbs. Schölmerich and Gehl (1951) put their subjects on a tilting table and found that the third sound was enhanced when the head was lowered and disappear-

ed when it was raised. Engelbertz (1953) obtained the same result in the case of the fourth heart sound. Lian and Hubert (1948a,b) found that presystolic gallop rhythm appeared after exercise in a number of cardiac patients but did not appear in any of the normal subjects submitted to the same test.

Experiments on animals

Animal experiment is limited by the variable occurrence of cardiac extra sounds in different species. Although the first and second heart sounds of other mammals resemble those of man (British Association, 1840; Halford, 1860), this is not always the case for extra sounds. D'Espine (1882) found it very difficult to produce cardiac extra sounds in the dog, although they were normally present in the horse. Thayer (1908) heard the third heart sound in some of his dogs, but Lewis (1913b) did not hear it in any of his. Melik-Gülnasarian (1932a) heard it at the chest wall in not more than 4% of his dogs and at the apex of the exposed heart in 25%.

Eyster (1911) recorded the third heart sound from dogs when the heart rate was slow. Boyer (1942) recorded it as a spontaneous phenomenon in some dogs; in others it could be produced by rapid infusion of saline or by asphyxia. Luisada and Mautner (1943) detected the third

sound by stethoscopic phonocardiography in some dogs and succeeded in producing it in others by injection of digitalis, ouabaine, or adrenaline. The fourth sound was usually obvious when the animal's heart rate was slow, especially if atrio-ventricular conduction were delayed by digitalis, moryl (carbamylcholine chloride), or mecholyl (acetyl-β-methyl choline), or by electrical stimulation of the vagus. Summation gallop was observed when tachycardia had been induced by the injection of adrenaline. Luisada, Weisz, and Hantman (1944), by stethoscopic phonocardiography, recorded third heart sound vibrations from dogs and donkeys but failed to record them from other domestic animals except, rarely, the horse. They recorded vibrations at the time of the fourth heart sound in dogs, oxen, swine, sheep, and guinea-pigs. Smith (1944) heard and recorded the third sound from the exposed left ventricle of a number of his dogs.

Fogelson (1932) and Braun-Menéndez and Solari (1936a,b) divided or crushed the atrio-ventricular bundle of the dog heart and observed the isolated fourth heart sound thereafter. Delayed atrio-ventricular conduction, achieved by partial destruction of the atrio-ventricular bundle or by stimulation of the left vagus nerve, gave rise to presystolic gallop rhythm.

Halford (1860) found that if both venae cavae and the pulmonary veins of a dog were occluded no sound was audible from the exposed heart, although it continued to beat. Krehl (1889) found that the first and second heart sounds could still be heard from the exposed dog heart after the venae cavae had been occluded. Wiggers (1919) demonstrated by 'direct' phonocardiography a reduction in amplitude of the first and second heart sounds in the dog when the posterior vena cava was clamped: with rapid intravenous infusion there was no increase in amplitude of the first sound but a definite increase in amplitude of the second sound.

Dock (1933) found that the first heart sound of the dog was diminished, but not abolished, by occlusion of the venae cavae, and Smith, Gilson and Kountz (1941) found that it was abolished by tightening a ligature round the atrio-ventricular sulcus.

Third heart sound

The mechanism of production of the third heart sound has been variously explained by various authors, as detailed in Chapter 7. Potain (1894) ascribed the protodiastolic gallop sound to vibrations set up in the ventricular wall by the impact of blood flowing in from the atria. He believed that this would occur particularly if the

ventricular wall were unduly rigid or unduly flaccid. Among others who believed that the third heart sound is initially due to vibrations of the ventricular wall are Gubergritz (1926), Leonhardt (1932), Braun-Menéndez and Orías (1934), Mannheimer (1940), Lian (1946), and Orías (1949). Boyer (1942) showed by phonocardiography of the exposed dog heart that a light tap on the ventricle produced vibrations like those of the third heart sound.

Ohm (1921), Routier and Van Bogaert (1934b) and Carlgren (1946) sustained Potain's further argument that the third sound vibrations of the ventricular wall occur more readily when there is deviation from the normal ventricular tone. Carlgren produced myocardial lesions in rabbits by the injection of sparteine or caffeine, followed by adrenaline; with the reduction in myocardial tone thus effected, a cardiac extra sound, usually the third heart sound, appeared in most of the animals.

The other popular theory for the origin of the third heart sound is that it is primarily due to vibrations of the cusps of the atrio-ventricular valves. Gibson (1907), Hirschfelder (1907), Sewall (1909), Henderson and Johnson (1912), Pezzi and Lutembacher (1913), Bridgman (1915), Lewis (1920), Levine and Harvey (1949), and Little (1951) held that these vibrations are due to the valve cusps floating into apposition as the ventricles fill, to be

separated again later by atrial systole. Henderson and Johnson (1912) demonstrated on the excised ox heart that valve closure could be effected by breaking the jet of blood from atria into ventricles in the absence of any ventricular contraction, and they showed that this is a highly efficient method of valvular closure since it prevents the initial regurgitation inevitable if the valves are closed by static back pressure. They affirmed that the third heart sound is due to apposition of the atrio-ventricular valve cusps, caused by sudden slowing of the flow of blood into the ventricles at the end of the rapid-filling phase of diastole. Little (1951) concluded from intra-atrial and intra-ventricular pressure curves that the atrio-ventricular valves do close in early diastole.

Thayer (1908, 1910), von Wyss (1911), Holt (1927), and Bramwell (1935b) associated the third sound with vibrations due to the sudden opening of the atrio-ventricular valves, but many investigators have since shown that the third sound occurs an appreciable time after the opening of these valves and that an obvious opening sound of the mitral valve is characteristic of mitral stenosis (Chapter 7).

The third heart sound has also been ascribed to vibrations of the semilunar valves (Einthoven, 1907b; Hess, 1915; Tigerstedt, 1921), to myocardial activity (Bard, 1922; Smith, 1944), and to impact of the heart against the

chest wall (Boyer, Eckstein & Wiggers, 1940). There is no experimental support for Einthoven's theory, but some for that of Bard and Smith that the vibrations may be due primarily to myocardial activity. Working on the exposed dog heart, Smith introduced through the mitral valve into the left ventricle a disc attached to a rod; this could then be drawn back to close the valve. When the venous inflow to the ventricle was arrested, either by occluding the great veins or by blocking the atrio-ventricular valve with the special device, Smith found that the first and third heart sounds persisted but the second sound disappeared. The last theory, that of Boyer et al., that the third heart sound is due to impact of the heart against the chest wall, is adequately refuted by those observers including Wiggers (1923) and myself, who have listened to or recorded this sound from the exposed heart.

Fourth heart sound

In his classic description of presystolic gallop rhythm, Charcelay (1838) attributed the extra sound, which we should now call the fourth heart sound, to audible contraction of a hypertrophied atrium. Autopsy on his patient revealed the expected atrial hypertrophy. Charcelay's theory was supported by Johnson (1876), Krehl (1889), Benjamins (1914), and Mond and Oppenheimer (1929). Ben-

jamins heard a loud fourth heart sound when the special end-piece of his stethoscope was passed down the subject's oesophagus to behind the atria.

Other observers, while believing the fourth heart sound to be due to muscular contraction of the atria, held that the most important factor in revealing the sound is delayed conduction of the cardiac impulse from atria to ventricles so that the ensuing first sound is delayed and does not mask the atrial sound. This view was expressed by von Wyss (1911), Reid (1921), Melik-Gülnasarian (1932b), Fogelson (1932), and Macleod, Wilson and Barker (1932). Melik-Gülnasarian failed to produce a fourth heart sound in dogs when atrio-ventricular conduction was delayed by vagal stimulation or by physostigmine, but Fogelson succeeded in demonstrating it in dogs when complete heart block was induced by division of the bundle of His. Delayed atrio-ventricular conduction is a rare finding in human cases of gallop rhythm (Bramwell, 1935b).

Potain (1894, 1900b) believed that presystolic gallop is due to the impact of inflowing blood on the ventricular wall during atrial systole. Bridgman (1914), Bard (1922), Braun-Menéndez and Orías (1934), and Bramwell (1935b) all supported Potain's theory, but without any experimental evidence.

Braun-Menéndez and Solari (1936a,b) induced complete heart block in dogs by crushing the bundle of His with forceps or by strong stimulation of the left vagus nerve. They noted three components of the isolated atrial sound, the first registrable from the atria and attributable to atrial contraction, the second registrable from the ventricles and coinciding with maximal dilatation, the third registrable from both atria and ventricles and attributable to a sudden partial closure of the atrio-ventricular valves. Dean (1916) had shown in the isolated beating heart of the cat that incomplete closure of the mitral cusps occurs at the end of atrial systole, before ventricular systole commences.

Other explanations of presystolic gallop rhythm mentioned in Chapter 7 include: asynchronous contraction of the two ventricles (Walshe, 1873; Barr, 1877; Bindley, 1878; Peter, 1883); two-stage ventricular contraction (Cuffer & Barbillion, 1887; Bouveret & Chabalier, 1889); and audible contraction of the papillary muscles an appreciable time before that of the ventricular wall (A. Chauveau, 1900; H. Chauveau, 1902). The first of these theories was disproved by Katz (1925) who demonstrated by careful experiment on dogs that asynchrony of the ventricles is of very minor degree; Holt (1927) pointed out

that this asynchrony would give rise to splitting of the first heart sound rather than to presystolic gallop rhythm. There is no evidence for two-stage contraction of the ventricles. A. Chauveau, by means of a catheter introduced through the aorta into the left ventricle of the horse, detected a ventricular pressure wave between the end of atrial and the beginning of ventricular systole; he ascribed this wave to contraction of the papillary muscles. But even if this be true there is still no adequate evidence that the phenomenon is related to the fourth heart sound.

Summation sound

Obrastzow (1905) observed that, in tachycardia, presystolic and protodiastolic extra sounds might coincide; this produces what is now called the summation sound. Separation of the two components of the summation sound by slowing the heart has been demonstrated in human subjects by Macleod, Wilson, and Barker (1932). Luisada and Mautner (1943) induced a summation sound in dogs by the injection of epinephrine to cause tachycardia.

Systolic extra sound

A protosystolic extra sound was described by Cuffer and Barbillion (1887), Bouveret and Chabalier (1889), and Laubry and Pezzi (1926), who all ascribed it to abnormal separation of the muscular and valvular components of

the first heart sound. None of these authors cited evidence in support of their contentions.

A mesosystolic extra sound was described by Potain (1894) and Wiedemann (1914), who both attributed it to rapid ejection of blood into the aorta, in cases where the elasticity of the aortic wall was reduced. This does not explain all the observed cases of the systolic extra sound, whose cause, in most cases, is still to seek.

Mitral opening snap.

Rouchès (1888) described the opening snap of the mitral valve and attributed it to sudden stretching of the cusps of a stenosed valve when they fail to open completely in early diastole. This explanation has been sustained by those subsequent observers, including Potain (1894), Wolferth and Margolies (1937), and Luisada (1953), who have not confused the mitral opening snap with either the third heart sound or the second part of a split second sound. Experimental investigation of the phenomenon is restricted by the difficulty of producing an artifidal mitral stenosis in experimental animals.

Summary

The actual mechanism of production of the several cardiac extra sounds is still in doubt.

Most observers consider the third heart sound to be primarily due to vibrations of the ventricular wall,

caused by the sudden impact of blood from the atria in early diastole, or to vibrations of the cusps of the atrio-ventricular valves when they float into apposition as the ventricle fills, or to both these factors.

The fourth heart sound has been ascribed to audible muscular contraction of the atria and to vibrations of the ventricular wall set up by the inrush of blood at atrial systole. Probably both these factors play a part.

The summation sound is, by definition, due to superposition of the third and fourth heart sounds.

The cause of the systolic extra sound is not known.

The mitral opening snap is probably due to vibrations set up in the cusps of a stenosed mitral valve when its opening in early diastole is suddenly checked.

## Chapter 10

## The effect of variations in the rate of filling of the heart on the amplitude of cardiac extra sounds in man

The third heart sound is usually ascribed , as noted in Chapters 5 and 9, to rapid filling of the ventricles in early diastole, whatever part of the heart may be primarily set into vibration. It is often referred to as the 'rapid-filling' sound because it occurs at the time in the cardiac cycle when the ventricles are filling most rapidly. If the sound is in fact dependent on this, one might expect it to become louder if the rate of ventricular filling were increased, and fainter if it were diminished. Modern phonocardiography makes it possible to demonstrate objectively any such increase or diminution in the amplitude of heart sounds.

This investigation was designed to illustrate any relationship in man between the amplitude of the third heart sound and the rate of filling of the heart (Sloan & Wishart, 1952, 1953a). It was hoped also to correlate with this any observed changes in other cardiac extra sounds which might be detected in the subjects of the experiment.

Logarithmic phonocardiography was performed as representing objectively the relative 'loudness' of the heart

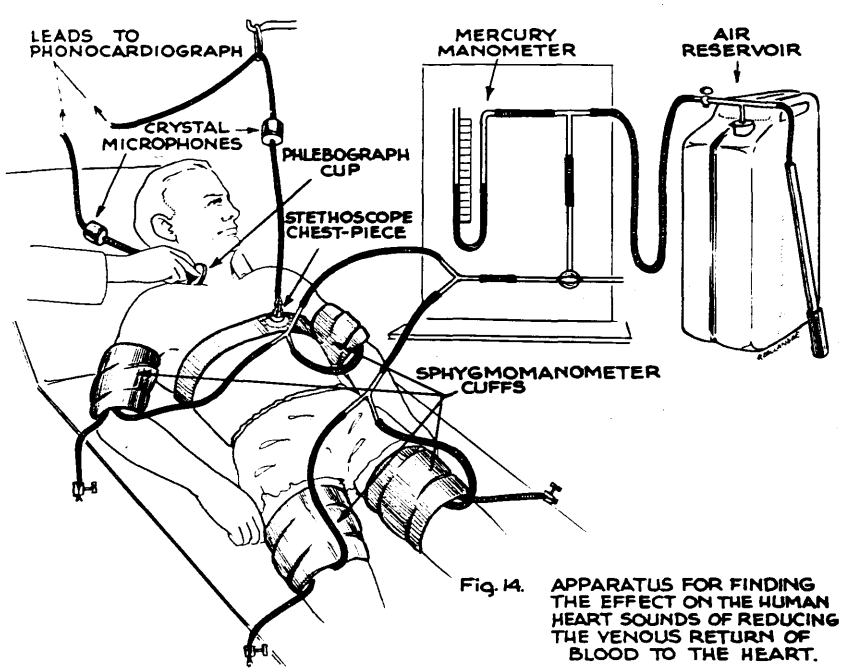


Fig. 14. APPARATUS FOR FINDING THE EFFECT ON THE HUMAN HEART SOUNDS OF REDUCING THE VENOUS RETURN OF BLOOD TO THE HEART.

sounds in contrast to the actual amplitude of their vibrations. The effects of reducing the venous return to the heart and of increasing it were studied on the same group of subjects.

Method

Records were taken with the subject supine on a couch. A standard stethoscope chest-piece, as described in Chapter 6, was attached by means of a rubber band round the chest to the subject's praecordium in the region of the apex-beat. The chest-piece was connected by a length of rubber tubing to a crystal microphone, leading to the sound channel of the phonocardiograph. Only logarithmic P.C.G.s were recorded (filter position 4 on the preamplifier).

Five records were obtained from each subject. On each one the amplitude of the deflections corresponding to each sound was measured over five cardiac cycles and the mean amplitude calculated.

The first two P.C.G.s on each subject were preliminary controls, one with the E.C.G. and the other with the jugular phlebogram as reference tracing; they were taken after the subject had been lying down for approximately five minutes. After the preliminary recording, the E.C.G. leads were removed.

Sphygmomanometer cuffs were then applied to both thighs and both arms, as shown in Fig. 14, and were in-

flated to the diastolic blood pressure of the individual. The use of an air reservoir permitted this inflation to be performed rapidly. Ebert and Stead (1940) and McMichael and Sharpey-Schafer (1944a,b) have shown that the return of venous blood to the heart is considerably reduced by the application of these 'venous tourniquets'.

The cuffs were maintained at diastolic blood pressure for five minutes, at the end of which time a further P.C.G. was recorded. The pressure was then released and five minutes were allowed to elapse for the circulation to return to normal before another control record was taken.

Finally the subject performed a cycling movement of the legs in the air for two minutes, a procedure which accelerates considerably the rate of filling of the heart. A final record was taken immediately on completion of this exercise.

The gain of the amplifiers was constant during each set of experiments but it was of necessity altered for different subjects owing to the wide individual variation in intensity of the heart sounds. Consequently the changes in amplitude of the P.C.G. deflections with the experimental procedures described were comparable within each individual experiment but were not comparable from subject to subject. For purposes of analysis the changes in amplitude are therefore expressed in terms of percentage

Table 24. Effect of venous occlusion on the amplitude of the heart sounds in 16 healthy young adults: logarithmic phonocardiography

| Sound | Mean change (per cent) | Standard error of mean change | Significance |
|---|---|---|---|
| First | + 3.1 | ± 8.2 | Not significant $P > 0.7$ |
| Second | + 8.1 | ± 8.9 | Not significant $P > 0.3$ |
| Third | - 72.0 | ± 4.8 | Highly significant $P < 0.001$ |
| Fourth (10 subjects) | - 41.5 | ± 17.2 | Significant $P < 0.05$ |

Table 25. Effect of exercise on the amplitude of the heart sounds in 14 healthy young adults: logarithmic phonocardiography

| Sound | Mean change (per cent) | Standard error of mean change | Significance |
|---|---|---|---|
| First | + 172.4 | ± 38.1 | Highly significant $P < 0.001$ |
| Second | + 14.2 | ± 8.2 | Not significant $P > 0.1$ |
| Third | + 72.1 | ± 19.3 | Highly significant $P < 0.01$ |
| Fourth (9 subjects) | + 46.1 | ± 12.3 | Highly significant $P < 0.01$ |

increase or decrease.

Subjects

The subjects selected for the investigation were 16 healthy young adults, undergraduates and apprentice technicians, each of whom showed an obvious third heart sound on the logarithmic P.C.G. The group consisted of 10 men and 6 women, and their ages ranged from 16 to 24 years.

Results

Plate 73 (Volume 2) shows a typical set of records from one of the subjects.

The effect of venous occlusion and of exercise on the amplitude of the heart sounds of each of the subjects is recorded in Tables 32-35 (Volume 2) and the effect on heart rate is noted in Table 36. The mean percentage change in amplitude of the several heart sounds, and the significance of these changes, are shown in Tables 24 and 25.

With venous occlusion the third sound was either abolished or diminished in every subject. The mean decrease was highly significant.

Of the 10 subjects, whose preliminary records showed a fourth heart sound, this was abolished or diminished with venous occlusion in 8, and in the remaining 2 the amplitude was unchanged. The mean decrease was significant.

The effect of venous occlusion on the first and second

heart sounds was negligible, the individual changes being small and variable.

The effect of exercise was observed in only 14 of the subjects; in the remaining two the chest-piece was dislodged during the exercise. In 10 out of these 14 subjects the third sound was increased in amplitude after exercise; the remaining four showed no change in the amplitude of the sound. The mean increase for the whole 14 subjects was highly significant.

The effect of exercise on the fourth heart sound was studied on the 9 subjects, who showed a fourth heart sound in the control record. In 7 of these the fourth sound was increased in amplitude after exercise, and in the remaining two it was unchanged. The mean increase was highly significant. Two other subjects, who showed no fourth heart sound in the control records, developed one after exercise.

The first heart sound was increased in amplitude after exercise in every subject, giving, over the series, a highly significant increase. The second sound showed a variable response; the mean change was positive but not significant.

The change in heart rate with venous occlusion was variable and of minor degree. The rate was increased after exercise in every case but one: this increase was

highly significant ($P < 0.001$). The individual alterations in heart rate with exercise did not correspond to the individual changes in amplitude of the third or of the fourth heart sound.

No summation or systolic extra sound was observed in any of the records.

Discussion

The third and fourth heart sounds were consistently diminished by reduction in the rate of filling of the heart, whereas changes in the first and second sounds were more variable. An increase in the rate of filling of the heart augmented the amplitude of the first heart sound in every case and of the third and fourth sounds in most cases, but the effect on the second sound was more variable. These findings suggest that the third and fourth heart sounds are related to the rate of filling of the heart. The close similarity between the changes in the third and in the fourth heart sounds with the experimental procedures adopted suggests a similar mechanism of production of these sounds, possibly the sudden filling of the ventricle which occurs in protodiastole and again in presystole.

Since there is no correlation between heart rate and the amplitude of individual third and fourth heart sounds, the occasional failure of these sounds to increase after

exercise cannot be explained by a reduced input per beat due to tachycardia. It seems therefore that other circulatory changes associated with muscular exercise, in addition to the rate of inflow of blood to the ventricles, must influence the production of the third and fourth heart sounds.

The balance of evidence seems to favour the relationship of the third and fourth heart sounds to the rate of filling of the heart, but the actual mechanism is still in doubt.

Summary

In 16 healthy human subjects with a well-marked third heart sound on the logarithmic P.C.G. the deflections corresponding to this sound were reduced in amplitude in every case when the venous return from the limbs was obstructed.

In the 10 subjects, whose preliminary P.C.G. showed a fourth heart sound, the deflections corresponding to this sound were reduced in amplitude in 8 cases and unchanged in 2 cases when the venous return was reduced.

In 10 out of 14 subjects the third sound was increased in amplitude after muscular exercise; in the remaining 4 subjects the deflections were unchanged.

In 7 out of 9 subjects the fourth sound was increased in amplitude after muscular exercise; in the remaining

2 subjects the deflections were unchanged. In 2 subjects, with no fourth heart sound at rest, the sound was present after exercise.

Changes in the amplitude of the first and second heart sounds were less consistent.

There was no relation between the changes in amplitude of the third and fourth heart sounds and the changes in heart rate with the experimental procedures described.

## Chapter 11

## The influence of various factors on cardiac extra sounds in the dog

The human experiments described in Chapter 10 indicated a relationship between the amplitude of the third and fourth heart sounds and the rate of filling of the heart. They failed, however, to show which part of the heart was primarily thrown into vibration in the production of these sounds.

I hoped, by animal experiments, to confirm the observations which I had made on human subjects and to obtain more information about the actual mechanism of production of the third and fourth heart sounds. In preliminary trials, dogs, cats, and rabbits were used, but I found the hearts of cats and rabbits inconveniently small for the experimental procedures adopted, so the experiments thereafter were performed on dogs.

The first experiments described in this chapter were planned to investigate more fully the relationship between the third and fourth heart sounds and the rate of venous inflow to the heart. The control of venous inflow was achieved by more direct means than in the human experiments, and the heart sounds were recorded directly from the surface of the heart.

The incidence of cardiac extra sounds was studied on

logarithmic and stethoscopic P.C.G.s. No linear records were made in this investigation since attachment of a heavy microphone directly to the heart was not considered practicable. In some cases records were taken from both right and left ventricles in order to find whether the sounds were louder from one side of the heart than from the other.

Since some previous investigators, as noted in Chapter 9, had found that cardiac extra sounds might be made manifest in the dog by vagal stimulation or by the administration of certain drugs, we repeated these procedures with some of our animals. The result of the experiments described above have already been published (Sloan & Wishart, 1953c).

Finally, in an attempt to elucidate the mechanism of production of the third heart sound I attempted to produce a similar sound artificially in the arrested heart by tapping the ventricle with a glass rod and by rapid filling of the ventricle with saline.

To summarize, the information which was sought in this series of investigations was as follows:

(a) The incidence of cardiac extra sounds and of extra sound vibrations on logarithmic and on stethoscopic phonocardiography respectively.

(b) The relation of cardiac extra sounds and of cardiac extra sound vibrations to the heart rate.

(c) The relative amplitude of heart sounds and heart sound vibrations as recorded from the right and from the left ventricle.

(d) The effect of variations in the rate of venous inflow to the heart on the amplitude of heart sounds and of heart sound vibrations.

(e) The effect of electrical stimulation of the right vagus nerve on the amplitude of cardiac extra sounds.

(f) The effect of administration of digoxin and of physostigmine salicylate on the amplitude of cardiac extra sounds.

(g) The effect of impact on the ventricular wall either of a glass rod or of a column of fluid.

Method

Anaesthesia. In the experiments described in this chapter, the anaesthetic procedure recommended by Luisada and Mautner (1943) was followed. Morphine sulphate (10 mg./kg. body weight) was administered subcutaneously, followed by urethane (1 g./kg. body weight) by stomach tube when the morphine had taken effect. On this dosage supplementary anaesthesia was usually required before the end of the experiment so, in the later experiments, the dose of each drug was increased by 25%.

Phonocardiography. For recording the heart sounds the vulcanite bell of a standard stethoscope chest-piece (2.5 cm. internal diameter) was connected by a short length

(30 cm.) of rubber tubing (8mm. bore) to the crystal microphone of the audio-frequency channel of the phonocardiograph. Both stethoscopic and logarithmic P.C.G.s were obtained (filter positions 0 and 4 on the preamplifier). As noted above, linear phonocardiography was not considered practicable, but the investigation described in Chapter 6 had shown that the distortion introduced by a short length of stethoscope tubing is negligible.

For the reference tracing either the electrocardiograph leads (lead II), or leads from the special phlebograph pick-up described in Chapter 4, were connected to the low-frequency channel of the phonocardiograph.

As in the human investigations described in Chapters 6, 8, and 10, the criterion for identification of heart sounds or heart sound vibrations was that they should be observed on the P.C.G. at the same period in at least three successive cardiac cycles. The gain of the phonocardiograph was set, for the preliminary records, so that the amplitude of deflections corresponding to the loudest sound was between 1 and 2 cm. on the record. In some of the experimental P.C.G.s, which followed, this gain was exceeded.

Since the deflections corresponding to cardiac extra sounds were, on the whole, smaller than in the human experiment, and the changes with experimental procedures less marked, the deflections on dog phonocardiograms were meas-

ured to the nearest 0.5 mm. and the percentage change was calculated to the first decimal place.

Operation. With the animal under general anaesthesia and the hair of the chest clipped very short, preliminary P.C.G.s were obtained from the chest wall in the region of the cardiac apex. A tracheal cannula was then introduced and the chest opened by excision of the 5th, 6th, and 7th ribs on the right side, from the costal cartilage to near the angle of the rib. Respiration was maintained by means of a Starling 'Ideal' pump attached to the tracheal cannula.

The pericardium was then incised and reflected, and a stethoscope bell was stitched to the muscle of the right ventricle. For this purpose six small holes had previously been drilled near the margin of the stethoscope bell. In the later animals the chest was opened from both sides and a stethoscope bell was stitched to each ventricle so that records could be taken from either.

Loose ligatures were placed round the anterior and posterior venae cavae for subsequent occlusion of these vessels.

The right vagus nerve and the right external jugular vein were then exposed in the neck. A loose ligature was passed round the nerve and a cannula was then introduced into the external jugular vein, through which slow intravenous infusion of Ringer-Locke solution was maintained

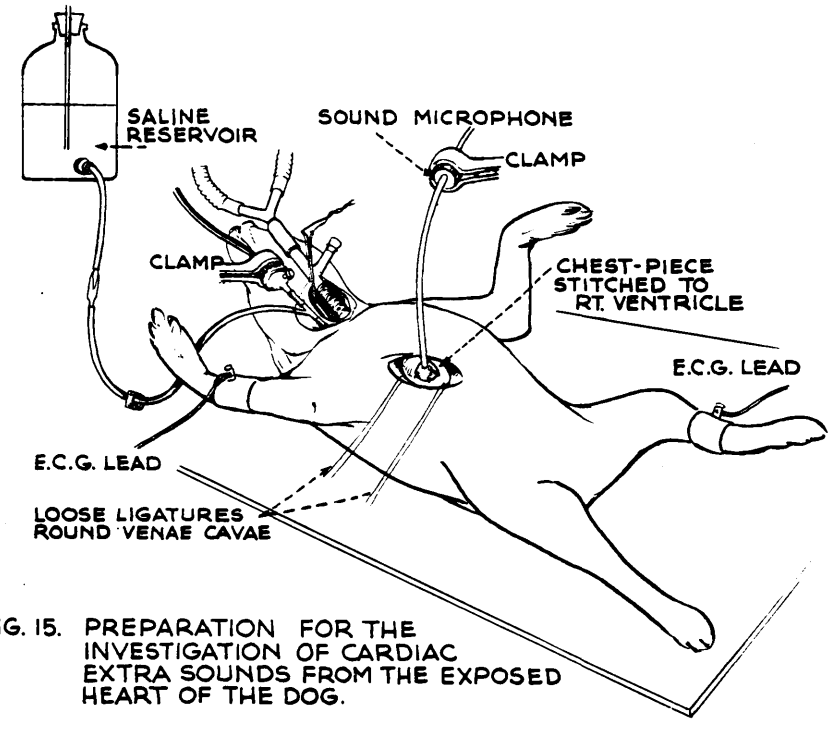


FIG. 15. PREPARATION FOR THE INVESTIGATION OF CARDIAC EXTRA SOUNDS FROM THE EXPOSED HEART OF THE DOG.

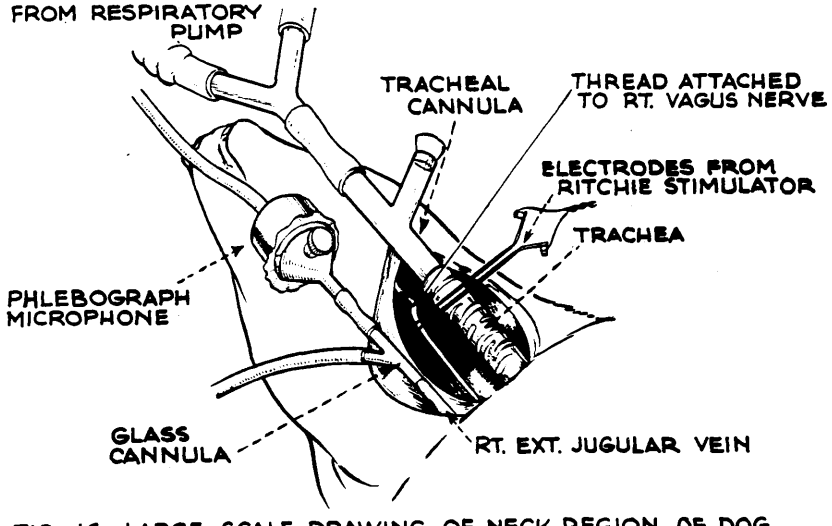


FIG. 16. LARGE-SCALE DRAWING OF NECK REGION OF DOG PREPARATION.

throughout the experiment. At the conclusion of all these operative procedures, heparin (1000 units/kg. body weight) was injected into the delivery tube of the infusion apparatus.

The phlebograph attachment was fitted to the straight arm of the jugular cannula so that subsequently either the venous pulse or the E.C.G. could be recorded as the reference tracing. Figs. 15 and 16 show the preparation at this stage.

P.C.G.s were then obtained from the exposed heart. The respiratory pump was stopped for a few seconds during the recording of these and of subsequent P.C.G.s.

Experimental procedures. Control P.C.G.s were recorded from the ventricle, with the phonocardiograph set at a suitable gain. Then, without altering the gain of the phonocardiograph, the venous return to the heart was reduced by pulling on the ligatures around one or both venae cavae, and a second record was taken.

To find the effect of increasing the venous return a second control record was taken, after which the venous inflow was increased by running Ringer-Locke solution rapidly into the external jugular vein. A P.C.G. was recorded during the rapid inflow, without any alteration of the gain of the phonocardiograph.

On each record the amplitude of deflection of each

sound was measured over five cardiac cycles and the mean amplitude was calculated. The mean change in amplitude with the experimental procedure was found and calculated as a percentage of the original mean amplitude.

Other experimental procedures employed were electrical stimulation of the peripheral end of the divided right vagus nerve and the administration of digoxin or physostigmine salicylate. One or other of these drugs was administered by slow intravenous injection into the delivery tube of the infusion apparatus. The dose, in the case of digoxin, was 1 - 3 mg., given slowly in divided dosage until some effect was apparent. In the case of physostigmine the dose was 1.3 - 4.6 mg., given slowly in divided dosage until there was an appreciable slowing of the heart rate. In each case logarithmic phonocardiograms were recorded at intervals for 30 - 45 minutes after administration of the drug.

For the final group of experiments the dog's heart was arrested by electrical stimulation of the peripheral end of the right vagus nerve, and venous inflow was stopped by occlusion of the venae cavae. Then attempts were made to produce artificially vibrations resembling those of a third heart sound. In some cases these attempts were made shortly after the animal's death.

First I observed the effect of tapping lightly with

a glass rod on the wall of the right ventricle at a little distance from the attached stethoscope chest-piece.

Secondly I studied the effect of squirting saline into the right ventricle. A purse-string suture was tied round the right auricular appendage, which was then incised. While loss of blood was controlled by tension on the purse-string suture, a rubber tube (5 mm. bore) filled with Ringer-Locke solution was introduced into the right atrium and the tip of the tube was guided through the tricuspid orifice to lie approximately 3 cm. from the wall of the right ventricle. The tube was then clipped and the purse-string suture was tied firmly to prevent loss of blood or further movement of the tube.

The heart was then arrested by electrical stimulation of the exposed right vagus and a quantity of Ringer-Locke solution was squirted rapidly down the tube to strike the ventricular wall, continuous phonocardiography being performed the while. Starting with the calculated stroke volume of the heart, estimated at 14 ml. for a 10 kg. dog (Dukes, 1943), progressively smaller quantities were injected in attempts to produce vibrations resembling those of the third heart sound.

Thereafter, a special deflector was passed through the rubber tube and then drawn back until it lay about

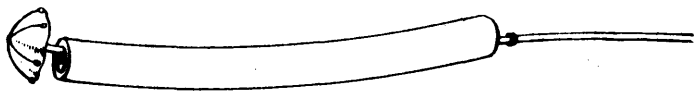

FIG. 17. DEFLECTOR FOR INTRODUCTION INTO THE VENTRICLE OF THE DOG HEART.

1 cm. in front of the opening of the tube. The deflector (Fig. 17) comprised an 'umbrella' of rubber dam, held in position by six small wire springs, tipped with knobs to prevent them catching on the papillary muscles or the columnae carneae, the whole mounted at the end of a firm but flexible wire (16 S.W.G.). The 'umbrella' was passed collapsed down the tube and opened out when it passed the end of the tube. Thereafter fluid squirted down the tube would not strike the ventricular wall but would be sprayed back on to the cusps of the tricuspid valve, tending to close them round the tube. Since the device could not be withdrawn from the heart once it had been introduced, this part of the experiment was necessarily performed last. In some of the dogs these experiments were performed shortly after death, before the onset of rigor mortis.

A post-mortem examination of the heart was carried out on every dog to confirm that the stethoscope chestpiece had in fact been applied precisely over the ventricle and, in the small group of experiments with the intracardiac tube and deflector, to confirm that the tip of the tube and the deflector were correctly positioned in the right ventricle.

Experimental animals

Two series of experiments are reported here. The

Table 26. Incidence of cardiac extra sounds in a series of 12 dogs: logarithmic phonocardiography.

| Sound | Recorded from | No. of animals | Sound present in | Incidence (per cent) |
|---|---|---|---|---|
| Third | Chest wall | 12 | 1 | 8 |
| | R. ventricle | 12 | 1 | 8 |
| | L. ventricle | 8 | 1 | 12 |
| Fourth | Chest wall | 12 | 0 | 0 |
| | R. ventricle | 12 | 8 | 67 |
| | L. ventricle | 8 | 0 | 0 |
| Systolic | Chest wall | 12 | 0 | 0 |
| | R. ventricle | 12 | 1 | 8 |
| | L. ventricle | 8 | 1 | 12 |

Table 27. Incidence of vibrations at the time of cardiac extra sounds in a series of 12 dogs: stethoscopic phonocardiography (x)

| Sound | Recorded from | No. of animals | Sound present in | Incidence (per cent) |
|---|---|---|---|---|
| Third | Chest wall | 12 | 12 | 100 |
| | R. ventricle | 12 | 9 | 75 |
| | L. ventricle | 8 | 5 | 62 |
| Fourth | Chest wall | 12 | 9 | 75 |
| | R. ventricle | 12 | 11 | 92 |
| | L. ventricle | 8 | 5 | 62 |

x The systolic extra sound cannot be distinguished in the stethoscopic P.C.G.

No summation sound was observed in these records.

first series was performed on 12 medium-sized dogs (7 - 20 kg. body weight) of various breeds and of both sexes. On these animals Miss Wishart and I studied the incidence of cardiac extra sounds and cardiac extra sound vibrations and the effect on the amplitude of these of alteration in the rate of venous inflow to the heart. In some of the animals of this series we observed the effect of vagal stimulation and of the administration of drugs.

Subsequently, in a small series of 5 dogs (11 - 20 kg. body weight) I attempted, by the methods described above, to produce something resembling a third heart sound.

Results

(a) The preliminary P.C.G.s from the exposed chest wall and from the exposed right and left ventricles of one of the dogs are shown in Plate 74 (Volume 2), and the occurrence of cardiac extra sounds and extra sound vibrations in the series of 12 dogs is detailed in Tables 37 and 38 (Volume 2) and summarized in Tables 26 and 27.

In logarithmic P.C.G.s from the chest wall, a third heart sound was recorded from one dog only. When the heart was exposed, a third heart sound was recorded from the right ventricle in another dog and from the left ventricle in yet another. The fourth heart sound was not recorded in any case from the chest wall or from the left ventricle but it was recorded from the exposed right ven-

tricle in 8 dogs. No systolic extra sound was recorded from the chest wall but it was recorded in one dog from the right ventricle and in another from the left ventricle.

Vibrations at the time of the third heart sound were recorded on the stethoscopic P.C.G. from the chest wall of all 12 dogs, from the right ventricle of 9, and from the left ventricle of 5. The corresponding incidence of fourth sound vibrations was 9 from the chest wall, 11 from the right ventricle, and 5 from the left ventricle. As noted in Chapter 6, the systolic extra sound cannot be identified on a stethoscopic P.C.G. since there is normally a continuous series of vibrations during systole on this record.

(b) No significant correlation was found between the heart rate and the presence or absence of cardiac extra sounds on the logarithmic P.C.G. or of the corresponding vibrations on the stethoscopic P.C.G.

(c) In the 6 dogs where the comparison was made, the heart sounds were of greater amplitude in the right ventricle than in the left in 4 cases and of greater amplitude in the left ventricle in one case. In the remaining case there was no appreciable difference in the amplitude of the heart sounds as recorded from the two ventricles. No obvious difference in the amplitude of particular cardiac extra sounds or extra sound vibrations was observed

except that, as noted above, the fourth heart sound was recorded on logarithmic P.C.G.s from the right ventricle only.

(d) The effects of variations in the venous inflow to the heart on cardiac extra sounds and extra sound vibrations were observed by logarithmic and stethoscopic phonocardiography but, owing to the low incidence of extra sounds on the logarithmic P.C.G.s, statistical analysis was performed only on the extra sound vibrations as shown on the stethoscopic records.

The effects of reduced and of increased venous return on the amplitude of the heart sounds was observed on logarithmic P.C.G.s from the right ventricle in only some of the dogs. The results are detailed in Tables 39 - 43. Plate 75 shows one of the sets of records obtained. In three cases in which a third heart sound was present in the control logarithmic P.C.G., occlusion of both venae cavae abolished it completely. In two cases the fourth heart sound was abolished by occlusion of the venae cavae but in a third case this procedure revealed a fourth sound, which was not present on the control record. The effect of increased venous return on the amplitude of third and fourth heart sounds, in the few cases where these appeared on the logarithmic P.C.G., was negligible. In two dogs a systolic extra sound was

Table 28. Effect of venous occlusion on the amplitude of vibrations at the time of occurrence of the heart sounds in dogs: stethoscopic phonocardiography from the right ventricle

| Sound | No. of dogs | Mean change (per cent) | Standard error of mean change | Significance |
|---|---|---|---|---|
| First | 12 | - 38.3 | ± 8.1 | Highly significant $P < 0.001$ |
| Second | 11 | - 56.7 | ± 10.9 | Highly significant $P < 0.001$ |
| Third | 11 | - 29.6 | ± 16.8 | Not significant $P > 0.1$ |
| Fourth | 10 | - 42.9 | ± 18.5 | Significant $P < 0.05$ |

Table 29. Effect of rapid intravenous infusion on the amplitude of vibrations at the time of occurrence of the heart sounds in dogs: stethoscopic phonocardiography from the right ventricle

| Sound | No. of dogs | Mean change (per cent) | Standard error of mean change | Significance |
|---|---|---|---|---|
| First | 11 | + 11.6 | ± 8.3 | Not significant $P > 0.1$ |
| Second | 10 | + 20.2 | ± 12.0 | Not significant $P > 0.1$ |
| Third | 9 | + 17.7 | ± 13.2 | Not significant $P > 0.2$ |
| Fourth | 9 | + 15.3 | ± 28.7 | Not significant $P > 0.5$ |

present in the control records; in one other it appeared only during rapid intravenous infusion.

The effects of reduced and of increased venous return on the heart sound vibrations recorded by stethoscopic phonocardiography from the right ventricle are detailed in Tables 44 - 47 (Volume 2) and summarized in Tables 28 and 29. Plate 76 is an example of the sets of records obtained.

Reducing the venous inflow to the heart caused a consistent reduction in the amplitude of the vibrations corresponding to first and second heart sounds and a more variable effect on the vibrations corresponding to third and fourth heart sounds. The mean change, in the case of the third and fourth sound vibrations, was a decrease; assessed as a percentage of the original amplitude it was significant only in the case of the fourth heart sound vibrations; expressed as an absolute change it was significant in both cases. Increased venous inflow caused no significant change in the amplitude of vibrations corresponding to any of the heart sounds.

The change in amplitude of the vibrations recorded from the left ventricle with the same experimental procedures are detailed in Tables 48 - 51 (Volume 2). These were in the same direction and of the same order of magnitude as those from the right ventricle, but the number

of experiments in which they were recorded was insufficient for the results to be significant.

The effect of reduction and of increase of the rate of venous inflow to the heart on the dogs' heart rate is noted in Table 52. The results are variable and fail to reach significance in either case.

(e) In five dogs the peripheral end of the divided right vagus nerve was stimulated electrically to cause appreciable cardiac slowing. This failed to produce a cardiac extra sound in logarithmic P.C.G.s from the right ventricle or to alter significantly the amplitude of existing extra sounds, except that a systolic extra sound appeared during vagal stimulation in one case (Table 53).

(f) The administration of digoxin to five dogs failed in every case to produce or alter a third heart sound on logarithmic P.C.G.s from the right ventricle. The fourth heart sound remained unaltered in two cases and was abolished in a third (Table 54). No systolic extra sound was recorded.

The administration of physostigmine salicylate to 6 dogs produced a third heart sound in the logarithmic P.C.G. in one case only. The fourth heart sound remained unaltered or absent in every case. A systolic extra sound appeared in two cases (Table 55).

Plate 77 (Volume 2) shows records obtained before and

after vagal stimulation and the administration of these drugs.

(g) When the heart's action was arrested by stimulation of the right vagus, a light tap on the outside of the ventricular wall usually produced an extra systole. Performed a few minutes after death, it produced vibrations resembling a heart sound.

When saline was squirted through the intra-cardiac tube against the wall of the right ventricle an extra systole was produced in most cases and , in the remainder, the vibrations recorded corresponded to a murmur rather than to a heart sound. With the deflector in position the vibrations again corresponded to a murmur rather than to a sound. When the injection was repeated after death of the animal, the vibrations produced again resembled those of a cardiac murmur.

Plate 78 (Volume 2) shows the effect of this last group of experimental procedures.

Discussion

(a) In common with D'Espine (1882) and Melik-Gülnasarian (1932a) I found in general a low incidence of cardiac extra sounds at the chest wall in dogs. Unlike Melik-Gülnasarian I found a low incidence also when recording directly from the exposed beating heart. An ex-

ception was the fourth heart sound, which was recorded in 8 out of 12 dogs on logarithmic P.C.G.s from the right ventricle. This suggests that the fourth sound may be produced mainly in the right side of the heart, but further evidence is lacking.

As in the observations on healthy human subjects, described in Chapter 6, the incidence of cardiac extra sound vibrations on stethoscopic phonocardiography was much higher than that of the actual sounds on logarithmic phonocardiography. This indicates that the strongest components of the vibrations produced at these times are below audio-frequency.

The suggestion that an audible fourth heart sound may be due to abnormal separation of the vibrations due to atrial systole from those due to ventricular systole is not borne out in these observations. Except after the administration of digoxin, the P-R interval on the E.C.G.s never exceeded 0.13 sec., the upper limit of normal conduction time in the dog (Luisada, Weisz, & Hantman, 1944).

(b) Neither tachycardia nor bradycardia was found to be essential for the production of cardiac extra sounds. This contradicts the belief of some of the earlier investigators of gallop rhythm but supports my own conclusions from observations on human subjects (Chapters 6, 8 and 10).

(c) No conclusions could be drawn from these ob-

servations as to whether one side or the other of the heart is predominant in the production of heart sounds. One might expect sounds of greater intensity to be produced in the left side of the heart, but the intensity of these would be reduced by conduction through the thick wall of the left ventricle.

(d) In spite of the greater facilities for control of the rate of venous return to the heart in the dog than in man, the results of the dog experiments were less conclusive. Unfortunately the low incidence of cardiac extra sounds in the dog, and the difficulty of obtaining dogs, made it impossible to obtain sufficient logarithmic P.C.G.s of cardiac extra sounds in the dog for adequate analysis.

The stethoscopic records obtained are, however, comparable to the 'total cardiac vibrations' recorded by Smith (1944). Unlike Smith I found, on blocking the venous inflow to the heart, that vibrations corresponding to all the heart sounds persisted in most cases. The vibrations were significantly diminished in amplitude by venous occlusion except in the case of the third heart sound, where the reduction, expressed as a percentage change, just failed to reach significance. The change produced in these cardiac extra sound vibrations by venous occlusion is therefore less marked than that in

the human heart sounds when the venous return is reduced (Chapter 10). It seems that the audio-frequency components of protodiastolic vibrations are influenced by the reduction of venous return more than are the vibrations of lower frequency.

With rapid intravenous infusion the change in amplitude of heart sound vibrations was variable. The mean change in each case was positive but failed to reach significance, as it might have done in a larger series. As with venous occlusion it would appear that it is the audio-frequency components of cardiac extra sound vibrations, rather than the lower-frequency components, which are mainly influenced by an increased rate of venous return.

(e) Unlike Melik-Gülnasarian (1932a,b) and Luisada and Mautner (1943) I failed to influence the third or fourth heart sound by stimulation of the right vagus nerve sufficient to cause cardiac slowing. Luisada and Mautner produced the fourth heart sound also by stimulation of the left vagus sufficiently strongly to cause increased atrio-ventricular conduction time, but I did not repeat this experiment. It appears either that hypotonia of the myocardium is not a very significant factor in the production of gallop rhythm, or that stimulation of the right vagus does not produce any great reduction of myocardial tone. The stethoscopic P.C.G. was not investi-

gated in this connection since the object was to produce the equivalent of audible extra sounds for comparison with the findings of previous investigators.

(f) Luisada and Mautner (1943) found digitalis the most effective drug for producing both third and fourth heart sounds. They attributed the augmentation of the third heart sound partly to direct stimulation of the myocardium and partly to stimulation of the vagus fibres to the heart; in the case of the fourth heart sound the effect was thought to be due to potential heart block.

In the present investigation digoxin, administered in large doses, failed to produce any significant change in either the third or the fourth sound on the logarithmic P.C.G.s. Similarly, physostigmine failed to exert any marked influence on the production of cardiac extra sounds. I expected that its administration, producing an effect similar to that of prolonged stimulation of both vagi, would cause some change, but only in one case did a third sound appear after administration of this drug. The appearance of a systolic extra sound in 2 out of 6 dogs after the administration of physostigmine is an interesting finding but the series is too small for any conclusions to be drawn from it.

(g) Like Boyer (1942) I found that a light tap on the ventricular wall of the dog heart produced vibrations

resembling those of the third heart sound. Boyer deduced from this that the third heart sound is primarily due to an impact on the wall of the ventricle. Since it can be detected in the exposed heart, this impact cannot be against the chest wall; it is therefore presumably due to the impact of inflowing blood.

The results of my intracardiac manipulations were disappointingly inconclusive. I had hoped to produce vibrations like those of the third heart sound either by the impact of fluid against the ventricular wall or by abrupt closure of the atrio-ventricular valve, but attempts with a wide range of injection volumes failed to produce anything resembling a heart sound in either case. Natural conditions were, of course, poorly simulated, since injection against the ventricular wall was performed into a ventricle already containing blood, and closure of the valve cusps was not into normal apposition but against a fairly wide tube passing through the tricuspid orifice. With these reservations the observations are evidence, albeit inconclusive, against the third heart sound being due simply to the impact of inflowing blood on the ventricle.

The next stage in this investigation, which I hope to carry out in the near future, will be to introduce a small crystal microphone directly into the normally beating heart and ascertain whether the heart sounds, and

especially cardiac extra sounds if present, are louder in the vicinity of the atrio-ventricular valve or of the ventricular wall opposite the valve orifice. That the recording of heart sounds by such a technique is technically possible has been shown by the work of Braun-Menéndez and Solari (1936a) and of Macleod and Cohn (1941). I am at present seeking a suitable microphone.

Summary

The incidence of cardiac extra sounds on logarithmic phonocardiography and of the corresponding vibrations on stethoscopic phonocardiography was observed in a series of 12 dogs. Records were obtained from the praecordium and from the exposed heart.

There was no significant relation between the presence or absence of cardiac extra sounds or extra sound vibrations and the heart rate.

The heart sounds were not consistently louder from one ventricle than from the other.

Reducing the venous inflow to the heart caused a reduction in amplitude of the heart sound vibrations recorded by stethoscopic phonocardiography from either ventricle. The change was statistically significant only in the case of the first, second, and fourth sounds recorded from the right ventricle. Increased venous inflow caused no significant change in the amplitude of vibrations corresponding to any of the heart sounds.

Neither electrical stimulation of the right vagus nerve nor the administration of digoxin or physostigmine had any consistent effect on the appearance or amplitude of cardiac extra sounds on the logarithmic P.C.G.

Vibrations resembling those of a heart sound were produced by a light tap on the ventricular wall. Rapid injection of fluid into the right ventricle, either against the muscular wall or against the ventricular aspect of the cusps of the tricuspid valve, failed to produce anything resembling a heart sound.

# SUMMARY AND CONCLUSIONS

## Heart sounds and murmurs

One must assume that from very early times men have been aware of sounds produced at the front of the chest by the beating heart. Yet medical literature paid scant attention to these sounds until Laënnec studied them with the aid of his newly invented stethoscope and described them in his great book, 'De l'auscultation médiate'. Although there was considerable opposition, especially in France, to the idea that heart sounds might have any clinical significance, the more enlightened physicians applied the new diagnostic technique to the clinical investigation of their cardiac patients. They did so to such good effect that by the end of the nineteenth century most of the important auscultatory signs of heart disease had been described.

Experimental workers, by auscultation of the exposed heart of various animals, established the time of occurrence of the heart sounds in the cardiac cycle. By ingenious experiments some insight was gained into the mode of production of the heart sounds and of murmurs.

The invention of the phonocardiograph in the last decade of the nineteenth century, and the subsequent development of this instrument, made possible the detailed

study of the actual vibrations which constitute heart sounds and murmurs. These vibrations were found to be so irregular that it was difficult to assign definite frequencies to them, but analysis showed that murmurs in general were of higher frequency than heart sounds and that both were considerably below the frequency of breath sounds. The fundamental tones of the heart sounds fall in the frequency range 25 - 50 cycles per second.

The beating heart produces vibrations below audio-frequency at the front of the chest; these vibrations are of much greater amplitude than the heart sounds. On an undistorted phonocardiogram (P.C.G.) the low-frequency vibrations mask the heart sounds: in order to display heart sounds or murmurs some high-pass filter, corresponding to the human auditory mechanism, must be incorporated in the instrument.

An undistorted record of praecordial vibrations is called a linear P.C.G. One in which the distortion normally produced by a stethoscope has been introduced is called a stethoscopic P.C.G. A record in which the vibrations have been modified as they would be in auscultation by the stethoscope and the auditory mechanism is called a logarithmic P.C.G. Only on the logarithmic P.C.G. do the deflections recorded correspond to auditory phenomena.

Phonocardiography

The earliest phonocardiographs merely indicated the occurrence of heart sounds but failed to record vibrations of audio-frequency. By the first decade of the twentieth century, however, reasonably accurate records of heart sounds were being obtained.

Most investigators appreciated the need to record some reference tracing simultaneously with the P.C.G. in order to determine the time of occurrence of sound phenomena in the cardiac cycle: the most popular reference tracings were and are the apical cardiogram (linear P.C.G.), the electrocardiogram, and the jugular phlebogram.

Two of the early methods of phonocardiography are still in use today, viz. the direct method, using Frank's segment capsules, and the string galvanometer introduced by Einthoven. Most modern phonocardiographs, however, employ either mirror galvanometers or a cathode-ray tube or tubes as the recording device. A great advantage of mirror galvanometers is the facility with which a number of these can be arranged to give simultaneous recording of a number of P.C.G.s and reference tracings. The cathode-ray tube is theoretically the ideal recording device but technical difficulties in its use have limited its popularity.

Calibration of the P.C.G. is a late development in

the history of phonocardiography. While of limited value in the assessment of cardiac activity, calibration serves to indicate the absolute loudness of recorded sound phenomena and helps to link phonocardiographic with auscultatory findings.

The phonocardiograph described in this thesis is a two-channel instrument incorporating piezo-electric crystal microphones, electronic amplifiers and filters, a loud-speaker monitor, and a double-beam cathode-ray oscillograph. In the sound channel the microphone is connected to a pre-amplifier, with electrical filters to give logarithmic attenuation of lower frequencies: this is followed by an audio-amplifier, which gives linear amplification of the signal from the preamplifier: the audio-frequency amplifier supplies the loud-speaker and the lower beam of the oscillograph. The venous pulse is picked up by a cup applied to the neck over the jugular bulb and connected by rubber tubing to a crystal microphone. By means of a switch either this microphone or electrocardiograph leads are connected to a low-frequency amplifier, which activates the upper beam of the oscillograph. The recording paper is run horizontally through the camera while the horizontal traverse of the beam is arrested. A small electric lamp, mounted in front of the oscillograph screen, flashes at regular intervals to record a time tracing on

the moving paper.

For experimental phonocardiography of dogs a special device was invented to permit recording of the venous pulse from the lumen of the external jugular vein.

A calibration device has recently been fitted to the phonocardiograph and is at present under trial.

Normal triple cardiac rhythm

Triple cardiac rhythm in a healthy young subject is most often due to the addition to the first and second heart sounds of the third heart sound (the 'rapid-filling' sound). The incidence of the physiological third heart sound diminishes with advancing age; it probably never occurs in health after the age of 40 years. Although the third heart sound can be heard , or recorded by logarithmic phonocardiography, in only a proportion of healthy young adults, vibrations below audio-frequency occur constantly in early diastole in these subjects and may be demonstrated by stethoscopic or linear phonocardiography.

The fourth heart sound (the atrial sound) is not normally audible in a healthy subject but may appear on the logarithmic P.C.G. The discrepancy between the findings by auscultation and by phonocardiography may be due to failure of the instrument to reproduce exactly the low-frequency cut-off of the ear, or to auditory masking of the fourth sound by the following, much louder, first sound.

The systolic extra sound is a rare phenomenon, which may be heard and recorded at any age, irrespective of the coexistence of heart disease.

A group of healthy young adults, students at Glasgow University, was investigated to determine the incidence of triple cardiac rhythm in the group, the agreement between auscultators as to the presence or absence of cardiac extra sounds, and the agreement between the results of auscultation and of phonocardiography.

The third heart sound, heard by a majority of three observers in approximately 10% of the subjects, was the only cardiac extra sound detected by auscultation. There was significant agreement between the three observers as to the presence or absence of this sound.

A third heart sound was noted on the logarithmic P.C.G. of 39% of the subjects, but agreement between the findings by auscultation and by phonocardiography was poor.

A fourth heart sound was recorded on the logarithmic P.C.G. of approximately 28% of the subjects, and a systolic extra sound in approximately 3%.

Apical pulsations below audio-frequency were recorded at the time of the third and fourth heart sounds on the stethoscopic and linear P.C.G.s of every case. It appears therefore that the rapid filling of the ventricles in early diastole and again during atrial systole constantly

sets up praecordial vibrations, although these are commonly below the threshold of audibility.

Pathological triple cardiac rhythm

Triple cardiac rhythm due to the addition to the first and second heart sounds of the third heart sound, the fourth heart sound, or the summation sound, in a patient with heart disease constitutes gallop rhythm. The third heart sound occurs at a constant time interval after the preceding second sound in any individual and the fourth heart sound occurs at a constant time interval before the following first sound. Third sound gallop is commonly called protodiastolic, and fourth sound gallop presystolic, although with tachycardia the third sound may occur in mid-diastole or even in the second half of diastole, and the fourth sound may occur in mid- or early diastole.

The commonest form of gallop rhythm, according to most previous investigators, is presystolic gallop. The extra sound (the fourth heart sound) is produced predominantly in the left ventricle and is associated with left ventricular failure, especially that of advanced cardiorenal disease. The fourth heart sound has been attributed to sudden filling of the ventricle to its elastic limit by atrial systole; this limit is more readily attained if the elasticity has been reduced by hypertrophy, passive dilatation, or both. Presystolic gallop rhythm is usually

considered a grave prognostic sign.

Protodiastolic gallop rhythm is commonly associated with right ventricular failure. The gallop sound is distinguished from the physiological third heart sound by its greater constancy during the respiratory cycle and with postural changes. It has been associated with a number of infectious or degenerative diseases and has been ascribed to the resulting reduction in myocardial tone. Protodiastolic gallop may indicate incipient or established heart failure, but it appears to be a less ominous sign than is presystolic gallop.

Summation gallop is rare and is due to superposition of the third and fourth heart sounds in tachycardia. The separate occurrence of these sounds, constituting train wheel rhythm, has been held to be a very grave prognostic sign.

Systolic gallop rhythm has been described by several investigators but there is no evidence that the systolic extra sound is a sign of heart disease.

The opening snap of the mitral valve is diagnostic of mitral stenosis and usually shows that this condition is still at an operable stage.

A series of patients with heart disease was investigated to find the incidence of the several types of triple cardiac rhythm, and the patients were followed up

for 6 - 9 months to find whether any significant deterioration occurred in those with gallop rhythm as compared with the rest. Protodiastolic gallop rhythm was commoner in this series than was presystolic gallop and occurred in patients in whom the right side of the heart was subjected to strain: presystolic gallop rhythm was associated with left heart strain. There was significant deterioration of the patients with presystolic gallop or train wheel rhythm but not of those with protodiastolic gallop rhythm. A few patients had a systolic extra sound, which appeared to be of no diagnostic or prognostic significance. The mitral opening snap was found only in patients with other evidence of mitral stenosis and appeared to be a favourable indication for mitral valvotomy: in some patients it persisted after an apparently successful valvotomy.

In patients with mitral stenosis a mitral opening snap was commoner in those with no other valvular lesion; in the presence of concomitant aortic incompetence a third heart sound was commoner than a mitral opening snap and was of bad prognostic significance. Gallop rhythm was not significantly commoner in patients with arterial hypertension than in the other cardiac patients, nor did it affect the short-term prognosis in these cases.

Experimental study of triple cardiac rhythm

The third heart sound appears to be primarily due either to vibrations of the ventricular wall set up by the impact of blood flowing in from the atria or to vibrations of the atrio-ventricular valve cusps as they float into apposition during rapid ventricular filling. There is some experimental evidence in support of each of these mechanisms, and indeed both may be responsible for the production of the third heart sound.

Experimental evidence supports the view that the fourth heart sound is due in part to muscular contraction of the atria and in part to the impact of blood expelled by atrial systole into the ventricles.

The summation sound has been produced experimentally, but little or no experimental work has been performed on the systolic extra sound or on the mitral opening snap.

In an attempt to ascertain whether the physiological third heart sound is in fact dependent on rapid ventricular filling a group of healthy young adults,,each with an obvious third heart sound on the logarithmic P.C.G., was investigated. The rate of filling of the heart was reduced by occluding the venous return from the limbs and increased by making the subject take active exercise. Reduction of the rate of filling significantly reduced

the amplitude of the third heart sound, whereas increase of the rate of filling significantly augmented it. Similar changes were observed in the fourth heart sound, where this was present, but changes in the amplitude of the first and second heart sounds were less consistent.

An attempt was made to repeat this experiment under more precisely controlled conditions in dogs. The heart sounds were recorded from the exposed heart; reduction of venous inflow was achieved by compressing the venae cavae, and the venous inflow was increased by rapid infusion of Ringer-Locke solution into an external jugular vein. Unfortunately cardiac extra sounds were recorded so seldom on the dogs' logarithmic P.C.G.s that no results statistically comparable to those of the human experiment could be obtained.

Cardiac extra sound vibrations on the stethoscopic P.C.G.s of dogs were less consistently related to the rate of venous inflow than were human heart sounds. Reduction of the venous inflow caused a significant diminution in the amplitude of vibrations corresponding to the fourth heart sound but an increase caused no significant change in the amplitude of any heart sound vibrations.

Experimental procedures, viz. electrical stimulation of the right vagus nerve or the administration of digoxin

or physostigmine, failed to produce the third or fourth heart sound or to influence these if they were already present. Vibrations resembling those of a heart sound could be produced by a light tap on the ventricle, but rapid injection of fluid into the ventricle failed to produce any such vibrations.

General conclusions

Phonocardiography is a valuable cardiological technique. It does not replace auscultation of the heart but is a most useful supplement to it. At present the main value of phonocardiography lies not so much in the detection of heart sounds and murmurs as in the determination of their exact time of occurrence in the cardiac cycle. The precise identification of diastolic extra sounds can only be achieved by phonocardiography.

It has been established that the third heart sound is a normal finding both by auscultation and by phonocardiography in young adults. Although the fourth heart sound is a normal finding on the phonocardiogram it is not audible in health. The systolic extra sound is rarer but occurs in healthy individuals as well as in cardiac patients.

Gallop rhythm is a common finding in heart disease. Protodiastolic gallop occurs more commonly when the right

side of the heart is under stress, and presystolic gallop when the left side of the heart is involved. Protodiastolic gallop may be of serious prognostic significance, and presystolic gallop appears to be an unfavourable prognostic sign, but further work is required to establish the truth of this.

The mitral opening snap, when it occurs, is diagnostic of mitral stenosis and usually indicates that this is at an operable stage.

The third and fourth heart sounds appear to be associated with rapid filling of the ventricles, but the precise mechanism of their production is still to be determined.

"The quest (understanding of Nature) is in a measure its own satisfaction. We receive the lesson that our advance to knowledge is of asymptotic type, even as continually approaching so continually without arrival. The satisfaction shall therefore be eternal."

Sir Charles Sherrington (1951)
Man on his nature. 2nd ed.
Cambridge: University Press.
p. 291

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# APPENDIX A

## CARDIAC MURMURS

Although cardiac murmurs are not a primary subject of study in this thesis, the murmurs recorded in some of the normal subjects and in most of the patients with heart disease merit brief comment.

As in the case of heart sounds so with murmurs there is often considerable discrepancy between the findings by auscultation and by phonocardiography. Since murmurs are of higher frequency than heart sounds they are more readily detected by the human ear, and it is not uncommon for a murmur to be clearly audible and yet fail to appear on the phonocardiogram.

Phonocardiography reveals no fundamental difference between innocent and organic murmurs, but diagnosis is aided by precise localization of the murmur in the cardiac cycle. The only murmur which does not lead one to suspect heart disease is a short systolic one. Evans (1947a,1948) found that an innocent murmur usually starts after the S wave of the simultaneously recorded electrocardiogram, but Cowen (1949) demonstrated that this is not a constant relationship.

Phonocardiography shows the amplitude of a murmur relative to that of the heart sounds, but a better guide to the significance of a murmur is probably its absolute loudness as detected by auscultation (Levine, 1948).

This may, in the future, be demonstrated by calibrated phonocardiography. Only the fainter murmurs (Levine's first and second degrees of loudness) may be innocent; louder murmurs (third to sixth degree) are always associated with heart disease.

For convenience of description systole and diastole may each be subdivided into three equal periods; a murmur is described as early, mid-, or late systolic, or early, mid-, or late diastolic, according to which period or periods of the cardiac cycle it occupies.

Innocent murmurs

In the series of 123 normal subjects described in Chapter 6 an apical systolic murmur was heard in 22 cases and was recorded on the logarithmic P.C.G. of 31; 13 were common to both groups. The murmur was of low amplitude and occurred in early or mid-systole. No diastolic murmur was observed in this series.

Mitral murmurs

In the series of 100 patients with heart disease described in Chapter 8 the commonest lesion was mitral stenosis. In the 29 patients diagnosed as having mitral stenosis as the only valvular lesion the commonest murmur on the logarithmic P.C.G. was pansystolic (11 cases), and the typical mid- and late diastolic murmur was next (6 cases). Two patients had a pandiastolic murmur; in ano-

ther two the diastolic murmur was limited to mid-diastole, and in another one to late diastole. Evans (1947a) found a mid-diastolic murmur to be the commonest auscultatory sign of mitral stenosis, but an audible mid-diastolic murmur may, as in seven cases in this series, fail to appear on the P.C.G. This is especially likely to occur in mitral stenosis, when the phonocardiograph is usually set at low gain because of the loudness of the heart sounds. Presystolic accentuation of a diastolic murmur was obvious in 8 out of the 16 cases of mitral stenosis with sinus rhythm in whom murmurs were recorded, and absent in all the cases with auricular fibrillation.

In five patients with mitral stenosis, three of whom had undergone mitral valvotomy, no murmur was recorded. In all of these a murmur was audible; this was mid-diastolic in time in the two patients who had not been submitted to operation and in one of the post operative cases. In one of the other post-operative cases it was pandiastolic and in the other pansystolic.

In three patients with mitral stenosis and well-marked murmurs, vibrations corresponding to these appeared also on the linear P.C.G. This is a not unusual occurrence (Weitz, 1918).

Of five patients with a diagnosis of mitral incompetence all had an audible pansystolic murmur and, in four of them, the murmur was seen on the logarithmic P.C.G.

The high incidence of systolic murmurs in the patients diagnosed as suffering from mitral stenosis suggests the existence of a considerable degree of mitral incompetence in these cases. Gerhartz (1911) and Cossio (1938) found that stenosis of the mitral valve is usually accompanied by incompetence. Logan and Turner (1953) and Brigden and Leatham (1953) affirmed that a long, loud apical systolic murmur suggests predominant mitral incompetence.

Aortic murmurs

In only four patients was a pure aortic lesion diagnosed. Two of these were aortic stenosis; both showed a pansystolic murmur on the logarithmic P.C.G. recorded at the cardiac apex, but in neither case did the murmur have the typical 'diamond' shape described by Leatham (1951). I heard no typical systolic murmur at the aortic area in either case and, unfortunately, no satisfactory P.C.G. was obtained from the aortic area. The other two patients, diagnosed as aortic incompetence, had early and mid-systolic murmurs, but the typical early diastolic murmur of this condition was neither heard by me nor recorded in either of them.

Murmurs of combined mitral and aortic valve disease

In 9 out of the 14 patients diagnosed as having lesions of both mitral and aortic valves, P.C.G.s were recorded not only from the mitral area but also from the aortic.

either at the sternal end of the third left intercostal space or at the second right costal cartilage or at the sternal end of the second right intercostal space.

All the patients with lesions of both mitral and aortic valves had clearly audible murmurs at both mitral and aortic areas; in 11 cases murmurs were recorded on the logarithmic P.C.G. at the mitral area and in 13 cases at the aortic area. In general the murmurs seemed to occupy more of the cardiac cycle on auscultation than was shown by phonocardiography. For instance in two cases where a murmur appeared on auscultation to be continuous throughout the cardiac cycle it was limited on the logarithmic P.C.G. to systole and early diastole.

In this group the commonest diagnosis (10 cases) was mitral stenosis and aortic incompetence. In three of these cases no murmurs were recorded at the mitral area; in two of these, early systolic and early diastolic murmurs were recorded at the aortic area, but in the third I found no evidence to support the hospital diagnosis of aortic incompetence. In five of the remaining seven cases the early diastolic murmur of aortic incompetence was clearly demonstrated on the logarithmic P.C.G. recorded at the cardiac apex. Being high-pitched this murmur is audible at very low sound intensity but a sensitive phonocardiograph, with adequate low-frequency cut-off, is required

to demonstrate it. Wells, Rappaport and Sprague (1949) considered that with modern equipment it should be possible to demonstrate all audible murmurs, but this ideal is seldom achieved. The presystolic murmur observed in three cases, and the presystolic accentuation of a long diastolic murmur in two more, are attributed to the mitral lesion and not regarded as Austin Flint murmurs. A murmur was recorded in early systole in all but one of the cases in whom murmurs were recorded at all. This murmur may be attributed to the mitral lesion in most cases but Evans (1947b) found that an early systolic murmur was recorded at the mitral area in all his cases of aortic valve disease. In seven cases of my series the murmur persisted throughout systole and was probably due to the mitral lesion.

In two patients the hospital diagnosis was of mitral stenosis and incompetence and aortic incompetence. One of these patients had pansystolic and early diastolic murmurs, whereas the other had early and mid-systolic and late diastolic murmurs; in the latter case the typical early diastolic murmur was heard and recorded only at the left sternal border.

In one patient diagnosed as having mitral and aortic incompetence without stenosis there was a pansystolic murmur at the mitral area, to which was added a pandiastolic

murmur at the aortic area.

In one patient with a diagnosis of stenosis and incompetence of both mitral and aortic valves the diamond-shaped systolic murmur described as typical of aortic stenosis was clearly seen in P.C.G.s from both mitral and aortic areas. An early diastolic murmur also was observed in all the records but the P.C.G. failed to show any murmur in mid- and late diastole although one was audible at this time.

Murmurs of congenital heart disease

There were only five cases of congenital heart disease in the series. Two of these had patency of the ductus arteriosus and are included on the assumption that the heart's function was affected to some extent by the abnormal work it was called upon to perform. One of the other cases was diagnosed as Fallot's tetralogy and the remaining two as pulmonary stenosis.

Both patients with patent ductus arteriosus had a long systolic murmur at the mitral area and a continuous or nearly continuous murmur at the pulmonary area. The murmur was loudest at the pulmonary area and reached its maximum intensity at the time of the second heart sound, which was masked by the murmur. The diagnosis was subsequently confirmed in both cases at operation.

The patient with Fallot's tetralogy had a pansystolic

murmur, loudest at the tricuspid area and probably due to the interventricular septal defect. Auscultation is of little diagnostic value in this condition (Taussig, 1947).

The two patients in whom congenital stenosis of the pulmonary valve was diagnosed had early and mid-systolic murmurs, and one had a soft mid- and late diastolic murmur at the apex, which suggests mitral stenosis. In neither patient did the systolic murmur continue up to or beyond the second heart sound as described by Leatham (1952) in cases of pulmonary stenosis.

Murmurs in patients with arterial hypertension

Patients with arterial hypertension were included in this series only if there was some evidence of cardiac disorder additional to or resulting from the hypertension. In 16 out of the 31 hypertensive patients this was limited to electrocardiographic evidence of left heart strain, but in 11 patients there were definite signs of cardiac failure.

In the patients with arterial hypertension there was good agreement between auscultatory and phonocardiographic findings. Of the 31 patients in the series, 20 had a systolic murmur by either means of investigation. Logarithmic phonocardiography showed this to be limited to early systole in 6 cases, and to early and mid-systole in another 7 cases; in the remainder it was pansystolic. The only diastolic murmur recorded in this group was a faint pre-

systolic one on the logarithmic P.C.G. of one patient.

Murmurs in other cardiac patients

No typical murmurs were observed in relation to the other cardiac lesions observed in this series of patients with heart disease.

# APPENDIX B

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# TRIPLE CARDIAC RHYTHM

BY

ARCHIBALD W. SLOAN

## VOLUME II

APPENDIX C Plates 1 - 79

APPENDIX D Tables 30 - 55

APPENDIX E Original publications

# APPENDIX C

## PLATES 1 - 79

| | |
|---|---|
| PLATES 1 - 8 | THE PHONOCARDIOGRAPH |
| PLATES 9 - 10 | PHONOCARDIOGRAMS OF NORMAL SUBJECTS |
| PLATES 11 - 72 | PHONOCARDIOGRAMS OF PATIENTS WITH HEART DISEASE |
| PLATES 73 - 79 | EXPERIMENTAL PHONOCARDIOGRAMS |

# APPENDIX C

## PLATES 1 - 8

## THE PHONOCARDIOGRAPH

Photograph, circuit diagrams, tests of venous pulse recorder.

## PLATE 1

Photograph of modern electronic phonocardiograph (Sloan, 1951; Campbell, Sloan & Andrew, 1952).

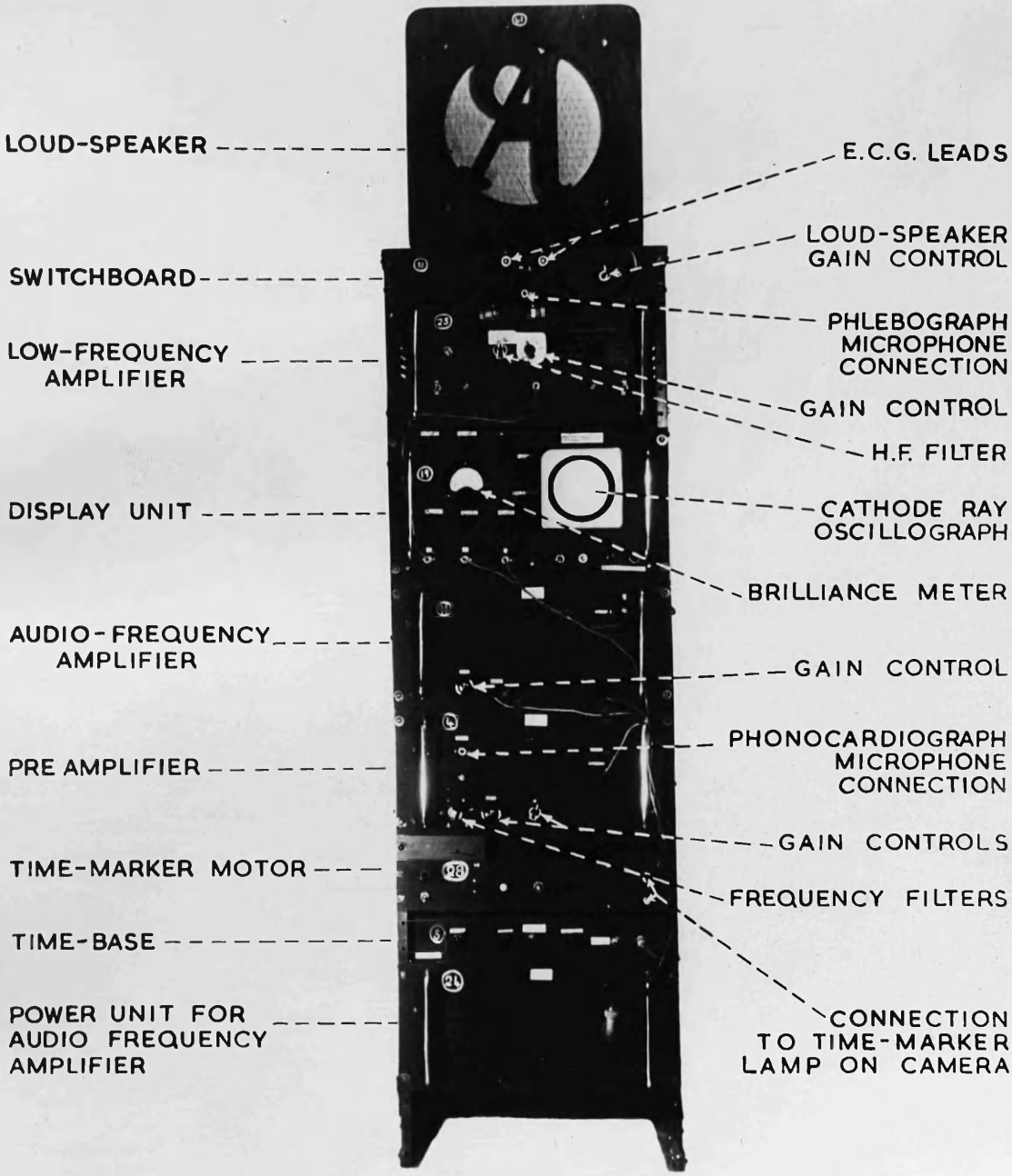
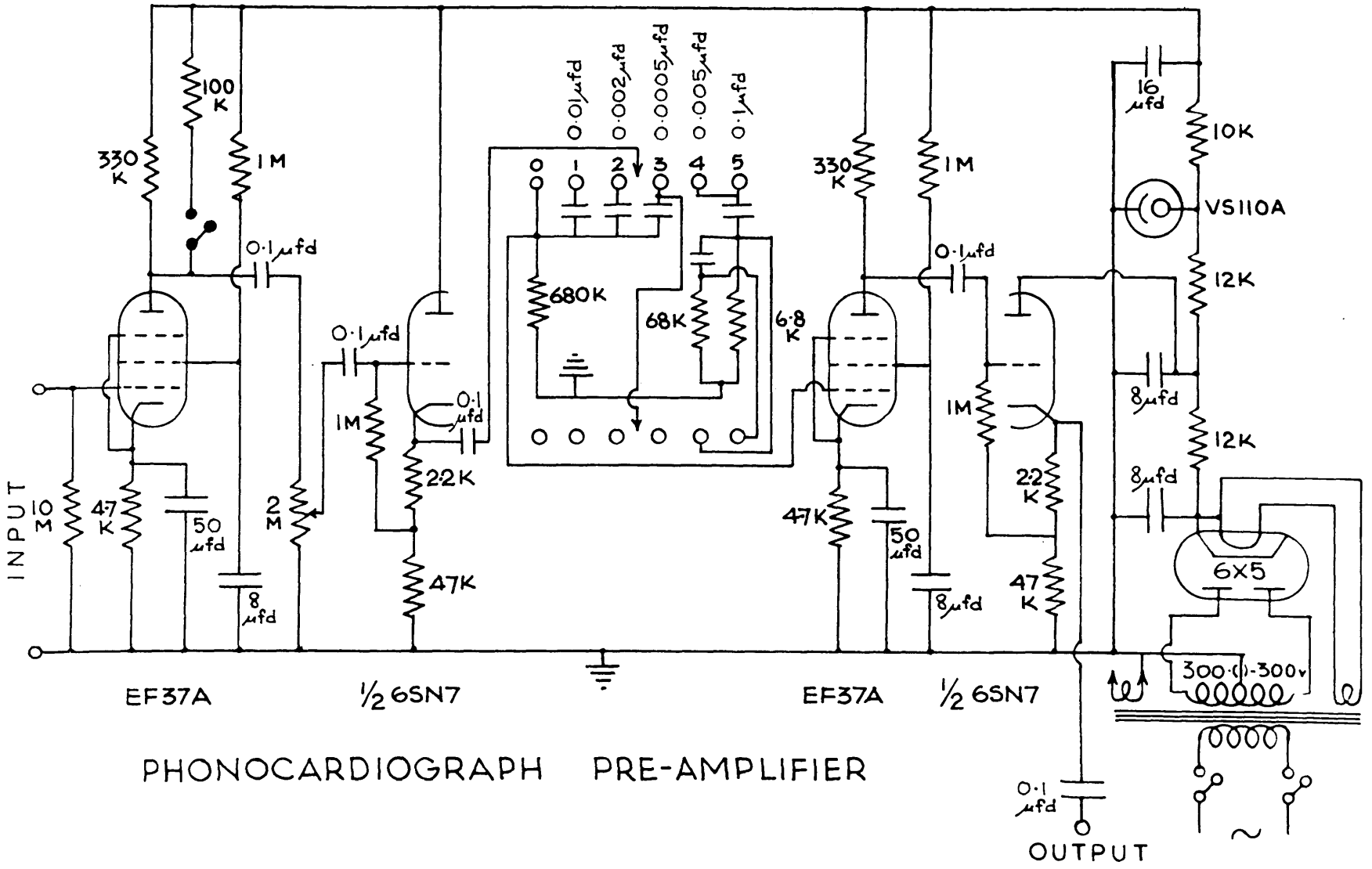

LOUD-SPEAKER
SWITCHBOARD
LOW-FREQUENCY AMPLIFIER
DISPLAY UNIT
AUDIO-FREQUENCY AMPLIFIER
PRE AMPLIFIER
TIME-MARKER MOTOR
TIME-BASE
POWER UNIT FOR AUDIO FREQUENCY AMPLIFIER
E.C.G. LEADS
LOUD-SPEAKER GAIN CONTROL
PHLEBOGRAPH MICROPHONE CONNECTION
GAIN CONTROL
H.F. FILTER
CATHODE RAY OSCILLOGRAPH
BRILLIANCE METER
GAIN CONTROL
PHONOCARDIOGRAPH MICROPHONE CONNECTION
GAIN CONTROLS
FREQUENCY FILTERS
CONNECTION TO TIME-MARKER LAMP ON CAMERA

PLATE 2

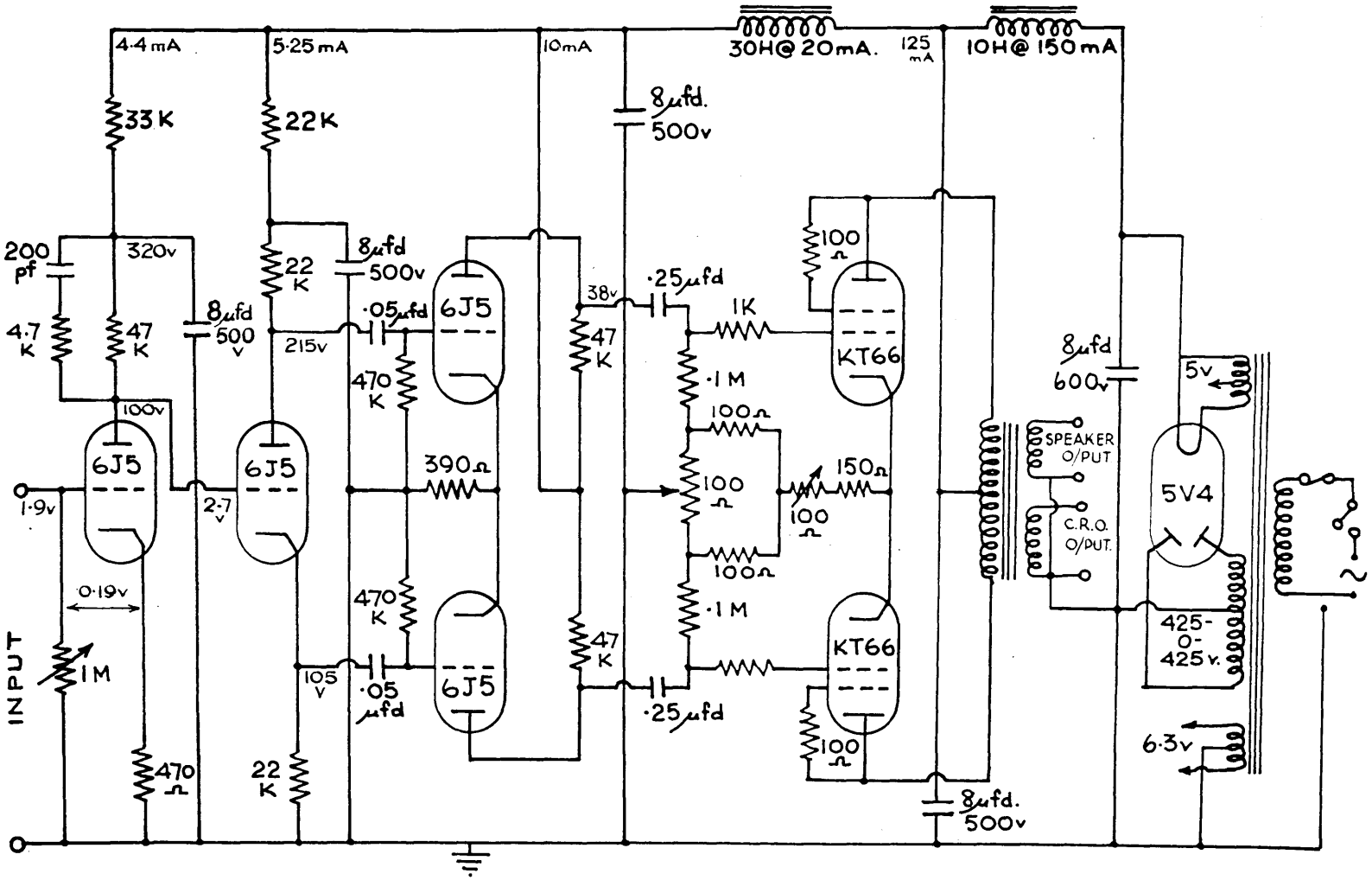
Circuit of phonocardiograph preamplifier (Cowen & Parnum, 1949; Campbell, Sloan & Andrew, 1952).




PHONOCARDIOGRAPH PRE-AMPLIFIER

## PLATE 3

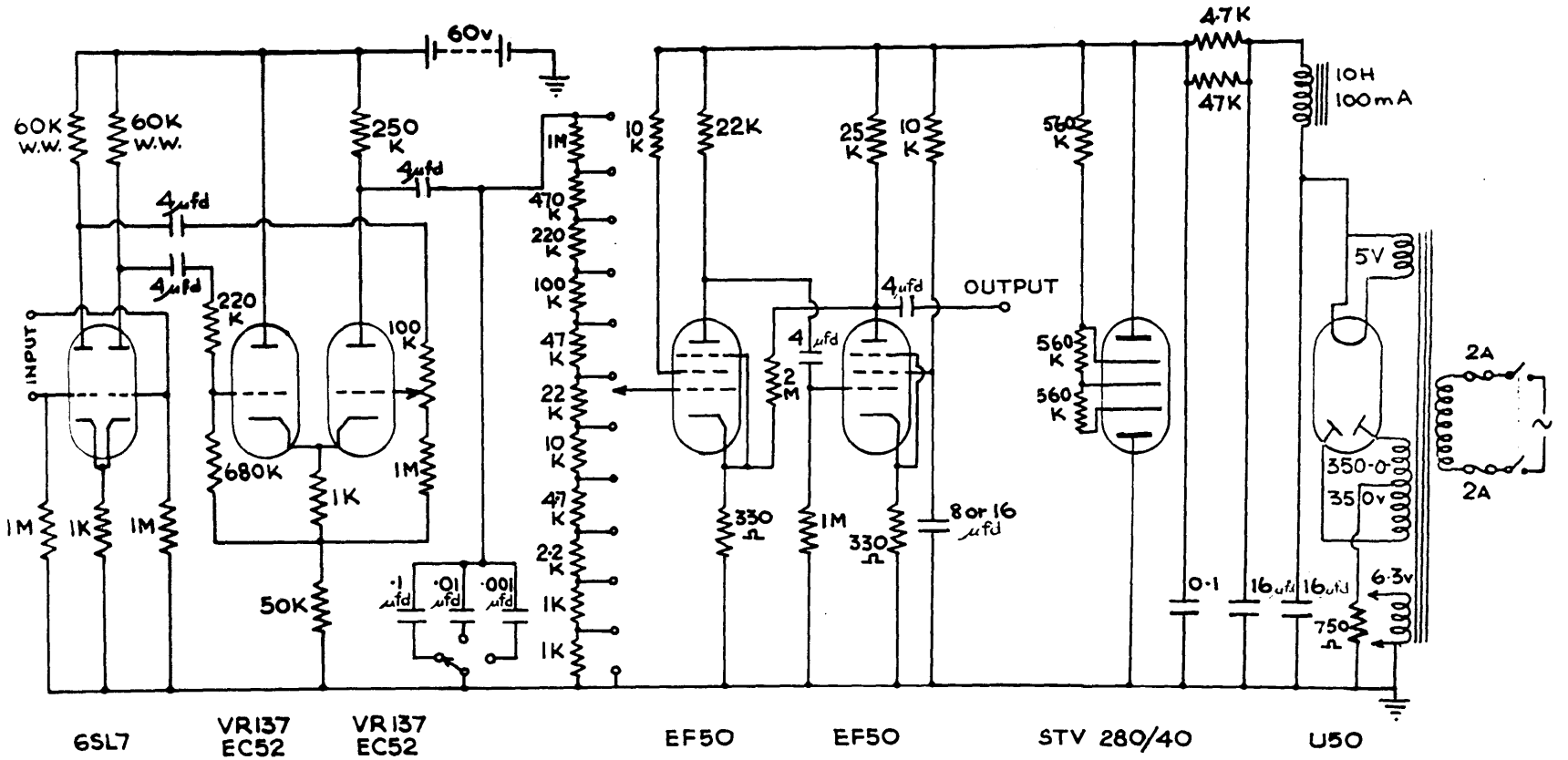
Circuit of audio-frequency amplifier, modified from Williamson (1951).




AUDIO AMPLIFIER

## PLATE 4

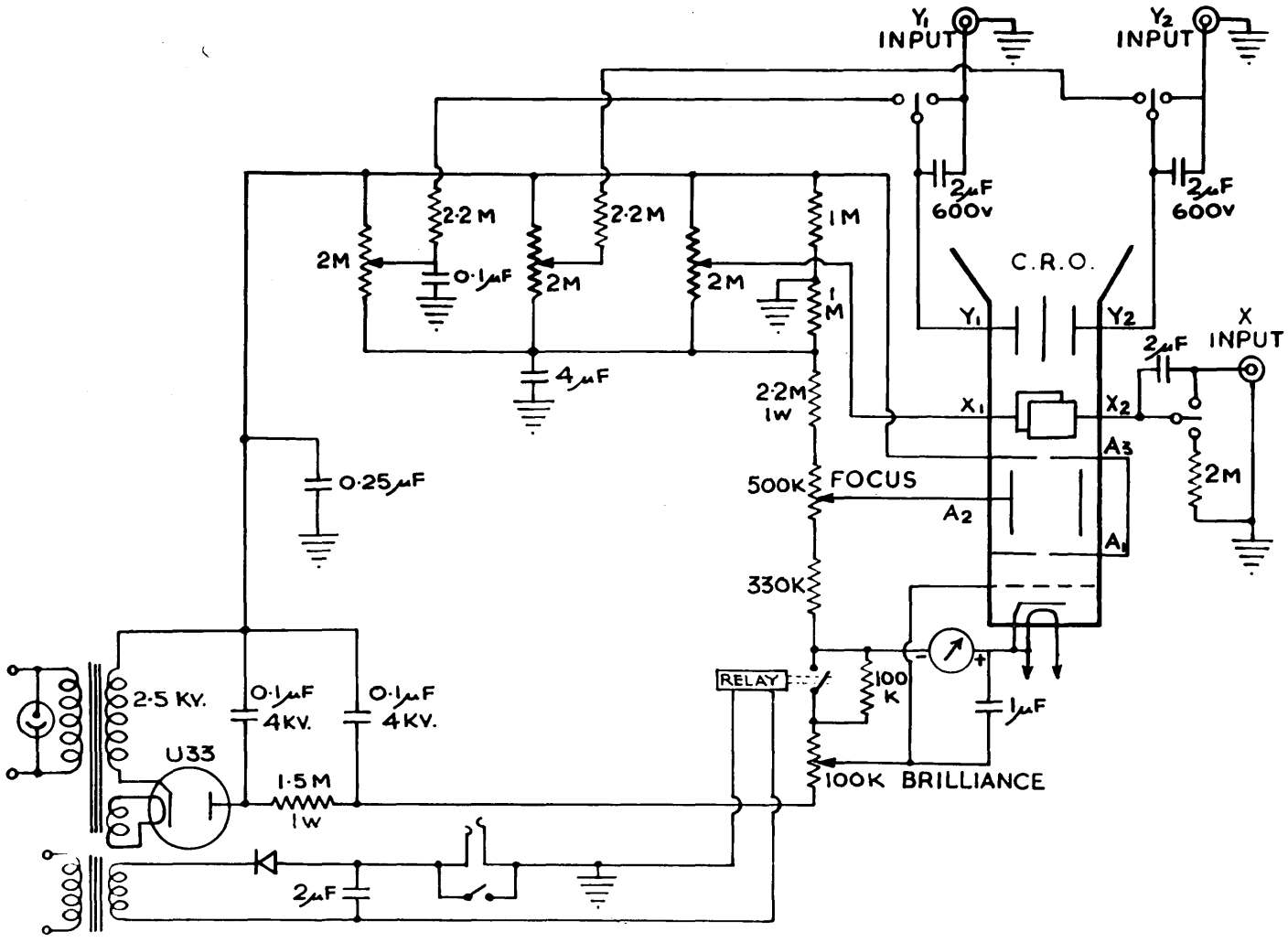
Circuit of low-frequency amplifier.




LOW-FREQUENCY AMPLIFIER

## PLATE 5

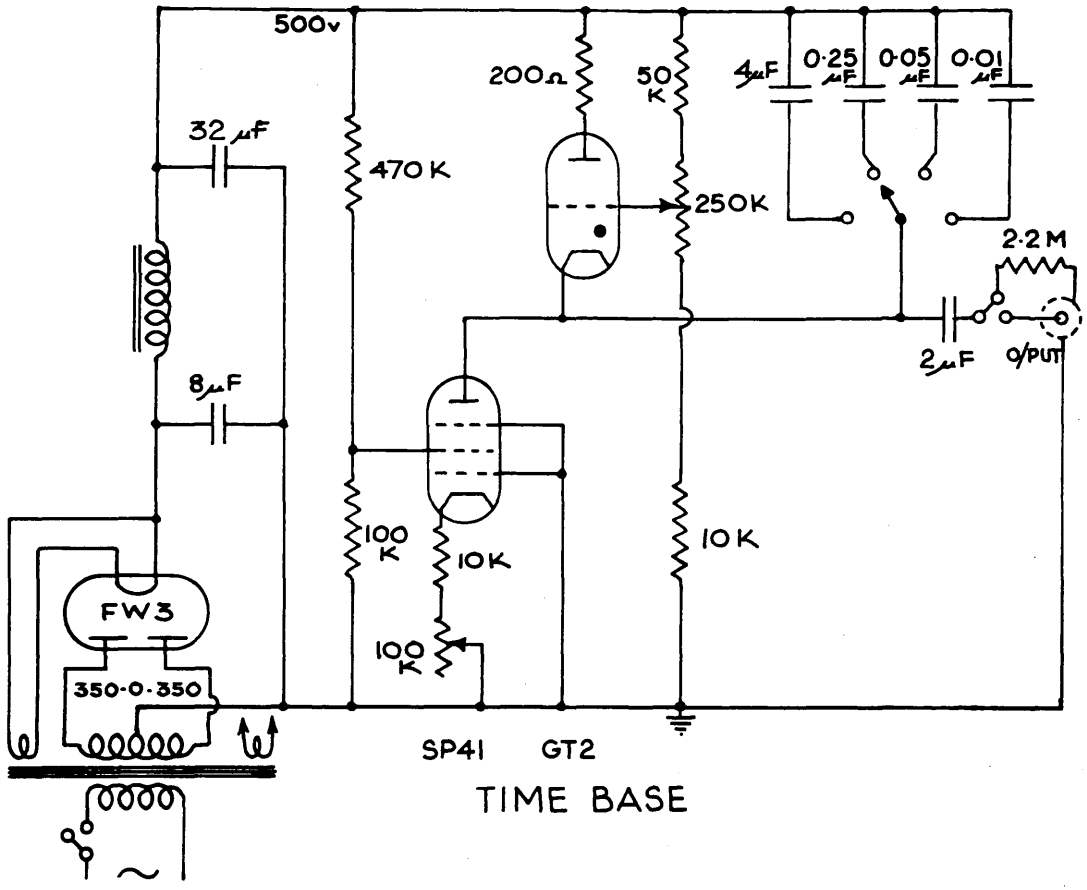
Circuit of display unit of phonocardiograph.




C.R.O. DISPLAY UNIT.

## PLATE 6

Circuit of time-base for cathode-ray oscillograph.


500v
200Ω
50 K
4μF
0·25 μF
0·05 μF
0·01 μF
32 μF
470 K
250K
2·2M
2μF
O/PUT
8μF
100 K
10K
10K
100 K
FW3
350-0-350
SP41
GT2

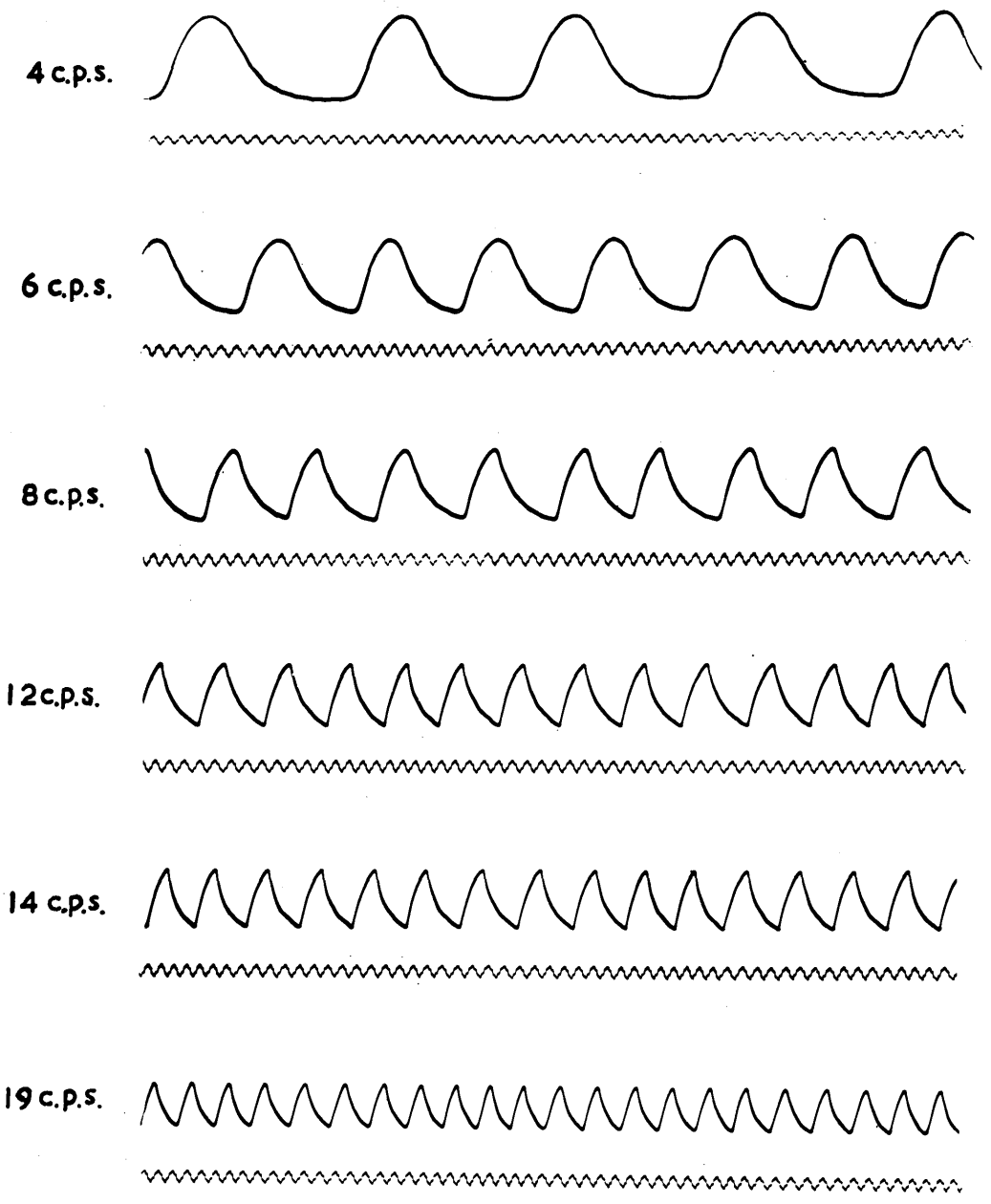
TIME BASE

## PLATE 7

Resonance test of venous pulse recorder

Upper records, oscillation of membrane

Lower records, time (50 cycles per sec.)




RESONANCE TEST OF VENOUS PULSE RECORDER

PART I

## PLATE 8

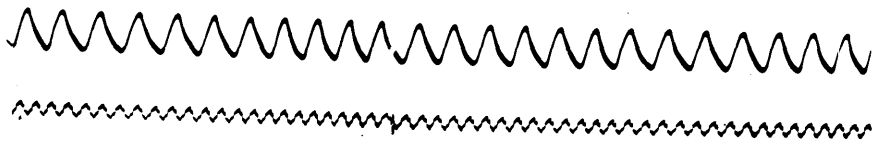
Resonance test of venous pulse recorder

(continued)

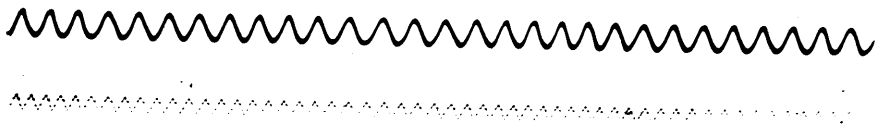
Upper records, oscillation of membrane

Lower records, time (50 cycles per sec.)

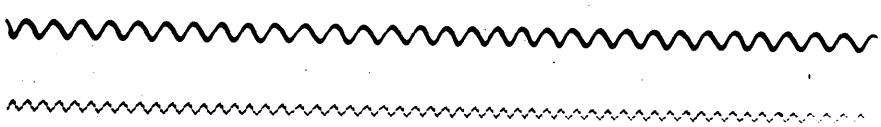
22 c.p.s.



27 c.p.s.

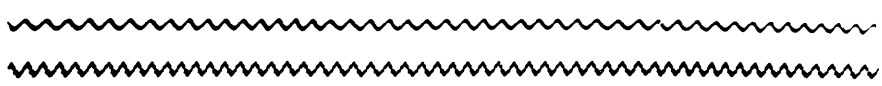


30 c.p.s.



35 c.p.s.

42 c.p.s.



# RESONANCE TEST OF VENOUS PULSE RECORDER

## PART 2

## APPENDIX C (continued)

## PLATES 9 - 10

### PHONOCARDIOGRAMS OF NORMAL SUBJECTS

In all records:

1. Jugular phlebogram
   Logarithmic P.C.G.

2. Jugular phlebogram
   Stethoscopic P.C.G.

3. Jugular phlebogram
   Linear P.C.G.

4. E.C.G. (Lead II)
   Linear P.C.G.

Time marking in 1/10 seconds.

The records were taken at the cardiac apex.

For details of interpretation see previous page.

## PLATE 9

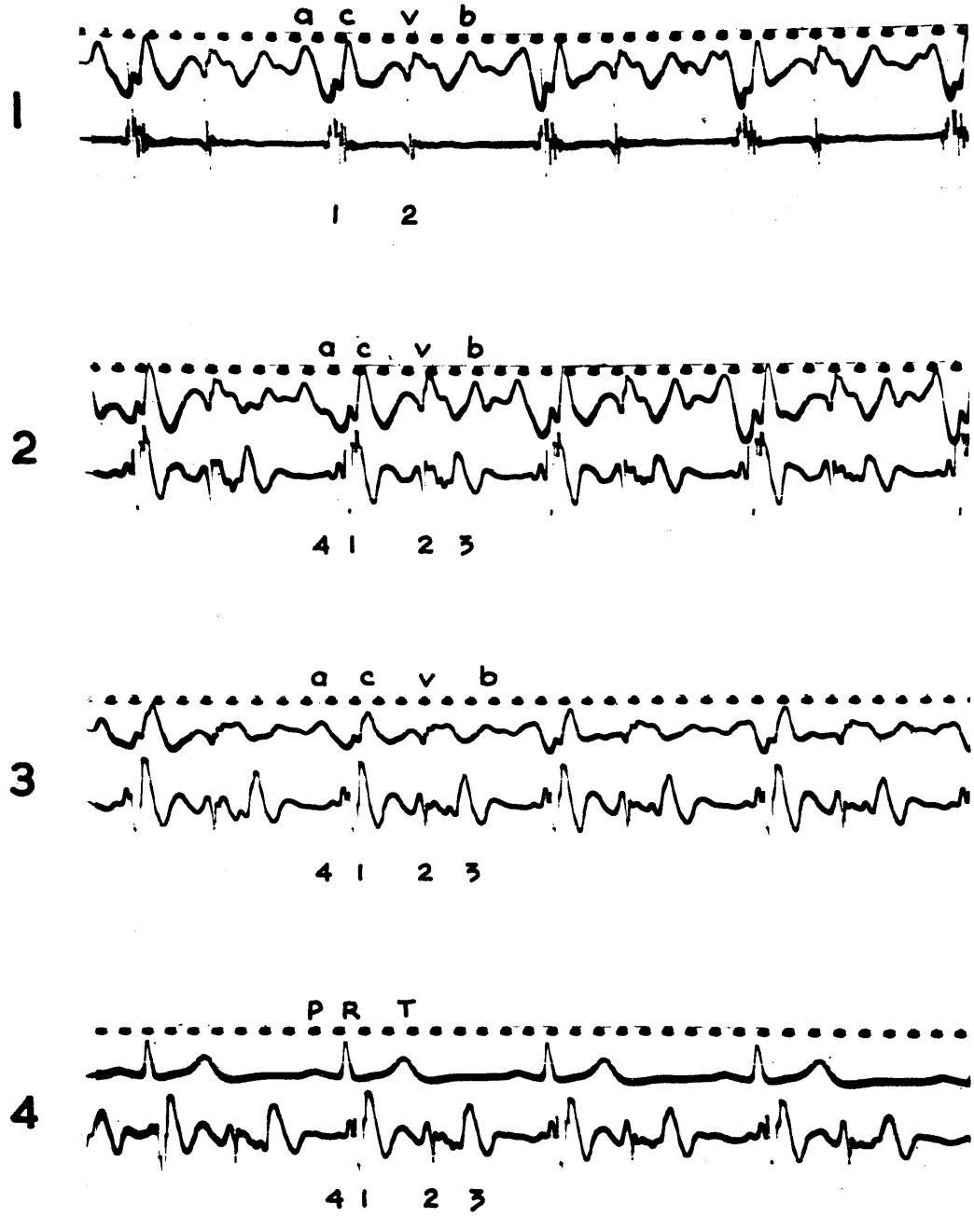
Normal subject No. 101 - male, age 27

Logarithmic P.C.G.

1st and 2nd heart sounds.

Stethoscopic and linear P.C.G.s

Vibrations at the time of occurrence of 1st, 2nd, 3rd and 4th heart sounds.




NORMAL SUBJECT NO. 101

## PLATE 10

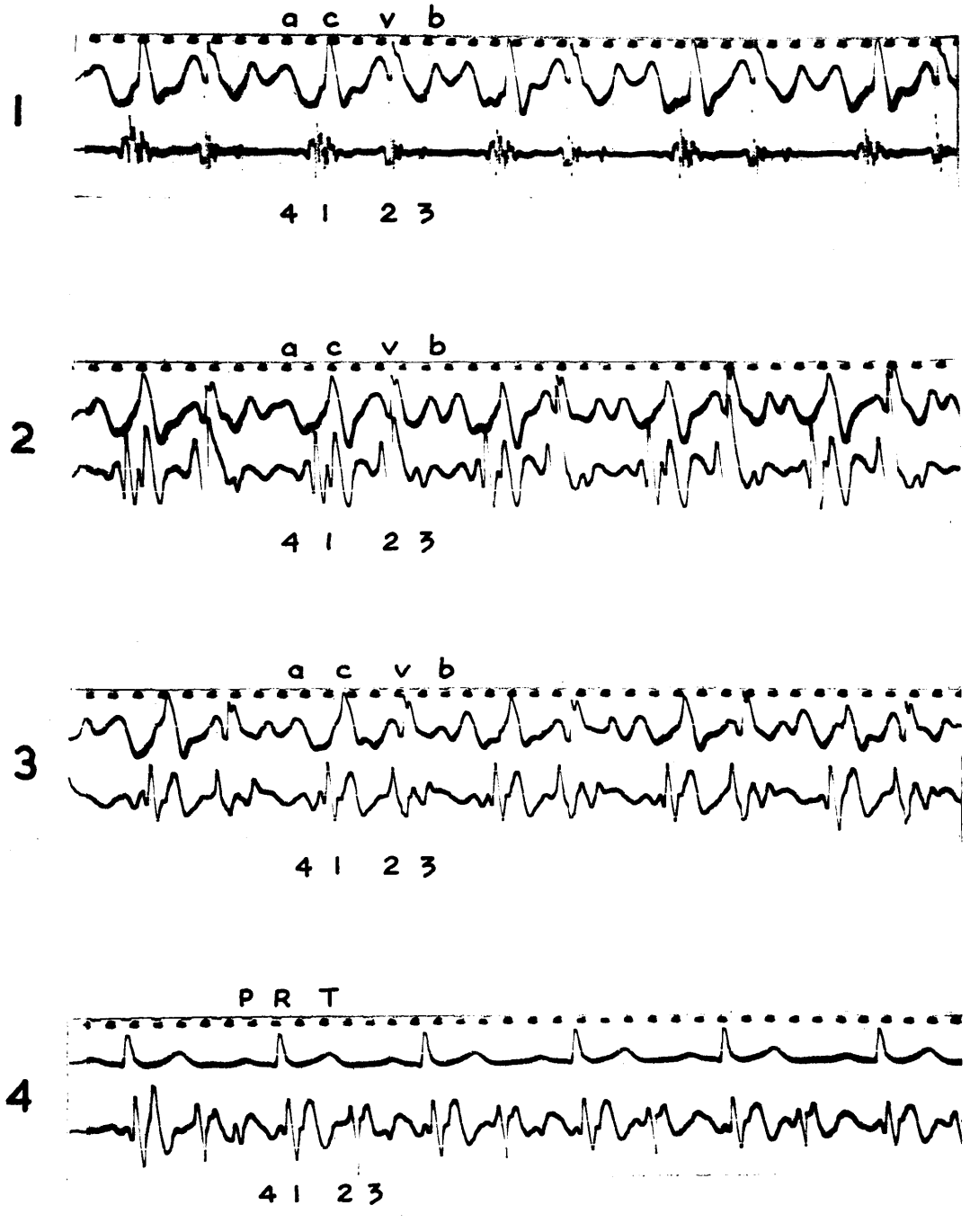
Normal subject No. 108 - male, age 24

Logarithmic P.C.G.

1st, 2nd, 3rd and 4th heart sounds.

Stethoscopic and linear P.C.G.s.

Vibrations at the time of occurrence of 1st, 2nd, 3rd and 4th heart sounds.




NORMAL SUBJECT NO. 108

## APPENDIX C (continued)

## PLATES 11 - 72

## PHONOCARDIOGRAMS OF PATIENTS WITH HEART DISEASE

In all records:

1. E.C.G. (Lead II)
   Logarithmic P.C.G.

2. Jugular phlebogram
   Logarithmic P.C.G.

3. Jugular phlebogram
   Linear P.C.G.

Time marking in 1/5 seconds

The photographs of the records are approximately the same size as the originals.

### CAPTIONS

The diagnosis is by the physician in charge.

The auscultatory findings reported are by the author.

Unless where otherwise stated, the notes refer to the region of the cardiac apex.

The results are summarized in Table 31.

For details of interpretation of the phonocardiograms of hospital patients see previous page.

## PLATE 11

### Patient No. 1 - male, age 44

Diagnosis
Mitral stenosis
Auricular fibrillation

Auscultation
1st and 2nd heart sounds; pansystolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; pansystolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.

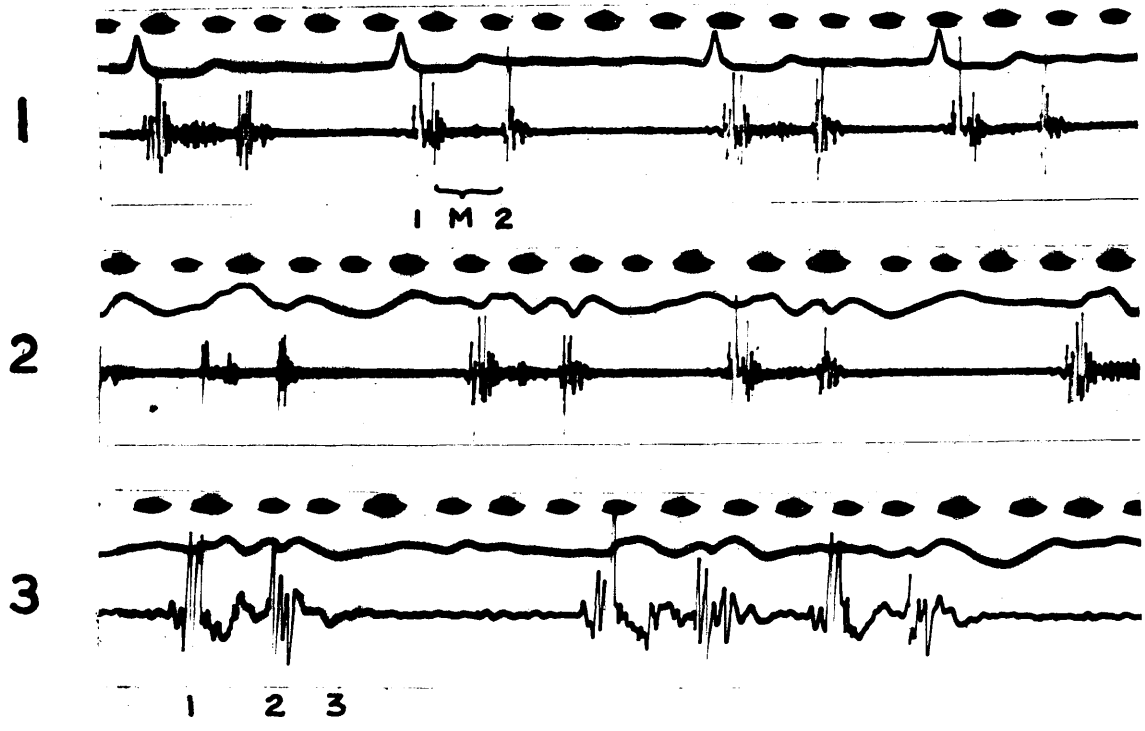
### Patient No. 2 - male, age 33

Diagnosis
Mitral stenosis
Auricular fibrillation

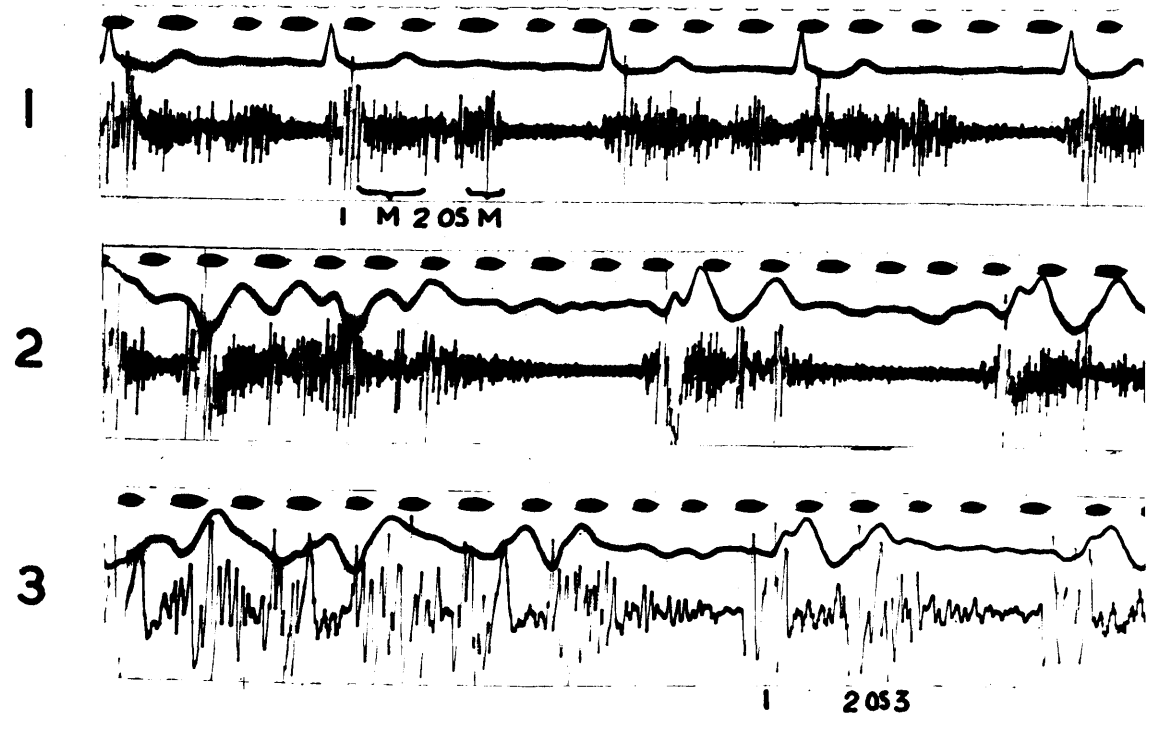
Auscultation
1st and 2nd heart sounds; early systolic and mid-diastolic murmurs.

Logarithmic P.C.G.
1st and 2nd heart sounds and mitral opening snap; pansystolic and mid-diastolic murmurs.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds and mitral opening snap.




PATIENT NO. 1 APEX




PATIENT NO. 2 APEX

## PLATE 12

### Patient No. 3 - male, age 9

Diagnosis

Mitral stenosis

Auscultation

1st and 2nd heart sounds; pansystolic murmur. At the pulmonary area, wide splitting of the 2nd heart sound; pansystolic murmur.

Logarithmic P.C.G.

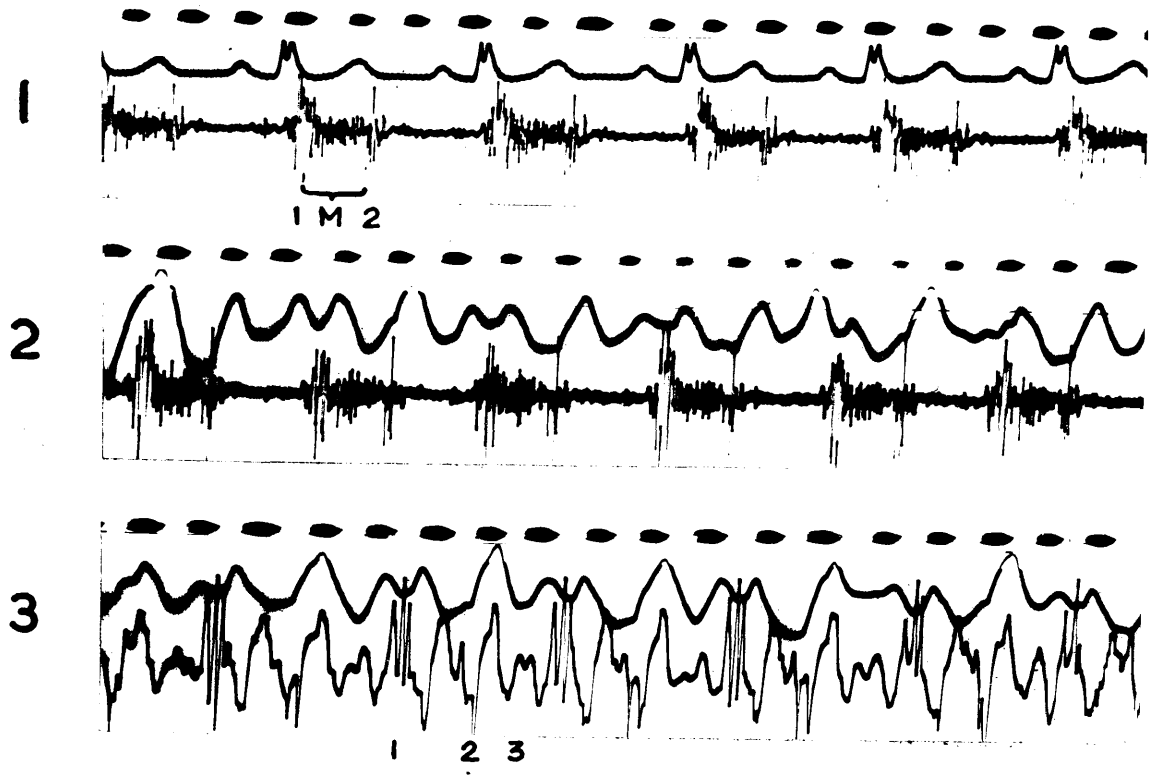
1st and 2nd heart sounds, with slight but definite splitting of the 2nd heart sound; pansystolic murmur. At the pulmonary area, wide splitting of the 2nd heart sound and mitral opening snap; pansystolic murmur continuing up to the second (the pulmonary) component of the split 2nd sound.

Linear P.C.G.

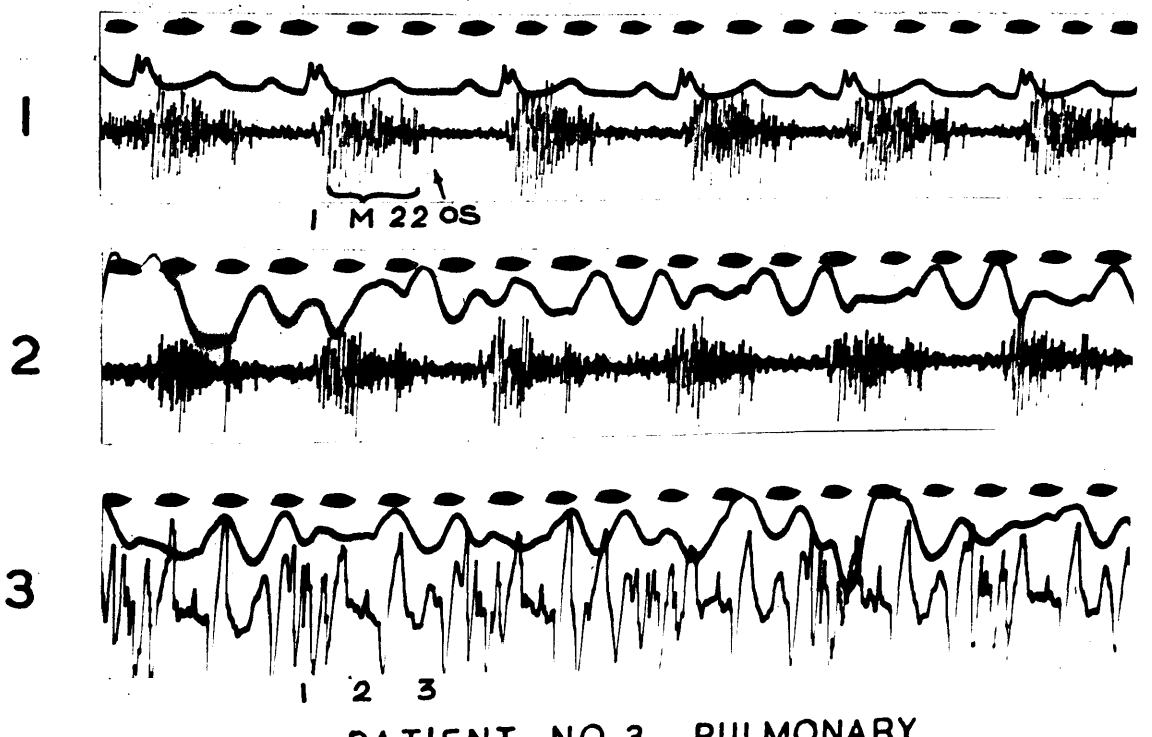
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.

Note

The prolongation of the systolic murmur up to the pulmonary component of the 2nd sound suggests that there is pulmonary stenosis.




PATIENT NO. 3 APEX




PATIENT NO. 3 PULMONARY

## PLATE 13

### Patient No. 4 - female, age 21

Diagnosis

Mitral stenosis
Aortic incompetence

Auscultation

Loud 1st and 2nd heart sounds; early and mid-systolic and early diastolic murmurs. At sternal end of 3rd left intercostal space, 1st and 2nd heart sounds; early systolic and early diastolic murmurs.

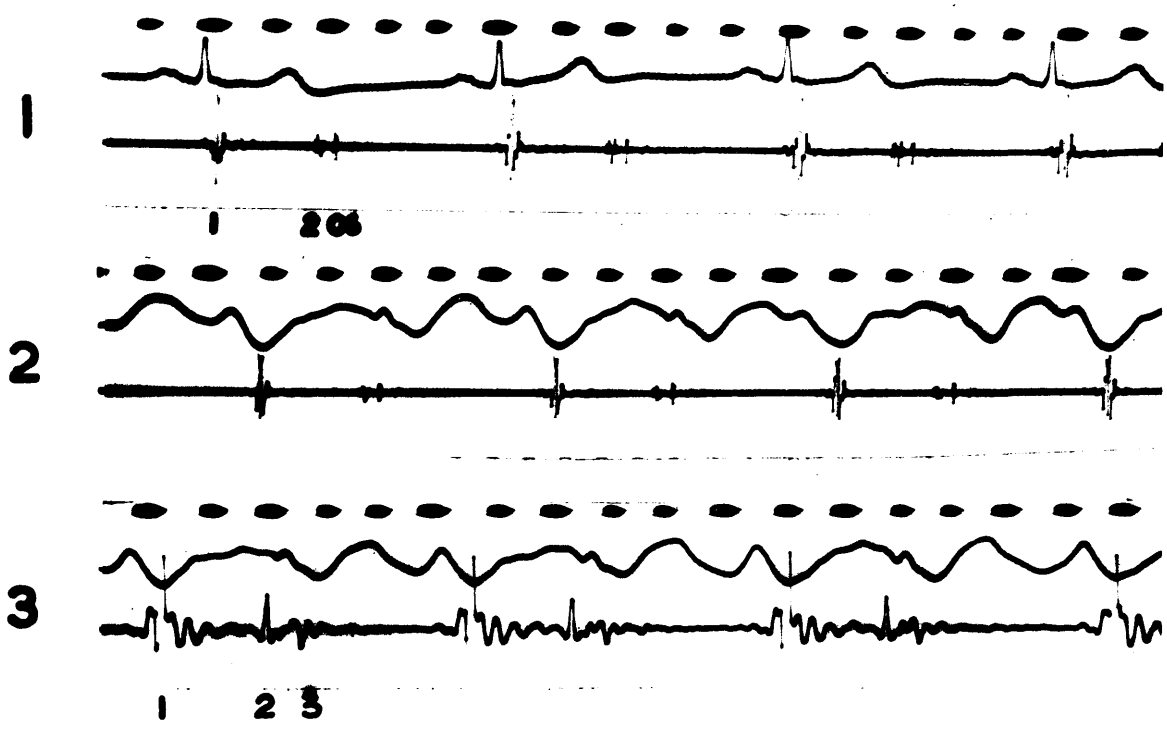
Logarithmic P.C.G.

1st and 2nd heart sounds and mitral opening snap; no murmur. At sternal end of 3rd left intercostal space, 1st and 2nd heart sounds and mitral opening snap; early systolic and early diastolic murmur.

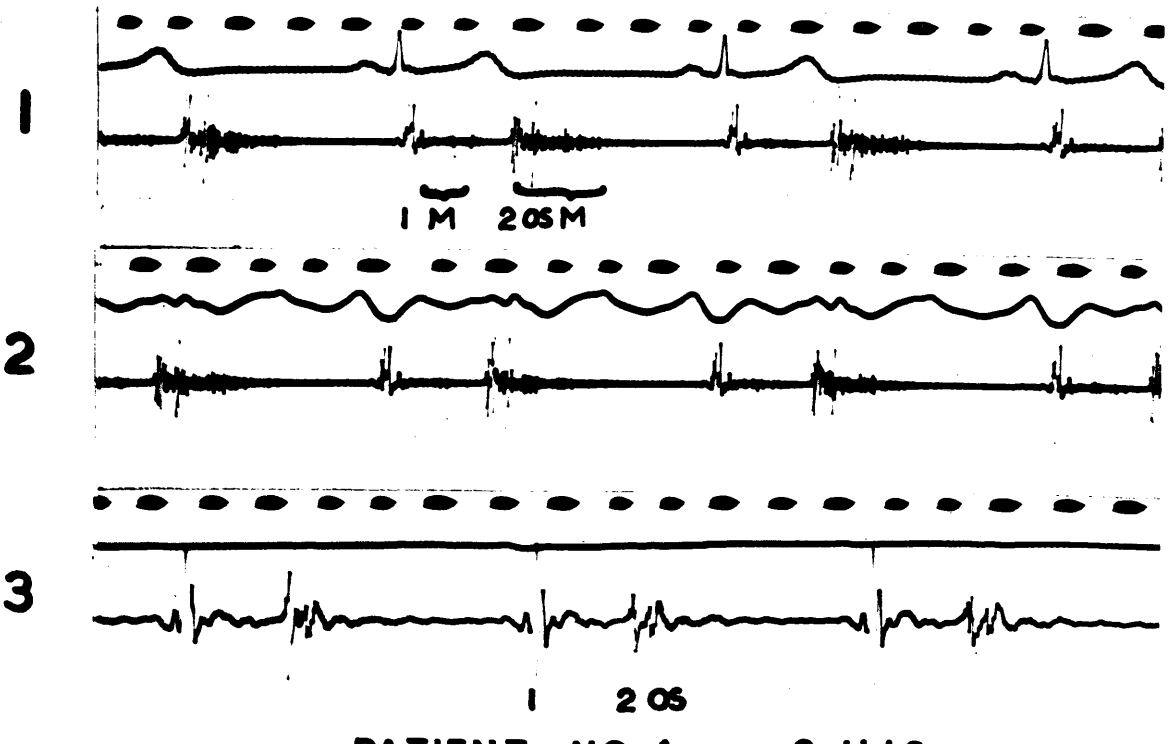
Linear P.C.G.

Vibrations at the time of 1st, 2nd, and 3rd heart sounds. At sternal end of 3rd left intercostal space, vibrations at the time of 1st and 2nd heart sounds and mitral opening snap.

The phlebogram tracing in the last records is defective owing to imperfect application of the phlebograph cup to the neck.




PATIENT NO.4 APEX




PATIENT NO. 4 3rd.L.I.S.

## PLATE 14

### Patient No. 5 - female, age 8

Diagnosis

Patent ductus arteriosus

Auscultation

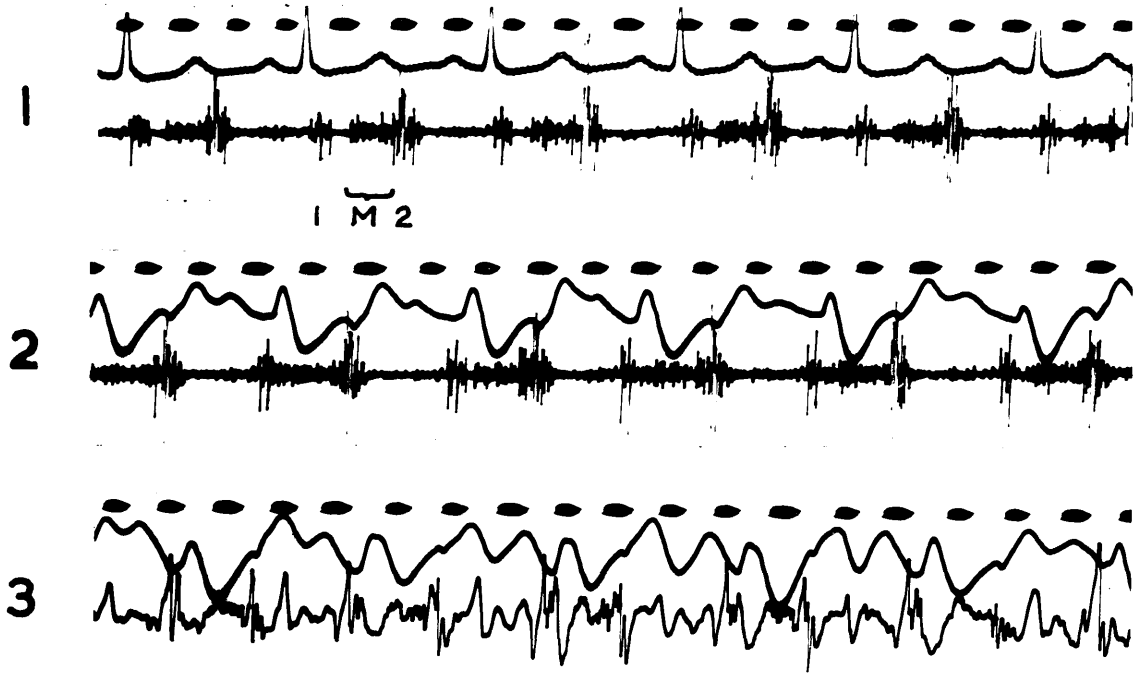
1st and 2nd heart sounds; pansystolic murmur. At the pulmonary area a continuous loud murmur, with maximum intensity in early diastole.

Logarithmic P.C.G.

1st and 2nd heart sounds; systolic murmur of varying length in successive cycles. At the pulmonary area, 1st heart sound; long, loud murmur, occupying the greater part of systole and diastole and masking 2nd heart sound.

Linear P.C.G.

Vibrations at the time of 1st, 2nd, and 3rd heart sounds.

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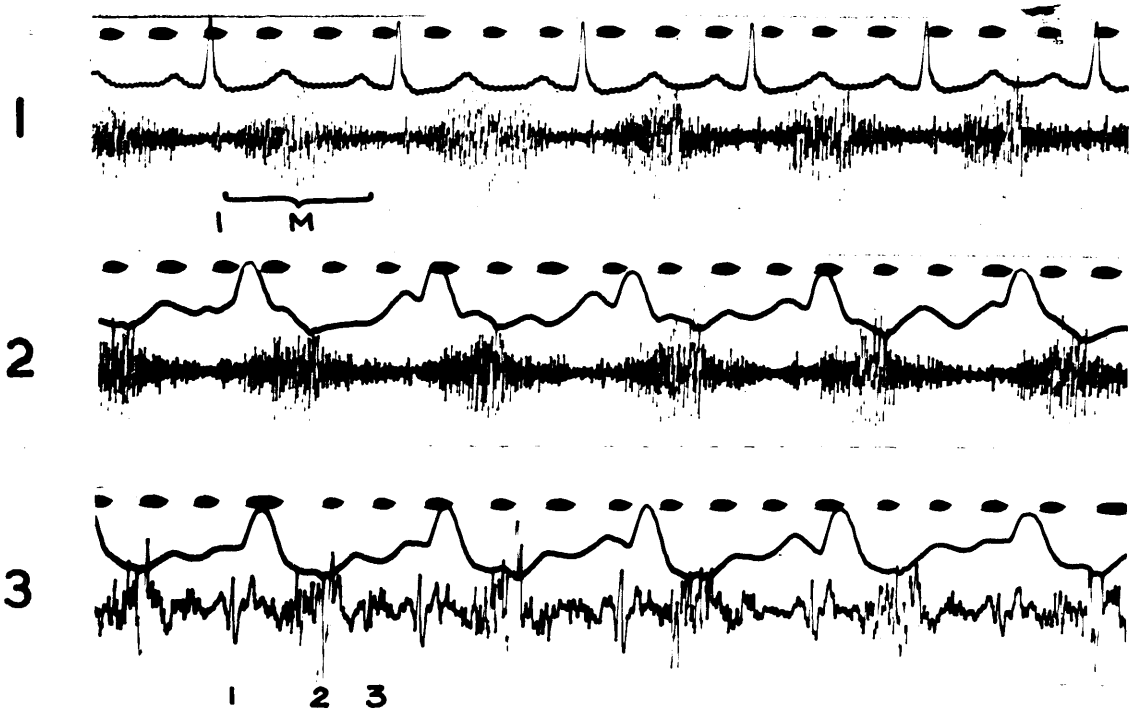



PATIENT NO.5 PULMONARY

## PLATE 15

### Patient No. 6 - female, age 23

Diagnosis

Mitral stenosis
Aortic incompetence

Auscultation

1st and 2nd heart sounds, the former much louder; long diastolic murmur with presystolic accentuation. No murmur at the aortic area.

Logarithmic P.C.G.

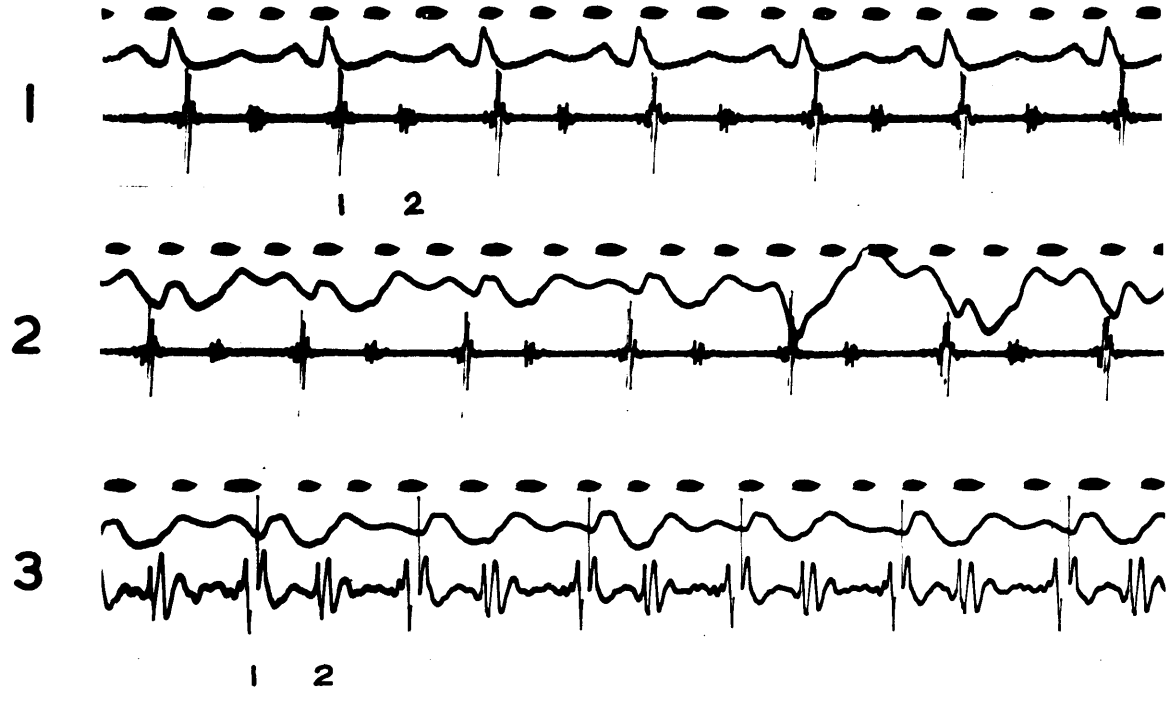
1st and 2nd heart sounds; no murmur. At sternal end of 3rd left intercostal space, 1st and 2nd heart sounds; no murmur.

Linear P.C.G.

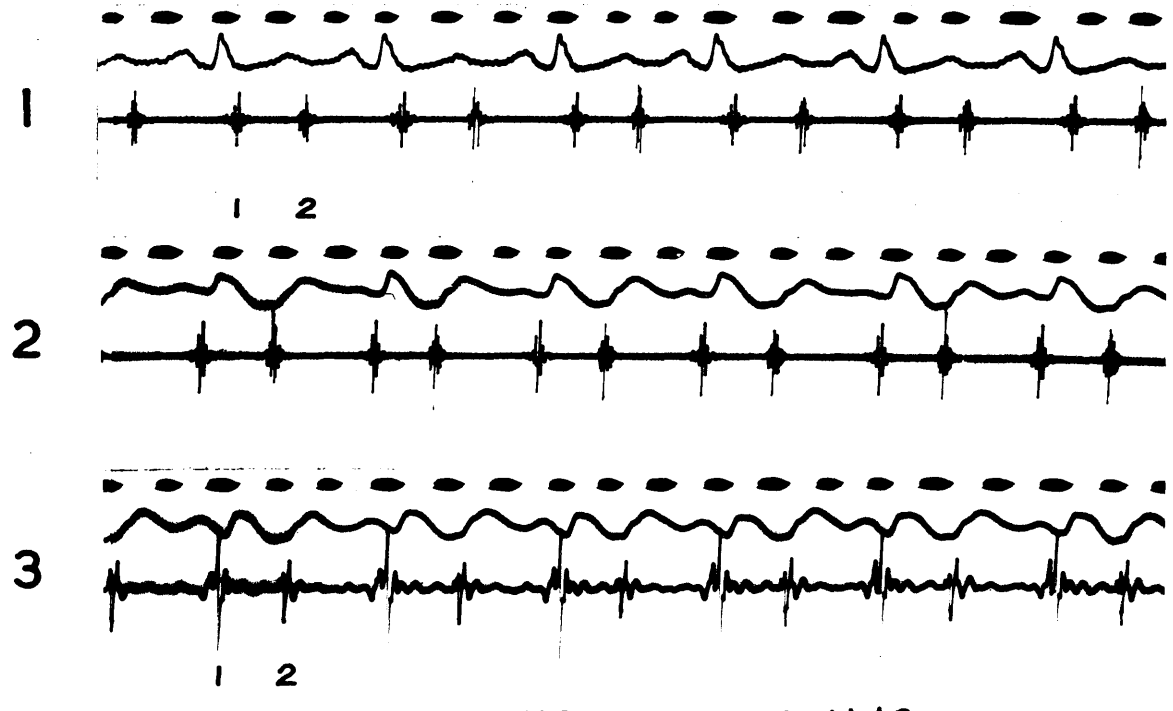
Vibrations at the time of 1st and 2nd heart sounds.

Note

No evidence was found to support the hospital diagnosis of aortic incompetence. The absence of the mitral diastolic murmur on the logarithmic P.C.G. can be attributed to the gain reduction necessary to record the whole extent of the deflections due to the loud 1st sound.




PATIENT NO.6 APEX




PATIENT NO.6 3rd.L.I.S.

## PLATE 16

### Patient No. 7 - female, age 20

Diagnosis
Mitral stenosis

Auscultation
1st and 2nd heart sounds; early systolic and pandiastolic murmurs.

Logarithmic P.C.G.
1st and 2nd heart sounds; mid- and late diastolic murmur with presystolic accentuation.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds; indication of low-frequency presystolic murmur.

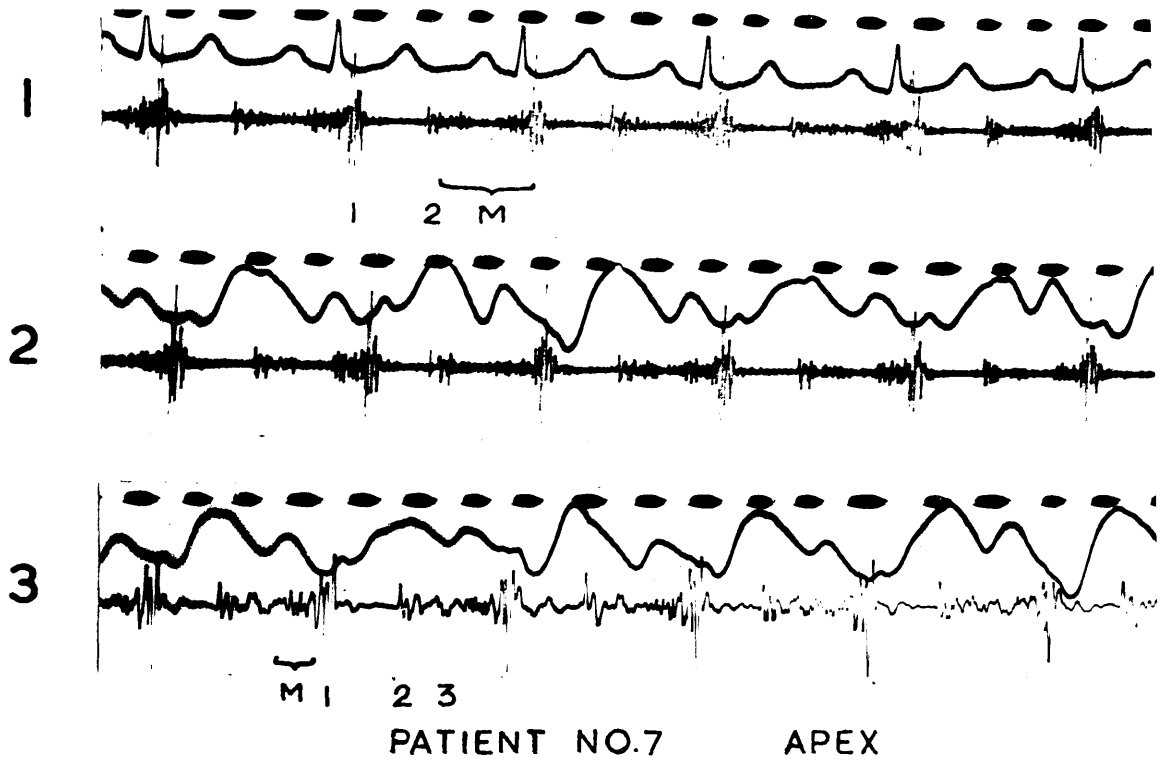
### Patient No. 8 - male, age 52

Diagnosis
Arterial hypertension
Anterior myocardial infarction
Cardiac failure

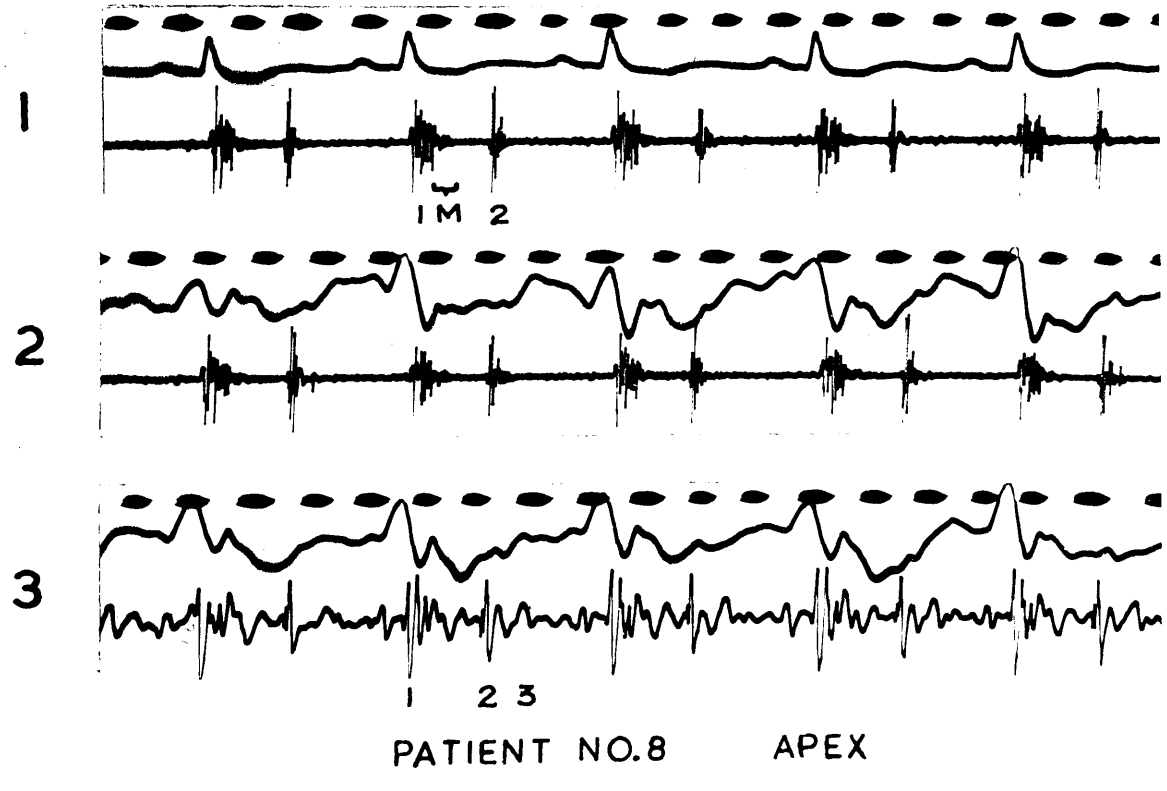
Auscultation
1st and 2nd heart sounds; early systolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; early systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.




PATIENT NO.7 APEX




PATIENT NO.8 APEX

# PLATE 17

## Patient No. 9 - female, age 23

Diagnosis

Mitral stenosis

Auscultation

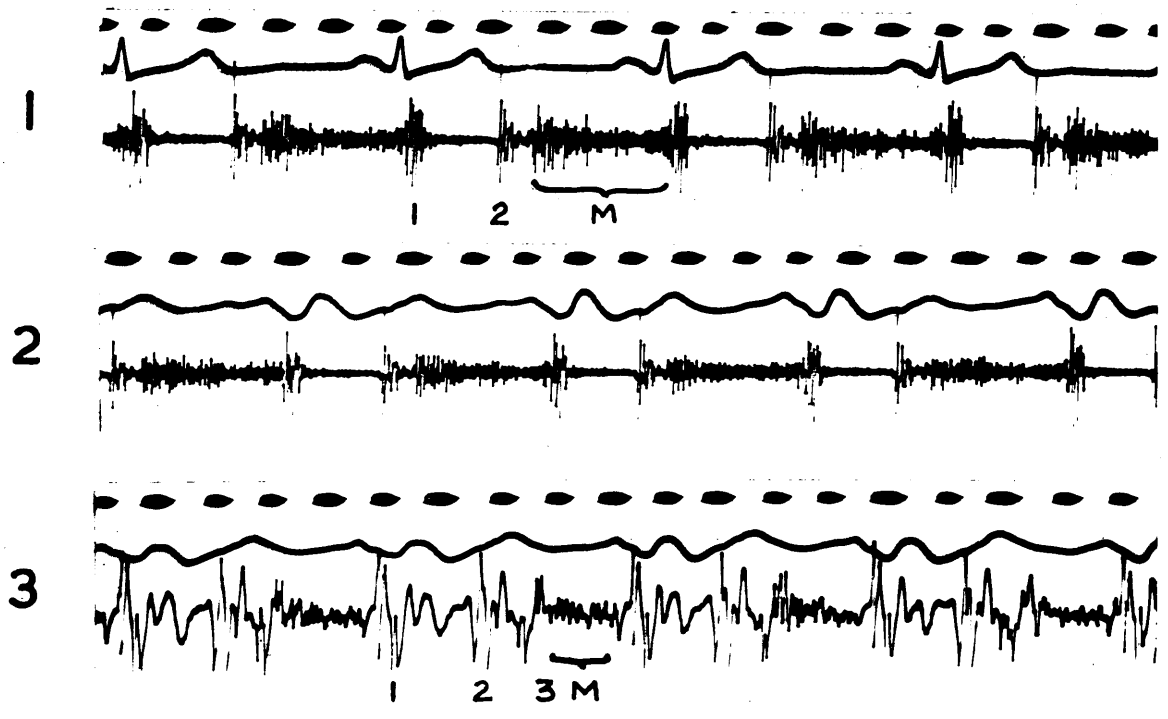
1st and 2nd heart sounds; loud pandiastolic murmur with presystolic accentuation.

Logarithmic P.C.G.

1st and 2nd heart sounds; long diastolic murmur, starting an appreciable time after 2nd heart sound.

Linear P.C.G.

Vibrations at the time of 1st, 2nd, and 3rd heart sounds; indication of diastolic murmur.




PATIENT NO. 9 APEX

## PLATE 18

### Patient No. 10 - female, age 38

Diagnosis

Mitral stenosis
Aortic incompetence

Auscultation

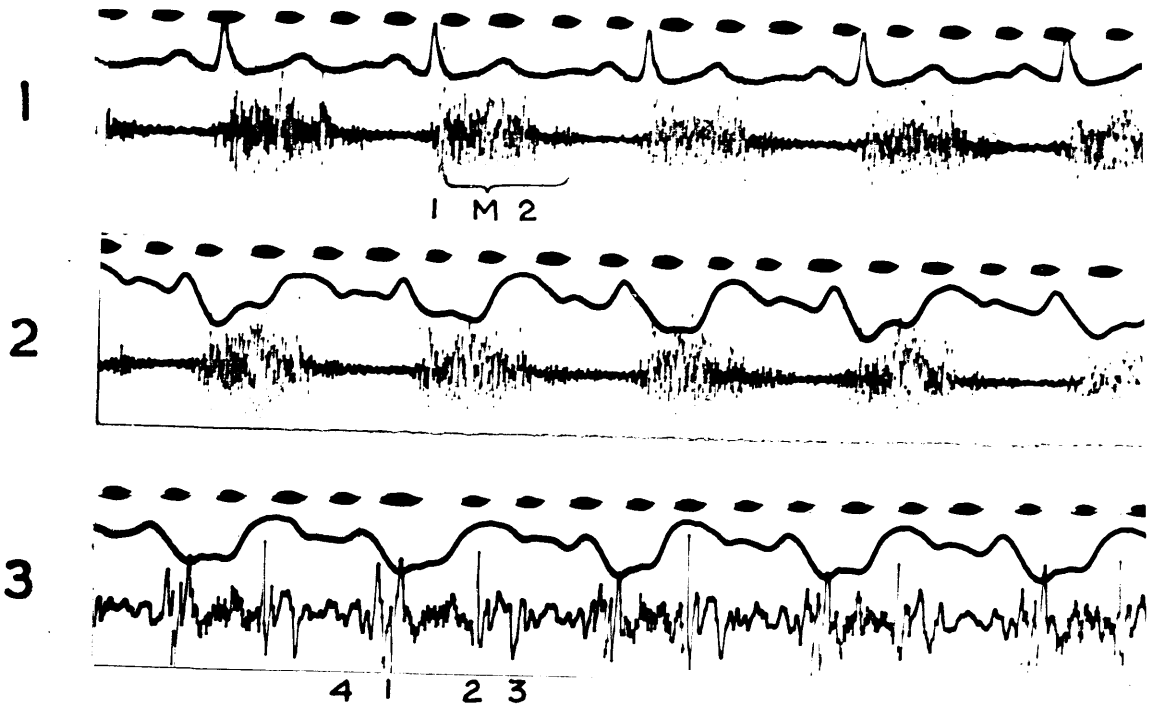
1st and 2nd heart sounds; continuous murmur. At sternal end of 3rd left intercostal space, 1st and 2nd heart sounds; systolic murmur.

Logarithmic P.C.G.

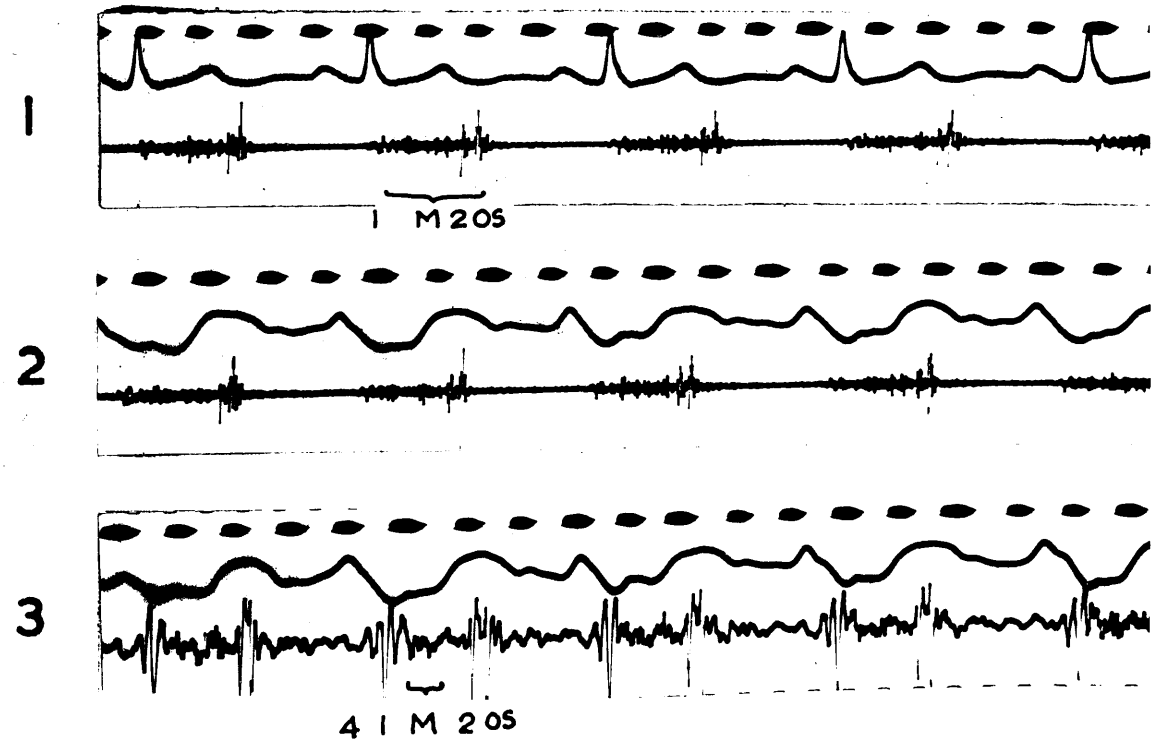
1st and 2nd heart sounds; pansystolic and early diastolic murmurs. At sternal end of 3rd left intercostal space, 1st and 2nd heart sounds and mitral opening snap; pansystolic and early diastolic murmurs.

Linear P.C.G.

Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds; indication of systolic murmur. At sternal end of 3rd left intercostal space, vibrations at the time of 1st, 2nd, and 4th heart sounds and mitral opening snap; indication of systolic murmur.




PATIE NT NO.10 APEX




PATIENT NO. 10 3rd.L.I.S.

# PLATE 19

## Patient No. 11 - female, age 60

**Diagnosis**

Arterial hypertension
Myocardial degeneration
Congestive cardiac failure
Diabetes mellitus
Hemiplegia (R)

**Auscultation**

1st and 2nd heart sounds; no murmur.

**Logarithmic P.C.G.**

1st, 2nd, and 3rd heart sounds (protodiastolic gallop); no murmur.

The 'mush' on the base-line is due to the high gain of the amplifier needed to record the faint heart sounds.

**Linear P.C.G.**

Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.

## Patient No. 12 - male, age 70

**Diagnosis**

Arterial hypertension
Left heart strain

**Auscultation**

1st and 2nd heart sounds, with 2nd sound much louder; soft, early systolic murmur.

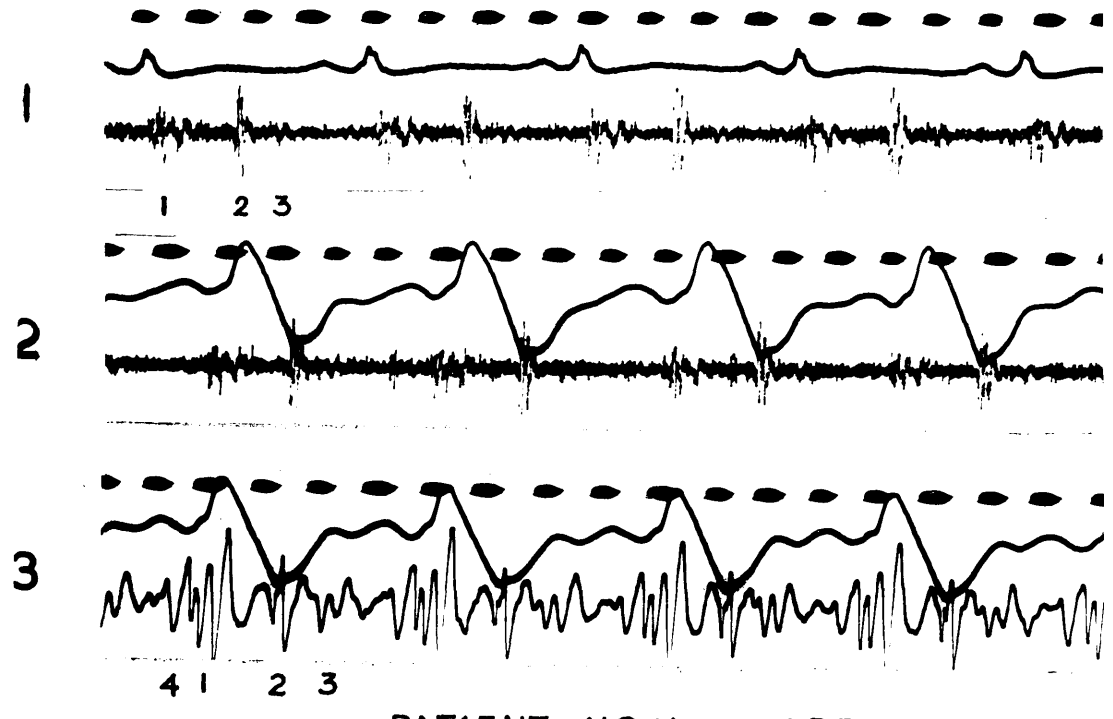
**Logarithmic P.C.G.**

1st and 2nd heart sounds and systolic extra sound; 1st sound is of low intensity and prolonged duration but no murmur is recorded.

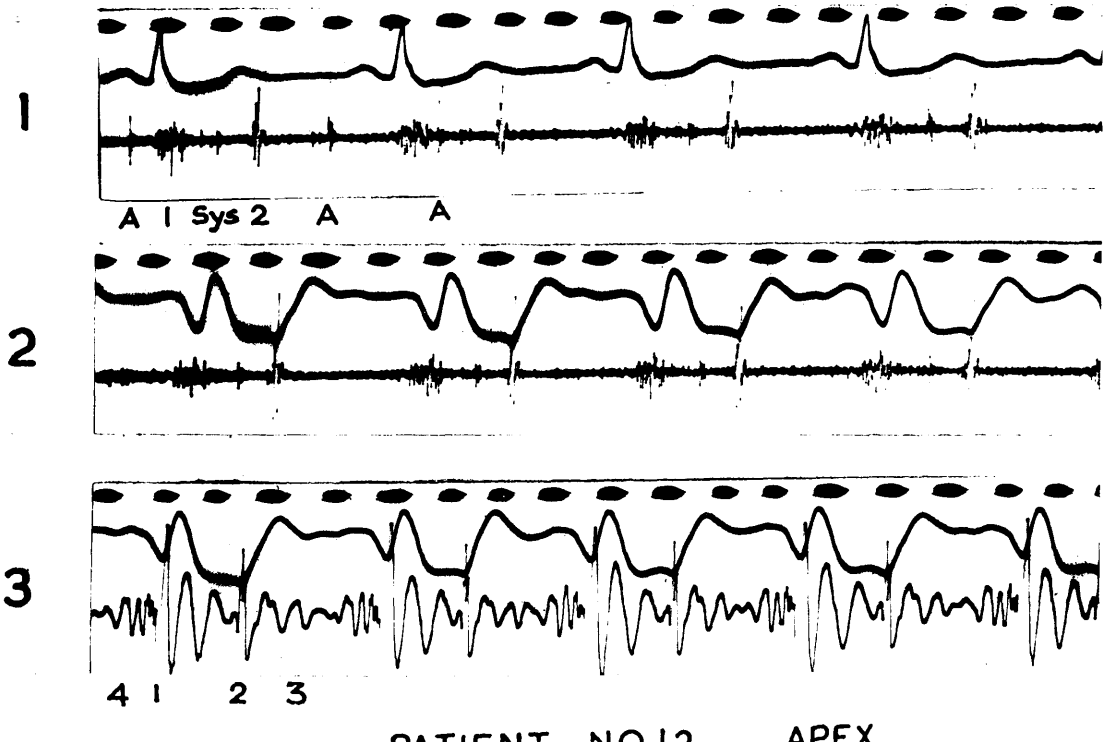
The deflections marked 'A' on record 1 are identified as artefacts because they are not repeated in successive cycles. They are probably instrumental in origin.

**Linear P.C.G.**

Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO.11 APEX




PATIENT NO.12 APEX

## PLATE 20

### Patient No. 13 - male, age 59

Diagnosis
Arterial hypertension
Left heart strain

Auscultation
1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.

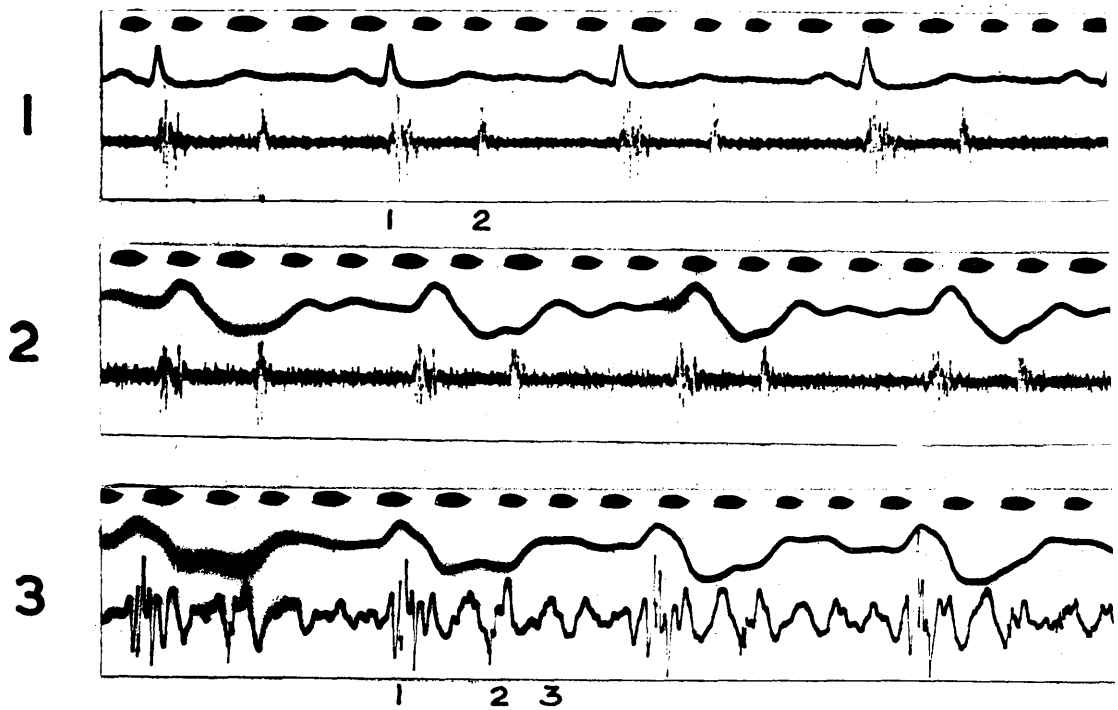
### Patient No. 14 - male, age 48

Diagnosis
Arterial hypertension
Cor pulmonale
Myocardial degeneration
Chronic bronchitis and emphysema

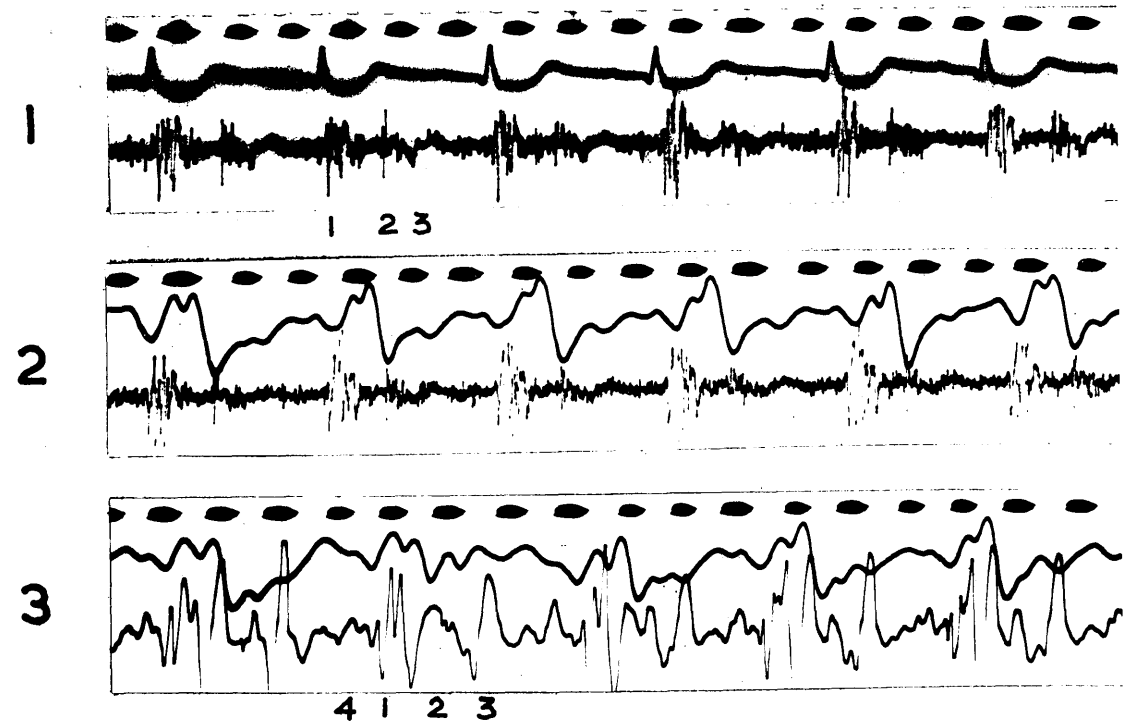
Auscultation
Very faint 1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st, 2nd and 3rd heart sounds (protodiastolic gallop); no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 13 APEX




PATIENT NO. 14 APEX

## PLATE 21

### Patient No. 15 - male, age 69

Diagnosis
Arterial hypertension
Anterior myocardial infarction
Congestive cardiac failure
Left pyonephrosis

Auscultation
1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 4th heart sounds.

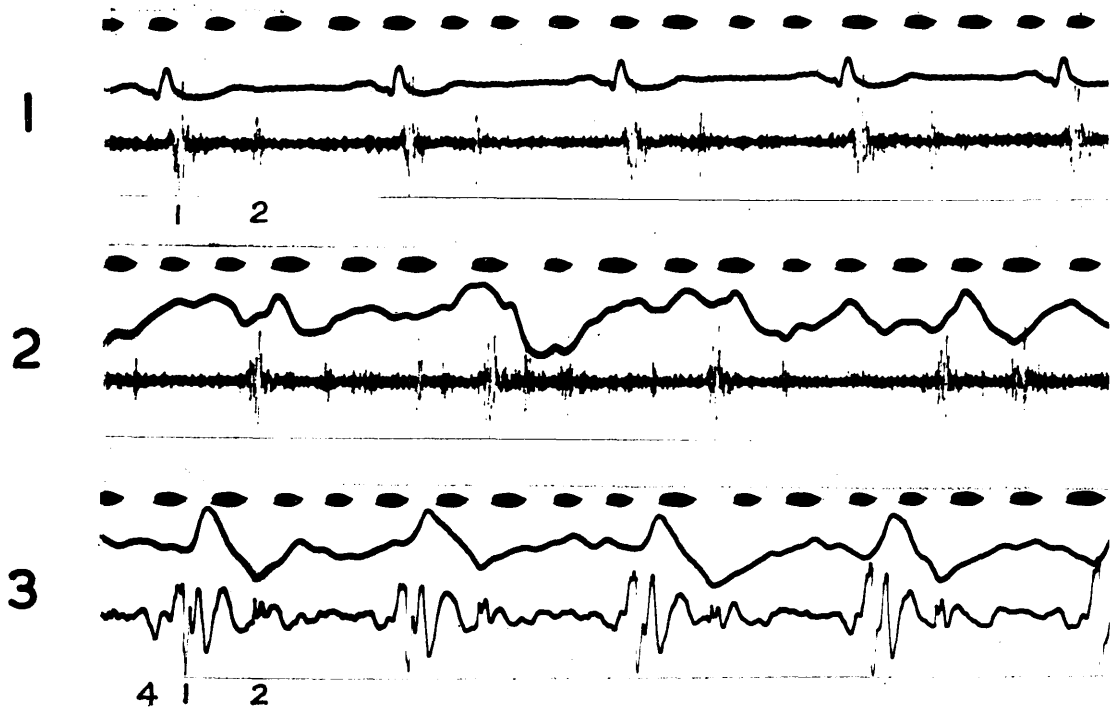
### Patient No. 16 - male, age 52

Diagnosis
Angina pectoris

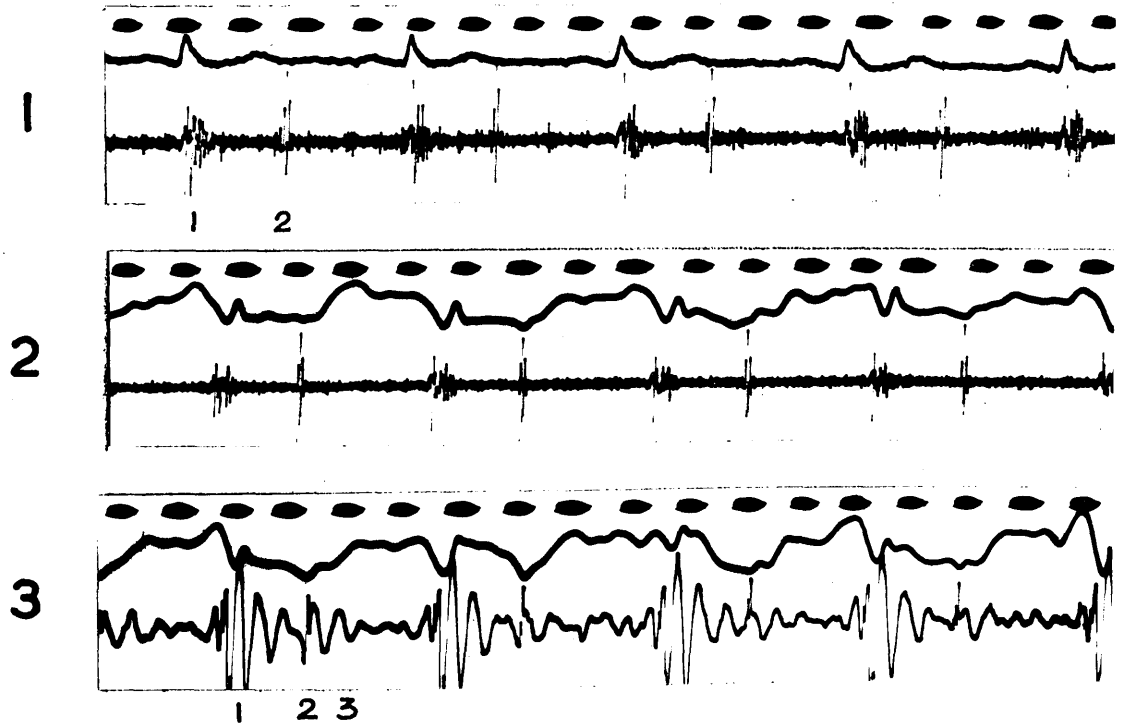
Auscultation
1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.




PATIENT NO. 15 APEX




PATIENT NO. 16 APEX

## PLATE 22

### Patient No. 17 - male, age 68

Diagnosis
Myocardial degeneration Duodenal Ulcer

Auscultation
1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.

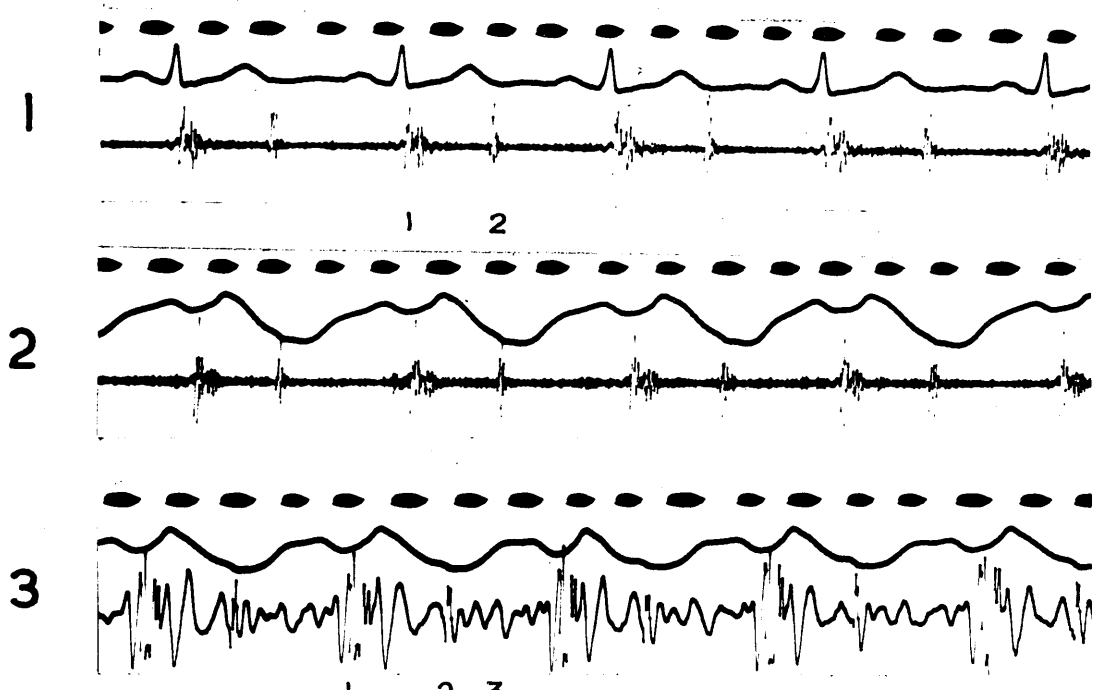
### Patient No. 18 - female, age 15

Diagnosis
Mitral incompetence.

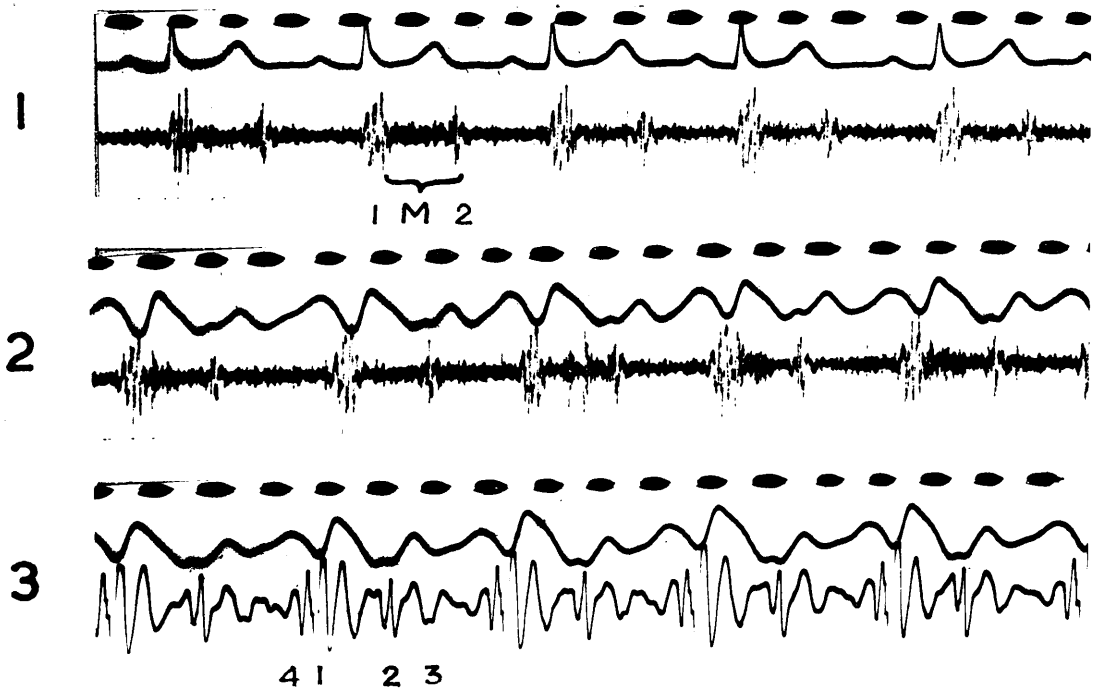
Auscultation
1st and 2nd heart sounds; pansystolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; pansystolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 17 APEX




PATIENT NO. 18 APEX

# PLATE 23

## Patient No. 19 - female, age 76

Diagnosis
Arterial hypertension
Myocardial disease
Left heart strain

Auscultation
Faint 1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; no murmur.
Record 2 shows artefacts (A) and a breath sound (BS).

Linear P.C.G.
Vibrations at the time of 1st and 2nd heart sounds.

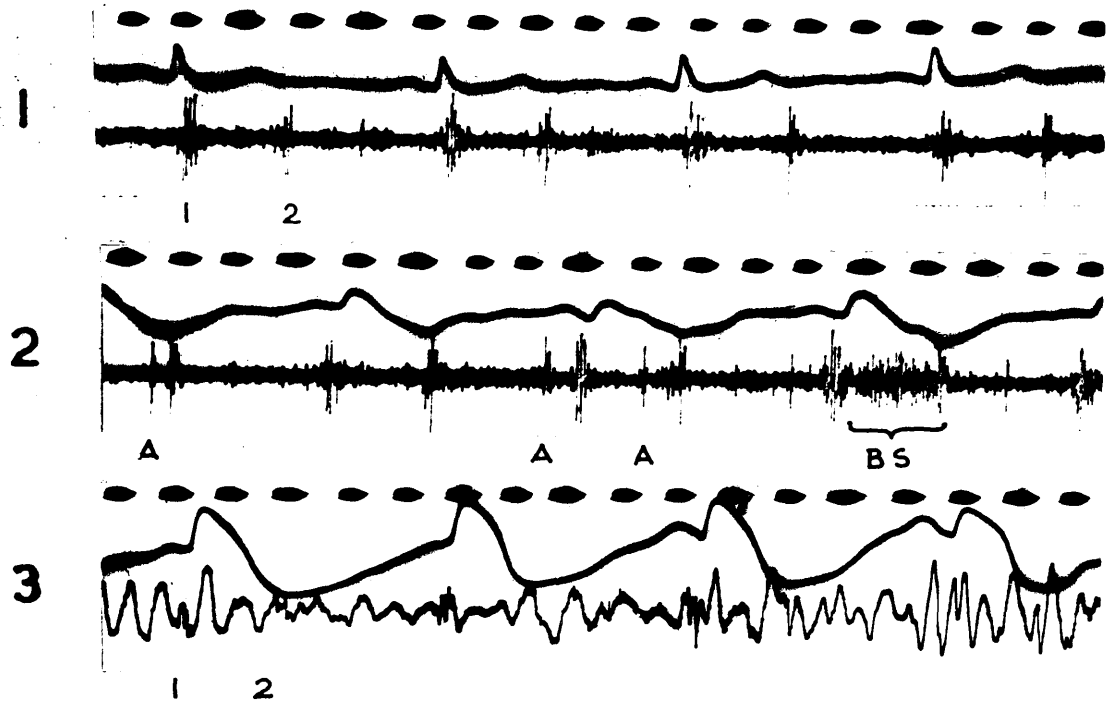
## Patient No. 20 - female, age 19

Diagnosis
Mitral incompetence

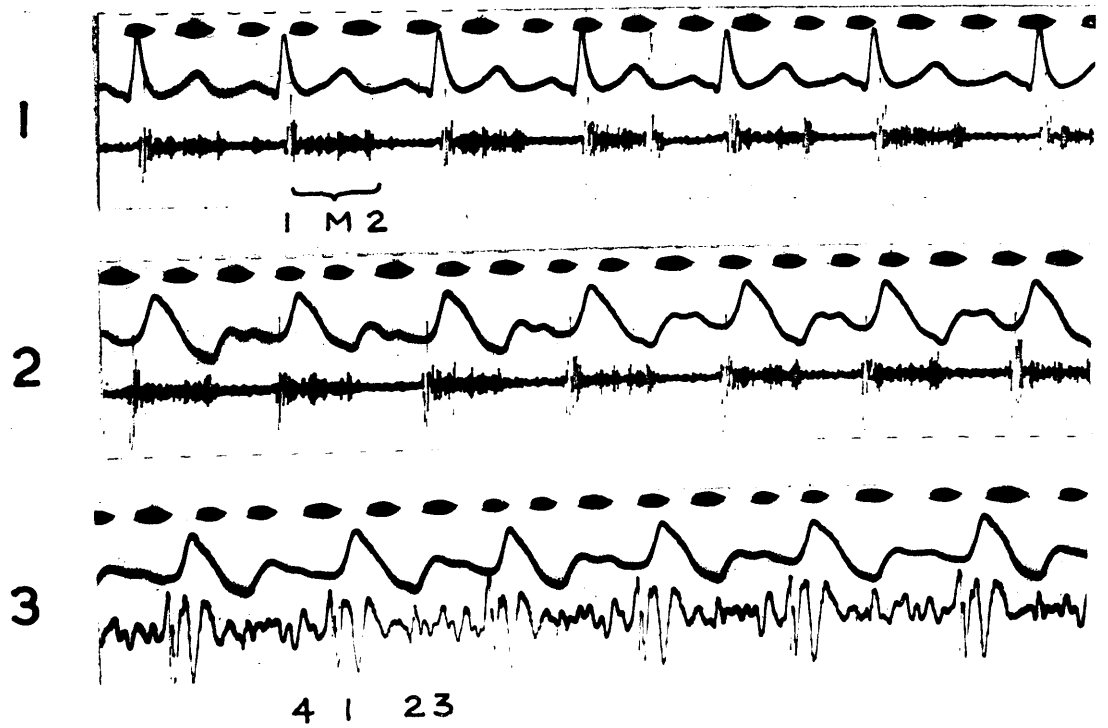
Auscultation
Loud 1st and normal 2nd sound; pansystolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; pansystolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 19 APEX




PATIENT NO. 20 APEX

## PLATE 24

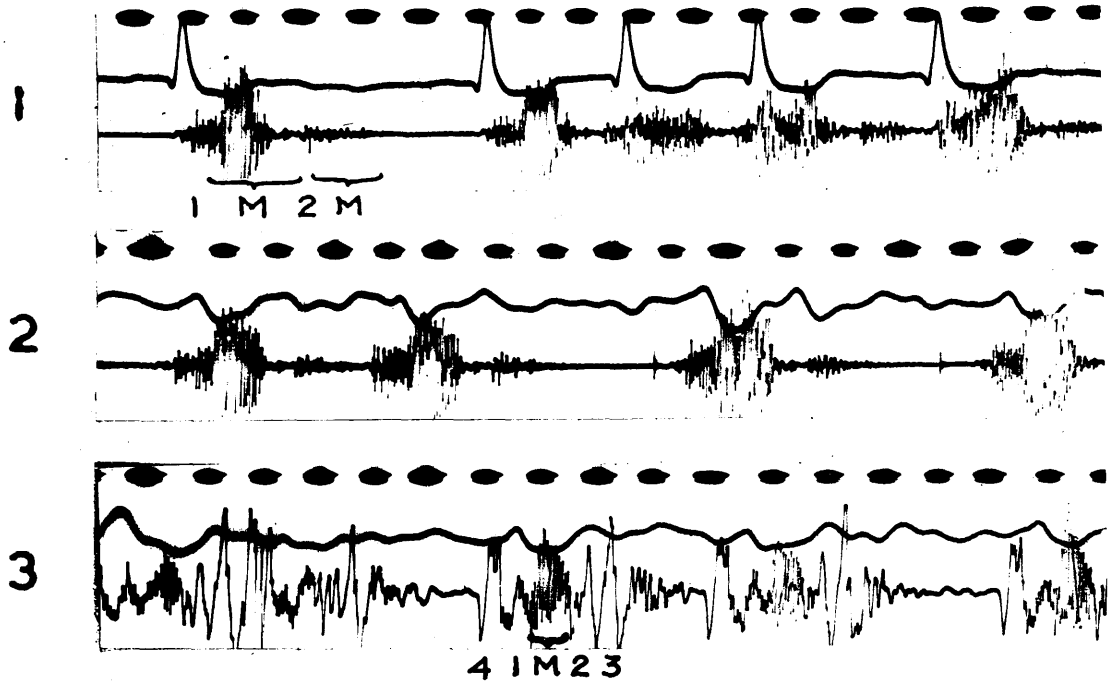
### Patient No. 21 - female, age 14

Diagnosis
Mitral stenosis
Auricular fibrillation

Auscultation
1st and 2nd heart sounds; continuous murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; pansystolic murmur, with mid-systolic accentuation, and early diastolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds; indication of mid-systolic murmur.




PATIENT NO. 21 APEX

## PLATE 25

### Patient No. 22 - female, age 26

**Diagnosis**

Mitral and aortic incompetence
Acute rheumatic carditis

**Auscultation**

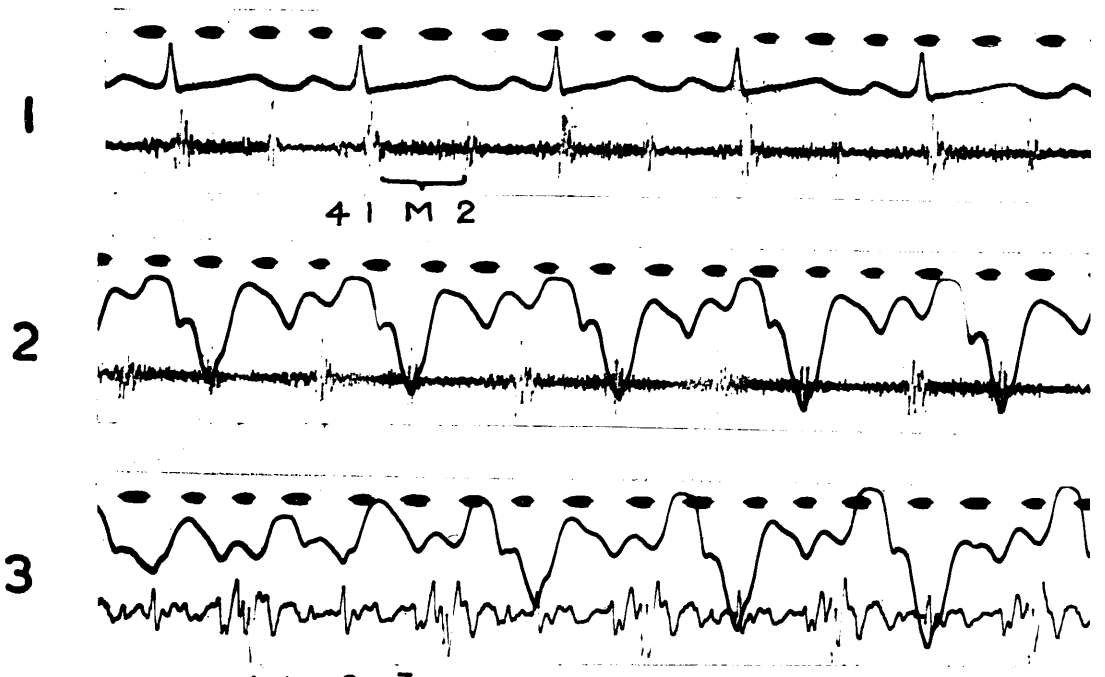
1st and 2nd heart sounds; pansystolic murmur. At sternal end of 2nd right intercostal space, heart sounds masked by loud, continuous murmur with systolic accentuation.

**Logarithmic P.C.G.**

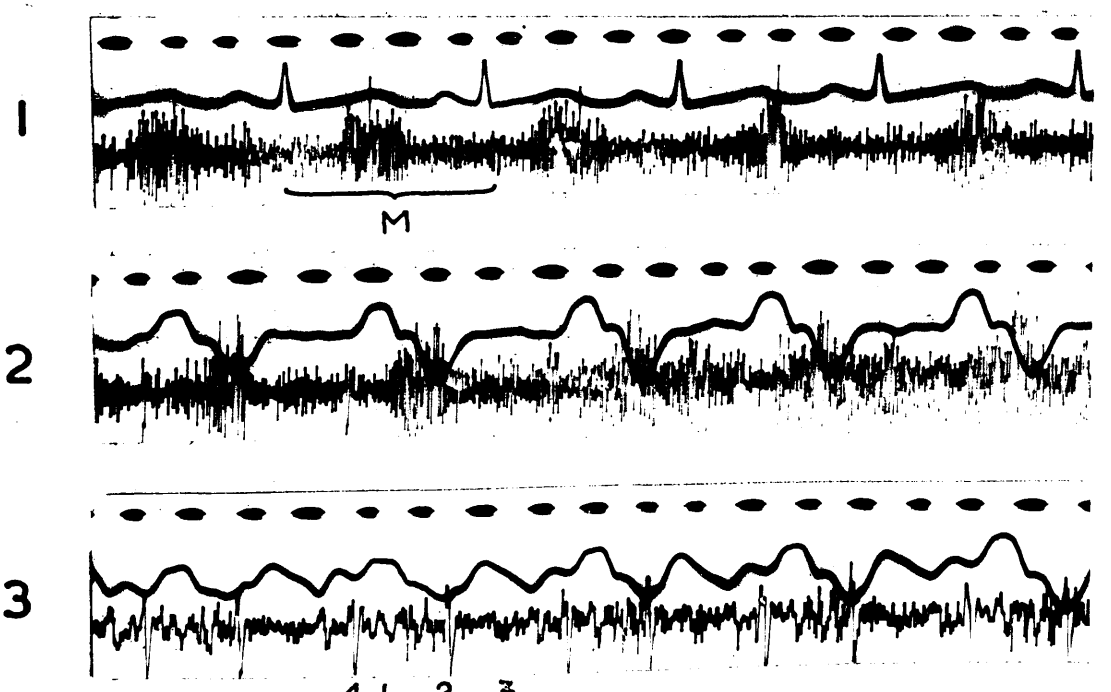
1st, 2nd and 4th heart sounds (presystolic gallop); pansystolic murmur. At sternal end of 2nd right intercostal space, heart sounds masked by continuous murmur, loudest in late systole and early diastole.

**Linear P.C.G.**

Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 22 APEX




PATIENT NO. 22 2nd. R.I.S.

## PLATE 26

### Patient No. 23 - female, age 15

Diagnosis
Acute rheumatic carditis

Auscultation
Soft 1st and 2nd heart sounds; pansystolic murmur. At aortic area, pansystolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.

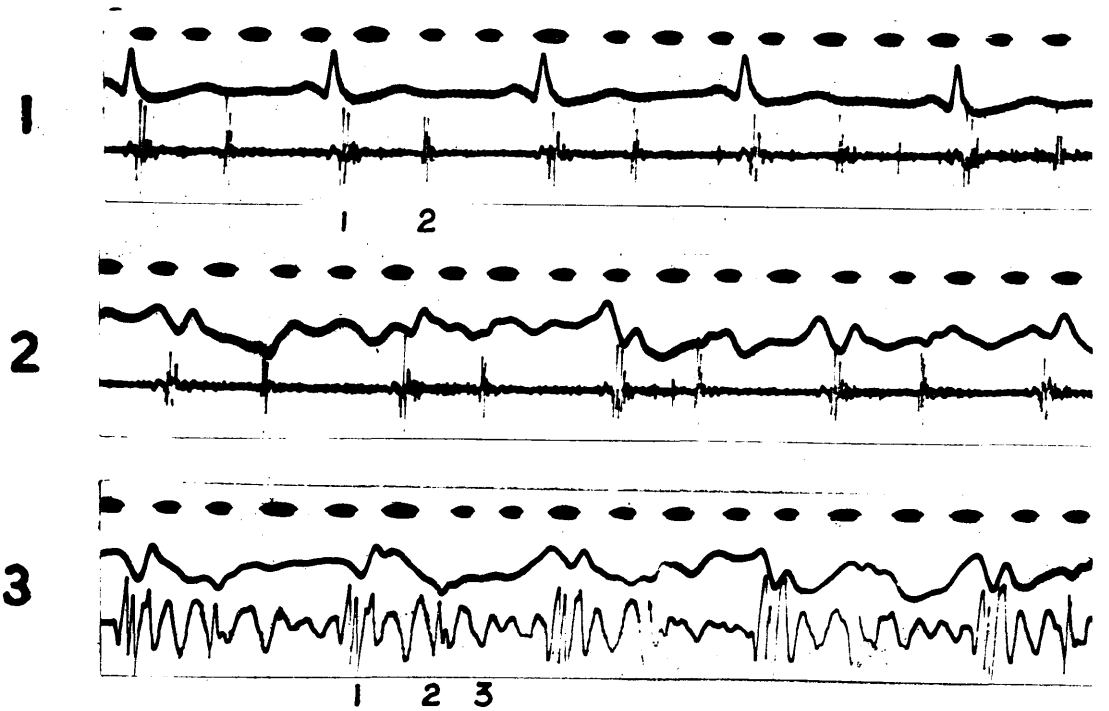
### Patient No. 24 - male, age 31

Diagnosis
Mitral stenosis
Auricular fibrillation

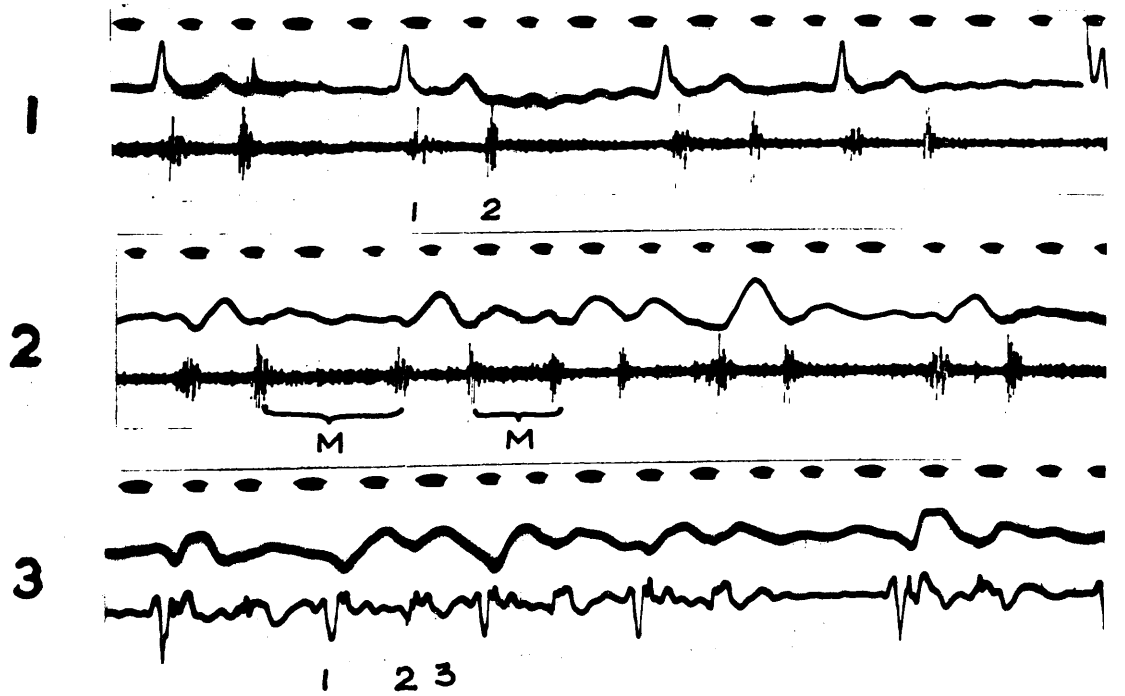
Auscultation
1st, 2nd and 3rd heart sounds (protodiastolic gallop); faint mid-diastolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; pandiastolic murmur, recorded in only two cardiac cycles.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.




PATIENT NO. 23 APEX




PATIENT NO. 24 APEX

## PLATE 27

### Patient No. 25 - male, age 58

Diagnosis
Anterior myocardial infarction

Auscultation
1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st, 2nd, and 4th heart sounds (presystolic gallop); no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 4th heart sounds.

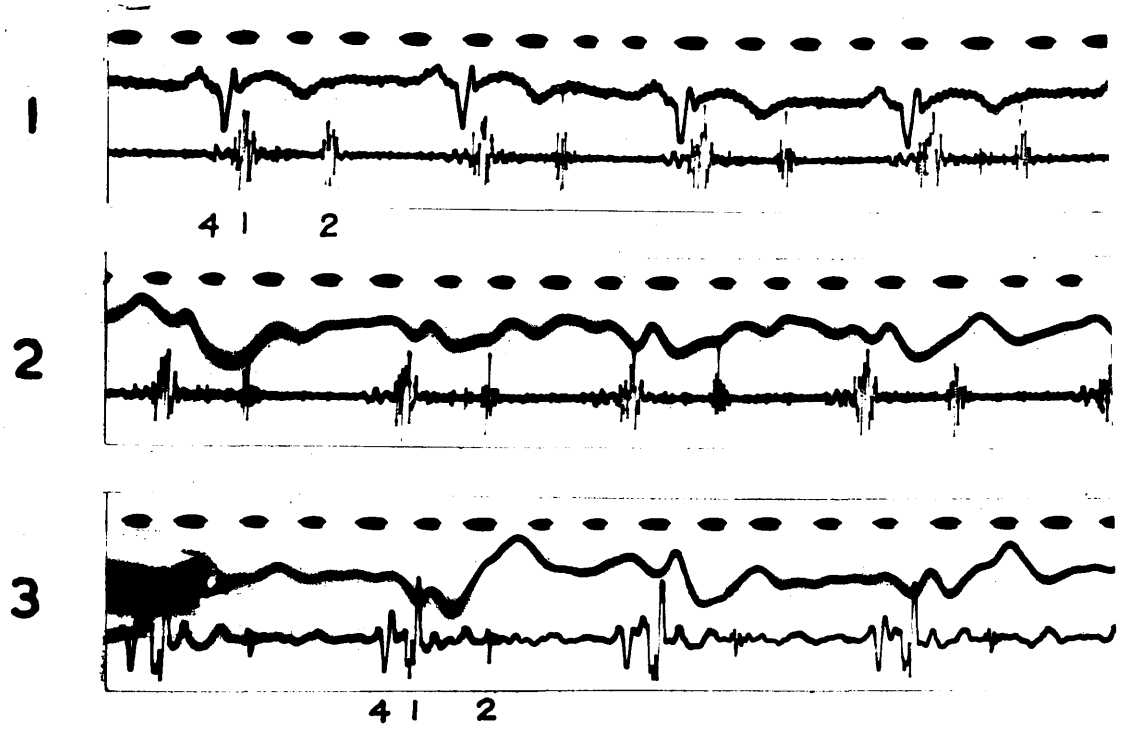
### Patient No. 26 - male, age 64

Diagnosis
Arterial hypertension Chronic nephritis
Left heart strain

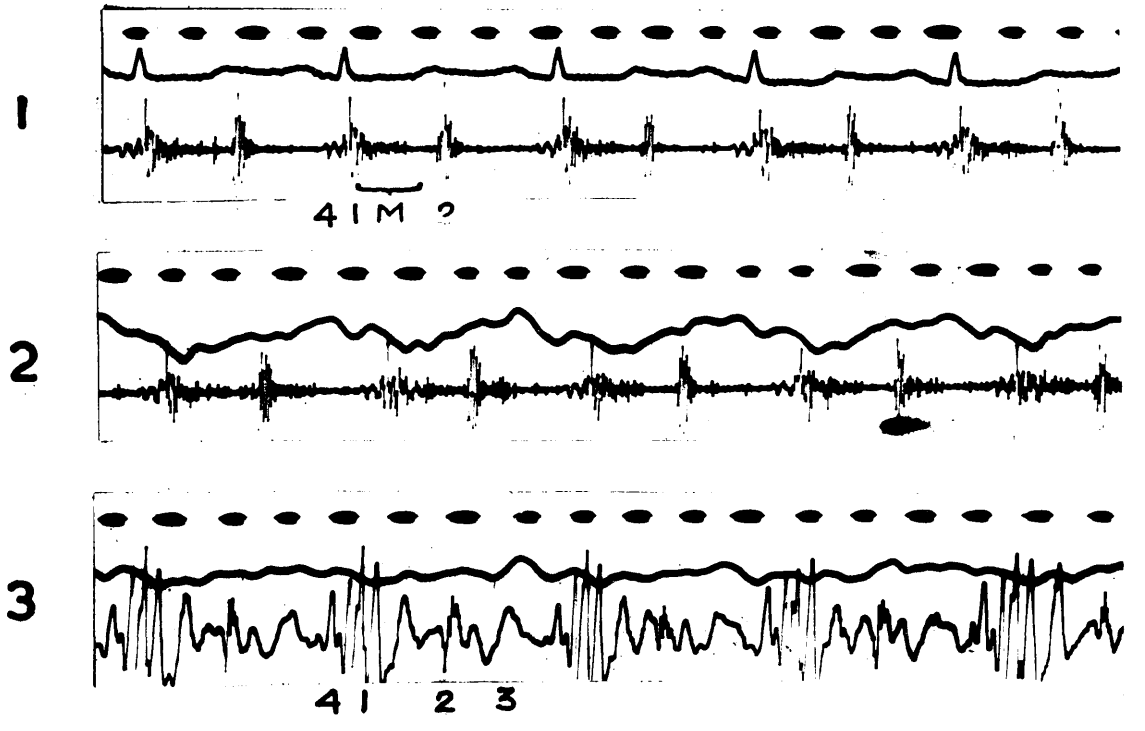
Auscultation
1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st, 2nd and 4th heart sounds (presystolic gallop); murmur in early and mid-systole.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd and 4th heart sounds.




PATIENT NO. 25 APEX




PATIENT NO. 26 APEX

## PLATE 28

### Patient No. 27 - female, age 27

Diagnosis
Mitral stenosis

Auscultation
1st and 2nd heart sounds; pansystolic and mid-diastolic murmurs.

Logarithmic P.C.G.
1st and 2nd heart sounds, with wide splitting of 2nd sound; pansystolic murmur.

Linear P.C.G.
Vibrations at the time of 1st heart sound and both components of split 2nd heart sound.

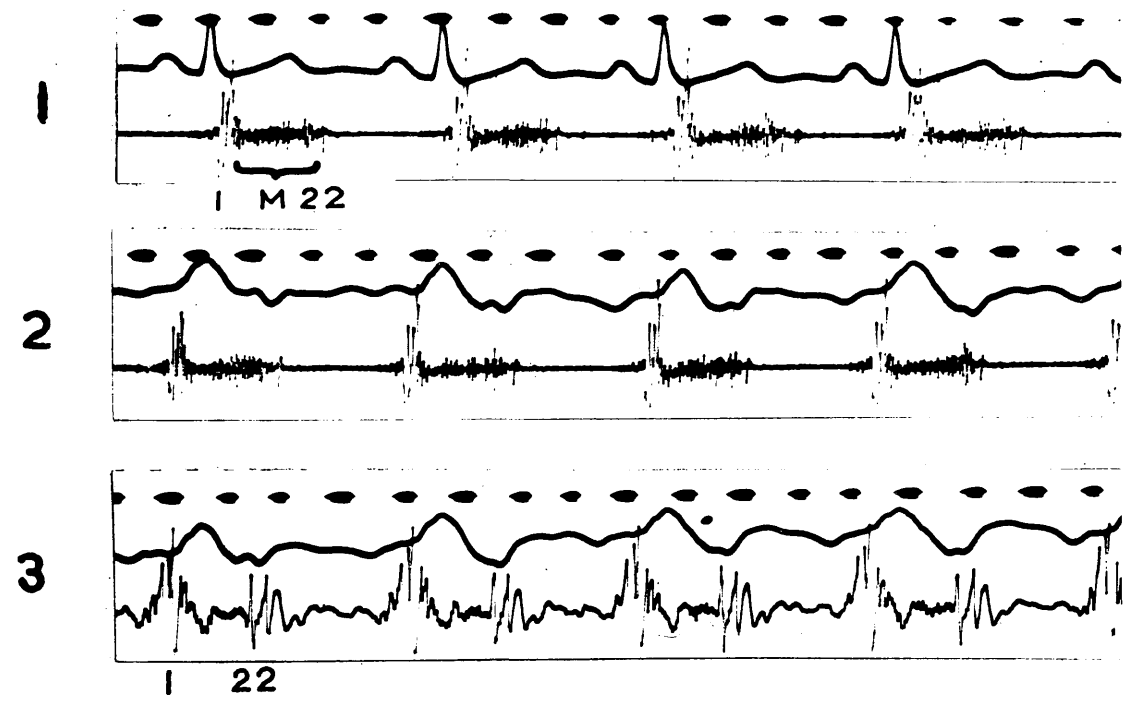
### Patient No. 28 - female, age 46

Diagnosis
Mitral stenosis
Auricular fibrillation

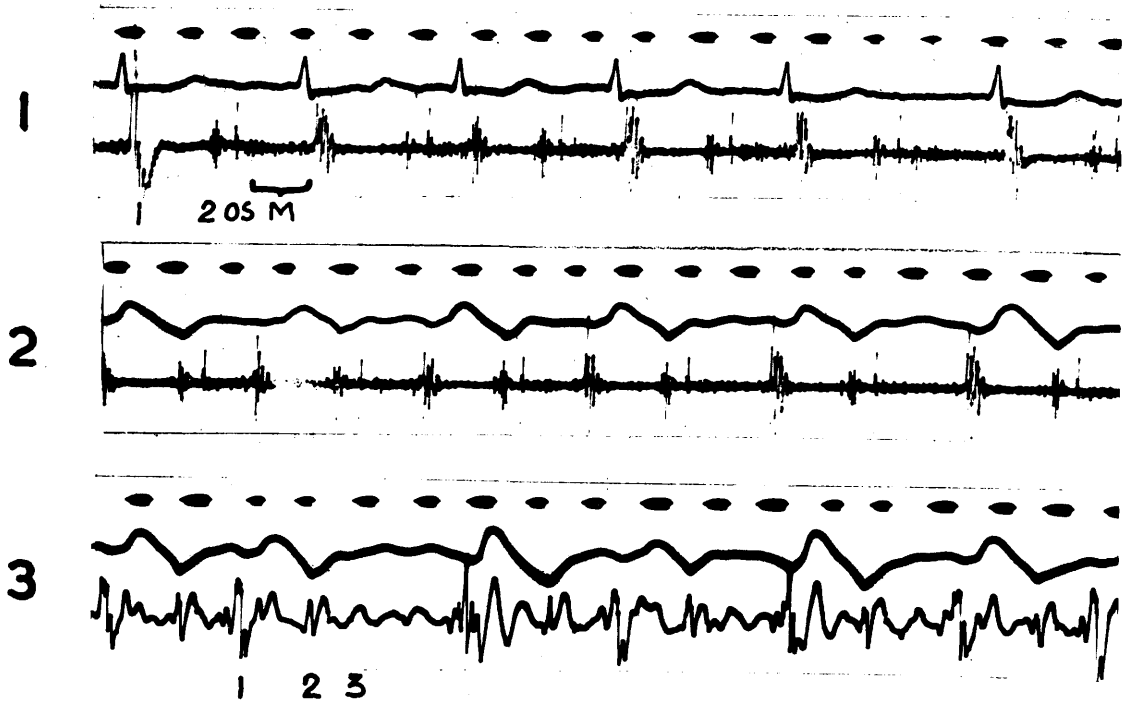
Auscultation
1st and 2nd heart sounds; pandiastolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds and mitral opening snap; mid- and late diastolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.




PATIENT NO. 27 APEX




PATIENT NO. 28 APEX

# PLATE 29

## Patient No. 29 - female, age 21

Diagnosis
Mitral stenosis
Acute rheumatic carditis

Auscultation
1st and 2nd heart sounds; early systolic and pan-diastolic murmurs.

Logarithmic P.C.G.
1st, 2nd, and 4th heart sounds (presystolic gallop); early systolic and late diastolic murmurs.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 4th heart sounds.

Note
In view of the tachycardia (105 beats per min.) the extra sound could be a summation sound, including an element of the 3rd heart sound.

## Patient No. 30 - female, age 39

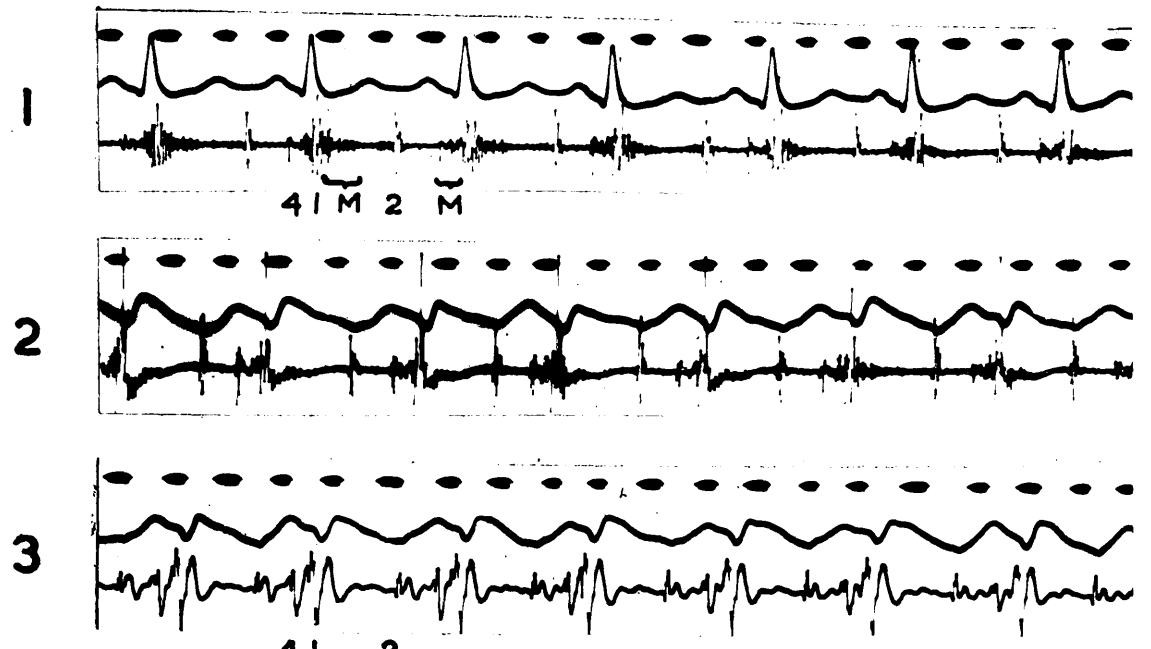
Diagnosis
Mitral stenosis (post-valvotomy)

Auscultation
1st and 2nd heart sounds; mid-diastolic and presystolic murmurs.

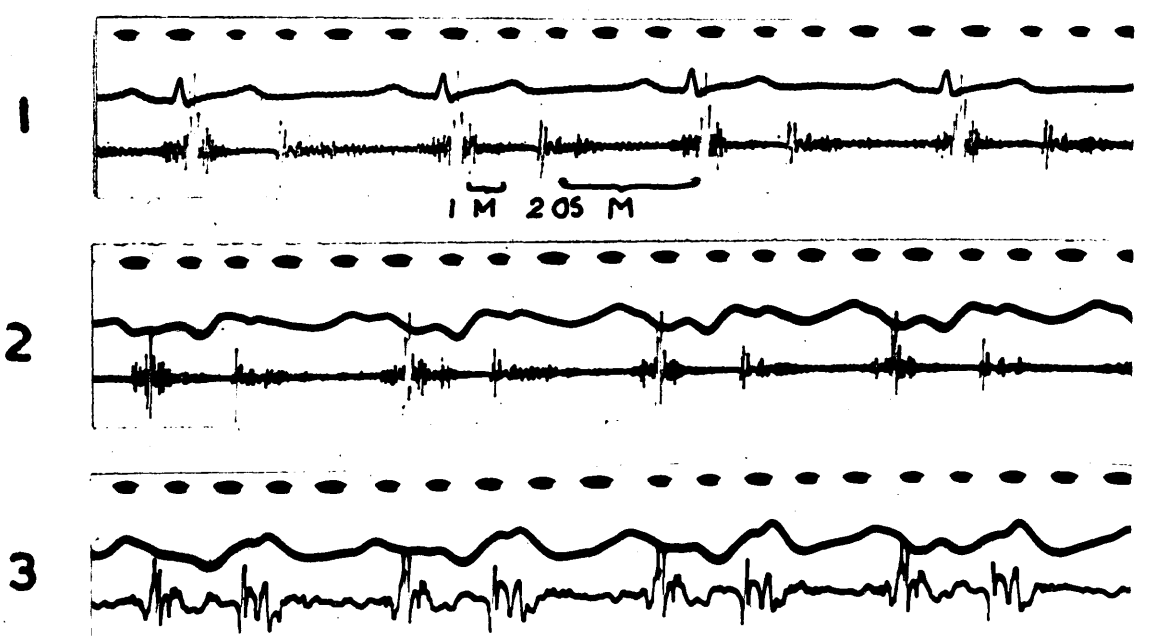
Logarithmic P.C.G.
1st and 2nd heart sounds and mitral opening snap; early systolic and pandiastolic murmurs, the latter with presystolic accentuation.

Linear P.C.G.
Vibrations at the time of 1st and 2nd heart sounds and mitral opening snap.

Note
After an apparently successful valvotomy the typical physical signs of mitral stenosis remain.




PATIENT NO.29 APEX




PATIENT NO.30 APEX

## PLATE 30

### Patient No. 31 - female, age 49

Diagnosis

Mitral stenosis and incompetence
Aortic stenosis and incompetence

Auscultation

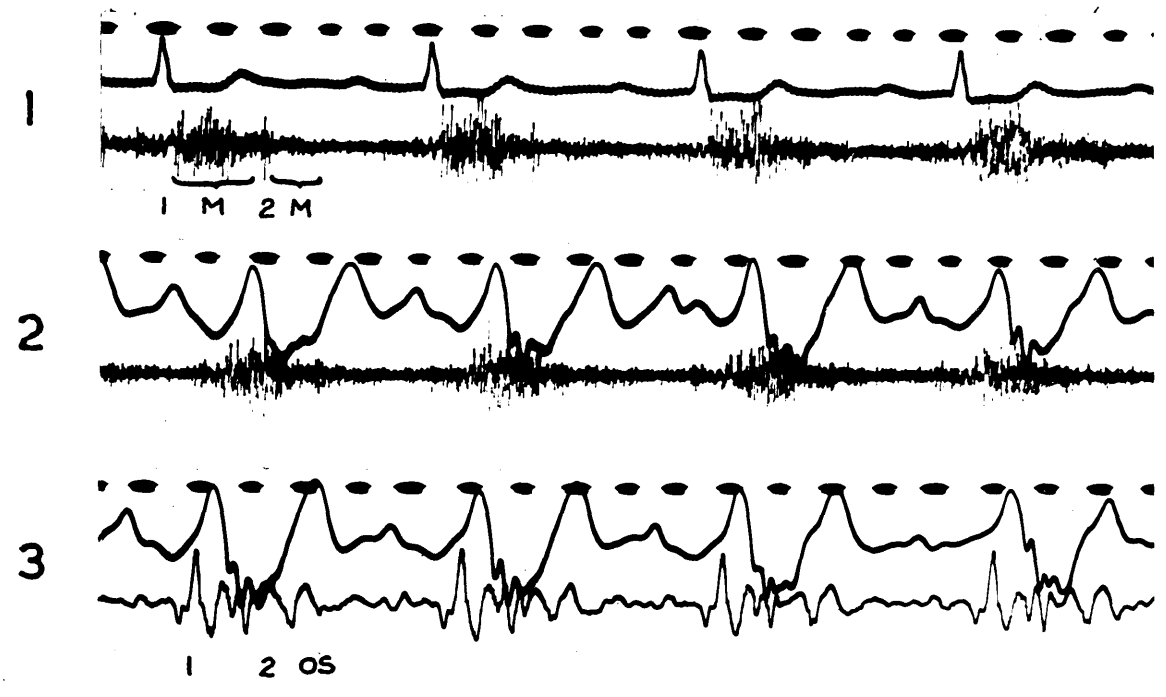
1st and 2nd heart sounds; continuous murmur. The same at aortic area (2nd right costal cartilage and left sternal border).

Logarithmic P.C.G.

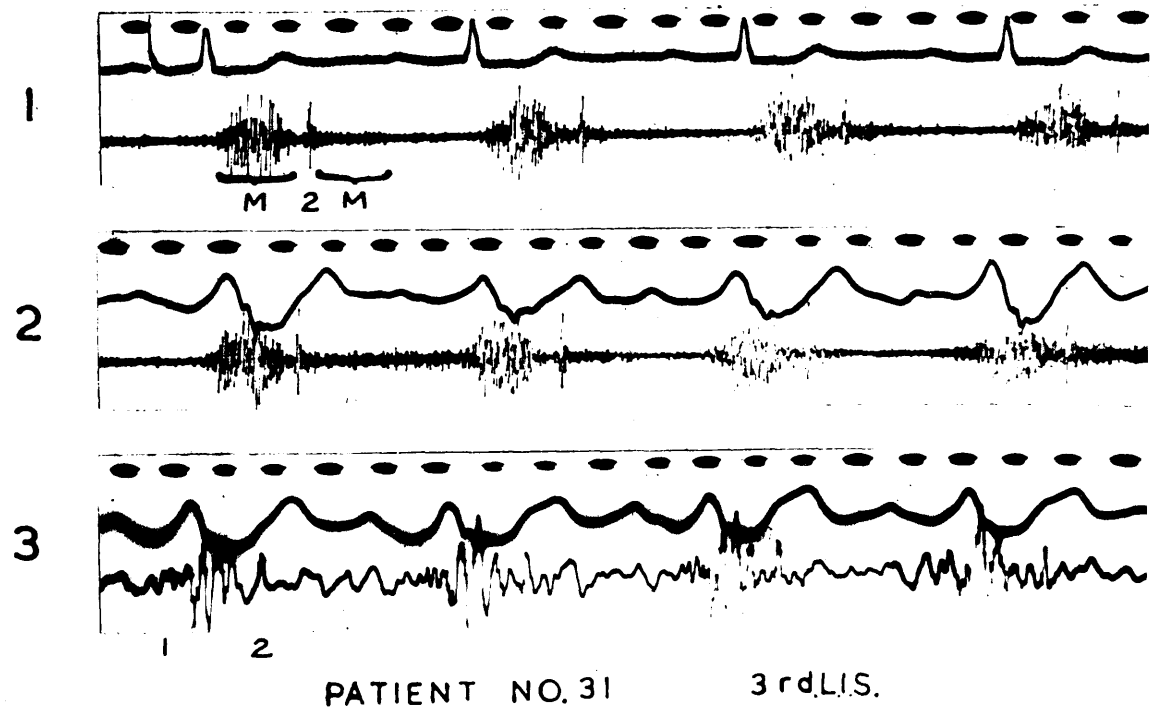
Low-amplitude 1st and louder 2nd heart sounds; pansystolic and early diastolic murmurs. At sternal end of 3rd left intercostal space, no obvious 1st heart sound; pansystolic murmur ('diamond-shaped', with maximum intensity in mid-systole) and early diastolic murmur.

Linear P.C.G.

Vibrations at the time of 1st and 2nd heart sounds and mitral opening snap. At sternal end of 3rd left intercostal space, vibrations at the time of 1st and 2nd heart sounds.




PATIENT NO. 31 APEX




PATIENT NO. 31 3rd L.I.S.

## PLATE 31

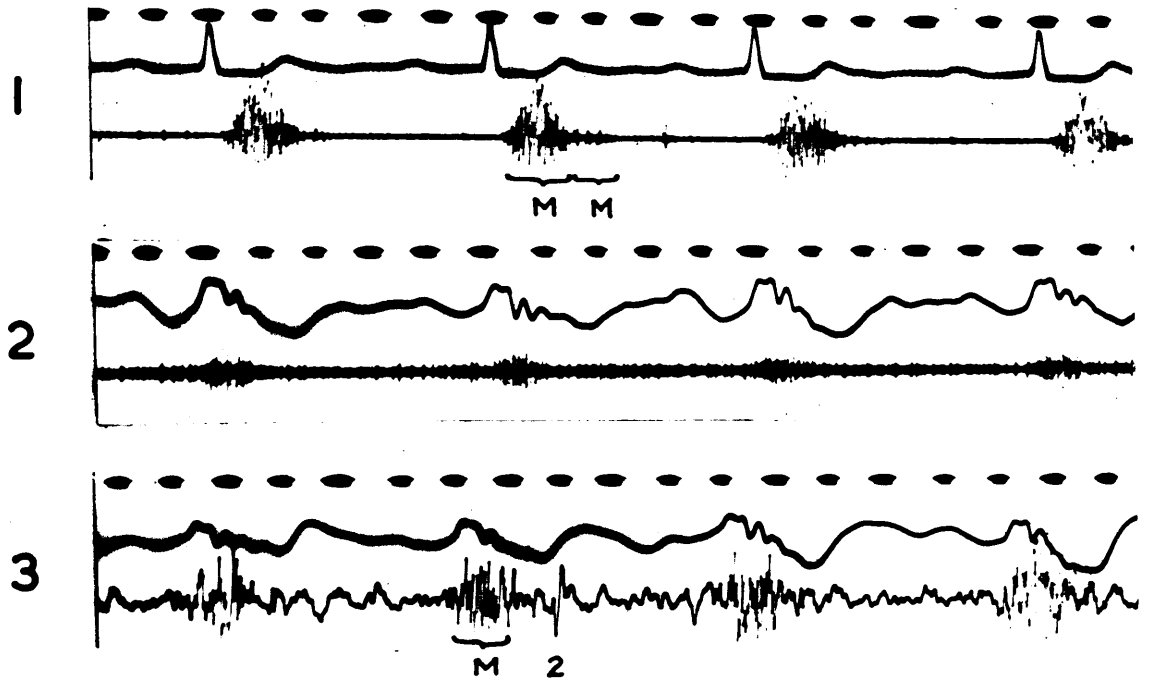
### Patient No. 31 - continued

Logarithmic P.C.G.

At 2nd right costal cartilage, no obvious heart sounds; 'diamond-shaped' pansystolic murmur and early diastolic murmur.

Linear P.C.G.

At 2nd right costal cartilage, vibrations at the time of 2nd heart sound and indication of systolic murmur.




PATIENT NO. 31 2nd R.C.C.

## PLATE 32

### Patient No. 32 - female, age 25

Diagnosis

Mitral stenosis Bronchiectasis

Auscultation

1st and 2nd heart sounds; pansystolic and early and late diastolic murmurs.

Logarithmic P.C.G.

1st and 2nd heart sounds and mitral opening snap; pansystolic and early and late diastolic murmurs.

Linear P.C.G.

Vibrations at the time of 1st and 2nd heart sounds and mitral opening snap.

### Patient No. 33 - female, age 44

Diagnosis

Arterial hypertension Chronic nephritis
Left heart strain Iron-deficiency anaemia

Auscultation

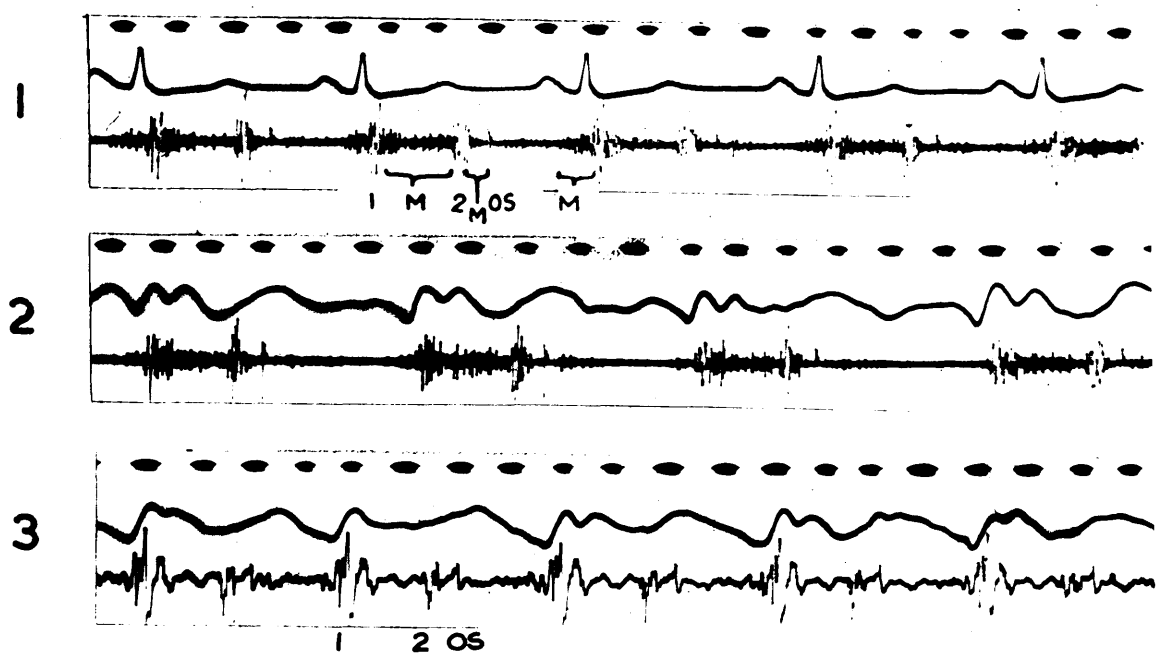
1st and 2nd heart sounds; early and mid-systolic murmur.

Logarithmic P.C.G.

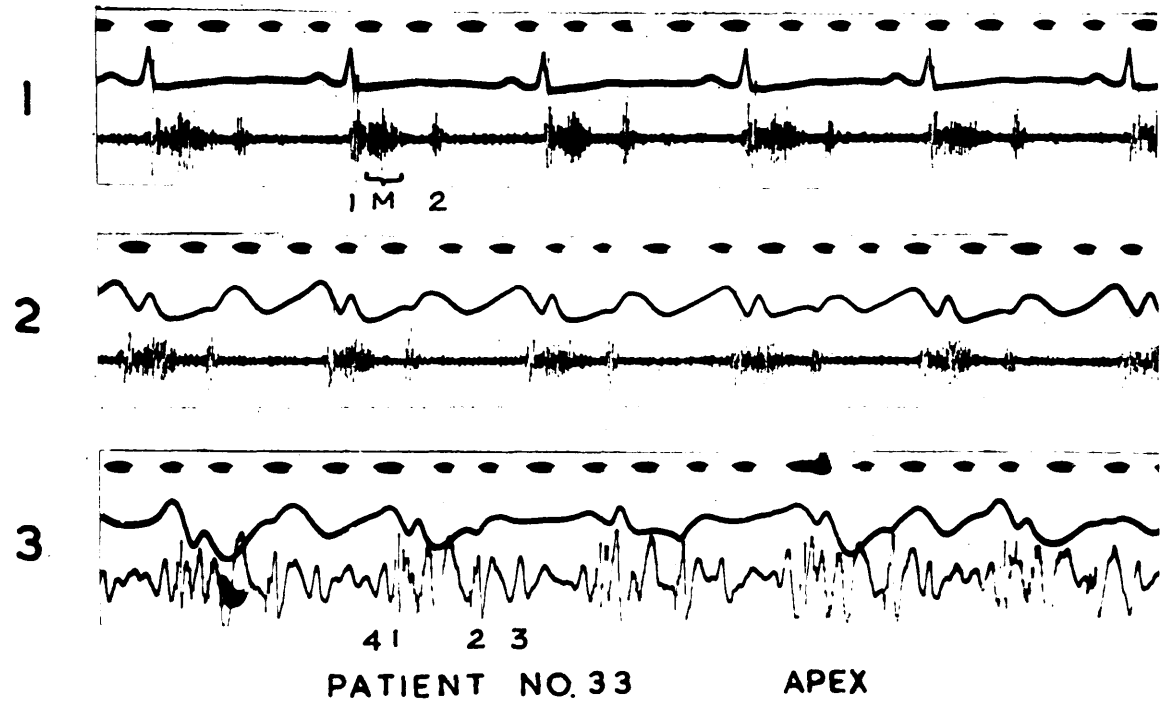
1st and 2nd heart sounds; early systolic murmur.

Linear P.C.G.

Vibrations at the time of 1st, 2nd, 3rd and 4th heart sounds.




PATIENT NO. 32 APEX




PATIENT NO. 33 APEX

## PLATE 33

### Patient No. 34 - female, age 14

Diagnosis
Mitral stenosis
Myocardial disease

Auscultation
1st and 2nd heart sounds and mitral opening snap; pansystolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds and mitral opening snap; early and mid-systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds and mitral opening snap.

Note
The predominant systolic murmur suggests a considerable degree of mitral incompetence.

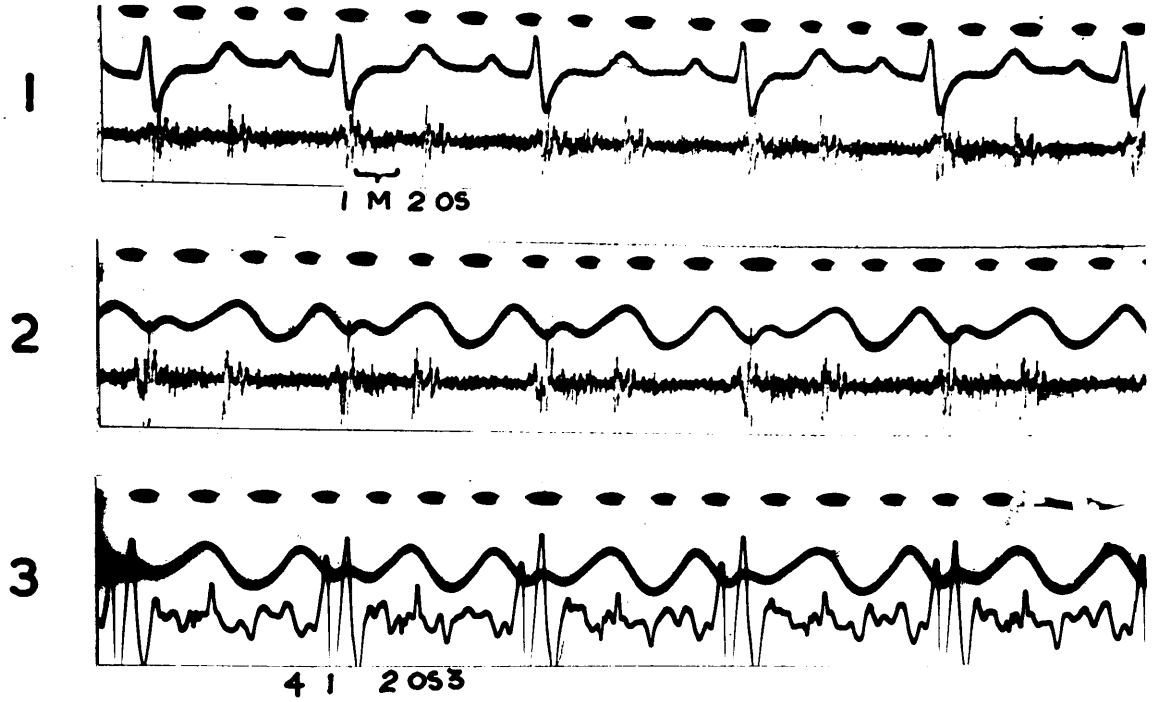
### Patient No. 35 - female, age 33

Diagnosis
Mitral stenosis (post-valvotomy)

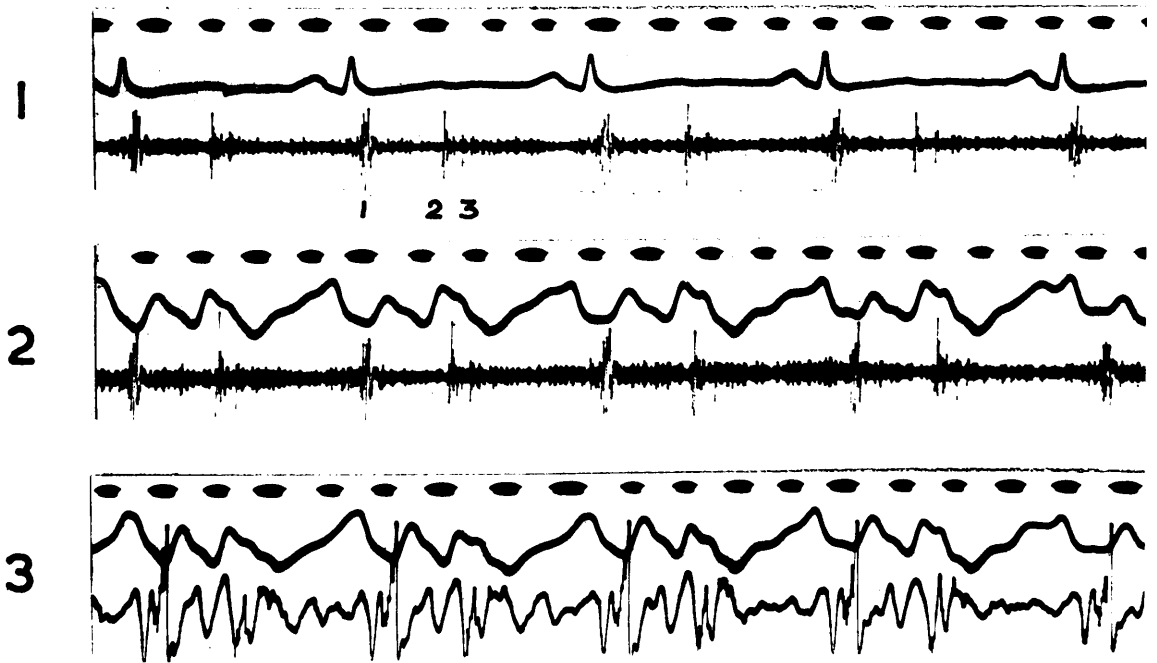
Auscultation
1st and 2nd heart sounds; mid-diastolic murmur.

Logarithmic P.C.G.
1st, 2nd and 3rd heart sounds (protodiastolic gallop); no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 34 APEX




PATIENT NO. 35 APEX

## PLATE 34

### Patient No. 36 - female, age 41

Diagnosis
Mitral stenosis (post-valvotomy)
Auricular fibrillation

Auscultation
1st and 2nd heart sounds; pansystolic and mid-diastolic murmurs.

Logarithmic P.C.G.
1st and 2nd heart sounds and mitral opening snap; early and mid-systolic and pandiastolic murmurs.

Linear P.C.G.
Vibrations at the time of 1st and 2nd heart sounds and mitral opening snap.

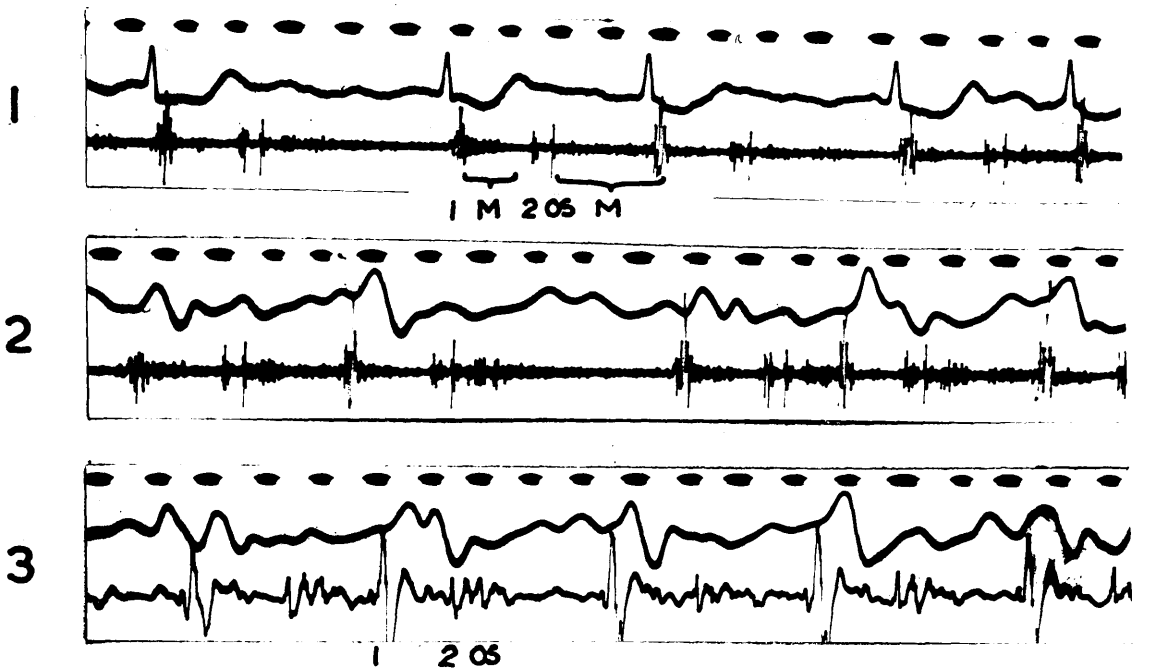
### Patient No. 37 - female, age 52

Diagnosis
Mitral stenosis
Aortic incompetence

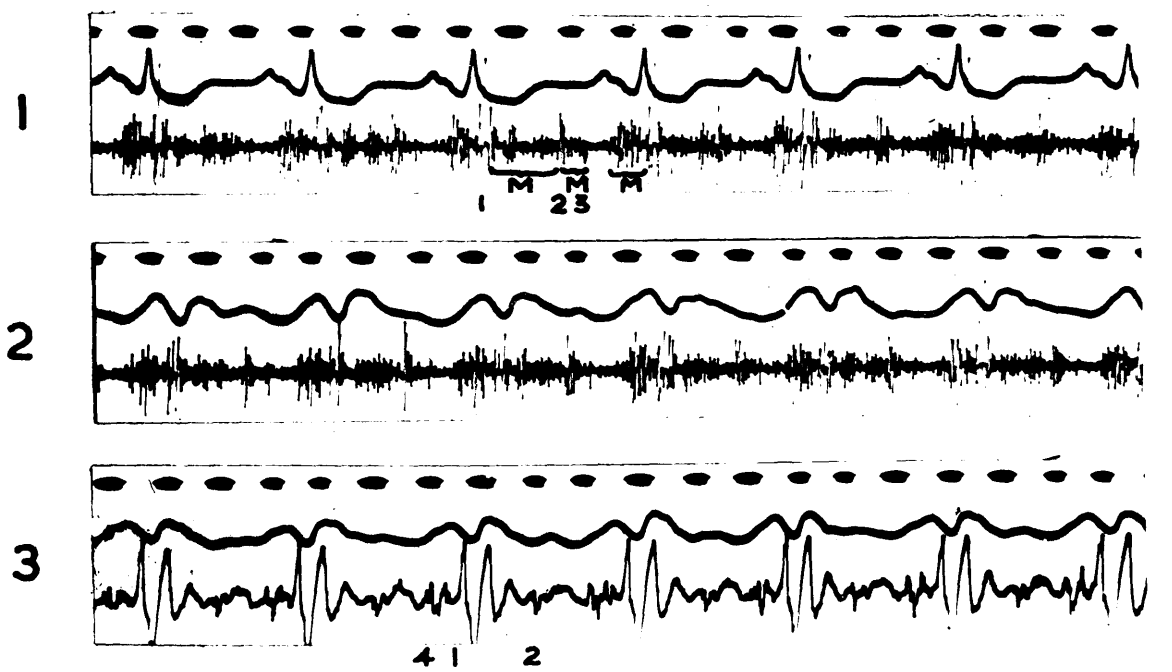
Auscultation
1st and 2nd heart sounds; pansystolic and mid- and late diastolic murmurs.

Logarithmic P.C.G.
1st, 2nd, and 3rd heart sounds (protodiastolic gallop); pansystolic and early and late diastolic murmurs.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 4th heart sounds.




PATIENT NO. 36 APEX




PATIENT NO. 37 APEX

## PLATE 35

### Patient No. 38 - female, age 35

Diagnosis

Mitral stenosis and incompetence
Aortic incompetence

Auscultation

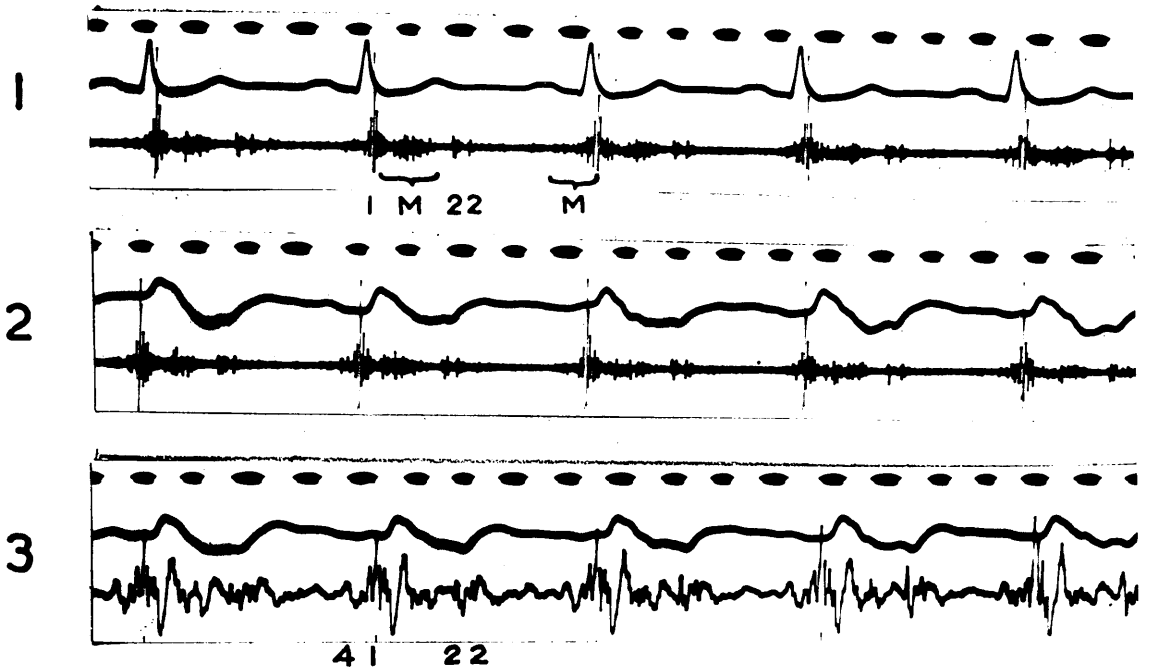
1st and 2nd heart sounds; pansystolic and mid- and late diastolic murmurs. Aortic pansystolic and early diastolic murmurs, the latter loudest at sternal end of 3rd left intercostal space.

Logarithmic P.C.G.

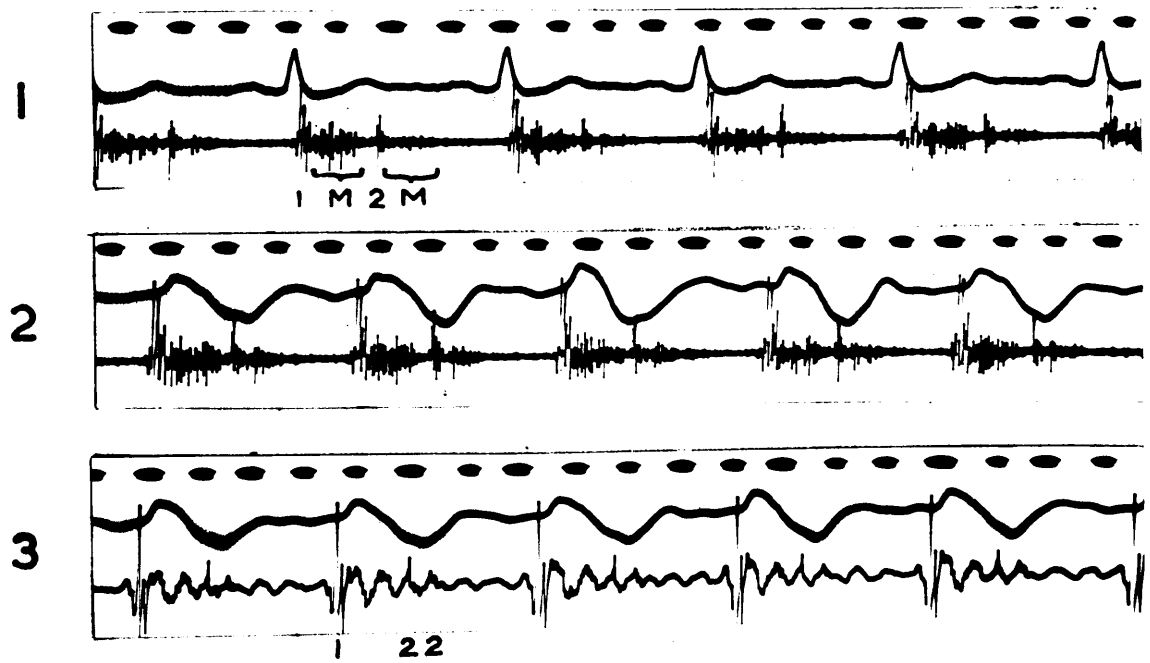
1st and 2nd heart sounds with wide splitting of 2nd sound; early and mid-systolic and late diastolic murmurs. At sternal end of 3rd left intercostal space, 1st and 2nd heart sounds; early and mid-systolic and early diastolic murmurs.

Linear P.C.G.

Vibrations at the time of 1st, both components of 2nd, and 4th heart sounds. At sternal end of 3rd left intercostal space, vibrations at the time of 1st heart sound and both components of 2nd sound.




PATIENT NO. 38 APEX




PATIENT NO. 38 3rd L.I.S.

## PLATE 36

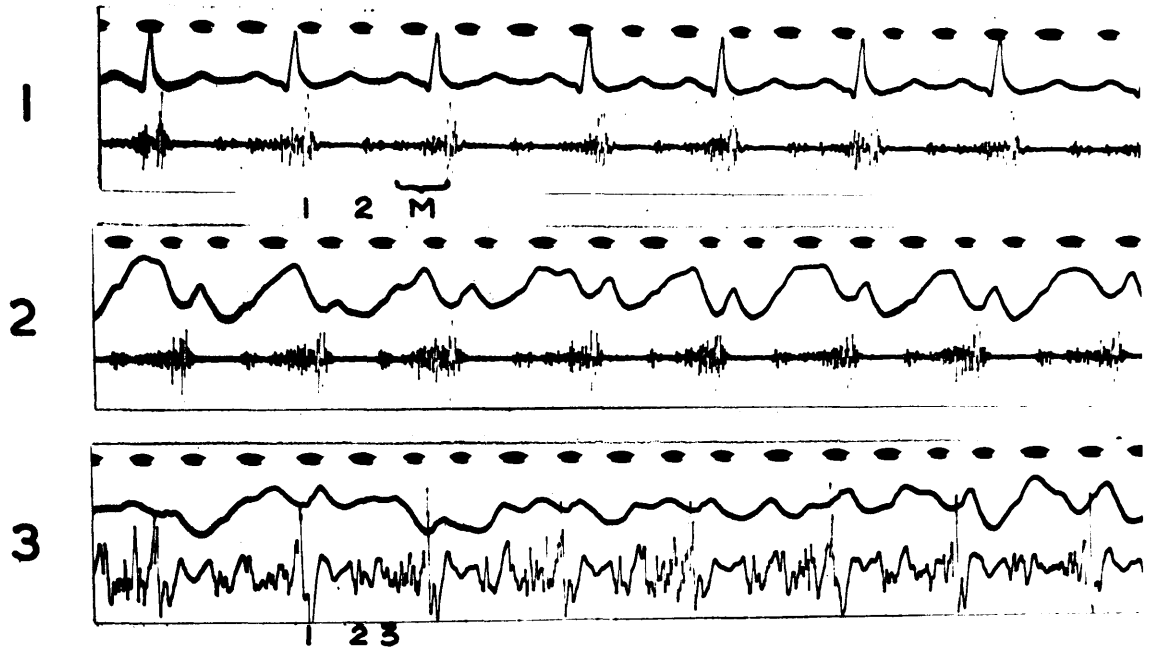
### Patient No. 39 - male, age 30

Diagnosis
Mitral stenosis

Auscultation
1st and 2nd heart sounds; mid- and late diastolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; mid- and late diastolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd and 3rd heart sounds.




PATIENT NO. 39 APEX

## PLATE 37

### Patient No. 40 - male, age 16

Diagnosis

Mitral stenosis
Aortic incompetence

Auscultation

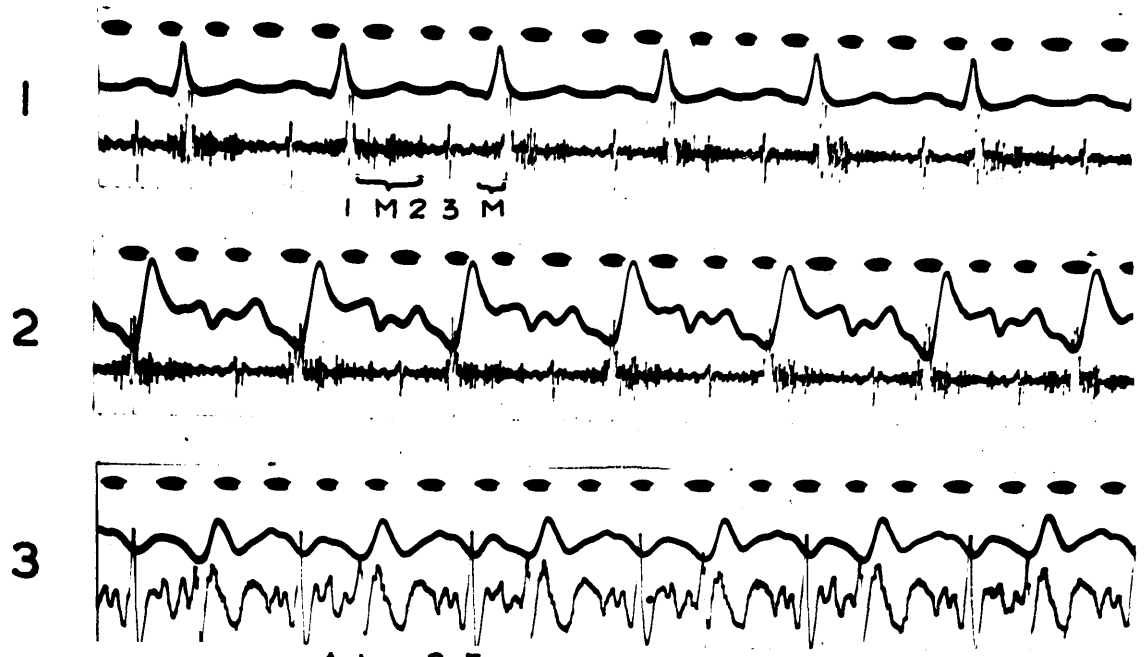
1st and 2nd heart sounds; pansystolic murmur. At 2nd right costal cartilage, 1st and 2nd heart sounds; continuous murmur.

Logarithmic P.C.G.

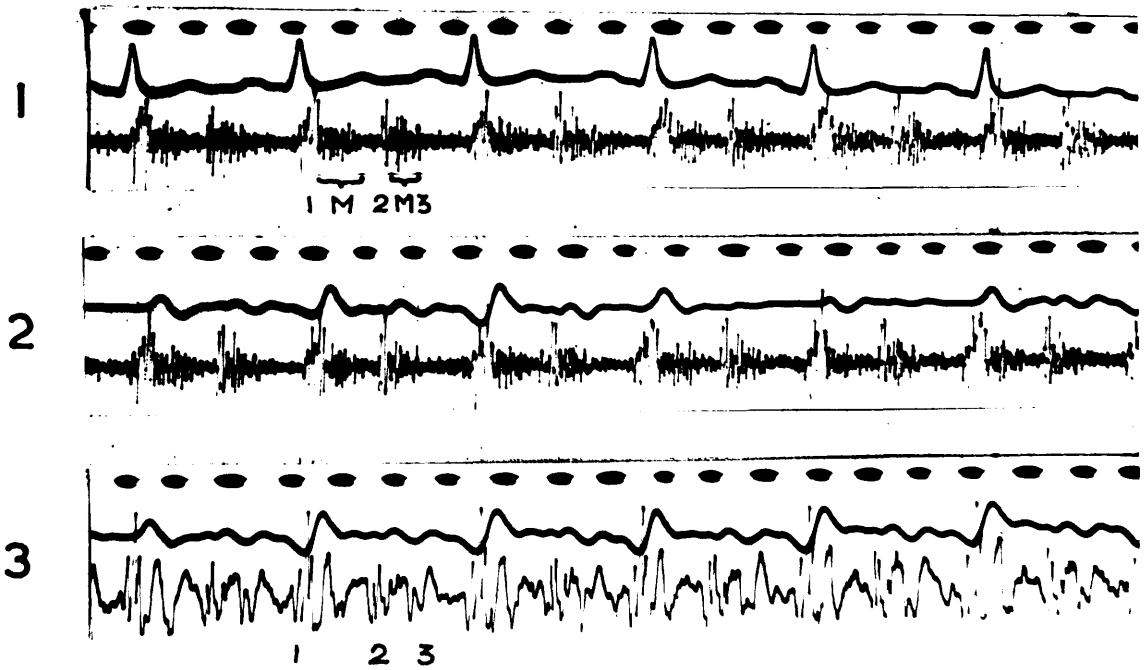
1st, 2nd, and 3rd heart sounds; pansystolic and late diastolic murmurs. At 2nd right costal cartilage, 1st, 2nd, and 3rd heart sounds; early and mid-systolic and early diastolic murmurs.

Linear P.C.G.

Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds. At 2nd right costal cartilage, vibrations at the time of 1st, 2nd, and 3rd heart sounds.




PATIENT NO. 40 APEX




PATIENT NO. 40 2nd.R.C.C.

## PLATE 38

### Patient No. 41 - male, age 66

Diagnosis

Aortic stenosis — Emphysema
Congestive cardiac failure

Auscultation

Very faint 1st and 2nd heart sounds; no murmur.

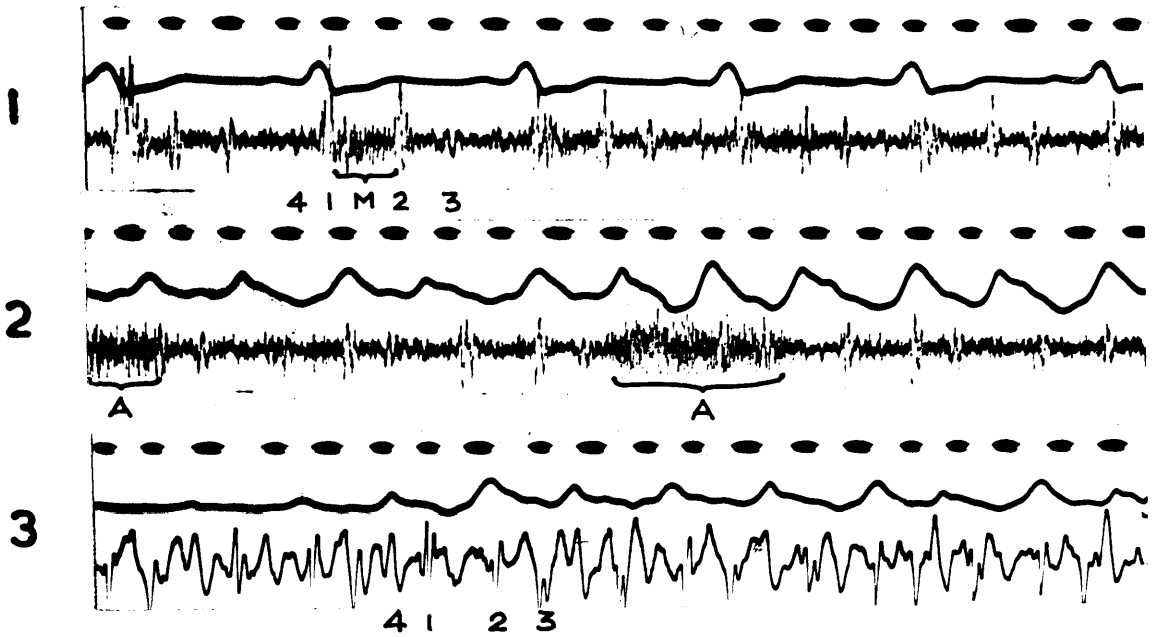
Logarithmic P.C.G.

1st, 2nd, 3rd, and 4th heart sounds (quadruple rhythm); pansystolic murmur.

Breath sound artefacts marked 'A' on record 2.

Linear P.C.G.

Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 41 APEX

## PLATE 39

### Patient No. 42 - male, age 15

Diagnosis

Fallot's tetralogy

Auscultation

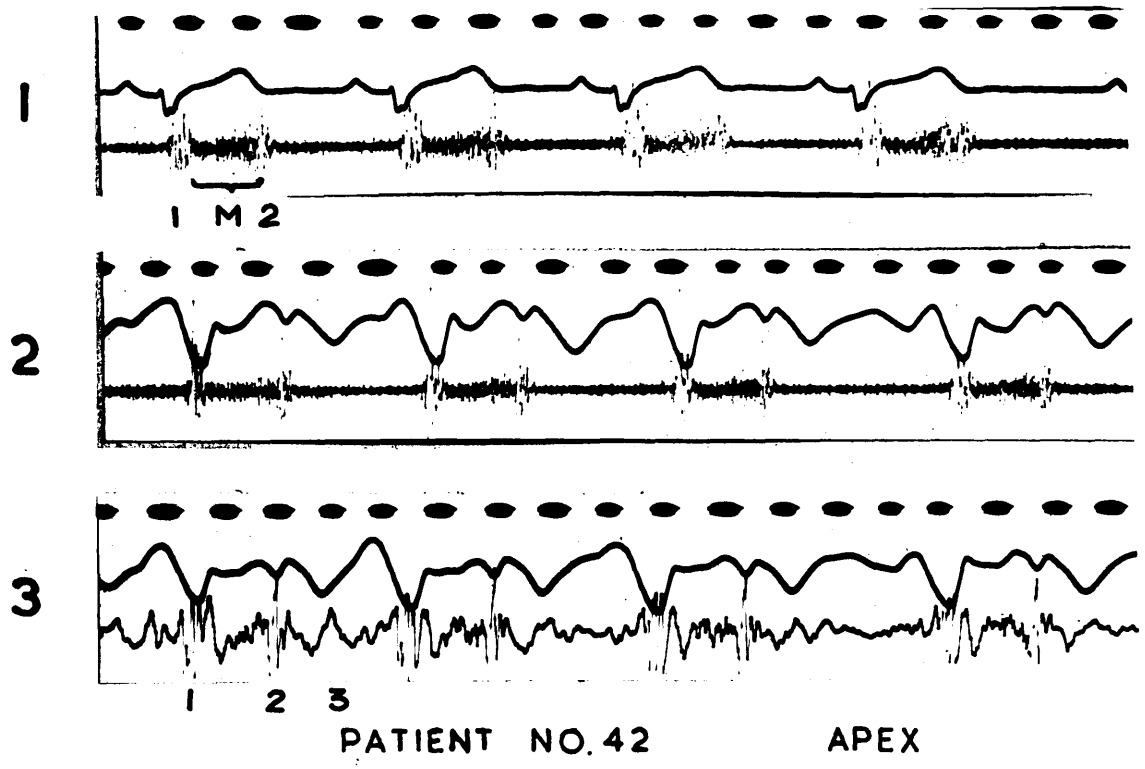
1st and 2nd heart sounds; pansystolic murmur. Murmur loudest at lower right sternal border.

Logarithmic P.C.G.

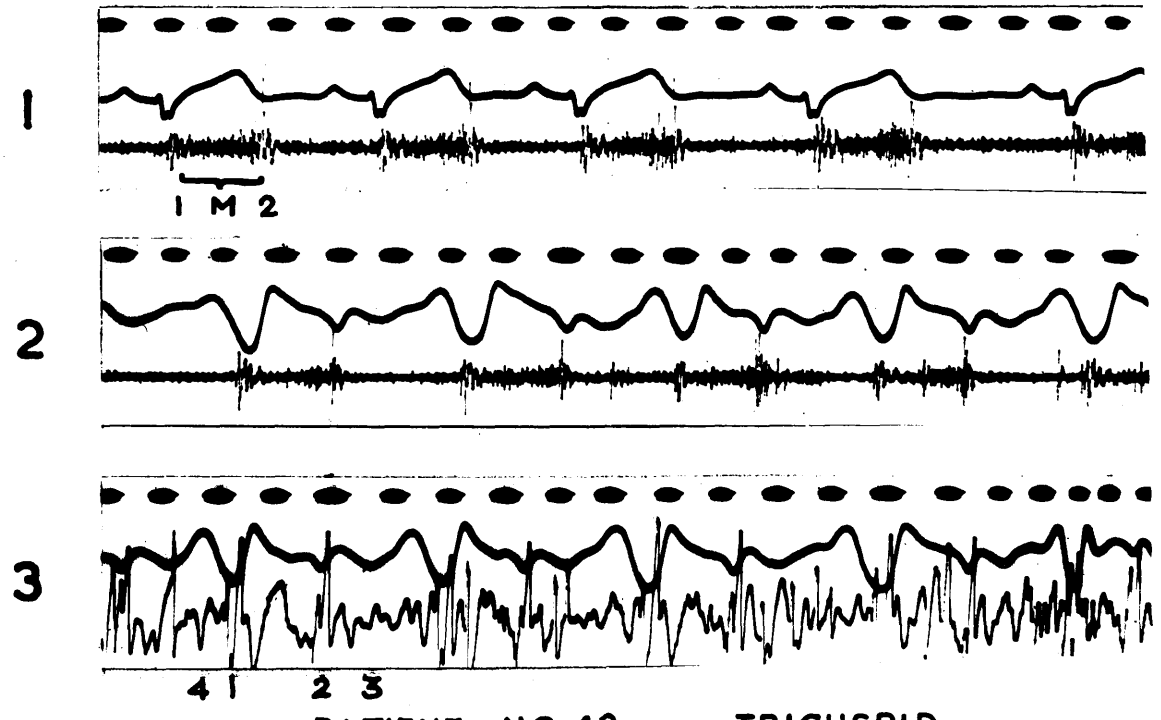
1st and 2nd heart sounds; pansystolic murmur. At tricuspid area, 1st and 2nd heart sounds, pansystolic murmur.

Linear P.C.G.

Vibrations at the time of 1st, 2nd, and 3rd heart sounds. At tricuspid area, vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 42 APEX




PATIENT NO. 42 TRICUSPID

## PLATE 40

### Patient No. 43 - male, age 24

Diagnosis
Mitral stenosis

Auscultation
1st and 2nd heart sounds; pansystolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; mid- and late diastolic murmur.

Linear P.C.G.
Vibrations at the time of 1st and 2nd heart sounds.

Note
The higher-frequency systolic murmur, which was heard, is not recorded: the lower-frequency diastolic murmur, which was not heard, is recorded.

### Patient No. 44 - female, age 28

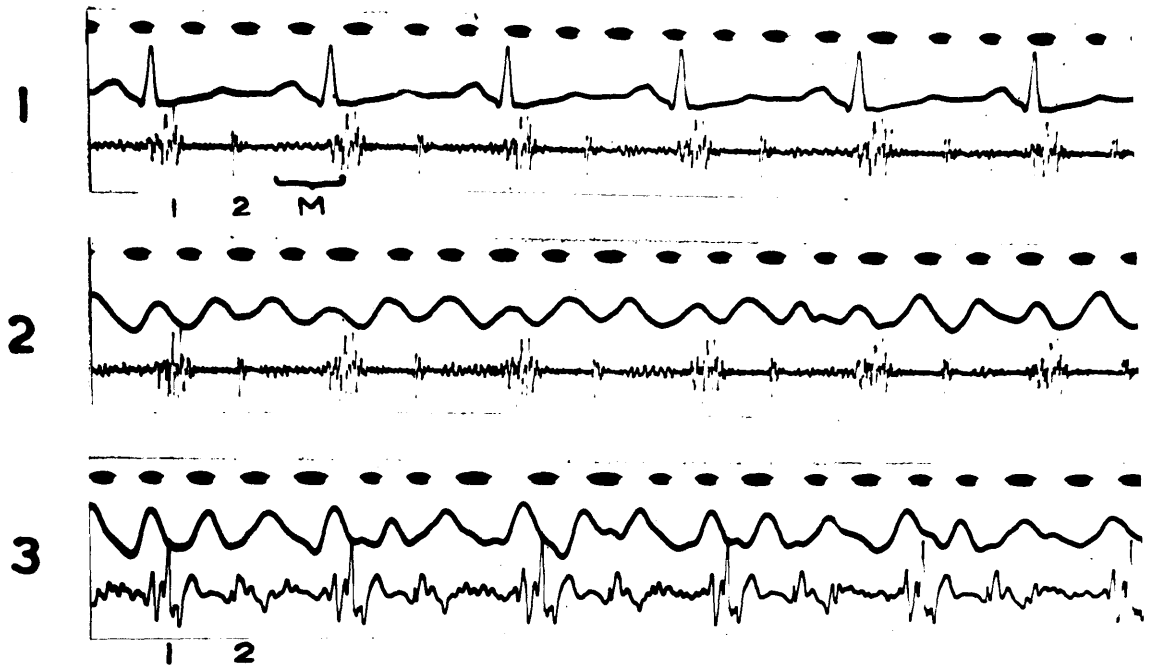
Diagnosis
Mitral stenosis

Auscultation
Very loud 1st and normal 2nd heart sounds; mid-diastolic murmur.

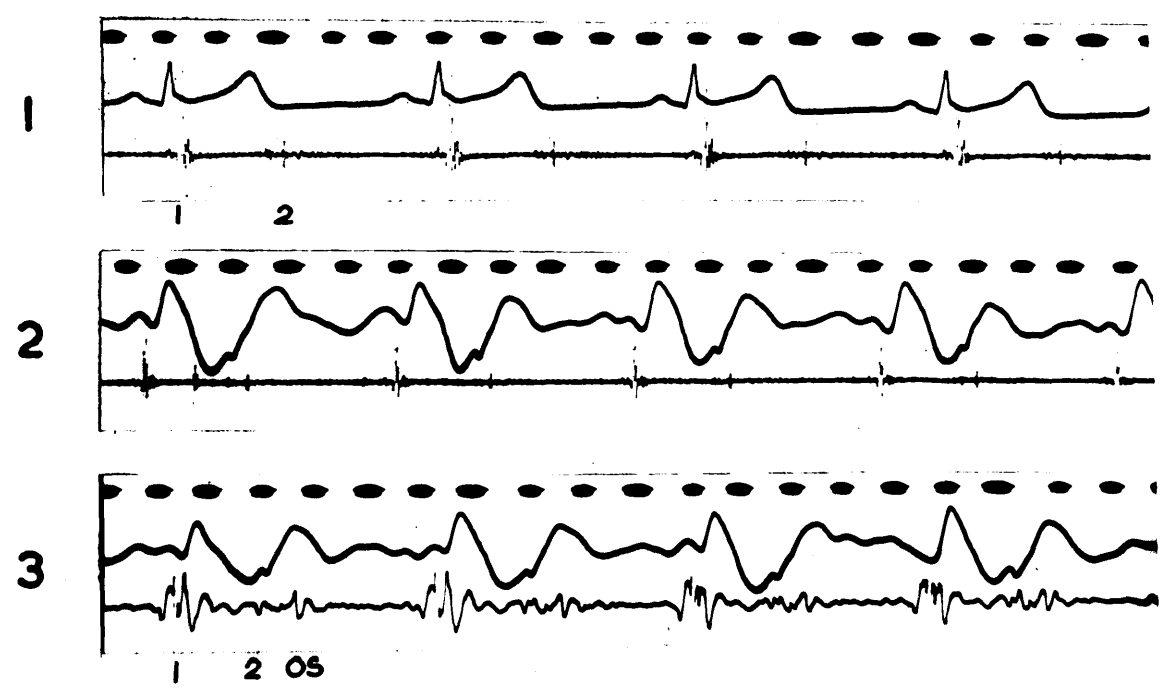
Logarithmic P.C.G.
1st and 2nd heart sounds; no murmur.

Linear P.C.G.
Vibrations at the time of 1st and 2nd heart sounds and mitral opening snap.

Note
At the reduced gain required to record the whole of the 1st sound deflections, the audible murmur is not recorded.




PATIENT NO.43 APEX




PATIENT NO.44 APEX

## PLATE 41

### Patient No. 45 - female, age 7

Diagnosis

Patent ductus arteriosus

Auscultation

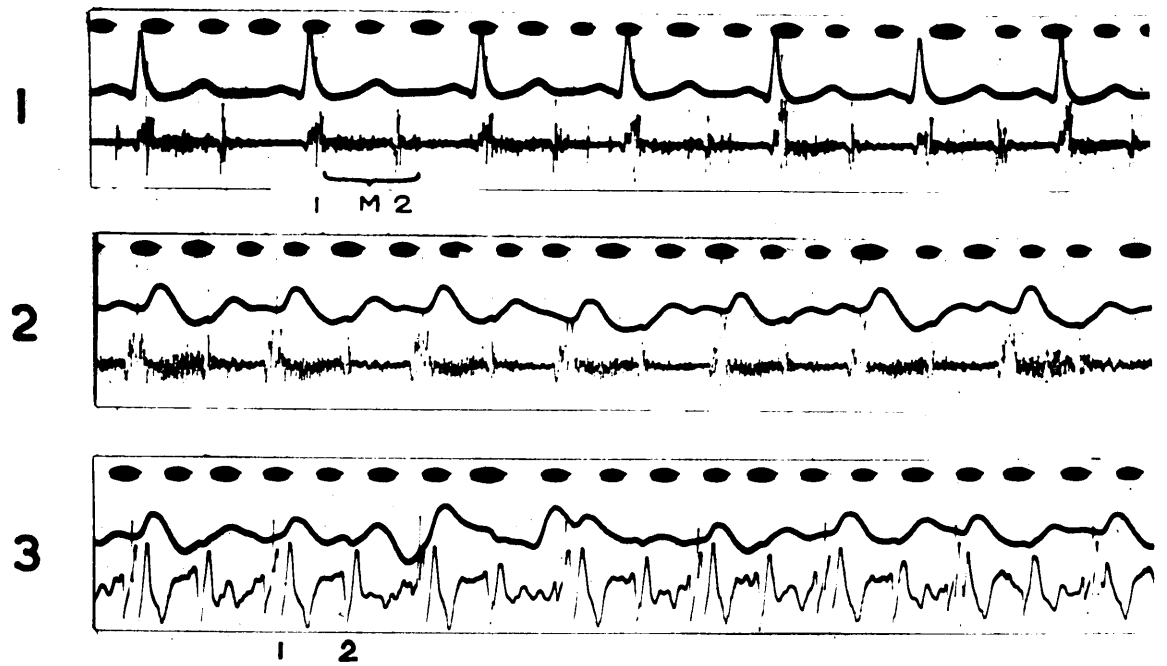
1st and 2nd heart sounds; pansystolic murmur. At pulmonary area, heart sounds masked by continuous murmur.

Logarithmic P.C.G.

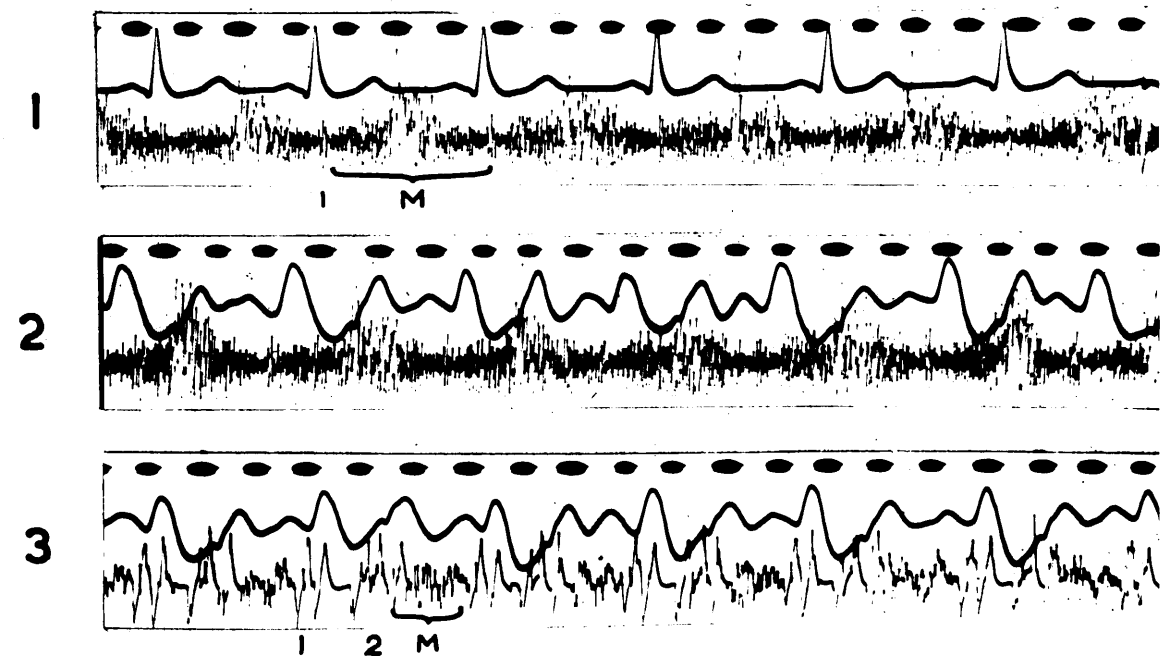
1st and 2nd heart sounds; pansystolic and early diastolic murmur. At pulmonary area, 1st heart sound; continuous murmur, loudest at the time of 2nd heart sound.

Linear P.C.G.

Vibrations at the time of 1st and 2nd heart sounds. At pulmonary area, vibrations at the time of 1st and 2nd heart sounds; indication of diastolic murmur.




PATIENT NO.45 APEX




PATIENT NO.45 PULMONARY

## PLATE 42

### Patient No. 46 - female, age 21

Diagnosis
Mitral stenosis

Auscultation
1st and 2nd heart sounds; pansystolic and mid-diastolic murmurs.

Logarithmic P.C.G.
1st and 2nd heart sounds; early and mid-systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st and 2nd heart sounds.

Note
The predominance of the systolic murmur suggests a predominance of incompetence rather than stenosis of the mitral valve.

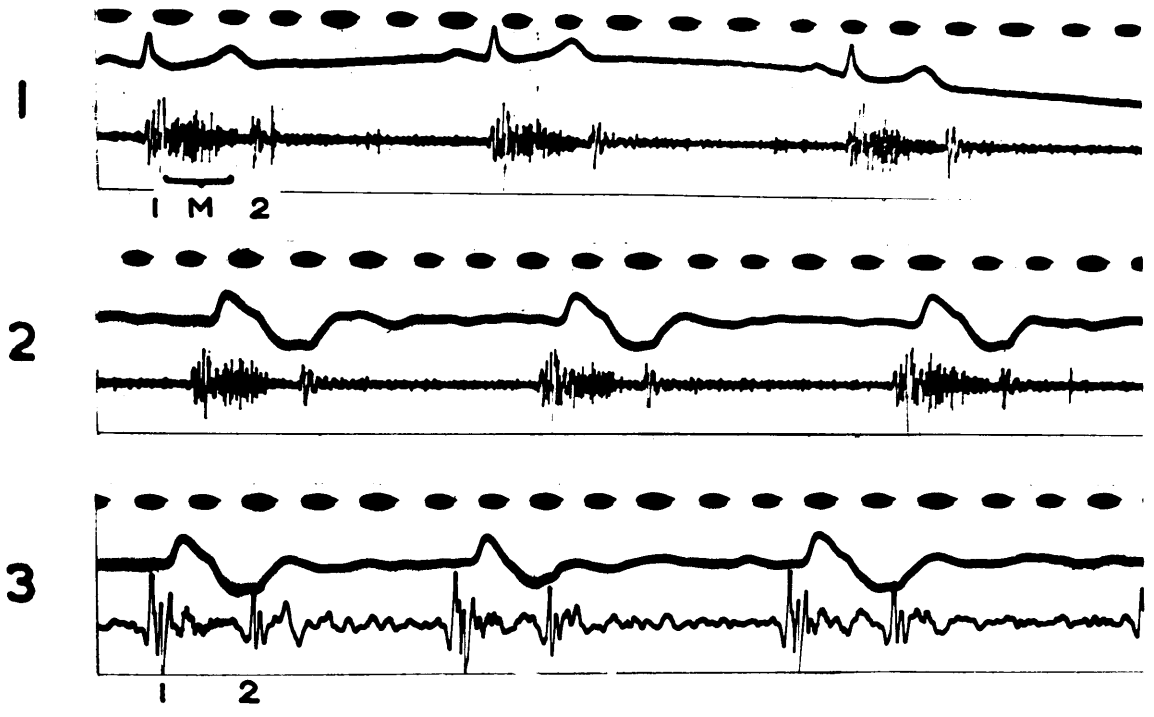
### Patient No. 47 - female, age 51

Diagnosis
Arterial hypertension
Cardiac failure

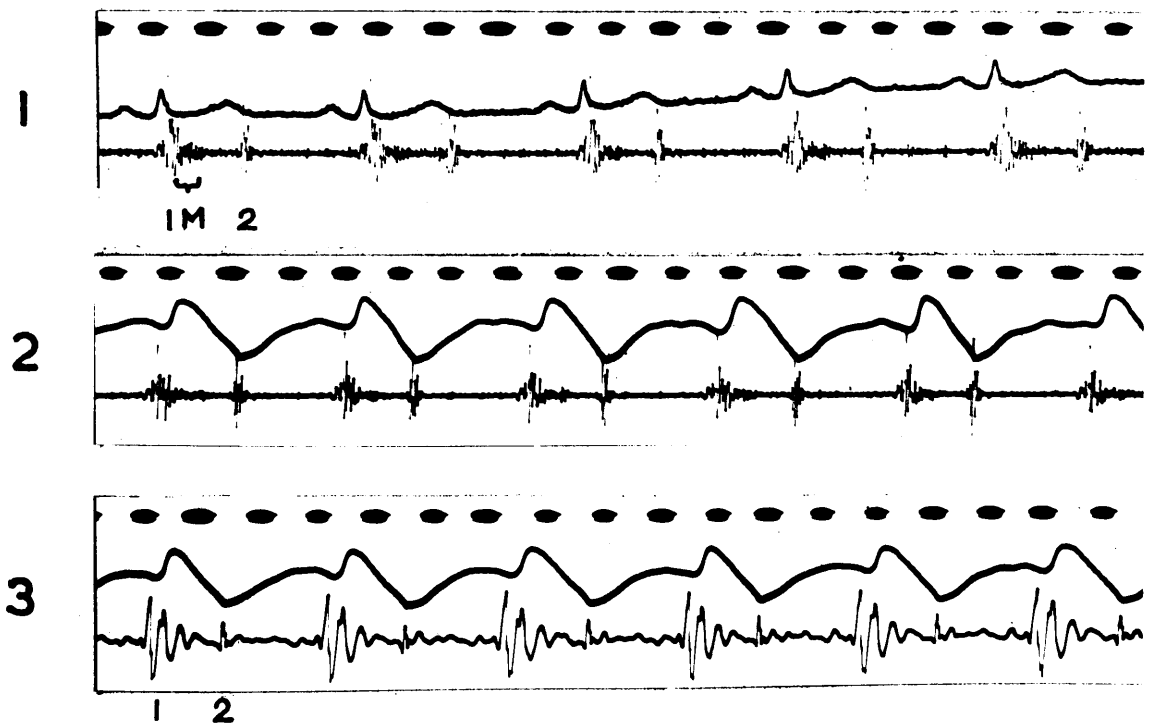
Auscultation
1st and 2nd heart sounds; early systolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; early systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st and 2nd heart sounds.




PATIENT NO.46 APEX




PATIENT NO. 47 APEX

## PLATE 43

### Patient No. 48 - female, age 63

**Diagnosis**

Arterial hypertension
Anterior myocardial infarction
Congestive cardiac failure

**Auscultation**

Very faint 1st and 2nd heart sounds; early systolic murmur.

**Logarithmic P.C.G.**

1st, 2nd, and 4th heart sounds (presystolic gallop); early systolic murmur.

The 'mush' on the baseline of records 1 and 2 is attributable to the high gain of the amplifiers necessary to record the sounds in this case.

**Linear P.C.G.**

Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.

### Patient No. 49 - female, age 44

**Diagnosis**

Arterial hypertension
Mitral stenosis
Cardiac failure

**Auscultation**

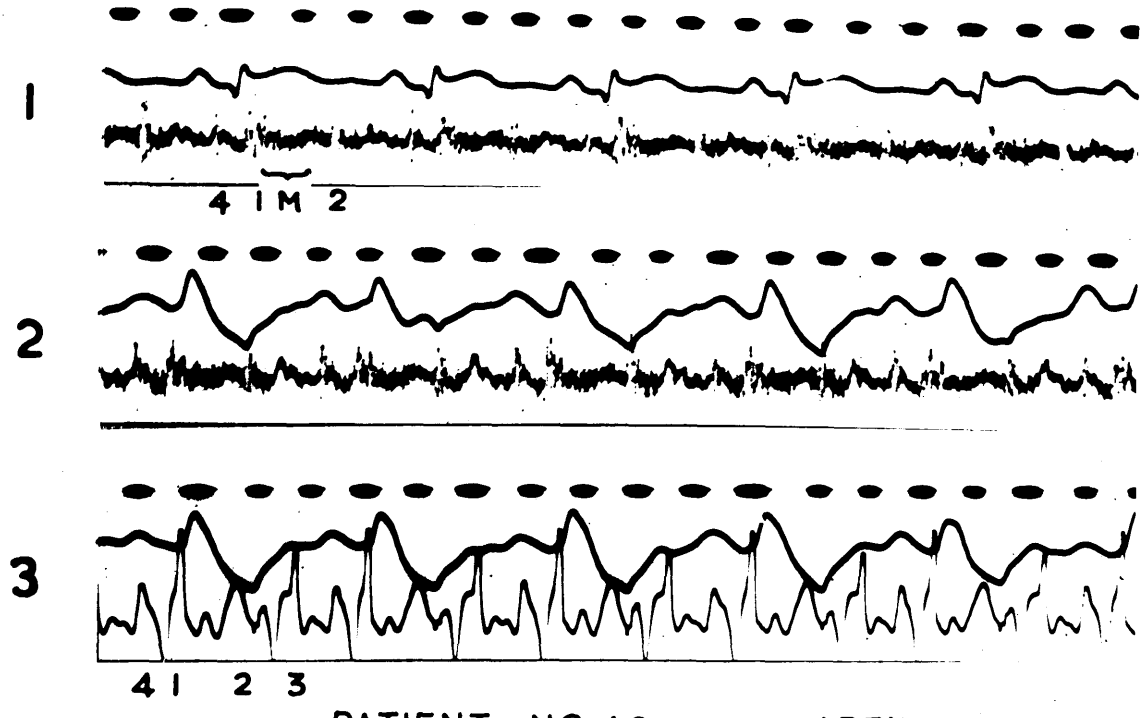
1st and 2nd heart sounds; pansystolic murmur.

**Logarithmic P.C.G.**

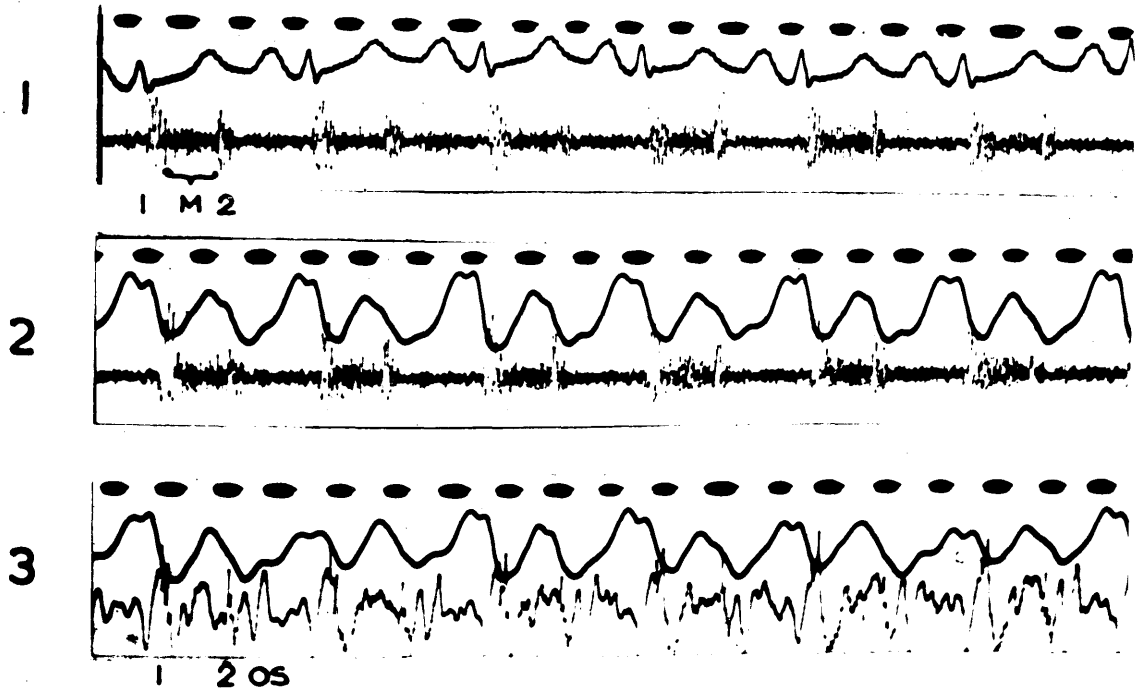
1st and 2nd heart sounds; pansystolic murmur.

**Linear P.C.G.**

Vibrations at the time of 1st and 2nd heart sounds and mitral opening snap.




PATIENT NO. 48 APEX




PATIENT NO. 49 APEX

## PLATE 44

### Patient No. 50 - female, age 42

Diagnosis
Mitral stenosis — Pneumonia (R)
Subacute bacterial endocarditis
Auricular fibrillation

Auscultation
1st and 2nd heart sounds; pansystolic and faint mid-diastolic murmurs.

Logarithmic P.C.G.
1st and 2nd heart sounds, with wide splitting of 2nd sound; pansystolic murmur.

Linear P.C.G.
Vibrations at the time of 1st heart sound and both components of split 2nd sound.

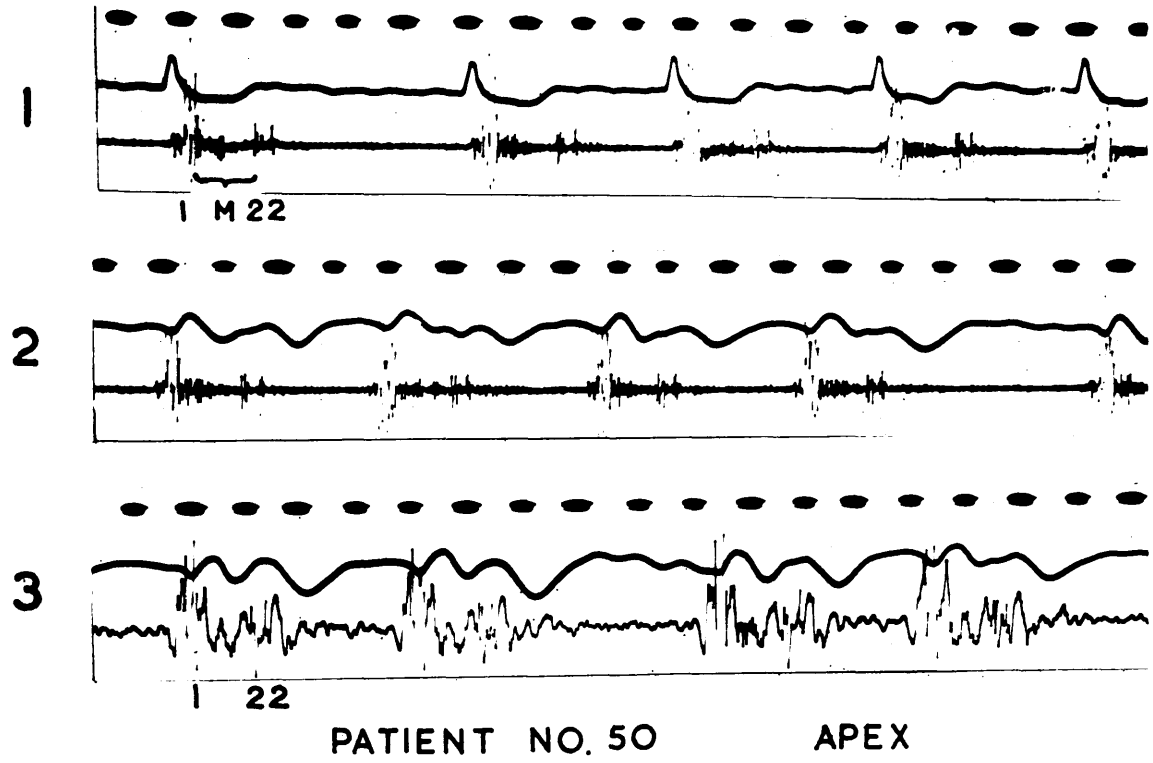
### Patient No. 51 - female, age 38

Diagnosis
Arterial hypertension — Chronic nephritis
Left heart strain

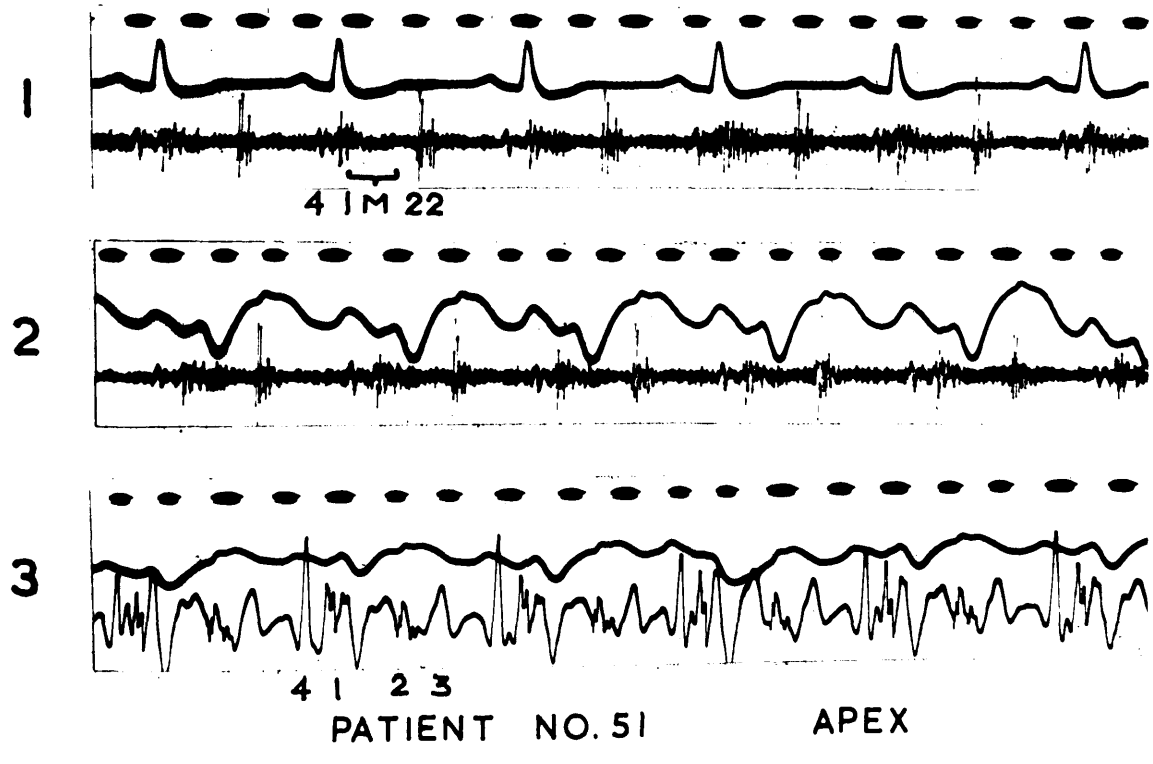
Auscultation
1st and 2nd heart sounds; early systolic murmur.

Logarithmic P.C.G.
1st, 2nd, and 4th heart sounds (presystolic gallop), with splitting of 2nd sound; early and mid-systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 50 APEX




PATIENT NO. 51 APEX

## PLATE 45

### Patient No. 52 - female, age 72

Diagnosis

Arterial hypertension — Chronic nephritis
Auricular fibrillation
Congestive cardiac failure

Auscultation

1st and 2nd heart sounds; early and mid-systolic murmur.

Logarithmic P.C.G.

1st and 2nd heart sounds; early and mid-systolic murmur.

Linear P.C.G.

Vibrations at the time of 1st and 2nd heart sounds.

Note

The reference tracing in records 2 and 3 is strongly arterialized, representing carotid rather than jugular pulsation.

### Patient No. 53 - female, age 31

Diagnosis

Mitral stenosis — Duodenal ulcer

Auscultation

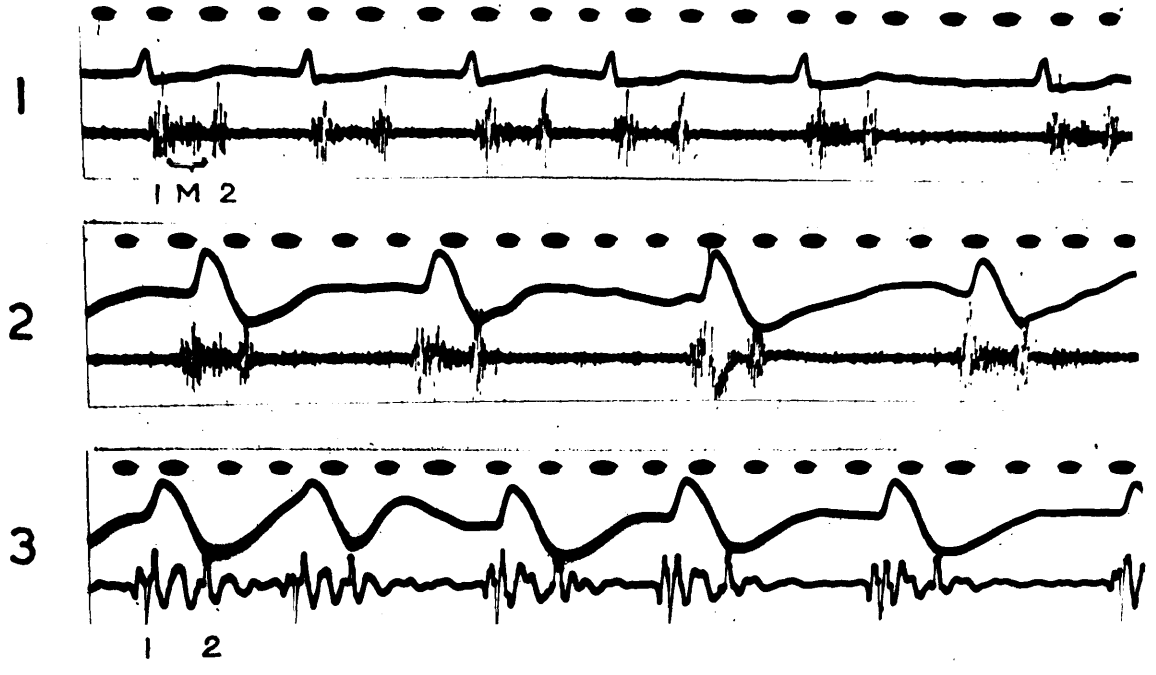
1st and 2nd heart sounds; early systolic murmur.

Logarithmic P.C.G.

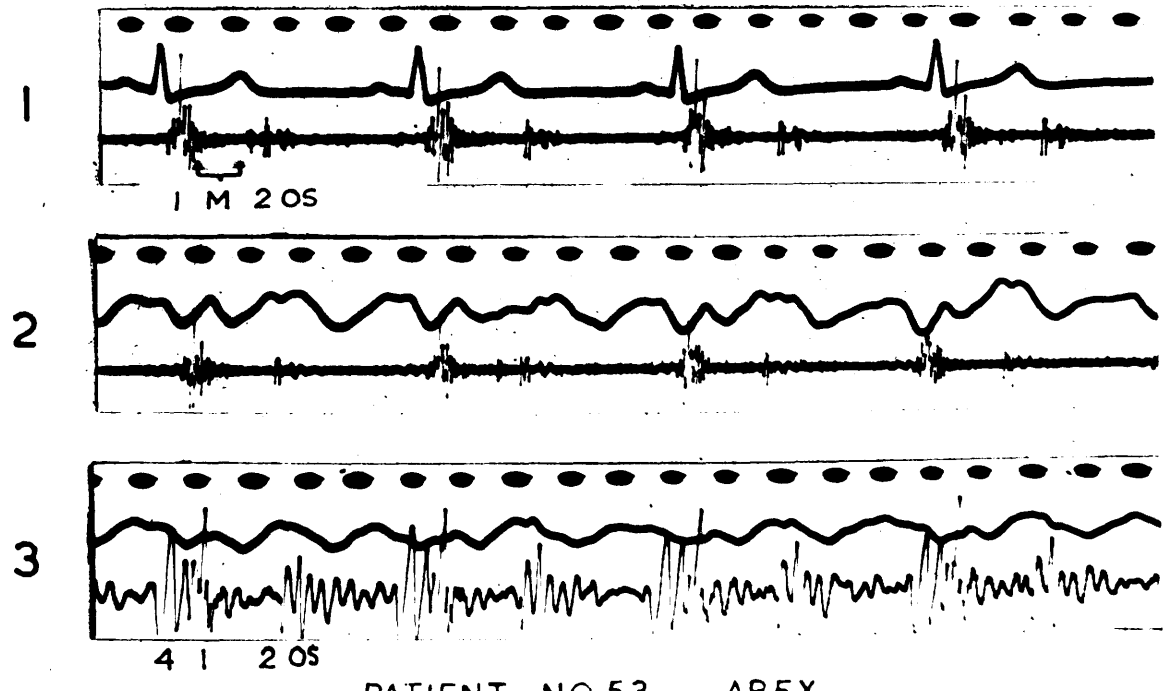
1st and 2nd heart sounds and mitral opening snap; early and mid-systolic murmur.

Linear P.C.G.

Vibrations at the time of 1st, 2nd, and 4th heart sounds and mitral opening snap.




PATIENT NO.52 APEX




PATIENT NO.53 APEX

## PLATE 46

### Patient No. 54 - female, age 48

Diagnosis
Arterial hypertension Chronic bronchitis
Congestive cardiac failure

Auscultation
1st and 2nd heart sounds; early systolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; early systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st and 2nd heart sounds.
The reference tracing shows carotid rather than jugular pulsation.

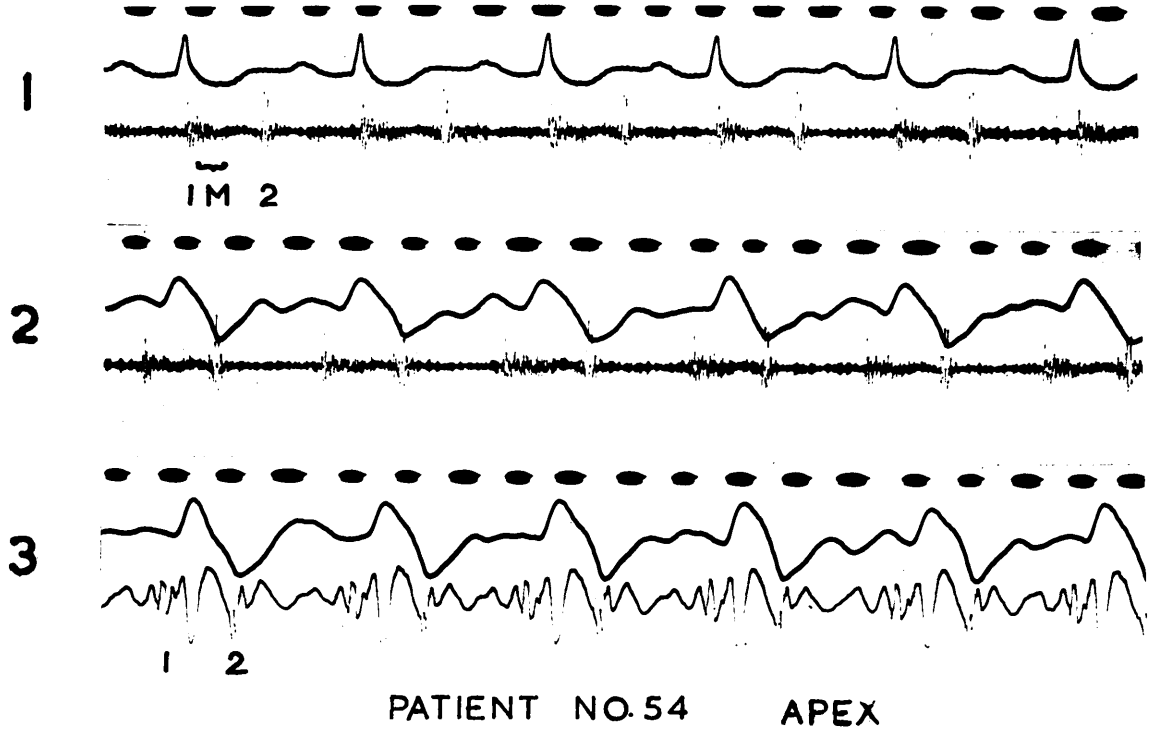
### Patient No. 55 - male, age 28

Diagnosis
Mitral stenosis (post-valvotomy)

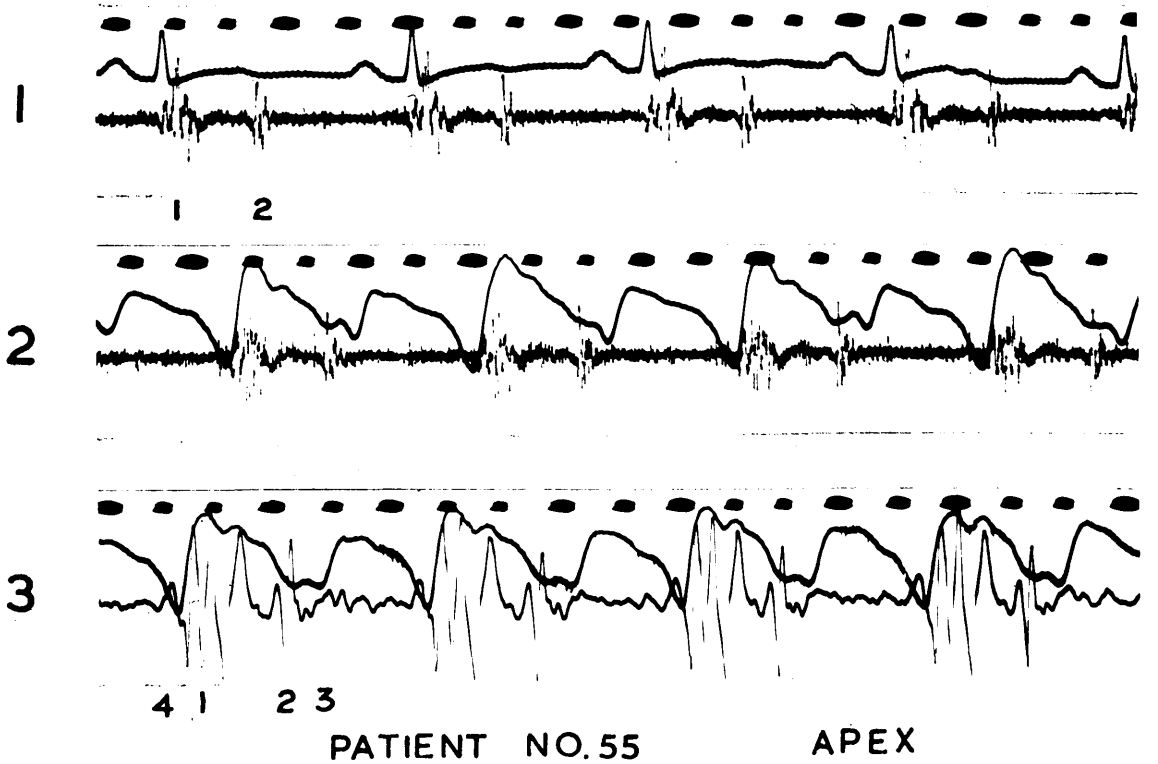
Auscultation
1st and 2nd heart sounds: pansystolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 54 APEX




PATIENT NO. 55 APEX

## PLATE 47

### Patient No. 56 - male, age 56

**Diagnosis**
Angina pectoris

**Auscultation**
1st and 2nd heart sounds; early systolic murmur.

**Logarithmic P.C.G.**
1st and 2nd heart sounds; no murmur.

**Linear P.C.G.**
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.

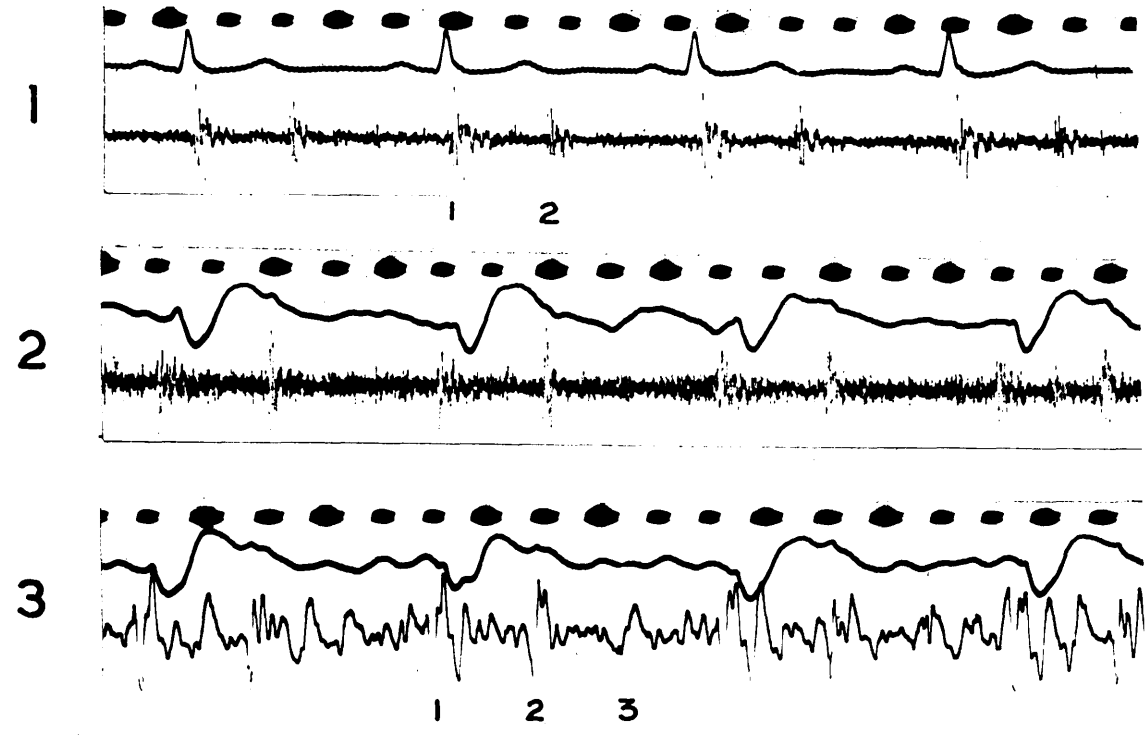
### Patient No. 57 - female, age 63

**Diagnosis**
Arterial hypertension
Left heart strain

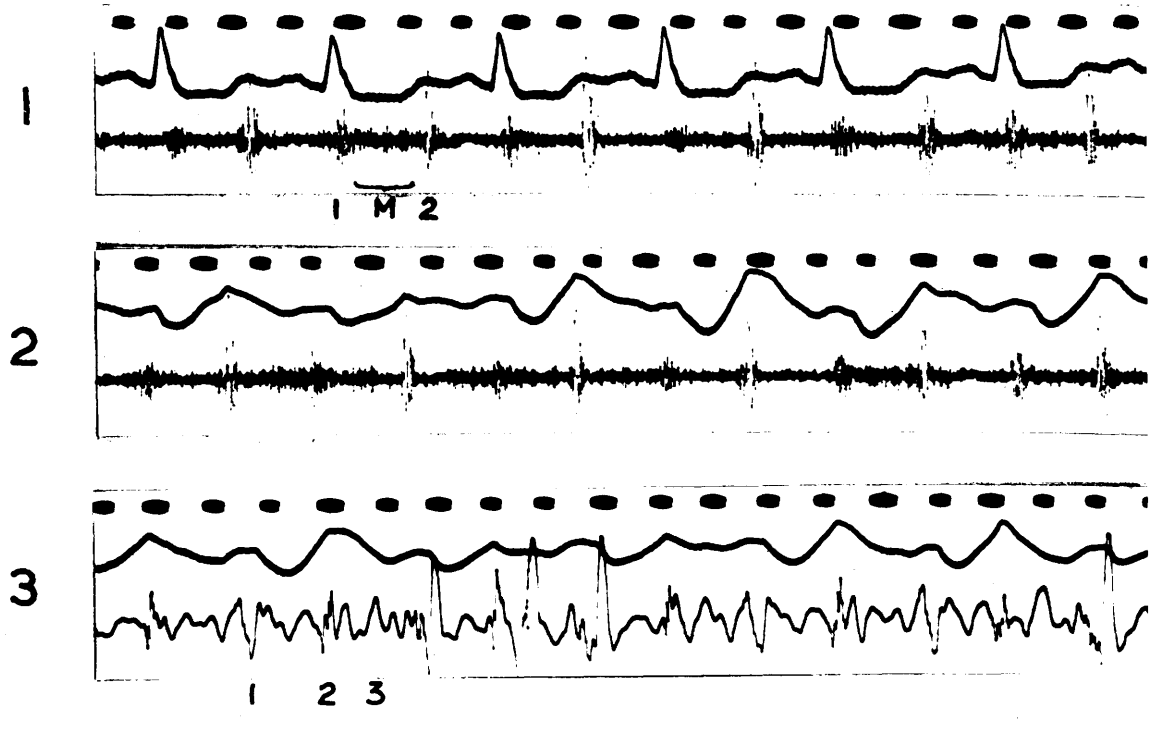
**Auscultation**
1st and 2nd heart sounds; pansystolic murmur.

**Logarithmic P.C.G.**
1st and 2nd heart sounds; pansystolic murmur.

**Linear P.C.G.**
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.




PATIENT NO.56 APEX




PATIENT NO.57 APEX

## PLATE 48

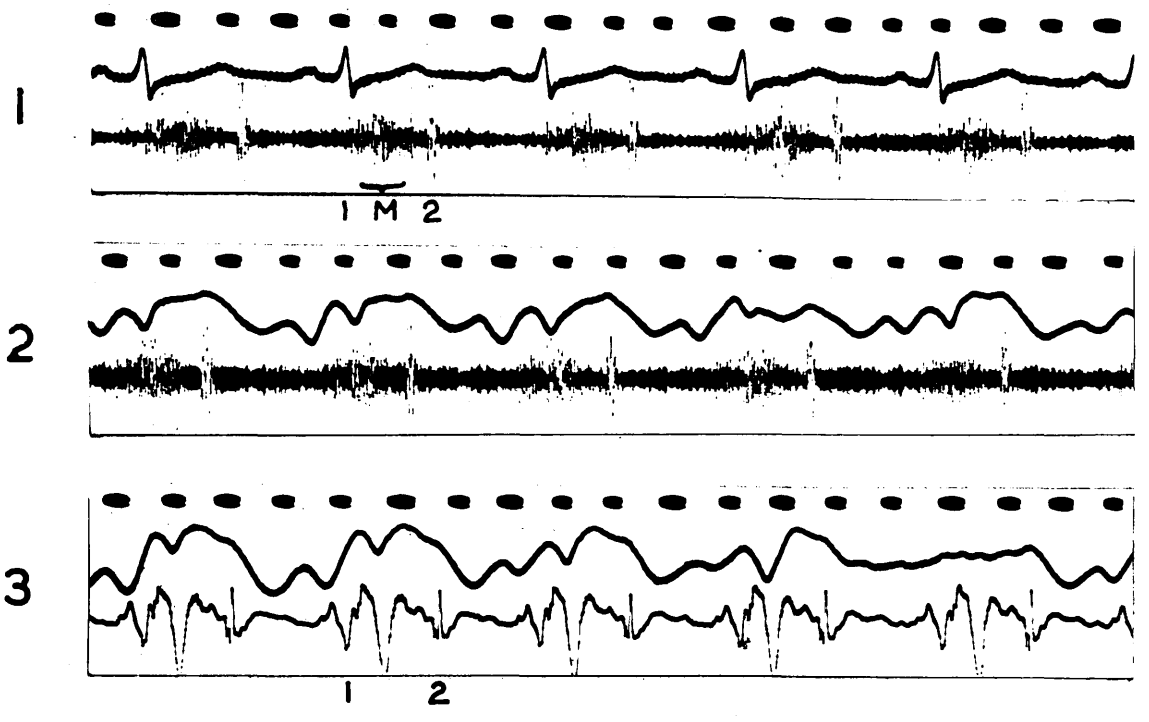
### Patient No. 58 - female, age 53

Diagnosis
Arterial hypertension
Left heart strain

Auscultation
1st and 2nd heart sounds; pansystolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; early and mid-systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st and 2nd heart sounds.




PATIENT NO. 58 APEX

## PLATE 49

### Patient No. 59 - female, age 32

**Diagnosis**

Mitral stenosis
Aortic incompetence
Auricular fibrillation

**Auscultation**

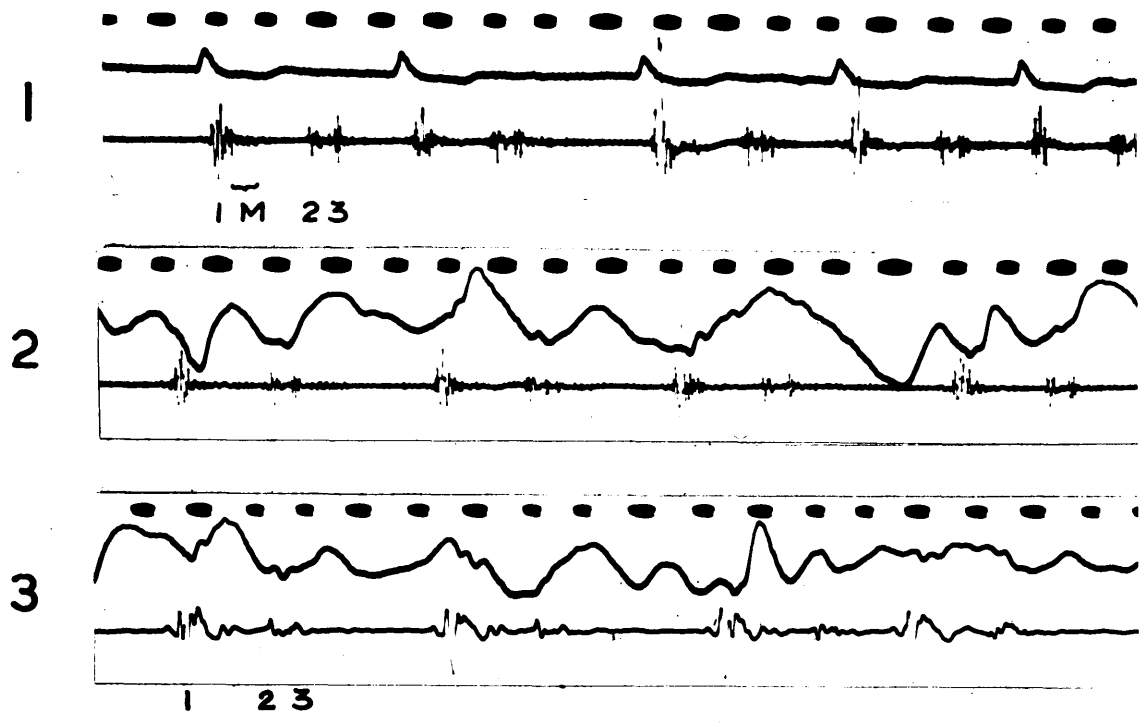
1st, 2nd, and 3rd heart sounds (protodiastolic gallop); early systolic murmur. At sternal end of 3rd left intercostal space, 1st and 2nd heart sounds; early and mid-systolic and early diastolic murmurs.

**Logarithmic P.C.G.**

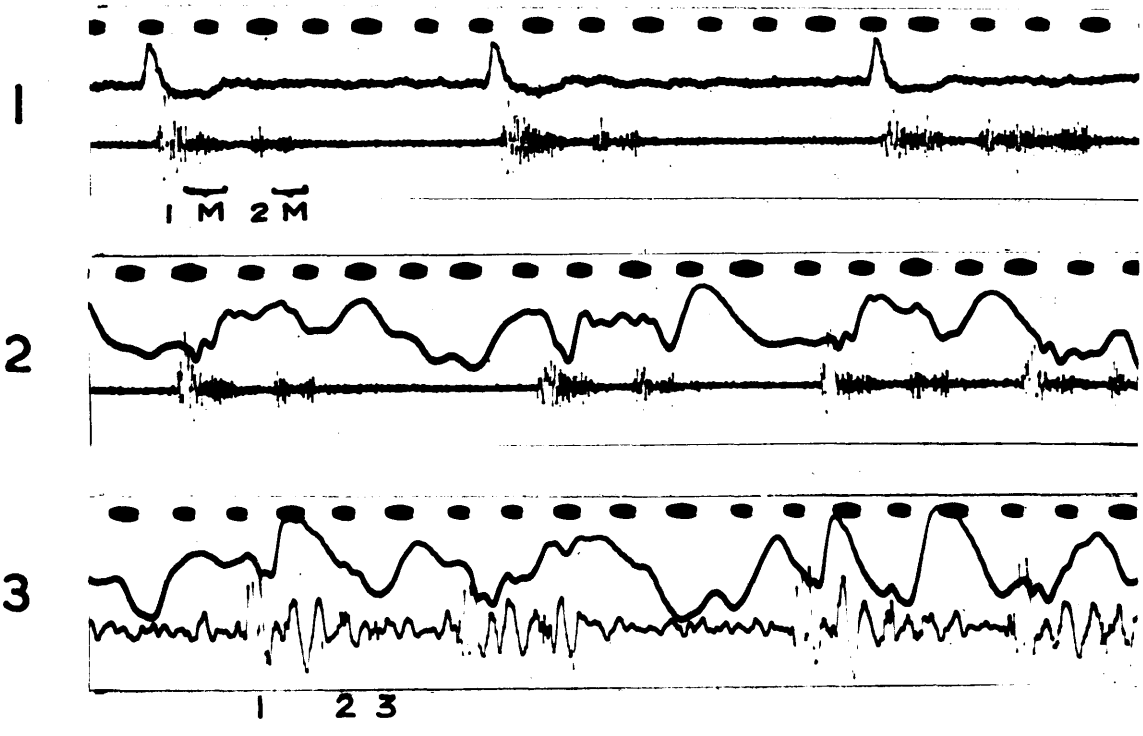
1st, 2nd, and 3rd heart sounds (protodiastolic gallop); early systolic murmur. At sternal end of 3rd left intercostal space, 1st and 2nd heart sounds; early and mid-systolic and early diastolic murmurs.

**Linear P.C.G.**

Vibrations at the time of 1st, 2nd, and 3rd heart sounds. The same at sternal end of 3rd left intercostal space.




PATIENT NO. 59 APEX




PATIENT NO. 59 3rd.L.I.S.

## PLATE 50

### Patient No. 60 - female, age 36

Diagnosis

Mitral stenosis
Aortic incompetence
Congestive cardiac failure

Auscultation

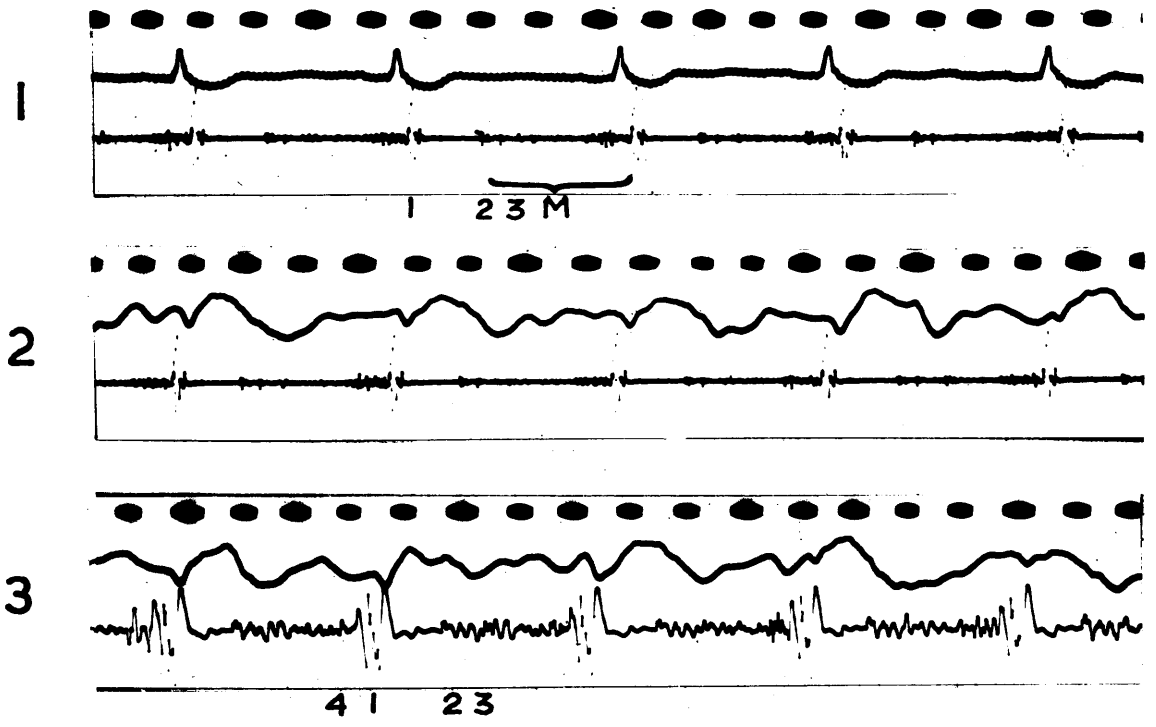
1st and 2nd heart sounds; pansystolic and early and late diastolic murmurs.

Logarithmic P.C.G.

1st, 2nd, and 3rd heart sounds; pandiastolic murmur with presystolic accentuation.

Linear P.C.G.

Vibrations at the time of 1st, 2nd, 3rd and 4th heart sounds.




PATIENT NO. 60 APEX

# PLATE 51

## Patient No. 61 - female, age 11

**Diagnosis**

Congenital pulmonary stenosis

**Auscultation**

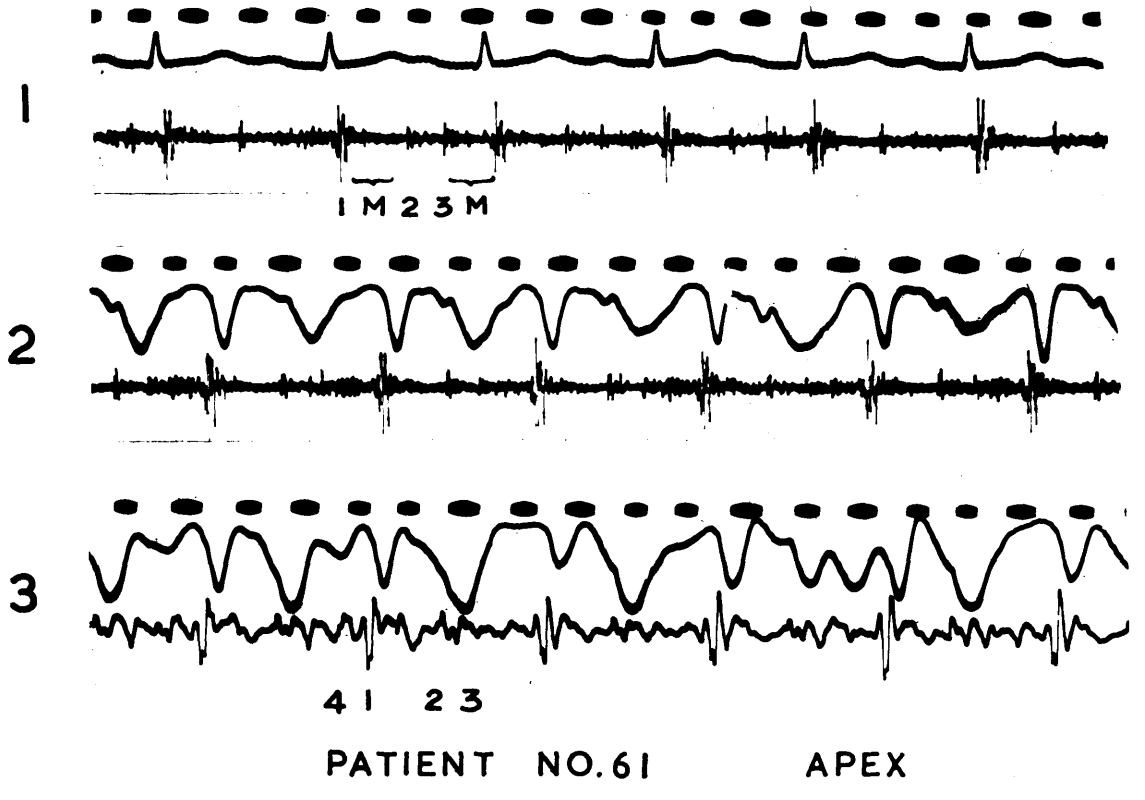
1st and 2nd heart sounds; early and mid-systolic and late diastolic murmurs. At pulmonary area, 1st and 2nd heart sounds with splitting of 2nd sound; pansystolic murmur.

**Logarithmic P.C.G.**

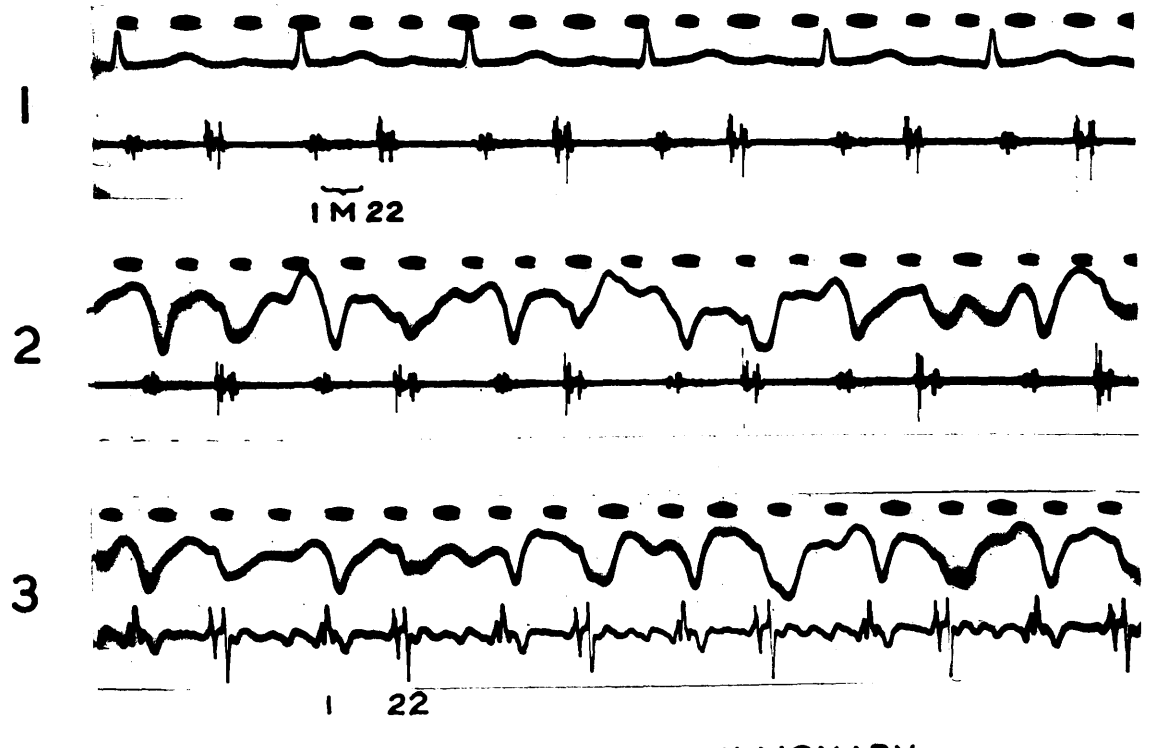
1st, 2nd, and 3rd heart sounds; early and mid-systolic and mid- and late diastolic murmurs. At pulmonary area, 1st and 2nd heart sounds with splitting of 2nd sound; early and mid-systolic murmur.

**Linear P.C.G.**

Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds. At pulmonary area, vibrations at the time of 1st heart sound and both components of split 2nd sound.




PATIENT NO. 61 APEX




PATIENT NO. 61 PULMONARY

# PLATE 52

## Patient No. 62 - female, age 52

Diagnosis
Mitral stenosis
Aortic incompetence
Auricular fibrillation
Congestive cardiac failure

Auscultation
1st and 2nd heart sounds; pansystolic murmur.

Logarithmic P.C.G.
1st, 2nd, and 3rd heart sounds (protodiastolic gallop); pansystolic and early diastolic murmurs.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.

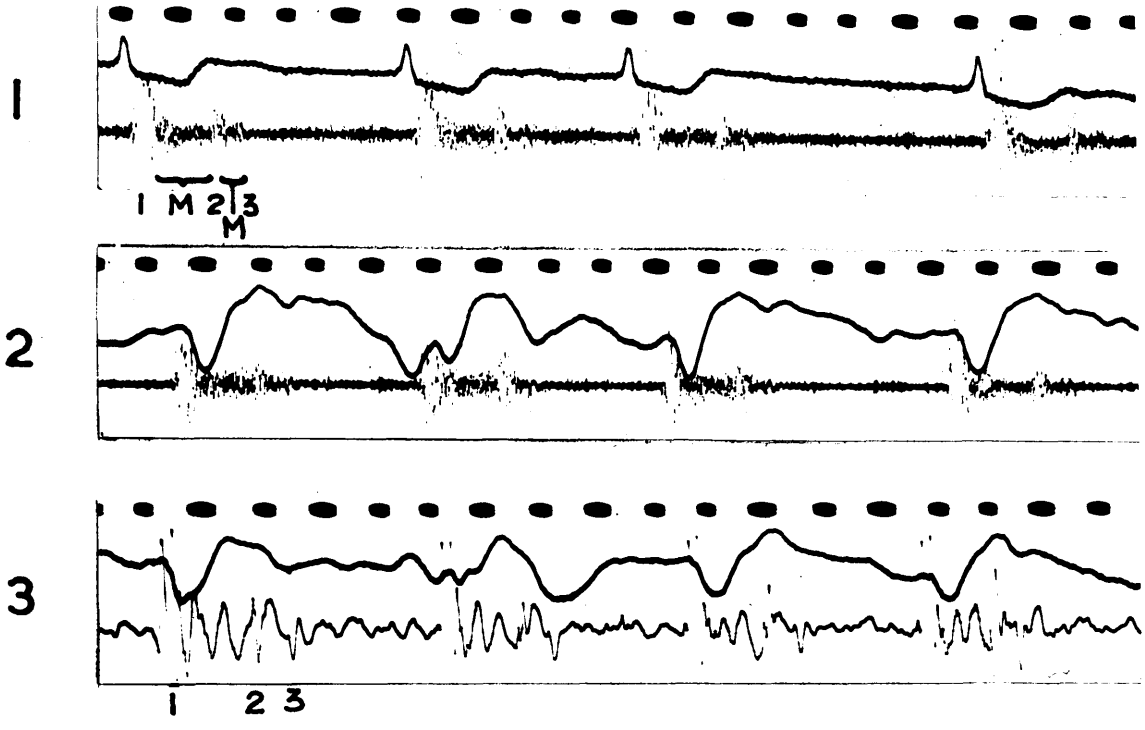
## Patient No. 63 - male, age 59

Diagnosis
Posterior myocardial infarction
Left heart strain

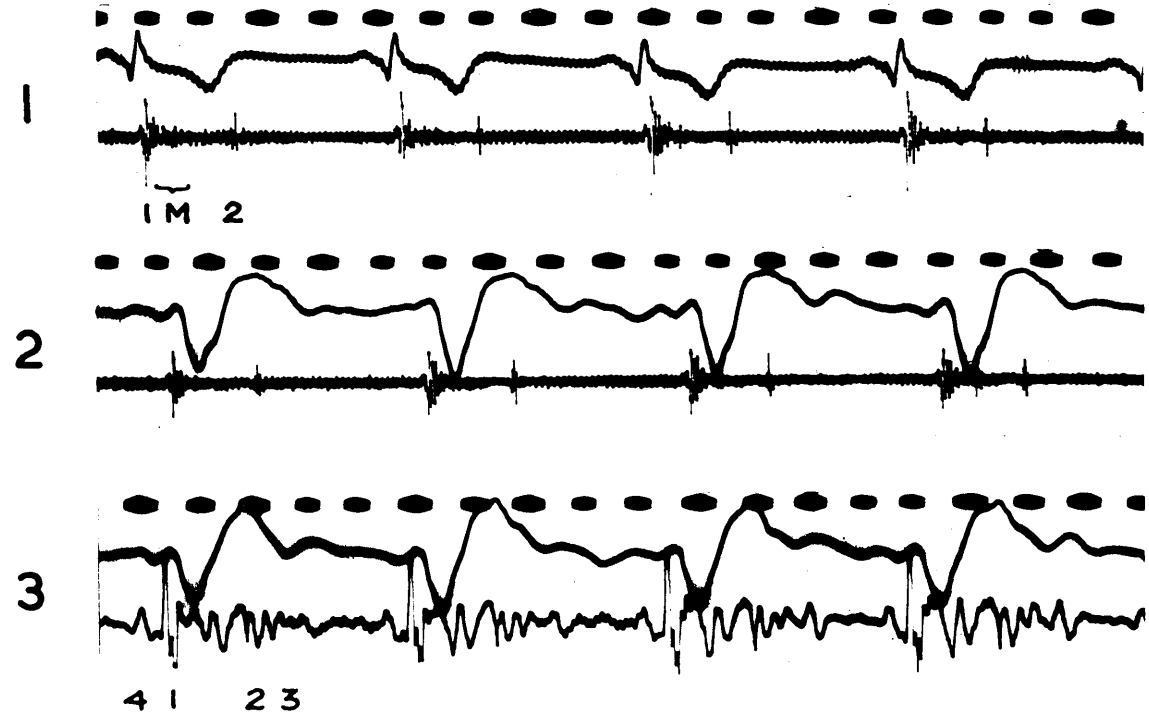
Auscultation
1st and 2nd heart sounds; soft early and mid-systolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; early systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 62 APEX




PATIENT NO. 63 APEX

## PLATE 53

### Patient No. 64 - male, age 59

Diagnosis
Arterial hypertension
Myocardial disease
Left heart strain

Auscultation
1st and 2nd heart sounds; early and mid-systolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; early and mid-systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.

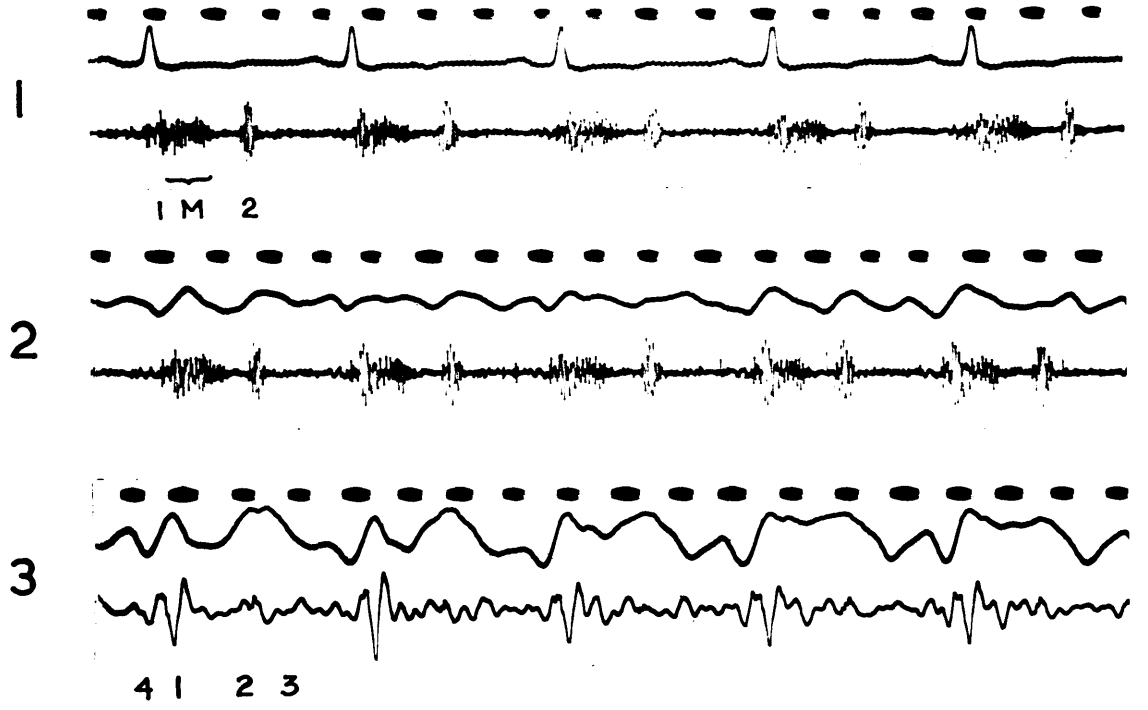
### Patient No. 65 - female, age 53

Diagnosis
Congenital pulmonary stenosis Duodenal ulcer

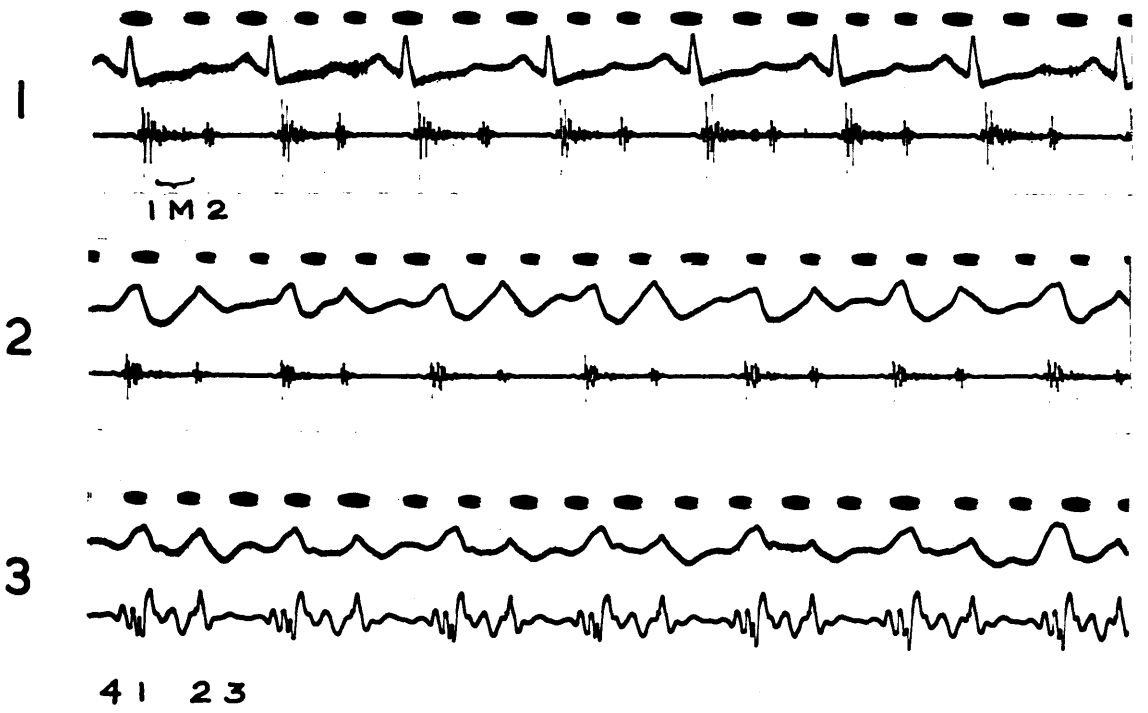
Auscultation
1st and 2nd heart sounds; pansystolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; early and mid-systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 64 APEX




PATIENT NO. 65 APEX

# PLATE 54

## Patient No. 66 - female, age 34

**Diagnosis**
Mitral stenosis (post-valvotomy)

**Auscultation**
1st and 2nd heart sounds; pandiastolic murmur.

**Logarithmic P.C.G.**
1st, 2nd, and 3rd heart sounds (protodiastolic gallop); no murmur.

**Linear P.C.G.**
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.

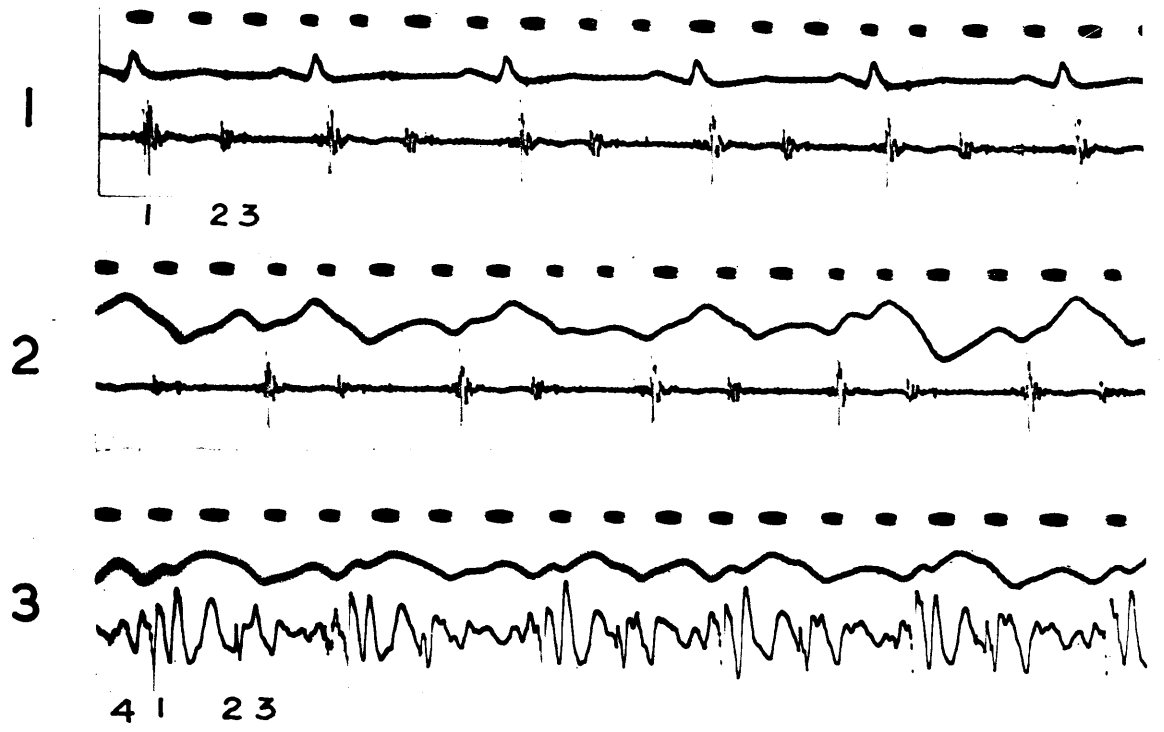
## Patient No. 67 - male, age 31

**Diagnosis**
Mitral incompetence
Acute rheumatic carditis

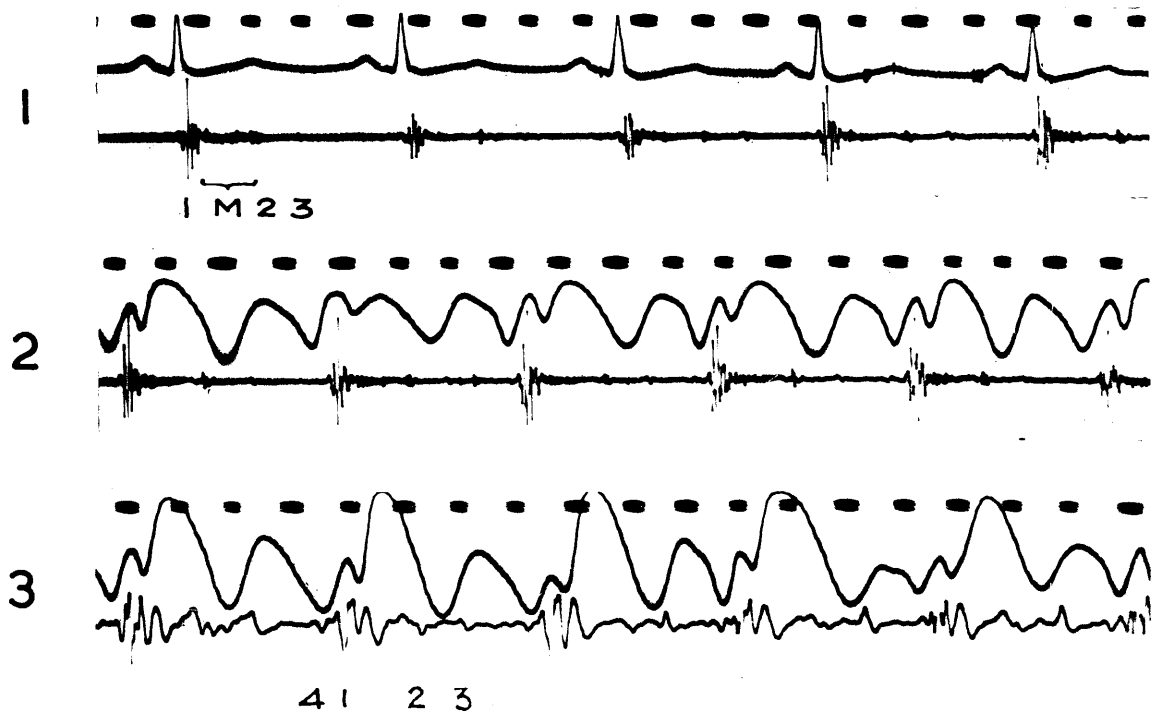
**Auscultation**
1st and 2nd heart sounds; pansystolic murmur.

**Logarithmic P.C.G.**
1st, 2nd, and 3rd heart sounds (protodiastolic gallop); pansystolic murmur.

**Linear P.C.G.**
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO.66 APEX




PATIENT NO.67 APEX

PLATE 55

## Patient No. 68 - male, age 58

Diagnosis
Anterior myocardial infarction

Auscultation
1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.

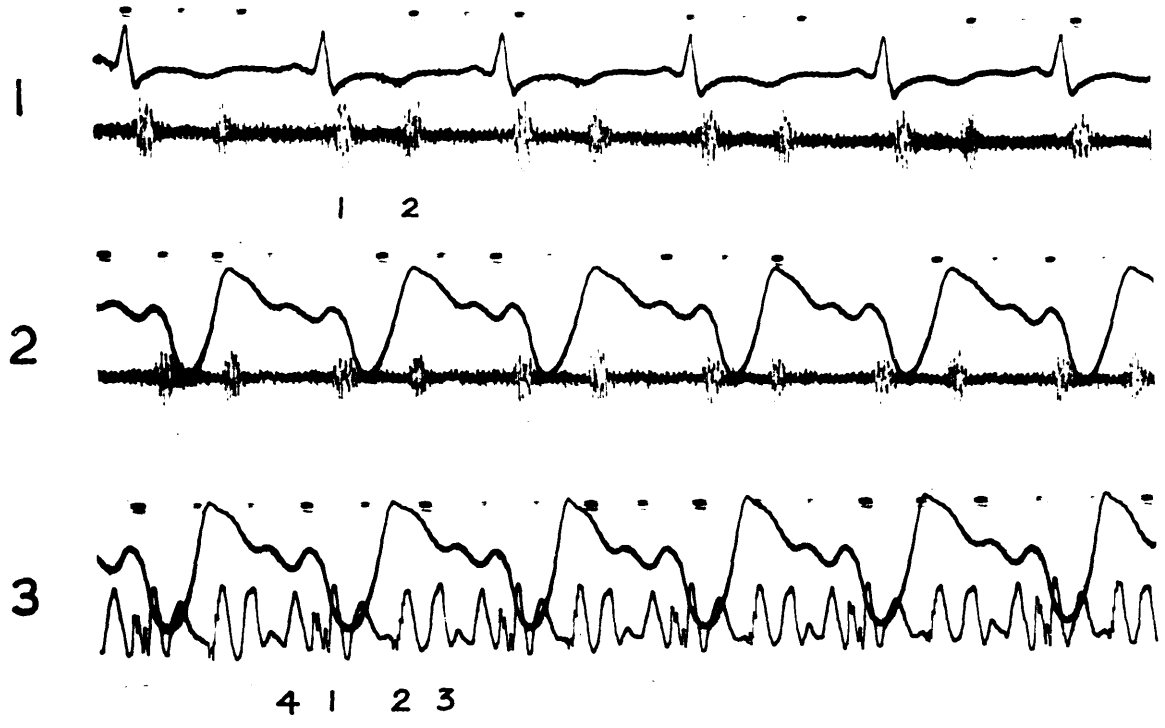
## Patient No. 69 - male, age 62

Diagnosis
Arterial hypertension
Myocardial disease
Left heart strain
Diabetes mellitus

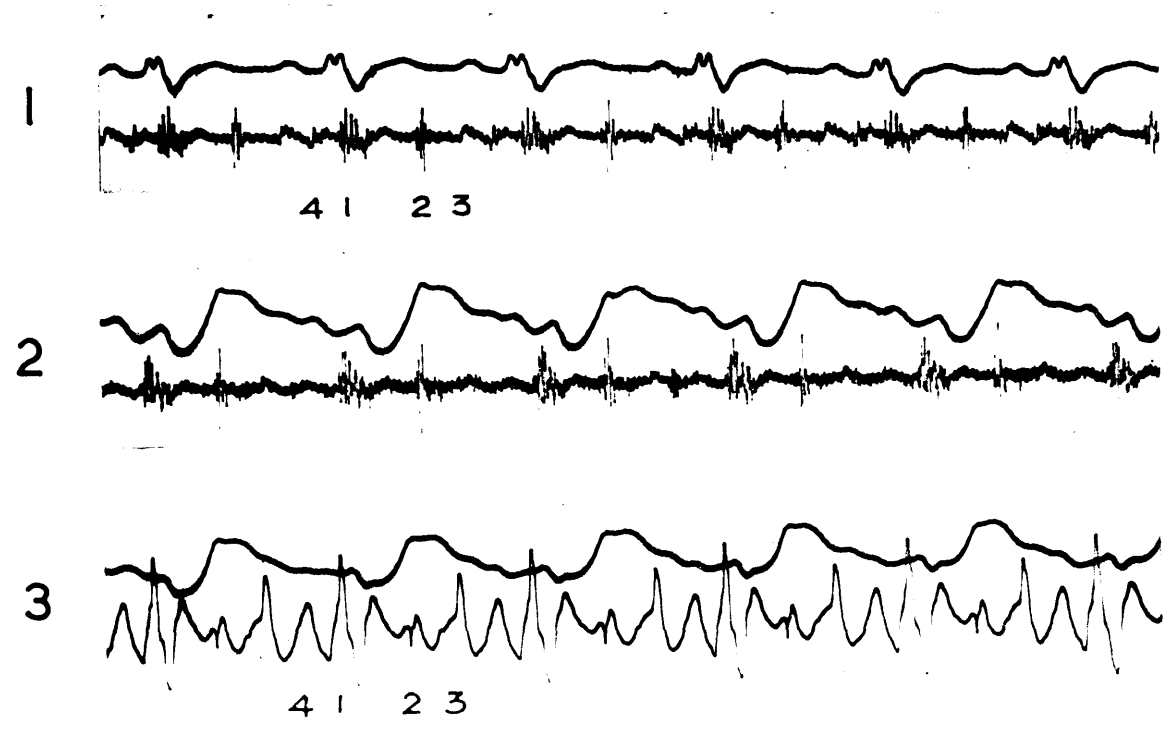
Auscultation
1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st, 2nd, 3rd, and 4th heart sounds; no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 68 APEX




PATIENT NO. 69 APEX

PLATE 56

## Patient No. 70 - male, age 76

Diagnosis
Anterior myocardial infarction

Auscultation
1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd and 4th heart sounds.

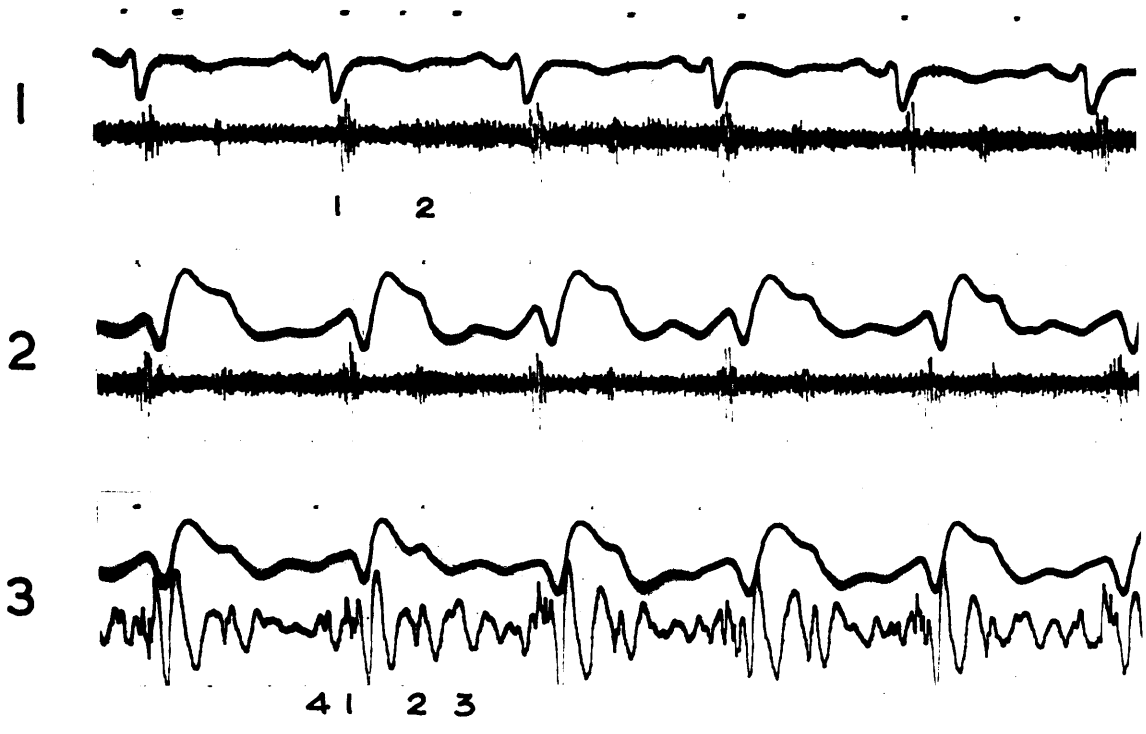
## Patient No. 71 - female, age 59

Diagnosis
Arterial hypertension Bronchial asthma
Left bundle branch block

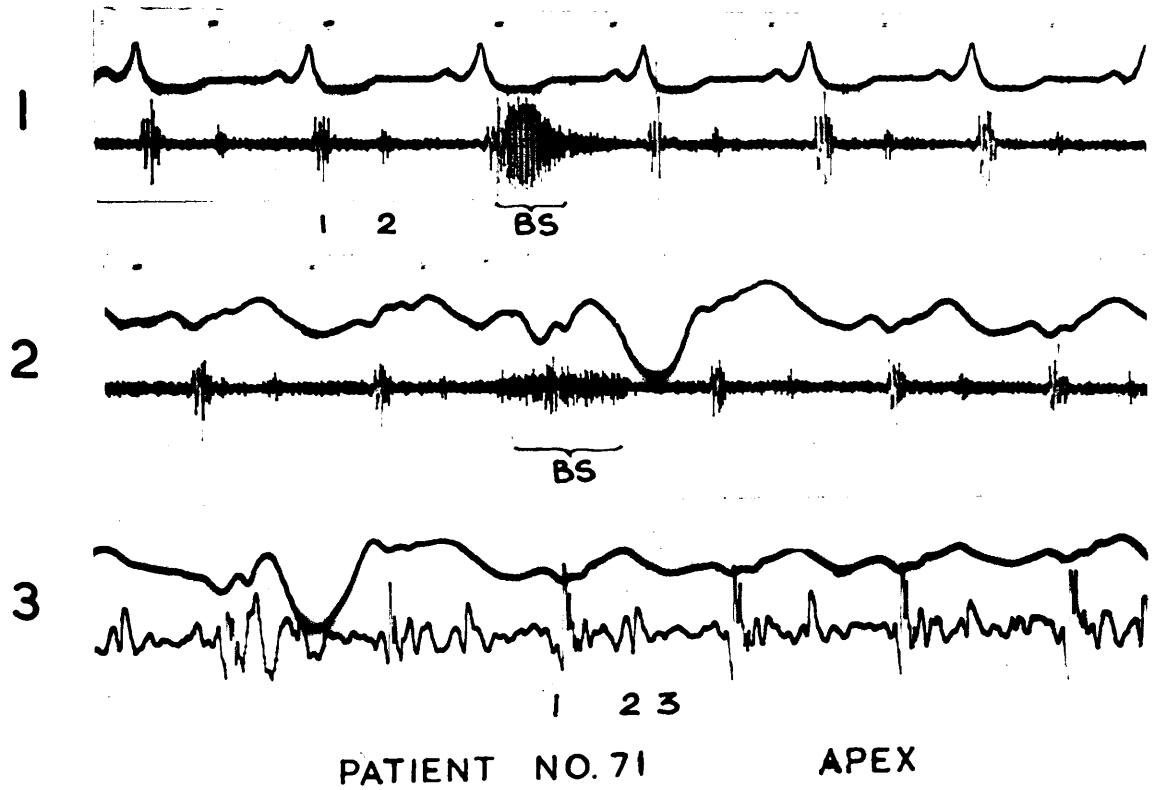
Auscultation
1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; no murmur.
Breath sounds (BS) on records 1 and 2.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.




PATIENT NO. 70 APEX




PATIENT NO. 71 APEX

## PLATE 57

### Patient No. 72 - male, age 45

**Diagnosis**
Posterior myocardial infarction

**Auscultation**
1st and 2nd heart sounds; no murmur.

**Logarithmic P.C.G.**
1st and 2nd heart sounds; no murmur.

**Linear P.C.G.**
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.

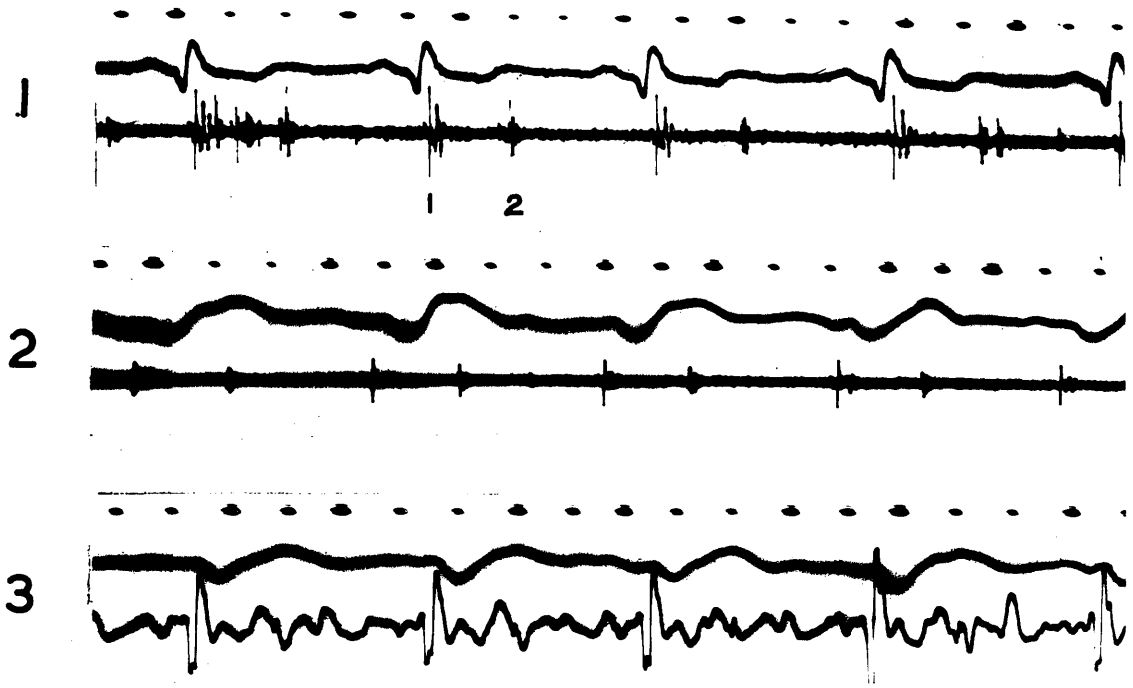
### Patient No. 73 - female, age 40

**Diagnosis**
Mitral stenosis

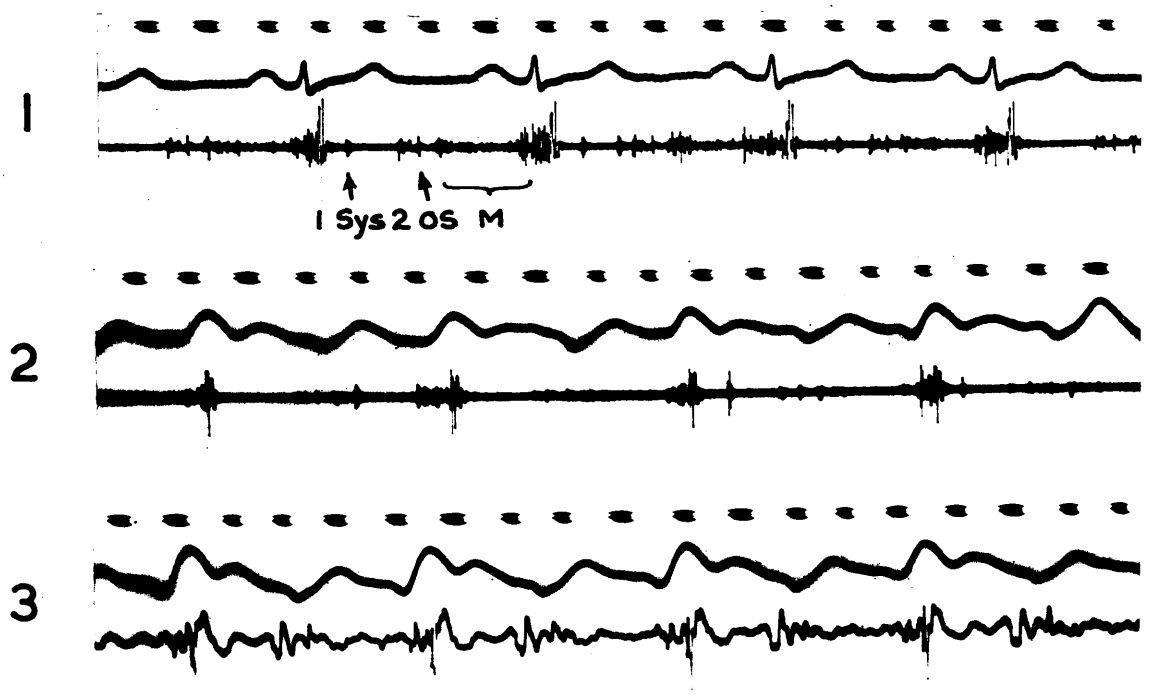
**Auscultation**
1st and 2nd heart sounds; mid- and late diastolic murmur.

**Logarithmic P.C.G.**
1st and 2nd heart sounds, systolic extra sound, and mitral opening snap; mid- and late diastolic murmur with presystolic accentuation.

**Linear P.C.G.**
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 72 APEX




PATIENT NO. 73 APEX

## PLATE 58

### Patient No. 74 - female, age 61

Diagnosis

Cor pulmonale — Bronchial asthma

Auscultation

1st and 2nd heart sounds; no murmur

Logarithmic P.C.G.

1st and 2nd heart sounds and systolic extra sound; no murmur.

Linear P.C.G.

Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.

### Patient No. 75 - male, age 49

Diagnosis

Mitral stenosis — Lobar pneumonia (L)
Myocardial disease — Acute nephritis
Auricular fibrillation
Congestive cardiac failure

Auscultation

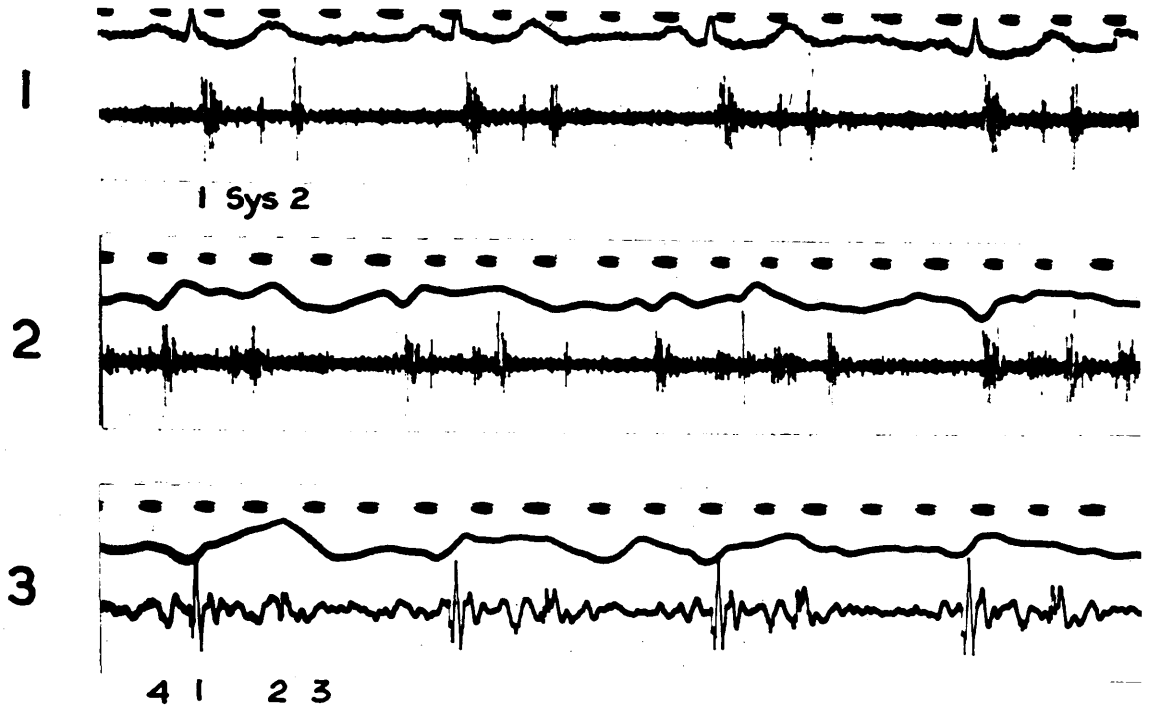
1st and 2nd heart sounds; pansystolic and early and mid-diastolic murmurs.

Logarithmic P.C.G.

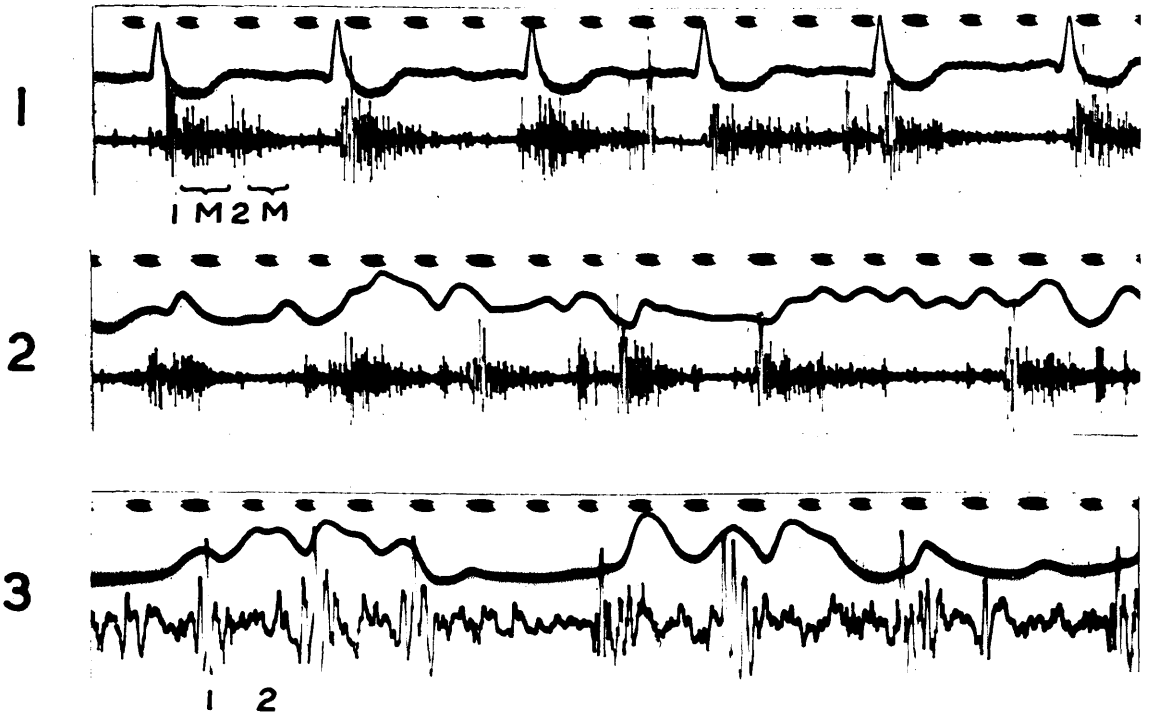
1st and 2nd heart sounds; pansystolic and early diastolic murmurs.

Linear P.C.G.

Vibrations at the time of 1st and 2nd heart sounds.




PATIENT NO. 74 APEX




PATIENT NO. 75 APEX

# PLATE 59

## Patient No. 76 - male, age 39

**Diagnosis**
Arterial hypertension Malignant hypertension
Cardiac failure

**Auscultation**
1st and 2nd heart sounds; pansystolic murmur.

**Logarithmic P.C.G.**
1st and 2nd heart sounds; pansystolic murmur.

**Linear P.C.G.**
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.

## Patient No. 77 - female, age 23

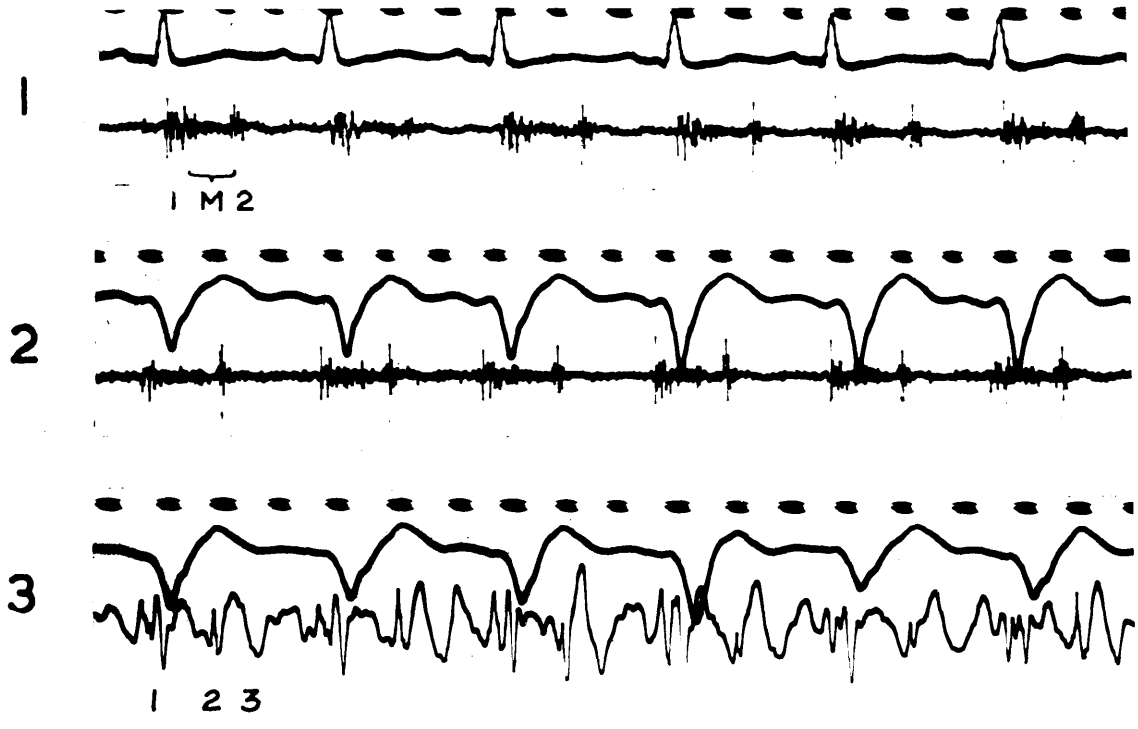
**Diagnosis**
Mitral incompetence

**Auscultation**
Loud 1st and 2nd heart sounds; pansystolic murmur.

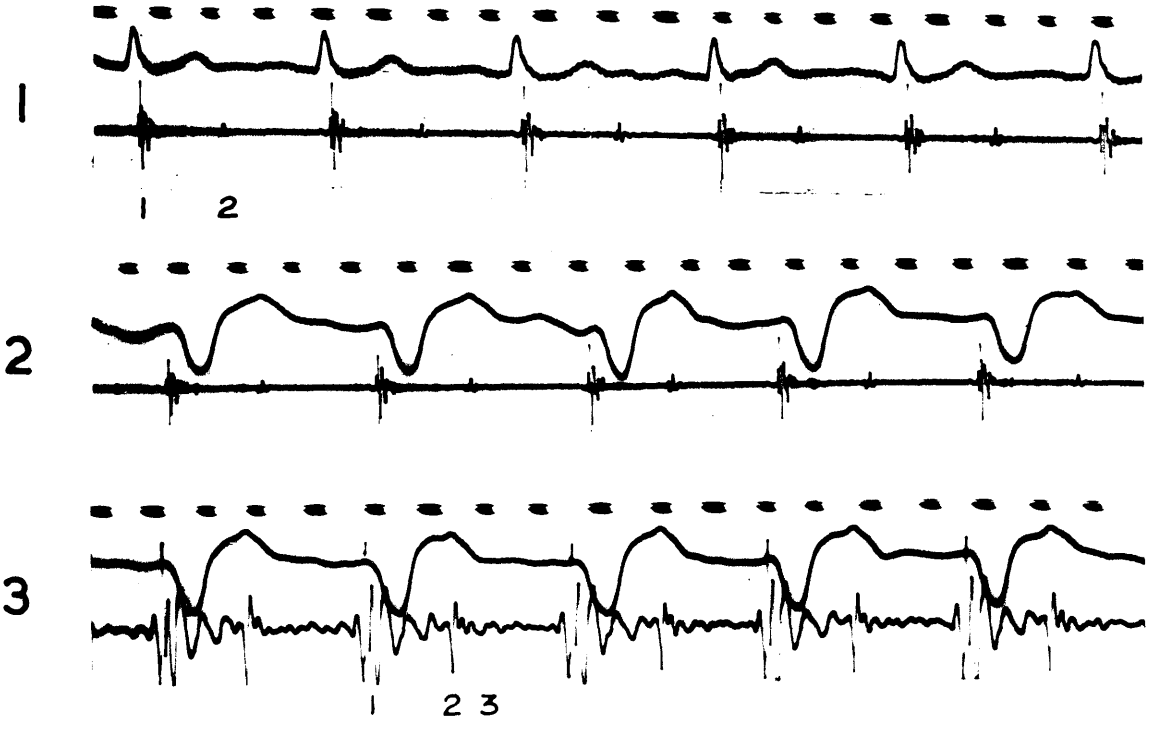
**Logarithmic P.C.G.**
1st and 2nd heart sounds; no murmur.

**Linear P.C.G.**
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.

**Note**
The absence of the mitral systolic murmur on the logarithmic P.C.G. can be attributed to the gain reduction necessary to record the whole extent of the deflections due to the loud 1st sound.




PATIENT NO.76 APEX




PATIENT NO.77 APEX

## PLATE 60

### Patient No. 78 - female, age 52

Diagnosis
Mitral stenosis Hemiplegia (R)

Auscultation
1st and 2nd heart sounds; early systolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; early systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.

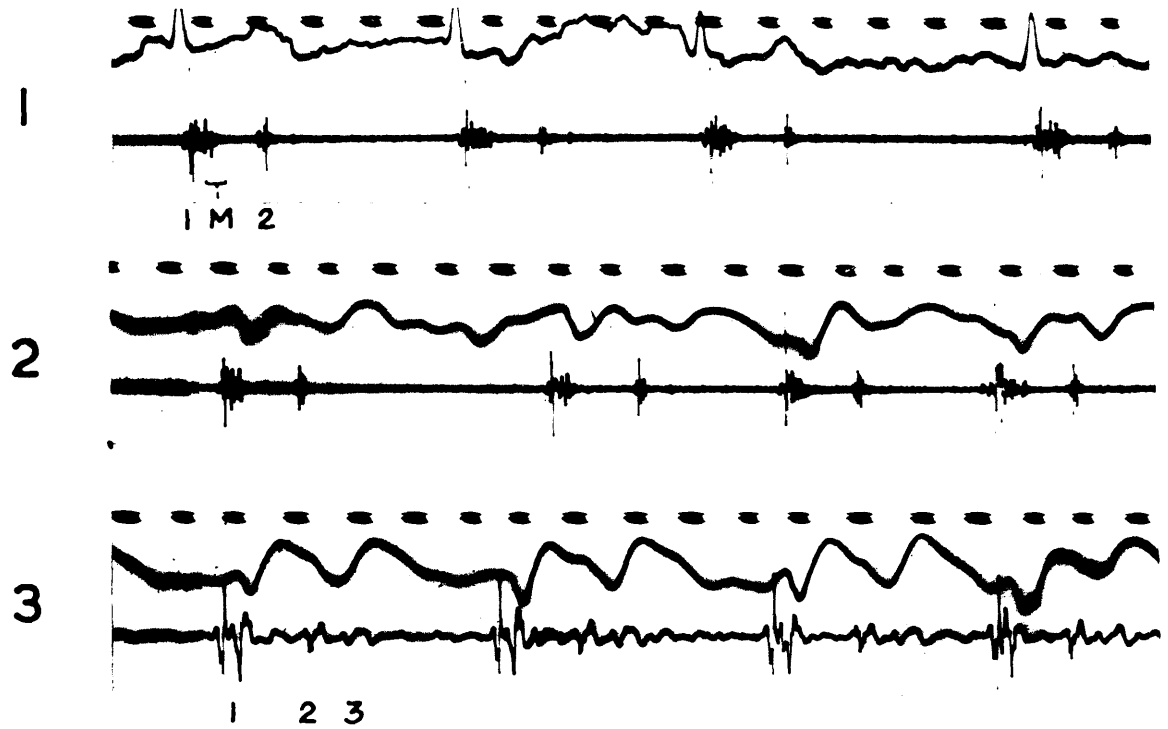
### Patient No. 79 - female, age 68

Diagnosis
Cor pulmonale Basal pneumonia

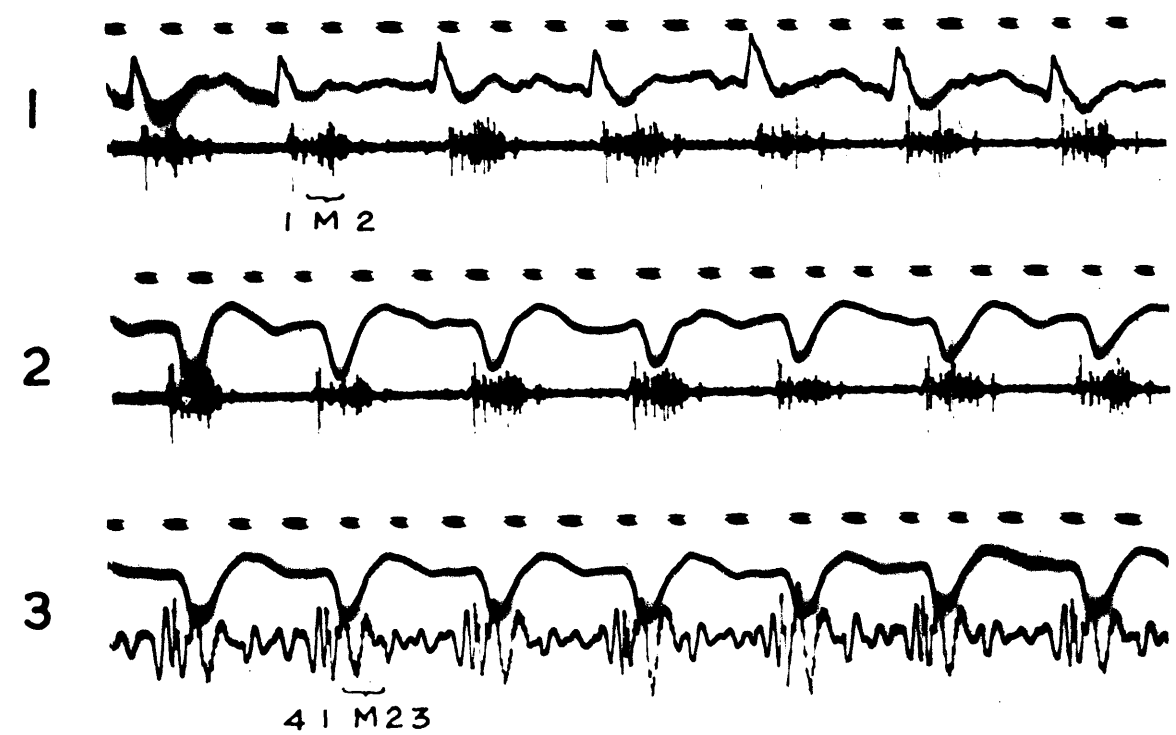
Auscultation
1st and 2nd heart sounds; pansystolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; early and mid-systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds; indication of systolic murmur.




PATIENT NO.78 APEX




PATIENT NO.79 APEX

# PLATE 61

## Patient No. 80 - male, age 61

Diagnosis
Left bundle branch block
Cardiac failure

Auscultation
Faint 1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st, 2nd, and 4th heart sounds (presystolic gallop); no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.

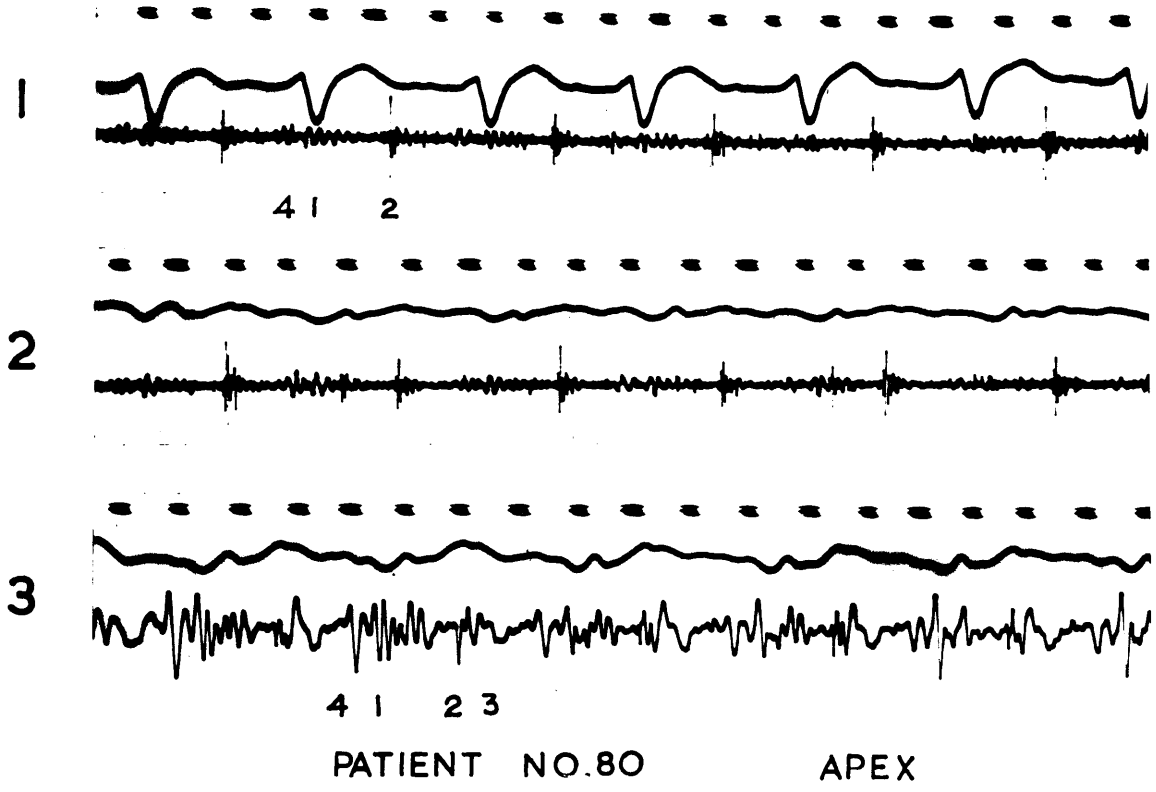
## Patient No. 81 - male, age 60

Diagnosis
Angina pectoris
Left bundle branch block

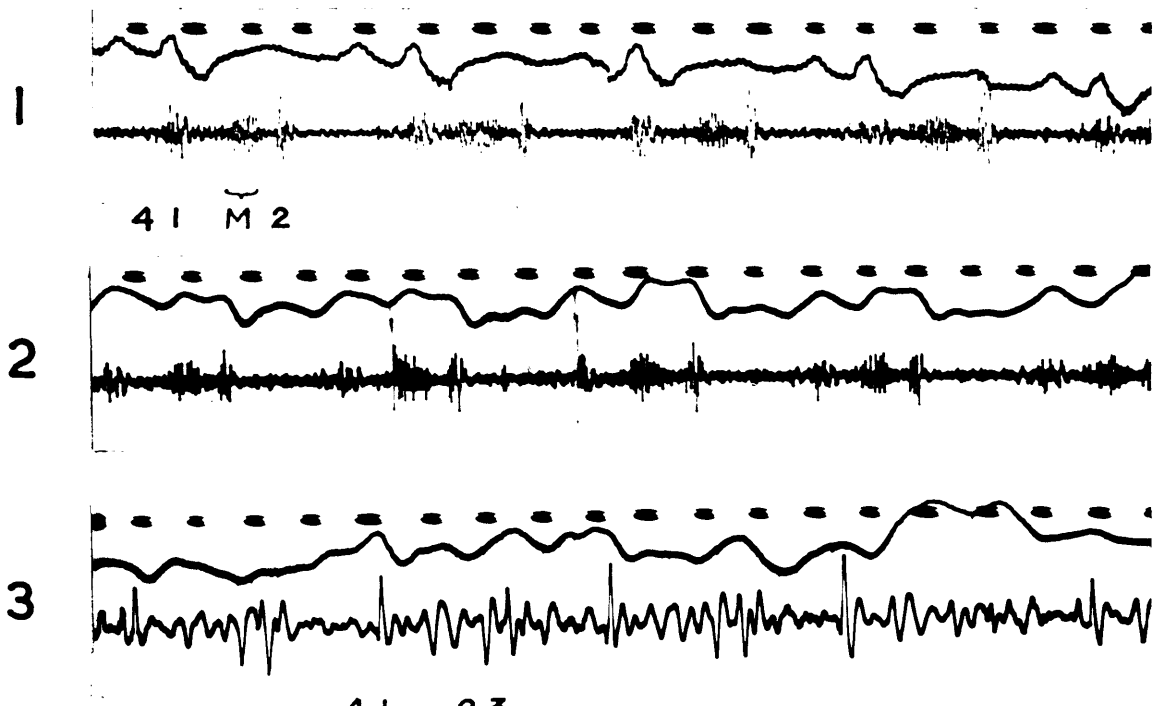
Auscultation
1st, 2nd, and 4th heart sounds (presystolic gallop); early systolic murmur.

Logarithmic P.C.G.
1st, 2nd, and 4th heart sounds (presystolic gallop); mid-systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO.80 APEX




PATIENT NO.81 APEX

# PLATE 62

## Patient No. 82 - male, age 50

Diagnosis
Right bundle branch block  Chronic bronchitis
Cor pulmonale

Auscultation
1st and 2nd heart sounds; early systolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; early systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.

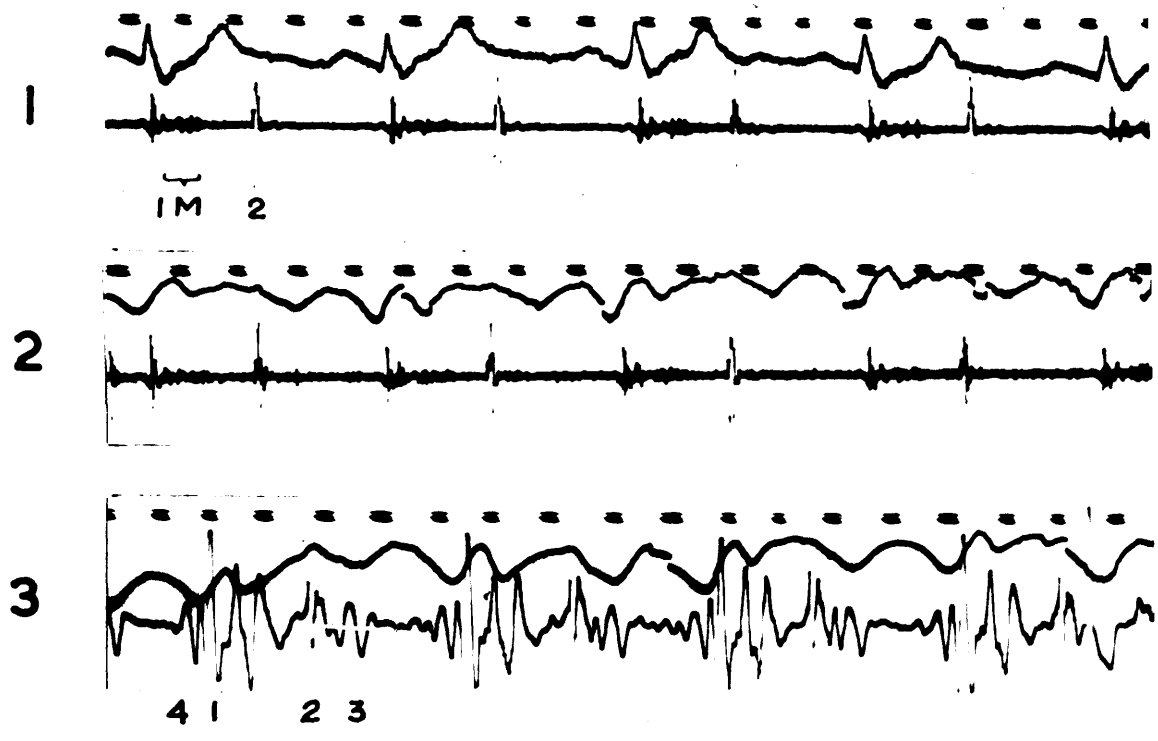
## Patient No. 83 - male, age 58

Diagnosis
Arterial hypertension
Myocardial disease
Left heart strain

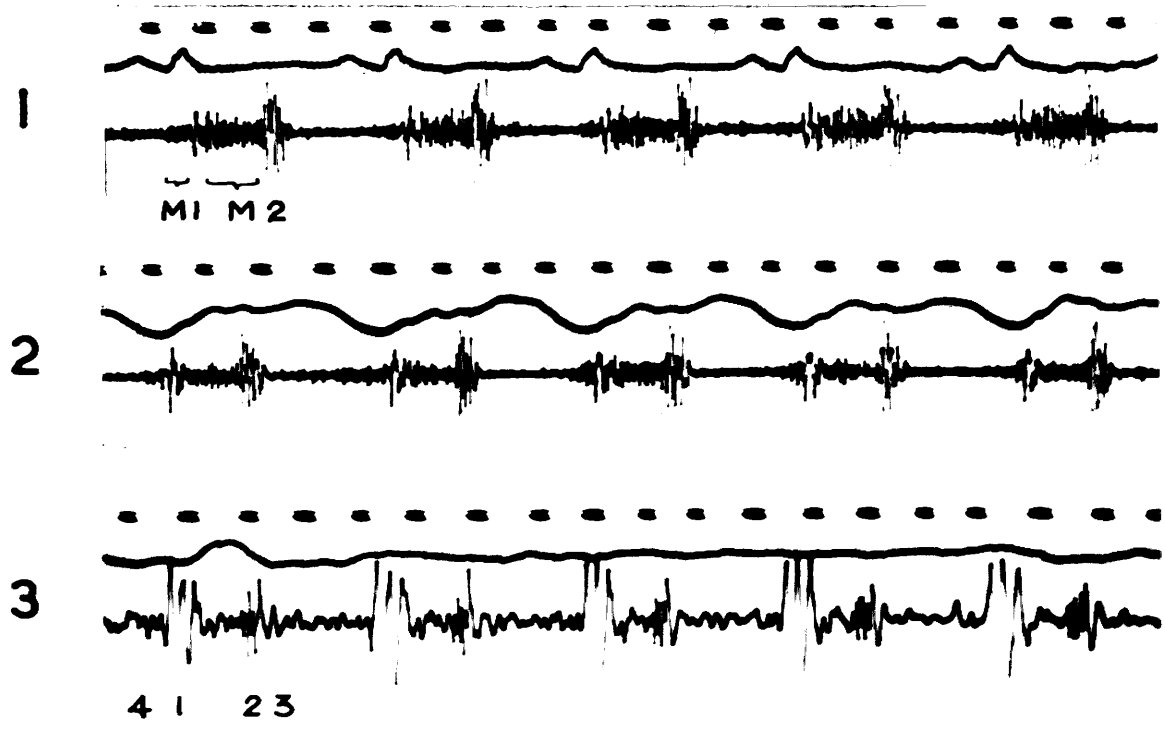
Auscultation
1st and 2nd heart sounds; early and mid-systolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; pansystolic and late diastolic murmurs.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 82 APEX




PATIENT NO 83 APEX

## PLATE 63

### Patient No. 84 - male, age 42

Diagnosis
Arterial hypertension
Myocardial disease
Angina pectoris

Auscultation
1st and 2nd heart sounds; no murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; no murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.

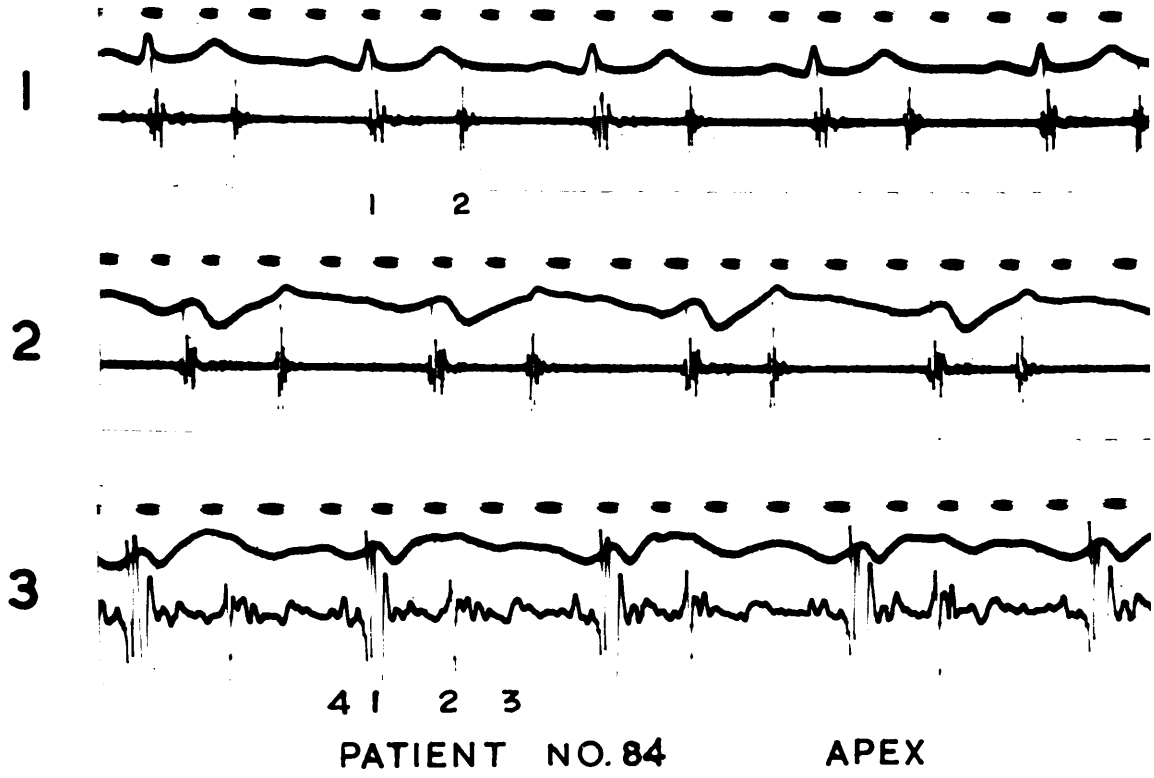
### Patient No. 85 - female, age 63

Diagnosis
Arterial hypertension
Aortic stenosis

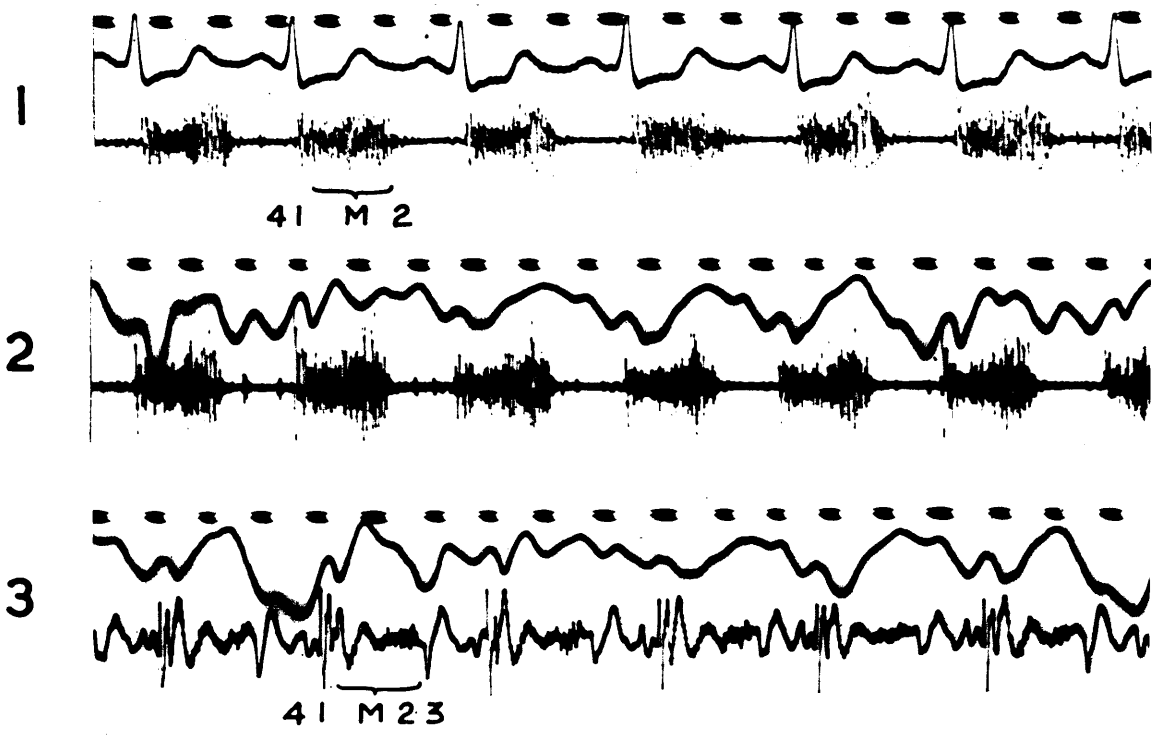
Auscultation
1st and 2nd heart sounds; pansystolic murmur.

Logarithmic P.C.G.
1st, 2nd, and 4th heart sounds (presystolic gallop); pansystolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds; indication of systolic murmur.




PATIENT NO. 84 APEX




PATIENT NO. 85 APEX

# PLATE 64

## Patient No. 86 - male, age 29

Diagnosis
Mitral stenosis
Aortic incompetence
Left heart strain
Subacute bacterial endocarditis

Auscultation
1st and 2nd heart sounds; pansystolic and mid- and late diastolic murmurs.

Logarithmic P.C.G.
1st, 2nd, and 3rd heart sounds (protodiastolic gallop); early and mid-systolic and pandiastolic murmurs.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.

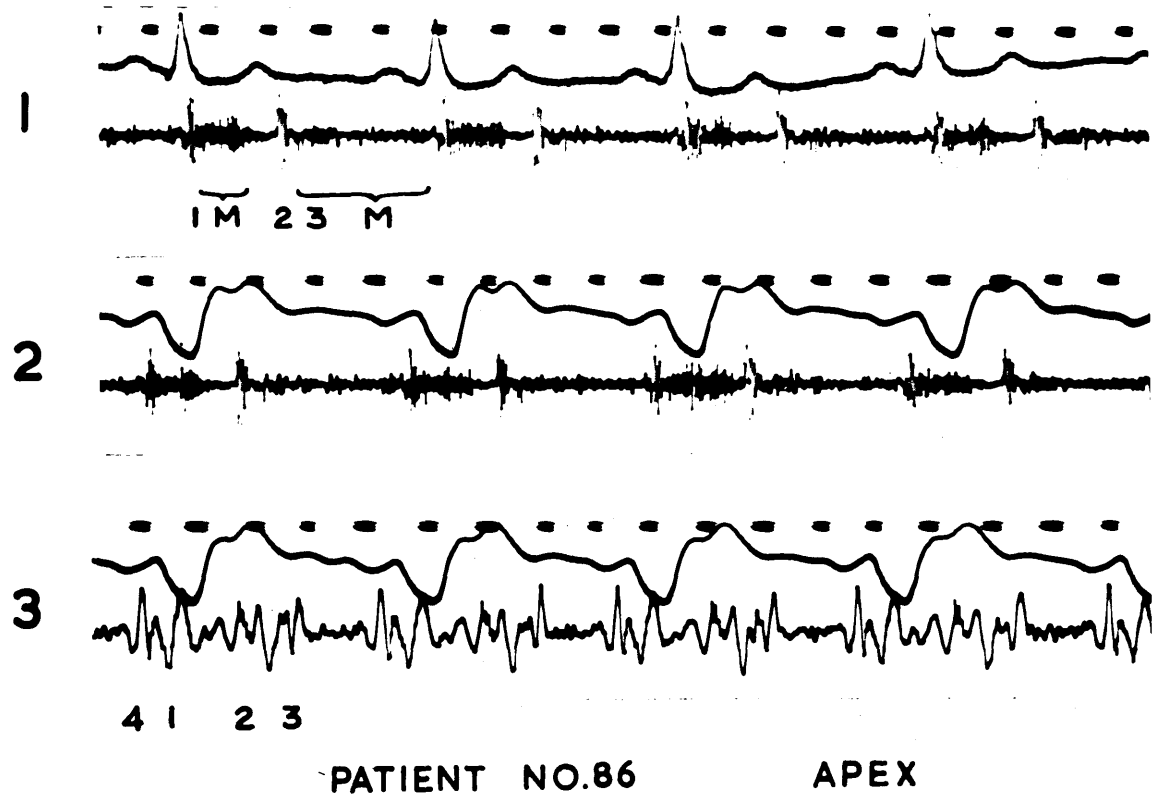
## Patient No. 87 - female, age 57

Diagnosis
Arterial hypertension Malignant hypertension
Myocardial disease
Congestive cardiac failure

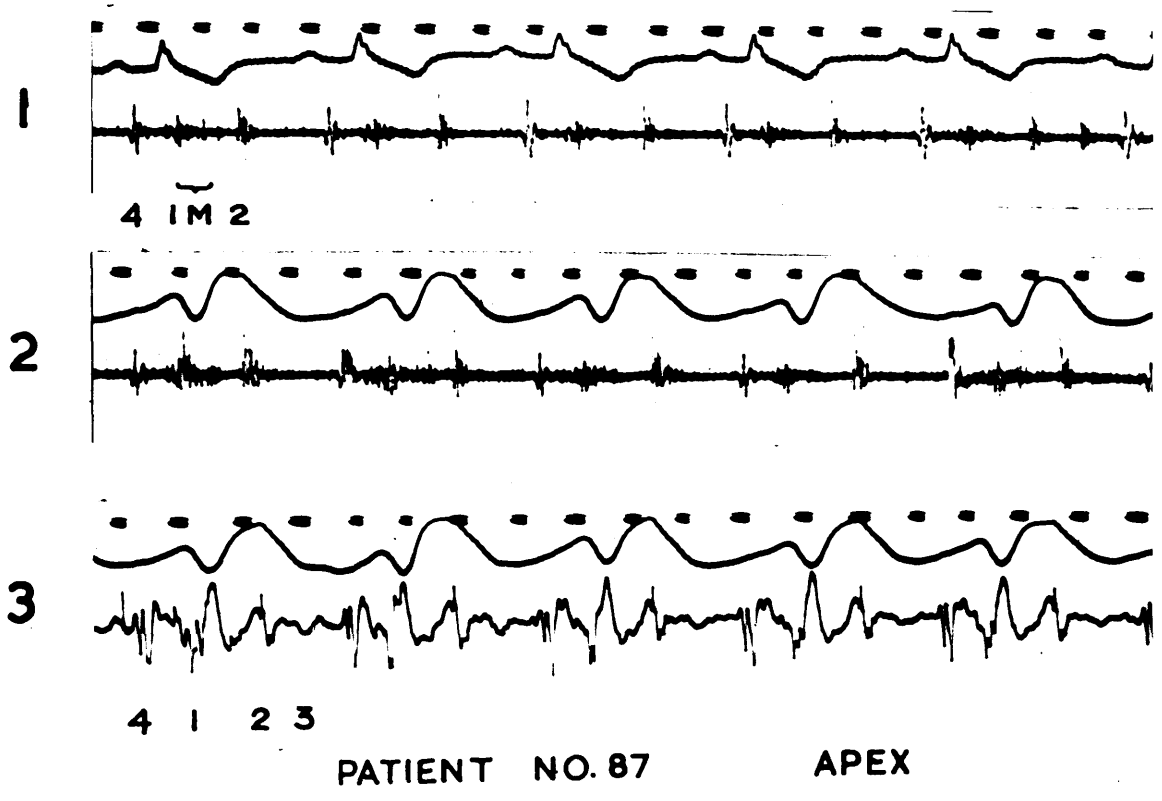
Auscultation
1st and 2nd heart sounds; early systolic murmur.

Logarithmic P.C.G.
1st, 2nd, and 4th heart sounds (presystolic gallop); early systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO.86 APEX




PATIENT NO. 87 APEX

# PLATE 65

## Patient No. 88 - male, age 55

Diagnosis

Mitral stenosis and incompetence
Aortic incompetence
Left bundle branch block

Auscultation

1st and 2nd heart sounds; pansystolic and mid- and late diastolic murmurs.

Logarithmic P.C.G.

1st, 2nd, 3rd, and 4th heart sounds (quadruple rhythm) with wide splitting of 1st sound; pansystolic and early diastolic murmurs.

Linear P.C.G.

Vibrations at the time of both components of 1st sound, and at the time of 2nd, 3rd, and 4th heart sounds.

## Patient No. 89 - male, age 66

Diagnosis

Arterial hypertension
Left heart strain

Auscultation

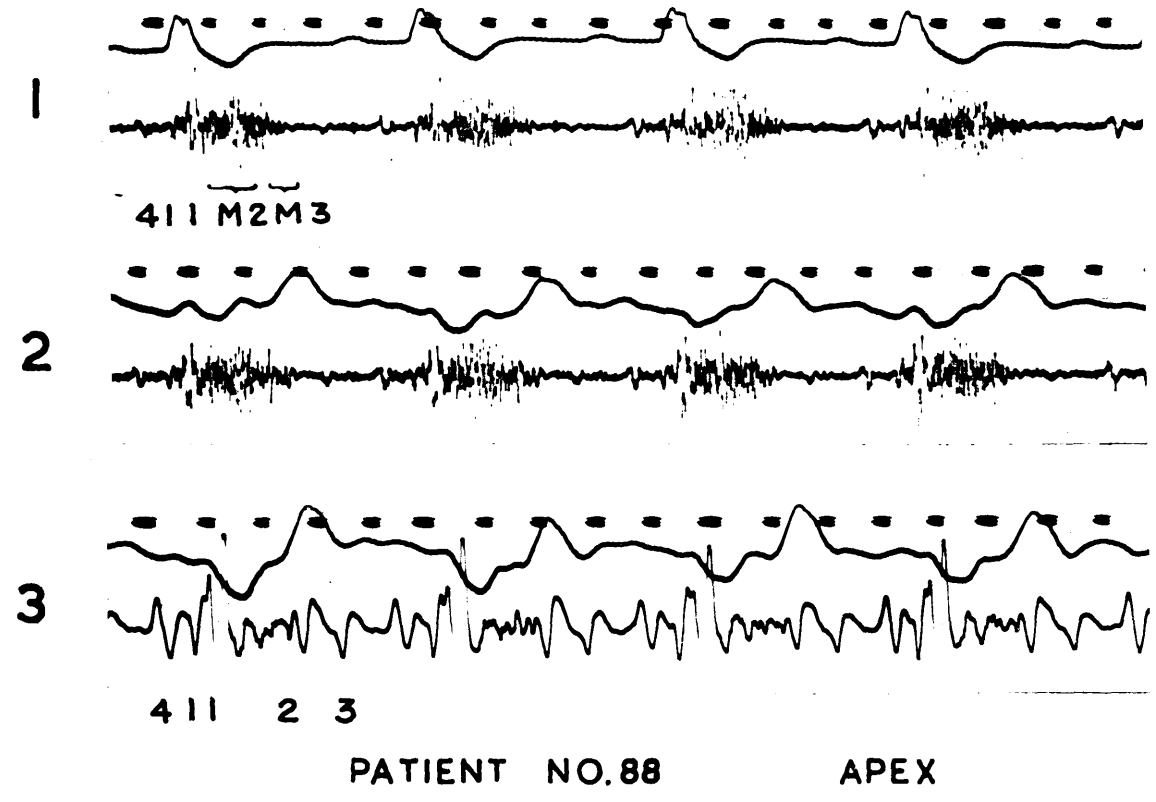
1st and 2nd heart sounds; pansystolic murmur.

Logarithmic P.C.G.

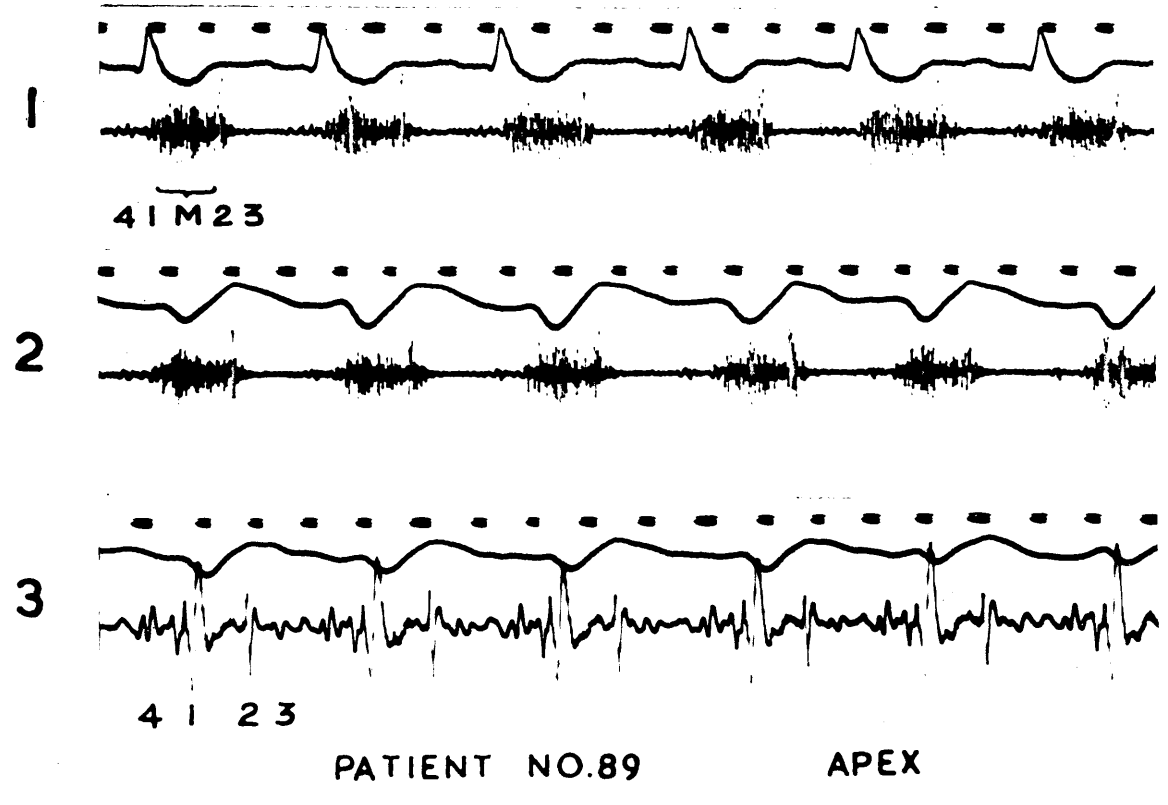
1st, 2nd, 3rd, and 4th heart sounds (quadruple rhythm); pansystolic murmur.

Linear P.C.G.

Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 88 APEX




PATIENT NO. 89 APEX

## PLATE 66

### Patient No. 90 - female, age 29

**Diagnosis**

Mitral stenosis
Auricular fibrillation
Cardiac failure

**Auscultation**

1st, 2nd, and 3rd heart sounds (protodiastolic gallop); pansystolic murmur.

**Logarithmic P.C.G.**

1st, 2nd, and 3rd heart sounds (protodiastolic gallop); pansystolic and early and mid-diastolic murmurs.

**Linear P.C.G.**

Vibrations at the time of 1st, 2nd, and 3rd heart sounds.

### Patient No. 91 - female, age 70

**Diagnosis**

Arterial hypertension — Kimmelstiel-Wilson syndrome
Congestive cardiac failure

**Auscultation**

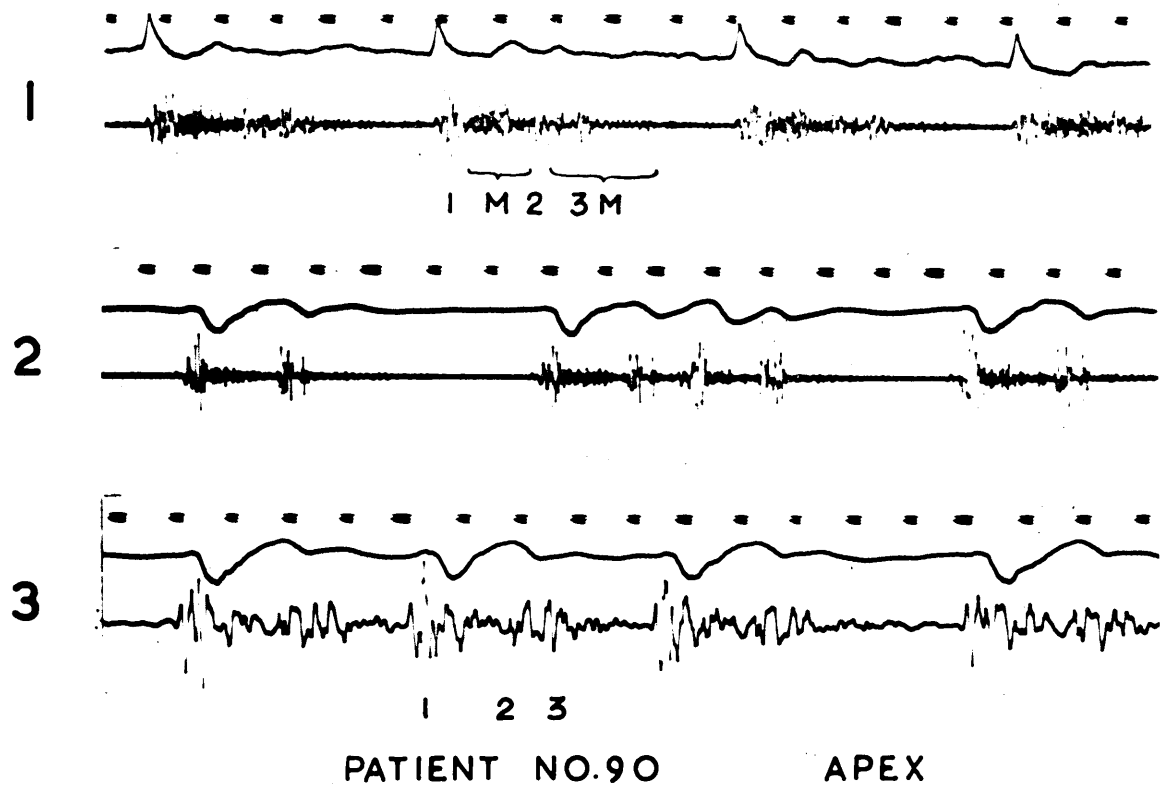
Soft 1st and 2nd heart sounds; no murmur.

**Logarithmic P.C.G.**

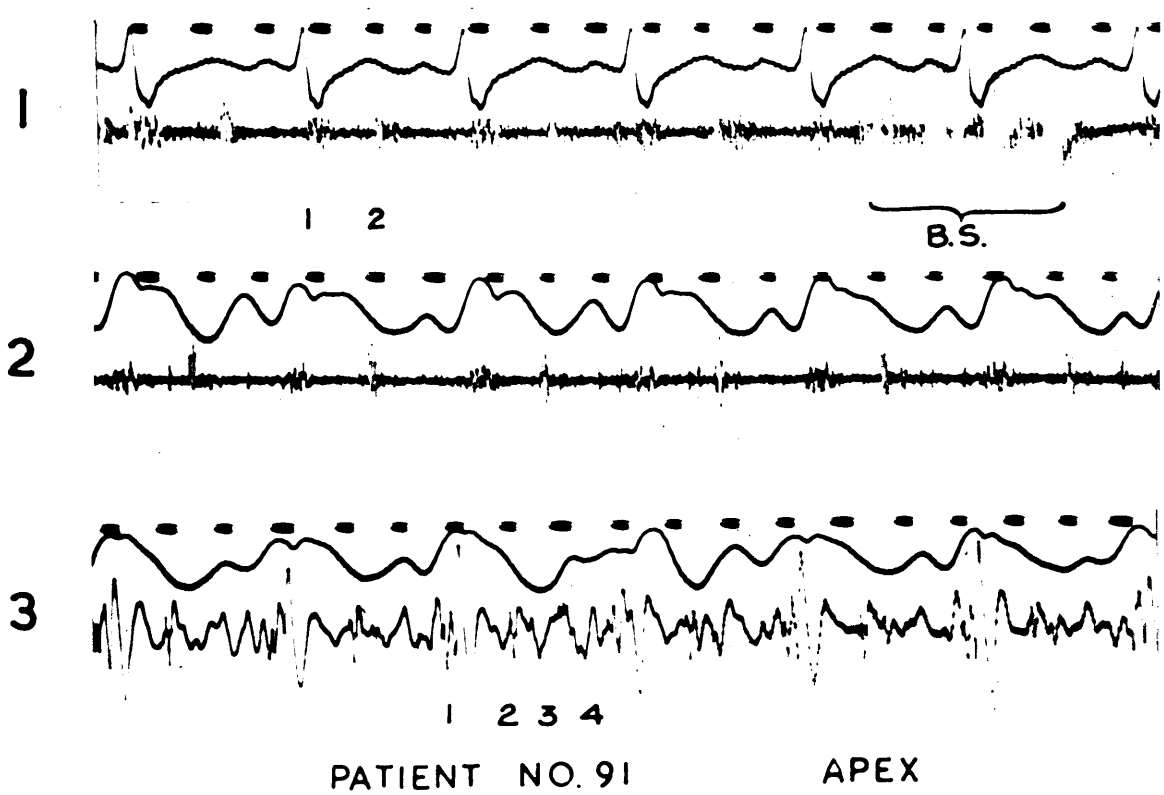
1st and 2nd heart sounds; no murmur.
Breath sound (BS) on record 1.

**Linear P.C.G.**

Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO.90 APEX




PATIENT NO. 91 APEX

# PLATE 67

## Patient No. 92 - female, age 45

Diagnosis
Arterial hypertension  Malignant hypertension
Left heart strain

Auscultation
1st and 2nd heart sounds; pansystolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; early and mid-systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 4th heart sounds; indication of systolic murmur.

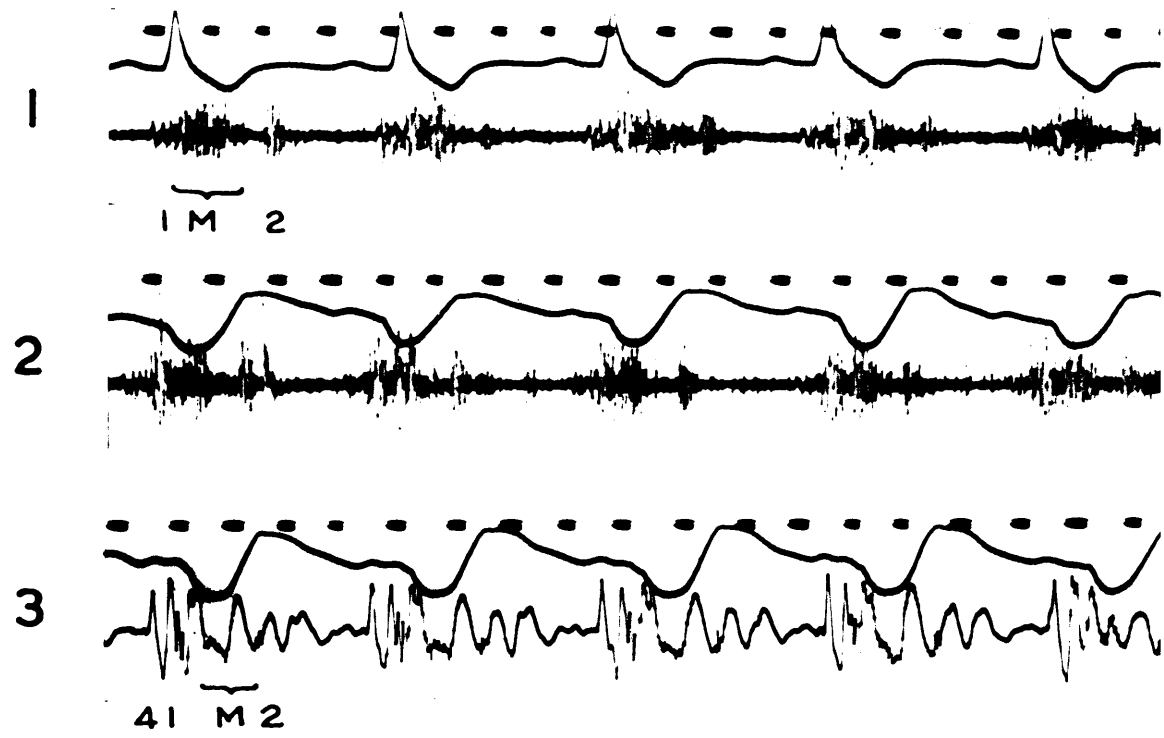
## Patient No. 93 - male, age 62

Diagnosis
Arterial hypertension  Cerebral vascular disease
Left heart strain

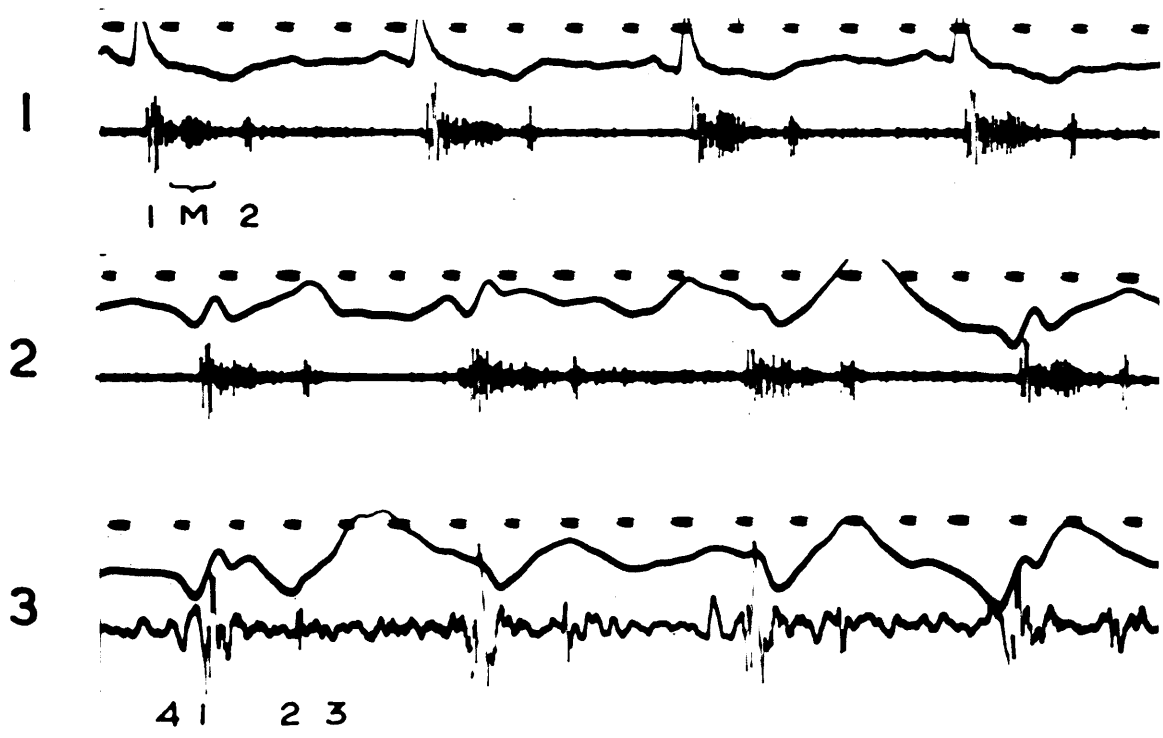
Auscultation
1st and 2nd heart sounds; early systolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; early and mid-systolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 92 APEX




PATIENT NO. 93 APEX

## PLATE 68

### Patient No. 94 - female, age 64

**Diagnosis**

Arterial hypertension
Left heart strain
Auricular fibrillation

Chronic bronchitis

**Auscultation**

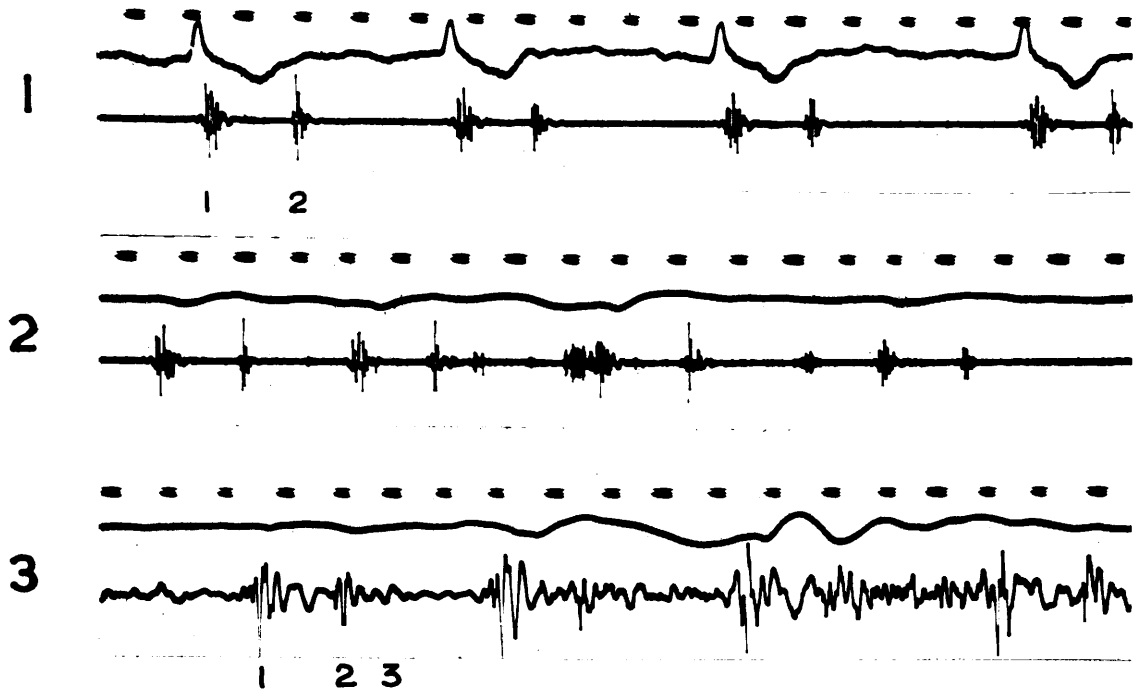
1st and 2nd heart sounds; no murmur.

**Logarithmic P.C.G.**

1st and 2nd heart sounds; no murmur.

**Linear P.C.G.**

Vibrations at the time of 1st, 2nd, and 3rd heart sounds.




PATIENT NO. 94 APEX

# PLATE 69

## Patient No. 95 - female, age 32

Diagnosis

Mitral stenosis
Aortic incompetence
Left heart strain

Auscultation

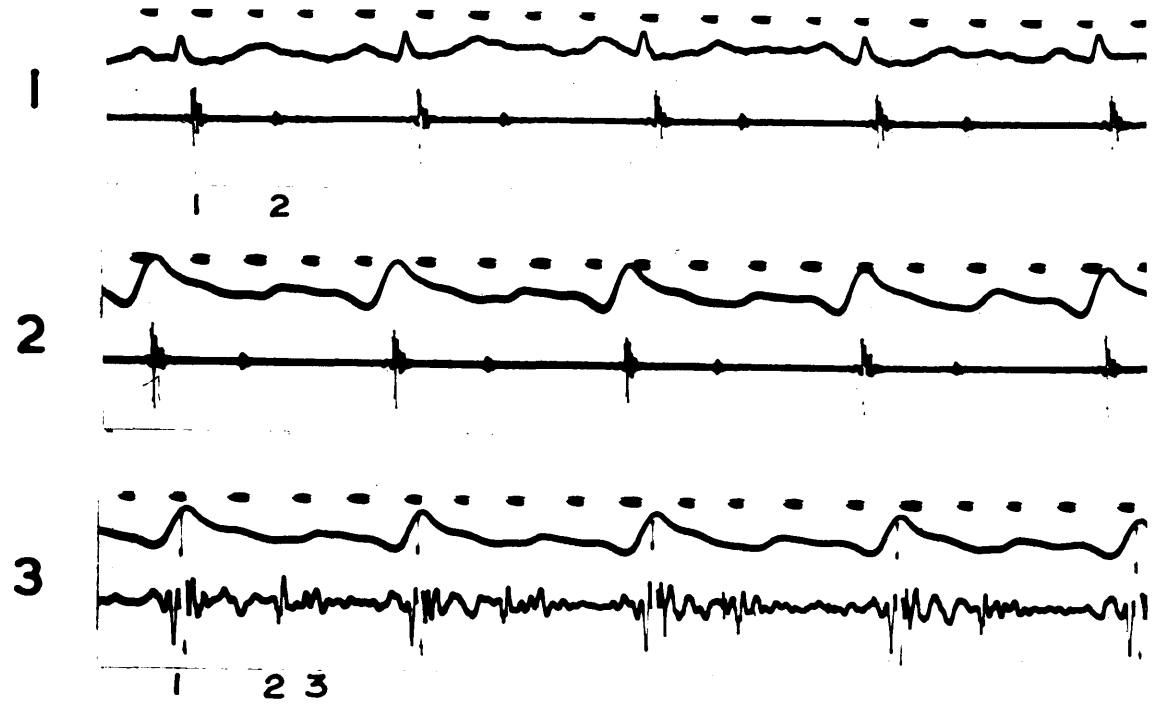
1st and 2nd heart sounds; pansystolic and early and late diastolic murmurs. At sternal end of 3rd left intercostal space, 1st and 2nd heart sounds; early and mid-systolic and early diastolic murmurs.

Logarithmic P.C.G.

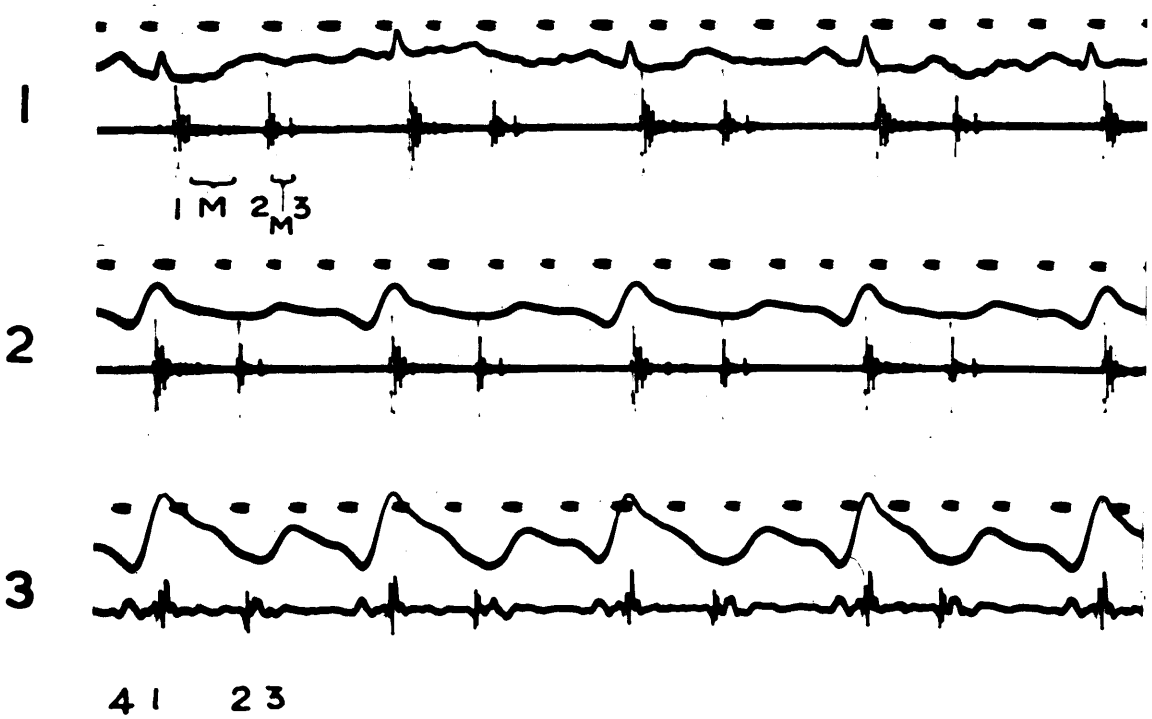
1st and 2nd heart sounds; no murmur. At sternal end of 3rd left intercostal space, 1st, 2nd, and 3rd heart sounds (protodiastolic gallop); early and mid-systolic and early diastolic murmurs.

Linear P.C.G.

Vibrations at the time of 1st, 2nd, and 3rd heart sounds. At sternal end of 3rd left intercostal space, vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.




PATIENT NO. 95 APEX




PATIENT NO. 95 3rd L.I.S.

## PLATE 70

### Patient No. 96 - female, age 53

Diagnosis
Arterial hypertension Bronchial asthma
Left heart strain

Auscultation
Faint 1st and 2nd heart sounds; early systolic murmur.

Logarithmic P.C.G.
1st and 2nd heart sounds; pansystolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.

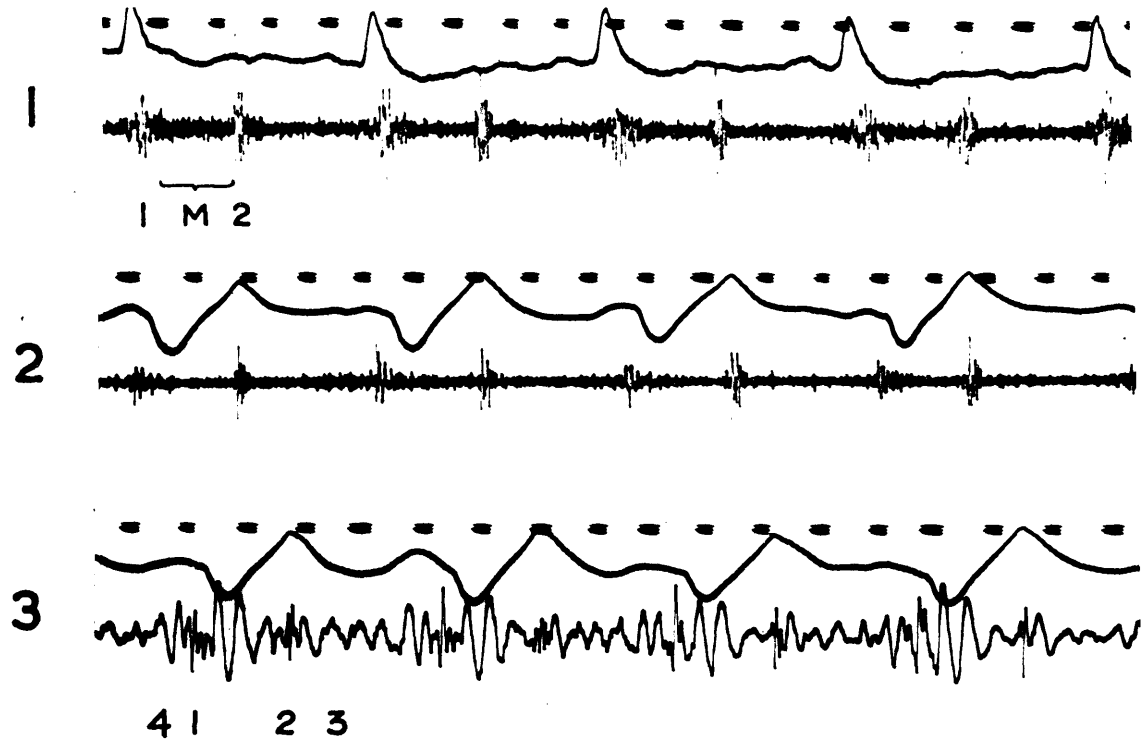
### Patient No. 97 - female, age 23

Diagnosis
Mitral stenosis

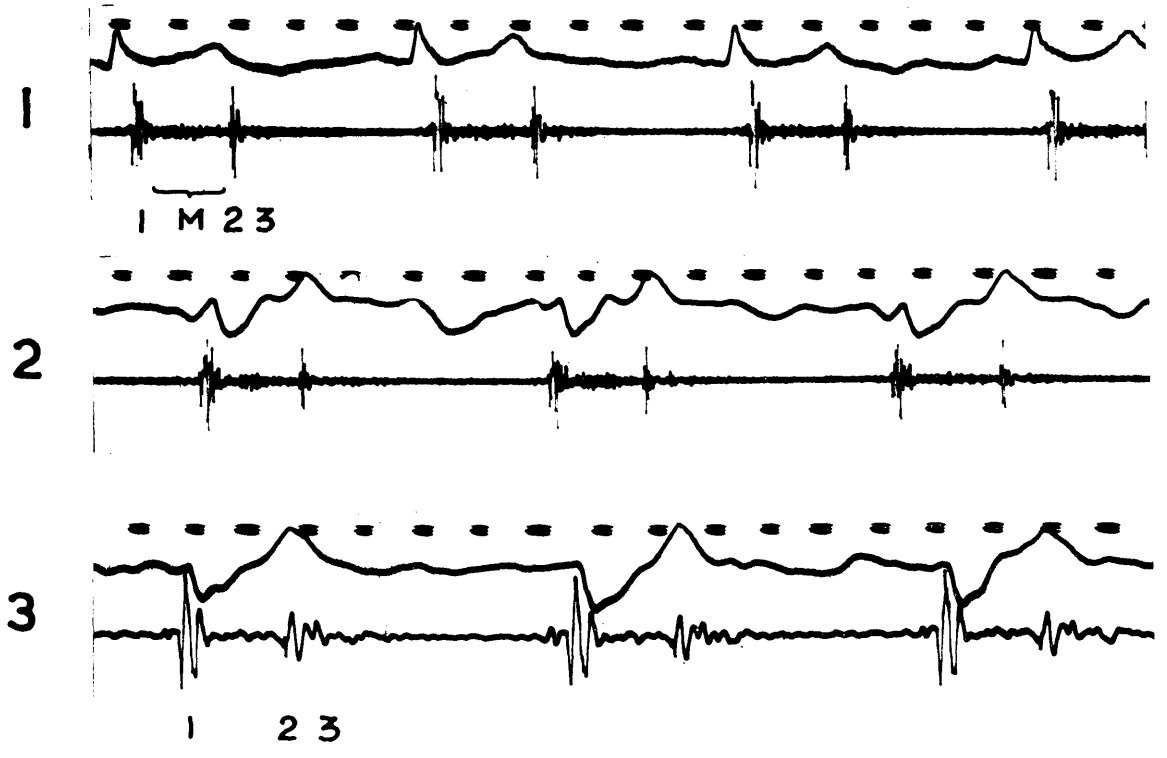
Auscultation
1st and 2nd heart sounds; pansystolic and mid-diastolic murmurs.

Logarithmic P.C.G.
1st, 2nd, and 3rd heart sounds (protodiastolic gallop); pansystolic murmur.

Linear P.C.G.
Vibrations at the time of 1st, 2nd, and 3rd heart sounds.




PATIENT NO. 96 APEX




PATIENT NO. 97 APEX

## PLATE 71

### Patient No. 98 - female, age 17

**Diagnosis**

Mitral incompetence
Subacute bacterial endocarditis
Iron-deficiency anaemia

**Auscultation**

1st and 2nd heart sounds; pansystolic murmur.

**Logarithmic P.C.G.**

1st, 2nd, and 3rd heart sounds (protodiastolic gallop); pansystolic murmur.

**Linear P.C.G.**

Vibrations at the time of 1st, 2nd, and 3rd heart sounds.

### Patient No. 99 - male, age 23

**Diagnosis**

Aortic incompetence
Myocardial disease
Left heart strain

**Auscultation**

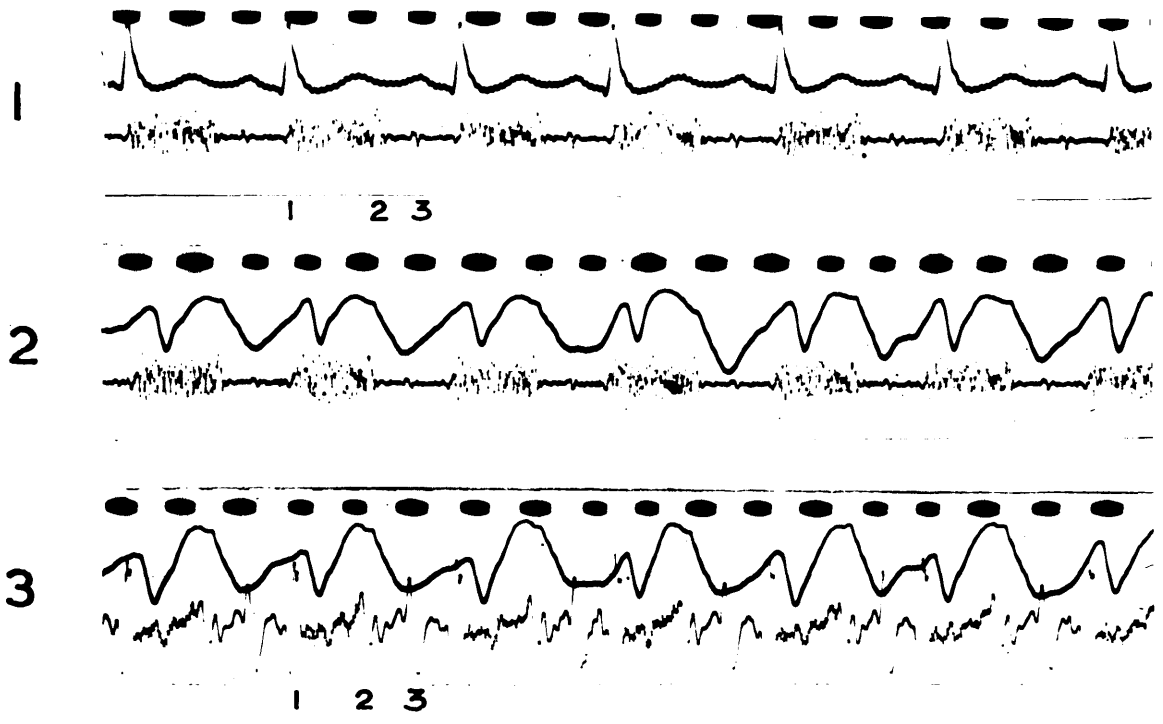
1st and 2nd heart sounds; continuous murmur, with systolic accentuation.

**Logarithmic P.C.G.**

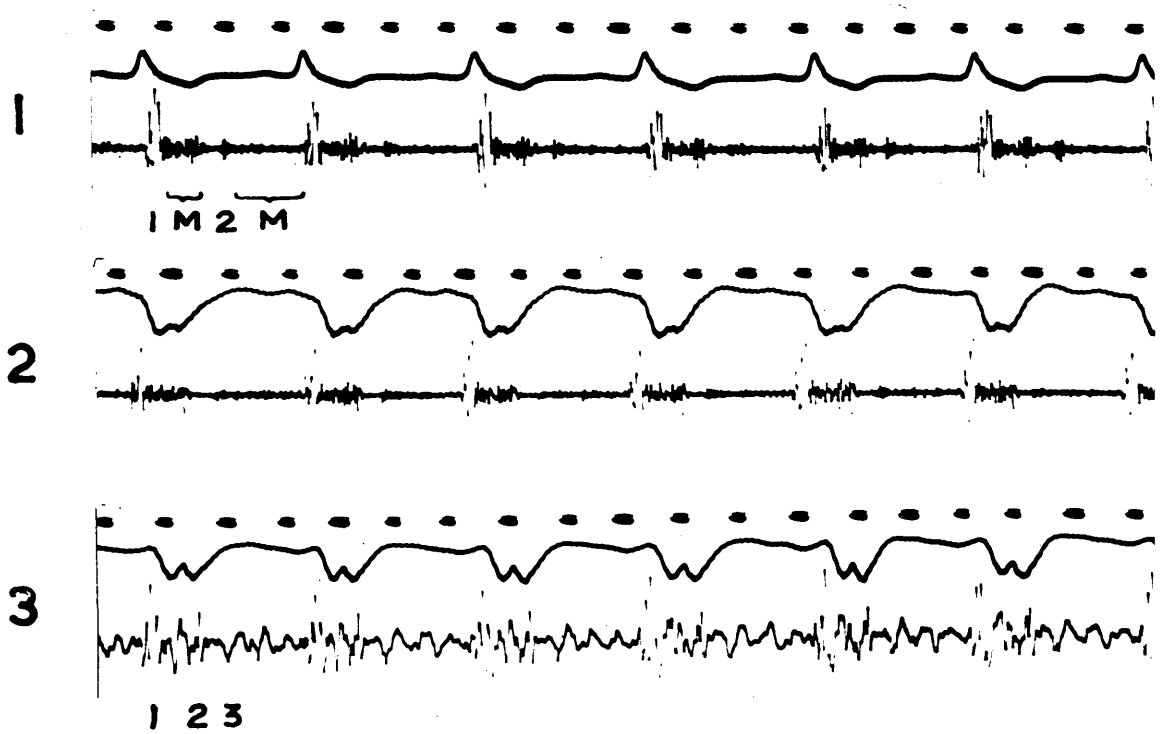
1st and 2nd heart sounds; early and mid-systolic and pandiastolic murmurs.

**Linear P.C.G.**

Vibrations at the time of 1st, 2nd, and 3rd heart sounds.




PATIENT NO. 98 APEX




PATIENT NO. 99 APEX

# PLATE 72

## Patient No. 100 - male, age 62

**Diagnosis**

Aortic incompetence
Cardiac failure

**Auscultation**

1st and 2nd heart sounds; pansystolic murmur.

**Logarithmic P.C.G.**

1st and 2nd heart sounds; early and mid-systolic murmur.

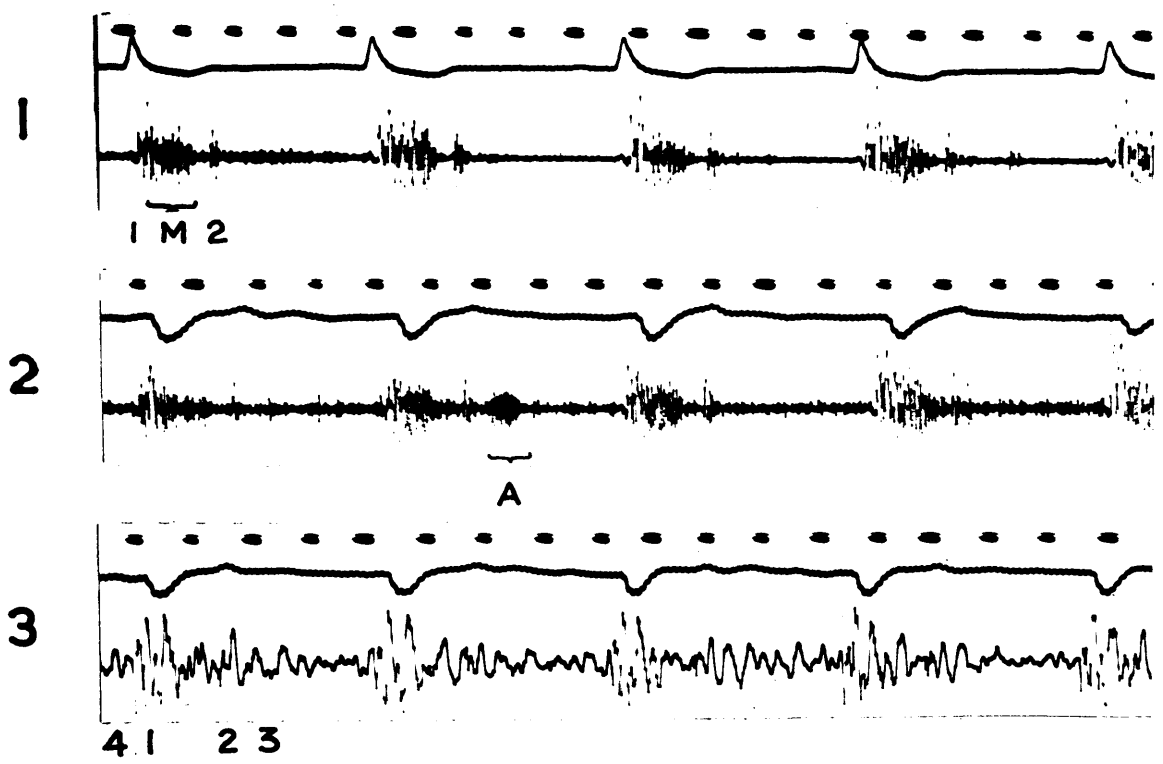
**Linear P.C.G.**

Vibrations at the time of 1st, 2nd, 3rd, and 4th heart sounds.

**Note**

No aortic diastolic murmur heard at any point on praecordium.

Artefact (A) on record 2.




PATIENT NO. 100 APEX

## APPENDIX C (continued)

## PLATES 73 - 79

### EXPERIMENTAL PHONOCARDIOGRAMS

| | |
|---|---|
| Plate 73 | Human experiment |
| Plates 74 - 78 | Dog experiments |
| Plate 79 | Calibrated human record |

# PLATE 73

Experimental subject No. 1 - female, age 20.

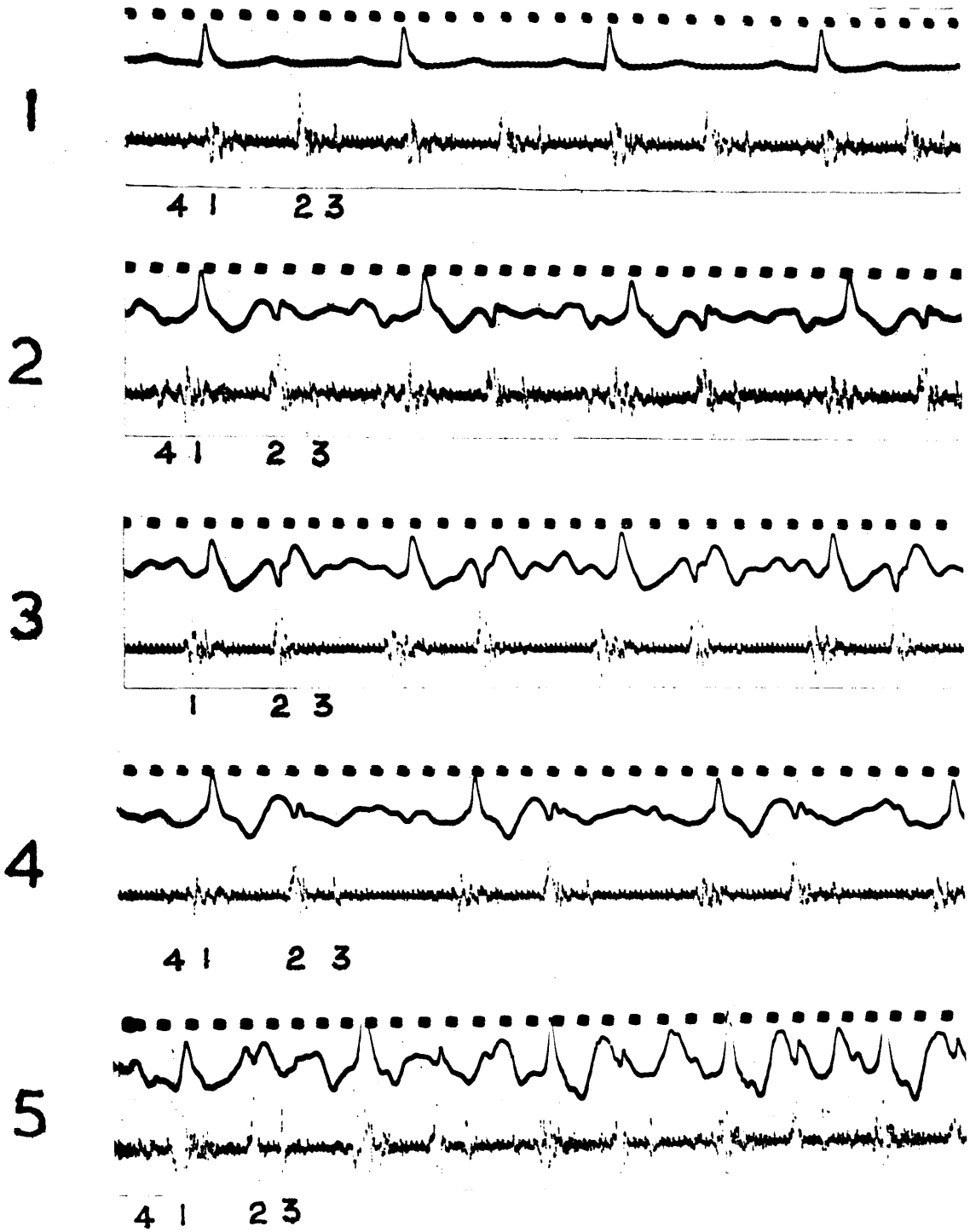
Logarithmic P.C.G.s from the mitral area.

Reference tracing in record 1 = E.C.G. (Lead II).
Reference tracing in records 2 - 5 = jugular phlebogram

1. Preliminary control
2. " "
3. With venous occlusion
4. Control after venous occlusion
5. Immediately after exercise

Time marking in 1/10 seconds

The records shown here are cut from the original (longer) records




EXPERIMENTAL SUBJECT NO.1

# PLATE 74

## Dog No. 11 - Collie - female, 11.3 kg.

Preliminary P.C.G.s

1. E.C.G. (Lead II)
   Logarithmic P.C.G. from left chest wall

2. E.C.G. (Lead II)
   Stethoscopic P.C.G. from left chest wall

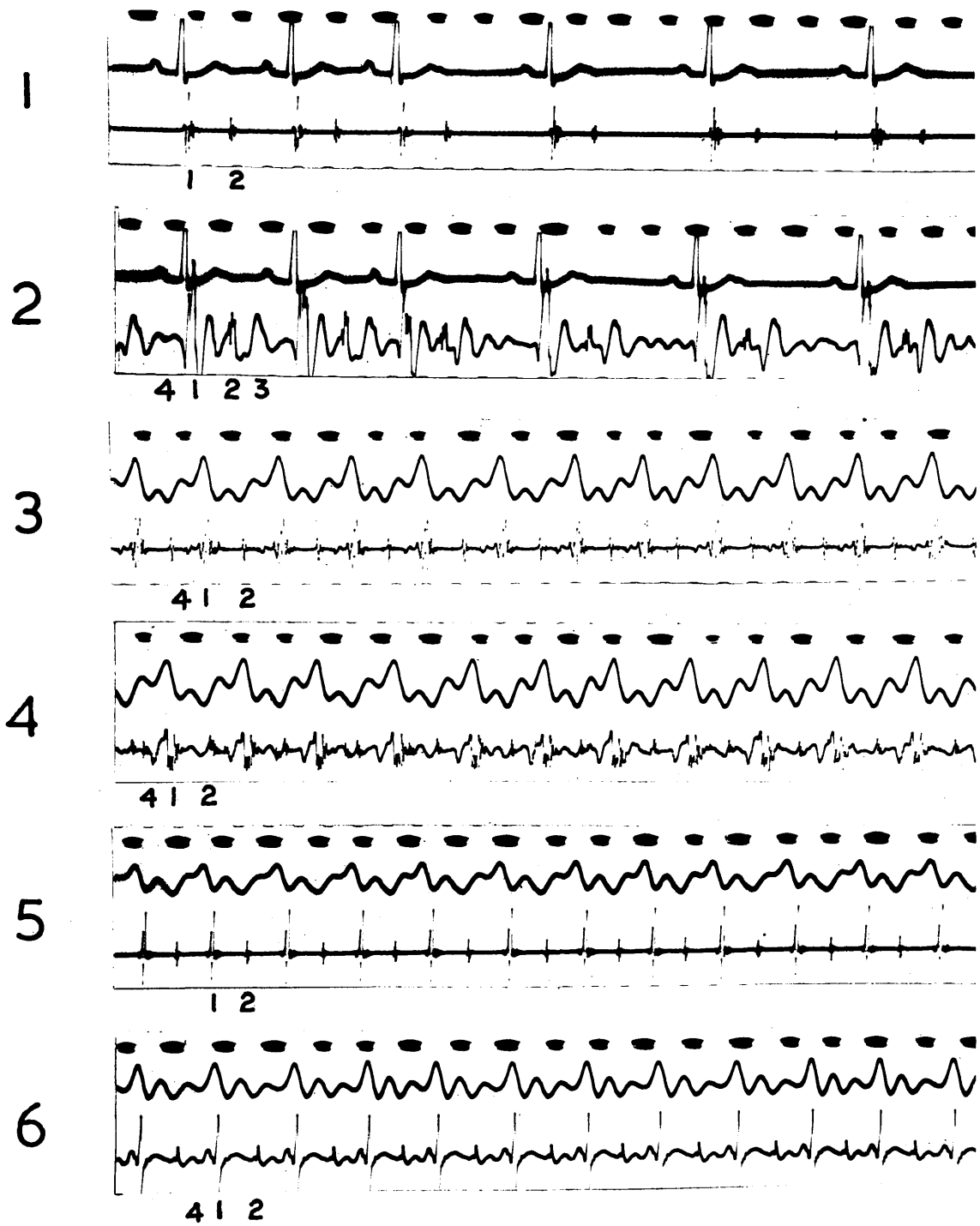
3. Jugular phlebogram
   Logarithmic P.C.G. from exposed right ventricle

4. Jugular phlebogram
   Stethoscopic P.C.G. from exposed right ventricle

5. Jugular phlebogram
   Logarithmic P.C.G. from exposed left ventricle

6. Jugular phlebogram
   Stethoscopic P.C.G. from exposed left ventricle

Time marking in 1/5 seconds




DOG NO. 11

# PLATE 75

Dog No. 3 - Labrador puppy - male, 12.1 kg.

Logarithmic P.C.G.s from the exposed right ventricle

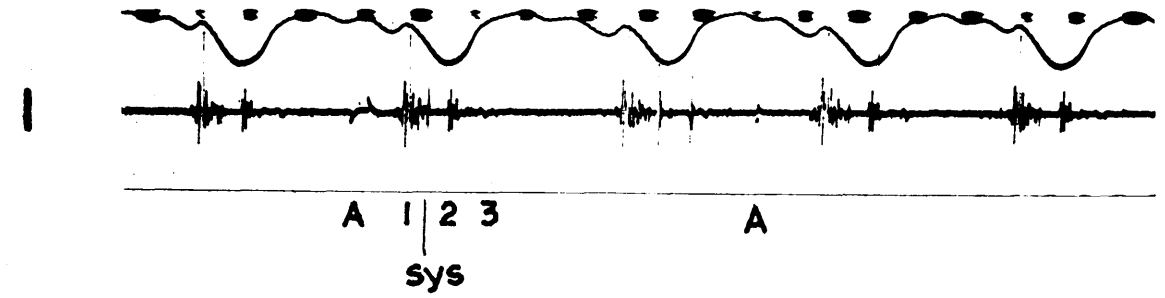
Reference tracings - No. 1 - jugular phlebogram
Nos. 2, 3, 4 - E.C.G. (Lead II)

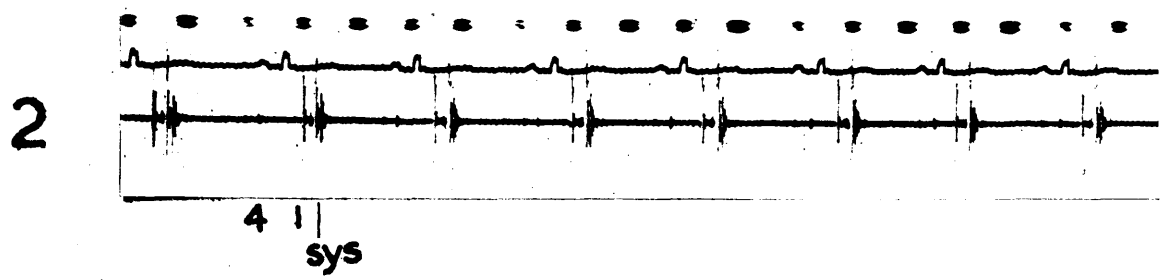
1. First control
2. During venous occlusion
3. Second control
4. During rapid intravenous infusion

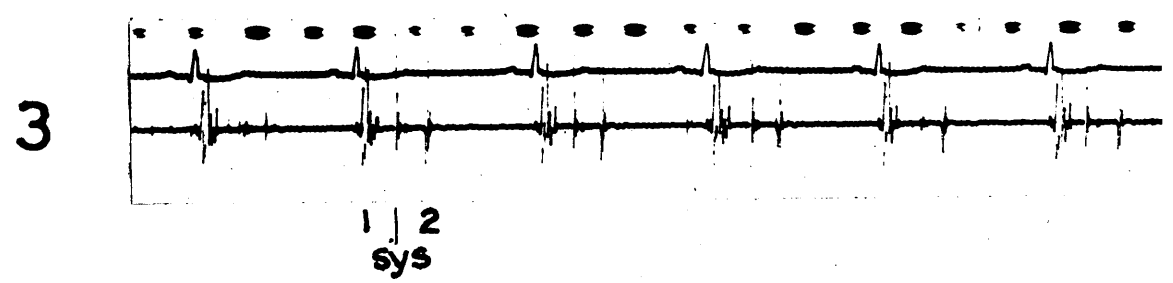
Note

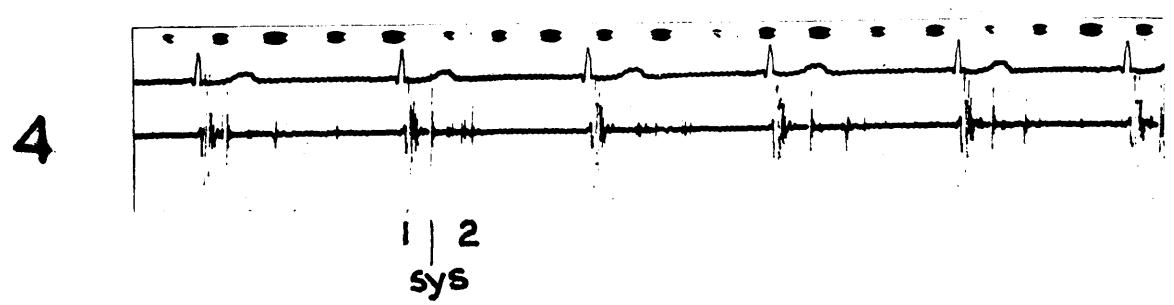
Artefacts, 'A', in record 1

Time marking in 1/5 seconds




DOG NO. 3

# PLATE 76

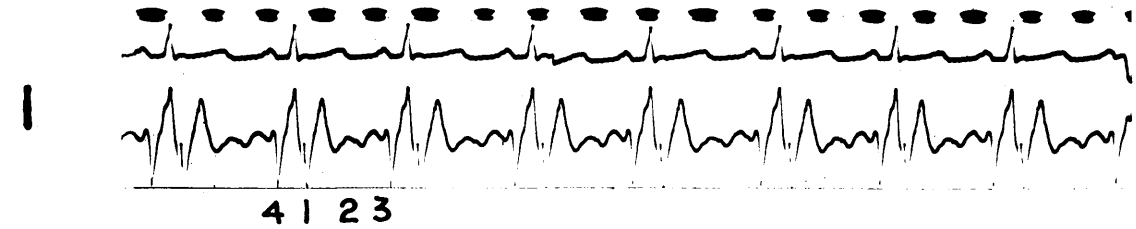
Dog No. 12 - Labrador puppy - female, 10.0 kg.

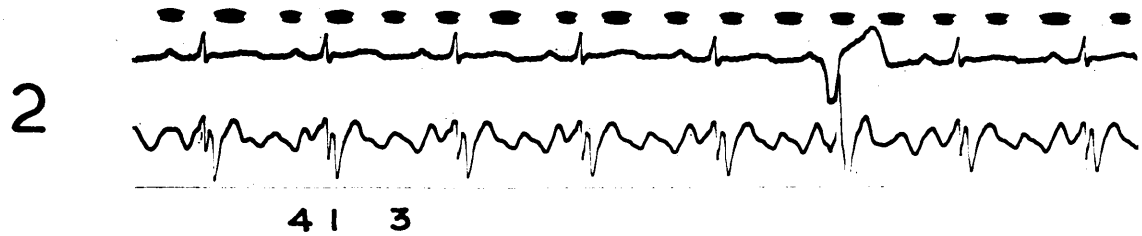
Stethoscopic P.C.G. s from exposed right ventricle

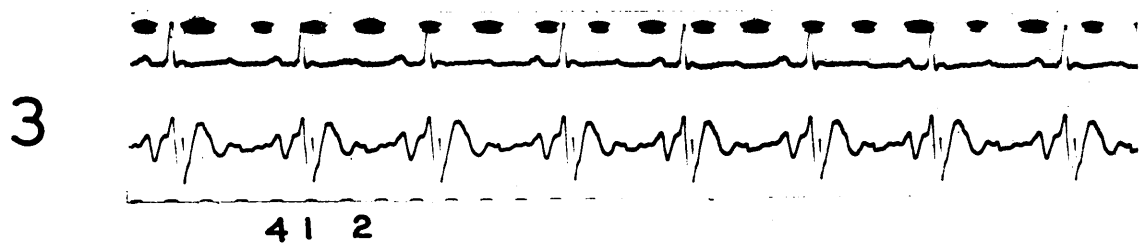
Reference tracings - E.C.G. (Lead II)

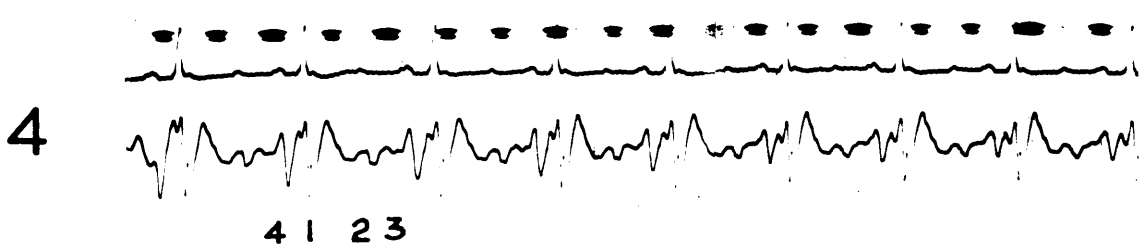
1. First control
2. During venous occlusion
3. Second control
4. During rapid intravenous infusion

Time marking in 1/5 seconds




DOG NO. 12

# PLATE 77

## Dog No. 1 - Collie - male, 10.3 kg.

Logarithmic P.C.G.s from the exposed right ventricle

1. E.C.G. (Lead II)
   Control

2. Jugular phlebogram
   P.C.G. immediately after administration of digoxin (slow intravenous injection of 1 mg. over a period of 4 minutes)

Time marking in 1/5 seconds

## Dog No. 12 - Labrador puppy - female, 10.0 kg.

Logarithmic P.C.Gs from the exposed right ventricle
Reference tracings - jugular phlebogram

1. First control

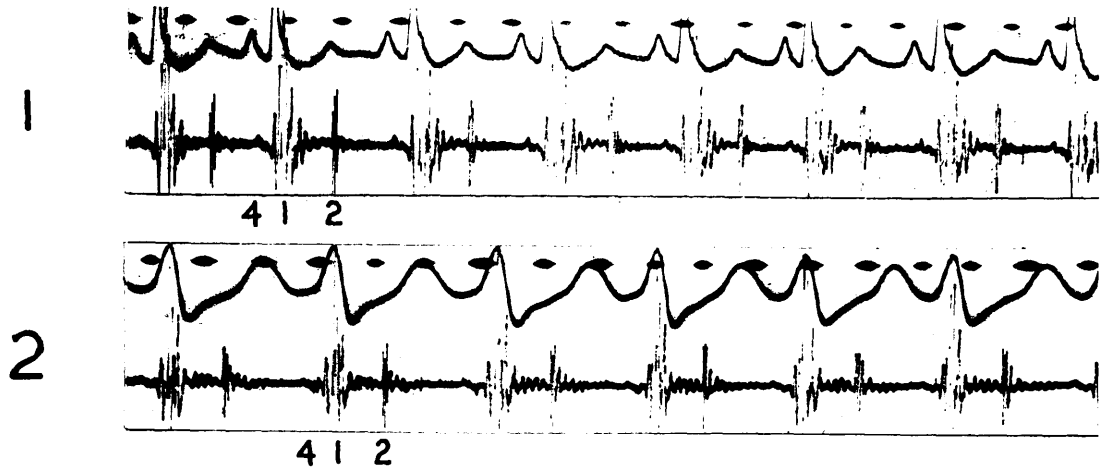
2. During stimulation of peripheral end of divided right vagus nerve (10 millisecond impulses at 3 volts and 50 impulses per second)

3. Second control

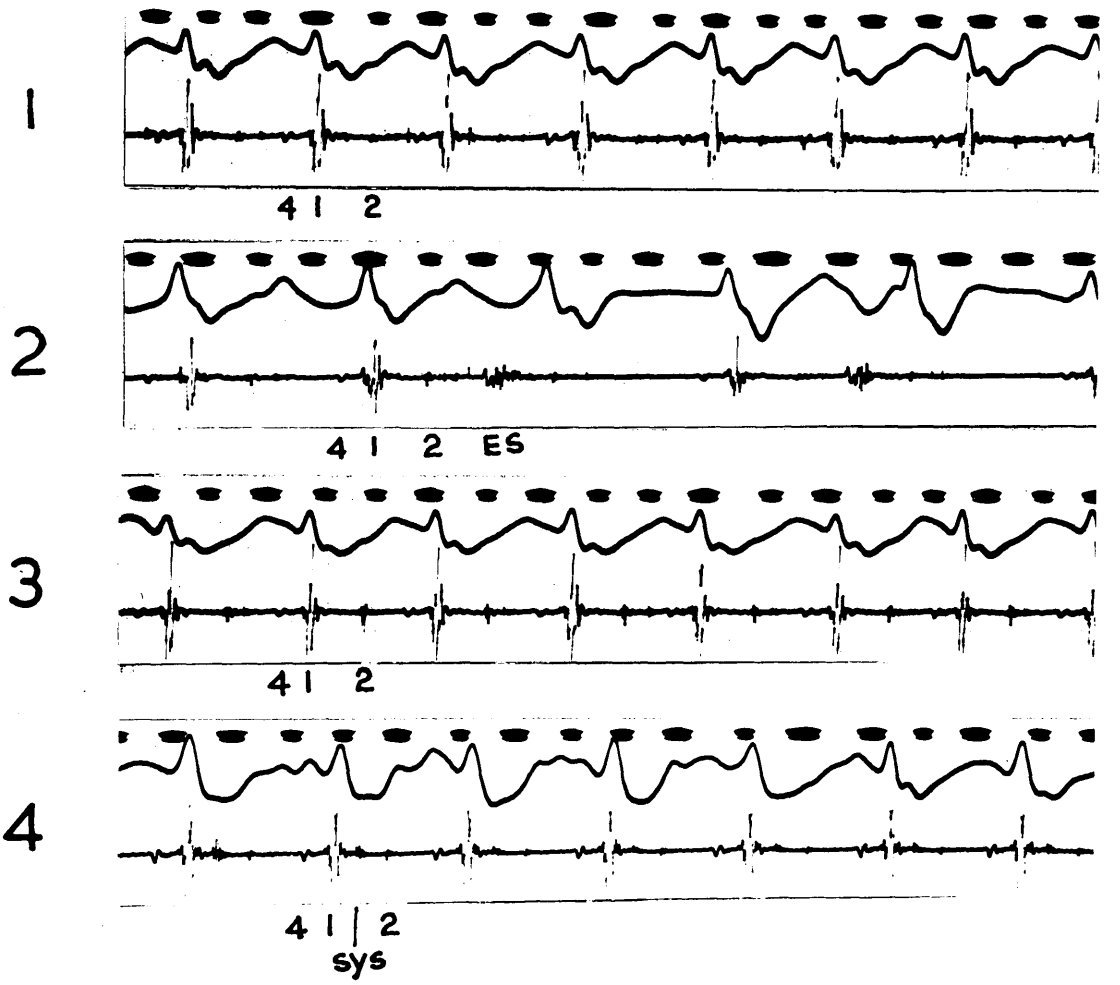
4. P.C.G. 10 minutes after administration of physostigmine (slow intravenous injection of 1.5 mg. over a period of 5 minutes)

Note
Extra systole (ES) in record 2

Time marking in 1/5 seconds




DOG NO. 1




DOG NO. 12

# PLATE 78

Dog No. 1 (2nd series) - Collie puppy - female, 6.5 kg.

1. Stethoscopic P.C.G. from exposed right ventricle
   Reference tracing - jugular phlebogram
   Vagal arrest of heart; between arrows, 9 ml. saline injected through cardiac catheter into right ventricle, causing extra systole (ES).

Dog No. 2 (2nd series) - Alsatian - female, 20.0 kg.

1. Stethoscopic P.C.G. from exposed right ventricle
   Reference tracing - jugular phlebogram
   Vagal arrest of heart; three taps (at arrows), producing two extra systoles (ES).

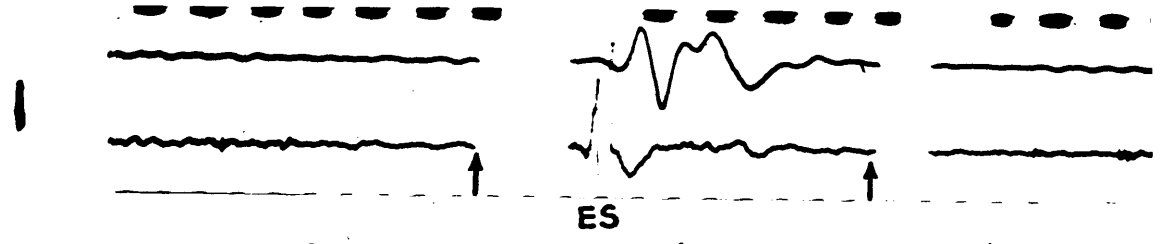
2. Logarithmic P.C.G. from exposed right ventricle
   Reference tracing - jugular phlebogram
   Vagal arrest of heart; between arrows, 14 ml. saline injected through cardiac catheter into right ventricle, causing murmur (M).

Dog No. 5 (2nd series) - Collie - male, 24.3 kg.

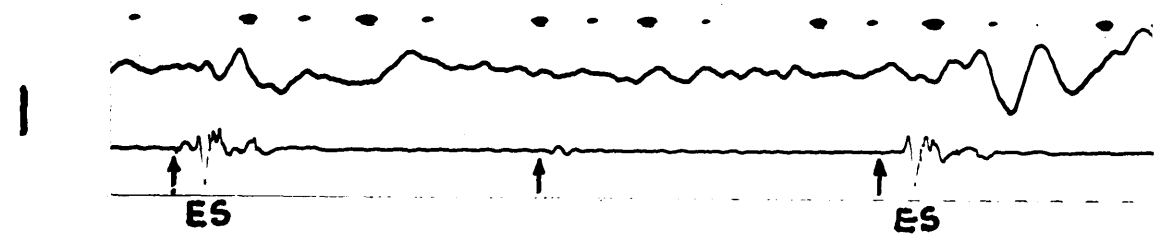
1. Logarithmic post-mortem record from exposed right ventricle
   Right ventricle tapped (at arrows), causing sound vibrations like heart sound.
   The calibration signal (CAL) at end of record corresponds to a sound signal of 6 dynes per sq. cm. at 50 cycles per second applied to the microphone.

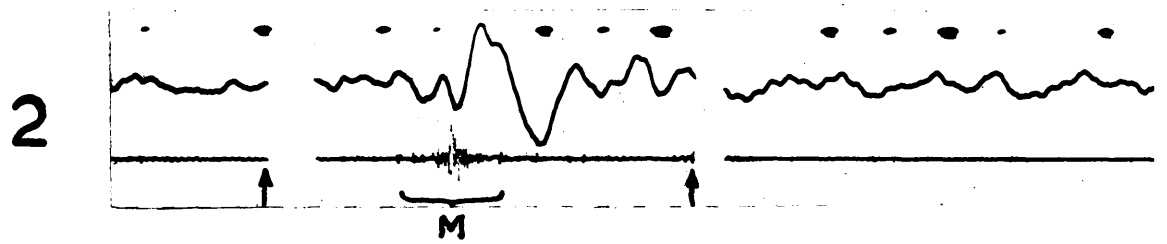
2. Logarithmic post-mortem record from exposed right ventricle
   10 ml. saline injected through cardiac catheter against deflector in right ventricle, causing murmur (M).
   Calibration signal (CAL) as before.

Time marking in 1/5 seconds in all records

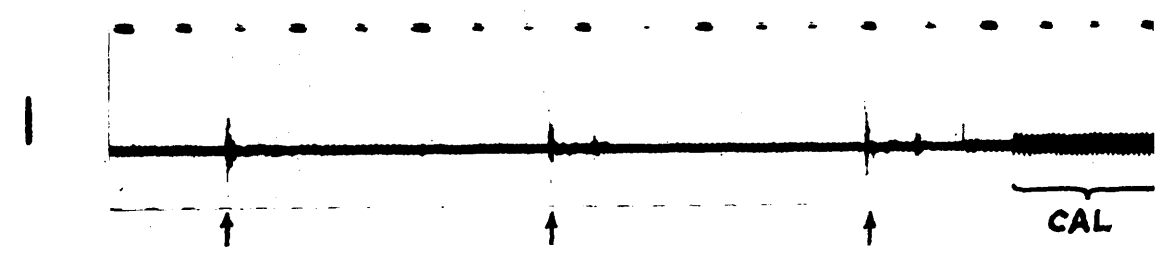


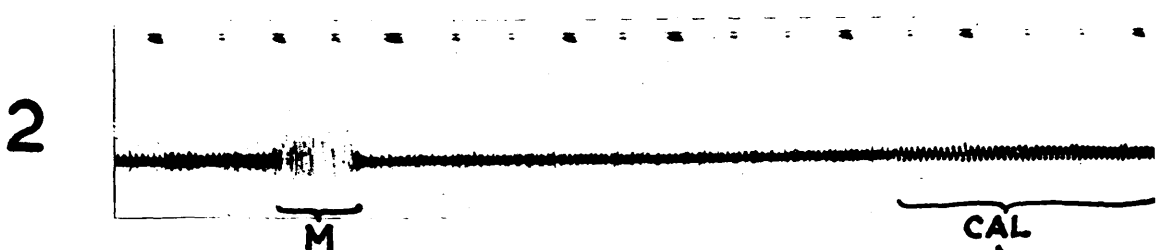

DOG NO.1 (SECOND SERIES)




DOG NO.2 (SECOND SERIES)




DOG NO.5 (SECOND SERIES)

## PLATE 79

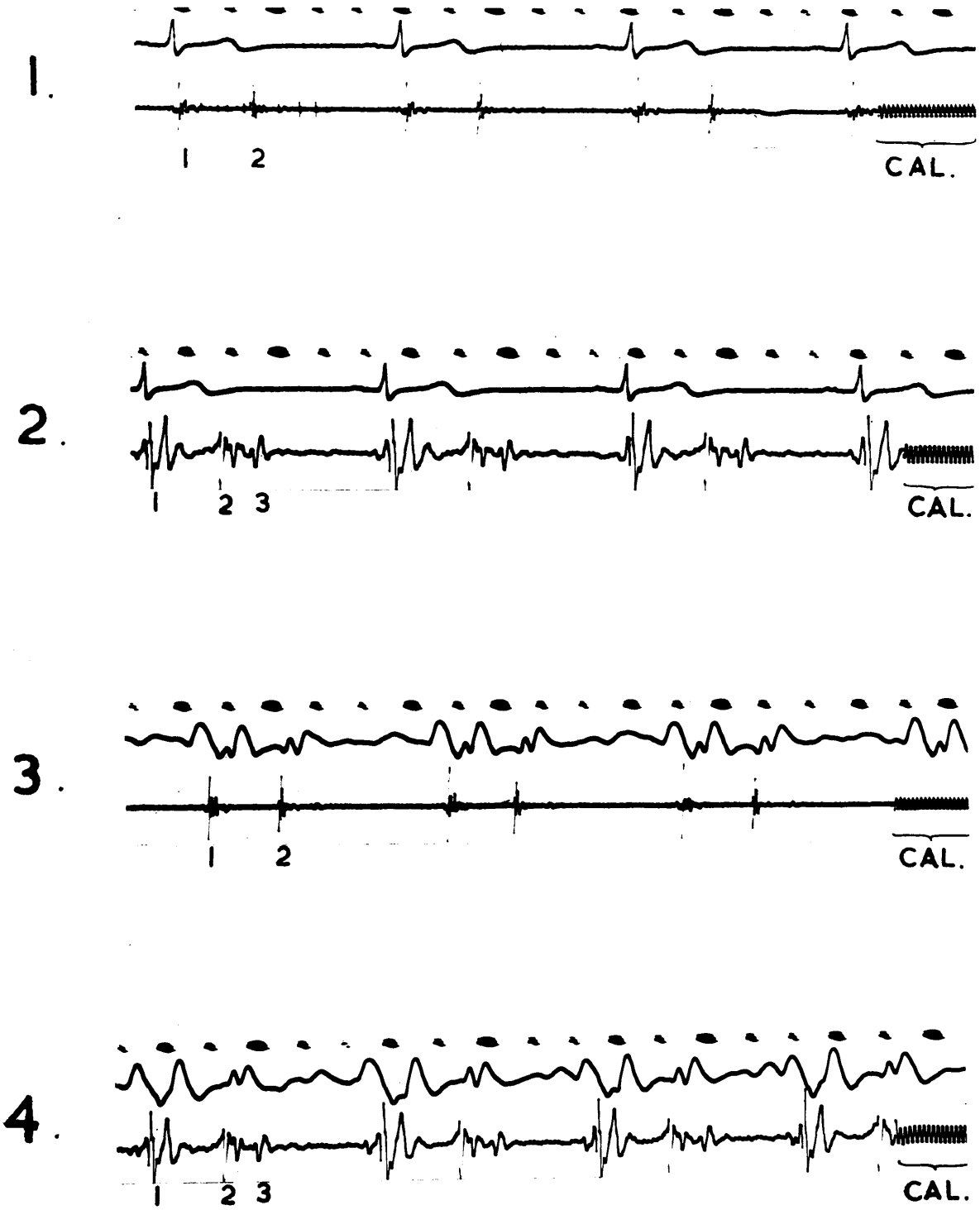
Calibration test record on normal subject - male, age 20.

P.C.G.s from the mitral area
Reference tracing in records 1 and 2 - E.C.G. (Lead II)
Reference tracing in records 3 and 4 - jugular phlebogram

1. Logarithmic
2. Linear
3. Logarithmic
4. Linear

Time marking in 1/5 seconds

The calibration signal is produced by an alternating potential of 2.4 mV. at 50 cycles per second applied to the input of the preamplifier. This corresponds to a sound pressure of 6 dynes per sq. cm. on the microphone employed, which is approximately 25 decibels above the threshold of audibility at this frequency.




CALIBRATION TEST RECORD
(NORMAL SUBJECT.)

# APPENDIX D

## TABLES 30 - 55

Table 30. Cardiac extra sounds at mitral area in healthy human subjects: auscultation and logarithmic phonocardiography

Observer 1 - A.S. Henderson
Observer 2 - A.W. Sloan
Observer 3 - F.W. Campbell

Heart rate per minute calculated from P.C.G.

| Subject No. | Age yrs. | Sex | Heart rate | 3rd heart sound | | | | 4th sound P.C.G. |
|---|---|---|---|---|---|---|---|---|
| | | | | Heard by obs. | | | P.C.G. | |
| | | | | 1 | 2 | 3 | | |
| 1 | 20 | M | 70 | - | - | + | - | + |
| 2 | 21 | F | 62 | - | - | - | + | - |
| 3 | 19 | M | 66 | - | - | + | + | + |
| 4 | 22 | M | 100 | - | - | - | - | - |
| 5 | 22 | M | 70 | - | - | - | - | - |
| 6 | 20 | F | 90 | - | - | - | - | - |
| 7 | 21 | M | 55 | - | - | - | - | - |
| 8 | 21 | F | 67 | - | - | - | - | - |
| 9 | 20 | M | 56 | - | - | - | + | + |
| 10 | 19 | M | 72 | + | - | + | - | - |
| 11 | 22 | M | 85 | - | - | - | - | - |
| 12 | 17 | M | 70 | - | - | - | - | - |
| 13 | 18 | M | 60 | - | - | - | - | - |
| 14 | 21 | M | 58 | - | - | - | + | + |
| 15 | 18 | M | 65 | - | - | + | + | + |

Table 30. (continued)

| Subject No. | Age yrs. | Sex | Heart rate | 3rd heart sound | | | | 4th sound P.C.G. |
|---|---|---|---|---|---|---|---|---|
| | | | | Heard by obs. | | | P.C.G. | |
| | | | | 1 | 2 | 3 | | |
| 16 | 31 | M | 54 | - | - | - | - | - |
| 17 | 18 | M | 80 | - | + | - | - | - |
| 18 | 20 | F | 68 | - | - | - | + | + |
| 19 | 20 | F | 64 | - | - | - | - | - |
| 20 | 18 | M | 72 | - | - | - | - | - |
| 21 | 18 | F | 74 | + | - | - | + | + |
| 22 | 21 | M | 64 | - | - | - | - | + |
| 23 | 18 | M | 82 | + | - | + | + | + |
| 24 | 24 | M | 82 | - | - | - | + | + |
| 25 | 18 | F | 86 | - | - | - | + | - |
| 26 | 21 | M | 68 | - | - | - | - | - |
| 27 | 19 | F | 62 | - | - | - | - | - |
| 28 | 19 | M | 56 | - | - | - | + | - |
| 29 | 20 | M | 66 | - | - | + | + | - |
| 30 | 21 | M | 48 | - | - | - | - | + |
| 31 | 19 | M | 70 | - | - | - | - | + |
| 32 | 22 | F | 58 | + | - | + | - | - |
| 33 | 19 | F | 90 | - | - | - | - | - |
| 34 | 27 | M | 50 | - | - | - | - | - |
| 35 | 19 | F | 75 | - | - | - | - | - |

Table 30. (continued)

| Subject No. | Age yrs. | Sex | Heart rate | 3rd heart sound | | | | 4th sound P.C.G. |
|---|---|---|---|---|---|---|---|---|
| | | | | Heard by obs. | | | P.C.G. | |
| | | | | 1 | 2 | 3 | | |
| 36 | 20 | M | 90 | - | - | - | + | + |
| 37 | 18 | M | 94 | - | - | - | + | - |
| 38 | 17 | F | 84 | - | - | - | - | - |
| 39 | 20 | M | 46 | + | - | + | + | - |
| 40 | 18 | M | 104 | - | - | - | - | - |
| 41 | 20 | M | 78 | + | + | + | + | + |
| 42 | 20 | F | 106 | - | - | - | - | - |
| 43 | 24 | M | 65 | - | - | - | + | - |
| 44 | 24 | M | 85 | - | - | - | - | - |
| 45 | 19 | M | 100 | - | + | + | - | + |
| 46 | 18 | M | 72 | + | + | - | + | + |
| 47 | 20 | M | 85 | - | - | - | - | - |
| 48 | 18 | M | 88 | - | - | - | - | + |
| 49 | 20 | M | 62 | + | + | - | + | + |
| 50 | 27 | M | 75 | - | - | + | - | - |
| 51 | 22 | M | 55 | - | + | + | - | - |
| 52 | 23 | M | 46 | - | - | - | + | - |
| 53 | 19 | M | 76 | - | - | - | + | + |
| 54 | 18 | F | 90 | - | - | - | + | - |
| 55 | 18 | M | 72 | - | - | - | + | + |

Table 30. (continued)

| Subject No. | Age yrs. | Sex | Heart rate | 3rd heart sound | | | | 4th sound P.C.G. |
|---|---|---|---|---|---|---|---|---|
| | | | | Heard by obs. | | | P.C.G. | |
| | | | | 1 | 2 | 3 | | |
| 56 | 26 | M | 95 | - | - | - | - | - |
| 57 | 20 | F | 96 | + | - | - | - | - |
| 58 | 20 | M | 95 | - | - | + | + | - |
| 59 | 25 | M | 64 | - | - | - | - | - |
| 60 | 21 | M | 72 | - | - | + | + | + |
| 61 | 19 | M | 66 | - | - | - | + | + |
| 62 | 20 | M | 70 | + | + | + | - | - |
| 63 | 23 | M | 76 | - | - | - | + | + |
| 64 | 23 | M | 70 | - | - | - | + | - |
| 65 | 23 | M | 75 | - | - | - | + | - |
| 66 | 23 | M | 85 | - | - | + | - | - |
| 67 | 26 | M | 70 | - | - | - | - | - |
| 68 | 18 | F | 46 | - | - | - | + | - |
| 69 | 19 | F | 96 | - | - | - | - | - |
| 70 | 21 | M | 76 | - | - | + | + | - |
| 71 | 20 | M | 64 | - | - | - | - | - |
| 72 | 19 | M | 95 | - | - | - | + | - |
| 73 | 22 | M | 80 | - | - | - | - | + |
| 74 | 23 | F | 78 | - | - | - | + | - |
| 75 | 32 | M | 68 | - | - | - | - | - |

Table 30. (continued)

| Subject No. | Age yrs. | Sex | Heart rate | 3rd heart sound | | | | 4th sound P.C.G. |
|---|---|---|---|---|---|---|---|---|
| | | | | Heard by obs. | | | P.C.G. | |
| | | | | 1 | 2 | 3 | | |
| 76 | 18 | F | 72 | - | - | - | - | - |
| 77 | 19 | M | 72 | - | - | - | - | - |
| 78 | 26 | M | 66 | - | - | + | + | - |
| 79 | 19 | M | 66 | - | - | - | + | + |
| 80 | 26 | M | 68 | - | - | - | - | + |
| 81 | 23 | M | 66 | - | - | - | - | - |
| 82 | 31 | M | 98 | - | - | - | - | - |
| 83 | 17 | M | 65 | - | - | + | - | - |
| 84 | 24 | M | 64 | - | - | - | - | - |
| 85 | 20 | M | 70 | - | - | - | + | + |
| 86 | 17 | F | 90 | - | - | - | + | - |
| 87 | 18 | F | 106 | - | + | - | + | - |
| 88 | 30 | M | 85 | - | - | + | - | - |
| 89 | 20 | M | 72 | - | - | - | - | - |
| 90 | 20 | M | 84 | - | - | - | + | - |
| 91 | 24 | M | 56 | - | - | - | - | - |
| 92 | 18 | F | 76 | - | - | - | - | - |
| 93 | 21 | M | 66 | - | - | + | - | - |
| 94 | 18 | M | 70 | - | + | - | + | + |
| 95 | 20 | F | 88 | - | - | - | - | - |

Table 30. (continued)

| Subject No. | Age yrs. | Sex | Heart rate | 3rd heart sound | | | | 4th sound P.C.G. |
|---|---|---|---|---|---|---|---|---|
| | | | | Heard by obs. | | | P.C.G. | |
| | | | | 1 | 2 | 3 | | |
| 96 | 25 | M | 75 | - | - | - | + | - |
| 97 | 21 | M | 80 | - | - | - | - | - |
| 98 | 19 | M | 73 | + | + | + | + | - |
| 99 | 23 | M | 66 | - | + | + | + | + |
| 100 | 17 | M | 95 | - | - | - | - | - |
| 101 | 27 | M | 70 | - | - | - | - | - |
| 102 | 22 | M | 68 | - | - | - | - | - |
| 103 | 20 | F | 74 | - | - | - | - | - |
| 104 | 19 | F | 104 | - | - | - | - | - |
| 105 | 29 | M | 78 | - | - | - | - | - |
| 106 | 20 | M | 66 | - | - | + | - | + |
| 107 | 17 | M | 85 | - | - | - | - | + |
| 108 | 24 | M | 76 | - | - | - | + | + |
| 109 | 21 | M | 87 | - | - | - | - | - |
| 110 | 22 | M | 66 | - | - | - | - | - |
| 111 | 20 | M | 90 | - | - | - | - | - |
| 112 | 23 | M | 66 | - | - | - | - | - |
| 113 | 18 | M | 86 | - | - | + | + | - |
| 114 | 20 | M | 85 | - | - | - | - | - |
| 115 | 24 | M | 78 | - | - | - | - | + |

Table 30. (continued)

| Subject No. | Age yrs. | Sex | Heart rate | 3rd heart sound | | | | 4th sound P.C.G. |
|---|---|---|---|---|---|---|---|---|
| | | | | Heard by obs. | | | P.C.G. | |
| | | | | 1 | 2 | 3 | | |
| 116 | 21 | M | 62 | - | - | - | - | - |
| 117 | 22 | M | 56 | - | - | - | - | - |
| 118 | 19 | F | 80 | - | - | - | - | + |
| 119 | 20 | M | 74 | - | - | + | - | - |
| 120 | 25 | M | 76 | - | - | - | - | - |
| 121 | 19 | F | 70 | - | - | - | + | - |
| 122 | 25 | M | 64 | - | + | - | + | - |
| 123 | 22 | M | 76 | - | - | - | + | - |

A systolic extra sound was seen on the logarithmic P.C.G. of subjects 4, 40, 58 and 96.

Table 31. Heart sounds and murmurs at mitral area in patients with heart disease (auscultation and logarithmic phonocardiography): follow-up

Diagnosis by physician in charge
Auscultation by A.W. Sloan
Follow-up assessment of patient's general health by doctor in charge of case (except where other source of information indicated)

KEY

Sounds:
3 - Protodiastolic gallop sound
4 - Presystolic gallop sound
Sys - Systolic extra sound
OS - Opening snap of mitral valve

Murmurs:
VS - Pansystolic murmur
ES - Early systolic murmur
MS - Mid-systolic murmur
LS - Late systolic murmur

VD - Pandiastolic murmur
ED - Early diastolic murmur
MD - Mid-diastolic murmur
LD - Late diastolic murmur

Con. - Continuous murmur

| Patient No. | Age yrs. | Sex | Diagnosis | Auscultation | | Phonocardiography | | Follow-up (6-9 months) | Notes |
|---|---|---|---|---|---|---|---|---|---|
| | | | | Sounds | Murmurs | Sounds | Murmurs | | |
| 1 | 44 | M | Mitral stenosis Auricular fibrillation | 1 2 | VS | 1 2 | VS | Improved | |
| 2 | 33 | M | Mitral stenosis Auricular fibrillation | 1 2 | ES MD | 1 2 OS | VS MD | Unchanged | Mitral valvotomy 4 months later |
| 3 | 9 | M | Mitral stenosis | 1 2 | VS | 1 2 | VS | Improved | |

Table 31. (continued)

| Patient No. | Age yrs. | Sex | Diagnosis | Auscultation | | Phonocardiography | | Follow-up (6-9 months) | Notes |
|---|---|---|---|---|---|---|---|---|---|
| | | | | Sounds | Murmurs | Sounds | Murmurs | | |
| 4 | 21 | F | Mitral stenosis<br>Aortic incompetence | 1 2 | ES MS ED | 1 2 OS | - | Unchanged | |
| 5 | 8 | F | Patent ductus arteriosus | 1 2 | VS | 1 2 | MS LS | Improved | Ligation of ductus 3 weeks later |
| 6 | 23 | F | Mitral stenosis<br>Aortic incompetence | 1 2 | VD | 1 2 | - | Improved | Pregnant: Spontaneous full-term delivery 4 months later |
| 7 | 20 | F | Mitral stenosis | 1 2 | ES VD | 1 2 | MD LD | Improved | Mitral valvotomy 7 months later |
| 8 | 52 | M | Arterial hypertension<br>Anterior myocardial infarction<br>Cardiac failure | 1 2 | ES | 1 2 | ES | Died 5 days later | |
| 9 | 23 | F | Mitral stenosis | 1 2 | VD | 1 2 | MD LD | Improved | Mitral valvotomy 3 months later |
| 10 | 38 | F | Mitral stenosis<br>Aortic incompetence | 1 2 | Con. | 1 2 | VS ED | Untraced | Left Scotland |
| 11 | 60 | F | Arterial hypertension<br>Myocardial degeneration<br>Congestive cardiac failure | 1 2 | - | 1 2 3 | - | Unchanged (patient) | Diabetes mellitus<br>Hemiplegia |
| 12 | 70 | M | Arterial hypertension<br>Left heart strain | 1 2 | ES | 1 2 Sys | - | Improved | |
| 13 | 59 | M | Arterial hypertension<br>Left heart strain | 1 2 | - | 1 2 | - | Unchanged | |
| 14 | 48 | M | Arterial hypertension<br>Cor pulmonale<br>Myocardial degeneration | 1 2 | - | 1 2 3 | - | Unchanged | Chronic bronchitis and emphysema |
| 15 | 69 | M | Arterial hypertension<br>Anterior myocardial infarction<br>Congestive cardiac failure | 1 2 | - | 1 2 | - | Died 4 months later | Pyonephrosis (L) |

Table 31. (continued)

| Patient No. | Age yrs. | Sex | Diagnosis | Auscultation | | Phonocardiography | | Follow-up (6-9 months) | Notes |
|---|---|---|---|---|---|---|---|---|---|
| | | | | Sounds | Murmurs | Sounds | Murmurs | | |
| 16 | 52 | M | Angina pectoris | 1 2 | - | 1 2 | - | Unchanged | |
| 17 | 68 | M | Myocardial degeneration | 1 2 | - | 1 2 | - | Unchanged | Duodenal ulcer |
| 18 | 15 | F | Mitral incompetence | 1 2 | VS | 1 2 | VS | Improved | |
| 19 | 76 | F | Arterial hypertension<br>Myocardial disease<br>Left heart strain | 1 2 | - | 1 2 | - | Improved | |
| 20 | 19 | F | Mitral incompetence | 1 2 | VS | 1 2 | VS | Improved (patient) | 7 months pregnant at follow-up |
| 21 | 14 | F | Mitral stenosis<br>Auricular fibrillation | 1 2 | Con. | 1 2 | VS ED | Improved (mother) | |
| 22 | 26 | F | Mitral and aortic incompetence<br>Acute rheumatic carditis | 1 2 | VS | 1 2 4 | VS | Improved | |
| 23 | 15 | F | Acute rheumatic carditis | 1 2 | VS | 1 2 | - | Improved (patient) | |
| 24 | 31 | M | Mitral stenosis<br>Auricular fibrillation | 1 2 3 | MD | 1 2 | - | Unchanged (patient) | |
| 25 | 58 | M | Anterior myocardial infarction | 1 2 | - | 1 2 4 | - | Worse | |
| 26 | 64 | M | Arterial hypertension<br>Left heart strain | 1 2 | - | 1 2 4 | ES MS | Unchanged | Chronic nephritis |
| 27 | 27 | F | Mitral stenosis | 1 2 | VS MD | 1 2 | VS | Died 2 months later | Anaesthetic death |
| 28 | 46 | F | Mitral stenosis<br>Auricular fibrillation | 1 2 | VD | 1 2 OS | MD LD | Untraced | Address unknown |
| 29 | 21 | F | Mitral stenosis<br>Acute rheumatic carditis | 1 2 | ES VD | 1 2 4 | ES LD | Improved | |
| 30 | 39 | F | Mitral stenosis (post-valvotomy) | 1 2 | MD LD | 1 2 OS | ES VD | Improved | |

Table 31. (continued)

| Patient No. | Age yrs. | Sex | Diagnosis | Auscultation | | Phonocardiography | | Follow-up (6-9 months) | Notes |
|---|---|---|---|---|---|---|---|---|---|
| | | | | Sounds | Murmurs | Sounds | Murmurs | | |
| 31 | 49 | F | Mitral stenosis and incompetence<br>Aortic stenosis and incompetence | 1 2 | Con. | 1 2 | VS ED | Unchanged | |
| 32 | 25 | F | Mitral stenosis | 1 2 | VS ED LD | 1 2 OS | VS ED LD | Improved | Bronchiectasis<br>Mitral valvotomy 1 week later |
| 33 | 44 | F | Arterial hypertension<br>Left heart strain | 1 2 | ES MS | 1 2 | ES | Improved | Chronic nephritis<br>Iron-deficiency anaemia |
| 34 | 14 | F | Mitral stenosis<br>Myocardial disease | 1 2 OS | VS | 1 2 OS | ES MS | Unchanged (mother) | |
| 35 | 33 | F | Mitral stenosis (post-valvotomy) | 1 2 | MD | 1 2 3 | - | Improved | |
| 36 | 41 | F | Mitral stenosis (post-valvotomy)<br>Auricular fibrillation | 1 2 | VS MD | 1 2 OS | ES MS VD | Unchanged | |
| 37 | 52 | F | Mitral stenosis<br>Aortic incompetence | 1 2 | VS MD LD | 1 2 3 | VS ED LD | Died 5 months later | |
| 38 | 35 | F | Mitral stenosis and incompetence<br>Aortic incompetence | 1 2 | VS MD LD | 1 2 | ES MS LD | Unchanged | |
| 39 | 30 | M | Mitral stenosis | 1 2 | MD LD | 1 2 | MD LD | Worse | |
| 40 | 16 | M | Mitral stenosis<br>Aortic incompetence | 1 2 | VS | 1 2 3 | VS LD | Died 3 months later | |
| 41 | 66 | M | Aortic stenosis<br>Congestive cardiac failure | 1 2 | - | 1 2 3 4 | VS | Died 3 months later | Emphysema |
| 42 | 15 | M | Fallot's tetralogy | 1 2 | VS | 1 2 | VS | Unchanged | |
| 43 | 24 | M | Mitral stenosis | 1 2 | VS | 1 2 | MD LD | Unchanged | Mitral valvotomy 6 months later |

Table 31. (continued)

| Patient No. | Age yrs. | Sex | Diagnosis | Auscultation | | Phonocardiography | | Follow-up (6-9 months) | Notes |
|---|---|---|---|---|---|---|---|---|---|
| | | | | Sounds | Murmurs | Sounds | Murmurs | | |
| 44 | 28 | F | Mitral stenosis | 1 2 | MD | 1 2 | - | Unchanged (patient) | Pregnant. Mitral valvotomy 3 weeks later. Full-term delivery 5 months later. |
| 45 | 7 | F | Patent ductus arteriosus | 1 2 | VS | 1 2 | VS ED | Improved | Ductus ligated 2 weeks later |
| 46 | 21 | F | Mitral stenosis | 1 2 | VS MD | 1 2 | ES MD | Unchanged | |
| 47 | 51 | F | Arterial hypertension Cardiac failure | 1 2 | ES | 1 2 | ES | Improved | |
| 48 | 63 | F | Arterial hypertension Anterior myocardial infarction Congestive cardiac failure | 1 2 | ES | 1 2 4 | ES | Worse | |
| 49 | 44 | F | Arterial hypertension Mitral stenosis Cardiac failure | 1 2 | VS | 1 2 | VS | Unchanged | |
| 50 | 42 | F | Mitral stenosis Subacute bacterial endocarditis Auricular fibrillation | 1 2 | VS MD | 1 2 | VS | Unchanged | Pneumonia (R) |
| 51 | 38 | F | Arterial hypertension Left heart strain | 1 2 | ES | 1 2 4 | ES MS | Improved | Chronic nephritis |
| 52 | 72 | F | Arterial hypertension Auricular fibrillation Congestive cardiac failure | 1 2 | ES MS | 1 2 | ES MS | Improved | Chronic nephritis |
| 53 | 31 | F | Mitral stenosis | 1 2 | ES | 1 2 OS | ES MS | Unchanged (patient) | Duodenal ulcer |
| 54 | 48 | F | Arterial hypertension Congestive cardiac failure | 1 2 | ES | 1 2 | ES | Unchanged | Chronic bronchitis |

Table 31. (continued)

| Patient No. | Age yrs. | Sex | Diagnosis | Auscultation | | Phonocardiography | | Follow-up (6-9 months) | Notes |
|---|---|---|---|---|---|---|---|---|---|
| | | | | Sounds | Murmurs | Sounds | Murmurs | | |
| 55 | 28 | M | Mitral stenosis (post-valvotomy) | 1 2 | VS | 1 2 | - | Improved | |
| 56 | 56 | M | Angina pectoris | 1 2 | ES | 1 2 | - | Unchanged | |
| 57 | 63 | F | Arterial hypertension Left heart strain | 1 2 | VS | 1 2 | VS | Unchanged | |
| 58 | 53 | F | Arterial hypertension Left heart strain | 1 2 | VS | 1 2 | ES MS | Unchanged | |
| 59 | 32 | F | Mitral stenosis Aortic incompetence Auricular fibrillation | 1 2 3 | ES | 1 2 3 | ES | Unchanged | |
| 60 | 36 | F | Mitral stenosis Aortic incompetence Congestive cardiac failure | 1 2 | VS ED LD | 1 2 3 | VD | Improved | |
| 61 | 11 | F | Congenital pulmonary stenosis | 1 2 | ES MS LD | 1 2 3 | ES MS MD LD | Improved (patient) | |
| 62 | 52 | F | Mitral stenosis Aortic incompetence Auricular fibrillation Congestive cardiac failure | 1 2 | VS | 1 2 3 | VS ED | Worse | |
| 63 | 59 | M | Posterior myocardial infarction Left heart strain | 1 2 | ES MS | 1 2 | ES | Unchanged | |
| 64 | 59 | M | Arterial hypertension Myocardial disease Left heart strain | 1 2 | ES MS | 1 2 | ES MS | Unchanged | |
| 65 | 53 | F | Congenital pulmonary stenosis | 1 2 | VS | 1 2 | ES MS | Improved | Duodenal Ulcer |
| 66 | 34 | F | Mitral stenosis (post-valvotomy) | 1 2 | VD | 1 2 3 | - | Improved (patient) | |
| 67 | 31 | M | Mitral incompetence Acute rheumatic carditis | 1 2 | VS | 1 2 3 | VS | Improved (patient) | |

Table 31. (continued)

| Patient No. | Age yrs. | Sex | Diagnosis | Auscultation | | Phonocardiography | | Follow-up (6-9 months) | Notes |
|---|---|---|---|---|---|---|---|---|---|
| | | | | Sounds | Murmurs | Sounds | Murmurs | | |
| 68 | 58 | M | Anterior myocardial infarction | 1 2 | - | 1 2 | - | Died 5 days later | |
| 69 | 62 | M | Arterial hypertension<br>Myocardial disease<br>Left heart strain | 1 2 | - | 1 2 3 4 | - | Worse (patient) | Diabetes mellitus |
| 70 | 76 | M | Anterior myocardial infarction | 1 2 | - | 1 2 | - | Improved | |
| 71 | 59 | F | Arterial hypertension<br>Left bundle branch block | 1 2 | - | 1 2 | - | Worse | Bronchial asthma |
| 72 | 45 | M | Posterior myocardial infarction | 1 2 | - | 1 2 | - | Improved | |
| 73 | 40 | F | Mitral stenosis | 1 2 | MD LD | 1 2 Sys OS | MD LD | Improved | Mitral valvotomy 6 months later |
| 74 | 61 | F | Cor pulmonale | 1 2 | - | 1 2 Sys | - | Unchanged | Bronchial asthma |
| 75 | 49 | M | Mitral stenosis<br>Myocardial disease<br>Auricular fibrillation<br>Congestive cardiac failure | 1 2 | VS ED MD | 1 2 | VS ED | Died 2 days later | Lobar pneumonia (L)<br>Acute nephritis |
| 76 | 39 | M | Arterial hypertension<br>Cardiac failure | 1 2 | VS | 1 2 | VS | Died 3 weeks later | Malignant hypertension |
| 77 | 23 | F | Mitral incompetence | 1 2 | VS | 1 2 | - | Unchanged (patient) | 4 months pregnant at follow-up |
| 78 | 52 | F | Mitral stenosis | 1 2 | ES | 1 2 | ES | Improved | Hemiplegia (R) |
| 79 | 68 | F | Cor pulmonale | 1 2 | VS | 1 2 | ES MS | Improved | Basal pneumonia |
| 80 | 61 | M | Left bundle branch block<br>Cardiac failure | 1 2 | - | 1 2 4 | - | Worse | |
| 81 | 60 | M | Angina pectoris<br>Left bundle branch block | 1 2 4 | ES | 1 2 4 | MS | Improved | |
| 82 | 50 | M | Right bundle branch block<br>Cor pulmonale | 1 2 | ES | 1 2 | ES | Improved | Chronic bronchitis |

Table 31. (continued)

| Patient No. | Age yrs. | Sex | Diagnosis | Auscultation | | Phonocardiography | | Follow-up (6-9 months) | Notes |
|---|---|---|---|---|---|---|---|---|---|
| | | | | Sounds | Murmurs | Sounds | Murmurs | | |
| 83 | 58 | M | Arterial hypertension<br>Myocardial disease<br>Left heart strain | 1 2 | ES MS | 1 2 | VS LD | Unchanged | |
| 84 | 42 | M | Arterial hypertension<br>Myocardial disease<br>Angina pectoris | 1 2 | - | 1 2 | - | Improved (patient) | |
| 85 | 63 | F | Arterial hypertension<br>Aortic stenosis | 1 2 | VS | 1 2 4 | VS | Unchanged | |
| 86 | 29 | M | Mitral stenosis<br>Aortic incompetence<br>Left heart strain<br>Subacute bacterial endocarditis | 1 2 | VS MD LD | 1 2 3 | ES MS VD | Improved (patient) | |
| 87 | 57 | F | Arterial hypertension<br>Myocardial disease<br>Congestive cardiac failure | 1 2 | ES | 1 2 4 | ES | Improved | Malignant hypertension |
| 88 | 55 | M | Mitral stenosis and incompetence<br>Aortic incompetence<br>Left bundle branch block | 1 2 | VS MD LD | 1 2 3 4 | VS ED | Died 7 months later | |
| 89 | 66 | M | Arterial hypertension<br>Left heart strain | 1 2 | VS | 1 2 3 4 | VS | Improved | |
| 90 | 29 | F | Mitral stenosis<br>Auricular fibrillation<br>Cardiac failure | 1 2 3 | VS | 1 2 3 | VS ED MD | Improved | |
| 91 | 70 | F | Arterial hypertension<br>Congestive cardiac failure | 1 2 | - | 1 2 | - | Died 5 months later | Kimmelstiel-Wilson syndrome |
| 92 | 45 | F | Arterial hypertension<br>Left heart strain | 1 2 | VS | 1 2 | ES MS | Unchanged | Malignant hypertension |
| 93 | 62 | M | Arterial hypertension<br>Left heart strain | 1 2 | ES | 1 2 | ES MS | Unchanged | Cerebral vascular disease |
| 94 | 64 | F | Arterial hypertension<br>Left heart strain<br>Auricular fibrillation | 1 2 | - | 1 2 | - | Died 3 months later | Chronic bronchitis |

Table 31. (continued)

| Patient No. | Age yrs. | Sex | Diagnosis | Auscultation | | Phonocardiography | | Follow-up (6-9 months) | Notes |
|---|---|---|---|---|---|---|---|---|---|
| | | | | Sounds | Murmurs | Sounds | Murmurs | | |
| 95 | 32 | F | Mitral stenosis<br>Aortic incompetence<br>Left heart strain | 1 2 | VS ED LD | 1 2 | - | Unchanged | |
| 96 | 53 | F | Arterial hypertension<br>Left heart strain | 1 2 | ES | 1 2 | VS | Worse | Bronchial asthma |
| 97 | 23 | F | Mitral stenosis | 1 2 | VS MD | 1 2 3 | VS | Unchanged | |
| 98 | 17 | F | Mitral incompetence<br>Subacute bacterial endocarditis | 1 2 | VS | 1 2 3 | VS | Improved | Iron-deficiency anaemia |
| 99 | 23 | M | Aortic incompetence<br>Myocardial disease<br>Left heart strain | 1 2 | Con. | 1 2 | ES MS VD | Unchanged | |
| 100 | 62 | M | Aortic incompetence<br>Cardiac failure | 1 2 | VS | 1 2 | ES MS | Improved | |

Table 32. Effect of venous occlusion and of exercise on the amplitude of the first heart sound at the mitral area in healthy human subjects: logarithmic phonocardiography

| Subject No. | Effect of venous occlusion | | | Effect of exercise | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Exercise (mm.) | Change (%) |
| 1 | 10 | 11 | + 10 | 8 | 25 | +213 |
| 2 | 14 | 13 | - 7 | 11 | 17 | + 55 |
| 3 | 20 | 19 | - 5 | - | - | - |
| 4 | 20 | 18 | - 10 | 20 | 22 | + 10 |
| 5 | 16 | 10 | - 38 | 16 | 23 | + 44 |
| 6 | 12 | 12 | 0 | 9 | 13 | + 44 |
| 7 | 13 | 11 | - 15 | - | - | - |
| 8 | 5 | 8 | + 60 | 5 | 26 | +420 |
| 9 | 9 | 15 | + 66 | 6 | 25 | +317 |
| 10 | 5 | 6 | + 20 | 5 | 22 | +340 |
| 11 | 20 | 10 | - 50 | 11 | 24 | +118 |
| 12 | 10 | 14 | + 40 | 13 | 21 | + 62 |
| 13 | 9 | 10 | + 11 | 12 | 24 | +100 |
| 14 | 6 | 5 | - 17 | 6 | 25 | +317 |
| 15 | 5 | 6 | + 20 | 6 | 25 | +317 |
| 16 | 20 | 13 | - 35 | 14 | 21 | + 50 |

N.B. With subjects 3 and 7 the stethoscope chest-piece was displaced by the exercise.

Table 33. Effect of venous occlusion and of exercise on the amplitude of the second heart sound at the mitral area in healthy human subjects: logarithmic phonocardiography

| Subject No. | Effect of venous occlusion | | | Effect of exercise | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Exercise (mm.) | Change (%) |
| 1 | 17 | 16 | - 6 | 9 | 9 | 0 |
| 2 | 14 | 12 | - 14 | 13 | 10 | - 23 |
| 3 | 6 | 10 | + 67 | - | - | - |
| 4 | 13 | 25 | + 92 | 15 | 17 | + 13 |
| 5 | 10 | 11 | + 10 | 11 | 13 | + 18 |
| 6 | 12 | 15 | + 25 | 12 | 13 | + 8 |
| 7 | 12 | 11 | - 8 | - | - | - |
| 8 | 12 | 11 | - 8 | 13 | 8 | - 39 |
| 9 | 7 | 4 | - 43 | 8 | 8 | 0 |
| 10 | 13 | 13 | 0 | 15 | 20 | + 33 |
| 11 | 11 | 9 | - 18 | 7 | 9 | + 29 |
| 12 | 6 | 9 | + 50 | 7 | 8 | + 14 |
| 13 | 9 | 10 | + 11 | 10 | 11 | + 10 |
| 14 | 9 | 6 | - 33 | 11 | 18 | + 64 |
| 15 | 7 | 7 | 0 | 9 | 16 | + 78 |
| 16 | 20 | 21 | + 5 | 17 | 16 | - 6 |

Table 34. Effect of venous occlusion and of exercise on the amplitude of the third heart sound at the mitral area in healthy human subjects: logarithmic phonocardiography

| Subject No. | Effect of venous occlusion | | | Effect of exercise | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Exercise (mm.) | Change (%) |
| 1 | 5 | 2 | - 60 | 4 | 12 | +200 |
| 2 | 4 | 2 | - 50 | 4 | 4 | 0 |
| 3 | 4 | 2 | - 50 | - | - | - |
| 4 | 8 | 2 | - 75 | 10 | 11 | + 10 |
| 5 | 3 | 1 | - 67 | 2 | 3 | + 50 |
| 6 | 4 | 1 | - 75 | 3 | 6 | +100 |
| 7 | 3 | 1 | - 67 | - | - | - |
| 8 | 5 | 1 | - 80 | 4 | 8 | +100 |
| 9 | 3 | 0 | -100 | 3 | 3 | 0 |
| 10 | 5 | 2 | - 60 | 3 | 5 | + 67 |
| 11 | 3 | 0 | -100 | 1 | 3 | +200 |
| 12 | 2 | 0 | -100 | 2 | 4 | +100 |
| 13 | 4 | 2 | - 50 | 3 | 4 | + 33 |
| 14 | 2 | 1 | - 50 | 2 | 5 | +150 |
| 15 | 3 | 0 | -100 | 3 | 3 | 0 |
| 16 | 10 | 3 | - 70 | 8 | 8 | 0 |

Table 35. Effect of venous occlusion and of exercise on the amplitude of the fourth heart sound at the mitral area in healthy human subjects: logarithmic phonocardiography

| Subject No. | Effect of venous occlusion | | | Effect of exercise | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Exercise (mm.) | Change (%) |
| 1 | 3 | 0 | -100 | 2 | 3 | + 50 |
| 4 | 7 | 5 | - 29 | 7 | 11 | + 57 |
| 5 | 2 | 0 | -100 | 2 | 3 | + 50 |
| 6 | 3 | 2 | - 33 | 2 | 2 | 0 |
| 7 | 2 | 1 | - 50 | - | - | - |
| 9 | 2 | 1 | - 50 | 2 | 2 | 0 |
| 12 | 1 | 1 | 0 | 1 | 2 | +100 |
| 13 | 3 | 3 | 0 | 3 | 4 | + 33 |
| 14 | 3 | 2 | - 33 | 2 | 4 | +100 |
| 16 | 5 | 4 | - 20 | 4 | 5 | + 25 |

N.B. With subjects 2, 3, 11, and 15, the fourth heart sound was absent throughout.

With subjects 8 and 10 the sound was present only after exercise.

Table 36. Effect of venous occlusion and of exercise on the heart rate of healthy human subjects

(heart rate from phonocardiograms)

| Subject No. | Effect of venous occlusion | | | Effect of exercise | | |
|---|---|---|---|---|---|---|
| | Control per min. | Occlsn. per min. | Change (%) | Control per min. | Exercise per min. | Change (%) |
| 1 | 67 | 79 | + 18 | 67 | 94 | + 40 |
| 2 | 88 | 77 | - 13 | 94 | 108 | + 15 |
| 3 | 51 | 64 | + 25 | - | - | - |
| 4 | 83 | 91 | + 10 | 83 | 142 | + 71 |
| 5 | 85 | 72 | - 15 | 73 | 72 | - 1 |
| 6 | 72 | 59 | - 18 | 59 | 67 | + 14 |
| 7 | 63 | 61 | - 3 | - | - | - |
| 8 | 87 | 91 | + 5 | 71 | 117 | + 65 |
| 9 | 92 | 92 | 0 | 77 | 92 | + 19 |
| 10 | 71 | 75 | + 6 | 66 | 101 | + 53 |
| 11 | 75 | 78 | + 4 | 79 | 111 | + 41 |
| 12 | 53 | 50 | - 6 | 56 | 77 | + 38 |
| 13 | 65 | 64 | - 2 | 61 | 77 | + 26 |
| 14 | 63 | 66 | + 5 | 59 | 113 | + 92 |
| 15 | 79 | 78 | - 1 | 63 | 100 | + 59 |
| 16 | 82 | 82 | 0 | 67 | 84 | + 25 |

Table 37. Incidence of cardiac extra sounds in a series of 12 dogs: logarithmic phonocardiography from chest wall (mitral area), from right ventricle, and from left ventricle

| Dog No. | Third heart sound | | | Fourth heart sound | | | Systolic extra sound | | |
|---|---|---|---|---|---|---|---|---|---|
| | Chest wall | Right vent. | Left vent. | Chest wall | Right vent. | Left vent. | Chest wall | Right vent. | Left vent. |
| 1 | - | - | | - | + | | - | - | |
| 2 | - | - | | - | + | | - | - | |
| 3 | - | - | | - | - | | - | - | |
| 4 | - | - | | - | - | | - | - | |
| 5 | + | - | - | - | - | - | - | - | + |
| 6 | - | - | - | - | - | - | - | + | - |
| 7 | - | - | - | - | + | - | - | - | - |
| 8 | - | - | + | - | + | - | - | - | - |
| 9 | - | + | - | - | + | - | - | - | - |
| 10 | - | - | - | - | + | - | - | - | - |
| 11 | - | - | - | - | + | - | - | - | - |
| 12 | - | - | - | - | + | - | - | - | - |

N.B. No records were obtained from the left ventricle in dogs 1 - 4.

Table 38. Incidence of vibrations at the time of third and fourth heart sounds in a series of 12 dogs: stethoscopic phonocardiography from chest wall (mitral area), from right ventricle, and from left ventricle

| Dog No. | Third heart sound | | | Fourth heart sound | | |
|---|---|---|---|---|---|---|
| | Chest wall | Right vent. | Left vent. | Chest wall | Right vent. | Left vent. |
| 1 | + | + | | - | + | |
| 2 | + | + | | + | + | |
| 3 | + | + | | + | + | |
| 4 | + | + | | - | + | |
| 5 | + | + | + | + | + | + |
| 6 | + | + | + | + | + | + |
| 7 | + | + | + | + | + | - |
| 8 | + | + | + | - | - | - |
| 9 | + | + | + | + | + | - |
| 10 | + | - | - | + | + | + |
| 11 | + | - | - | + | + | + |
| 12 | + | - | - | + | + | + |

N.B. No records were obtained from the left ventricle in dogs 1 - 4.

Table 39. Effect of venous occlusion and of rapid intravenous infusion on the amplitude of the first heart sound in dogs: logarithmic phonocardiography from right ventricle

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Infusn. (mm.) | Change (%) |
| 1 | 13.0 | 5.5 | -57.7 | 18.0 | 22.0 | +22.2 |
| 3 | 20.0 | 16.5 | -17.5 | 14.0 | 15.0 | + 7.1 |
| 4 | 21.0 | 18.5 | -11.9 | - | - | - |
| 5 | - | - | - | 11.0 | 14.0 | +27.3 |
| 6 | 14.5 | 4.0 | -72.4 | - | - | - |
| 9 | - | - | - | 7.5 | 8.5 | +13.3 |
| 10 | 17.0 | 4.0 | -76.5 | - | - | - |

N.B. The effect of venous occlusion was not studied by logarithmic phonocardiography in dogs 5 and 9.
The effect of rapid intravenous infusion was not studied by logarithmic phonocardiography in dogs 4, 6, and 10.

Table 40. Effect of venous occlusion and of rapid intravenous infusion on the amplitude of the second heart sound in dogs: logarithmic phonocardiography from right ventricle

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Infusn. (mm.) | Change (%) |
| 1 | 2.0 | 0.0 | -100.0 | 11.5 | 15.0 | +30.4 |
| 2 | 6.0 | 0.0 | -100.0 | 13.0 | 5.5 | -57.7 |
| 4 | 3.0 | 1.5 | - 50.0 | - | - | - |
| 5 | - | - | - | 12.5 | 14.5 | +16.0 |
| 6 | 4.5 | 1.5 | - 66.7 | - | - | - |
| 9 | - | - | - | 1.5 | 2.0 | +33.3 |
| 10 | 5.0 | 1.0 | - 80.0 | - | - | - |

Table 41. Effect of venous occlusion and of rapid intravenous infusion on the amplitude of the third heart sound in dogs: logarithmic phonocardiography from right ventricle

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Infusn. (mm.) | Change (%) |
| 3 | 1.5 | 0.0 | -100.0 | - | - | - |
| 5 | - | - | - | 1.5 | 1.5 | 0.0 |
| 6 | 3.0 | 0.0 | -100.0 | - | - | - |
| 9 | - | - | - | 1.5 | 1.5 | 0.0 |
| 10 | 1.5 | 0.0 | -100.0 | - | - | - |

Table 42. Effect of venous occlusion and of rapid intravenous infusion on the amplitude of the fourth heart sound in dogs: logarithmic phonocardiography from right ventricle

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Infusn. (mm.) | Change (%) |
| 1 | 2.0 | 0.0 | -100.0 | 2.0 | 1.5 | -25.0 |
| 10 | 5.0 | 0.0 | -100.0 | - | - | - |

N.B. With dog 3 the sound was present only during venous occlusion.

Table 43. Effect of venous occlusion and of rapid intravenous infusion on the amplitude of the systolic extra sound in dogs: logarithmic phonocardiography from right ventricle

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Infusn. (mm.) | Change (%) |
| 3 | 5.5 | 14.5 | +163.6 | 10.5 | 12.5 | +19.0 |
| 9 | - | - | - | 4.0 | 1.5 | -62.5 |

N.B. With dog 1 the sound was present only during rapid infusion.

Table 44. Effect of venous occlusion and of rapid intravenous infusion on the amplitude of vibrations at the time of the first heart sound in dogs: stethoscopic phonocardiography from right ventricle

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Infusn. (mm.) | Change (%) |
| 1 | 14.5 | 7.0 | -51.7 | - | - | - |
| 2 | 13.0 | 13.0 | 0.0 | 13.0 | 17.0 | +30.8 |
| 3 | 22.0 | 19.0 | -13.6 | 22.0 | 20.0 | - 9.1 |
| 4 | 11.0 | 10.0 | - 9.1 | 11.0 | 16.0 | +45.5 |
| 5 | 13.0 | 5.0 | -61.5 | 22.0 | 22.0 | 0.0 |
| 6 | 16.0 | 14.0 | -12.5 | 17.0 | 14.0 | -17.6 |
| 7 | 13.5 | 12.0 | -11.1 | 13.0 | 15.0 | +15.4 |
| 8 | 14.0 | 3.0 | -78.6 | 12.0 | 15.0 | +25.0 |
| 9 | 10.5 | 6.0 | -42.9 | 10.0 | 10.0 | 0.0 |
| 10 | 10.0 | 5.0 | -50.0 | 11.0 | 9.5 | -13.6 |
| 11 | 15.0 | 3.0 | -80.0 | 15.0 | 25.0 | +66.7 |
| 12 | 14.0 | 7.5 | -46.4 | 14.0 | 12.0 | -14.3 |

N.B. The effect of rapid intravenous infusion was not studied in dog 1.

Table 45. Effect of venous occlusion and of rapid intravenous infusion on the amplitude of vibrations at the time of the second heart sound in dogs: stethoscopic phonocardiography from right ventricle

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Infusn. (mm.) | Change (%) |
| 1 | 1.5 | 0.0 | -100.0 | - | - | - |
| 2 | 5.0 | 2.5 | - 50.0 | 5.0 | 10.0 | +100.0 |
| 3 | 4.0 | 0.0 | -100.0 | 4.0 | 4.0 | 0.0 |
| 4 | 5.0 | 2.0 | - 60.0 | 5.0 | 5.0 | 0.0 |
| 5 | 3.0 | 1.0 | - 66.7 | 7.5 | 8.0 | + 6.7 |
| 6 | 5.0 | 3.5 | - 30.0 | 7.0 | 5.0 | - 28.6 |
| 7 | 3.0 | 1.5 | - 50.0 | 2.0 | 2.0 | 0.0 |
| 8 | 3.0 | 0.0 | -100.0 | 1.0 | 1.0 | 0.0 |
| 9 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| 10 | 1.0 | 1.0 | 0.0 | 2.0 | 2.5 | + 25.0 |
| 11 | 3.0 | 1.0 | - 66.7 | 4.5 | 6.0 | + 33.3 |
| 12 | 1.5 | 1.5 | 0.0 | 1.5 | 2.0 | + 66.7 |

Table 46. Effect of venous occlusion and of rapid intravenous infusion on the amplitude of vibrations at the time of the third heart sound in dogs: stethoscopic phonocardiography from right ventricle

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Infusn. (mm.) | Change (%) |
| 1 | 1.5 | 0.0 | -100.0 | - | - | - |
| 2 | 5.0 | 3.0 | - 40.0 | 5.0 | 5.5 | + 10.0 |
| 3 | 9.5 | 7.5 | - 21.1 | 9.5 | 7.0 | - 26.3 |
| 4 | 5.0 | 2.0 | - 60.0 | 5.0 | 3.5 | - 30.0 |
| 5 | 1.0 | 2.0 | +100.0 | 5.0 | 6.0 | + 20.0 |
| 6 | 3.5 | 2.5 | - 28.6 | 3.0 | 3.0 | 0.0 |
| 7 | 5.0 | 4.0 | - 20.0 | 5.0 | 5.5 | + 10.0 |
| 8 | 5.0 | 3.5 | - 30.0 | 2.0 | 3.0 | + 50.0 |
| 9 | - | - | - | - | - | - |
| 10 | 1.0 | 0.0 | -100.0 | 2.0 | 2.5 | + 25.0 |
| 11 | 2.0 | 1.0 | - 50.0 | 1.5 | 3.0 | +100.0 |
| 12 | 2.0 | 2.5 | + 25.0 | - | - | - |

N.B. Summation sound on the control records of dog 9.

No third sound on the second control record of dog 12.

Table 47. Effect of venous occlusion and of rapid intravenous infusion on the amplitude of vibrations at the time of the fourth heart sounds in dogs: stethoscopic phonocardiography from right ventricle

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Infusn. (mm.) | Change (%) |
| 1 | 6.0 | 1.5 | - 75.0 | - | - | - |
| 2 | 3.5 | 1.0 | - 71.4 | 3.5 | 6.0 | + 71.4 |
| 3 | 5.0 | 5.5 | + 10.0 | 5.0 | 2.0 | - 60.0 |
| 4 | 1.0 | 2.0 | +100.0 | 1.0 | 3.0 | +200.0 |
| 5 | 2.0 | 1.0 | - 50.0 | 4.0 | 1.5 | - 62.5 |
| 6 | 1.5 | 1.0 | - 33.3 | 1.5 | 2.0 | + 33.3 |
| 7 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| 8 | 5.0 | 1.0 | - 80.0 | 3.0 | 2.0 | - 33.3 |
| 9 | - | - | - | - | - | - |
| 10 | 8.0 | 3.0 | - 62.5 | 5.0 | 3.0 | - 40.0 |
| 11 | 7.0 | 0.0 | -100.0 | 6.0 | 4.0 | - 33.3 |
| 12 | 7.5 | 2.5 | - 66.7 | 4.0 | 6.5 | + 62.5 |

N.B. Summation sound on the control records of dog 9.

Table 48. Effect of venous occlusion and of rapid intravenous infusion on the amplitude of vibrations at the time of the first heart sound in dogs: stethoscopic phonocardiography from left ventricle

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Infusn. (mm.) | Change (%) |
| 3 | 18.0 | 9.5 | -47.2 | 18.0 | 16.1 | -10.6 |
| 5 | 23.0 | 21.5 | - 6.5 | 23.0 | 21.5 | - 6.5 |
| 6 | 15.5 | 5.0 | - 6.8 | 22.0 | 22.0 | 0.0 |
| 7 | 17.5 | 22.5 | +28.6 | 12.5 | 20.5 | +64.0 |
| 11 | 17.0 | 10.0 | -41.2 | 11.5 | 10.5 | - 8.7 |
| 12 | 15.0 | 15.0 | 0.0 | 15.0 | 16.5 | + 6.7 |

Table 49. Effect of venous occlusion and of rapid intravenous infusion on the amplitude of vibrations at the time of the second heart sound in dogs: stethoscopic phonocardiography from left ventricle

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Infusn. (mm.) | Change (%) |
| 3 | 7.5 | 4.5 | -40.0 | 7.5 | 8.0 | + 6.7 |
| 5 | 10.5 | 6.5 | +38.1 | 10.5 | 10.5 | 0.0 |
| 6 | 5.0 | 2.0 | -60.0 | 10.5 | 12.0 | + 14.3 |
| 7 | 10.5 | 1.0 | -90.5 | 12.0 | 12.0 | 0.0 |
| 11 | 6.0 | 1.0 | -83.3 | 4.5 | 3.5 | - 22.2 |
| 12 | 3.0 | 2.0 | -33.3 | 2.0 | 4.0 | +100.0 |

Table 50. Effect of venous occlusion and of rapid intravenous infusion on the amplitude of vibrations at the time of the third heart sound in dogs: stethoscopic phonocardiography from left ventricle

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Infusn. (mm.) | Change (%) |
| 3 | 7.0 | 4.0 | -42.9 | 7.0 | 4.0 | -42.9 |
| 5 | 9.0 | 7.5 | -16.7 | 9.0 | 8.5 | - 5.6 |
| 6 | 5.0 | 2.5 | -50.0 | 6.0 | 10.0 | +67.0 |
| 7 | 2.0 | 0.0 | -100.0 | 2.0 | 3.0 | +50.0 |
| 11 | 6.0 | 1.5 | -75.0 | 4.5 | 5.0 | +11.1 |

N.B.

With dog 12, the sound was present only during venous occlusion and during rapid infusion.

Table 51. Effect of venous occlusion and of rapid intravenous infusion on the amplitude of vibrations at the time of the fourth heart sound in dogs: stethoscopic phonocardiography from left ventricle

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Occlsn. (mm.) | Change (%) | Control (mm.) | Infusn. (mm.) | Change (%) |
| 3 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| 5 | 3.0 | 0.5 | -83.3 | 3.0 | 1.0 | - 67.0 |
| 6 | 2.5 | 1.5 | -40.0 | 5.0 | 2.5 | - 50.0 |
| 7 | - | - | - | 1.0 | 2.0 | +100.0 |
| 11 | 2.5 | 1.5 | -40.0 | - | - | - |
| 12 | 2.0 | 2.0 | 0.0 | 1.5 | 4.5 | +200.0 |

N.B. No fourth sound on the first control record of dog 7 or on the second control record of dog 11.

Table 52. Effect of venous occlusion and of rapid intravenous infusion on the heart rate of dogs

(heart rate from phonocardiograms from right ventricle)

| Dog No. | Effect of venous occlusion | | | Effect of rapid infusion | | |
|---|---|---|---|---|---|---|
| | Control per min. | Occlsn. per min. | Change (%) | Control per min. | Infusn. per min. | Change (%) |
| 1 | 179 | 179 | 0.0 | - | - | - |
| 2 | 189 | 189 | 0.0 | 189 | 131 | -30.7 |
| 3 | 110 | 162 | +47.3 | 110 | 100 | - 9.1 |
| 4 | 234 | 189 | -19.2 | 234 | 170 | -27.4 |
| 5 | 121 | 126 | + 4.1 | 115 | 110 | - 4.3 |
| 6 | 148 | 139 | - 6.1 | 145 | 139 | - 4.1 |
| 7 | 184 | 184 | 0.0 | 184 | 184 | 0.0 |
| 8 | 206 | 206 | 0.0 | 212 | 179 | -15.6 |
| 9 | 200 | 200 | 0.0 | 189 | 206 | + 9.0 |
| 10 | 179 | 174 | - 2.8 | 166 | 126 | -24.1 |
| 11 | 189 | 194 | + 2.6 | 189 | 162 | -14.3 |
| 12 | 142 | 126 | -11.3 | 131 | 139 | + 6.1 |

N.B. The effect of rapid intravenous infusion was not studied in dog 1.

Table 53. Effect of electrical stimulation of the right vagus on the amplitude of cardiac extra sounds in dogs: logarithmic phonocardiography from right ventricle

| Dog No. | Third sound | | Fourth sound | | Systolic sound | |
|---|---|---|---|---|---|---|
| | Control (mm.) | Stim. (mm.) | Control (mm.) | Stim. (mm.) | Control (mm.) | Stim. (mm.) |
| 8 | - | - | 4.5 | 4.5 | 2.0 | 2.0 |
| 9 | 1.5 | 1.5 | 2.5 | 3.0 | - | - |
| 10 | - | - | 6.5 | 5.5 | - | - |
| 11 | - | - | - | - | 0.0 | 2.0 |
| 12 | - | - | 2.0 | 1.5 | - | - |

Table 54. Effect of slow intravenous administration of digoxin on the amplitude of cardiac extra sounds in dogs: logarithmic phonocardiography from right ventricle

| Dog No. | Third sound | | Fourth sound | | Systolic sound | |
|---|---|---|---|---|---|---|
| | Control (mm.) | After Admin. (mm.) | Control (mm.) | After Admin. (mm.) | Control (mm.) | After Admin. (mm.) |
| 1 | - | - | 2.0 | 0.0 | - | - |
| 2 | - | - | - | - | - | - |
| 3 | - | - | 1.0 | 1.0 | - | - |
| 5 | 1.5 | 1.5 | - | - | - | - |
| 8 | - | - | 4.5 | 4.5 | - | - |

Table 55. Effect of slow intravenous administration of digoxin on the amplitude of cardiac extra sounds in dogs: logarithmic phonocardiography from right ventricle

| Dog No. | Third sound | | Fourth sound | | Systolic sound | |
|---|---|---|---|---|---|---|
| | Control (mm.) | After Admin. (mm.) | Control (mm.) | After Admin. (mm.) | Control (mm.) | After Admin. (mm.) |
| 4 | - | - | - | - | - | - |
| 6 | 0.0 | 2.0 | - | - | 0.0 | 7.0 |
| 8 | - | - | 6.0 | 6.0 | - | - |
| 9 | - | - | - | - | - | - |
| 10 | - | - | 2.5 | 2.5 | - | - |
| 12 | - | - | 2.0 | 2.0 | 0.0 | 2.0 |

# APPENDIX E

## ORIGINAL PUBLICATIONS

## List of original publications


1. Sloan, A.W. (1951). Phonocardiography. Glasg. med. J. 32, 159-174.

2. Campbell, F.W., Sloan, A.W. & Andrew, A.M. (1952). An electronic phonocardiograph employing a double-beam cathode-ray oscillograph as the recording device. Brit. Heart J. 14, 271-275.

3. Sloan, A.W. & Wishart, Mary (1952). The relationship of the physiological third heart sound to the rate of venous return of blood to the human heart. J. Physiol. 116, 7P.

4. Sloan, A.W., Campbell, F.W. & Henderson, A.S. (1952). Incidence of the physiological third heart sound. Brit. med. J. ii, 853-855.

5. Sloan, A.W. & Wishart, Mary (1953a). The effect on the human third heart sound of variations in the rate of filling of the heart. Brit. Heart J. 15, 25-28.

6. Sloan, A.W. & Wishart, Mary (1953b). A device for recording the jugular phlebogram of the dog. J. Physiol. 121, 25-27P.

7. Sloan, A.W. & Wishart, Mary (1953c). Cardiac extra sounds in the dog. J. Physiol. 122, 135-143.

8. Sloan, A.W. (1953). The phonocardiograph: development and clinical applications. Med. Ill. 7, 687-696.

9. Sloan, A.W. & Greer, J.R. (1954). Calibration of an electronic phonocardiograph. In preparation.