The use of the sphygmograph in clinical medicine / by Graham Steell.

THE SPHYGMOGRAPH
IN
CLINICAL MEDICINE

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OF THE

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BY

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1899.

F. A. MAHOMED

In Memoriam.

THE USE OF THE SPHYGMOGRAPH IN CLINICAL MEDICINE.

Chapter I.

Introductory.

In the following pages it is intended to give a short account of the practical use of the sphygmograph at the bedside or in the consulting room. The instrument (which owes most of such popularity as it enjoys in England to the eloquent advocacy of the late lamented Dr. F. A. Mahomed) is often spoken of as if it were the mere toy of faddists and an invention devoid of practical usefulness. I must protest against such opinions, which belong generally to those who have had but little experience with it.

Again, we are told that the physician's "finger" should be educated, and instrumental aid eschewed. My answer is that as an educator of the "finger" ("finger" standing, of course, for the cerebral centre that receives the impressions conveyed by the finger) the sphygmograph is *facile princeps*. This fact alone should render the instrument at least deserving of respect. To those who would trust the "finger" absolutely, I might say that I believe the most cultured "finger" will occasionally err without an appeal to instrumental aid.

Moreover, the sphygmograph gives us valuable records of the pulse at definite dates. It is idle to trust to the recollection of such and such sensations, and even the verbal description of them, written at the time of observation, gives later but a feeble representation of the *status quo antea*.

Lastly, I unhesitatingly affirm that the sphygmograph is of greatest value as an aid to treatment, of less value in prognosis, and of least value in diagnosis.

A description of the sphygmograph itself would be worse than useless in this place. The student must study the instrument with it in his hand, and learn to apply it and adjust it himself. In doing the former he must take care to turn the index on the pressure dial to zero before fixing the strap round the wrist. The actual measurement of pressure indicated on the dial is, however, hardly trustworthy for reasons that need not be discussed here. The simplest rule is to take the tracings at the degree of pressure that gives the longest sweep of the writing point. This degree of pressure will usually be found to be considerable in high-tension pulses and small in low-tension pulses.

It is difficult to define the characters of a normal pulse-tracing, inasmuch as there are physiological differences in the pulses of different individuals. One man has normally a fairly high-tension pulse, another, equally healthy, a fairly low-tension pulse, and the pulse of the same man at different times and under the influence of temporary circumstances may vary as greatly in its characters.

It is necessary for purposes of description to make use of certain terms in describing the curves of a tracing. These are very simple: (1) An imaginary line drawn through the inferior extremities of the upstrokes is called the *respiratory line*. (2) The upstroke may be said to reach from it to the apex of the curve. (3) The peak at the top of the curve or first wave is called the *percussion wave*.* (4) The wave immediately

*Drs. Roy and Adami in their well-known paper "Heart Beat and Pulse Wave," proposed to call this the *Papillary Wave*.

following is the *tidal wave.** Its degree of development is of chief importance in estimating the tension of the pulse. In high-tension pulses it is well developed and (what is perhaps of greater importance than its height) it is *sustained* or prolonged. (Fig. 1.) On the other hand in low-tension pulses, the tidal

Fig. 1.

wave is either absent or ill developed (Fig. 2), and it is never

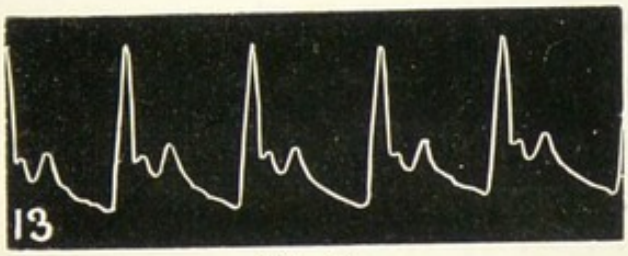


Fig. 2.

sustained or prolonged. (5) The wave that follows is the *dicrotic wave.* It is best developed in low-tension pulses, in some of which it may actually be felt with the finger as a second and feebler impulse. In low-tension pulses the tidal wave may disappear altogether so that the dicrotic wave follows the percussion wave immediately (Figs. 3, 4, 5), whether this happen

Fig. 3.

*Drs. Roy and Adami proposed to call this the *Outflow-remainder Wave*: for the reasons in favour of such change in nomenclature their paper referred to must be consulted.

Fig. 4.

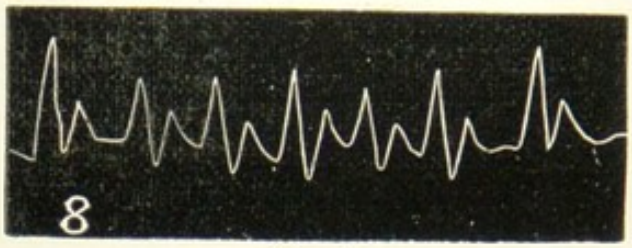


Fig. 5.

or not, there will be a notch, the "aortic" between the tidal or percussion wave, as the case may be, and the dicrotic wave. In low-tension pulses this notch is situated low down, *i.e.*, it approaches the respiratory line, while in high-tension pulses it is situated at a considerable distance above the respiratory line; in the former type of pulse the dicrotic wave is apt to be exaggerated while the opposite is the case in true high-tension pulses.

These are the points in a sphygmogram that demand our attention in studying pulse-curves. For instance: When a tracing is taken of a low-tension pulse, while the patient is suffering from severe dyspnœa, the respiratory (Fig. 3) line is often seen to become undulating instead of being straight. In obstruction of the aortic orifice the upstroke often slopes instead of being perpendicular. In the same lesion the percussion wave is often practically abolished, the top of the curve being formed by the tidal wave (Fig. 6) while in incompetence of the aortic

Fig. 6.

valves it is exaggerated. (Fig. 7.) In the high-tension pulse the tidal wave is well developed and sustained, while the

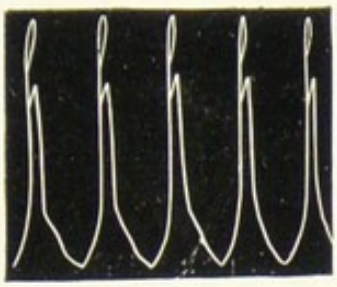


Fig. 7.*

"aortic" notch is situated at a distance from the respiratory line. (Fig. 1.) In the low tension pulse, on the other hand, the tidal wave is small or absent while the dicrotic wave is well developed, and the "aortic" notch approaches the respiratory line. (Fig. 2.) When it reaches the respiratory line the pulse is said to be *fully dicrotic* (Fig. 4), and when it actually passes below this line the pulse is said to be *hyperdicrotic.* (Fig. 5.) Very rarely the tidal wave—a peculiarly modified one, we shall see—becomes perceptible by the finger. This is called the *bisferiens* pulse, and is met with most commonly in cases of aortic obstruction. (Fig. 8.) Without the sphygmograph it

Fig. 8.

would be impossible to say in these cases which wave it is that has become so exaggerated as to simulate a second pulse-beat. In aortic incompetence the dicrotic wave is usually ill-developed or absent. (Fig. 7.)

*The loop formed at the top of this curve is a defect in the tracing, but it is not uncommon in Aortic Incompetence, and is due to the great jerk given to the lever.

We have spoken of the pulse-curves as yet as if in a given case they were all alike, but this does not always happen so that we have to consider a tracing as a series of curves the individual members of which may be very different. This introduces the important subject of *irregularity* of the pulse. (Figs. 9 and 10.) Again, the interval between a couple of

Fig. 9.

Fig. 10.

curves may be prolonged, producing *intermission* of the pulse. (Fig. 11.) The pulses may occur in couples, *bigeminal*, a large beat being followed by a small abortive one, whose curve appears in the tracing on the line of descent of the preceding curve.

Fig. 11.

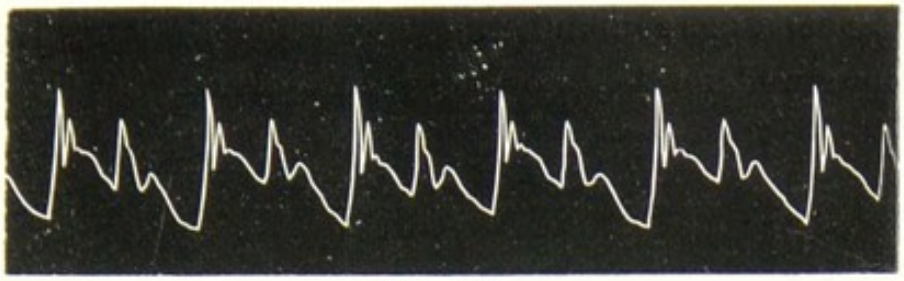


Fig. 12.

(Fig. 12.) This second beat may or may not be perceptible by the finger. In the *alternating* pulse there is a regular alternation of long and short upstrokes. (Fig. 13.)

Fig. 13.

We have, then, to consider the individual curve of the pulse-beat in detail, next the *series* of curves, and lastly we have to compare the tracings of the right and left pulses. The radial artery, which usually serves for our observation of the pulse-beat, may be irregularly distributed, only a small vessel being found in the usual situation of the "pulse"—quite unsuited for the sphygmograph—while the larger branch curves round the radius to get to its outer side. When difficulty is experienced in finding the pulse, this is the first explanation that should occur to our mind. As a routine practice we should feel both "pulses" or radials. If there is a difference it may depend on aneurysm or upon local disease—atheroma—of the subclavian artery at its origin. In aneurysm, the pulse-tracing of one side

may become a mere undulating line, while that of the other side shows well-developed ordinary pulse curves. (Figs. 14 and

Fig. 14

Fig. 15.

15.) A radial pulse may be obliterated suddenly from embolism.

Chapter II.

The Pulse in Aortic Disease of the Heart.

I use the term Aortic Disease of the Heart to designate cases of disease of the aorta implicating the heart. Aneurysms confined to the 2nd and 3rd parts and some aneurysms of the 1st part of the arch do not come under the category, and we shall consider all aneurysms of the arch together in a separate section.

The subject "aortic disease of the heart" is naturally divisible into two sections accordingly as the essential lesion is (1) Stenosis or narrowing of the aortic orifice, or (2) Incompetence of the aortic valves. The two conditions are often combined, but usually one or other is predominant with regard to its influence on the pulse. It rarely happens that the two lesions are so equally balanced that the influence of the one negatives that of the other on the pulse.

(A) *Aortic Stenosis.* There are two types of sphygmogram commonly associated with aortic stenosis, which lesion must be pronounced in order to impress special characters on the pulse. The two types in question are commonly known by the names (*a*) Anacrotic, and (*b*) Bisferiens. The former is the more common and perhaps the more characteristic, but it must not be supposed that either is pathognomonic; we are not entitled to make the diagnosis of aortic stenosis as soon as we find a characteristic example of either of the pulses in question, moreover, the tracing of any pulse that affords a tidal wave can readily be rendered anacrotic by the application of too great pressure, and this possible fallacy must be borne in mind.

The features of the (*a*) anacrotic pulse-curve may be enumer-

ated as follows:—(1) The upstroke slopes instead of being perpendicular; (2) the percussion wave is practically abolished; (3) the tidal wave forms the summit of the curve; and (4) the dicrotic wave is usually ill-developed. From a consideration of these features of the sphygmogram, a good idea may be obtained of the impression such a pulse affords on palpation with the finger. The anacrotic pulse of aortic stenosis is characteristically infrequent. The patient from whose pulse Fig. 16 was taken,

Fig. 16.

however, was subject to temporary rapid action of the heart, during which the sphygmogram lost most of it characteristic features. (Fig. 17.) At the same time the systolic thrill

Fig. 17.

usually to be felt over the aorta disappeared, while the murmur became comparatively feeble, and it is doubtful if the lesion could have been recognised clinically had the pulse remained frequent. As already indicated, diagnosis of the lesion must never be based alone upon the sphygmogram, although the latter may supply most valuable evidence. The auscultatory sign associated with the lesion is a systolic murmur, with maximum

intensity in the aortic region, well conducted in the course of the circulation. Such murmur is often accompanied by palpable thrill. The course of the aorta, as the vessel forms the great stem of the heart, from the centre of which it springs, to the aortic area (2nd rt. costal cartilage at its junction with the sternum) must be kept in mind, and a thrill need not be limited to or even best felt in the "aortic area." A systolic aortic murmur is *by itself* of little or no diagnostic value, for it is often present, when the *stenosis*—if such it can be called—is nominal only as the result of a tiny nodule on a valve, or slight thickening and stiffening of a valve, or when the orifice of the aorta is actually larger than in health, though the current of blood spreads out as it passes into the still wider channel of the vessel beyond in cases of dilated atheromatous aorta. Lastly,

Fig. 18.

a systolic murmur, though such a murmur is usually best developed in the "pulmonary area," is commonly enough heard in the "aortic area" independently of any lesion, in cases of simple anæmia. It follows that the diagnostic value of a systolic murmur by itself in the aortic area is exceedingly small. A thrill accompanying the murmur, and due to the coarseness of its vibrations, adds some degree of diagnostic value to the latter. In actual stenosis of the aortic orifice the 2nd sound, however, is usually defective, and may be absent altogether, and when this is the case, and at the same time the systolic murmur is very prolonged and accompanied by thrill, while the pulse is

anacrotic and slow, a good basis for the diagnosis of aortic stenosis is afforded. A history of rheumatism in long past years offers contributory aid in diagnosis, but the lesion is not always due to rheumatic endo-carditis and may be congenital. In some cases it is produced by the chronic inflammatory and degenerative process known as aortitis deformans. In most cases of aortic stenosis there is some amount of regurgitation, but the murmur due to the latter is usually feeble. In a few cases there is neither 2nd sound nor diastolic murmur audible: a loud prolonged harsh systolic murmur (accompanied by thrill) being all that is to be heard in the aortic area—a significant auscultatory condition. In the cases of dilated aorta, above referred to, the systolic murmur may be of great loudness, and possibly accompanied by thrill, but the 2nd sound is markedly accentuated.

(*b*) The bisferiens pulse is characterised by (1) a well-marked percussion wave, (2) an elevated but unsustained tidal wave, and (3) usually an ill-developed dicrotic wave. (Figs. 19, 20, 21.)

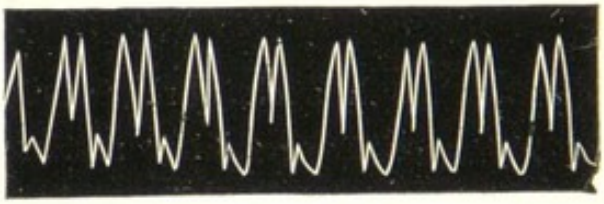


Fig. 19.

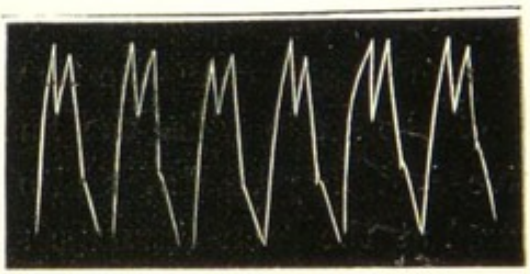


Fig. 20.

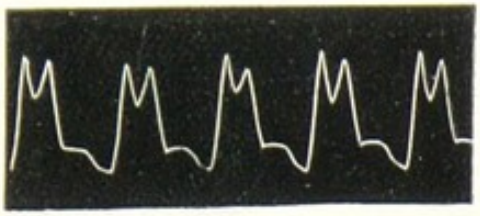


Fig. 21.

This last is the only character it has in common with the anacrotic pulse. It often happens that the percussion and tidal waves can be felt separately with the finger, in the same way as the percussion and dicrotic waves may become separately perceptible in certain low tension "dicrotic" pulses. The impression received through the finger is very similar in the two cases, so much so that without the sphygmograph one could not identify the nature of the second beat. In certain cases of aortic incompetence the sphygmogram may closely resemble that of an ill-developed bisferiens pulse although there is no stenosis. (Fig. 22.) A thrill in the aortic region accompanying

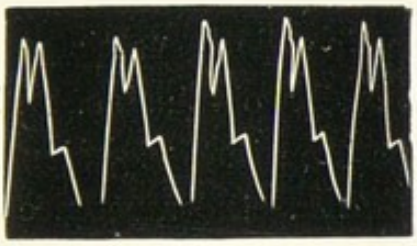


Fig. 22.

the systolic murmur is of the same diagnostic value as in cases with anacrotic pulse. A very remarkable fact, with reference to the bisferiens pulse, is its occasional development in one radial only. I have looked in vain for an anatomical explanation of the fact in fatal cases.

When the two lesions aortic stenosis and aortic incompetence are present together, and neither is predominant, the effects of

the one lesion neutralise those of the other on the pulse, so that the pulse curve is not characteristic of either. Fig. 23 was

Fig. 23.

taken from a young patient who had a marked systolic thrill and loud systolic murmur as well as a diastolic murmur in the aortic region.

With regard to the bisferiens pulse, the cleft of the curve between the percussion and tidal waves, to be of diagnostic value, must be very deep. Minor degrees of such character of the curve must not be allowed diagnostic value in favour of stenosis, and are common enough in cases of aortic incompetence without stenosis. (Fig. 22.) I have already given a similar warning concerning the anacrotic pulse, pseudo-anacrotic curves being only too easily obtainable. The diagnosis of the case must never be based on the pulse alone.

(B). *Aortic Incompetence.* Incompetence of the aortic valves may be brought about by several pathological processes which may be enumerated as follows:—1. Rheumatic and (rarely) choreic endocarditis; 2. Septic endocarditis; 3. The chronic inflammatory process that results from excessive strain on the valves and ends in their shrinking; 4. The chronic inflammatory and degenerative process known as "aortitis deformans" or atheroma. This leads to aortic incompetence in either or both of two ways: (*a*) by diminishing the elasticity of the aorta, whereby the vessel becomes permanently dilated, the dilatation

of the channel of the vessel being ultimately followed by dilatation of the orifice, so that the valves are rendered incompetent even though sound in themselves, and (*b*) direct implication of the valves in the chronic inflammatory process of the vessel wall. Syphilis produces aortic incompetence by a process similar to 4.

Rupture of an aortic cusp by violence is so rare an event that we may leave it out of account, and the incompetence that is associated with aneurysm involving the first part of the arch is of the same nature as that referred to under 4*a*.

It may be said that rheumatic endocarditis prefers the mitral to the aortic valves for its attack, and although it frequently produces aortic incompetence, the mitral lesion usually overshadows the aortic in the production of symptoms, but the total number of cases in which aortic incompetence is the essential lesion is well made up by the cases I term *degenerative* in type, inasmuch as they are met with in patients who have passed the prime of life, or in younger subjects who may be regarded as prematurely aged as the result of the action of syphilis on their vascular system. Aortitis not rarely leads to attacks of angina pectoris and interference with the nutrition of the heart-muscle by narrowing the coronary orifices. (Fig. 25.)

We must keep before our minds, then, the association of aortic incompetence with disease of the endocardium and with disease of the aortic wall. It was at one time supposed that the type of disease which had produced the valve lesion in a given case could be recognised by the sphygmogram. This is not so, unless it be in the early stage of cases referred to under 4*a*, in which, however the degree of incompetence may be believed to be small, so small as to fail to impress its special characters on the tracing (Fig. 24). A valuable sign of dilatation of the arch of the

aorta is accentuation of the 2nd Sound, and when such accentuation is pronounced we should listen frequently and carefully for the faint diastolic murmur that denotes commencing leakage. Commonly this murmur, when it first develops, is not persistent but comes and goes, under various circumstances, for a time before it is permanently established. Then, there is the well attested fact that even when the lesion is great and the regurgitation free, the cardiac diastolic murmur may be lost for a time, even when the diagnosis can be easily made from the visible pulsation of the larger arteries, and the diastolic murmur elicited by pressure with the stethoscope over the femoral artery. This may happen in acute febrile disease or intense disturbance of the circulation such as is often present for a day or two after severe aortic cases have been admitted to hospital. It is easy in such cases to predict the return of the diastolic murmur provided the patient rallies.

There is no lesion that so rapidly produces dilatation of the left ventricle as aortic incompetence. Its effects fall directly upon the ventricle when it is relaxed, and dilatation of the cavity ensues. Hypertrophy of the heart-muscle can only come to stay such dilatation and to enable the ventricle to empty itself of its excessive contents. The first change resulting from the lesion must be dilatation—that unmixed cardiac evil : till it has occurred hypertrophy has no place.

Supposing that hypertrophy of the left ventricle has occurred, and that the ventricle is able to complete its systole, an abnormally large quantity of blood is shot into the arterial system. Is it surprising that an unusually long upstroke should be the rule in aortic incompetence? More than this the blood pressure in the arteries during the diastole of the heart has been running down with most abnormal rapidity, there being a

backward as well as forward vent during the *arterial* systole. What wonder that the percussion wave should be exaggerated and the tidal wave unsustained?

The notch preceding the dicrotic wave is commonly known as the "aortic" notch, and marks the closure of the semilunar valves—the end of systole and the beginning of diastole. The dicrotic wave may be regarded as the result of the rebound from these valves. But if the valves are incompetent and leak, such rebound must be deficient. Hence we find that the dicrotic wave is diminished in aortic incompetence, and in cases of very free regurgitation, in which the valves are practically destroyed, the dicrotic wave is with difficulty discerned, or absent. (Figs. 27, 28, 29, 33.)

The peculiarity of the pulse in aortic incompetence may be said to consist in the great difference between the maximum and minimum blood pressure and the rapidity of transition from the one to the other. One moment the pulse is "full" the next "empty." This character of the pulse, first described by Sir Dominic Corrigan, of Dublin, is intensified if the patient's wrist is raised above his head while the finger is on the radial. Again, if the forearm is grasped with the hand the pulsation of the radial and ulnar arteries can be easily felt. For inspection the larger arteries should be chosen, as the carotids and subclavians, for in them the remarkable jerkiness of the beat is most characteristic, but the smaller vessels are often seen to pulsate with extraordinary distinctness, as, for instance, the palmar arch.

In conclusion: the characters given to the sphygmogram by aortic incompetence are a long upstroke, an exaggerated percussion wave, an unsustained tidal wave, and a deficient dicrotic wave.

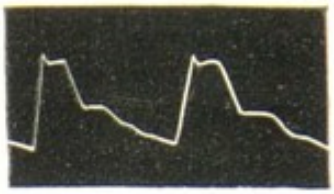
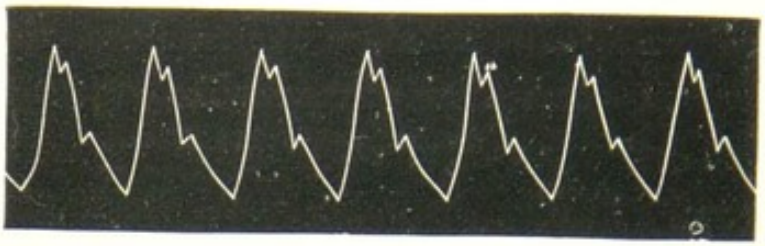


Fig. 24



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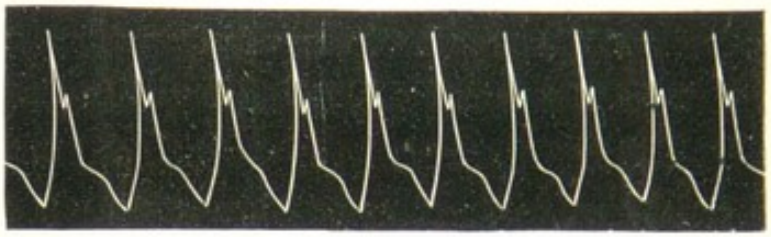
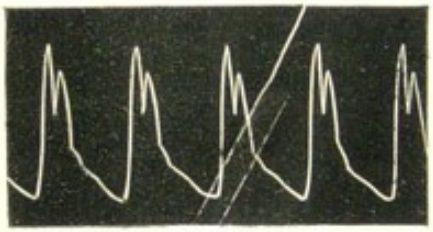


Fig. 26.



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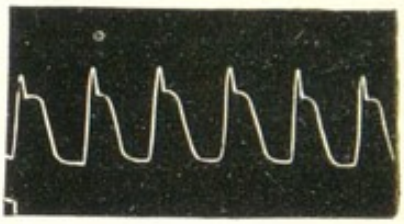


Fig. 28.

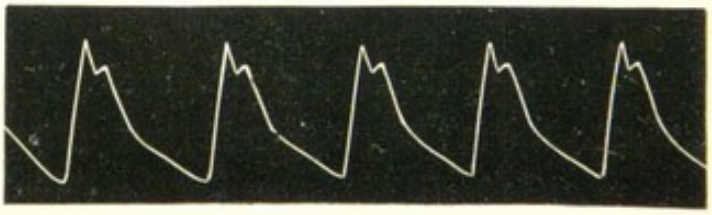


Fig. 29.

Fig. 30

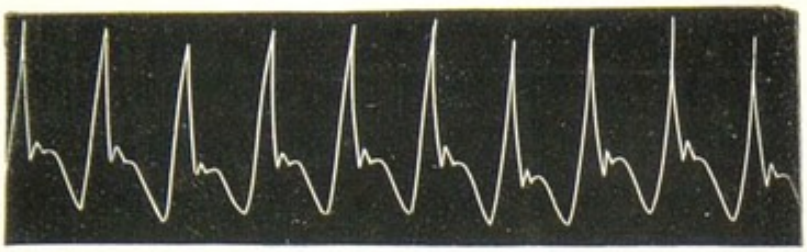
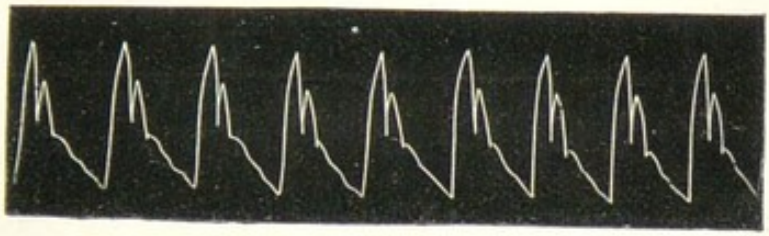


Fig. 31.



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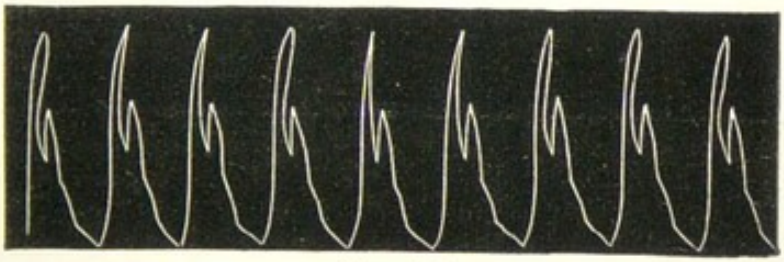
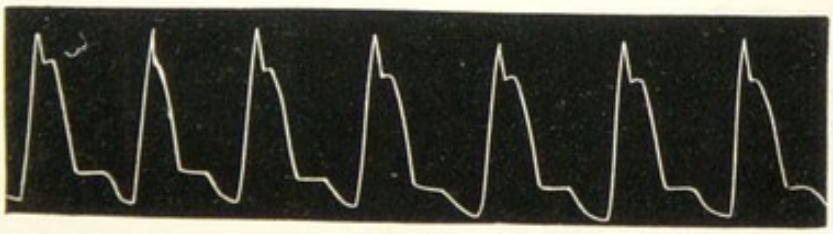


Fig. 33.



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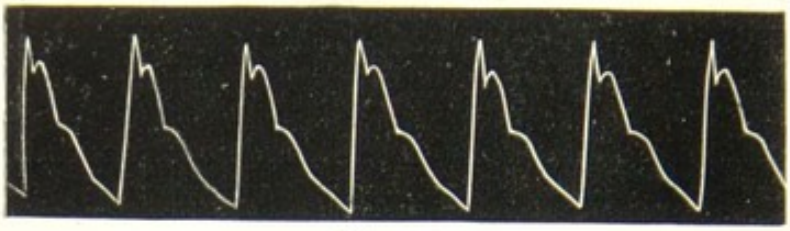


Fig. 35.

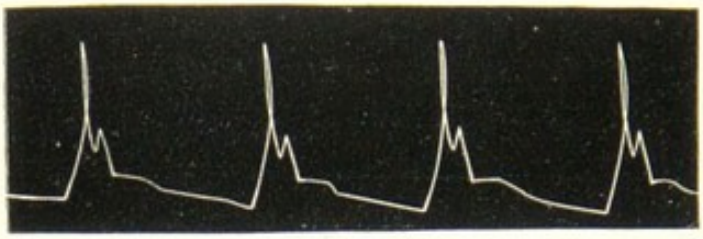


Fig. 36.

Fig. 37.

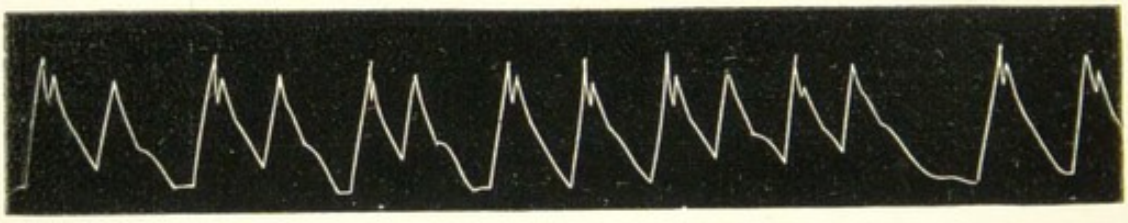


Fig. 38.

Fig. 24. Aortic dilatation, with incompetence of valves. Fig. 25. Syphilitic Aortitis, slight incompetence of valves, and angina pectoris from narrowing of coronary orifices. Female æt. 27. Fig. 26. Exaggerated percussion wave, small tidal wave, and defective dicrotic wave. Rheumatic case æt. 35. Figs. 37 and 38. Showing irregularity—inequality and intermission—in case of "degenerative" type of Aortic Incompetence.

CHAPTER III.

The Pulse in Mitral Stenosis.

Mitral Stenosis may be regarded as the great mitral *lesion*. No doubt endocarditis—rheumatic, and especially septic—may so damage the valve-curtains that they can no longer completely close the auriculo-mutricular orifice even with the help of a sound heart-muscle, but in the great majority, if not in all such cases, it is impossible to guarantee the functional integrity of the heart-muscle under the circumstances, while we know that failure of the heart-muscle, quite independently of its cause, which may be important and permanent or unimportant and transient, can render the valves, though perfectly healthy in themselves, incompetent. In the next section I hope to demonstrate that there is no pulse that has any claim worthy of consideration to be called the pulse of mitral incompetence, as was at one time supposed. When the mitral orifice is stenosed it goes without saying that the valve-curtains are deformed, and it is only likely that they should, either occasionally or permanently, leak. And so we find that the murmur most commonly present in cases of mitral stenosis is the murmur of mitral incompetence, which *directly* has nothing to do with the essential morbid condition present, namely, the *stenosis*.

In the early stages of the case—not of the *lesion*—the sphygmogram that is usually found associated with mitral stenosis is that of a regular pulse of good, one might almost say high, tension showing a well-developed tidal wave, and in fact nothing *per se* abnormal—a pulse compatible with good, nay, robust health. This is the pulse that is usually associated with the best development of the most characteristic ausculta-

tory sign of the lesion, namely, the presystolic murmur, but there is no necessary association between the two conditions, and I have found the presystolic murmur present with a very low tension pulse.

What may be termed the second stage of the pulse in mitral stenosis is that most characteristic of the lesion, but one must rid one's mind of any idea of its being *pathognomonic* of the lesion. A precisely similar pulse is commonly present in cases of simple muscle-failure of the heart. What I term the second stage pulse of mitral stenosis is characterised by irregularity and inequality of the curves. We see side by side and in all degrees of preponderance of one or other, high-tension and low-tension curves, the latter being generally more or less abortive, or ill-developed in magnitude at any rate. A series of such ill-developed curves is often interrupted by a well-developed curve of the first stage, or the opposite condition may be met with : that is to say, a long series of regular high-tension curves is rarely interrupted by an abortive low-tension curve. If any pulse is entitled to be called the pulse of mitral stenosis, it is this "second stage," or irregular pulse, but, as I have already stated, a precisely similar pulse occurs in cases of simple muscle-failure of the heart, without any *valve-lesion* whatsoever.

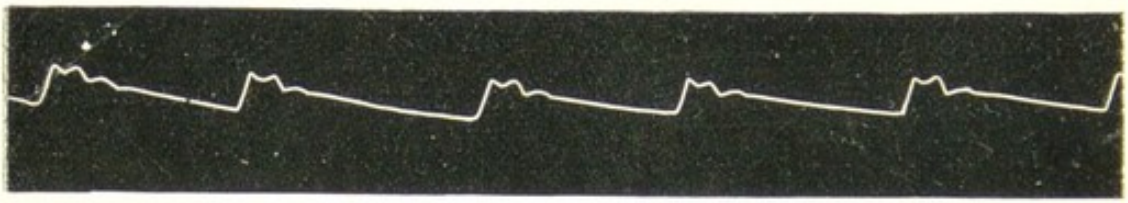
What I term the third stage pulse is simply a low-tension pulse, and generally regular.

Now it is most important that the three kinds of pulses I have described as if they were phases in the clinical evolution of mitral stenosis, should not receive a degree of prognostic importance to which they have no title. Speaking very generally, it is the rule for the first and second stages to be represented, and the same may be said of the third stage if observations are made up to the end of the case. But a patient

quite early in the evolution of the case may have a low-tension —third stage—pulse, and yet be in no danger as far as his heart is concerned. Often there will be an evident cause for what we may call the "abnormal" condition, such as the presence of fever, or there may be disturbed innervation of the heart, such as produces temporary tachycardia. Again, unfortunately, the retention of a regular high-tension pulse does not of necessity justify a good prognosis. Lastly, in some cases the second stage pulse is never observed. Far better that I should never have mentioned *stages* in the description of the pulse in mitral stenosis than have led—misled—my readers to an erroneous belief fraught with the risk of erroneous prognosis. Nevertheless, the three kinds of pulse described are usually present in the course of cases of mitral stenosis, and in the sequence indicated, and to a limited extent prognostic value cannot be denied to them. Prognosis, however, any more than diagnosis, can never safely be based on one feature of a case. A comprehensive view of all the features presented is as necessary in the making of a prognosis as of a diagnosis.

As most of my readers know, towards the end of a case of mitral stenosis the direct murmurs—the presystolic perhaps invariably, the diastolic frequently—disappear for a longer or shorter period before the patient's death, and the only mitral murmur present is a systolic one, in the majority of cases not conducted to the back but there replaced by the first sound, whether or not the first sound is present with the murmur at the apex. A precisely similar auscultatory condition is met with in that class of case which of all others resembles cases of mitral stenosis, namely, simple muscle-failure of the heart. A man may have had rheumatic fever in early manhood, and escaped severe endocarditis and mitral stenosis, and in the late period

of life become the subject of muscle-failure (which may be regarded as almost physiological). He may present at the cardiac apex just such a systolic murmur as I have described in cases of mitral stenosis, and his pulse may be typically the second stage pulse of this lesion, while the usual symptoms of disturbed circulation—venous stasis—are present. Under such circumstances the history of the acute rheumatic attack is apt to be a stumbling block for the physician, but the rule should be to regard the case as a simple "muscle" one (senile) unless there is a distinct history of cardiac disability in early middle age or even before this. I have seen the two conditions—mitral stenosis and muscle-failure—in several instances as it were, independently combined, the kidneys at the same time having undergone a certain amount of granular degeneration—so common in the old.



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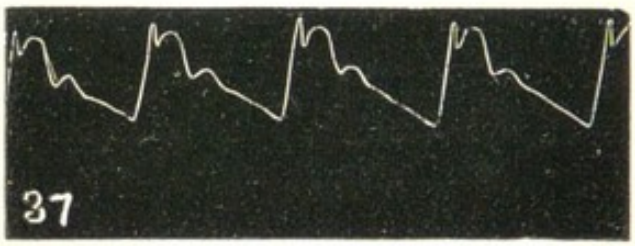

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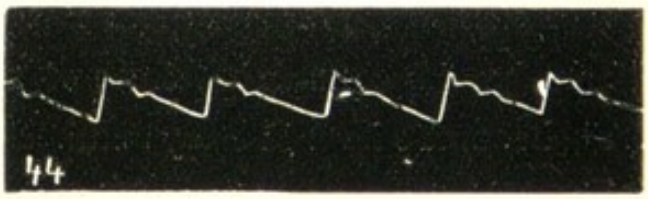

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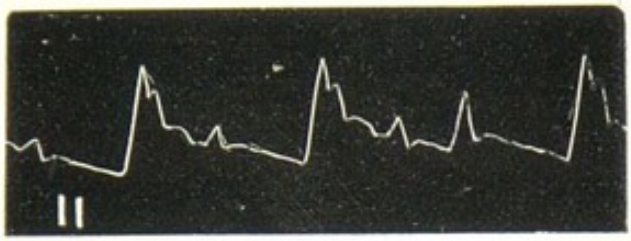

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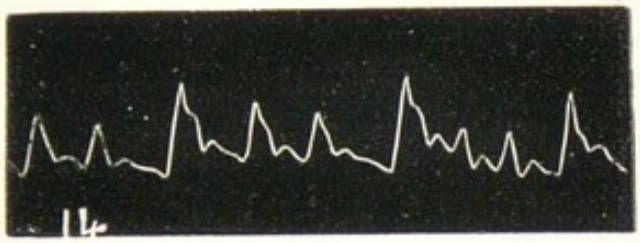

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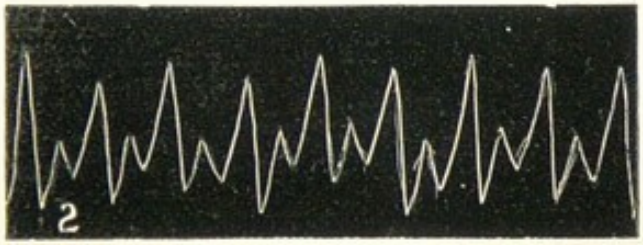

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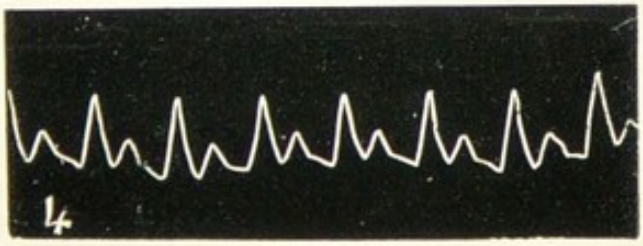

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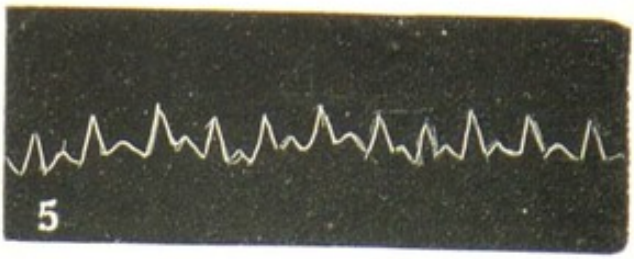

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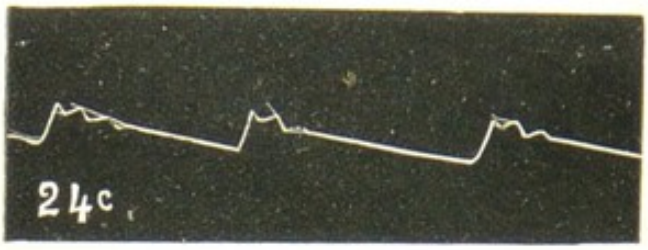

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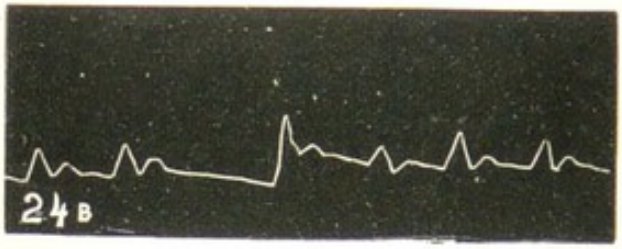

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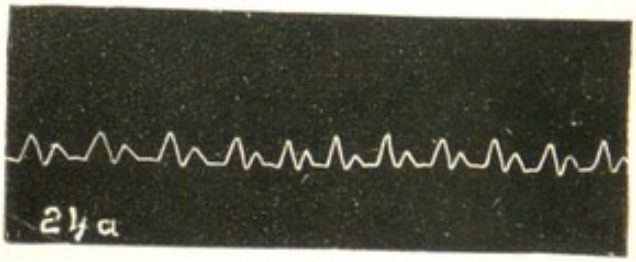

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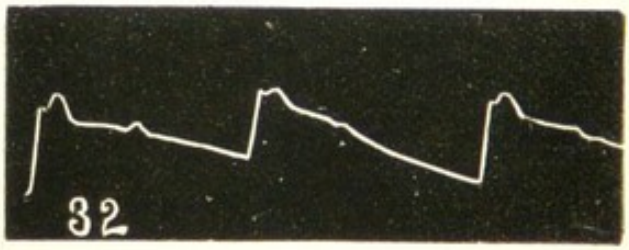

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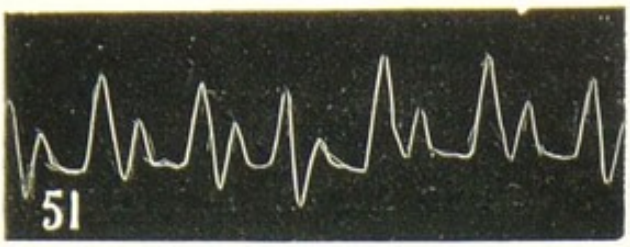


Fig. 52.

Fig. 53.

Figs. 39, 40, 41. "First stage" pulses in mitral stenosis.

Figs. 42, 43, 44 "Second stage" pulses in mitral stenosis.

Figs. 45, 46, 47. "Third stage" pulses in mitral stenosis.

Figs. 48, 49, 50. Show three stages of pulse occurring in the course of same case.

Fig. 51. Bigeminal pulse in mitral stenosis. Heart and vessels at time under influence of Digitalis.

Figs. 52, 53. Third stage and second (almost first) stage pulses. The latter (53) taken during convalescence from symptoms of "venous stasis.

Chapter IV.

The Pulse in Mitral Incompetence.

Our views with regard to the pulse in cases of mitral regurgitation have undergone considerable modification during the last quarter of a century. Notwithstanding statements by high authority to the contrary, I affirm without hesitation that there is no type of pulse entitled to be termed "the pulse of mitral incompetence." The fact is that incompetence of the mitral valves is met with under a great variety of conditions having little or nothing in common apart from the mitral incompetence.

It must be borne in mind that *mitral incompetence frequently occurs without there being any disease of the valves whatsoever.* To appreciate the fact one has to bear in mind that the mitral valves, for the perfect fulfilment of their function, require the co-operation of the contraction of the heart muscle. In two ways at least the muscle of the heart is complemental in the valve apparatus. Its contraction diminishes the size of the orifice the valves have to close, and sustains the curtains in action by shortening the musculi papillares. Long ago physiologists were acquainted with what they termed "the safety valve action of the tricuspid valves" on the right side of the heart, by which term was understood the fact that under strain the tricuspid valves permitted a certain amount of regurgitation of blood, and that the occurrence was for the immediate benefit of the organism and the temporary relief of the ventricle. We now know that a similar occurrence is common on the left side of the heart also, and that under the influence of high arterial tension the mitral valves frequently leak, although sound in structure and supported by a

practically sound—even hypertrophied—heart muscle. There is a degree of arterial pressure in face of which the heart-muscle, however vigorous, cannot be expected to maintain its perfect function. Moreover, considering the case as a whole, there seems to be a limit to the continuance of hypertrophy when established. As we found that it is the heart-muscle that alone renders possible the full function of the auriculo-ventricular valves on both sides of the heart, it follows that muscle-failure of the heart renders incompetence of the valves likely. Whether we increase the arterial tension or weaken the heart-muscle—the driving force—the result will naturally be much the same. We cannot therefore be surprised to find that a patient with a manifestly strong heart and very high arterial tension may suffer from most distressing breathlessness—the first cardinal symptom of heart failure—in the same way as the patient with a debilitated heart and low arterial tension may do. In the former case the burden is too grievous even for a vigorous heart; in the latter case, though the burden is light speaking generally, it is no less than in the former case too great for the heart thas has to bear it.

In discussing the subject of mitral incompetence as the result of muscle-failure, it is impossible to omit reference to that other, and, in my opinion, more important condition usually involved, in greater or less degree, in muscle-failure, namely, the *non-completion of systole—asystole or systole catalectic.* We know now that this condition, in which the ventricle fails to *empty itself in the normal manner, is not limited to a short period preceding the patient's death, but may be a chronic condition lasting for years, now better, now worse, or a transient one, passing away with recovered power and energy on the part of the heart-muscle.

*The supra-papillary space contains blood physiologically at the end of systole.

I think I can convince my readers of the recoverableness of this, once supposed most lethal, condition by reference to a common type of sphygmogram. A "missed beat" occurs, either a complete intermission or a wretched abortive curve utterly unlike its fellows, then comes an upstroke of exaggerated length to form a curve that is a giant among the rest. The obvious explanation of this occurrence in a case of simple muscle-failure without murmur surely is that the left ventricle has become distended during the intermission and prolonged diastole even though this be interrupted by an attempt at systole. The ventricle, however, is roused to supreme effort by the distension it suffers and succeeds in completing, or nearly so, its systole, throwing, at the same time, of course, an abnormally large amount of blood into the aorta. It is impossible, in considering mitral incompetence, to ignore the cases of muscle-failure of the heart which are accompanied by all the disturbance of the circulation, characteristic of heart-failure resulting from or accompanied by valve defect, but which never develop a murmur, and therefore presumably mitral incompetence. Such cases are not very rare, and the obvious explanation of the disturbance of the circulation in them is that the muscle-failure is manifested alone by the production of systole catalectic. Why the auriculo-ventricular valves do not become incompetent in these cases I do not know. On the other hand, it is probable that in many, if not most of the cases of mitral incompetence from muscle-failure the incompetence is associated with systole catalectic in some degree. Of a *silent* mitral regurgitation we have no knowledge.

Cases of mitral incompetence fall naturally into groups.

(1) Cases in which the valve-structures are themselves damaged by rheumatic, choreic, or septic endocarditis in such degree that their function is impaired.

(2) Cases of mitral stenosis, with the necessary deformity in some degree of the valve curtains. These cases were considered in last section, and need not be further referred to. The essential condition is the stenosis.

(3) The great group of cases of simple muscle-failure, in considering which it is necessary to bear in mind (*a*) the work to be done by the heart-muscle, and (*b*) the capability of the heart-muscle to perform it.

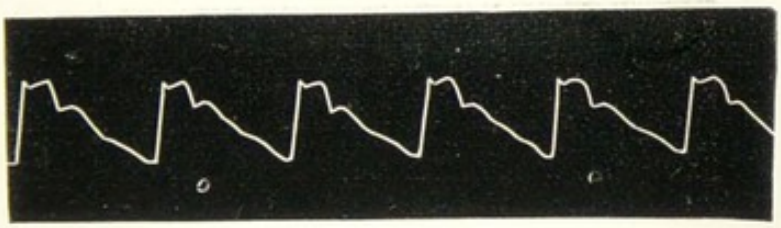
(1) Five-and-twenty years ago rheumatic crippling of the mitral valves, without stenosis of the orifice, was supposed to be a common cause of mitral incompetence in such degree as seriously to interfere with the circulation. The majority of cases of severe rheumatic endocarditis end in mitral stenosis; in the remainder the disturbance of the circulation that may follow cannot be attributed alone to the damage of the valves, inasmuch as such damage is associated with other changes quite as likely to have caused the disturbance of the circulation. Among these changes are to be found adherent pericardium and an evidently impaired heart-muscle irrespective of the valve-lesion. The importance of adherent pericardium has probably been over-rated, for the condition is not very rarely found with a sound muscle. Of late, on the other hand, we have been forced to recognise the fact that rheumatism can exert an injurious influence upon the myocardium itself, and I am inclined to think that the evident muscle-failure and dilatation, found post-mortem, have been too often attributed to an associated but altogether trifling valve-lesion. Many years ago, when I had not been long Resident Medical Officer in the Manchester Royal Infirmary, my belief in the importance of the changes wrought by rheumatic endocarditis as the cause of mitral incompetence and consequent disturbance of the circulation, received a severe

shake by a P.M. examination of a rheumatic patient who had shown the ordinary clinical signs of mitral incompetence from valve lesion. The examination revealed practically sound mitral curtains, with a markedly dilated heart, the patient being a woman in early middle age, in whom the ordinary causes of simple muscle-failure, such as anæmia, Bright's disease, alcoholism, &c., were absent. Subsequent experience has only served to emphasise the lesson I received from this case. Before writing this paper, however, I asked Dr. Kelynack to be so good as to look over his records of P.M. examinations of my cases, and to state his impressions received therefrom for quotation here. He writes: "At your suggestion I have gone over the P.M. notes of your cases for the past few years. It is remarkable that out of the very large number of cardiac cases examined—a very considerable number being examples of mitral stenosis—I have hardly been able to select a single case of what I might call straightforward mitral incompetence from mitral lesion pure and simple. From the list of cases I have sent you, you will have noticed that several were to a great extent 'muscle' rather than 'valve' cases."

It seems quite clear that rheumatic endocarditis can no longer maintain the high place formerly accorded to it as a cause of mitral incompetence without stenosis. The latter is, as I have said, the great rheumatic mitral *lesion*, although incompetence of the valve in some degree is so often in subordinate association with it.

Septic endocarditis no doubt is abundantly capable of so damaging the mitral curtains that they must become not only impaired in function but practically useless. Septic endocarditis, however, is a disease so terrible in its other results that this one becomes of quite minor importance.

(3) Our last group of cases owe their mitral incompetence to a great variety of pathological states which have little or nothing in common except the element of muscle-failure of the heart, and consequent mitral incompetence. The nutrition of the heart-muscle may be interfered with by anæmia—one of the most commonly met with examples of this group —or by atheroma of the aorta implicating and narrowing the coronary orifices—or by excessive work, as in conditions associated with high arterial tension, such as Bright's disease, gout, and plumbism. Severe fever, as typhus, exerts a most debilitating effect upon the heart-muscle, as was long ago shown by Stokes, whose classical description of the auscultatory signs present must be read later, and no doubt the results of such a degree of weakening of the heart-muscle would be more permanent were it not that fever is a most potent reducer of arterial tension, thus lightening the load the heart has to bear during its debility. In Bright's disease, however, there may be anæmia and high arterial tension in combination for a time, the result often being rapid dilatation of the heart. Of course, as the heart fails, the tendency is towards lowering of the arterial tension.



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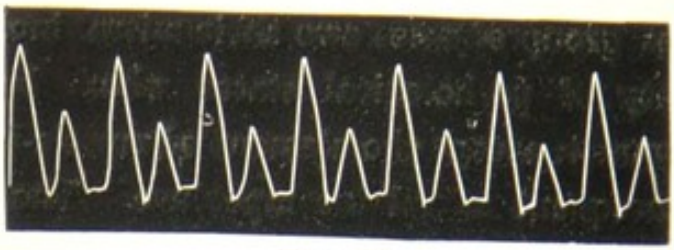

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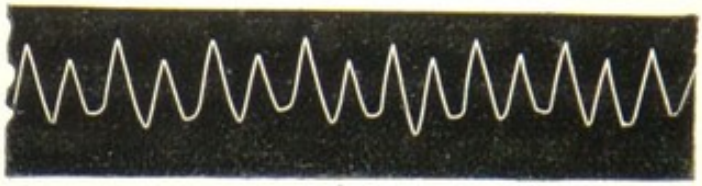

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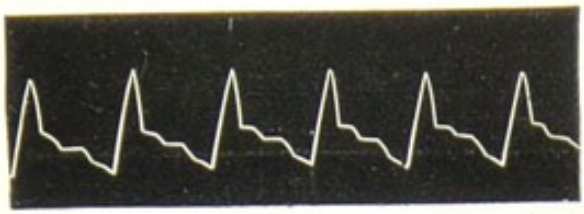

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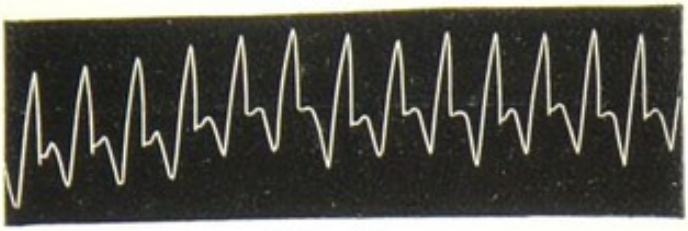

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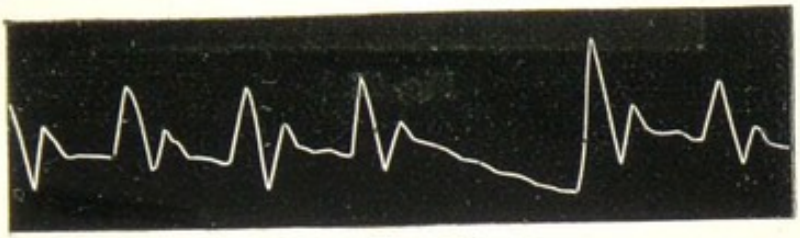

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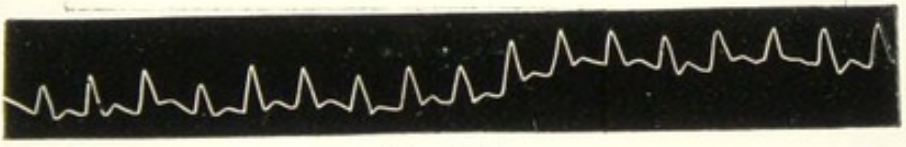

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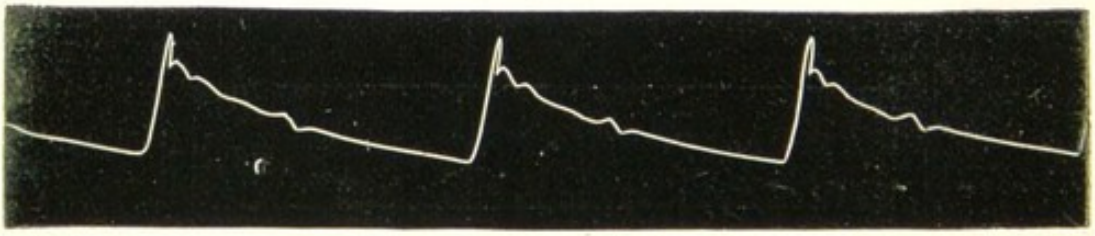


Fig. 74.

Fig. 75.

Fig. 76.

Fig. 77.

Fig. 54. High tension pulse in Bright's disease: mitral incompetence. Male aet. 38.

Fig. 55. High tension pulse in Pernicious Anæmia: mitral incompetence. P.M. Male aet. 43.

Figs. 56 and 57. Low tension pulse in case of Septic Endocarditis: mitral incompetence—pyrexia and progressive heart failure. 57. Dicrotic wave nearly as high as percussion wave. P.M.

Figs 58, 59, 60. Alcoholic muscle-failure of heart: mitral incompetence—gradual recovery of tension during convalescence.

Figs. 61 and 62. Low tension: alcoholic muscle-failure of heart: mitral incompetence. (62) Lowering of tension towards death.

Fig. 63. Common type of pulse-curve in simple anæmia (Chlorosis). Mitral incompetence. Well-marked percussion wave; small tidal wave.

Fig. 64. Low tension pulse of unusual form—in case of rheumatic valve-lesion with mitral incompetence but without stenosis. Boy aet. 14 pericardium adherent. P.M.

Fig. 65. Showing "missed beat" in case of muscle-failure with systole catalectic and mitral incompetence. P.M.

Fig. 66. Bigeminal pulse. Case of alcoholic muscle-failure: mitral incompetence. P.M.

Figs. 67—76. Showing changes in pulse during the course of the muscle-failure of Bright's Disease (granular kidney P.M.). Patient was nearly nine years under observation. When tracing Fig. 67 (lowest tension) was taken there was no mitral incompetence, which developed later: no doubt there was systole catalectic. Figs. 67—73 were taken during patient's first stay in hospital, and show progressive recovery of tension. Fig. 74 shows the pulse to be bigeminal, probably due to digitalis. Figs. 75 and 76 show irregularity of the pulse, developed only late in the case. Patient usually came into Manchester Royal Infirmary with mitral incompetence, the murmur disappearing as his heart regained strength. Towards the end it became persistent.

Fig. 77. Low tension: rheumatic endocarditis without stenosis: adherent pericardium. Rheumatic fever a year before. P.M. Man aet. 25.

Chapter V.

Irregularity and Intermission of the Pulse, &c.

Irregularity of the pulse occurs in a great many conditions. It may be met with in cases of pericarditis, myocarditis, and endocarditis, although these diseases are more usually accompanied by a regular pulse. More frequently it occurs in cases of disturbed innervation of the heart and of degenerative changes in the heart-muscle, which are often the result of chronic arteritis of the coronary vessels. As instances of the first variety may be mentioned, the irregularity often experienced by excessive smokers, and that observed in cases of tubercular meningitis. Irregularity of the pulse may be due, again, to various toxic agents absorbed from the alimentary canal and the glands of the body generally or to various nervous impressions received from other organs acting upon the heart in a reflex manner. In the complicated cardiac nervous system the influence of the pneumogastric nerves is probably most often at work in the production of irregular and intermittent pulses. In some animals, as in dogs, the pulse is often found to be irregular although they are in perfect health and have excellent "wind." Patients who have habitually an irregular pulse may acquire a regular pulse during pyrexial while those with habitually a regular pulse may acquire an irregular pulse under the same condition. This latter occurrence is very common in the old and in those with degenerated vessels and heart-muscle. Lastly, certain individuals apparently in perfect health have always an irregular pulse, like most dogs. I know several such individuals who lead very active lives before the public and who yet have

habitually irregular pulses. Figs. 80, 81, and 82 are tracings from the pulses of such individuals.

The simplest form of irregularity may be considered to be occasional intermission. Usually a beat is omitted—the so-called missed beat—but the sphygmogram may indicate an attempt at systole on the part of the ventricle which is imperceptible by palpation of the radials. Most healthy individuals at one time or another have had experience of the sensation in the chest produced by the momentary halt of the heart's beats and by the throb of the beat that follows such halt. I have already commented on the state of the left ventricle during the intermission—a state probably highly dangerous to the sufferer from aortic incompetence. In one case of the kind, I was particularly struck by the duration of the intermissions, and the patient died suddenly a few hours later.

Irregular pulses have been already described as common in cases of mitral stenosis and of muscle failure of the heart, and it has been shown that tracings from such pulses in the two conditions are indistinguishable. It may be said that the two elements of the tracing of this type constitute the irregular pulse in general, but their degree of development and their grouping vary indefinitely and a tracing often shows both irregularity and intermission. The term "inequality" is used by some writers to describe the different size of the curves, but such difference in size is often associated with a difference in type as well—the large curves tending towards the high tension type, the small ones towards the low tension type. There may be a definite arrangement of the irregular beats, so that the pulse is "regularly irregular," if one may so say: this has been called "rhythmic irregularity." The commonest type is the "bigeminal pulse," the occurrence of which is so

often promoted by Digitalis, although it is common enough independently of any drug. It is peculiar to no one lesion and frequently occurs in simple muscle cases. One of the examples, Fig. 83, was taken from the pulse of a gentleman, whom I had passed for assurance at the ordinary rate a few months previously, his pulse and heart being then apparently quite normal. Subsequent experience has shown that the attacks of this peculiar derangement are, in his case, quite transitory, although they cause him considerable discomfort for the time. Possibly they are of toxic origin, as the patient is comparatively young: about 40. Not long ago I took a tracing of the pulse of a chlorotic girl, that is a perfect specimen of the bigeminal pulse. In this case, likewise, the condition was of short duration, and the muscle-failure of the heart seemed to be no more than is common in pronounced cases of anæmia.

I have met with the trigeminal pulse in one case only—a case of simple muscle-failure in a man over middle-age. (Fig. 84.)

Fig. 85 represents a pulse occurring in groups of four beats (pulsus quadri-geminus), each group being separated by short intermissions, but it will be seen that these groups of four are very different from the bigeminal and trigeminal groups of two and three, inasmuch as the curves that compose the groups of four are of equal magnitude, while in the other cases they diminish onwards. I should regard this pulse rather as an example of intermission occurring at regular short intervals.

It is often impossible to recognise any order in the arrangement of the unequal curves of irregular pulses, "arhythmic irregularity," individual curves of most different type and magnitude being huddled together in the wildest disorder. For such condition the fanciful term "delirium cordis" has been used. (Fig. 89.)

The Pulse in Aneurysm. It is common to find one radial pulse diminished in intra-thoracic aneurysm. The usual explanation given is the interposition of the elastic sac between the heart and the pulse, but there are obviously other ways in which such an aneurysm may interfere with the pulse of one side, as by the pressure of the sac on the outside of the left sub-clavian trunk. Moreover, as already pointed out, local disease—atheroma—of the origin of the subclavian may interfere with the pulse on one side quite independently of any aneurysm. In rare cases of intra-thoracic aneurysm both radial pulses may be abolished, although the circulation and nutrition are well maintained in the parts. Such a case came under my observation a few years ago, and a similar case is described in Dr. Fagge's "Practice of Medicine."* The usual sphygmogram of a radial pulse interfered with by an intra-thoracic aneurysm is characterised by a sloping up-stroke and down-stroke and rounded top, the different waves becoming obliterated, till finally the tracing is represented by a merely undulating line. (Fig. 101.) It is evident that the attainment of the maximum height of the curve will be delayed on the affected side.

Bradycardia, or slow pulse, is sometimes an individual peculiarity. Napoleon the First, it is recorded, was the subject of bradycardia. No better evidence of the fact that bradycardia need exert no injurious effect upon the nutrition of the brain could be adduced. I think I have read of Napoleon that he had a singular power of going to sleep again after being aroused in the night. His brain seemed to spring at once into its normal marvellous activity, and its function for the time having been discharged it would pass almost at once into repose again. If

*p. 107, Vol. II, 3rd Edition.

this were so, one cannot help associating the faculty with the slow pulse. Fig. 99 represents the bradycardiac pulse in a case recently in the wards:—pulse 30 to 40. This patient, like many sufferers from bradycardia, was subject to syncopal attacks or faints as they are popularly termed, but the relation of such to epileptic seizures is a close one. Occasionally in severe fever, as typhus, a bradycardiac pulse is met with, and in diphtheria it is a very dangerous condition. It is common in jaundice.

Tachycardia, or frequent pulse, occurs in many circumstances. An attack of "palpitation" may usually be regarded as an attack of tachycardia, but they are not equivalent terms. A patient with tachycardia may be quite unaware of the condition while a patient with a quite moderate frequency of his pulse may be distressingly aware of his heart's beats. Fig. 91 represents the pulse of a patient with diabetes, who experienced a short attack of tachycardia—rate 200 per minute. His normal pulse is represented in Fig. 92. In diphtheria a condition of tachycardia as well as bradycardia may be met with, and the one is of as bad prognosis as the other.

There is no pulse that is characteristic of *Pericarditis*. A great degree of irregularity in severe cases is not rare (Fig. 78), while the accompanying fever exerts its usual influence in lowering tension. Dry *pericarditis* is common in granular disease of the kidney with enlarged heart, and under the circumstances there may be little or no pyrexia, while the pulse shows no material departure from *its* normal.

The typical pulse-tracing of *pyrexia* (Fig. 100) is simply a low-tension one, often showing well marked dicrotism, the dicrotic wave being so well developed that it often becomes perceptible to the finger. As already stated, pyrexia is a powerful reducer of arterial tension. I think one of the most hyperdicrotic pulses, of which I ever took a sphygmogram,

occurred in a man with granular disease of the kidneys who was brought into the Infirmary apoplectic from cerebral hæmorrhage. As the coma deepened his temperature rose to a great height, and he had no doubt paralysis of his vaso-motor controlling nerve-centre, resulting in dilatation of the arterioles throughout the body. In all probability this patient had had habitual high arterial tension before the cerebral hæmorrhage occurred. In such cases heart failure is often the less of two evils, as it diminishes the risk of apoplexy in the presence of habitual peripheral resistance in the vessels and high tension. A great clinicist went so far on this account as to regard the failing heart of advanced years as a "conservative lesion." The irregularity and intermittence and certain rarities of the pulse in fevers have already been referred to.

When Dr. Bright first described the disease that bears his name he recognised the cardiac hypertrophy that is so marked a feature of most chronic cases, but perhaps he hardly dwelt sufficiently upon the termination of the hypertrophic condition he described. In most cases the period of hypertrophy is a limited one, and sooner or later the mass of muscle tends to fail in vigour, so that all the common indications of muscle-failure of the heart are apt to arise, and in this relation it is necessary to bear in mind the vascular tension with which the heart has to cope. But I have sufficiently in the foregoing chapter referred to the matter. Here I wish only to point out that cases vary greatly as to the duration of the stage in which the heart-muscle remains vigorous and the sphygmogram one of high tension, that the state of hypertrophy may never be established, that dilatation may be rapidly induced with lowering of arterial tension, and lastly that not all cases of Bright's Disease—not even of the granular form—are associated with circulatory disturbance.

I have already described the pulse of simple *anæmia*—the exaggerated percussion wave, small tidal wave, and well-marked dicrotic wave of its sphygmogram (Fig. 63)—also the pulse I have observed in several cases of pernicious anæmia. (Fig. 55.) But lest I should lead anyone into error by seeming to imply that I regard the latter pulse as pathognomonic, I add in the present section the tracing of a patient with this grave disease taken towards the end of the case when the heart had greatly failed. (Fig. 95.) I had no means of ascertaining the state of the pulse in the earlier stages of the disease.

Influence of inspiration (especially deep) on certain pulses:—This occurs in striking development in cases of Mediastinitis a rare disease that is met with specially in children. My colleague, Dr. Harris, has kindly permitted me to make use of one of the sphygmograms contained in his well-known monograph on the subject. (Fig. 96.) During inspiration it will be noticed that the pulse-curves become markedly diminished. A less degree of the same phenomenon may occasionally be seen in cases of (probably) free mitral regurgitation and Sir Wm. Broadbent has suggested that in these latter cases the regurgitation is freer during the act of inspiration so as to diminish the output of the left ventricle into the arterial system. In the former cases there is probably interference with the great arteries during inspiration owing to the adhesions and matting together about them.

I show illustrations of the pulse in Cheyne-Stokes respiration. (Figs. 97 and 98.) In these it will be seen that the arterial tension rises during the dyspnœal period. The accompanying phenomena of Cheyne-Stokes respiration seem to vary in detail. In one case the pulse was slowed in so marked a manner during the dyspnœal period that the respiration became more frequent than it.

I have been unable to demonstrate the rise of blood-pressure that is alleged to be the determining cause of attacks of angina pectoris. It does not follow necessarily that because vaso-dilators relieve such seizures that rise of pressure is the cause of the attacks. The heart is undoubtedly embarrassed in action in seizures of the kind, and any relief afforded to it by vaso-dilators would no doubt be beneficial. It has always seemed to me, in discussions on angina pectoris in relation to a rise of arterial pressure as its immediate cause, that the fact of the pain itself being capable of acting as a vaso-constrictor has been too much overlooked. Certainly morphia is the great remedy that one has usually to fall back upon when vaso-dilators have spent their powers of affording relief.

There are few conditions of disease in which a sphygmogram cannot be taken, but it is open to question if the time and labour spent on proving the truth of the rule: "there is no *pulse* of which a tracing cannot be taken," are fairly compensated seeing that the result is likely to be of only very moderate value. In such circumstances it may be legitimate to take the tracing while the radial is compressed below, a proceeding which does not fundamentally alter the character of the tracing, that is to say: high-tension curves remain high-tension curves and low-tension curves remain low-tension curves.

A few months ago in a case of heart-failure in Bright's disease with distressing dyspnœa the pulse could only be *felt* with difficulty, but a carefully-taken tracing showed a pronounced tidal wave.

Again, the walls of the radial arteries may be so profoundly changed in structure that the vessel feels like a row of beads owing to calcareous deposition and in such a case sphygmography is inapplicable.

I possess a large number of sphygmograms from cases of various diseases not directly associated with the circulation: the tracings have often shown characters that one would not have expected them to show. The subject, however, is too large a one to enter upon here and it would besides be out of place. I do not think, however, that many varieties of pulse-curve will be found to have escaped reference entirely. After all, the whole subject of sphygmography is composed of simple elements. These simple elements, their more important modifications and combinations in the single curve and in the series of curves that constitutes a pulse-tracing, I have attempted to sketch only. The study of the subject in detail must be made at the bedside. "La vérité est dans les choses."

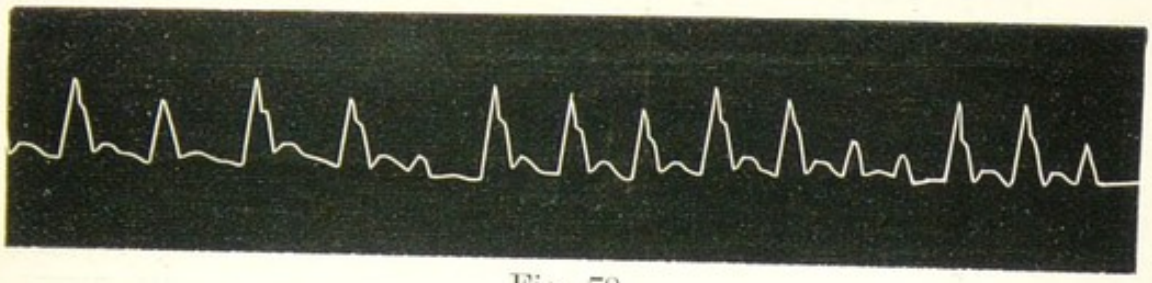


Fig. 78.

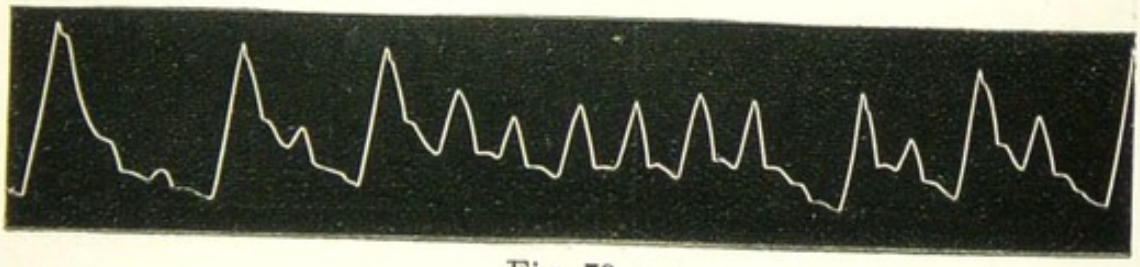


Fig. 79.

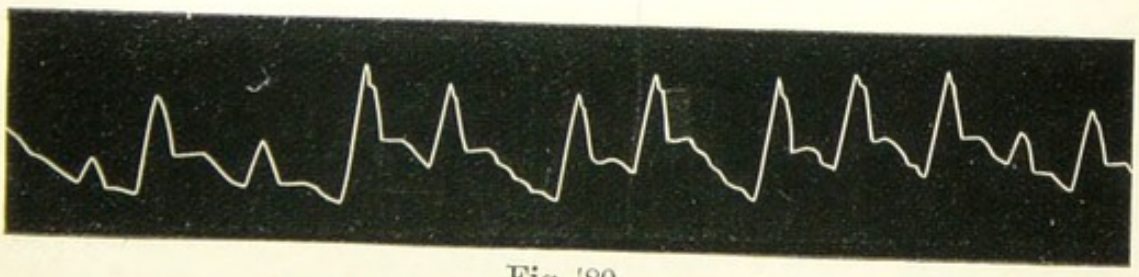
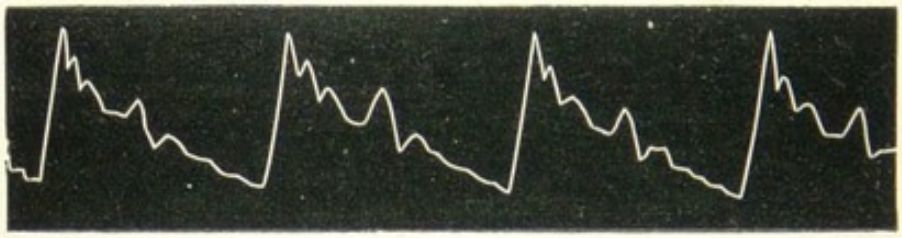


Fig. 80.

Fig. 81.

Fig. 82.



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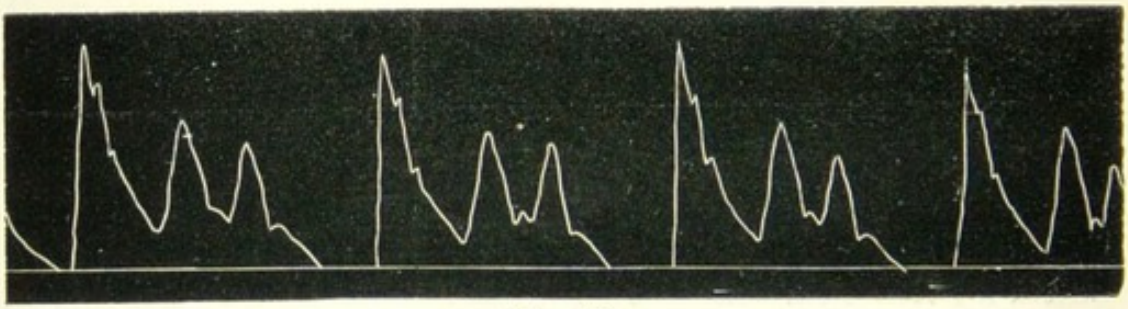
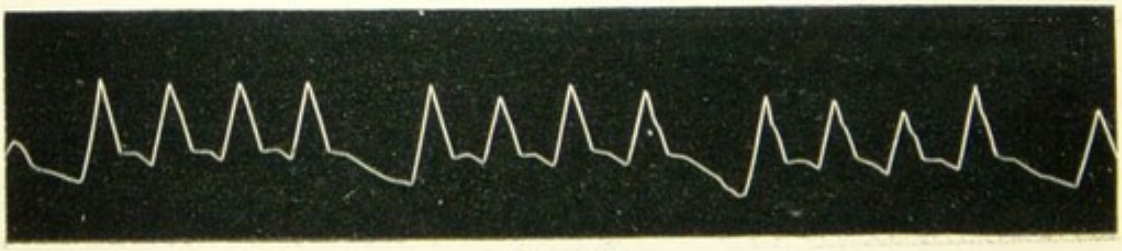


Fig. 84.



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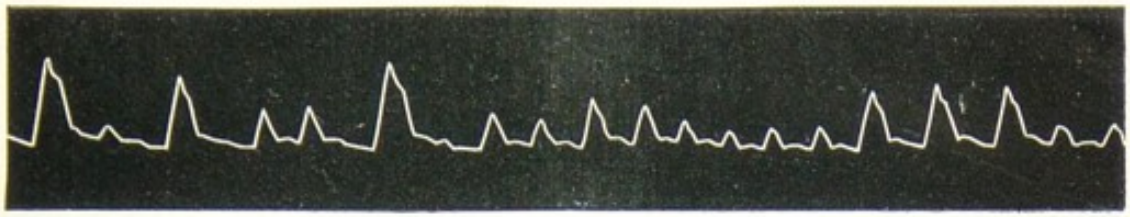


Fig. 86.

Fig. 87.

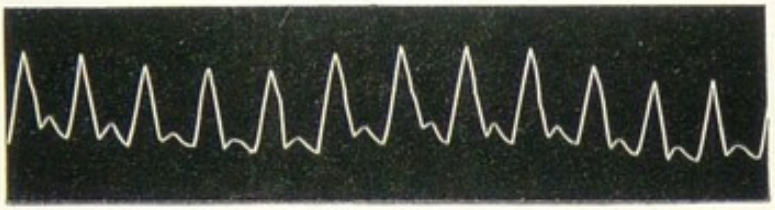


Fig 88.

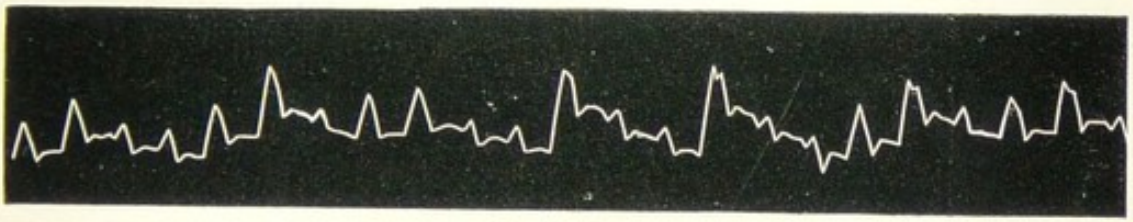
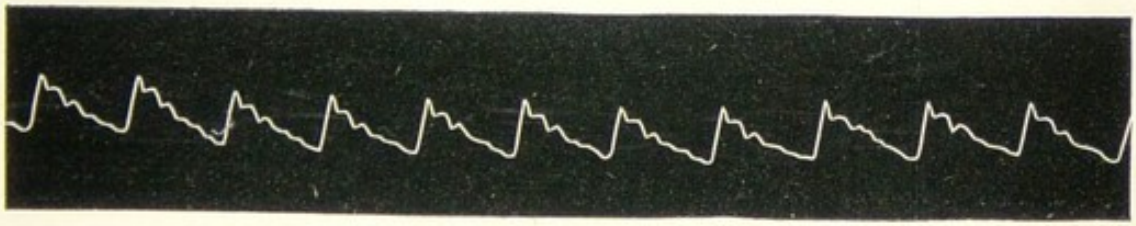


Fig. 89



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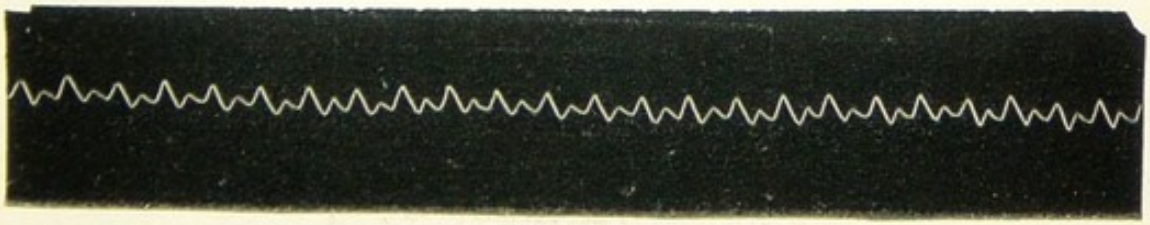


Fig. 91.

Fig. 92.

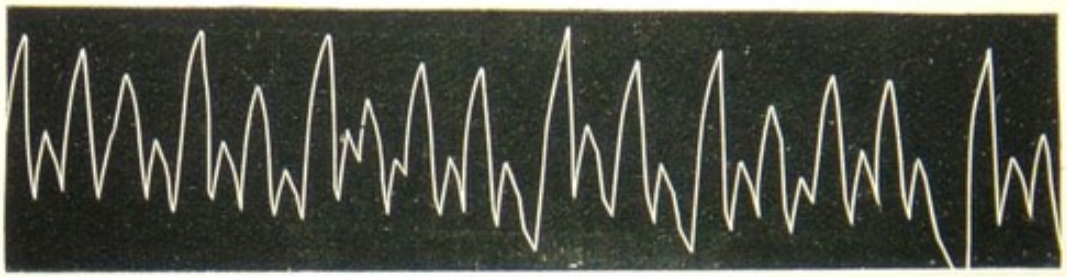
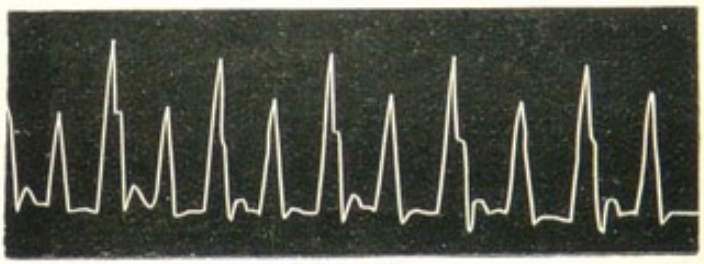


Fig. 93.



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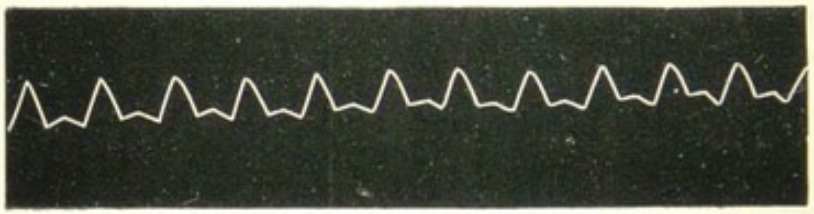

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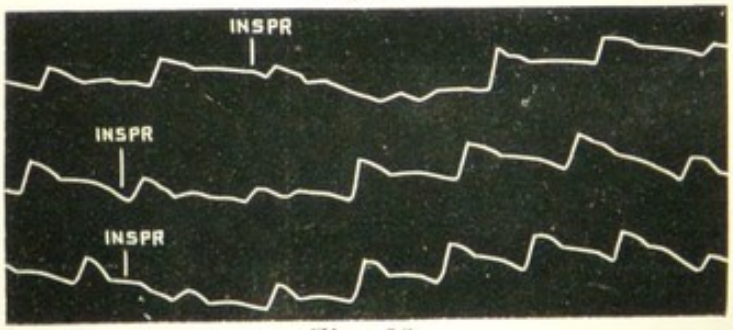


Fig. 96.

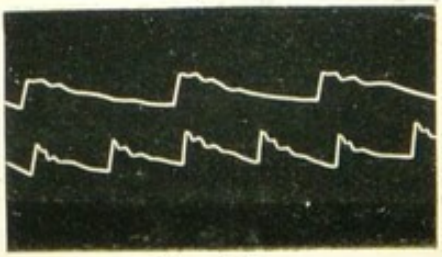
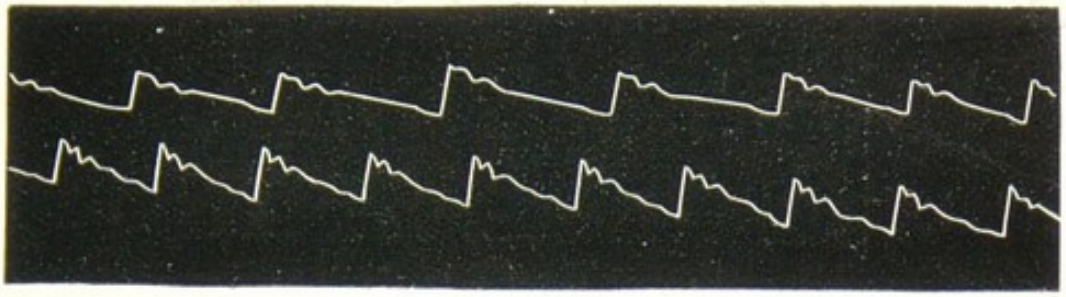


Fig. 97.



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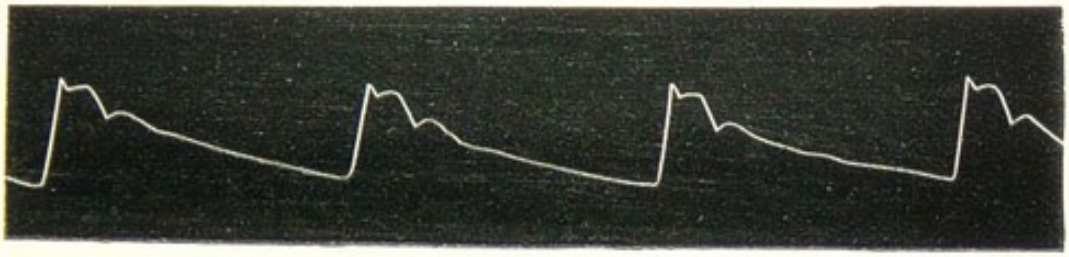


Fig. 99.

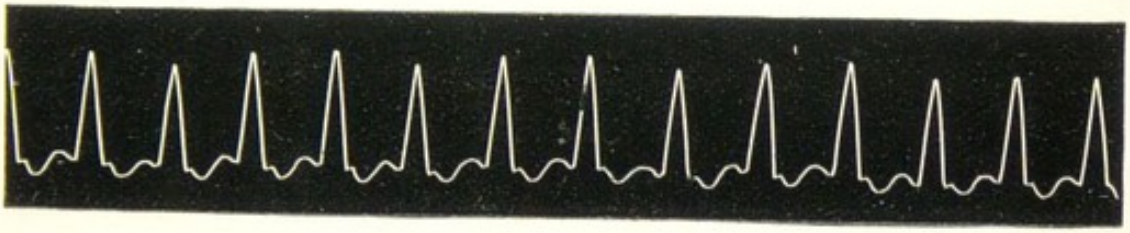
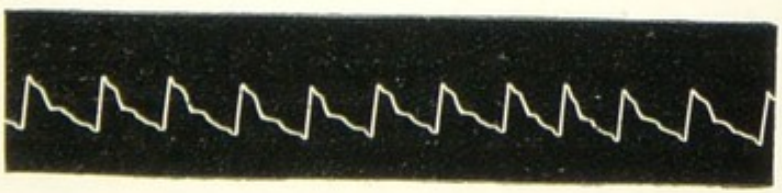


Fig. 100.

Fig. 101.



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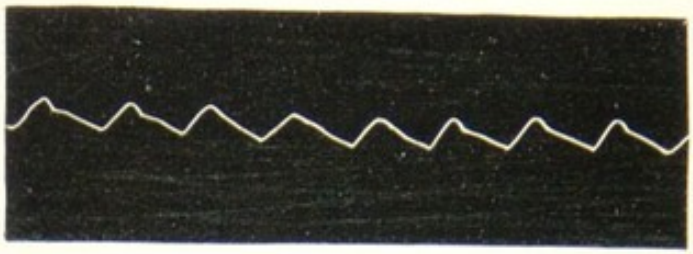

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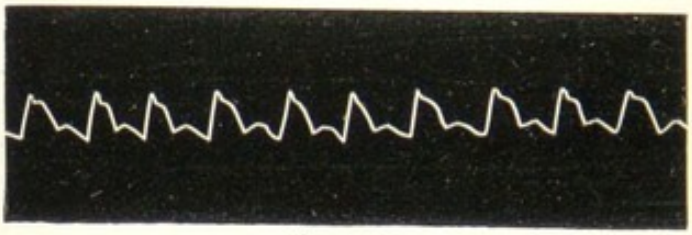

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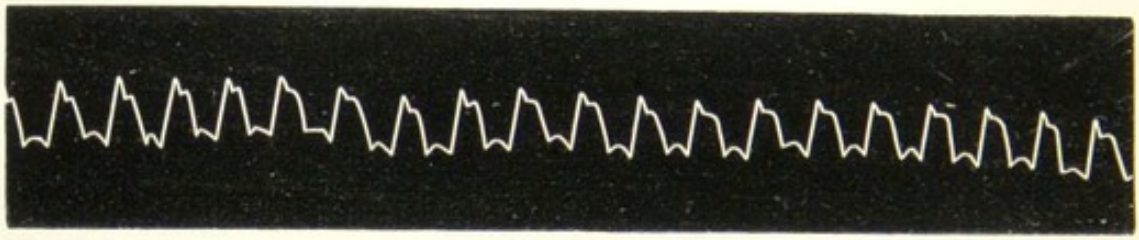

Fig. 105.

Fig. 78. Irregular pulse in case of pericarditis.

Fig. 79. Irregular pulse in case of senile muscle-failure of heart with slight mitral incompetence.

Figs. 80, 81, and 82. Irregular pulses in *apparently* quite healthy active individuals.

Fig. 83. Bigeminal pulse: temporary condition in an *apparently* sound man. Case referred to in text.

Fig. 84. Trigeminal pulse in man with muscle-failure of his heart.

Fig. 85. Pulse-beats occurring in groups of four. Case of rheumatic mitral disease in young woman.

Figs. 86, 87, and 88. From case of muscle-failure due to alcoholism, showing at different times during patient's stay in Hospital, an irregular pulse, a regular pulse of good tension, and a low tension pulse during tachycardia—160 per minute. The transition from a condition amounting to "delirium cordis" to a normal state as in Fig. 87 was often very abrupt, so that in quarter of an hour Figs. 86 and 87 might have been taken.

Figs. 89 and 90. From case of "Gouty Kidney" (granular) with heart-ure. Showing temporary irregular pulse and the normal pulse of the patient.

Figs. 91 and 92. From a case of Diabetes. Showing transient tachycardia and normal pulse of individual.

Fig. 93. From case of mitral and aortic disease with much enlargement of heart in a young man. Tracing shows the up-strokes commencing before the dicrotic wave is completed. Sir William Broadbent has called attention to this feature.

Fig. 94. Alternating pulse in a case of senile muscle-failure.

Fig. 95. Low tension pulse from case of pernicious anæmia with advanced heart-failure.

Fig. 96. Pulse in Mediastinitis. From Dr. Harris's work on that disease.

Figs. 97 and 98. Tracings taken during the apnœal and dyspnœal periods of Cheyne-Stokes respiration in a case of "Granular Kidney" with heart-failure. During the latter period the pulse is slowed and of higher tension (the upper tracing).

Fig. 99. Tracing of pulse in case of Bradycardia referred to in text.

Fig. 100. Low tension (febrile) pulse in Enteric Fever.

Fig. 101. Aneurysmal pulse.

Fig. 102. Tracing of unaffected radial pulse of same patient.

Fig. 103. Aneurysmal pulse.

Fig. 104. Tracing of unaffected radial of same patient.

Fig. 105. Pulse in Graves' Disease (exophthalmic Goitre).