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The Pulse

W. H. Broadbent, M.D.

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CLINICAL MANUALS

FOR

PRACTITIONERS AND STUDENTS OF MEDICINE.

THE PULSE.

BY

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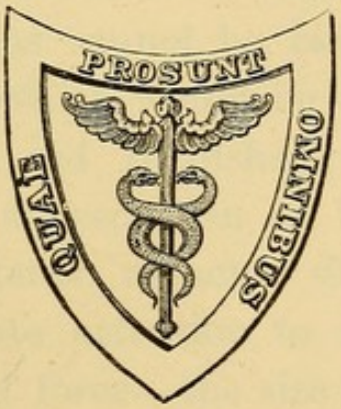
ILLUSTRATED WITH 50 SPHYGMOGRAPHIC TRACINGS.



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PREFACE.

This little book is, for the most part, a reproduction of the Croonian Lectures on the Pulse delivered before the College of Physicians in 1887, with some amplifications and additions—with the addition, in particular, of a chapter on the Sounds of the Heart, which must always be taken into consideration if the full significance of variations in the character of the pulse is to be estimated.

More, probably, will be expected than is here found with regard to the pulse and its indications in different forms and at various stages of acute disease. But such indications cannot be laid down in words with the precision which would be required to make them useful. The aid furnished in the following pages towards an appreciation of the variations of the pulse in the course of acute disease will consist in directing separate attention to other points than mere frequency and force—the size of the artery and the character and duration of the individual beats and intervals by which the experience and observation, which are absolutely essential, will be guided and rendered more fruitful.

An apology is necessary for the absence of reference to and due acknowledgment of the work of others who have contributed to our knowledge of

the pulse. Had this been attempted, the production of this book, small and imperfect as it is, would have been impossible; and it may be said that while no opportunity of gaining knowledge or insight by reading has been neglected, the subject has been worked out by personal research and observation.

I should be wanting in gratitude if I did not take this opportunity of acknowledging my indebtedness to my late teacher and colleague, Dr. Francis Sibson, under whom, more than thirty years ago, I entered upon the serious study of the problems of the circulation. His patient investigation and re-investigation of every point, experimental or clinical, his unwearied efforts for the attainment of minute accuracy, his complete subordination of theory and preconceived opinion to observation, were a lesson to me then, and have remained impressed on my mind ever since as an example which has had a determining influence on my thought and work.

W. H. BROADBENT.

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THE PULSE.

CHAPTER I.

HISTORY.

THERE can have been no true comprehension of the significance of the pulse, or intelligent appreciation of the relation between its variations and the physiological or pathological conditions associated therewith, until the discovery of the circulation of the blood by Harvey, which, it will be remembered, was only announced in 1628. It was long after this, indeed, before the physiology of the circulation began to influence the ideas of physicians and to enter into the consideration of disease; and anything like an adequate knowledge of the physics of the circulation, of the rate of movement of the blood, or of the varying degree of pressure within the vessels, and of the effects of such pressure, is of quite modern acquisition Many of the indications to be obtained from the pulse, however, are matters of simple observation, and are in a great measure independent of a knowledge of the physiology of the circulation; the frequency and force of the pulse in fever, for example, were perfectly well known, and their indications as to the actual condition of the sufferer and as to the probable course and issue of the disease were well understood, when the most erroneous notions of the cause of the arterial beat were entertained. It may be doubted, indeed, whether even now the medical man consciously refers to the movement of the blood while estimating the

significance of varying states of the pulse, and does not rather infer directly from the pulse the severity of the fever, or the degree of general weakness, or what not. For this reason, therefore, and because it is always interesting to trace the course of ideas and the progress of knowledge in any subject, the history of the pulse will be briefly followed; and it may be stated at once that, although I had devoted much time to this many years since, I here take advantage of the complete and admirable account given by Dr. Ch. Ozanam in his recent work.*

The earliest idea of the pulse appears to have been that the arteries were filled, not with blood, but with a vital air or spirit, the movements of which, independent in every part of the body and independent of the heart, gave rise to the pulsations. The pulse in the temples, for example, was supposed to be independent of, and to have a different signification from, that at the wrist. The veins were believed to carry the blood into every part of the body, and to have their origin in the liver, which was the great formative source of the blood.

It was then discovered that the pulse was everywhere synchronous, and later that it coincided with the beat of the heart, and was dependent upon it. Now, also, it was the dilatation of the heart and arteries which drew the vital air into all parts of the body, and no longer the throbbing of the vital spirit in the vessels which gave rise to the pulsations. The heart was not supposed to contain blood any more than the arteries, and the septum between the ventricles was believed to be perforated.

Galen's great discovery was that the heart and arteries contained blood. He did not remove the errors with regard to the perforation of the interventricular septum, or with regard to the office of

* "La Circulation et le Pouls" (Baillière et Fils, 1886).

the liver as the manufactory of blood, and of the veins as its distributors. Both in veins and arteries there was supposed to be a flux and reflux, and the dilatation of the heart and of the vessels was still supposed to be the active phase of their function, causing the pulsations and drawing the blood into them.

This view prevailed from the time of Galen, A.D. 164, to 1628, when Harvey showed not only that the blood circulated, but that the ventricular systole supplied the force which drove the blood through the arteries and caused the pulsation felt in them.

The distinction between the arteries and veins was made before the time of Hippocrates by Diogenes of Apollonia and Euryphon, but it is only in the writings of Hippocrates (400 B.C.) and of his school of successors that reference to the pulse under the names of σφυγμός and παλμός becomes frequent and definite. It appears to have been noted in the arteries generally, but it is most frequently mentioned as observed in the temples, where it was considered to be an important indication of fever. Hippocrates was the originator of the idea that the arteries did not contain blood during life, since they were always found empty after death in animals killed for sacrifice, and that they were in communication with the trachea. His observations, however, with regard to the pulse are entirely clinical and practical, and various features of the pulse are recognised and named.

Aristotle (384 B.C.) gave its name to the aorta, and ascertained that the pulse was due to the movement of the blood, and was synchronous throughout the body.

Praxagoras and his disciple Herophilus (344 B.C.), the latter of whom gave his name to the occipital confluence of the cerebral sinuses (torcular Herophili), treated more fully of the pulse, recognising the synchronism of the pulse and cardiac impulse.

Herophilus named the pulmonary artery the arterial vein, and the pulmonary veins venous arteries. The pulse he considered to be communicated from the heart to the arteries, but he believed that there was an active dilatation of these vessels which drew in vital air. He described four qualities in the pulse, size, frequency, force, and rhythm, and gave special attention to the rhythm of the arterial pulsation, by which was meant the relative duration of the wave and the interval, not the regularity or frequency of the beats. This he compared to musical time and to the measures of verse. It is interesting to note this early attention to the character of the pulse as distinguished from its force and frequency.

Erasistratus, who lived about 280 B.C., discovered the valves of the heart and described their action; he founded at Smyrna a school of followers, which flourished till the time of Galen.

In the first century of the Christian era Archigenes wrote a treatise on the pulse, which is often referred to by Galen in a controversial spirit, with the effect of exciting regret that the book has been lost.

Rufus of Ephesus, who lived a little later, also wrote a treatise on the pulse, which, after being lost, was discovered and translated by Daremberg in 1845. His description of the different characters of the pulse leaves little to be added at the present day. It is frequent or infrequent, as regards time or rate; quick or slow, as regards the individual pulsations; strong or weak, as regards intensity of beat; hard or soft, as regards the body of the artery.

The intermittent, dicrotous, vibratory (formicans), and vermicular pulses are described and named, and, under the term P caprizans—which was already adopted by Herophilus, but apparently applied differently by different writers—is described a pulse with a secondary beat, different from that of dicrotism,

which it is not easy to identify; while the Pulsus myouros appears to be one in which the respiratory variations of pressure are recognisable by the finger.

Galen, whose writings dominated the entire art of medicine for fourteen centuries, was born in A.D. 131 at Pergamos, and went to Rome in 164. His industry was extraordinary, and besides remarkable intellectual faculties, he must have possessed great force of character and much enthusiasm. He established the facts that the arteries did not contain air, but blood, and that there was no communication between them and the trachea, as had been taken for granted. He showed also that there must be some communication between the arteries and veins at their ultimate distribution, since, on bleeding an animal to death from an artery, the veins as well as the arteries were found to have been drained of blood. He supposed, however, that blood traversed the intraventricular septum from the right to the left side of the heart, and he considered the diastole of the heart and the pulsatile distension of the arteries to be corresponding phenomena, and to constitute the active phase of their action. The left ventricle, according to Galen, attracted the blood vitalised by the pneuma, which had entered it in the lungs, as a magnet attracts iron, and then distributed it by the aorta, the arteries again drawing it in by dilating.

He wrote voluminously on the pulse, as will be seen by an enumeration of his works on this subject:

1. "Libellus de Pulsibus ad Tirones."
2. "Libri Quatuor de Pulsuum Differentiis."
3. "Libri Quatuor de Pulsibus Dignoscendis."
4. "Libri Quatuor de Causis Pulsuum."
5. "Libri Quatuor de Præsagitione ex Pulsibus."
6. "Synopsis Sexdecim Librorum de Pulsibus."
7. "Pulsuum Compendium."

The general effect of his writings, however, is to

confuse the essential features of the important variations of the pulse by overwhelming them in minute distinctions of no practical significance. Indeed, his point of departure is not observation, but theory, and the varieties are not described from nature, but deduced from axioms.

It is not necessary to follow Galen in the enumeration of the varieties of pulse which he names. His account of them is characterised by extreme verbal subtlety, and one cannot wonder that his terms furnished matter for inexhaustible discussions by his followers in successive generations.

It is interesting, however, to find Galen noting carefully the relative duration of the periods of distension and relaxation of the arteries, which he does under the head of Pauses.

It will not be without interest, or indeed instruction, even in the present day, to reproduce an example of Galen's writing on the pulse, and a translation of a part of his treatise for beginners is here given.

GALEN ON THE PULSE.

ELEMENTARY FACTS.

Chapter 1.—I shall here speak merely of the elementary facts concerning the pulse, as I have treated of the whole subject elsewhere.

The heart and all the arteries pulsate with the same rhythm, so that from one you can judge of all; not that it is possible to feel the pulsations of all to the same extent, for those in the fleshy portions are much less distinct than those in the more superficial areas. For you could not perceive the pulsations of arteries which are thickly enveloped in flesh, or which lie within bones, or which have other bodies in front of them in an animal in natural health. But when the body is wasted away, the pulsations felt in the artery that lies along the vertebral column frequently indicate the throbbing of the abdomen, and arteries in the limbs previously indistinguishable have been felt.

But in all cases the pulsations of the arteries in the soles of

the feet and the wrist are easily felt. Not so distinct, yet by no means indistinguishable, are the pulsations of the arteries behind the ears and in the arms, and others that do not lie deep in the flesh. But you could not find any arteries more convenient or better or more suitable for the pulse than those in the wrists, for they are easily visible, as there is little flesh over them, and it is not necessary to strip any part of the body of clothing for them, as is necessary with many others, and they run in a straight course; and this is of no small help to the accuracy of diagnosis.

CHAPTER 2.—The artery will seem to the touch to be distended in every dimension. There are three dimensions to every body—length, depth, and breadth.*

In an animal in a normal state of health you will find the artery quite moderately distended; but in abnormal conditions sometimes the tension is too low, sometimes too great in every dimension. Now you must remember what a normal pulse is like, and if you find an abnormal pulse of excessive breadth, you should term it "broad," and if of excessive length "long," and if of excessive depth "deep," and in like manner the opposite of these "narrow," "short," and "shallow." And a pulse that is in all these dimensions abnormally diminished is termed "small," and one that is abnormally augmented "large." Such, then, are the varieties of pulse as far as dimension goes.

CHAPTER 3.—As regards special characteristics, there is swiftness and slowness. In the former case the movement is free and unrestrained, in the latter case enfeebled. These conditions you must judge by comparison with the normal.

The strength of the pulse or the reverse is determined by the force with which it repels the touch; if it repels violently it is strong, if weakly the reverse.

And there are variations in the softness or hardness of the arterial coat; it is soft when the artery appears, so to speak, flesh-like to the touch; hard when it seems dry and hard, like leather.

So then you notice differences in pulses such as this at once, as you observe the movement of the artery, though they

* In his larger treatise Galen enumerates all the possible permutations and combinations of these dimensions in the three degrees of large, moderate, and small, to the number of twenty-seven, as varieties of the pulse—an over-refinement on purely theoretical or transcendental grounds which led to extreme confusion. Deserting the path of observation, he did not see that a cylindrical tube would expand equally in all directions, and that there could not be any difference between its breadth and depth.

are not, however, specially characteristic of it, as were the three before mentioned.

For the speed or slowness of the pulse depends, we said, on the rate of movement, and the strength or feebleness on the character of the pulsation, and the largeness or smallness on the length of the diastole. But the diastole is not devoid of movement, and there is no need of movement in a soft or hard body for it to be such. These four variations in pulses you will find according to the beat.

Chapter 4.—Besides, there is a fifth variety depending on the pauses between the beats. For such is the term usually given by medical men to the space of time between the beats, within which the artery expands and contracts. Moreover, I think that beginners should practise themselves as though the systole could not be felt. The two terms I shall use are the pulsation and the pause. By the pulsation I mean the feeling the artery strike against the finger as it is expanded; by the pause I mean the period of quiescence between the pulsations, according to the length of which normal pulses are rapid, slow, or medium. These you will determine by the length of the pause. For a pulse is rapid when the interval of quiescence is short, slow when the interval is long. You may call it indifferently quiescence or pause between the pulsations or systole.*

Chapter 5.—Regularity and irregularity occur in the above-mentioned variations. By regularity is meant an even and unbroken series. For example, when the dimension of a series of pulsations continues the same, the pulse would be termed regular in size; and if the rate were unaltered, regular in rate. The same holds good in speaking of violence, feebleness, and frequency of pulse. Irregularity means the destruction of even rhythm in whatever varieties of pulse it occurs.

For one may be irregular in size, another in rate, another in violence, feebleness, and frequency, and so on.

Chapter 6.—Sometimes, too, when a number of beats are definite and regular, an uneven pulsation occurs in the midst of the even ones; and this may happen in various ways. For there may be three regular beats, then the fourth irregular,

* It is the individual pulsation which is here spoken of, not the pulse rate or frequency. In his larger treatise Galen describes two pauses—one after the diastole of the vessel, the other in systole; and it must be borne in mind that the diastole or pulsation was believed to be a more or less sudden expansion of the artery, and not, as we know it to be, a distension of the vessel by blood.

and so on continuously; or there may be four regular and the fifth irregular. The same thing may occur with any other number, for frequently the sixth is irregular after five regular beats, or the seventh after six. So, then, in these cases, a normal rhythm is not preserved, and so the pulse is not normal; and yet, as a certain fixed order of beat is maintained, it is regular.

For though the number is always constant, yet an irregular beat occurring in the midst of regular beats destroys the normal rhythm; but the recurring cycle insures a certain regularity. But if no period recurs, such a pulse is termed irregular.

CHAPTER 7.—And abnormality may occur even in a single pulsation, owing to the different relations which the parts of the artery bear to one another in rest and in movement, and owing to the special movement of each separate pulsation.

When the parts are at rest the abnormality consists in the artery seeming to have been drawn out of position upwards and downwards, and forwards and backwards, and to the right and to the left; but when in movement from the movement of the parts being too quick or too slow, or too soon or too late, too violent or too feeble, too long in duration or too short, being in perpetual movement or not moving at all.

But in cases of irregular flow, when it is at first too swift and afterwards too slow, and again when it is first too slow and then too swift, and in the same way with regard to violence and feebleness, and diminutiveness and excessive size, when the flow is not divided into two periods only, but into more, as far as can be discerned by the touch. Such, then, are the irregularities occurring in each separate pulsation.

CHAPTER 8.—The pulses are arranged, as far as it is possible for one to be taken with another, one with many, many with many, and some of them have a name; for instance, the worm-like (vermiform), the ant-like (formicans), and the hectic pulses. The worm-like pulse is a condition in which it seems as though a worm were creeping along the artery which is in waves of pulsation, the whole of the artery not being distended at the same time. If this takes place, accompanied by a short relaxation, it is called worm-like; but if with a long interval merely wave-like. The worm-like pulse, too, is readily seen to be feeble and beating quickly. But the pulse that has sunk to the extreme limits of feebleness, frequency, and smallness is called ant-like, and this, though it appears to be swift, is not really so.

So, too, the pulse is termed hectic, just as we apply the term to a fever, when it does not vary greatly, but remains much the same continuously, being entangled and never

getting free, as the whole condition is one of disease in fevers and pulses of this sort. I think I have said enough for beginners on the subject of varieties of pulses.

For if any one wishes for more accurate knowledge, there is the entire book on Differences of Pulses written by me. So I need not here speak of a full and empty pulse or of rhythms; for in my book I have given an accurate account of them, and the subject is rather difficult for beginners.

Let me then now sum up shortly what I have been speaking of, and then proceed to the subject next in order.

An excessive pulse is that which occurs when the artery is greatly distended in length, depth, and breadth; a pulse is long when the artery is distended only in length, broad when distended in breadth, deep when in depth. A violent pulse is one that strikes strongly against the finger; a soft gentle pulse occurs when the coat of the artery is soft. The pulse is rapid when the artery is distended in a short space of time; frequent when there is little interval; regular when each successive beat is the same; constant when each recurring cycle of beats is the same; a pulse that is uneven in one beat is termed irregular in one beat.

Clearly the opposites of these would be the small, short, narrow, low, feeble, hard, slow, infrequent, irregular, inconstant. Clearly, too, there is a mean between each of the other opposites; but there is no mean between a regular and irregular, a constant and inconstant pulse; and the means between all the others are the normal pulses, but in the latter cases the regular pulse alone is normal; the others—namely, the irregular and inconstant pulses—are abnormal.

CHAPTER 9.—Since pulses are readily altered in various ways—in fact, I might say there is no cause that does not change them—I have determined to take a threefold and very general difference in their change, and to speak specially about each in turn. The first change I shall speak of is that occurring in a normal pulse; the second is one which is not natural, but is not abnormal; the third is the abnormal.

All these changes take place as well as the natural variations, for there are many normal varieties of motion in arteries; and he who would fain determine the cause of the change in motion of a pulse and its magnitude must first be familiar with these. But the special peculiarities of each must be learnt by accurate trials and observations. And the artery should frequently be fingered, especially in a state of health and in the absence of all violent movement, and also, of course, in other states. But, since it is not in the power of all to attain this knowledge by experience—for many have often felt the need

of doctors at one time with whom when in sound health they had no intercourse—it is certainly best, even in this case, for the professional to excel the amateur; and he would do so if he has a knowledge of things which occur similarly in many cases.

Men, in short, possess different natures from women; those of a warm temperament from those of a cool. And each of these have a common nature, just as thin and fat people have. And there is rarely a trait in these common properties taken singly that is not similar to many.

So the man who has an accurate knowledge of general common features will rarely make a mistake.

The knowledge of the circulation and of the pulse remained at the point at which Galen left it for many centuries. Then, early in the sixteenth century, the function of the valves of the heart was made clear by Berengario di Carpi; the valves of the veins were described and their uses explained by Fabricius D'Aquapendente in 1625; and the mitral valve was described and named, and the perforation of the intra-ventricular septum disproved, by the great Vesalius in 1555.

Realdo Columbo now also clearly described the pulmonary circulation in his work on "Anatomy," published in 1558, having already propounded the discovery in his "Lectures," through which, perhaps, Servetus, who first published it, had obtained the idea.

Finally comes Cesalpinus, who, writing in 1543, seems first to have conceived the notion of the systemic circulation, but without appreciating its importance, and without any recognition of the heart as the agent in the propulsion of the blood, and who, moreover, instead of devoting his energies to the demonstration and establishment of so great a discovery, buried his idea in a mass of other speculations ("Questiones Peripateticæ"), so that it was unknown to his contemporaries and to the professors who immediately followed him in his own country, leaving to

Harvey the incontestable glory of the real discovery and proof of the circulation.

This is not the place to explain once more the steps of Harvey's complete and lucid experimental demonstration of the circulation, but it is worth while mentioning that in his treatise "Exercitationes Anatomicæ de Motu Cordis et Sanguinis Circulatione," the contraction of the ventricles was, for the first time, shown to be the agent in the propulsion of the blood along the arteries throughout the body. Harvey began to teach the circulation in 1619, but did not publish his book till 1628.

The porosities in the tissues by which Harvey concluded that the blood made its way from the arteries to the venous radicles, were shown by Malpighi to be a network of capillary vessels—a discovery which completed the demonstration of the circulation.

Nothing is to be gained by attempting to obtain an idea of the critical pulses of Solano and Nihell, by which the occurrence of epistaxis, purgation, etc., was foreseen, or of the organic pulses of Bordeu and Fouquet, a special variety of pulse for disease in each organ. These fantastic descriptions had no reference to the physiology of the circulation, and might more easily have been written before its discovery.

It is, however, interesting to note that it was Kepler who originated the idea of counting the pulse by the minute. Before this there was no standard of frequency to which reference could be made.

With the complete overthrow of the theory which had obtained from the time of Galen as to the movement of the blood, his teachings with regard to the pulse fell into discredit, and the minute and exact observation which had established distinctions between the different varieties of pulse, scarcely surpassed in accuracy and completeness by the teachings of the sphygmograph, and which furnished ready-made almost

all the terms required by the records of this instrument, was neglected. For the most clear and true terminology we must, indeed, go farther back than Galen, since he overlaid it with trivial and transcendental refinements, and confused the results of simple and accurate observation by verbal and theoretical considerations.

To-day the study of the pulse cannot be better begun, or a better description of its variations be more clearly given than by considering separately—(1) the rate or frequency of the beats; (2) the character of the individual pulsations; (3) the intensity of the beats; (4) the size of the vessel and its hardness or softness, as given by Rufus, and probably by Archigenes. No doubt physicians have continued to estimate with care and accuracy the diagnostic and prognostic significance of the indications furnished by the pulse, but it was a great loss to medical science when vague descriptions, such as quick, or rapid, and slow, full and bounding, firm, and the like, capable of various interpretations, took the place of the definite terms employed by the early writers named. It thus became impossible to transmit by writing the knowledge gained by experience.

A new era, however, set in when the circulation began to be studied as a physical problem. The early attempts to estimate the work done by the heart were, it is true, mere random guesses; but when Hales in 1748 and Poiseuille in 1828 applied the first rude apparatus for measuring the fluid pressure in the arteries, and setting it down as equivalent to a column of so many inches of blood or water, or so many millimetres of mercury, a real step was taken, and the way was opened for the complete and more exact investigations of Volkmann, Vierordt, Ludwig, Fick, Chauveau, Marey, and others.

The fluid pressure has been measured not only in

the arteries, but in the different chambers of the heart itself by means of instruments devised by Marey ; the rate of movement of the blood along the vessels has also been approximately determined.

In another direction the action of the nerves upon the heart, accelerating or slowing its beats, and rendering them more or less energetic, and upon the arteries, narrowing or dilating their channels, was gradually ascertained by successive experimenters.

From the fact that the heart continues to beat after its removal from the body, it was believed by early experimenters to be altogether independent of the nervous system, and there can be no doubt as to the independence of the property of rhythmic contraction which it possesses. But the Brothers Weber in 1845, and Budge in 1846, discovered the controlling influence of the pneumogastric ; the excitant influence of the sympathetic and of the spinal cord, by means of communicating branches to the sympathetic, in which Prochaska and Brachet appear to have led the way, was also established. The most recent investigations, while they have contributed details and refinements, and have shown that the action of the heart is indirectly affected through variations in the blood pressure, as well as directly by the pneumogastric and sympathetic nerves, have only established these discoveries, which were the earliest results of physiological experimentation.

Perhaps even more important was the discovery of the contraction and dilatation of the arterioles under nervous stimulation. The knowledge of the vaso-motor mechanism to which it led, while explaining the old maxim "Ubi stimulus ibi fluxus," is essential to any true comprehension of the varieties of pulse. The influence of the nerves upon the diameter of the arteries had been asserted by Henle and Stilling, and the latter had introduced the term vaso motor, but it

is to Claude Bernard, whose discoveries were quickly extended by Brown-Sequard, that science owes the complete demonstration of the vaso-motor function of the sympathetic. This was only in 1851.

While in this way the physiology of the circulation was elucidated, the application of the graphic method by means of the admirable sphygmograph invented by Marey led to a scientific study of the pulse and to a comprehension of its indications never before possible. The object of this little book is to set forth these indications, and to bring to pass their application to the everyday practice of medicine.

CHAPTER II.

THE PULSE, ITS PRODUCTION AND SIGNIFICANCE.

Introductory remarks.—The blood is the source from which the tissues draw their supply of nutrient material, and of oxygen which is consumed in the oxidation necessary for the evolution of energy, heat, motion, or nerve-force, and the object of the circulation is to renew this supply according to the needs of the structures and the demands for force. The renewal is effected by the propulsion into the aorta of a certain amount of blood by each systole of the ventricle, and this is distributed by the arteries to the network of capillaries which are in intimate relations with the tissue elements.

The amount of blood delivered by the ventricular systole is variously estimated. Professor Foster accepts six ounces (180 grammes) as approximately correct.

The velocity of its movement in the carotid is estimated from experiments on animals at about 12 inches per second (300 millimetres), or at the outside 18 to 20 inches; but in the carotid and vessels of this size the movement is rapid during the systole of the heart, almost suspended during the diastole. The average motion of the blood will therefore be less than this, even in the large arteries; and since, with few exceptions pointed out by Mr. Nunn, the sectional area of the branches at each division of the arterial tree is together greater than that of the dividing trunk, the blood will move more and more slowly as it gets nearer the periphery; in the radial the rate is probably three or four inches per second. In the capillaries the motion of the blood is very slow, in the

human retina it is estimated by Vierordt at ·75 mm. (about $\frac{3}{100}$ths of an inch in the second). This great fall in the rapidity of the blood is due partly to the much larger collective capacity of the capillary channels, which may be said to form a lake in the course of the stream, partly to the friction in the tortuous and inter-communicating channels, but probably also, in part, to cohesion between the blood elements and the capillary walls, which will intensify the friction. There is, then, in the capillary network considerable obstruction to the flow of blood, requiring considerable pressure to force it through. Now this pressure is an indispensable element in the adaptation of the circulation to the purposes of nutrition. While the blood is within the capillaries, it is out of reach of the tissues, and its albuminoid constituents are not available for their nutrition. These blood albuminoids are not in a diffusible form, and we have no right to suppose that they will diffuse through the capillary walls just because they are wanted outside. Here the pressure comes in ; it forces a certain amount of albuminoid matter to exude out of the capillaries into the interstices of the tissues where it is in immediate relation with their nutritional elements, and forms the "milieu" in which they live. Of course these albuminoids cannot get back into the blood against pressure, but the interstices of the tissues are continuous with the lymphatics, and the office of the lymphatics is to afford a channel by which nutrient material, which has escaped from the capillaries in excess of the demands of the tissues, may be restored to the circulation. With regard to the diffusible constituents of the blood, its oxygen, its salines, its sugar, and the diffusible products of tissue waste, the carbonic acid and urea, the case is different. For them the intertextural fluid and the blood in the capillaries are in free communication, since the capillary membrane

offers little or no obstacle to their interchange, and the time occupied by the blood in filtering through the capillary network of blood-vessels, estimated to be a full second, is sufficient to allow of the mutual diffusion.

We examine habitually the pulse at the wrist, and at first sight it seems strange that the radial artery, which supplies merely the structures of a part of the hand—a few small bones with their articulations, a few muscles and tendons, the skin and nerves distributed to it—should afford the varied and far-reaching knowledge we look for in the pulse. The hand is not essential to life, it contains no organ of any importance, and *à priori* it might have been supposed that the variations in the circulation of the blood in so small a member could have no significance. We know as a matter of observation, however, that the pulse of the wrist indicates the condition of the circulation generally, and on reflection it is seen that, as a branch of the great arterial system, it receives every impulse starting from the heart, and reveals the frequency and force of its beats; and not only this, but as fluid pressure is equal, or tends to become equal, in all parts of a freely communicating system of tubes, it shows the degree of freedom of the general outflow of the blood through the capillaries as well as the energy of its propulsion by the heart. The very fact that the hand has no special circulation of its own liable to extreme variations for functional purposes—such as, for example, those which occur in the salivary glands—makes the radial pulse a more trustworthy index of the general circulation.

What the pulse really is.—A preliminary question upon which a distinct understanding must be arrived at is the following: The pulse, what is it? and what is the exact information which it furnishes?

Now it is not, as is commonly understood, an expansion of the artery. This, at any rate, is not what we feel or what is recorded by the sphygmograph. A moment's reflection as to the volume of blood discharged by the left ventricle into the aorta, and a comparison of this with the capacity of the entire arterial system, will convince us that it is altogether inadequate to produce any such expansion of the smaller arteries as will be appreciable to the touch. The aorta and its primary branches are, it is true, dilated somewhat by the injected blood; but even in a vessel of the size of the carotid it is difficult to measure the increase of diameter, so minute is it; whereas in the radial, in which it must be much less, the sphygmograph, if its trace were taken to indicate actual enlargement of the artery, would show the expansion to be considerable. Nor is the pulse a sinuous movement of the artery in its bed from elongation which throws it into curves. To feel the pulsation in an artery, or to take a sphygmographic trace, a certain degree of pressure must be applied to the vessel, and, as is well known, there must be a bone behind it against which it can be compressed. What happens, then, is as follows:—In the intervals between the pulsations, when the resistance by the contained blood is at its lowest, the tube of the artery is more or less flattened by the pressure of the finger upon it; then comes the so-called wave of blood propelled by the systole of the left ventricle, or, to speak more accurately, the fluid pressure in the vessel is increased, and this forces the artery back into the circular form (Fig. 1). It is this change of shape from the flattened condition impressed upon the vessel by the finger, or by the sphygmographic lever, to the round cylindrical shape, which it assumes under the distending force of the blood within

Fig. 1.

it which constitutes for us the pulse. Such a pulsation can be felt on a large scale by placing the foot on the inelastic leather hose of a fire-engine in action, in which there can be no expansion, or shown in a schema of the circulation with inelastic vessels. It is not, then, an increase in the diameter of the vessel, but an increase of the blood-pressure within it, created by the systole of the ventricle of the heart, which constitutes the pulse.

Another common misconception must be cleared up—namely, that the pulse necessarily signifies onward movement of the blood in the artery. Since a certain amount of blood is normally injected into the aorta at each systole, it would seem, at first sight, that there must be a corresponding propulsion of blood along the vessel which is under the finger, and misapprehension has been carried so far that the pulse-wave has been understood to mean the actual transport of the blood, and even to indicate the rapidity of such motion. Short of this, it is more commonly taken for granted that the rate of movement of the blood in the vessels is directly proportionate to the strength of the pulse, that a good strong pulse implies a vigorous rush through the capillaries, and a weak pulse a languid flow. The stream which issues from a divided artery and the pulsatile jet seem to countenance this conclusion. It is, however, an erroneous idea. If the radial is compressed close to the hand, the pulsation above is not extinguished, but exaggerated; and when an artery is tied, the pulsation up to the ligatured point is more vehement than before. Pulsation is thus no evidence of onward movement of the blood. Now, resistance in the arteries and capillaries will have *pro tanto* the effect of a ligature, hindering or even arresting the onward current. And there can be no doubt that peripheral obstruction does at times reach a point which almost stops the flow

from the arteries to the veins, the pulse appearing to be all the stronger on this account. The heart acts with increasing energy in order to combat the obstruction, but may fail so far to overcome it as to propel an average amount of blood into the aorta, although it raises the pressure throughout the arterial system. This is a consideration which, it seems to me, is not adequately borne in mind. I think it enters into the explanation of dropsy, and especially of the varying amount of dropsical effusion under apparently similar conditions, and that it also helps to clear up obscurities in the relation between circulatory conditions and head symptoms.

The pulse, then, indicates simply the degree and duration of increased pressure in the arterial system caused by the ventricular systole. There is a certain mean blood-pressure maintained by the elasticity of the large arteries, varying greatly in different individuals, which keeps up the flow through the capillaries, and the level of which is determined by the resistance in the capillaries and the amount of force received from the heart, and stored up by the elastic walls of the large arteries. This pressure is lowered during the diastole of the heart by the outflow through the capillaries into the veins, and is reinforced by the successive contractions of the left ventricle, and the pulse marks and indicates the minimum and the maximum pressures, with the gradation from one to the other. The term "tension," as applied to the pulse, means simply the degree of fluid pressure within the artery, putting its walls on the stretch.

Arterial tension and blood-pressure mean exactly the same thing. Distension might perhaps be more expressive than tension if less exact and technical.

Factors of the pulse.—With these preliminary observations we may proceed to consider the factors of the pulse.

There are three factors in the production of the pulse, and the influence of each on the variations observed in it must be understood. The three factors are :—

1. The action of the heart.
2. The elasticity of the great vessels.
3. The resistance in the arterioles and capillaries.

The heart determines unconditionally the frequency and regularity or irregularity of the pulse, and, with certain qualifications, its force or strength. The great vessels, acting as an elastic reservoir, convert the intermittent jet issuing from the ventricle into a more or less continuous stream, impressing at the same time certain characters upon the pulse according as the elasticity of their walls is perfect or impaired, and according as they are kept fully distended, or only slightly on the stretch. The capillaries and arterioles, by the varying resistance which they offer to the passage of blood through them, determine the mean pressure maintained in the arterial system and the character of the pulse, and influence materially the action of the heart. Each of these must be considered in some detail.

I.—The Action of the Heart.

1. **Frequency.**—It has just been said that the heart determines absolutely the frequency of the pulse ; and this is true in so far that the number of beats of the pulse corresponds with the number of heart beats, except when a certain proportion of the latter are too weak to reach the wrist, or when very little blood enters the ventricle during its diastole ; with the exception again of that curious modification of the heart's action in which there are two heart beats coupled together for every beat of the pulse. It must not be lost sight of, however, that resistance in the peripheral circulation re-acts upon the heart's

action, as well as upon its character, the frequency being increased as resistance is lessened, and *vice versâ*; subject, however, in both cases to the intervention of the nervous system.

2. **Rhythm.**—The rhythm, as well as the rate, of the pulse is determined by the heart, and the pulse, generally speaking, is regular or irregular according as the action of the heart is regular or irregular. The pulse, however, may be made irregular when the heart is acting regularly by beats failing to reach the wrist, and irregularity of the heart's action may be greatly exaggerated in the pulse.

3. **Force.**—With regard to the strength or force of the pulse, again, this must be directly dependent upon the strength of the ventricular systole. The pulse cannot be strong or forcible when the heart's action is weak, and it will not, as a rule, be weak when the heart's action is vigorous. But the volume of blood discharged by the ventricle into the aorta is another element in the production of the pulse. If from any cause the ventricle is not properly filled, as may be the case when the total volume of the blood has been reduced by hæmorrhage or other cause, or when there is obstruction in the pulmonary circulation, from disease of the lungs or extreme constriction of the mitral orifice, or when the ventricle has not time to dilate, as may happen in palpitation, the systole, however forcible, will have little effect in increasing the pressure in the arterial system; and there may, under such circumstances, be powerful action of the heart, with a feeble pulse. It will also be seen, when the influence of peripheral resistance is discussed, that the apparent strength of the pulse may not correspond with the energy of the ventricular contraction, even when the amount of blood propelled is normal; and the blood-pressure or arterial tension—*i.e.* the degree of distension of the arteries—

while it is maintained by the heart, and is dependent upon the degree of pressure supplied by the ventricular systole, is by no means necessarily proportionate to its vigour.

II.—Elasticity of the Great Vessels.

The principal effect of the large arteries is to act as an elastic reservoir, which converts the intermittent jet of blood which issues from the ventricle into a continuous stream. They are kept by the resistance to the outflow through the capillaries in a state of continual distension, which is increased momentarily by each ventricular systole, and runs down to some extent in the intervals, but never during life to a point at which the elastic coats of the vessel cease to exercise some compression on its contained blood. The force of the heart is thus stored up and delivered out gradually in the form of a steady pressure, which keeps up an almost uniform flow through the vessels of the periphery. The regular current of blood sustained in this way is essential to the functional activity of the central nervous system.

A collateral result of the elasticity of the large arteries is an economy of the force of the heart, since the outflow from elastic tubes under an intermittent supply is greater than if the tubes, being of the same size, were rigid; but this effect is not appreciable in the pulse.

Another effect, however, is a certain delay of the pulse wave. In a rigid system of tubes increase of pressure from the injection of fluid would be simultaneous at every point, and, were the arteries inelastic, there would be no loss of time between the heart and the pulse. The result of the elasticity of the great vessels is that the increase of pressure from the systole of the ventricle is partly expended in dilating them, and, in proportion as this obtains, there

is delay in the transmission of the pressure onwards. The pulse wave, as the increase of pressure is called, travels, according to Dr. A. Waller, at about the rate of thirty feet a second. The want of synchronism between the heart and the pulse is quite perceptible, and the pulse cannot therefore be taken as a point of reference by which to determine the position of a murmur or sound in the cardiac revolution, as has often been recommended. This is all the more liable to give rise to confusion, as the loss of time is variable, being greatest when the mean blood-pressure in the arteries is low, less when the tension is high, least when the great vessels have lost their elasticity from degeneration.

Further, the elasticity of the great vessels is concerned in the production of the dicrotism of the pulse.

Dicrotism—*i.e.* a double or re-duplicated beat—is, in a small or great degree, a constant feature of the normal pulse. As felt by the fingers, it may be

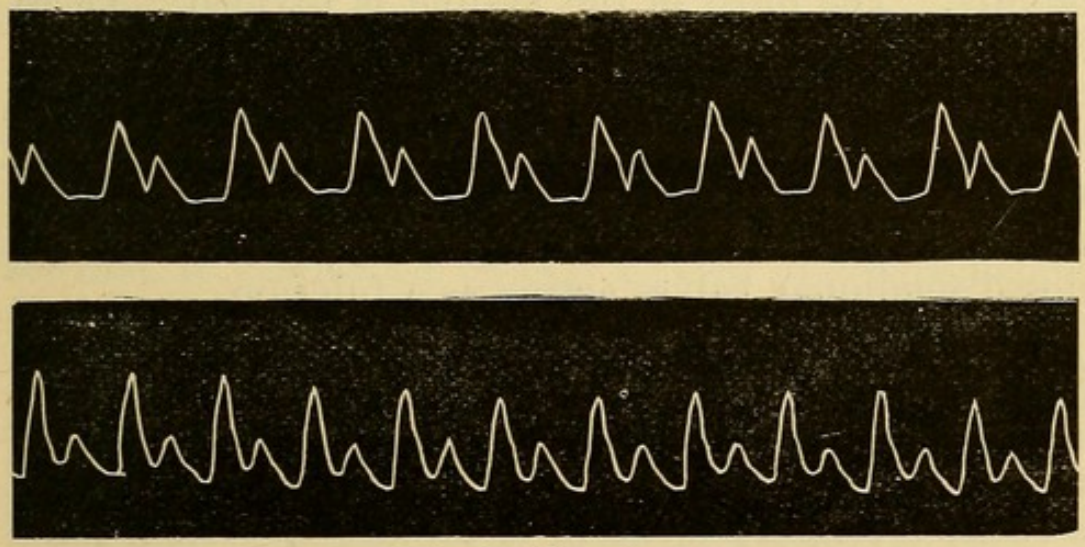


Fig. 2.—Dicrotism.

described as a sort of echo of the main beat following it at a very brief interval, and it is represented in the sphygmographic trace by a secondary rise of varying height occurring in the course of the down stroke at a fairly constant point. (Fig. 2.) Dicrotism

has been the subject of much discussion. It has been considered to be produced by a wave reflected by the periphery. But this view is now quite abandoned; had it been true, the reflected wave would have followed the primary wave more quickly near the periphery than near the heart, and the contrary is the case. The dicrotic wave is a secondary wave of pressure, due to the elastic recoil of the aorta from its expansion by the blood injected during the systole of the left ventricle.

The conditions favourable to its manifestation are dilatation of the arterioles and sharp contraction of the heart, which commonly go together. When the outflow by the capillaries is rapid, the pressure in the aorta and great vessels will run down speedily during the cardiac diastole, and there will be a great and sudden rise with the systole. There being, moreover, comparatively little resistance to the blood entering the aorta from the ventricle, its systole takes place rapidly. The force of the systole is divided. There is at the same time a launching forwards of the column of blood and an expansion of the great vessels laterally, their elastic coats not being on the stretch, and so yielding easily. A contractile rebound follows, which starts the dicrotic wave along the arteries, the closed semilunar valves acting as a fulcrum.

Another variety of double beat, the pulsus bisferiens—which may easily be mistaken for dicrotism, but which must be distinguished from it—is met with under totally different conditions. This second beat is really a reinforcement of a prolonged systole near its close, and is represented by a sphygmogram, such as those here given. (Fig. 3.)

It is most common as a result of aortic stenosis, but is met with in senile degeneration of the arteries. The artery is usually rather small, always full between the beats, and generally resisting compression; the

pulse wave is gradual in onset and long, and just before it begins to subside a second beat is felt.

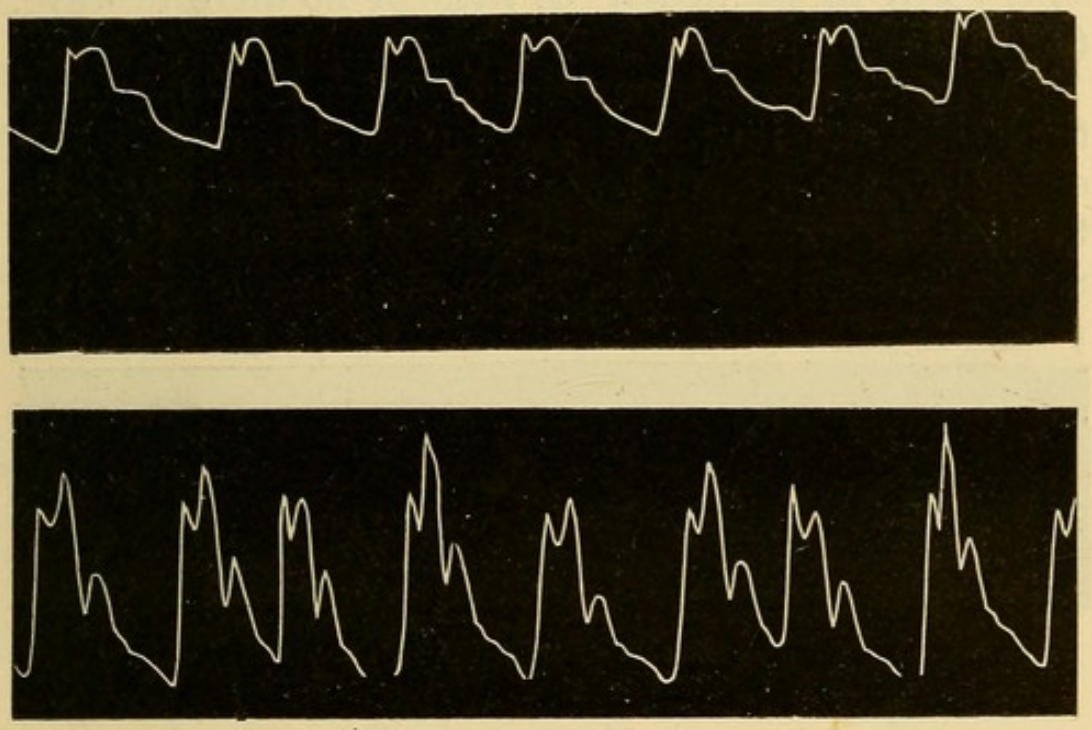


Fig. 3.—Pulsus Bisferiens.

When the arterioles are contracted and the onward movement of the blood in the periphery is obstructed, the pressure in the great vessels cannot reduce itself by rapid outflow through the capillaries. The aorta is distended, and its coats are on the stretch, so that it cannot be quickly dilated to accommodate the contents of the ventricle: the force of the systole

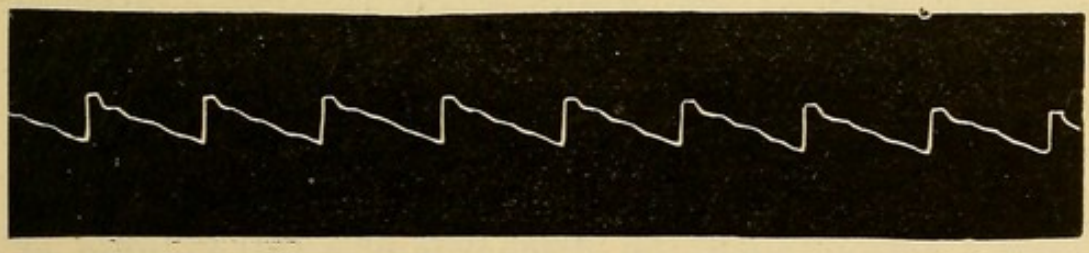


Fig. 4.—Normal High Tension.

will thus be expended mainly in pushing forwards the column of blood, and the exact converse of the effects above enumerated as giving rise to dicrotism will

hold. The systole will be slowed, there will be no great expansion and contraction of the aorta. There will in particular be little or no dicrotic wave. (Fig. 4.)

When from atheromatous or other senile changes the aorta and its primary branches have lost their elasticity, the resemblance to a system of inelastic tubes is more complete, and the pulse curve not being modified resembles that of the ventricle. (Fig. 5.)

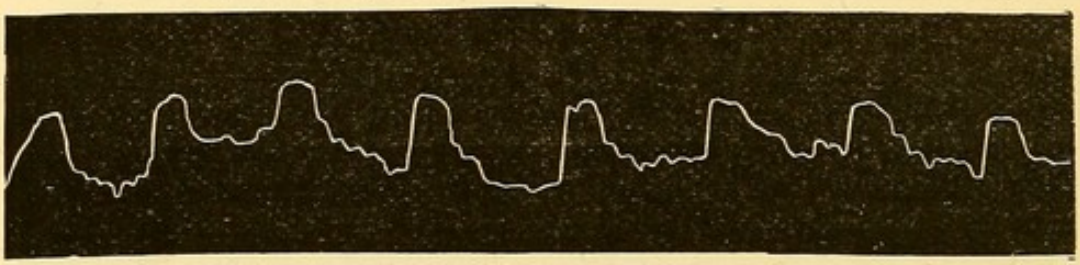


Fig. 5.—Senile Pulse.

III.—Arterio-Capillary Resistance.

Not less important than the action of the heart in its influence on the circulation of the blood, and even more important in the modifications it produces in the character of the pulse, is the resistance in the arterioles and capillaries. It is by their resistance in front and the force of the heart's action behind that the mean pressure in the arterial system is determined. If the outflow through the capillaries is free, then no amount of blood which the heart in a normal condition can pour into the arteries, and no degree of energy with which this is projected into the aorta, will maintain the general arterial pressure at a high point. It is possible that great frequency of the heart's action with the discharge of a full volume of blood by each systole may pour blood into the arteries more quickly than it can run off by the capillaries even when relaxed, in which case the pressure would rise; but this is a rare occurrence, except for brief

periods in excitement or the early stage of effort. On the other hand, when the passage through the capillary network is obstructed, the blood is dammed back in the arteries, and the pressure within them is raised, it being understood always that the heart is capable of supplying the requisite force, which must of course be sufficient to overcome the peripheral obstruction and keep up some onward movement of the blood, or life would cease. The average blood-pressure or mean tension present in a given case is a most important part of the knowledge to be obtained from the pulse; it is measured by the degree of fulness and resistance in the artery between the beats; the variations in the character of the pulse contributing to an accurate estimate of it.

It follows from theoretical considerations, which need not be here discussed, and it is proved both by observation and experiment, that the higher the constant or mean pressure in the arteries, the less is the difference between the maximum and minimum; or, in other words, the more full and firm the artery is between the beat, the less marked will be the pulsation, and *vice versâ;* the lower the tension and the more conspicuous the pulsation.

The significance, again, of the constant or mean, and the variable or pulsatile, pressure is different. The mean pressure tells most with regard to the circulation and the circulatory system. The variable pressure or pulse tells most with regard to the general state of the individual.

When the peripheral vessels oppose little resistance to the passage of the blood through them, the pressure in the arteries will not only be low, but it will also be variable. The blood flowing off quickly, the pressure will rapidly run down in the intervals of the ventricular systole, and will rise suddenly with each systole. This would be the case even were the

celerity of the individual contractions constant and uninfluenced by the amount of force to be overcome; such, however, is not the case, for when it meets with diminished resistance the ventricle expels its contents more rapidly. The pulse, then, will be more sudden or sharp, and will seem to be more vehement. The artery at the wrist and elsewhere will allow itself to be flattened more easily and completely, as there is little pressure of blood within it to resist the external pressure; it is then suddenly distended, and resumes its cylindrical form, repelling the finger and jerking up the sphygmographic lever. The effect is all the more marked from the fact that the walls of the artery will be relaxed and its diameter increased. It is shown in the following diagrams, the first of which (Fig. 6)

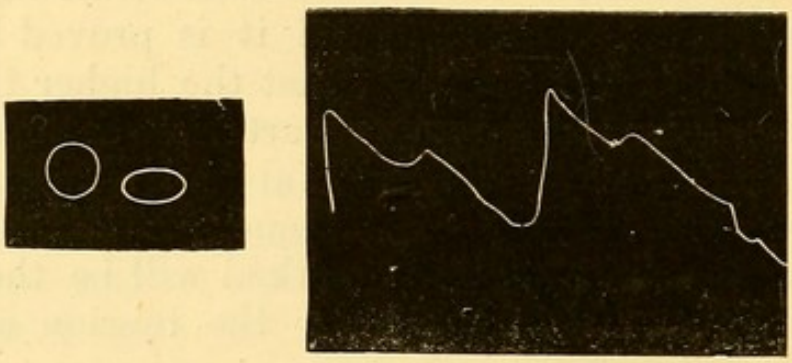


Fig. 6.

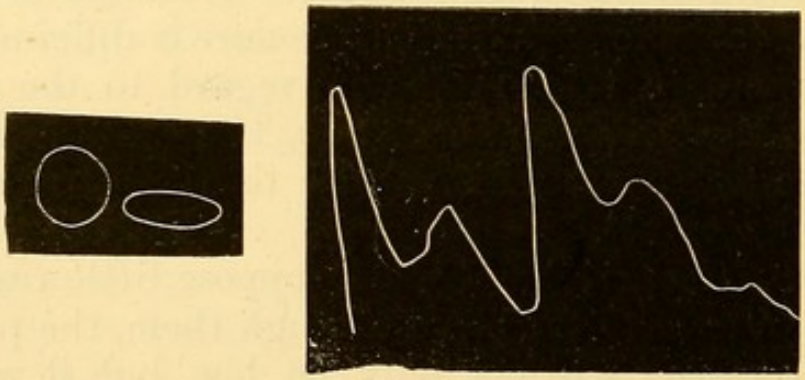


Fig. 7.

is supposed to represent an artery of normal size, with its pulse-trace, the second (Fig. 7) the same artery when relaxed, with its pulse-trace.

But another consequence flows from the varying resistance in the peripheral arterioles and capillaries. A given volume of liquid will pass more rapidly through a large channel than through a smaller under the same pressure. When, then, the resistance in the periphery is weak—which is equivalent to the channel being large—the pulse will be short: that is, the wave is sudden and soon over.

The converse effect will be produced by peripheral resistance. With a higher mean pressure the variations will be less, and the rise of pressure which constitutes the pulse more gradual, this difference being accentuated when the artery is contracted. The wave again will be long (Fig. 8).

Fig. 8.

It is easy to confound the fulness of the artery between the beats with a prolonged beat; but although these two conditions are often met with together, they are distinct, and must be kept so in our minds.

The sphygmograph.—In recent teachings with regard to the circulation and the pulse, the constant reference to the sphygmograph has been an obstacle to the application of the newly-obtained knowledge to clinical work, and especially to everyday practice.

It is not every student who can thoroughly familiarise himself with this instrument and acquire the requisite skill for bringing out its indications, and the busy practitioner has still less chance of doing this; nor has he the time to employ it constantly, while without constant use the results are untrustworthy.

It will, therefore, be an object in this little book to describe the variations in the character of the pulse as they are felt by the finger, and the sphygmograph will be relegated to a position of secondary importance. It has been objected that the distinctions are too refined to be appreciated by the touch. But the answer to this is that every important variety of pulse revealed by the sphygmograph was recognised, described, and named before the Christian era, and it may be added that resident physicians and clinical clerks of the present day learn to do this without difficulty.

The sphygmograph has been invaluable in research; it has given precision to our ideas, and in the hands of Marey and others has made clear and comprehensible many intricate and doubtful problems of the circulation. It is capable, too, of rendering important aid in clinical investigation, especially where demonstration and records of changes in the circulation are required. To me personally the sphygmograph has been of immense service. I worked with it under the eye of Sibson, and shared Anstie's enthusiasm with regard to it when he and Professor Burdon Sanderson took up the instrument with which Marey endowed medical science. It is not, therefore, from ignorance of or want of familiarity with the sphygmograph that I have come to the conclusion that it is not specially useful in practice—that in any form known to me it is not a clinical instrument for every-day work. It is rarely necessary for diagnosis, and scarcely ever to be trusted in prognosis. The indications obtained from it are not, like those of the thermometer, independent of the observer. Skill and practice are required in applying it; judgment is called for in determining the position and pressure which give the best trace, and indeed in deciding which of the traces obtainable is the best representative

of the particular pulse; the personal equation of the observer, therefore, comes in, and if any special result is expected or wished for, an enthusiastic investigator can obtain it, and may, without the least conscious intention, twist facts in the desired direction. It is necessary also before a trace can be interpreted with any degree of confidence to know what form of sphygmograph has been employed. Marey's is still, in my opinion, the best, and his traces appear to me to correspond most closely with traces taken without instrumental multiplication, and magnified by the lens. English modifications of Marey's sphygmograph often magnify the pulsation too much, and in doing so introduce exaggerations due to the rapid movement of the writing lever. Pond's and Dudgeon's instruments are extremely handy and convenient, but a gratuitous provision for exaggerations and for extraneous jerks and vibrations exists in the loose and unmechanical way in which the motion of the intermediate lever is communicated to the writing lever, and in the weight which acts as counterpoise in the last-named lever.

The pretence, again, to measure the exact pressure employed in taking the trace, and thereby to obtain corresponding knowledge of the intra-arterial pressure, is illusory. To say nothing of the varying thickness and resistance of the skin, which would of itself vitiate all conclusions of this kind in at least three ways—by difference of flexibility, by affecting the position of the spring, and by varying the area of the button or pad actually in contact with the skin—the size of the artery would introduce an element of uncertainty. Hydraulic pressure is equal at every point of the containing surface, and its force is multiplied by increase of area; the greater surface, therefore, of a large vessel would exercise greater lifting power, so that the same blood pressure would appear to be

higher or lower according to the diameter of the artery. For the same reason buttons or pads of different size resting on the vessel would affect the degree of force required in order to compress it, as would slight differences of adjustment. A knife-edge resting transversely across the vessel eliminates some of these uncertainties, and gives a more accurate trace, but requires delicate adjustment of the strength of the spring. The above would be true even if the pressure exercised by the spring were accurately graduated; but this is far from being the case in any sphygmograph known to me, and graduation by the eccentric commonly employed is ridiculously inexact. While, then, I think that every student ought to be familiar with the sphygmograph, and will gain from a study of its indications a comprehension of the pulse in its different forms, obtainable in no other way, I am of opinion that we learn by means of the educated finger all that the sphygmograph can teach, and more. This instrument is invaluable as a means of educating the sense of touch and of cultivating the faculty of observation; it is most useful in resolving doubts as to the difference between the pulse of the two sides in some cases of aneurism, and in recording pulses in the graphic form; but it is not an infallible court of appeal, and there are niceties of information which are out of its reach. I need only point to the groups of pulse tracings exhibited, which are by experts, to show that a sphygmogram does not speak for itself, but requires interpretation. One set is a series of normal traces, made for me by the late Dr. Mahomed to illustrate my lectures at St. Mary's; the others are copies—one from Marey, the other from Hayden, of traces from cases of aortic regurgitation or insufficiency. (Figs. 9, 10, 11.) Unless it were so stated it would be difficult to believe that the latter were all taken from the same form of disease.

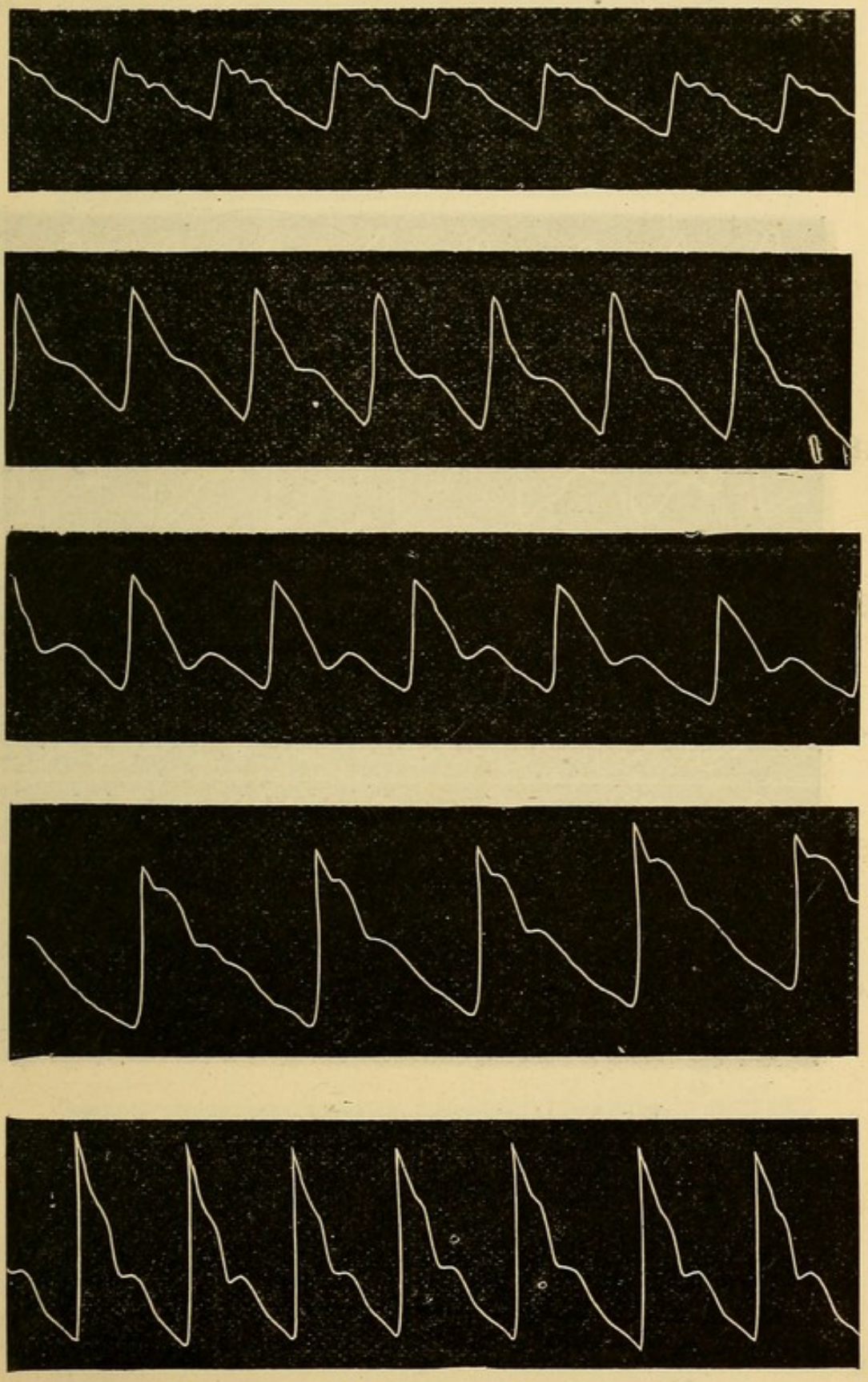


Fig. 9.—Normal Forms of Pulse (Mahomed).

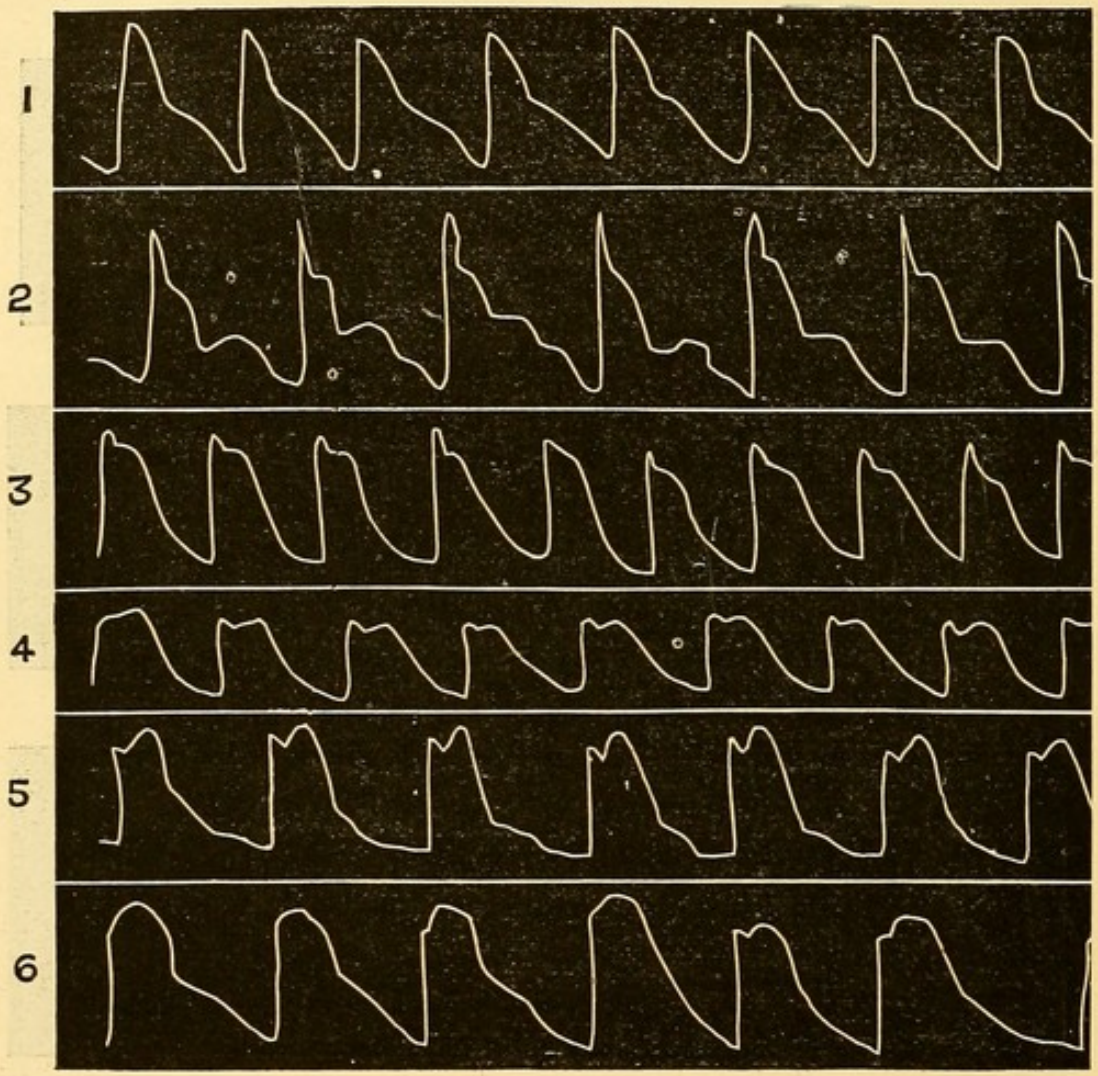
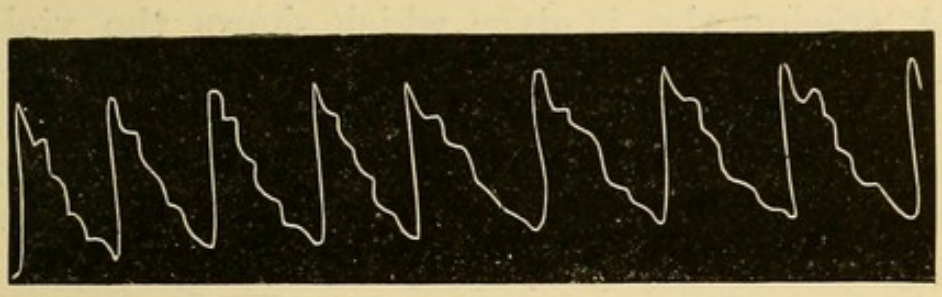
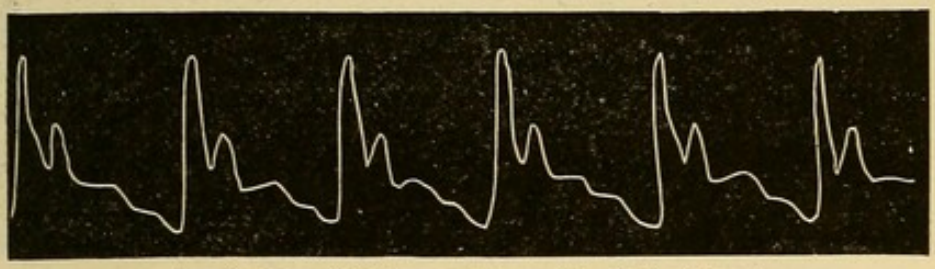


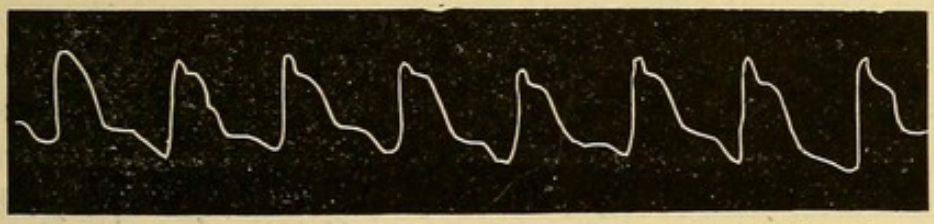
Fig. 10.—Aortic Regurgitation (Marey).



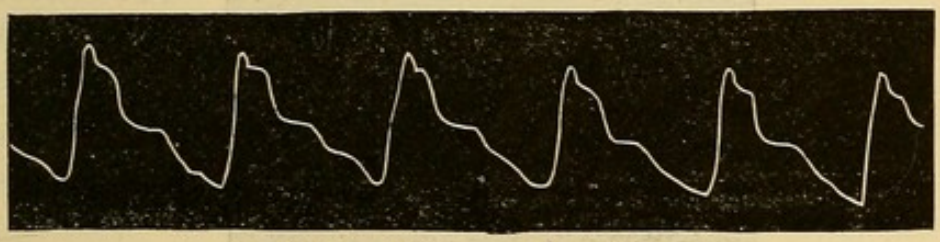
Aortic Insufficiency.



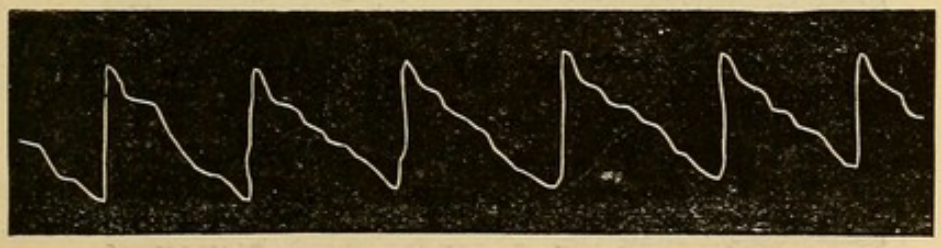
Aortic Insufficiency. (From Dr. Grimshaw.)



Aortic Insufficiency. Cardiac Hypertrophy. (Dr. Boileau.)



Aortic Insufficiency. (Dr. Boileau.)



Aortic Insufficiency. (Dr. Boileau.)

Fig. 11.—Pulse Tracings from Hayden; totally different representations of aortic insufficiency by different observers and different kinds of instruments.

A tracing also is given which shows the violent oscillations sometimes yielded by Dudgeon's sphygmograph: the instrument employed was one which I have used for years, and was generally fairly trustworthy. The case was a severe example of aortic regurgitation, with collapsing pulse. (Fig. 12.)

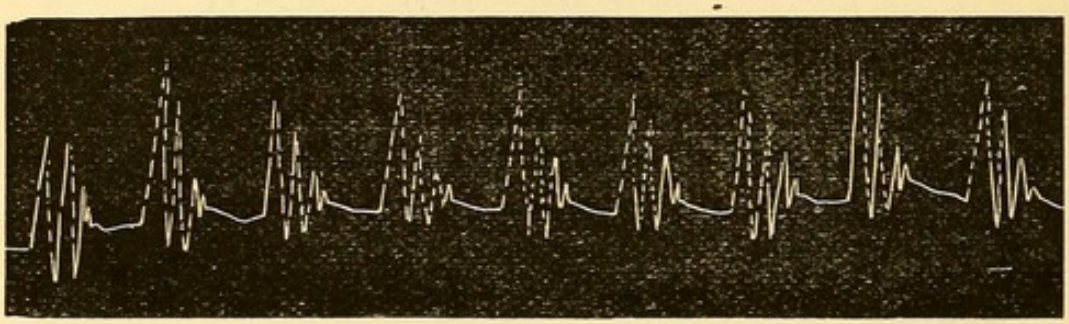


Fig. 12.

To illustrate the changes in the form and character of a sphygmogram produced by increase of pressure by the spring, tracings are copied from Landois and Stirling's "Physiology," showing the effects of a gradual increase from 100 to 450 grammes. (Fig. 13.)

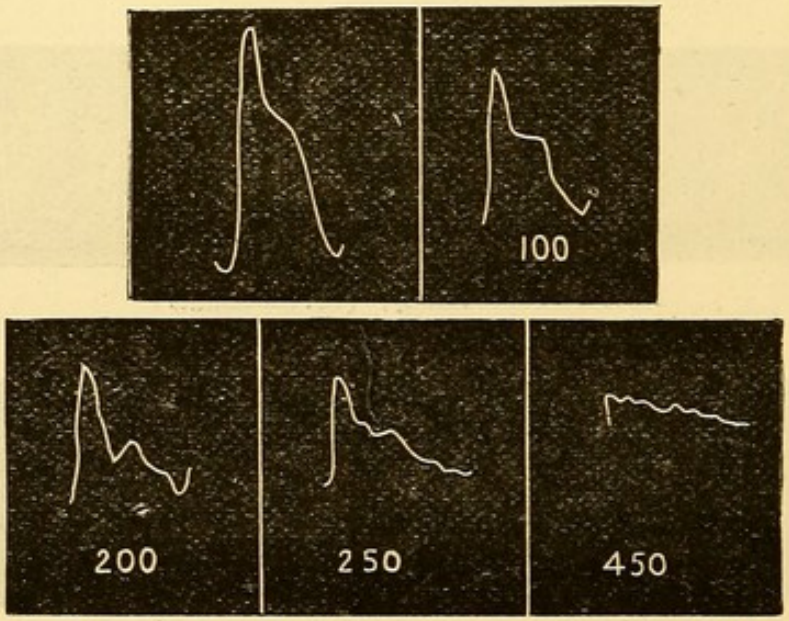


Fig. 13.—Influence of Pressure upon the Form of the Pulse-curve. (From Landois and Stirling.)

CHAPTER III.

MODE OF FEELING THE PULSE.

In examining the pulse, our object is to obtain the most complete and exact knowledge attainable as to the circulation and to interpret accurately the facts we observe; the method to be followed must therefore be carefully described. Three fingers should be placed on the artery, and it will not be amiss to observe the old-fashioned rule of letting the index always be nearest the heart; the different points with regard to the pulse should then be ascertained, each by a distinct and separate act of attention.

The point first to be noted is the frequency—the number of beats per minute,—the regularity or irregularity of the beats as to time, and their equality or inequality in force. This is simple and easy.

We should naturally wish, in the next place, to estimate the force or strength of the pulse, but considerations which modify the idea derived from the impression made on the fingers may first be conveniently discussed. It will be well, therefore, after counting the pulse, to give attention to the size of the artery. This varies greatly in different individuals, and may differ in the two wrists of the same person. It varies, again, greatly according as the muscular coat is relaxed or contracted. We have, then, as a preliminary to any further inference, to distinguish between congenital differences in the diameter of the vessels and variations induced by physiological or pathological influences. Now, a large artery will communicate a more perceptible impression to the fingers placed lightly upon it than a small one, and the beat will seem more forcible. On the other hand,

the pulse wave in a large artery can usually be arrested more readily by pressure, and the pulse is more compressible; one mode of examination thus controls or corrects the other. When the artery is small, and especially when it is rendered small by contraction of its muscular coat, there appears to be little pulsation in it, and the pulse may easily be set down as weak; but let an attempt be made to obliterate it by compression, and it often seems as if the pulse grew stronger as the pressure on the vessel increased.

An important point to be investigated is the degree of constant pressure prevailing in the arteries. The constant intra-arterial pressure or pulse tension is manifested by the degree of fulness of the artery between the beats. To determine this, the artery must first be rolled transversely under the three fingers, or the attempt must be made to do so.

In a pulse of average tension the vessel only stands out, so as to be felt distinctly, during the actual beat, and subsides gradually or rapidly in the interval; it cannot, therefore, be rolled by the fingers at all periods, though it may generally be distinguished with care between the beats, especially when the skin is thin and flexible.

In a pulse of low tension the vessel can scarcely be said to be felt as such at all; it starts up with the beat, and is at once lost again when the brief wave has passed.

In a pulse of high tension, on the other hand, the artery stands out among the structures of the wrist like another tendon, and can be rolled like a cord under the fingers, and followed for some distance up the forearm. While the vessel is thus being rolled about, the pulsation in it may scarcely make itself felt, and the artery can often be seen distinctly, if the skin is thin, projecting on the surface, without any appearance of

pulsation, except where it is thrown into curves. Pressure, however, brings out the pulsation and develops its force.

The character of the beat is another matter for study; and brief as is the period occupied by it, each pulse wave presents a rise, duration, and fall. It may strike the finger suddenly or lift it deliberately; the distension of the artery may be momentary only, or it may persist for a time; the fall of pressure finally may be abrupt or gradual. For the most part, a sudden rise, brief duration, and abrupt fall go together, and constitute the short pulse of large arteries and low tension; while a gradual rise, persistent fulness, and slow decline are usually associated, and give the long pulse of contracted arteries and high tension. Exceptions, however, occur, and they are often of great significance. There may be a large vessel and sudden pulse, when the tension is relatively high, in cases of dilatation of the left ventricle; and a small artery, slowly and feebly filled, in extreme low tension with cardiac weakness.

We are now prepared to estimate the strength of the pulse. Three fingers are placed on the vessel, as is supposed to be the case from first to last. With that nearest to the heart, pressure is made till the wave is arrested, so as not to be felt by the other fingers, or, if necessary, two fingers are employed to extinguish the pulsation. In this way, by the degree of pressure required, and by varying the pressure with one, two, or all three fingers, an idea is obtained of the force with which the heart is propelling the blood onwards. Account will have been taken already of the size of the vessel, and the method of noting the tension will have brought out other points which enter into the consideration.

Here reference may again be made to the statement, already put forward in the preceding chapter,

that the pulse is not an actual expansion of the artery, but a resumption of its cylindrical form by the vessel, which has been flattened by the pressure of the fingers in the interval. It will render more clear some of the differences produced in the character of the beat by relaxation and contraction of the arteries—*i.e.* between low and high tension pulses.

Let the sectional area of the radial artery in a normal condition of average tension be represented diagrammatically by a circle of the size here given.

Between the pulsations it is more or less completely flattened, as shown by the adjoining figure;

and when filled by the increased pressure, which constitutes the beat, it resumes the circular form, yielding again gradually, as the pressure within it subsides. This gives the normal sphygmographic trace—a sharp rise, a moderate height, and a gentle fall, slightly broken by a dicrotic elevation; and a normal pulse as felt by the fingers—a moderately sudden access and moderate vehemence of the beat, which is felt under the finger for a very brief period, and then gradually subsides. (Fig. 14.)

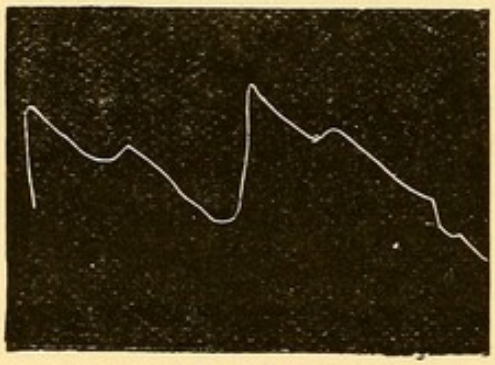


Fig. 14.

When the arterial walls are relaxed the lumen will be larger, and, the mean blood pressure being lower, the vessel will allow itself to be more completely flattened between the beats. The filling of the artery, therefore, and its restoration to the cylindrical form will jerk up the lever of the sphygmograph more rapidly, and will make a greater impression on the fingers. Again, as has already been explained,

the wave will pass more quickly, and the pressure will diminish rapidly. This gives to the sphygmographic trace a more perpendicular and higher upstroke, a sharper top, an abrupt fall, and a marked dicrotic

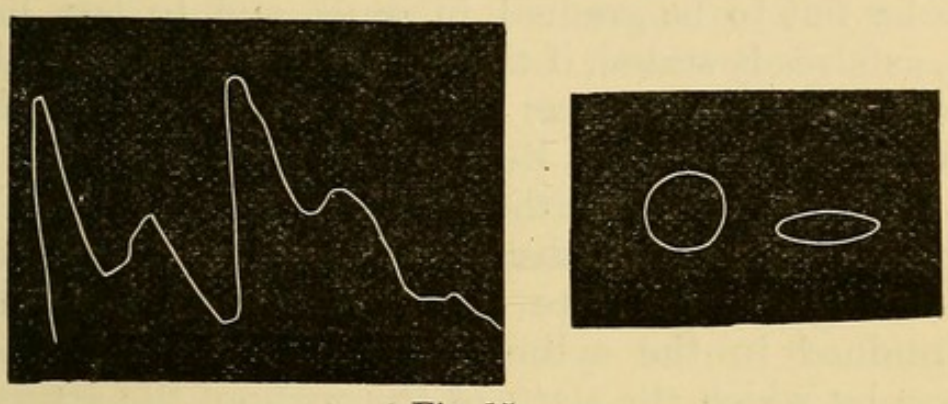


Fig. 15.

rebound. (Fig. 15.) To the fingers the beat is sudden and vehement, but short and dicrotic; while between the beats the artery cannot be felt, or at any rate cannot be rolled at all under the fingers.

Let the artery be contracted, and not only will the difference between its cylindrical and flattened state be less, but, the mean blood pressure being high, the vessel refuses to be flattened by moderate compression. The actual pulsatile movement is small and

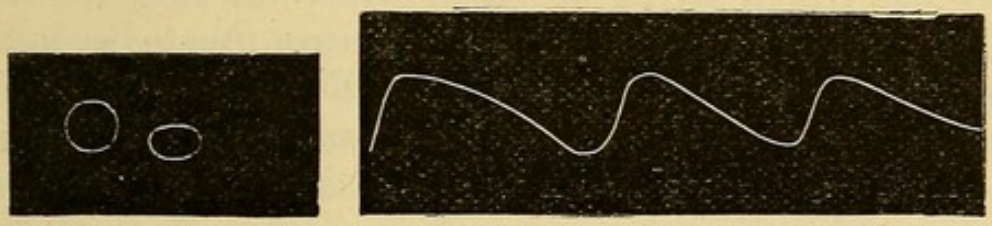


Fig. 16.

inconspicuous, and, not making much impression on the fingers, is often taken to be weak, unless and until the attempt be made to compress the vessel and arrest the beat, when the greater the pressure employed the stronger the pulse seems to be. Then the wave being long, for reasons before given, the sphygmogram will have a sloping upstroke of no great height,

a round top, and a gradual unbroken descent. (Fig. 16.) To the fingers the artery will be distinct between the beats, and capable of being rolled like a cord or tendon, and will not be easily compressed; the beat will be felt to be gradual in onset and to last long, and, as already stated, if the fingers are lightly applied, it will be inconspicuous and apparently weak, while pressure will bring out its real vigour.

I have spoken of the manœuvre of rolling the vessel transversely under the fingers as a means of estimating the pressure and tension by which it is maintained in the cylindrical form. Another manœuvre by which the state of the coats of the artery is ascertained is to carry the skin along it longitudinally, with varying pressure; curves in its course and bulgings in its walls are thus detected, and when the compression is carried so far as to exclude the blood, any inequalities of thickness and density in the coats which may exist are felt, sometimes mere thickening and hardening, at others actual patches of rigidity and calcareous deposit; or the entire vessel may be found to have a thick, leathery, inelastic feel, or may be converted into an irregular, hard, calcareous tube, or may feel like a string of beads under the finger.

One more question of observation must be taken up—the recognition of dicrotism. For this purpose the fingers must rest as lightly as possible on the vessel, and uniform gentle pressure must then be made by all three. The dicrotic wave, when well within physiological limits of variation, will be felt like an echo of the principal beat, and when well marked is extremely distinct.

A complete account of the pulse, then, should specify (1) the frequency: that is, the number of beats per minute, with a note of any irregularity or intermission or instability of the rhythm; (2) the size of the vessel; (3) the degree of distension of the

artery between the beats; (4) the character of the pulsation—whether its access is sudden or gradual, its duration short or long, its subsidence abrupt or slow, note being taken of dicrotism when present; (5) the force or strength of both the constant and variable pressure within the artery, as measured by its compressibility; (6) the state of the arterial walls.

How far the description of the pulse usually given, even in cases in which much turns upon it, falls short of these requirements it is unnecessary to state.

Here a word must be said as to the terminology to be employed in speaking of the pulse; the words quick and slow are capable of two applications: either to the rate at which the beats follow each other or to the character of the individual beats. This ambiguity may be avoided by the use of frequent and infrequent to indicate the number of beats, and of long and short to describe the individual pulsations, as was the practice with old writers on the pulse. A pulsation is long when the increased pressure due to the cardiac systole can be felt by the fingers to last for an appreciable time; it is short when the pressure is quickly gone.

The words strong and weak are even more vague when applied to the pulse than the words quick and slow; they are apt to be used at one time in reference to the mean pressure in the artery, at another in reference to the pulse-wave, the significance being totally different in the two cases. The terms compressible and incompressible are scarcely more definite unless it is stated whether they apply to the artery between the beats or to the pulsations themselves. We must have terms which are incapable of this indiscriminate application. We might speak of the pulse being hard or firm without much risk of this being understood to apply otherwise than to

the general feel of the artery, and therefore to the state of fulness and degree of resistance to pressure in the intervals between the beats. It would scarcely, however, be accurate to say that the *pulse* was firm, although this would be justified by the example of the ancients, who were very exact in their employment of terms, and it is well to avoid as far as possible all latitude of expression. If it is well understood that by tension we mean mean pressure, and that this is estimated by the fulness of the artery and the degree of resistance to pressure between the beats, there can be no better term for conveying an idea of this mean pressure than high, moderate, or low tension. We may then describe the beat as vehement or sluggish, or as forcible or weak, always bearing in mind that the wave may be abrupt or gradual.

The standard or typical pulse of the adult male may now be described. It will have a frequency of 72 beats per minute, will be perfectly regular in time, and the beats will be of equal force. The artery will be of medium size ; with care it can be distinguished among the surrounding structures between the beats, but it yields to pressure, does not give the idea of a cord, and cannot be rolled as such under the fingers ; it is flattened by moderate force, and does not then feel thick or hard. The individual pulse-waves reach the finger nearest the heart with a definite stroke, which can scarcely be described as sudden, still less as sharp; they have sufficient vehemence to be felt by all three fingers, unless decided pressure is made on the vessel, but they can be arrested without difficulty by one finger, the beat then feeling both more sudden and more vehement. The wave, or expansion, or distension of the artery, does not instantly drop, but subsides gently and without perceptible dicrotism.

The sphygmogram corresponding to this description will have a nearly perpendicular rise, a moderate

elevation, a rounded summit, and a gradual, almost unbroken, fall. (Fig. 17.)

It is impossible to examine with attention a large number of pulses, whether among the healthy or the sick, without being struck by the extraordinary diversity of frequency, size, character, tension, and

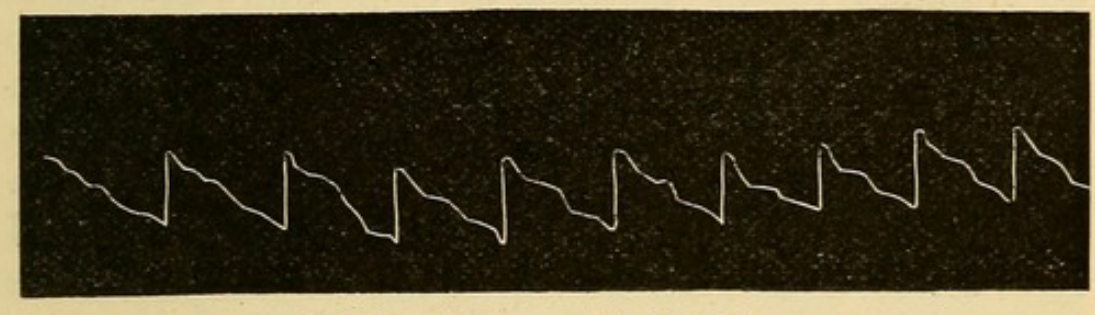


Fig. 17.—Pulse of Adult Male.

force met with. This diversity prevails quite independently of disease in both sexes and at all ages, especially with regard to diameter of vessel and tension and force of pulse. If we judged only according to the size of the blood-channels, together with the pressure within them, we should be compelled to estimate the amount of blood flowing through the arterial system as three or four times as much in some persons as in others. But the amount of blood actually put in circulation is determined by the volume of blood discharged at each systole and the number of contractions in the minute, and, assuming that the ventricles empty themselves at each contraction, there is no such difference in the capacity of the ventricle or in the rate of the heart's action as would corroborate the above estimate. Still, taking everything into account, there must, when we compare the small, short, compressible pulse of one man with the large, firm, and long pulse of another, be great differences in the velocity and energy of the movement of blood through the capillaries in different individuals, and clearly there are great differences in the circulation of the

same person at different times. The fact that such differences are compatible with health and vigour is conclusive evidence that nutrition and functional efficiency, even of the nerve centres, are not in such close relation with and intimate dependence upon the blood supply as we are sometimes apt to suppose; and additional evidence pointing in the same direction is furnished by certain cases of valvular disease of the heart, as, for example, mitral stenosis, and of such functional derangements, for example, as that in which the pulse-rate is only 35 or 40 a minute, and there are two beats of the heart to one of the pulse, which will render the transit of blood through the heart more slow. There must, in effect, be provision for a very large margin of excess beyond the minimum blood supply required for the purposes of the tissues and organs generally. It is a noteworthy fact, too, that in acute disease, which is attended with weakness and wasting, the circulation of the blood is accelerated, as is indicated by the larger diameter of the vessels and the increased frequency of the heart's action.

I have not been able to connect differences in the circulation with any constant bodily or mental character. A marked tendency to obesity appears usually to be associated with small arteries and low tension; and the thin, wiry individual commonly has large arteries, which are conspicuous, not only from the thinness of the skin and absence of subcutaneous fat, but also because, together with their size, they are markedly full between the beats. People with low arterial tension seem to wear out less quickly and to furnish most of the examples of longevity; but they appear to have less power of resistance in acute disease. These, however, are given as mere impressions. Speaking generally, I have found physical strength, energy, and endurance impartially associated with small low tension pulse, and with

large arteries and high tension, and the reverse. I can say the same of intellect, perseverance, courage, and force of character generally ; they appear to be absolutely independent of circulatory conditions. It is, indeed, clear that the circulation is not the determining influence in the production of the differences which are found to exist in respect of bodily or mental energy. The circulation is the servant, and not the master ; and, physiologically, it is tissue activity which conditions the blood supply, and not the blood supply which conditions the tissue changes. In disease, again, the modifications of the circulation which are observed are more frequently effects than causes, and the pulse is an index, not so much of a more or less rapid movement of the blood, to be taken into account as a factor in the morbid processes, as of the state of the nervous system, and of the body generally, which has determined its rate and character.

In order to discuss systematically the indications afforded by the pulse in disease, some sort of analysis must be made of the elements which enter into the consideration. The basis for this is found in the factors of the pulse already enumerated, variations in which constitute the deviations from the normal pulse.

These factors, leaving out for the moment the elasticity of the great vessels, are the action of the heart and the degree of resistance in the peripheral circulation, which, if not entirely dependent upon the degree of contraction of the arterioles, is usually associated with variations in their diameter.

The heart determines the frequency, rhythm, and force of the beats, and to it are traceable any deviations from the normal in respect of these elements in the production of the pulse. The vessels determine the size of the channels by which the blood driven into the aorta at each systole reaches the capillaries, and therefore the rate of its flow and the duration of

the pressure—*i.e.* the character of the individual pulsations.

The following is a tabular view of the variations from the normal pulse referred to the heart and arteries respectively :—

			Pulse.
Heart	Frequency		Frequent.
			Infrequent.
	Rhythm		Irregular.
			Intermittent.
	Force		Excessive.
			Defective.
Arteries.	Relaxed.		Large.
			Short.
			Low-tension.
	Contracted.		Small.
			Long.
			High-tension.

This classification of the deviations from the normal pulse will serve as a guide in their consideration, but it would be impossible, even if it were convenient, to discuss quite separately and independently the variations due to the heart and those due to the vessels. The heart and arteries are, after all, only parts of the same system correlated throughout by mutual interdependence and by the vaso-motor nerves, and abnormal frequency of the heart's action is usually associated with relaxation of the peripheral vessels, and contracted arteries with deliberate heart-beats. Some of the more common combinations may indeed, with advantage, be enumerated.

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Frequency and force of the heart's action, with dilated arteries, give the pulse of sthenic fever, and of violent but not excessive muscular exertion, which is frequent, sudden, vehement, large, short, and dicrotous.

Frequency and force, with arteries moderately contracted, give the pulse of excitement, of an early

stage of effort, and of the pyrexia attending some forms of inflammation; with extreme contraction of the arteries, the pulse of peritonitis and of severe rigor.

Frequency, with deficient force on the part of the heart and relaxed arteries, gives the pulse of asthenic fever and of exhaustion, which, while frequent and sudden and short, has no vehemence; the dicrotic wave is sometimes so marked that it is almost as distinct as the primary wave. The artery is large, except when the cardiac weakness is extreme.

A normal or slightly slowed heart-rate, with diminished force and relaxed arteries, gives the pulse of fatigue and of convalescence from acute disease, both force of heart and arterial tone increasing as convalescence advances.

Normal or slightly diminished frequency and increased force of the cardiac systole, with contraction of the arterioles, constitutes the so-called renal pulse, of which much will be said hereafter, but it is common to kidney disease and to a variety of other conditions with which high tension of the pulse is associated.

It may be well to mention here the visible pulse and the recurrent pulse.

The radial pulse may be visible simply from the fact that the artery takes an abnormal course, and runs very superficially immediately beneath the skin, or when the patient is spare and the skin thin. The pulse, however, becomes visible from pathological causes, and this has serious significance. The visible pulse is most marked in aortic regurgitation, the vessel becoming empty and collapsed between the beats, and the blood rushing into it with extreme suddenness and violence. It is rendered particularly distinct by holding the hand up above the level of the head, when the column of blood, not having the support of the aortic valves, drops out of the artery and is shot into it alternately. It may, indeed, be overlooked when the

hand is hanging down, as is the case when the patient is in the sitting position while the pulse is being felt.

The radial pulse is usually audible as well as visible, and is associated with conspicuous throbbing of the carotids, and with visible pulsation in the temporals, facials, brachials, etc.

In the circumstances described, the arterial tension is at its lowest, but the pulse is often visible at the opposite extreme of very high tension, when from persistence of the pressure within the vessels they have become enlarged and tortuous. It is in virtue of the tortuosity that the pulsation is seen, and the visibility consists of an exaggeration of the curves present in the course of the vessel.

The recurrent pulse is also met with in exactly opposite conditions of the circulation. What is meant by the term is, that when the radial artery is completely closed by pressure with one or more fingers, and the direct pulse is arrested, a feeble and retarded beat can be felt in the distal part of the vessel, which is produced by a wave which has come round from the ulnar through the palmar arch. The abnormal patency of this communication occurs, on the one hand, when the arteries are relaxed and the tension is low, as in pyrexia. A vigorous cardiac systole is necessary to propel the wave, and accordingly it is in pneumonia rather than in fevers that it is felt. It will often be present in aortic regurgitation. The palmar circuit is often the channel for recurrent pulsation, on the other hand, in chronic high tension, when the arteries are dilated and worn out by protracted distension.

CHAPTER IV.

THE HEART-SOUNDS IN RELATION TO THE PULSE.

The heart and arteries forming together one system and being in close mechanical as well as functional relation, the varying conditions of the circulation manifested by the pulse will be attended with modifications of the sounds of the heart corresponding in significance with the variations in the character of the pulse. The pulse and heart-sounds, in effect, corroborate, and often extend, each other's indications, and it is always useful and often necessary to combine the information to be obtained from both in order to realise fully the state of the circulation.

This is all the more true from the fact that there are in effect two hearts, pulmonary and systemic, bound up together in the one organ, and changes in the pulmonary circulation are intimately associated with and throw light upon changes in the systemic circulation. There is no pulse of the pulmonary artery accessible to the finger, and we have only the sounds of the right heart to inform us as to the varying conditions of blood pressure in this vessel. In examining the heart, then, with the object of connecting modifications of its sounds with variations in the character of the pulse, it is necessary to bear in mind not only that each ventricle, left and right, and each artery, aortic and pulmonary, has its own sounds, which must not be confounded, but that comparison and even contrast of the sounds of the two sides of the heart may be required in order to bring out the full significance of the pulse.

The left ventricle first sound.—The first sound of the left ventricle is best heard at the apex,

where it has the prolonged and so-called dull character which has led to the employment of the imitative syllable "lub" to represent it in the familiar "lub-dup" which is supposed to convey an idea of the heart sounds. A little to the left of the apex beat it is usually quite as distinct and loud, and it is here less liable to confusion with the first sound of the right ventricle.

It is heard again in the aortic area, to which point on the surface of the chest it must be conducted by the aorta; but at this spot its intensity is extremely variable, not corresponding necessarily with the degree of loudness at the apex. Apparently, indeed, the character of the sound affects its conduction along the aorta, the dull and prolonged first sound of hypertrophy, as heard at the apex, being lost at the aortic area, while the shorter first sound of dilatation is distinctly heard here. Whatever the explanation, the clinical fact is that with high arterial tension, with or without hypertrophy of the left ventricle, its first sound becomes weak, and at times inaudible at the base of the heart, while with low arterial tension and, again, with dilatation of the left ventricle, the first sound is distinct.

The aortic second sound.—The second sound of the left side of the heart is best heard close to the right edge of the sternum at the level of the second intercostal space or third costal cartilage. This, which is called the aortic area, is, of course, not immediately over the valve, which is deeply situated behind the conus arteriosus of the right ventricle, and beneath the left side of the sternum at about the level of the third intercostal space, but marks the spot at which the aorta approaches or touches the chest-wall. It is short and sharp, varying in loudness or intensity in different individuals, and according to the vehemence and sharpness of the ventricular systole, and according to the degree

of pressure in the arterial system. The aortic second sound is again heard at and to the left of the apex, conducted thither by the wall of the left ventricle, and it is an important fact that it is frequently as distinct here as in the aortic area, sometimes far more so, while frequently the modifications which it undergoes, especially in association with high arterial tension, are better marked. At the base it is often difficult to make sure that the pulmonary second sound does not invade the aortic area, and it may be necessary to listen over the carotids in order to exclude this source of confusion. Of the second sounds it is the aortic which alone is audible at, and especially to the left, of the apex; however loud and accentuated the pulmonary second sound may be, it is not conducted by the walls of the left ventricle, and does not, therefore, reach the surface of the chest where this part of the heart only is in contact with the chest-wall. When the right ventricle is so far enlarged by dilatation and hypertrophy as to displace the left and usurp the situation of the normal apex, the pulmonary second sound will, of course, be here heard, but even then it is not conducted to the left of the apex beat.

The right ventricle first sound.—The right ventricle first sound is heard over a considerable area, from the mid-sternal line outwards to within an inch of the apex beat, and from the seventh costal cartilage upwards as high as the third, but best over the lower costal cartilages from the left edge of the sternum upwards and outwards for about an inch. It is always shorter and usually louder than the first sound of the left ventricle, notwithstanding that the pressure in the pulmonary circulation, and therefore the force of the systole, is less. This is probably due to the close contact of the right ventricle with the chest-wall, but, perhaps, also to its own thinner wall, which will lend itself better, either to

the production, or to the convection of sound, than a thicker mass of muscular substance.

The pulmonary second sound.—The second sound of the pulmonary artery is most distinct close to the sternum in the third left intercostal space (the valve lying behind the sternum at this level), and along the course of the pulmonary artery upwards and outwards as high as the second space, and for an inch to the left of the edge of the sternum. It is audible also over the right ventricle generally, and is more or less conducted by the sternum. It is usually louder than the aortic second sound, probably because the pulmonary artery is superficially situated. This, at any rate, is the conclusion resulting from my observation; but some observers consider the aortic second sound to be the louder of the two. The relative intensity of the sounds of course varies under different conditions of the circulation.

Further description or account of the sounds will be unnecessary, as it is assumed that the reader is familiar with them.

The variations to which they are subject will be as follows, and they must be considered seriatim, although they cannot be altogether disentangled one from another:—

1. Variations in intensity, collective and individual.
2. Variations in character.
3. Variations in rhythm or time.

The collective variations in intensity will be such as are due to the varying force of the heart's action in excess of or below the normal average. The second sound of the two sides of the heart does not necessarily correspond with the first; in most circumstances it will be louder and more distinct when the first is louder—it is especially loud and clear when the ventricular systole is rapid, but when the heart is

acting frequently as well as forcibly the first sound of both ventricles may be loud and the second feeble, the amount of blood discharged by the ventricles being small; and, on the other hand, in feeble action of the heart, whether due to functional debility, or to structural disease, fatty or other, the second sound at both orifices may be distinct when the first is inaudible.

It must always be borne in mind that the sounds may be disguised by overlapping lung either in deep-chested individuals, when the heart may normally be partially or completely covered, or when the lungs are brought over the heart by emphysema. The mitral and aortic sounds are soonest and most effectually muffled, the apex, on the one hand, being covered by a thick cushion of the left lung and the aorta, on the other, displaced from its proximity to the chest-wall by the overlapping edge of the right lung. This source of error having been pointed out, will not further be alluded to when the individual valves are under consideration.

Modifications of the left ventricle first sound.—Taking the valves separately and in order. The first sound of the left ventricle is seldom much louder than normal, except when the heart is acting powerfully and the sounds are collectively intensified. When there is obstruction to overcome, either at the aortic orifice or in the peripheral vessels, and greater force of systole is required, the contraction of the ventricle is more deliberate, and this prevents the sound from being correspondingly loud; such is especially the case when the ventricle is hypertrophied.

It is in dilatation of the left ventricle that its first sound is most liable to be loud from the sudden tension of the thin walls. When the pulse tension is low, and the heart's action excited, as in pyrexia, there is also usually a disproportionate intensity of

the first sound. In mitral stenosis, again, for reasons not thoroughly understood, it is loud. Whenever loud it is short, but this is particularly marked in the last-named valvular affection.

The left ventricle first sound is often weakened and sometimes lost in typhoid fever, as a result of the granular degeneration in the muscular structure of the heart which takes place in this disease; its complete extinction is a grave element in the prognosis. In mitral incompetence, when the regurgitation is considerable, the first sound ceases to be audible, and is replaced by a murmur. It is not that the sound is masked or overpowered by the murmur; it is lost altogether. When the regurgitation is small in amount, there may be a loud murmur, but the first sound is distinctly heard, together with the murmur.

Accentuation of the aortic second sound.—The aortic second sound is intensified relatively to the sounds generally, and to the pulmonary second sound in particular, or accentuated, as it is usually termed, whenever there is high blood pressure in the arterial system—*i.e.* in the whole range of conditions attended with high pulse tension, and especially when the ventricular systole is reinforced by hypertrophy. The valves are carried back more forcibly by the high pressure in the aorta, the walls of the aorta are more tightly stretched, and therefore more ready to produce or to transmit sonorous vibrations. A sort of accentuation of the aortic second sound is producible in an exactly opposite state of the circulation: when the pressure in the arterial system is low the ventricular systole, if at all vehement, meeting with little resistance, is carried through very rapidly, and its contents being shot quickly into the aorta the elastic rebound is sharp, causing the second sound to be loud. The distinction is that the accentuation is not relatively marked; the

first sound will be loud and short, and the pulmonary second sound intensified as well as that at the aortic valves.

The accentuated aortic second sound is heard not only at the right second space, but often in the space below, and always, or nearly always, in the carotids; it is extremely distinct also at and to the left of the apex, as already said. With accentuation there is often a change of pitch and character of this sound, which is of great importance, and will be spoken of later.

It is relatively weaker when the left ventricle is dilated; again, and in a greater degree, in stenosis of the mitral orifice in consequence of the diminished amount of blood which enters and leaves the ventricle; it is again weakened in aortic stenosis, and especially in aortic regurgitation, in which affection it may be altogether absent, and the presence or absence of the aortic second sound in the neck over the carotids is a point of diagnostic and prognostic significance.

Intensification of the right ventricle first sound.—The first sound of the right ventricle is intensified whenever there is decided obstruction in the pulmonary circulation, so long as its muscular structure is sound and so long as it is not overpowered by the resistance. All diseases of the left side of the heart sooner or later tell back upon the pulmonary circulation and produce an effect on the right ventricle; mitral stenosis and regurgitation from a very early period, aortic regurgitation and obstruction at a later stage especially when secondary mitral incompetence has been induced, and usually before this is indicated by a murmur. Aortic regurgitation affects the right ventricle more quickly than obstruction. Dilatation of the left ventricle, again, very soon makes its effects felt in the pulmonary circulation and in the right heart. When the right ventricle is over-distended,

or when tricuspid regurgitation has been established, its first sound may lose the intensity which it has possessed at an earlier period.

To these cardiac causes of intensification of the right ventricle first sound must be added obstruction to the passage of blood through the lungs by affections of the lungs themselves or of the air passages, by bronchitis and emphysema, pneumonia, and effusion into the pleural cavity, more rarely by malignant disease in the lung or mediastinum, or pressure on the root of the lung by aneurismal or other tumour. Imperfect aëration of the blood from whatever cause gives rise to resistance in the pulmonary capillaries, and to more powerful action of the right ventricle, and in this way laryngitis, croup, and other affections of the air-passages, which produce cyanosis, cause intensification of its first sound.

The right ventricle first sound may be weak or absent in fatty degeneration of the heart at a comparatively early period of the disease, and when the existence of this affection is suspected special attention should be given to the sounds of the right heart.

Accentuation of the pulmonary second sound.—The pulmonary second sound is still more sensitive to increase of pressure in the pulmonary circulation than the right ventricle first sound, and no examination of the heart can be considered complete unless the relative intensity of this sound has been carefully estimated. Accentuation of the pulmonary second sound, it is true, is produced by slight causes, and may be simulated by uncovering of the conus and artery from retraction of the lung; but these sources of fallacy are easily eliminated by the exercise of a little thought and care, and then the pulmonary second sound becomes a means of recognising increased pressure in the pulmonary circulation at its earliest stage, long before this tells on the right ventricle, and

of estimating the extent of valvular or other disease of the left side of the heart by its effects on the passage of the blood through the lungs. All the causes of intensification of the right ventricle first sound will give rise to accentuation of the pulmonary second sound, and need not again be enumerated.

Heart-sounds: variation in character.— The first sound of the left ventricle may be prolonged and dull as compared with the normal; or, on the other hand, short and sharp. The former modification is usually associated with hypertrophy, the latter with dilatation; but the first sound is short whenever the heart is acting frequently, especially when the arterial tension is low, and there is little resistance to the contraction of the ventricle; it is often specially short and sharp in the early stage of pericarditis. But the most remarkable modification of the first sound as heard at the apex, and presumably therefore that of the left ventricle, is that met with in the late stages of narrowing of the mitral orifice, when it is loud, short and sharp, and high-pitched, and so completely resembles the second sound as often to be taken for it.

The principal modification in the character of the aortic second sound, which requires notice so as to mark the distinction between it and accentuation, is a change in the pitch and its having a musical tone or ring.

In order that the significance of this change may be fully understood it will be well to explain exactly the production of the second sound. The idea, not altogether extinct, that it is the click of the semilunar valves as they meet and are forcibly driven together by recoil of the blood in the aorta is mentioned only to be dismissed; the sound is one of sudden tension and vibration, not of sharp contact. The vibration and tension, however, which give rise to the second

sound, whether at the aortic or pulmonary orifice, are not limited to the valvular cusps, but affect the arterial walls for some distance from the valves. The second sound thus gives information as to the state of the aorta as well as of the valves. Were it not that the entire root of the aorta forms part of the vibrating membrane, dilatation of this part of the vessel and thinning of its walls would have no great effect upon the second sound; whereas, modifications of the second sound are diagnostic of such changes, and in this way are of extreme importance. Just as a cord of longer length, when suddenly stretched, yields a lower pitch, so does the larger diameter of a dilated aorta lower the pitch of the second sound; just, again, as the note becomes clearer and more musical as the cord is thin and homogeneous, so does the second sound acquire a musical tone when the coats are attenuated, and the three tunics are fused into one membrane in disease.

Dilatation of the aorta rarely takes place except when there has been protracted arterial tension; when, therefore, there has long been accentuation of the aortic second sound—and with accentuation the pitch of the sound is lower—it may be inferred that dilatation, simple or aneurismal, has supervened. When the second sound has a distinct musical tone or ring it may be inferred that the dilatation is associated with thinning of the coats of the vessel, probably uniform, and therefore, not aneurismal, except indeed, in case of aneurism of one of the sinuses of Valsalva, which may be attended with a ringing second sound.

Another point to be noted in connection with the lowered pitch and sonorous tone-like character of the aortic second sound when the aorta is dilated is that it is audible over a very large area. This is not merely because the enlargement of the aorta brings it into more extensive relations with the wall of the

chest; but because the area of contact with the lung is increased, and from the larger conducting surface the sound is transmitted in all directions. As it is the right lung which lies in contact with the ascending aorta, it is over the right side of the chest that the sound is most extensively heard, but very commonly it is again caught on listening over the upper part of the left thorax just beyond the pulmonary artery.

The modifications, independent of the intensification already described, of the first and second sounds of the right side of the heart are less noteworthy and have no particular significance; they need not, consequently, occupy our attention. Mention may be made of a peculiar click which sometimes accompanies the first sound just to the left of the ensiform cartilage when the right ventricle is acting forcibly, and of a kind of scratching sound sometimes heard at the same spot. The latter occasionally gives rise to a suspicion of pericarditis, and it may perhaps be due to the friction which is supposed to contribute to the formation of a white patch often seen on the corresponding part of the heart. The click appears to be endocardial, but it means nothing.

Modifications of the rhythm of the heart-sounds.—The term rhythm is here employed in its strict sense, as denoting the time-relations of the first and second sounds of individual beats, and not the regularity of the succession of heart-beats.

The two sounds of the heart so follow each other as to correspond normally with the first two beats of triple time in music, "one, two, three"—*i.e.* if in counting "one, two, three; one, two, three," the "three" were silent, the "one, two, —, one, two, —; one, two, —;" would represent the first and second sounds, the silent "three" the long silence. The accent would be on the "one" or the "two:" "**one**′, two, —," or "one, **two**′, —," according to the way

in which the heart was acting, or according to the point at which the stethoscope was applied.

The relative length of the two intervals between the first and second, and between the second and the next first, giving the triple musical time, is maintained with remarkable constancy, whatever may be the rate at which the heart is beating—whether, that is, the pulse is 60, 90 or 120 in the minute, and this whether the increased frequency is from exercise, excitement, warmth, stimulants, or fever. Under certain conditions, however, the triple time is departed from in contrary directions. The cardiac systole may be prolonged, and thus the interval between the first and second sounds increased, and this may be carried so far that the sounds become equidistant. The musical time would then be double instead of triple, and we should count "one, two; one, two;" instead of "one, two (three); one, two (three);" and the sounds may be compared to the "tick-tack" of a pendulum, or of a watch, according as the heart's action is slow or rapid.

The same result might come about by a shortening of the diastole, and an abbreviation of the interval between the second sound and the first.

The spacing of the first and second sounds by the prolongation of the interval between them indicates, of course, that the systole is longer than usual in completing itself, and this occurs, as might be inferred, when there is resistance in the peripheral circulation. But physiological resistance, while rendering the systole more deliberate, is accompanied by a corresponding increase in the duration of the diastole, so that the effect is to diminish the frequency of the heart's action, the rhythmic succession of the sounds in triple time not being disturbed. It is when the peripheral resistance is such as to affect the heart injuriously, and dilatation of the ventricle has set in, or is impending,

that the systole is prolonged at the expense of the diastole, and the sounds tend to become equidistant. This occurs as a result of protracted high arterial tension, especially in kidney disease, and the effect on the ventricle is induced all the more readily when the heart is weakened, together with the muscular tissues generally, by pyrexia. In acute renal dropsy, therefore, when there is at the same time the debilitating influence of acute disease upon the heart, and sudden increase of the resistance in the circulation, spacing of the sounds is often observed.

Equidistance of the sounds, from shortening of the diastole, occurs in palpitation with extreme frequency of the heart's action. The sounds then resemble the ticking of a watch under the pillow, or the sounds of the fœtal heart in utero.

On the other hand, the systolic interval may be shortened or the diastolic interval lengthened till the rhythm is that of common time—"one, two, (three, four); one, two, ––, —;" or even "one, two, (three, four, five, six)." In extreme cases the second may follow the first quite precipitately.

The approximation of the first and second sounds may be due to the rapid completion of the systole, when the ventricle contracts energetically and meets with little resistance, as may occur sometimes in pyrexia, and in states of excitement; but this is not common without a corresponding abbreviation of the diastole, which maintains the rhythm. More frequently, when the second sound follows the first too rapidly, it is that the systole is not completed at all, from weakness of the ventricular muscle or from resistance in the arterial system which it is unable to overcome. The ventricle discharges only a portion of the contained blood, sometimes a very small proportion of its contents, when the contraction is brought up short, so to speak, and ends unfinished; then, as

soon as the pressure in the aorta exceeds that in the ventricle, which may be the case before the effort on the part of the ventricle ceases, the second sound occurs.

Associated with the approximation of the sounds is a corresponding shortness of the pulse, but this is not so striking or so significant as the rapid succession of the sounds, the latter being an extremely serious prognostic sign. In diphtheria it is often the first recognisable indication of the fatal cardiac asthenia which attends this disease, preceding sometimes by forty-eight hours or more other symptoms of heart-failure, and under various circumstances it may for a time stand almost alone as a warning of impending peril.

Occasionally the hurried succession of the two sounds succeeds to an exactly opposite departure from the cardiac rhythm in high arterial tension, the sounds, from being equidistant, going to the other extreme, the second coming close upon the first.

In a case recently seen with Dr. Habershon and Mr. Pedler the serious prognostic significance of this sign was verified by rapidly fatal failure of the heart three weeks after apparent recovery from an attack in which it was present for some time. The patient, aged fifty-four, a tall and powerful man, of remarkable mental vigour, was seized with cardiac symptoms after an indiscretion in diet and copious draughts of iced water on a hot day. There was at first severe pain in the epigastrium and in the region of the colon, apparently from flatulence, but without much distension, and with this a sense of oppression in the chest and extreme prostration; frequent deep sighs and great restlessness. It was observed also that when he spoke, which was usually in rather a loud voice, the abdominal muscles contracted forcibly to reinforce the thorax in producing the requisite pressure of air, and that he could say only a few words

at a time without taking breath. There was not, however, paralysis of the diaphragm giving rise to reversed abdominal movements in respiration. The mental condition was peculiar and extremely variable; he would be excited and almost violent at one time, at another quite himself; the memory was greatly impaired. The pulse, which in health was 60 or under, ranged between 70 and 80. It was large for the most part, regular, and did not seem to be specially weak, but it was extremely short; and on examining the heart the second sound was found to follow the first very hurriedly, the first being itself short and for a time weak. The shortness of the pulse was the more remarkable from the fact that the history of the patient and his appearance were suggestive of high tension. Stimulants had to be given freely at times to avert threatened syncope, but the average daily amount was moderate. The remedies employed were chiefly digitalis with ammonia and strophanthus with bark or nux vomica. After a fortnight of serious danger the patient gradually recovered, and was able to leave town after an illness of about a month. Three weeks later another attack came on, which proved fatal in a few days.

The patient was gouty, and had had several attacks of thrombosis of veins; this led to a suspicion of the formation of a coagulum in the heart, but no evidence, direct or indirect, of any such occurrence was present. The cerebral symptoms were no doubt the result of anæmia of the cortex caused by the inefficient contraction of the left ventricle.

Reduplication of the heart sounds.—There remains to be noticed the reduplication of the heart sounds which is so frequently met with. Either the first or the second sound may be reduplicated; occasionally, but rarely, both at the same time.

Reduplication of the second sound may be taken first, since there is no great difference of opinion as to its causation, and it is generally recognised as being due to a want of synchronism between the aortic and pulmonary second sounds; this asynchronism, again, being the effect of increased resistance in the pulmonic or systemic circulation (usually the pulmonic), which retards the completion of the systole of the corresponding ventricle, and therefore the closure of the semilunar valves. Instead of "lub-dup" we have "lub-dullup," or "one, two-two," instead of "one, two." Sometimes the two second sounds are so far apart as to give rise to a resemblance to the foot-fall of a horse at the canter; and we have the *bruit de galop* or cantering heart-sounds, and different varieties of the canter are imitated according to the relative intensity of the sounds and the fall of the accent on one or other. We may have "**one**′, two-two; **one**′, two-two;" or "one, **two**′-two; one, **two**′-two;" or "one, two-**two**′; one, two-**two**′." At other times they succeed each other with the utmost rapidity, and are only distinguished by the practised ear. The reduplication of the second sound is usually most distinct at the base of the heart, and best heard over the left edge of the sternum at the level of the third space; but it may be audible to the right of the sternum in the second space and across the sternum between these two spots, or at the fourth left space, rarely upwards along the course of the pulmonary artery. Occasionally the reduplication is more distinct near the apex than elsewhere. Sometimes it is the aortic second sound which is first, sometimes the pulmonary, and it is not at all an easy task to determine which of them leads. Fortunately, though it is interesting to make this out, it is not important, and it is almost always the pulmonary second sound which is displaced, whether forwards or backwards, as regards the aortic sound

That such is the case is seen from the fact that reduplication of the second sound can be induced by merely holding the breath; this fact shows also that it is producible by comparatively slight causes.

The causes of reduplication of the second sound are mostly such as tell upon the pulmonary circulation. Bronchitis and emphysema or fluid in the pleural cavity may give rise to it. Far more commonly it is produced by disease, valvular or structural, of the left side of the heart, and particularly by mitral stenosis, which it may attend at almost all stages, but especially early. Reduplication of the second sound, indeed, is probably heard as frequently in association with obstruction at the mitral orifice as with all other causes put together. It is often heard, again, in pericarditis, which may be accounted for by the inflammation of the serous covering which paralyses subjacent muscular fibres, affecting more seriously the thin walls of the right ventricle than the thick walls of the left. Occasionally reduplication of the second sound is present in renal disease, and is apparently due to the high systemic arterial tension to which this gives rise. Occasionally, again, reduplication of the most marked character is heard in cases of cerebral tumour and of cerebral disease of other kinds, and is one of the many effects on the circulatory system produced by affections of the nervous system. It is probable, however, that it is indirect, and that the intermediate agency is high arterial tension.

Reduplication of the first sound is generally considered to be strictly analogous to reduplication of the second, and to be due to a want of synchronism between the first sounds of the two ventricles, and therefore between the first moment of their contraction, since tension of the auriculo-ventricular valves, which is the most important element in the first

sound, must occur as soon as the ventricular walls begin to exercise pressure on the contents. This seemed to be conclusively demonstrated by my late teacher and colleague Dr. Sibson; but Dr. Geo. Johnson does not accept the explanation, and considers that the reduplication is caused by the auricular systole giving rise under certain circumstances to an audible sound. The question must be discussed, but before entering upon the arguments adduced in favour of one or other of the two views, it will be well to state the facts of observation with regard to the reduplication.

The first is as to the point at which the double sound is best heard. This is near the apex of the heart, and especially about an inch to the inner side of the apex beat. The spot indicated corresponds very nearly with the interventricular septum, and the stethoscope applied here would be over both ventricles. Very commonly to the right or left of a given spot the first sound seems to be prolonged or confused, and is sometimes described as impure, but at the point itself the prolongation is distinctly resolved into the two elements which constitute reduplication.

Sometimes the reduplication is quite distinct along the lower border of the right ventricle from near the apex-beat to the edge of the sternum; more rarely it is recognisable in an upward direction as high as the third space.

Reduplication of the first sound is not, as a rule, so striking as reduplication of the second, and in most cases must be sought for, but it may be so well marked as to produce a cantering rhythm "one-one′, two; one-one′, two; one-one′, two." Occasionally it is difficult to say at the first moment whether cantering sounds are due to reduplication of the first or second; whether they are represented by "one,

two-two; one, two-two;" or by "one one, two; one-one, two;" and some care may be required to establish the distinction.

The most common cause of reduplication of the first sound is high pressure in the systemic circulation or high arterial tension, and it is met with most frequently in renal disease. It may also be produced by obstruction in the pulmonary circulation, such as attends bronchitis and emphysema; but this is not frequent, and it does not occur when the obstruction is due to valvular or other disease of the left side of the heart.

On the hypothesis that the reduplication is due to loss of synchronism between the first sounds of the two ventricles, one explanation is that the resistance in the systemic or pulmonary circulation delays the action of the corresponding ventricle. Dr. Barr in a very interesting paper in the *Liverpool Medico-Chirurgical Journal* argues very forcibly that, on the contrary, the ventricle which is working against undue pressure is the first to contract; "relatively greater blood supply," he says, "to one or other ventricle does not retard the closure of the auriculo-ventricular valve, but more quickly overcomes the inhibitory action of the vagus, stimulates that ventricle to initiate contraction, and first apply tension to its auriculo-ventricular valve, which perhaps may be the more readily effected on account of the hyper-distension of the ventricle, and thus produce the first element of a duplex sound." I am more disposed to accept Dr. Barr's conclusion than his reasons; I do not see that the left ventricle has any relatively excessive blood-supply, or that it undergoes hyper-distension during diastole in Bright's disease; and I distrust explanations which turn upon the influence of the pneumogastric or sympathetic nerves.

But it is not the mere existence of abnormal

resistance which has to be overcome which gives rise to reduplication. The occurrence of reduplication shows that the resistance is overtaxing the heart, and that the hypertrophy, which for a time meets the increased resistance, is no longer equal to the task, and is beginning to give place to dilatation.

It has been already said that Dr. Geo. Johnson considers one of the two elements of the reduplicated first sound to be auricular and the other ventricular The starting-point and basis of his hypothesis is that in pericarditis the auricular friction gives rise at the base of the heart to a triple instead of a to and fro rub, and that the auricular systole, preceding as it does the systole of the ventricle, were it audible would, with the second sound, give exactly the triple sound produced by the reduplication in question. It may be said in passing that basic friction sounds are often still more complex, and that they are suggestive rather of the sound of a distant loom than of a cantering horse. Dr. Johnson then endeavours to show that the contraction of the auricle may under certain circumstances give rise to a sound, and that the reduplication is most marked at the point where this would be heard. Here I am at issue with Dr. Johnson on a matter of observation: his hypothesis requires that the reduplication should be heard at the base of the heart near the left or right auricle respectively, and he states that it is frequently, if not constantly, audible here. This is certainly not in accordance with my experience. For some years—ever since, in fact, the publication of Dr. Johnson's views—I have rarely omitted, on hearing the first sound reduplicated, to ascertain its seat of maximum distinctness and intensity, and to compare the sounds as heard at the base and near the apex; and the result of repeated careful and conscientious examination has been to confirm the original observation of

Dr. Sibson that the seat *par excellence* of the reduplication of the first sound is along the line of the interventricular septum where the stethoscope is over both ventricles. The duplicated sound is often heard along a horizontal line from the apex towards the sternum as far as the edge of this bone, less frequently upwards to the third intercostal space. When thus audible over the right ventricle the explanation is that the first sound of the left ventricle is audible through the shallow right ventricle which overlies it; and when it is heard in an upward direction it is that, in consequence of hypertrophy and dilatation of the left ventricle, the septum comes to run nearly along the left border of the heart instead of going round to its posterior surface.

As regards theoretical considerations, again, the conditions under which reduplication of the first sound arises are not such as to put stress upon the auricle, or to render its contraction audible, were this liable to occur. The high arterial tension and resistance in the peripheral circulation affect the ventricle, not the auricle; the extra effort required to expel the blood from the ventricle in systole does not imply hindrance to its influx from the auricle during diastole and, until the mitral valve gives way and becomes incompetent, there is nothing to give rise to dilatation or hypertrophy of this cavity. Having frequently seen and felt and applied the stethoscope to the naked auricle, when assisting Dr. Sibson in his investigation of the changes of form which the heart undergoes in systole and diastole, I cannot understand how its contraction could under any circumstances give rise to an audible sound.

That the reduplication of the first sound is due to asynchronism of the initial systolic contraction of the ventricles appears to me to be placed beyond the reach of doubt by two facts. The first is one which was

originally adduced by Dr. Sibson—namely, that if the two cusps of a double stethoscope are placed one over the right and the other over the left ventricle, at points where on separate examination only a single sound, that of the subjacent ventricle, is audible, the two sounds are found not to coincide and the reduplication is heard. The second is that the asynchronism can often actually be felt, as well as heard, by pressing the fingers or the ball of the hand well into the intercostal space just to the inner side of the apex beat. When the impulse is distinct and the reduplication well marked, in a thin subject, two beats, clearly recognisable as belonging, one to the left, the other to the right ventricle, are felt quite distinctly.

This I have habitually pointed out in the wards of St. Mary's Hospital for many years, and the observation has been corroborated by a long series of resident physicians and clerks.

CHAPTER V.

INCREASED FREQUENCY OF THE PULSE.

As has been already said, many of the indications obtained from the pulse do not depend upon a comprehension of the circulatory conditions which the varieties of the pulse denote, or, indeed, upon a knowledge of the circulation at all. The ordinary diagnostic and prognostic indications of the pulse are learnt only by experience. Observant physicians before the time of Harvey could gauge thoroughly the state of the patient in fever from the pulse, and it is not for the purpose of estimating the movement of the blood that we ourselves, in a case of fever, count the beats and note their force and volume. We calculate from the data thus obtained the strength of the sufferer and the effect upon him of the disease; and we might or might not do this more accurately than our predecessors. On the other hand, it is only through a knowledge of the conditions which govern the circulation that such facts as the connection between kidney disease and cerebral hæmorrhage can be understood, and that the prognostic significance of the hard pulse, which betrays this connection, can be appreciated.

I propose, then, to consider first deviations from normal frequency, and abnormalities of the rhythm; next variations of tension, their causes and consequences, and the indications for treatment which they furnish.

The average frequency of the pulse in the adult male is 72 beats per minute; in the female about 80; in the child it is much more frequent, and it gradually loses in frequency from infancy onwards. There are

slight diurnal variations, traceable perhaps to meals and exercise, but, independently of any such influences, the pulse is more frequent in the evening than in the morning, and it would appear from various considerations, but principally from what is observed in certain forms of disease, that during a long night's sleep the circulation runs down in vigour as well, and not only in frequency. It is in the early morning that depression of spirits is liable to be at its worst in nervous debility, so called; or there is the morning headache which is relieved by the bath and breakfast, or wears off as the day advances; or the subject of this affection is more tired on waking up than on going to bed. In heart disease, again, the sufferer wil wake up gasping for breath after sleep, and paroxysms of asthma and the fits in epilepsy choose this time for coming on.

The rate of the heart's action, with which the frequency of the pulse corresponds, is governed by various influences. Resistance to the onward current of blood in the arteries, or, in other words, increase of pressure in the arterial system, whether produced by compression of large vessels, such as the femorals and brachials, or by obstruction in the arterioles and capillaries, tends to slow the action of the heart and render the pulse less frequent, and, conversely, diminished resistance, or lowered tension, accelerates the heart and pulse rate. But more direct and powerful than these variations of the arterial pressure and entirely overruling their tendency, are nervous influences, of which the channels are the pneumogastric and sympathetic nerves, the former inhibitory, the latter exciting. It is not my intention to enter at all upon a discussion or explanation of the respective action of these nerves; this would help us very little clinically and knowledge with regard to them has not yet reached a perfectly stable condition. The question, too, is rendered complex by the fact that nervous

influences reach the arterioles and capillaries as well as the heart, and modify the outflow of blood and the arterial tension; and an effect upon the heart, apparently direct, may be brought about indirectly through variations in the degree of resistance in the circulation.

Exertion of any kind at once sends up the pulse rate, and the action of the heart is more forcible as well as more frequent. At first the tension in the arteries is raised and the pulse is vehement; but in a short time the artery is no longer full between the beats, and these, while still sudden, lose their force, becoming short and unsustained.

The regulating nervous apparatus no doubt plays an important part in the acceleration of the heart's action produced by exercise, but there is a physical necessity and cause for it which would explain it. The first effect of powerful general muscular action is to drive the blood along the veins towards the heart, and the right auricle and ventricle are at once distended. To pass on the blood through the lungs as fast as it arrives, the right heart must act more frequently and powerfully and the stimulus to this exists in the increased pressure on its inner surface. For a time there is accumulation in the pulmonary circulation and while this is the case there is dyspnœa and shortness of breath and panting; but when the individual is vigorous, the circulation in the lungs and system becomes equalised and he gets his second wind, as the term is.

Position influences frequency, there being, on an average, a difference of eight beats per minute between the standing and the recumbent posture. The greater frequency in the upright position is not due to the muscular exertion required to assume and maintain it. The general blood pressure in the arteries has been found to be greater in the horizontal position of the

body, and this probably is the influence which slows the action of the heart.

Excitement of any kind accelerates the pulse, as do all powerful emotions. For the most part they also cause contraction of the arterioles and so give rise to an increase of tension. There is, however, great diversity in this respect between emotions of different classes and also in respect of the force of the heart's action. Fear, for example, while increasing the frequency of the heart's action will render it feeble, while anger will make it violent. The effect of excitement must always be taken into account when examining the pulse, and it must be borne in mind that the tension as well as the frequency may be increased; the sudden acceleration of the heart's action may of itself give rise to a temporary fulness of the arterial system and increased blood pressure, by the greater number of times per minute which the ventricle discharges its contents into the aorta.

Food accelerates slightly the heart's action, relaxing also the peripheral vessels. Stimulants do the same in a more marked degree. External warmth, again, increases slightly the frequency of the pulse and relaxes the arteries.

There are certain drugs which increase the frequency of the heart's action, and therefore of the pulse; these belong mostly to the class of stimulants. Some produce this effect through the central nervous system, such as the alcoholic and ethereal stimulants; but, as most of them also dilate the peripheral vessels, the lowered tension and diminished resistance so brought about may contribute to the result. It may also be maintained that there is direct stimulation of the heart itself. Such agents as nitrite of amyl, nitro-glycerine and the nitrites, while causing acceleration of the pulse, act primarily and chiefly upon the arterioles and capillaries.

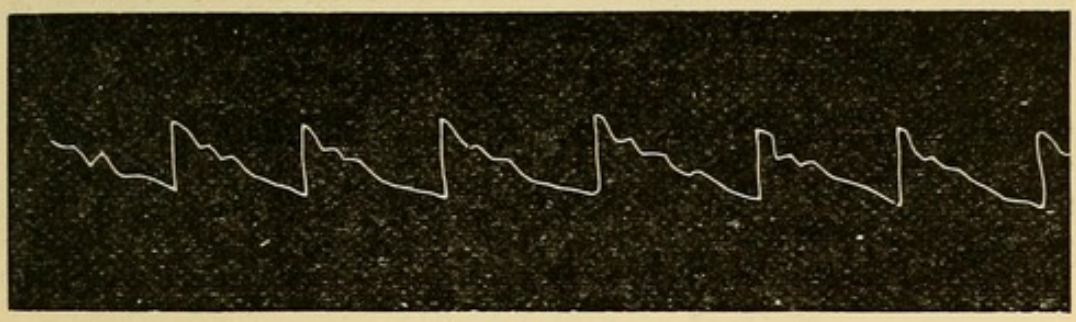
Other stimulants excite the heart to more rapid action mainly by excitation of peripheral nerves. They are characterised by pungency and comprise the essential oils; ammonia also belongs to this group, for, although its salts relax the arterioles and accelerate the pulse slightly, their action in this respect is not to be compared with that of free ammonia, which possesses pungency. Ammonia applied to the nostrils, when only the most minute quantity can be absorbed, excites the heart's action.

In another class of cardiac excitants will come belladonna and its alkaloid, atropine.

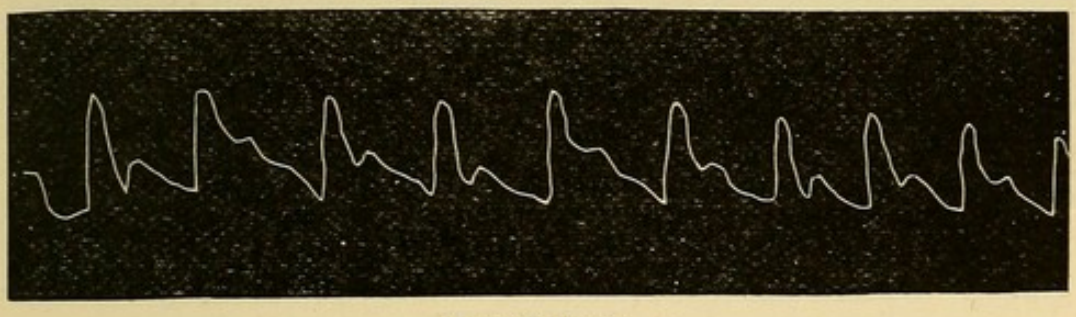
The influence of the respiratory movements and of variations of intrathoracic pressure upon the pulse is a question of considerable interest. In disease it often becomes manifest, as in emphysema, when the intrathoracic pressure is very different in inspiration and expiration; it is seen again in the pulsus paradoxus, and it probably plays an important part in producing the irregularity of the heart's action in mitral disease. Neither the results of the respiratory variations of pressure nor the way in which these results are brought about, are set forth very clearly in the works on physiology which I have consulted. Such effects must be due entirely to the influence of pressure-variations on the veins and auricles, and on the movement of blood through the pulmonary capillaries; perhaps in some small degree to influence on the ventricles in diastole. The normal difference of pressure between inspiration and expiration can have scarcely more direct effect on the aorta or on the pulmonary artery, than if their walls were made of metal, the pressure within these vessels being so great; and the same may be said of the ventricles during systole: the principal effect on the pulse will be due to the influence of the respiratory movements on the filling of the left ventricle, and this will depend on their

influence on the auricles. If a deep inspiration be taken and the breath be then held, the glottis being closed and pressure exerted on air contained in the chest, the pulse rapidly becomes small and weak; the pressure on the thin-walled auricles interferes with their action and with the supply of blood to the ventricles. The pulse rate may be increased or diminished. Similar effects are produced by holding the breath after a deep expiration. During an ordinary inspiration, venous blood is drawn into the chest along the great systemic veins and the right auricle is well filled, and the right ventricle well supplied, since the slight general negative pressure will scarcely affect the auricular contraction. The converse will take place in expiration. The effect on the left auricle is not so simple, as the pulmonary veins which supply it are exposed to the same negative pressure; the greater surface of the auricle as compared with the veins will, however, promote influx into its cavity and a larger charge will be delivered to the ventricle.

In investigating the effect of varying respiratory pressure, however, no account has been taken of the degree of tension in the arterial system of the subjects of the observation, and as this appeared to me likely to influence the result, I requested Mr. Eustace M. Callender, while my house physician, to take sphygmographic tracings from two young men of good physique, both notable football players, one of whom had physiological high tension, the other low tension. The observations were taken first during ordinary tranquil breathing and afterwards when the intrathoracic pressure was varied in a positive and negative direction and in different degrees. It will be seen that the high tension pulse takes little or no notice of variations, which produce extraordinary perturbations in the low tension pulse. (Figs. 18—26.)

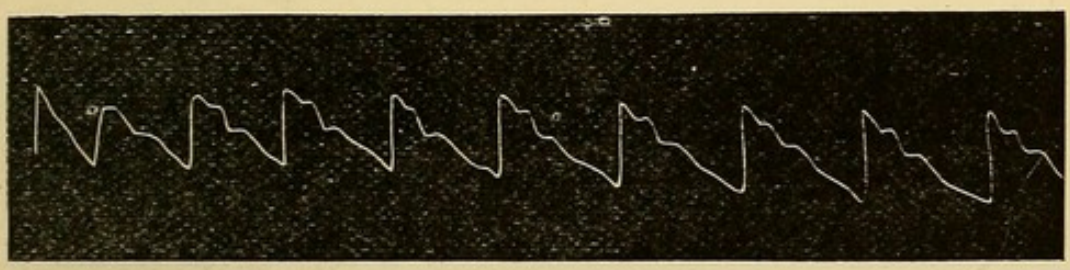


High Tension.

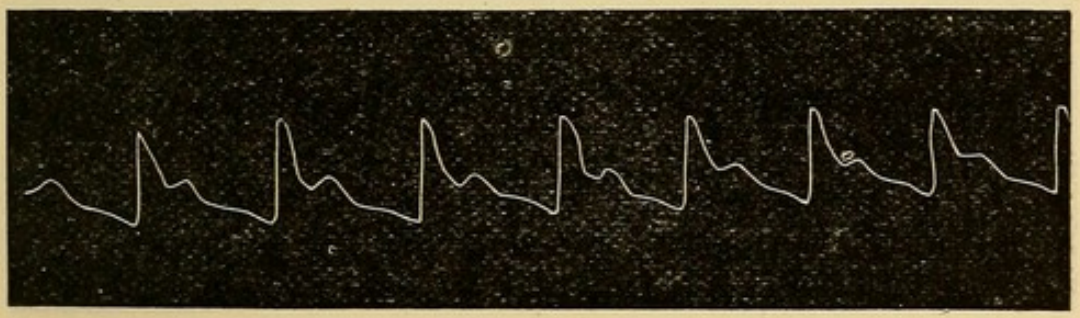


Low Tension.

Fig. 18.—While sitting at ease and breathing normally.

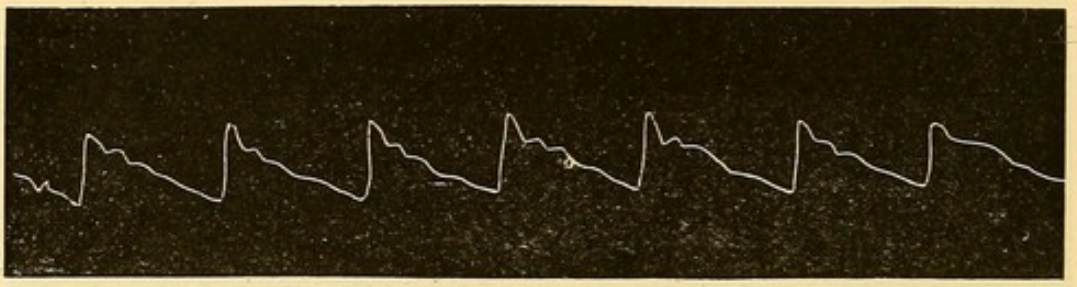


High Tension

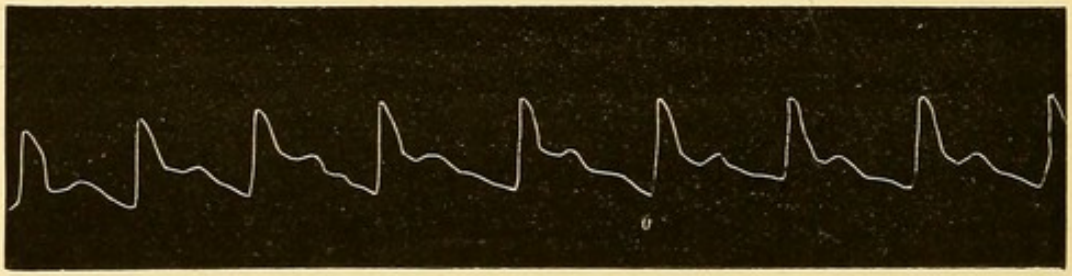


Low Tension.

Fig. 19.—While holding the Breath at the end of ordinary Inspiration.

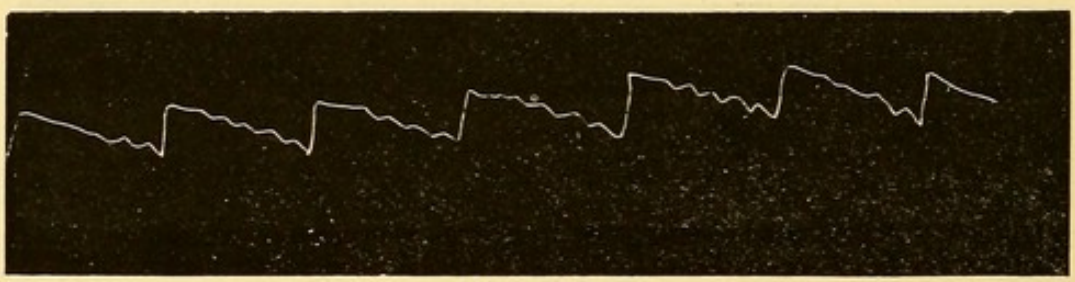


High Tension.

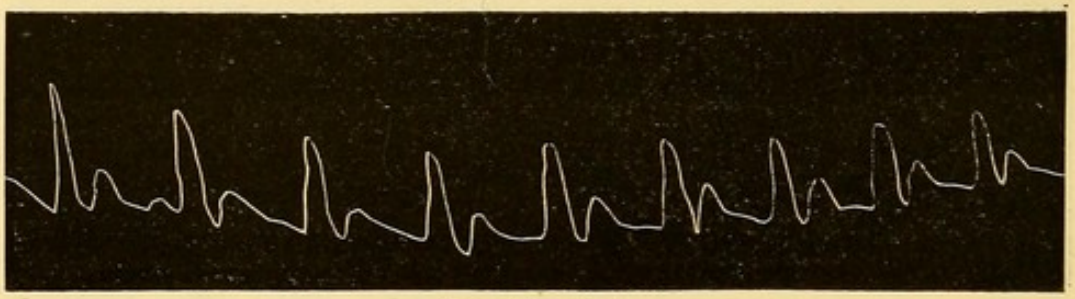


Low Tension.

Fig. 20.—While holding the Breath at the end of ordinary Expiration.

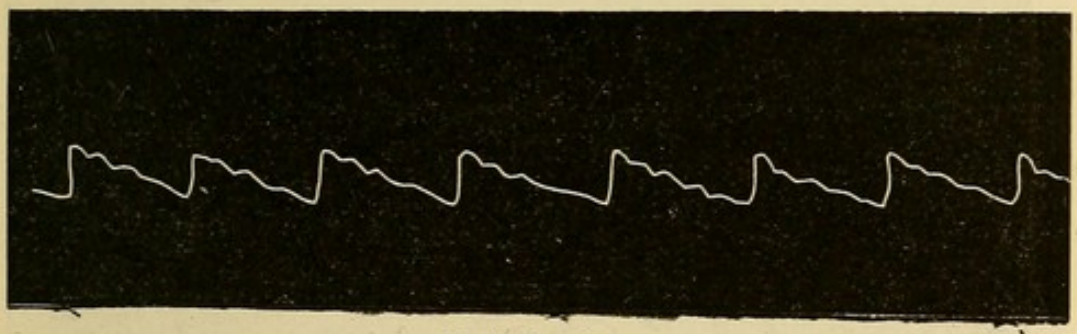


High Tension.

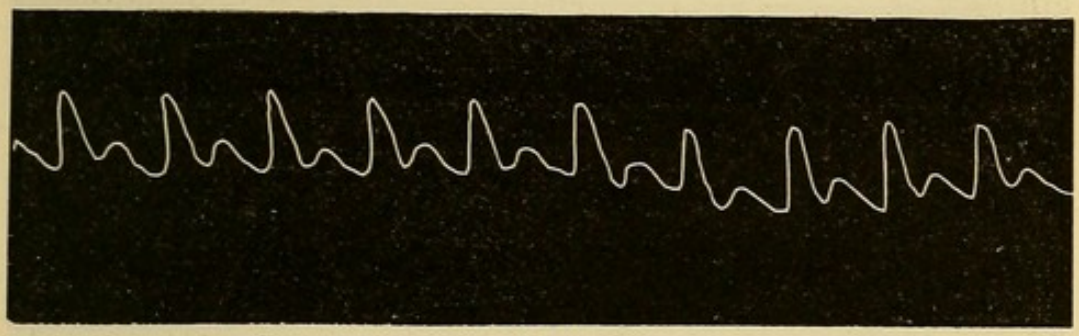


Low Tension.

Fi . 21.—Breath held at the end of *deep* Inspiration.

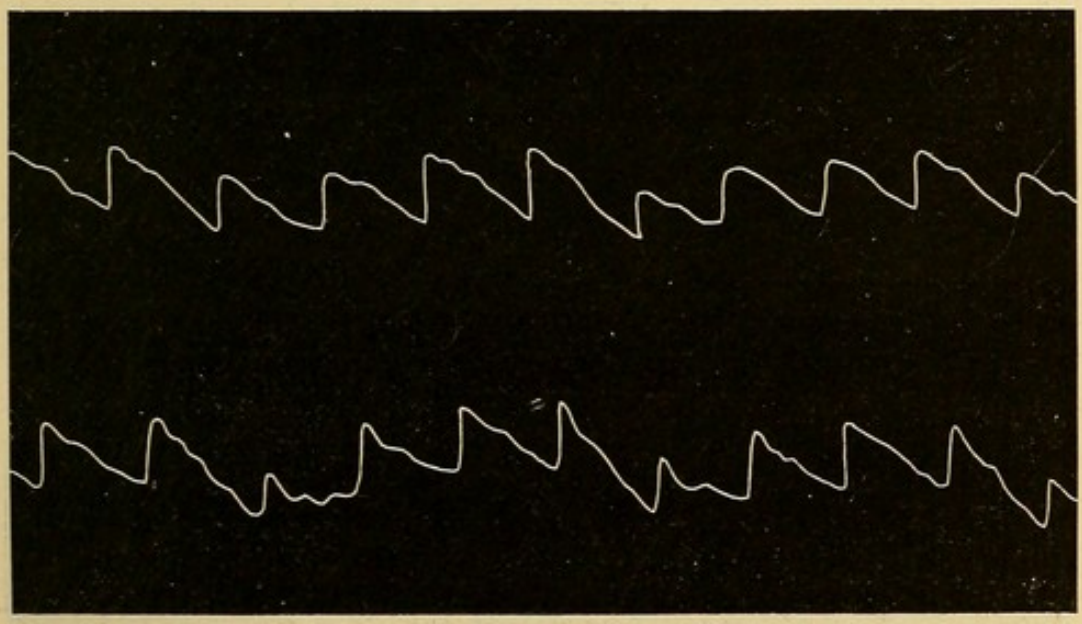


High Tension.



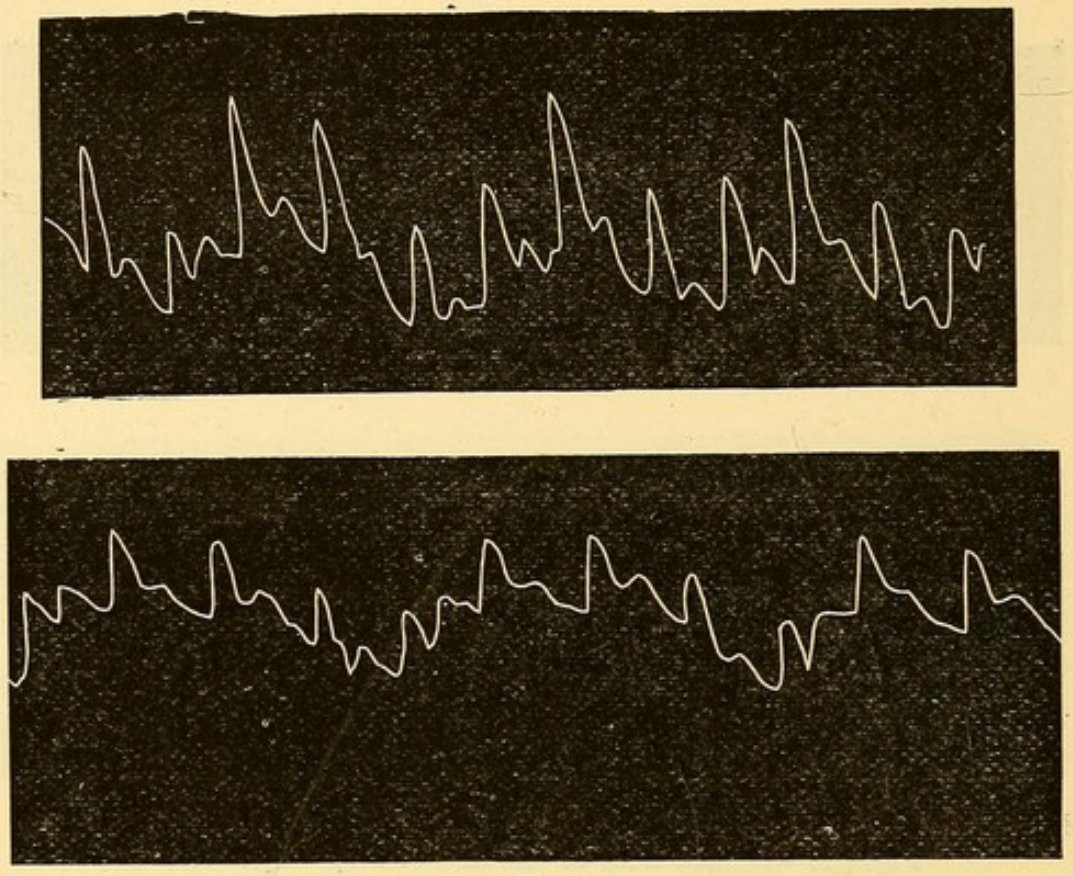
Low Tension.

Fig. 22.—Breath held at the end of *deep* Expiration.



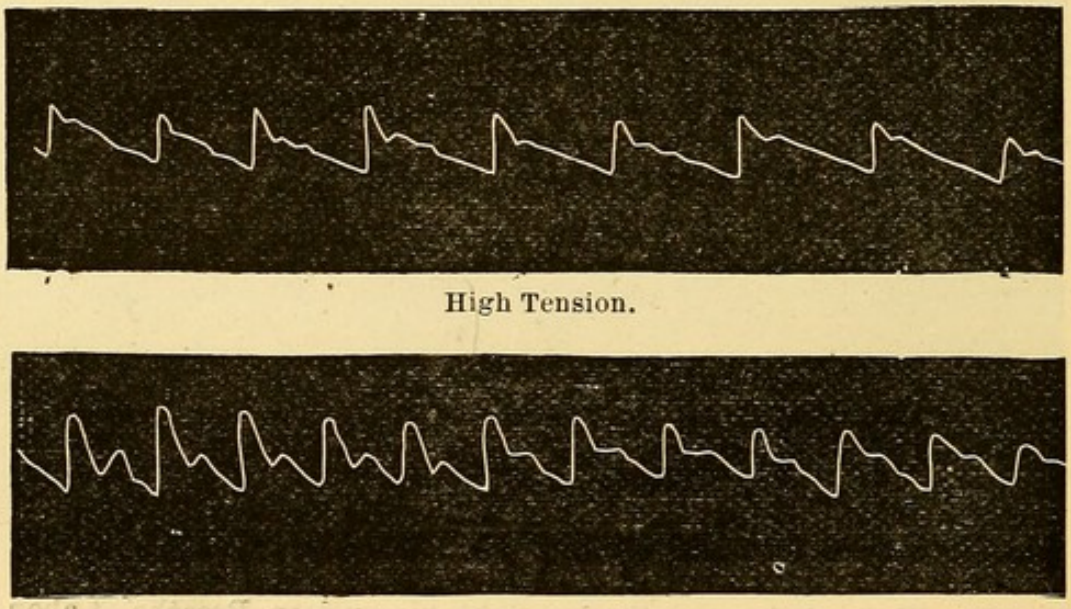
High Tension.

Fig. 23.—While sitting quietly, but taking very deep Breaths at about the rate of Twenty per Minute.



Low Tension.

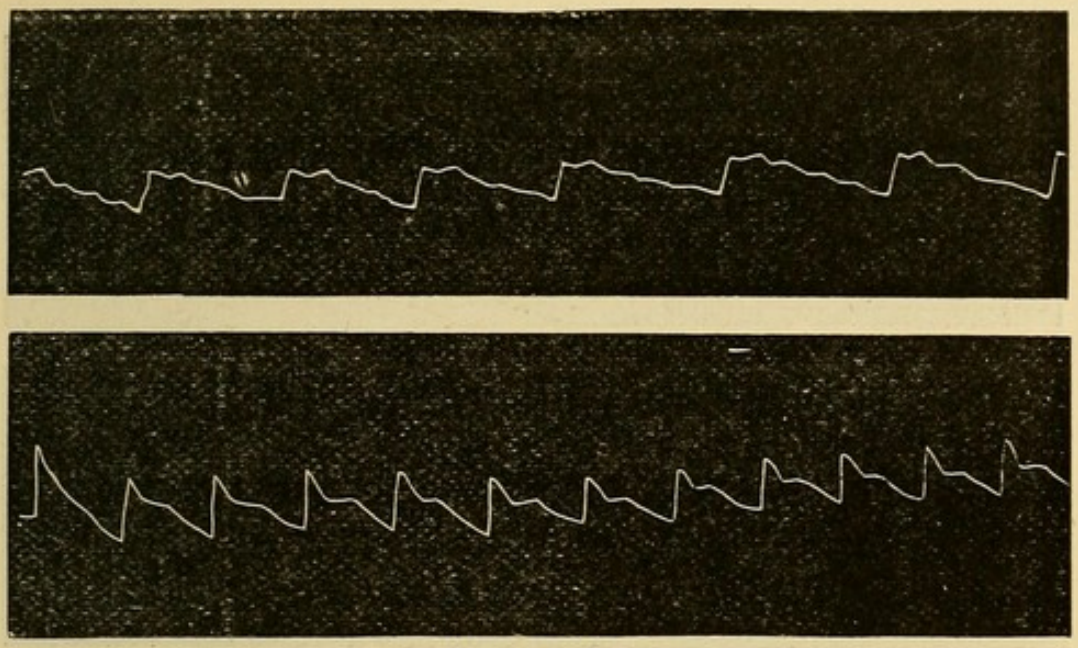
Fig. 23.—While sitting quietly, but taking very deep Breaths at about the rate of Twenty per Minute.



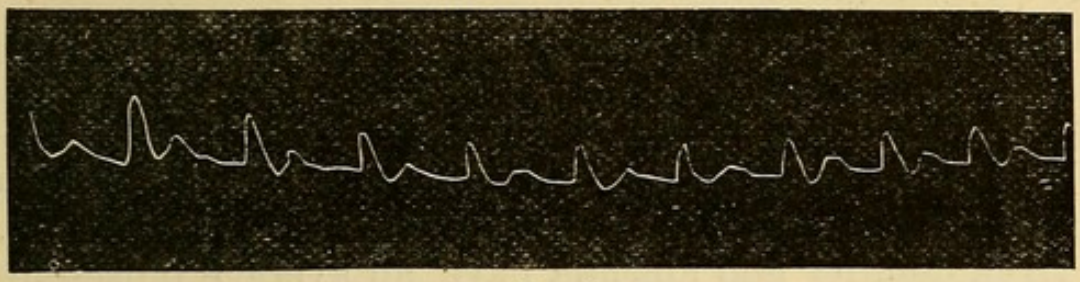
High Tension.

Low Tension.

Fig. 24.—While Breathing naturally after taking Forty such deep Breaths.

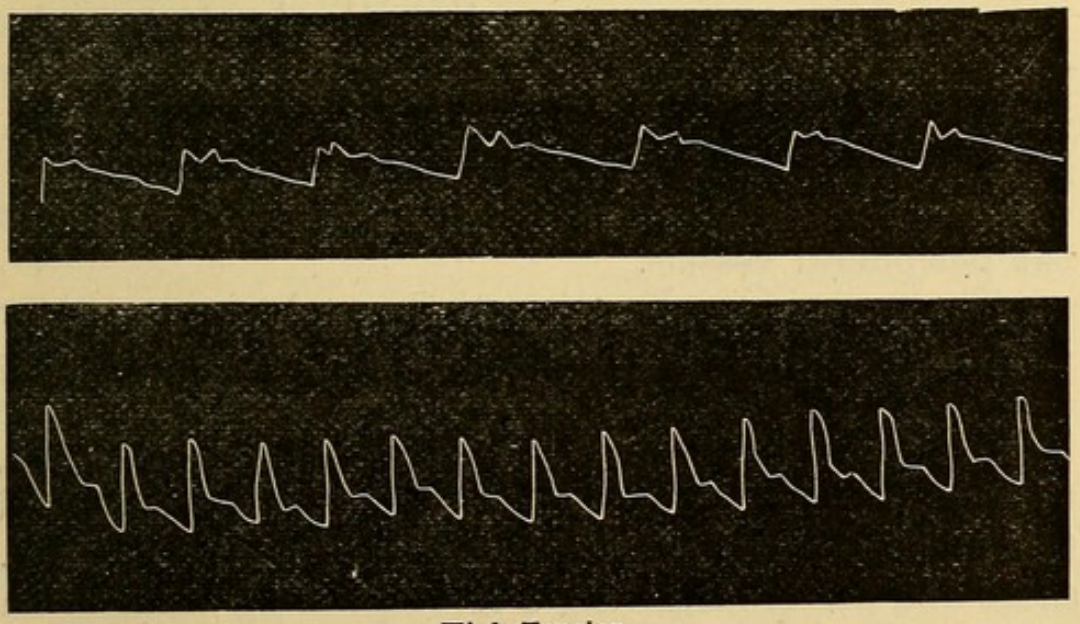


High Tension.



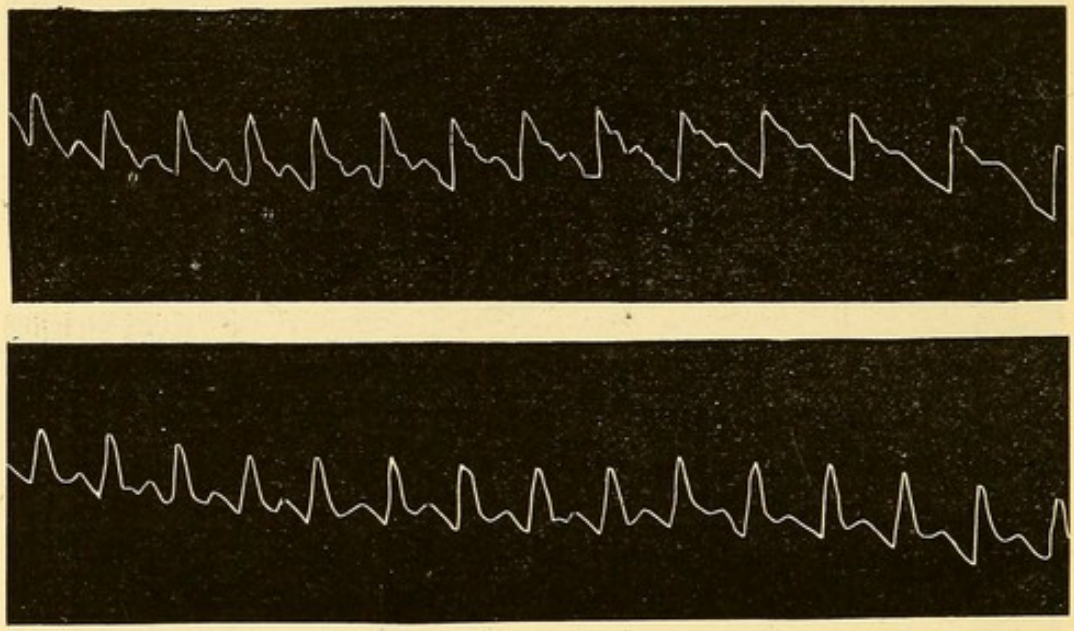
Low Tension.

Fig. 25.—Breath held at the end of deep Inspiration after Forty deep Breaths.



High Tension.

Fig. 26.—Breath held at the end of Expiration after Forty deep Breaths.



Low Tension.

Fig. 26.—Breath held at the end of Expiration after Forty deep Breaths.

Increased frequency of pulse in disease.—Almost all departures from a normal state of health are attended with increased frequency of the pulse. Even debility and anæmia have an average pulse-rate above the normal, but more marked than the frequency in these conditions is the increased mobility of the pulse under exertion or emotion, the least muscular effort or change of position sending up the frequency inordinately.

Pyrexia, as such, gives rise to marked and continuous acceleration of the pulse and to great relaxation of the peripheral vessels.

The force of the heart's action may be increased or diminished, and the vehemence of the beats will vary accordingly. In sthenic pyrexia, such as attends acute inflammation, and generally in the early stages of fever, the force of the cardiac systole will be augmented and the pulse will be frequent, sudden, vehement, large, short and dicrotous, giving a corresponding trace when the sphygmograph is applied. When the action of the heart is weak the pulse loses in vehemence, and, when

the weakness goes beyond a certain point, in suddenness and size, dicrotism being still well marked.

The frequency of the pulse bears some sort of relation to the pyrexial temperature, being usually greater as the temperature is higher in fever of the same character, and the pulse and temperature taken together mark the severity of the attack and the impression made upon the system. It might, perhaps, almost be said that, while the temperature shows the degree of fever, the pulse indicates its effects on the system, and the efficiency of the constitutional reaction. The frequency of the pulse, then, is often of great prognostic importance. At all stages of the specific fevers it is noted carefully; at the outset it denotes the severity of the attack; towards the end it tells whether the strength is sustained or is failing; the increase of a few beats per minute daily at this period will be a source of grave anxiety, even independently of the vigour of the heart's action.

The degree of frequency of the pulse, again, is one of the points by which septic fever is distinguished in surgical cases, and extreme frequency is often the first indication of deadly puerperal septicæmia. This extreme frequency may be regarded as a sign of shock, or as denoting the serious impression made upon the vital powers by the poison. Shock from other causes may be marked by a racing pulse, and, when such is the case, there is always danger.

Among the causes of unusual frequency of pulse, scarlet fever must not be forgotten. At the onset of this disease a pulse of 120 to 140 is not uncommon in adults, and in children it may reach 160 or 200 beats per minute.

So far, increased pulse frequency has been one among many other symptoms and has been a consequence and not in any sense a cause of the disease. But there are many affections in which the unduly

frequent pulse is, so to speak, of the essence of the disease, the disorder of the circulation of which it is the index constituting indeed sometimes its most important factor, and not being a mere sign of its existence. These must now be considered.

Persistent frequency of pulse.—Persistent frequency of pulse is one of the signs or consequences of overstrain of the heart by exertion. It is met with in young men who have overtaxed their powers in rowing, training for races, or by heavy gun drill, and was observed on a large scale in the American war, among men taken from sedentary or other occupations not attended with great exertion, and called upon to undergo protracted drilling or to make long and trying marches. The name "irritable heart" employed as a descriptive term may very well be accepted. Besides the beating of the heart of which the patient is conscious, there are breathlessness on exertion, nervousness, depression of spirits and anxiety, sensations of faintness, sleeplessness, and incapacity for sustained exertion. In all cases of this kind which I have seen the pulse-tension has been high.

The great remedy for this condition is rest, and from one to three weeks may be well spent in bed, however irksome and wearisome it may be at this period of life, in allowing the heart to settle down.

During and after middle age persistent frequency of pulse may be induced by a single act of excessive exertion, such as running to catch a train. The effects upon the heart of such an imprudence vary; there may be dilatation of one or other ventricle or of both, with or without insufficiency of the mitral valve; or a valve may be actually damaged, or the action of the heart may become irregular; but the heart's action may become, as the result of strain, hurried, without irregularity and without obvious lesion, and the frequency may persist till the strength of the patient is

worn out. Here, again, the arterial tension is, according to my experience, high, resistance in the peripheral vessels contributing to the effect, so that the distension of the arteries is not simply the result of blood being driven into the arterial system in consequence of the increased frequency of the heart's contractions. No satisfactory explanation has been given of these cases, and I have none to offer. Perhaps the most plausible is that the plexus of minute nerve-ganglia and network of fibres so copiously distributed beneath the endocardium may have been stretched and rendered unduly irritable.

Graves's or Basedow's disease, Exophthalmic Goitre. — Inordinate frequency of the pulse is one of the tripod of symptoms which constitute Graves's disease or exophthalmic goitre. The three are—enlargement of the thyroid gland, proptosis, and excitement of the heart's action. Together with the protrusion of the eye, there is retraction of the eyelids, and also a want of readiness in the upper lid to follow the eye downwards, so that the white sclerotic shows above the cornea, adding to the oddity of the patient's appearance. The protrusion of the eyes, however, may be absent, and occasionally the goitre may be small. The frequent and violent action of the heart is, perhaps, the most constant and the most characteristic of the symptoms. The pulse is seldom under 100 per minute, often 120 or over, sometimes 160 to 200. In a case recently under observation the pulse was never under 160 for many weeks, and in paroxysms of palpitation it sometimes numbered 240 for several days, reaching at times 300. The beat is sudden, sharp, and vehement; and its very frequency renders it short. The artery has not time to subside; but, though it is full between the beats, there is no peripheral resistance and no real pulse-tension.

This affection is evidently a neurosis, and a very plausible theory with regard to it is that it is due to disease of one or more of the cervical ganglia of the sympathetic. This, however, cannot be looked upon as established.

Further evidence of the neurotic origin of exophthalmic goitre is found in the extraordinary pigmentation of the skin, which is sometimes present. It may simulate in tint the bronze skin of disease of the suprarenal capsules, but has not the same distribution.

The heart-sounds are loud and short, the first especially, the second being relatively weak. The relative length of the intervals is little disturbed.

This disease is most commonly met with in young women, but may come on in middle life and may affect men. Its causes are mostly such as are attended with excessive wear and tear of the nervous system: long hours, over-work, confinement to close rooms, unnourishing food—conditions to which sempstresses, shop-girls, and other women following similar occupations in towns are subjected, aided very often by uterine derangements. Other causes are frequent child-bearing and over-suckling, anxiety, and distress of mind.

It usually comes on slowly, but may develop rapidly, and has been known to do so after mental shock.

Its duration is long, and may be reckoned by months and years. Two years is, perhaps, a low average, but not uncommonly it resists all treatment and is incurable.

It is mostly attended with considerable wasting, and indeed usually comes on in thin subjects. The sufferers are anæmic, breathless, liable to palpitation, dyspeptic, and subject to constipation; they are nervous, and sleep badly.

One of the painful effects sometimes met with is destructive inflammation of the eye from exposure, the lids not being able to meet and cover it. Conjunctivitis and ulceration of the cornea from this cause are often troublesome.

The treatment required consists of rest, which is of the first importance, so that in bad cases the patient should be kept in the recumbent posture for the greater part of the day; good food, iron, and arsenic, aloetic and tonic aperients, and other means which may be required for the improvement of the general health. The special remedies suggested by the circulatory and other characteristic symptoms are digitalis, strophanthus, caffeine, and medicines belonging to the same class. These are, perhaps, the most generally useful, and sometimes seem to have an immediate palliative effect. Sedation of the heart and pulse by aconite has been tried, but this drug is dangerously depressing. A line of treatment which has sometimes been attended with marked success is the administration of belladonna or atropine in gradually increasing and ultimately very large doses. Galvanisation of the sympathetic in the neck has been suggested, and has been reported to do good. For the most part, however, it is time and the gradual re-establishment of the general health and nervous tone by rest, food and tonics, and the removal of functional derangements, gastro-intestinal and uterine, which effect a cure.

Aortic pulsation.—Cases are met with, so far as is known to me, only among women, and usually at or after middle life, in which, with remarkable rapidity and violence of the heart's action, and, of course, a corresponding frequency of the pulse, there is an extraordinary vehemence in the pulsation of the abdominal aorta. The beating is a source of constant discomfort, sometimes of actual pain, to the

patient, and is conspicuous both to the eye and hand of the observer. I have known it to be so violent as to be seen and felt through the dress, stays included. It is not to be wondered at that this condition is often taken for aneurism, but on reflection it will occur to the mind that aneurism is not attended with the constant excitement of the circulation, the violent action of the heart, and the frequent pulse, which are present; and on examination, although the pulsation seems to extend laterally beyond the normal limits, the pulsation is not localised in any particular part of the aorta, but can be followed along its whole length, and is found to extend beyond its division into the common iliac arteries. Where the pancreas lies across the vessel the thickness of the gland causes the pulsation to be felt over a larger area, especially laterally to the left, which may simulate a local dilatation of the aorta, and the pulse of the splenic artery may contribute to this.

The explanation of the violent throbbing of the aorta is not clear. Of course, the action of the heart is the primary cause, but there may be powerful and abrupt cardiac contraction without this effect. In some respects the aortic pulsation resembles the exaggerated beat in a ligatured artery, but the condition of the arterioles is not such as to corroborate this comparison. It might, again, be an effect of extreme low tension in the visceral arteries and in the arteries of the lower extremities, allowing of extreme alternations of distension and relaxation of the abdominal aorta. The easiest hypothesis would be to suppose that the coats of the aorta itself were relaxed, from deranged vaso-motor influence or loss of vaso-motor control; but the muscular element in the arterial walls subject to such control is at a minimum in the large arteries.

The general condition of the patient is very similar

to that of the sufferer from exophthalmic goitre, and the two affections belong probably to the same class. The causes are much the same, and the treatment would be conducted on the same principles.

Palpitation of the heart.—Palpitation of the heart has not a very definite signification. It may mean a beating of the heart of which the subject is conscious, whether this is unduly frequent or not, and whether it is unduly forcible or not, as felt or heard by the observer. Again, it may mean undue rapidity and violence of the heart's action, whether attended or not with conscious discomfort to the patient. We are here, however, concerned only with such forms as are attended with frequency of the pulse.

Palpitation, so understood, varies extremely in its significance; it may be a mere passing disturbance set up by flatulence or indigestion; it may be a severe recurrent affection easily provoked by emotion or excitement, symptomatic of a weak and disordered state of the nervous system, or of anæmia and debility, or attendant on a gouty state of system; and is often a cause of extreme discomfort and of uncontrollable nervous dread, sometimes rendering the sufferer incapable of all business and enjoyment; it may, again, be a symptom of complete and irremediable cardiac or nervous breakdown, and will be one of the effects of such breakdown which will help to wear out the strength.

In the minor form of palpitation the attacks may come on at different times—soon after a meal, or at an interval of some hours; very often in the night. There may or may not be antecedent discomfort of one kind or another—a feeling of fulness and distension in the epigastrium, or a sense of oppression in the chest. In the night there may have been dreams, usually, but not necessarily, of an unpleasant

character, or actual night-mare, out of which the patient wakes with the heart beating violently, sometimes as if from fright or other powerful emotion arising out of the dream. Usually some discomfort has lasted for a time, when the heart suddenly goes off with a kind of leap into rapid action, sometimes appearing to the subject to roll or turn over. There is more or less of a feeling of breathlessness and oppression, sometimes of anxiety, in severe cases of faintness and giddiness, so that the patient must lie down. On these symptoms we need not dwell. The pulse is frequent, short, variable in fulness and strength, rarely at all vehement, sometimes irregular.

The attacks may be over in a few seconds or minutes, and pass off spontaneously, or may last an indefinite time, unless some remedy is applied.

A teaspoonful or two of brandy taken neat, half a drachm or so of sal volatile with a little carbonate of soda; or, in more severe cases, the well-known haustus stimulans, compounded of ammonia, ether, chloroform, spirit of lavender, with camphor water, will generally put an end to the attacks, usually with more or less eructation. At times an emetic will be required, especially when the stomach has been overladen, or food of a particularly indigestible kind has been taken.

The more serious kind of palpitation may also be started by gastric derangement, flatulence, an ill-digested meal, or the like; but when the predisposition has been generated, no care in diet will entirely prevent the attacks, and there is danger lest the fear of taking food should increase the debility on which they depend. A more common exciting cause is emotion or excitement, which may be extremely slight, the mere apprehension of an impending attack being sufficient to bring it on, or the expectation of an interview, or the mere idea of seeing a friend will

have this effect. A sudden noise, again, such as a banging door, or a step, or the rustle of a leaf may start a paroxysm. Where the predisposing condition is gout, slight exertion, or a particular position, or a fit of temper will perhaps induce an attack; but acid and flatulent dyspepsia is common as a cause. Hysterical palpitation might either be classed with the severe forms on account of the violence and duration of the paroxysms, or with the slight forms, as leaving the patient little the worse when the attacks are over.

It is late in life that paroxysmal palpitation with frequency of pulse is most commonly a cause of suffering and danger, shortening life and rendering it miserable. It may complicate heart disease of any kind, and may possibly sometimes be one of the consequences of the disease; but it may occur independently of valvular affection or of any structural change sufficiently advanced for recognition, and it has seemed to me that when disease of the heart is present, the palpitation is often rather a complication than a consequence. The exciting cause may be indigestion and flatulence or the mere act of taking food, lying down, emotion, apprehension of an attack at a particular hour, or under given circumstances associated with former attacks; but whatever this may be, the onset of the paroxysm is frequently accompanied by a sudden relaxation of the arteries, and the palpitation seems to resemble the excited action of the heart set up by nitrite of amyl or nitroglycerine. Resistance to which it is habituated is suddenly removed and the heart starts off like the engine of a locomotive when the wheels fail to bite the rails. In the course of a prolonged attendance upon a medical man advanced in years and long subject to gout, who suffered greatly from palpitation, this occurred more than once when my hand was actually

on the pulse; the artery became large and soft, and there was a flutter of the heart, which then bounded off. A remarkable complication occurred in this case which is worthy of being related. Early one morning the patient began to bring up bloody fluid from the lungs, and in the course of twenty-four hours expectorated several pints of it. A pink froth covered the bright red liquid to the depth of half an inch, and the whole looked very much like the boiling red currant juice in the process of making jelly. There was no rise of temperature, the flux of blood-stained serum gradually ceased and the palpitation did not recur for several months, when after imprudent fatigue and exposure it returned, and ultimately wore out the patient. It should be added that there was no valvular disease and only moderate dilatation and hypertrophy with old-standing high arterial tension.

In the severe palpitation now under consideration, the action of the heart is generally irregular, sometimes extremely so, and this irregularity is exaggerated in the pulse by the beats being very unequal in strength and by many of them failing to reach the wrist. The heart sounds are short and confused, defying description and often analysis, so that it is impossible to say which is most distinct, the first sound of the right or left ventricle, or whether the pulmonary or aortic second sound is the more accentuated. A short, smooth, high-pitched systolic whiff is often audible near the apex, or in the tricuspid area on careful listening. The paroxysms are attended with extreme distress and suffering, and there are not only greater oppression, more vertigo, worse faintness, with pallor and cold perspiration, but the attacks last longer, and when they have passed off leave the patient pale, weak and depressed, perhaps for the rest of the day or till after sleep.

In the worst cases the attacks of palpitation become

so frequent that they almost run into each other and become continuous. This condition may be reached gradually, the paroxysms, which at first seemed to be attributable to some exciting cause or other, coming on without provocation; or it may be established abruptly with or without apparent cause. No description can convey an idea of the suffering and misery of the patient. Awake he is breathless, or rather, has a *besoin de respirer*, which it is impossible to satisfy, and which makes him take deep voluntary inspirations; he has an abiding sense of oppression and anxiety and faintness; he feels painfully in need of support, yet dare scarcely eat or drink; not uncommonly, however, the conscious sense of palpitation is absent, or is present only at intervals. Asleep in his chair, which happens from time to time from utter exhaustion, the face twitches and the limbs jerk, and he wakes up with a start and is not only unrefreshed, but feels as if the nap had started the palpitation. He is usually able to lie down in bed at night, but tosses restlessly and cannot sleep, or, if he does so, will often wake with a sense of impending death, feeling as if the heart had stopped and could only be started again by an effort of the will. Death may be sudden in the sleep or during some slight exertion, or it may come as the result of gradual exhaustion of the most distressing character.

I have more than once in a gouty condition of the system known a severe attack of palpitation to establish mitral regurgitation, the usual effects of which have been speedily developed: a systolic apex murmur, never before heard, has appeared and persisted, the result, no doubt, of dilatation of the left ventricle.

In the treatment of the attacks, the remedies already enumerated will be tried, with, in some cases, bromides, in others, valerian; or oil of cajuput may be given, three to five drops on a piece of sugar.

Digitalis and ammonia, strophanthus, or convallaria may be given and sometimes afford relief, as may ether or chloroform. Ether hypodermically may produce striking alleviation, but it is generally fugitive. A sinapism or turpentine stupe, or chloroform and belladonna liniment sprinkled on spongio-piline wrung out of hot water as an application over the heart and epigastrium will often quiet the palpitation for some time and afford an opportunity for sleep; a belladonna plaster may be employed as a preventive, and it often seems to keep off the attacks; it is decidedly more efficacious when so applied as to give support. A dozen very deep breaths may stop an attack at the outset; this is especially the case in hysteria.

The most important point in the treatment is, however, to remove the constitutional condition which predisposes to the attacks by such measures as are adapted to the purpose.

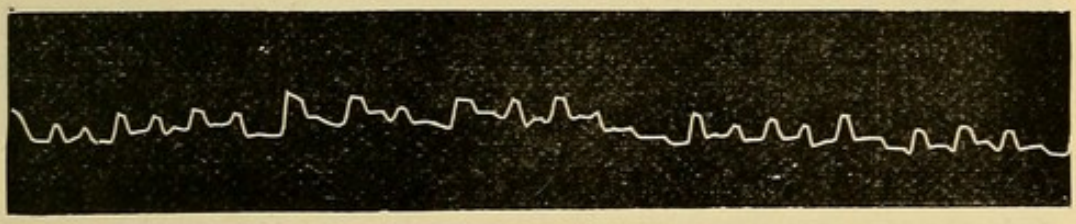
A careful mode of life, with attention to the diet, and to the regular action of the bowels, none but very gentle exercise being taken—this, however, not being neglected—may enable the heart to recover itself, and small doses of digitalis or strophanthus will contribute to this. Convallaria may in such cases do better than either.

Differing from ordinary palpitation in many respects is an extraordinary rapid action of the heart lasting for days or weeks, or even months, with or without severe distress, to which Dr. Bristowe called attention in *Brain* for July, 1887.

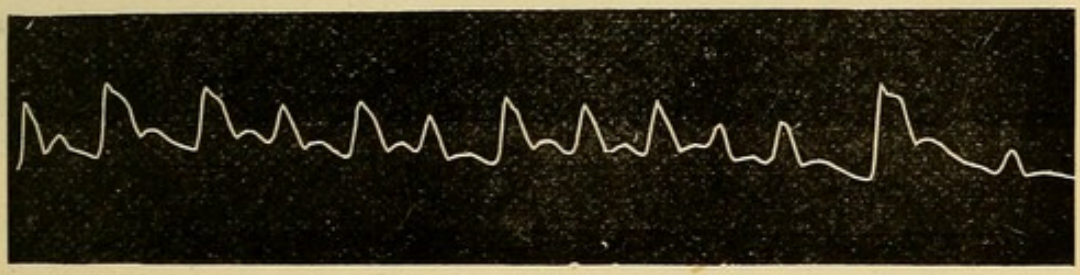
A case of the kind has recently been under my care at St. Mary's hospital. The patient, a married woman from the country, a fruit-gatherer, aged 49, was well-nourished, and had a good colour; she had had rheumatic fever fifteen years before, but, with this exception, had never had a day's illness till nine weeks before admission, when she began to suffer every three

or four days from attacks of pain under the right shoulder-blade, with sickness, and then for a few days she had felt weak and trembling.

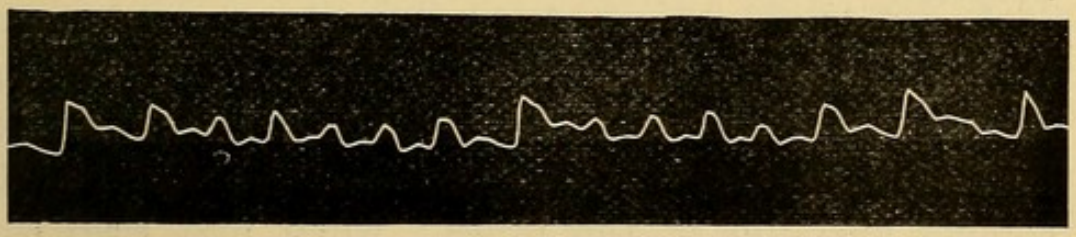
The pulse was found to be extremely frequent, and on this account she was sent into the hospital. After admission, the heart beats were 152 in the minute; the pulse could not be accurately counted on account of inequality and slight irregularity. She was not conscious of any heart trouble, and after a few days looked and felt well. There were no cardiac murmurs. She was made to remain in bed and strophanthus was given. The progress of the case will be best shown by a series of sphygmographic traces. (Fig. 27.)



Before Strophanthus, May 4th.

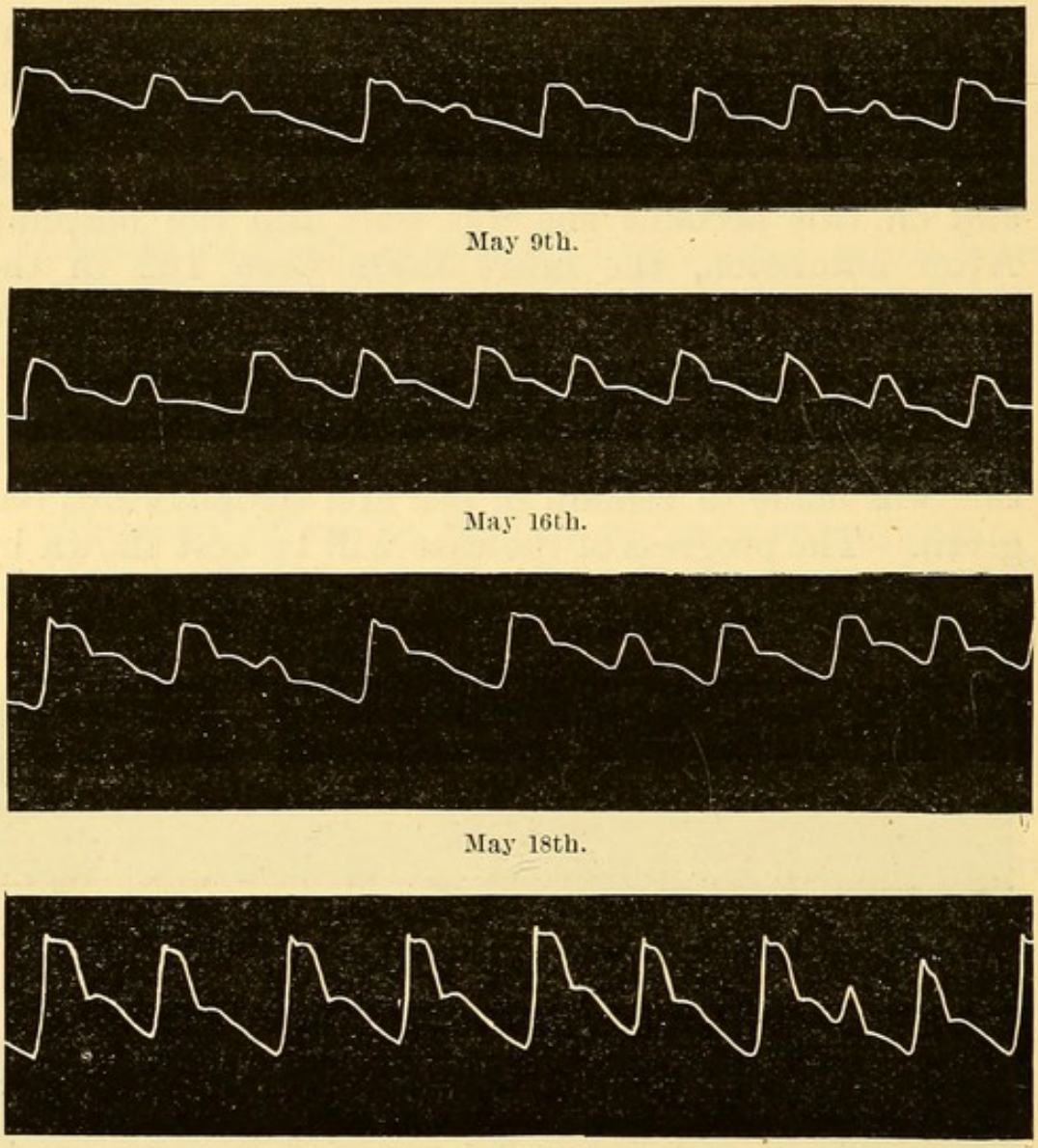


After Strophanthus, May 5th.



May 6th.

Fig. 27.—To illustrate Case of rapid Action of the Heart.



May 9th.

May 16th.

May 18th.

June 1st.

Fig. 27.—To illustrate Case of rapid Action of the Heart.

There was great improvement, but the heart was in a very unstable condition when she left the hospital.

Other examples have come under my notice from time to time. One is related in a lecture published in the *Lancet* for 1875 (vol. ii. p. 442). The patient, a lady's maid, was supposed to be suffering from some sudden affection of the lungs; when first seen she was sitting up in bed with a pale and anxious face,

almost gasping for breath, and from time to time coughing a single short cough. Placing my finger on the pulse I found the radial artery persistently full; but the only pulsation to be felt in it was an occasional feeble beat or rapid succession of several such weak small beats followed by a pause. When I came to examine the heart, however, it was beating at the rate of nearly 200 a minute, regularly and equally, but somewhat feebly, which was not to be wondered at, as the attack had already lasted more than twelve hours. This rapid action kept the artery at the wrist full of blood; but few of the individual pulsations reached this vessel, either because they were too weak, or more probably because the brief diastole afforded no time for blood to enter the left ventricle. The usual heart sounds were not to be distinguished; what was heard was a rapid series of short puffs, reminding me of the sound of a distant locomotive. There was nothing the matter with the lungs. The attack had been induced by over-exertion. I gave belladonna with ammonia and ether, and the excitement of the heart gradually subsided in the course of another twenty-four hours, and it was then seen that there was no heart disease. The patient was very much shaken, and it was long before she was able to resume her work.

In another case I saw the patient, a lady of about forty-five, almost daily for more than three weeks. The pulse was never under 200, usually 240, and there was every reason to believe that the heart was beating at the same rate during the whole of the time. The artery was small, full between the beats, not easily compressible, and the pulsation felt more like a vibration than a beat, and gave the impression of there being little or no onward propulsion of the blood. The heart sounds were short and equidistant. There was no opportunity of examining the pulse or heart

during sleep, but the sleep was broken, and the feelings attendant on the palpitation were always present. This was a second attack after an interval of some years. I saw her in the first, which lasted about five days, and left her looking old and worn. She survived the second for at least two years, since which time I have not heard of her.

Another case was brought to me by Dr. Seccombe in April, 1886. The patient, a gentleman aged fifty-six, was stout and had a good colour, had lived freely in all respects, and worked hard. He had been suffering from palpitation night and day for three weeks, sleeping for two hours only and then restless all night. Pulse was 160, and had the character described in the last case. A tracing of it is here given. (Fig. 28.)

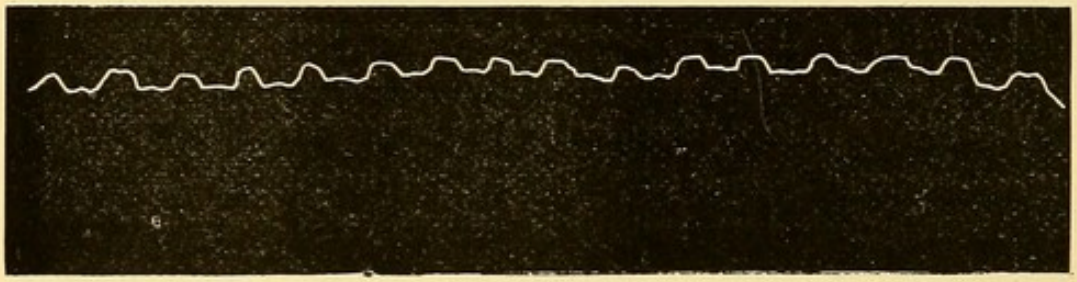


Fig. 28.

There was a reduplication of the second sound of the heart best heard near the apex. The liver and spleen were large. Three weeks later the patient still looked extremely well, and his appetite was good, but he was subject to feelings of suffocation, during which he flushed, and his eyes had an expression of distress; these were brought on by very slight exertion or by stooping. He refused to consider himself an invalid, and went about much as usual. He could lie in any position, but when on his left side required a pillow under the side. He usually slept till three a.m. and then woke up suddenly. The palpitation had never ceased; the pulse was always 160; the

heart was not much enlarged, the sounds equidistant, the aortic second having a ringing note at the right second space; slight jugular pulsation was visible on the right side, and the liver was large; crepitation was heard in both lungs posteriorly as high as the angle of the scapula. In June, when seen again, the palpitation continued, having lasted now three months. He had been out of town and looked as well as ever, but evidences of stasis in the circulation had made their appearance, the legs had suddenly swollen, he could not lie down, and the liver was extremely large. The pulse was still 160, the heart-beats, as before, equidistant. Various lines of treatment had been tried without effect, and digitalis seemed to do harm. I have not seen the patient since, but I learn from Dr. Seccombe that he continued to suffer very much until November, when improvement began, and the patient was recently said to be so much better as to wish to resume his official duties. A later account, however, describes him as having had an attack of hemiplegia.

In another, seen with Mr. Stanley Smith in the course of 1888, the patient, a lady aged sixty-four, died after seventeen days' illness, during which the pulse ranged from 150 to 200, the heart being apparently worn out. There was no other disease.

For particulars of another interesting case with post-mortem examination I am indebted to Dr. Dreschfeld and Dr. Robert Maguire. The patient, a labourer, aged thirty-six, who had had syphilis and had been a heavy drinker, had suffered, when first seen in October, 1883, from attacks of palpitation for twelve months, which came on suddenly after a heavy supper, and were attended with vomiting, and usually lasted from fourteen to twenty days. The pulse-rate was found to be from 215 to 230. No cardiac or other disease was discoverable. He improved under digitalis, the pulse falling rapidly to eighty-six. He was again seen in

December, 1883, and March, 1884, with relapse of palpitation and a pulse of from 200 to 215. On this last occasion, while under treatment by convallaria, he died in an attack of convulsions. The heart was large, and its structure presented degenerative changes, but nothing was found which could explain the rapid heart-action.

Dr. Bristowe has related a series of nine cases under the head of "recurrent palpitation of extreme rapidity in persons otherwise apparently healthy," which had come under his observation since 1885. One of the most remarkable of these was that of a young man, aged nineteen, in whose case there was reason to think that the affection dated from the age of eight, the attacks of rapid action of the heart recurring from time to time. When he entered St. Thomas's hospital he was suffering from anasarca and pulmonary apoplexy, which had supervened in the course of an attack which had lasted some months. The pulse varied, but often numbered 200 or 240. Under the influence of iron and digitalis the action of the heart quieted down to about the normal, but was easily excited, and during one visit to the hospital the beats were counted at the rate of 304 or 308 a minute. He had resumed work as a draper's assistant, and on February the 26th was at work till midnight. Next morning he did not seem much the worse for it, but at midday, while playing the piano, he suddenly stopped and fell down dead. The heart was somewhat large and dilated, but the valves were normal, and no other disease was discovered.

Four of Dr. Bristowe's patients died obviously from the effects of the rapid action of the heart itself, and two from other causes. No changes in the heart or in the pneumogastric or sympathetic nerves were discovered on examination after death. The only cause traceable was bodily or mental strain, and there

was not in every case evidence of extraordinary stress of either kind. Dr. Bristowe points out that at first there are usually remissions, both attacks and remissions varying in duration, and in some of his cases the liability to the palpitation had existed for fifteen years or more. He dwells also on the remarkable fact that there is often wonderfully little distress while the extreme rapidity of the heart is present, and that the patient may be capable of going about as usual.

This rapid action of the heart is, so far as I know, unexplained except by referring it to some obscure neurotic influence. It is met with at almost all ages (one of Dr. Bristowe's patients appears to have been subject to it from the age of eight, and died of it at nineteen, and I have seen it in a child of ten) although it is more common after middle life than before; usually there is some apparent exciting cause, such as would put stress upon the heart. Over-work and anxiety are the most common, but the predisposition, which is of more consequence, eludes us; sooner or later sudden death is a frequent result, or the heart is gradually worn out.

It is interesting to speculate on the condition of the circulation. Clearly the motion of the blood is not accelerated, and the dropsy in Dr. Bristowe's and Dr. Seccombe's cases shows that the tendency is to stasis. The pulse, too, gives one the idea of vibratory alternations of pressure with little onward movement. The question then arises, what is the condition of the left ventricle? Either it does not fill during diastole or it fails to empty itself in systole. The very rapidity of the heart's action would of itself interfere with the filling of the ventricles independently of spasm of their muscular walls, the extreme brevity of the diastole not giving time for the influx of a normal amount of blood. It may be that in some cases the ventricle is contracted and refuses to dilate to receive blood from the auricle, and that in others it is in a

state of over-distension. Dr. Bristowe's idea is that the ventricles are distended with blood during the attacks, but it seems to me that the former is the more probable condition. I have usually found the heart of normal size, and the apex-beat at or near the normal situation, whereas persistent distension would sooner or later give rise to dilatation. It might be asked, again, whether the peripheral arterioles and capillaries played any part in the phenomenon, or the pulmonary circulation; but these are questions which I am unable to answer.

Treatment is not often efficacious. Rest in bed will be necessary in severe cases or in weakly persons, and in all cases rest and care, protection from excitement of all kinds should be secured, together with suitable food. Digitalis, strophanthus, convallaria, have all appeared to be useful, but they do not at once manifest any control over the rapidity of the heart's action, and when the heart settles down this often appears to come from lapse of time rather than from the remedies employed.

CHAPTER VI.

INFREQUENT PULSE.

THE discussion of diminished frequency of the pulse will not occupy much time, although there are questions of great interest connected with it.

It has already been remarked that the effect upon the pulse of nearly all departures from a normal state of health is to increase its frequency, and the diseases are extremely few which reduce the pulse-rate in any marked degree. High tension in the aorta—induced experimentally in animals or man by pressure upon the abdominal aorta or upon a number of the larger arteries—slows the pulse, and the high tension of renal disease has some effect in the same direction, but less than might have been anticipated. In jaundice the pulse falls in frequency, and in various affections of the nervous system this effect is observed; but, except under circumstances which will be discussed later, the reduction is not striking. A pulse is really infrequent when the number of beats is below forty per minute. A pulse of sixty is not uncommon; of less than sixty rare. Persons are met with, however, in the enjoyment of vigorous health, with a pulse of less than forty. I have known an athletic young man, tall, well-built, and muscular, who distinguished himself at football and other violent games, whose pulse-rate was thirty-six. Some years later than I last saw him I heard of his death while swimming; he was taken with "cramp," so-called, and sank. In this cramp it is the heart which is brought to a standstill by the resistance in the peripheral circulation, due to the combined effects of arterio-capillary contraction from contact with the cold water, and of pressure on

the surface from immersion, and it is not unlikely that slow action of the heart would predispose to this result, as may also exhaustion or a weak state of the heart.

Infrequency of the pulse to the degree under consideration is sometimes met with in fatty degeneration of the heart, and is enumerated among the indications of this disease; but it is far more commonly absent than present, even in an advanced stage of fatty change in the heart.

The infrequent pulse, however, is so closely associated with the bigeminal pulse, and with a peculiar modification of the rhythm of the heart, in which only every other beat reaches the wrist, that it will be well to consider them together. In the interesting variety of pulse-rhythm called "pulsus bigeminus," the beats come in couples, a strong beat being followed quickly by a somewhat weaker one, after which there is a pause. The second of the two beats may vary greatly in strength, being sometimes almost as distinct as the first, sometimes scarcely perceptible, and the interval between the first and the second may vary somewhat in length. On examining the heart, its action is found to correspond; there are two unequal impulses, the second weaker than the first, and usually felt at a different point; and on auscultation the sounds associated with the second impulse are less loud and of a lower pitch. If a murmur is present, usually a mitral systolic, it will be louder and higher pitched with the first, and may be inaudible with the second. I have sometimes heard a mitral murmur with one beat, and a tricuspid with the other.

Closely allied to this condition is that in which there are two beats of the heart to one of the pulse, one beat being dropped. Not that transitions are common, for, in the absence of valvular disease, the

pulse may number forty one minute, the heart beating eighty in couples, and the next may be perfectly regular at eighty. In the form of valvular disease, however, mitral stenosis, in which the pulsus bigeminus and the dropped beat are most frequently met with, the former may be a stage in the return towards a regular mode of action from the latter; and although, in the absence of valvular disease, a systematic bigeminal pulse does not succeed the dropping of a beat, yet an occasional bigeminal beat occurs when the weaker systole happens to raise the aortic valves, so that there is a clinical similarity which permits of their being considered together.

Both, as it has just been said, are met with in mitral stenosis, and especially when under treatment by digitalis. I have had several cases in which the pulsus bigeminus, or dropping of one beat, could be induced at will by administering this drug.

Taking the more advanced condition, when there are two beats of the heart to one of the pulse, a careful examination of the heart is most instructive. It is often difficult to resist the impression that the two ventricles are acting alternately. The impulse in the first of the two beats will give the sharp apex tap, characteristic of an advanced stage of this form of heart disease; the second will be felt only or mainly over the right ventricle. On auscultation, if there is regurgitation as well as stenosis, the systolic murmur and short sharp first sound will be heard at the apex with the first beat, but will be absent at the second. *Vice versâ*, the sounds of the right side of the heart will be heard alone at the second beat with the tricuspid regurgitant murmur, if one is present, and will be comparatively weak with the first and stronger of the two beats; the pulmonary second sound, however, bearing witness that the right ventricle has contracted. When there is no regurgitation

the first beat will have the loud, short, sharp, first sound at the apex and both sounds at the base; the second the low-pitched first sound of the right ventricle and a loud pulmonary second sound, but no aortic second sound. Usually the presystolic murmur will have disappeared before the coupled beats under consideration set in.

In the absence of mitral stenosis, and of the influence of digitalis, the double heart-beat with only one pulse is considered by my friend, Dr. Raymond Tripier, of Lyons, to occur only in connection with epileptiform attacks, and to be of very serious import. The association of a slow pulse and epileptiform convulsions has been observed not infrequently, and Professor Tripier's results would explain such association and define more strictly the conditions under which it holds.

In an important and interesting memoir published in the *Revue de Médecine* 1883-4, he relates two cases observed by himself. In one the patient, a man aged forty-five, given to excess of alcohol, had, from time to time, a series of attacks of *petit-mal*, in which the pulse, which was habitually sixty, fell to eighteen, and even twelve beats per minute. In the region of the apex indistinct sounds were heard corresponding in time and number with the pulse; over the right ventricle beats were audible to the number of eighty-four, and the same number of jugular pulsations were seen in the neck.

The patient died suddenly, and no important change was found in the heart.

In the second case the patient, a woman aged fifty-three, the subject of chronic albuminuria, became subject to syncopal attacks of an epileptiform character, and was found to have a pulse of forty-four and heart-beats eighty-eight per minute. When these attacks were absent the pulse varied from

seventy-six to 100. Careful inquiry elicited the information that she had had *petit-mal* from time to time since the age of fifteen. She gradually sank and ultimately died comatose. There was an excess of subpericardial fat, some dilatation of the ventricles, and atheromatous changes in the mitral and aortic valves, which, however, were competent. The kidneys were fatty.

With these he collates cases recorded by other observers to the number of nearly thirty, which, after criticism, he considers to support, or, at any rate, not to be inconsistent with, the conclusions which he formulates as follows: "Il semble donc qu'il n'y a pas de pouls lent avec épilepsie sans déviation du rhythme cardiaque, de même qu'il n'y a pas de déviation du rhythme cardiaque avec ralentissement du pouls sans épilepsie ou sans l'action du digitale, tout au moins d'après les faits dont nous avons connaissance." My own experience, however, does not accord with his; but, before relating the cases which seem to me to be in opposition to Dr. Tripier's argument, I may add one more to the series which he has collected with so much industry and employed with so much ability, illustrating the connection which he insists upon between epileptiform attacks and the dropped beat and bigeminal pulse.

C. W., a carman, aged forty-seven, but looking ten years older, was admitted into St. Mary's Hospital under my care, March 2nd, 1877. He had had rheumatic fever eight years before, and had suffered from occasional attacks of bronchitis. For eighteen months he had complained of palpitation after hard work, and he had had fits, of which no exact description was obtained. After a recent attack of bronchitis, from which he had recovered about three weeks, he had had for a fortnight an increasing sense of palpitation of the heart, with pain which he called

"working pains," across the upper part of the abdomen. On admission he had an anxious look, and the skin was clammy; the temperature 97°; the urine had a

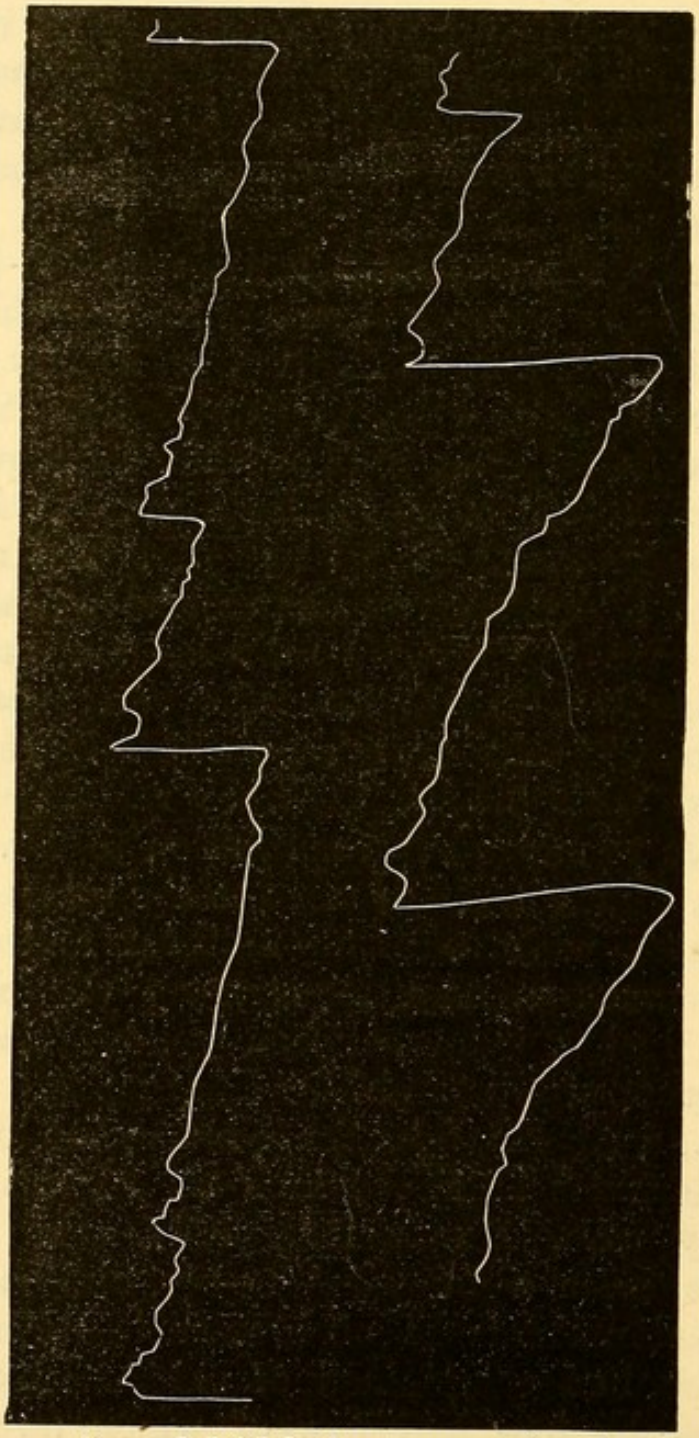


Fig. 29.—Infrequent Pulse and Pulsus Bigeminus.

specific gravity of 1010, but contained no albumen; the pulse was 31, large and strong, not quite regular either in force or frequency; at times there was an echo of the beat (pulsus bigeminus). (Fig. 29.) The

heart was of normal size; the apex beat was distinct, and often followed at a brief interval by a second beat. At the apex a low-pitched systolic murmur, beginning with an accent, was heard; it was soon lost beyond the apex, but within it was audible over the right ventricle and up to the pulmonary area. The aortic second sound was heard over the entire heart. As a rule, the heart-beat was single, but from time to time there was a double beat. When he was made to walk rapidly the patient felt faint, but did not stagger, and secondary beats were more numerous. He was in the hospital two months, and improved much in strength, no fits occurring: the pulse, however, remaining at 28. At one time, when suffering more than usual from "working pains" all over the abdomen, the pulse was 59, and quite regular. His subsequent history is unknown.

A well-marked example of infrequent pulse with double heart-beat, but without epileptiform attacks or serious effects on the health, came under my notice in November, 1883, in a tall, strong, broad-chested man, aged fifty-two years, who had been engaged in exciting political agitation, and had suffered a severe domestic loss. He was complaining chiefly of continuous dreaming and of depression and loss of energy, with heaviness and confusion of the head. Having found a pulse of 40, I wished to examine the chest, but he said this was quite unnecessary, since, whatever else might be amiss, the heart and lungs at any rate were all right, and he declared that he could walk up-hill as well as ever, and even run. There was no impulse or apex beat, but on auscultation a normal first and second sound were heard all over the heart, followed rather sooner than usual by a first sound without a second, a weak pulmonary second sound, however, being audible when listened for with care—that is, at the apex the succession of sounds

heard was "one, two, one; one, two, one;" instead of "one, two; one, two; one, two," with a slight emphasis on the final first "one." Over the right ventricle "one, two, one, two; one, two, one, two." The first only of the two beats gave a pulse at the wrist; the second, indeed, not raising the aortic valves. (Fig. 30.) All the symptoms disappeared; but two years later, after another period of excitement and anxiety, he again consulted me, and with similar complaints he had a return of the peculiar action of the heart. The pulse was 34, quite regular. There was a diffused double cardiac impulse without defined apex beat, one of them strong, giving a pulse, followed quickly by a weaker one, which sent no wave to the wrist. The sounds were much as they have been described before, but the first

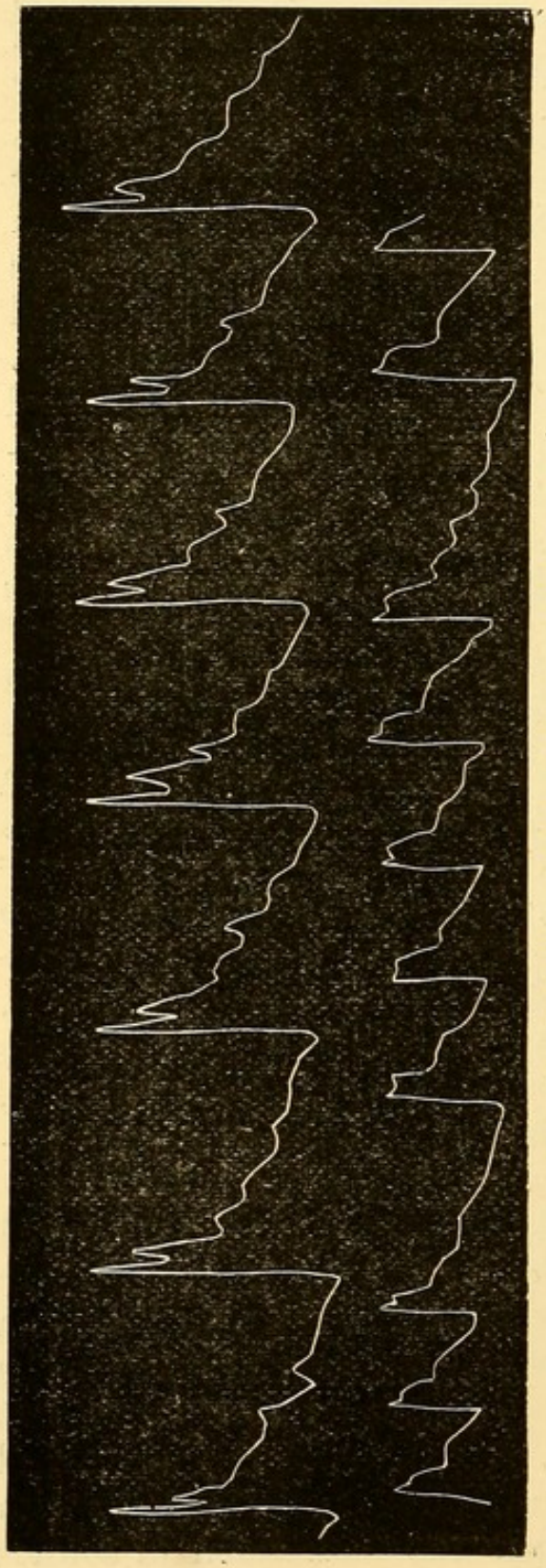


Fig. 30.—Infrequent Pulse, with Double Heart-beat.

sound of the second weaker beat was almost inaudible at the apex and distinct over the right ventricle. This patient again recovered, and is still making much noise in the world.

In the same year I saw a medical man of about sixty, who, when rather overtaxed by work, had run some distance on being called to an urgent case. This was followed by strange faint feelings and an uneasy sensation about the heart. He was not otherwise out of health. He had discovered that his pulse was 40 instead of 70, and on examination this was found to be due to the heart beating in couples—a strong beat with normal sounds which gave a pulse, followed quickly by a weaker beat with sounds which appeared to belong to the right ventricle only. This had been going on for ten days when I saw him, and continued for ten or fourteen days longer, coming and going towards the end of this time. Rest and change restored the equilibrium of the circulation, and this gentleman is in active practice at the present moment.

In November, 1883, I saw a gentleman, aged fifty-three, who had enjoyed good health until the previous June, when he had an attack of congestion of the lungs and mitral regurgitation was found to exist. He looked well, but complained of shortness of breath on exertion. The pulse was large, rather tense, perfectly regular at 72 beats a minute, except that from time to time the rate was 36, which was found to be due to the fact that, while this continued, the beats of the heart were in couples, one only of which reached the wrist. The apex beat was well-defined and forcible, and near the normal situation. A long, smooth, high-pitched, musical murmur was heard at and to the left of the apex, and in the back; another murmur of lower pitch was audible along the right edge of the upper part of the sternum up to the sterno-clavicular articulation; the aortic sound was

accentuated, the pulmonary masked by overlapping lung. When the coupled beats were present, no second sound followed the later of the two, so that the sounds ran "one, two, one; one, two, one." Both the mitral and aortic murmurs were louder with the first beat than when the heart's action was regular, and absent, or scarcely audible, with the second; this second beat, however, gave a loud first sound, so that it was not abortive from mere weakness, but the absence of the murmurs and of the second sound was probably due to an imperfect diastole. A week later, after rest in bed, the physical signs were unchanged, except that when a series of coupled beats occurred, which was as frequently as before, the second of the two beats was weaker and more precipitate. This patient, after a Mediterranean trip, again came under my care in February, 1884, and was then observed to have Cheyne-Stokes' respiration, which, however, did not prevent him from going to the City until thrombosis of the deep tibial veins came on. He died from the effects of thrombosis in the right innominate vein. The peculiar heart-rhythm was never heard again; but the case is the more interesting from the occurrence of this peculiarity in the action of the heart, and later of Cheyne-Stokes' respiration, which Tripier considers to belong to the same order of phenomena.

The double heart-beat for each beat of the pulse was present again in the case of a lady seen in October, 1885. She had worked very hard in her father's parish, which involved much walking up-hill, from April to June, when she began to look ill, and was breathless, which, however, she did not notice herself. She then suddenly broke down, and had been resting up to the time when she consulted me. She looked well, and all the functions were natural, but she felt as if she could sleep continually. The pulse was infrequent, and two beats of the heart—one like an echo

of the other—were felt and heard for every pulsation at the wrist. The aortic, as well as the pulmonary, second sound seemed to be audible in the second weaker beat, although no evidence of the systole having raised the aortic valves is recognisable in the tracing. (Fig. 31.) Under the excitement produced by the application of the sphygmograph, the rhythm gradually became normal—not suddenly, as is usual.

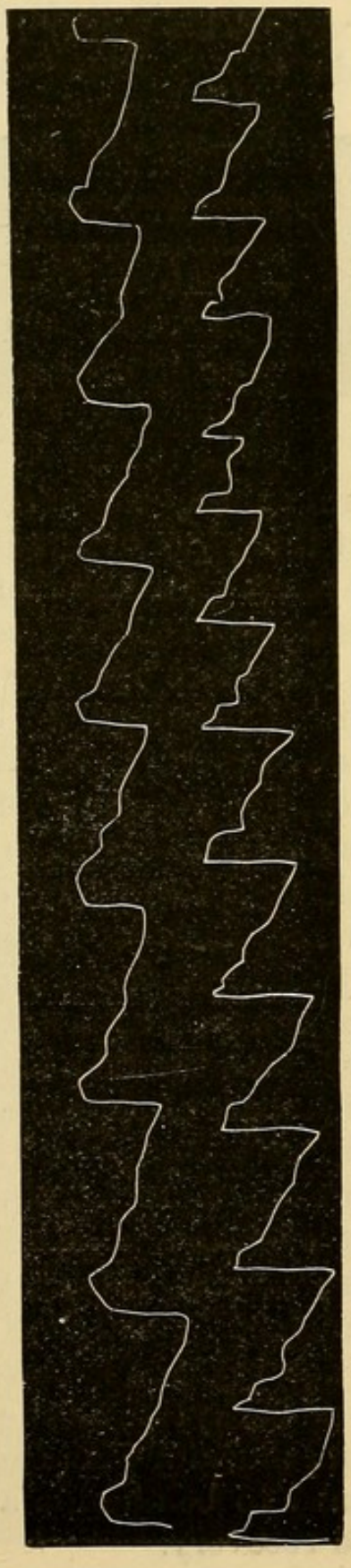


Fig. 31.—Infrequent Pulse, with Double Heart-beat, gradually becoming Normal.

Another case has recently come under my notice in a gentleman, aged sixty-three, who consulted me on account of faintness and flatulence. He had been nervous about his heart for thirty years or more, and had been afraid of exercise. He had neither overwork nor anxiety; but some months before he consulted me he had had a fall from his horse, and had broken his collar-bone, since which he had not ridden. The pulse was at first 108, and regular; but from agitation, or from the effort of taking off his coat, it fell to 54, the number of heart-beats being exactly double, a strong and weak beat being coupled together, with sounds belonging

predominantly to the left and right ventricle respectively. This continued to the end of the interview, and a tracing of it is here given. (Fig. 32.) I have

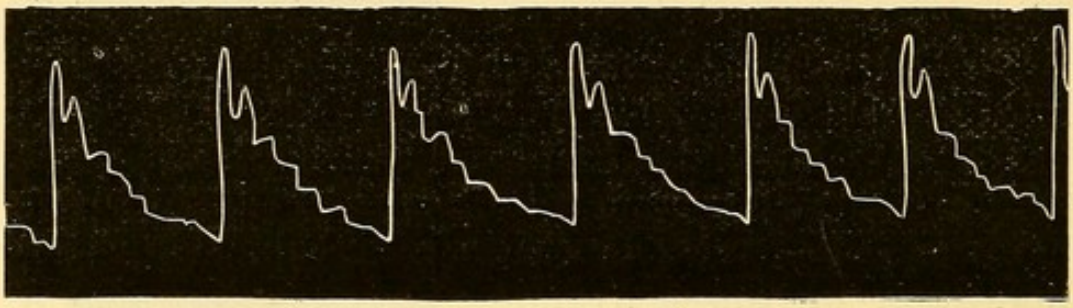


Fig. 32.—Infrequent Pulse with Double Heart-beat.

learnt subsequently from his regular medical attendant that this rhythm has been observed from time to time for years. In none of these cases have epileptiform or syncopal attacks been known to occur.

Still more recently, I have seen, with Mr. Stanley Smith, an example in which the bigeminal pulse had followed the dropped beat. An old lady, aged seventy-seven, had been to "Olympia" on February 11th. She was ailing on the 12th and 13th, and prostrate on the 14th, complaining of oppression in the chest and respiratory distress. She was found to have a pulse of only 35 to 40, instead of 76, and the double heart-beat. She improved under stimulant treatment, and when I saw her on the 17th had a pulse of 60 in coupled beats, which were about equal in strength and followed each other rather quickly, the pause being long enough for two more beats at the same rate. The rhythm was that of common time in music—"one, two, three, four;" "one" and "two" being beats, and "three" and "four" silent. No cardiac impulse could be felt, but the beats as followed by means of the sounds had the same rhythm as the pulse, and there was a systolic mitral murmur with each. A doubtful abortive systole seemed to be audible at times. This old lady made a good recovery.

A single instance of the pulsus trigeminus may be given. The patient, a needlewoman, aged forty-one, employed in St. Mary's Hospital, had been in better circumstances before taking this situation, and after fifteen months in it, was set to do laundry work; lifting and carrying heavy baskets caused blood to well into her mouth, but in spite of this and of feeling weak and languid, she persisted without complaining for a fortnight, when she was sent into the ward on Sept. 14th, 1885. She looked languid and had from time to time sharp pain in the cardiac region. The pulse was trigeminal with two distinct and similar beats and then an intermission, the third beat, however, being very faintly indicated by the sphygmograph. On auscultation of the heart, which was of normal size, a triple rhythm was observed. The first beat gave perfectly normal sounds; the second, a good first sound,

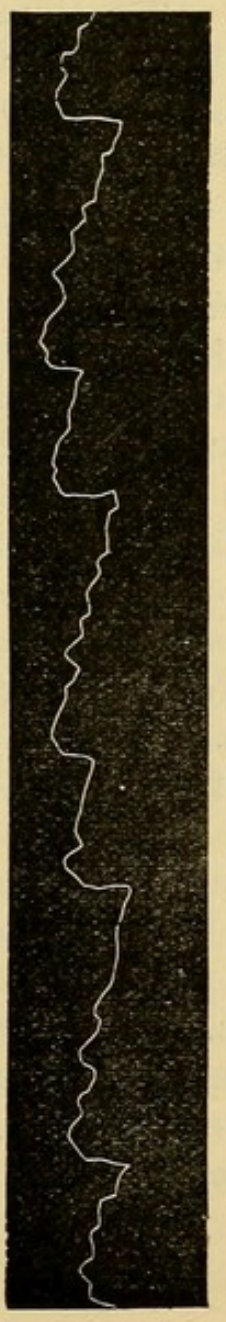


Fig. 33.—Trigeminal Pulse.

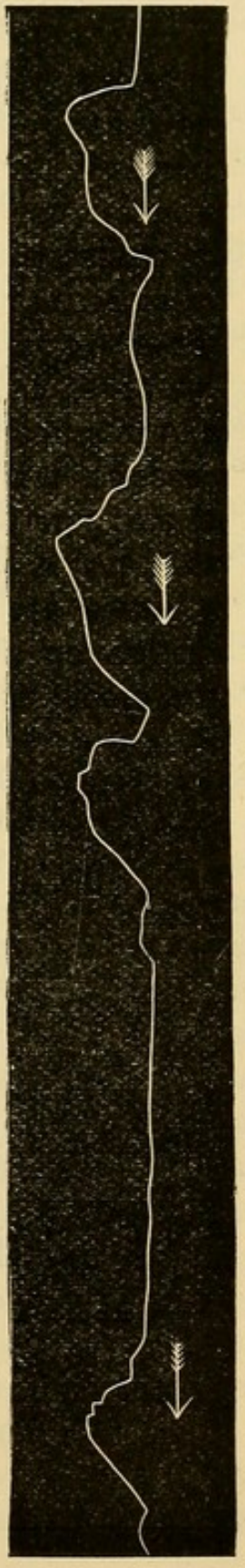


Fig. 34.—Cardiogram, showing the Rhythm of the Heart in case with Trigeminal Pulse.

but a reduplicated second sound at the base; the third beat had no second sound at all, and the first was rather precipitate. A cardiogram taken as well as the sphygmogram, by my clerk, Mr. S. A. Tidey, shows the rhythm of the heart. (Figs. 33 and 34.) As the patient gained strength, the cycle was extended to four and five beats, while in bed; but reverted to the trigeminal type when she began to walk about, and it had this character when she returned to her duties as needlewoman.

It seems to me that the cases cited invalidate Tripier's conclusion that the peculiar modification of the cardiac rhythm, in which every other beat is dropped, is always associated with, and due to, epilepsy, and usually of very serious import; nor can I accept the other conclusion that a slow pulse with epileptiform attacks is always due to dropping of beats. The decision is important, since upon it turns the question of whether, as Tripier believes, it is the epilepsy which affects the cardiac rhythm, or whether, as I think, it is the slowing of the circulation which causes the convulsions. Before entering upon any discussion on the subject, however, I may bring evidence to show that there may be a slow pulse with syncopal or epileptiform attacks which is not due to abortive heart-beats. I have myself seen a case of fatty degeneration of the heart attended with syncopal attacks which soon after proved fatal suddenly, in which the pulse was infrequent, without any interposed feeble heart-beats being detected on careful examination. The patient, however, was seen only once in consultation. The notes of a hospital case with numerous tracings which I think would have corroborated this are, unfortunately, missing.

The causes of the bigeminal pulse and the dropped beat are varied, but mental or bodily strain is one of the most common. Dr. Tripier finds in the severe

and fatal cases attended with epileptiform attacks, which he describes, evidence of profound cachexia and deterioration of the nervous structures.

The treatment will consist of rest from work of all kinds, protection from anxiety, and measures for the restoration of the general health. The bromides are sometimes of service, especially when there are frequent epileptiform attacks, and perhaps chloral may be useful in some cases.

In the *Lancet* for 1885 cases of slow pulses with epileptic seizures are related by Mr. F. St. George Mivart and Dr. A. T. Gibbings. Mr. Mivart's patient was a man aged sixty-one, who had been subject to rapid and violent palpitation of the heart. His first fit was six years before he came under Mr. Mivart's care, and this and most subsequent ones came on while he was stooping. His pulse-rate was twenty-four, and there is no record either of interposed weak beats or of abortive systoles. In Dr. Gibbings's case, the patient, aged sixty-five, had for some time had an intermittent pulse of about sixty. After an attack of bronchitis he suffered from dyspnœa and faintness, and the pulse was found to be only forty-four. Still later, he became subject to frequent epileptiform attacks and the pulse had now fallen to twenty-two, but was full and regular, the heart-beats corresponding in time. It is noteworthy that the epileptiform attacks would come on when the patient sat up. The pulse continued to fall and it reached twelve or thirteen, the attacks being now so frequent that he went out of one into another. The urine also became albuminous. With a subsequent return of the pulse to thirty-one, the albumen disappeared, and he was so much better as to be able to do a little work. While apparently better, he fainted at stool and died. Nothing abnormal was found either in the heart or in the nervous centres. This patient was seen by Sir

Andrew Clark and the late Dr. Moxon, and it is distinctly stated that the heart-sounds were normal. We may conclude, therefore, that there were no abortive beats; it is true that it is not expressly stated that they were absent, but in a case of such interest they could not have been overlooked.

The infrequent pulse of fatty heart may fairly be attributed to the structural degeneration of the organ; but an infrequent pulse, when the heart is sound, and especially if it is due to the occurrence of alternate weak beats, can scarcely be explained in any other way than by the intervention of the nervous system. This does not bring us much nearer a true comprehension of the phenomenon; and, as I have already stated, I do not accept the conclusion at which Tripier has arrived, that it is an effect of epilepsy. On the contrary, I look upon convulsive attacks, when they occur in connection with an infrequent pulse, as a result of cerebral anæmia, produced exactly in the same way as the convulsions after great hæmorrhage. In Mr. Mivart's case, the convulsions came on when the patient stooped, in Dr. Gibbings's when he sat up; changes of attitude being sufficient in the extremely feeble state of the circulation to determine such an arrest of the blood-supply to the brain as would give rise to an attack. The late Dr. Moxon, it will be remembered, went so far as to suggest that the initial event in common epilepsy was a stoppage of the heart.

Another question is suggested by some of the cases of coupled heart-beat. In almost all, while the aortic second sound of the weaker beat which fails to reach the wrist is absent, the pulmonary sound is audible. In many the impulse and first sound of the right side of the heart belonging to the second beat are more distinct than those of the left, and occasionally there is such a difference in the

situation of the impulse and in the character of the sounds as to suggest that the two sides of the heart are beating alternately; there may even be a mitral murmur with one beat, a tricuspid with the other. The extreme case is met with only in mitral stenosis under digitalis. Now, while it must be taken as demonstrated that there is no such occurrence as an alternating action of the two ventricles, it is certain, from numerous observations, that in the beat which does not raise the aortic valves, the systole of the right ventricle is often forcible, and the pulmonary second sound is usually distinct. This last sound—the pulmonary second sound—means, of course, that blood has been thrown into the pulmonary artery; and the absence of the aortic second sound means that none has entered the aorta; and as the amount of blood which passes through the right and left heart in a given time must be equal, so long as the equilibrium between the pulmonary and systemic circulation is maintained, the propulsion of blood from the right ventricle only must be supplementary. In mitral stenosis, it is easy to understand that such an extra contraction of the right ventricle might be useful in maintaining the pressure in the pulmonary circulation which is necessary in order to force the blood through the constricted mitral orifice; or, regarding it in another aspect, the abortive contraction of the left ventricle is due to its being inadequately filled during one diastole, so that a second is needed. No such necessity, however, is apparent when both valves and walls of the heart are normal.

CHAPTER VII.

INTERMITTENT AND IRREGULAR PULSE.

THE rhythm of the heart's action is deranged chiefly in two ways: by the omission of a beat from time to time, and by the beats generally coming at unequal intervals. The rhythm of the pulse will vary with that of the heart; and in the latter case the beats of the pulse are also of unequal force. Occasionally inequality in the force of the pulse is more marked than the irregularity in its time.

Intermittent pulse.—The term "intermittent" is employed to designate the pulse when a beat is completely missing from time to time, while the pulse in the intervals is perfectly regular. It is a remarkable variety of the pulse, and is, perhaps, the least capable of explanation of any. It is not, so far as I am aware, producible by experiment. We are compelled to fall back on nervous influence as the agent in its causation; but, while it is easy to understand a general disturbance of the action of the heart producing complete irregularity, the omission of a beat at stated or varying intervals is a curious exercise of the power of the nervous system.

The intermission may happen at regular and definite periods every four, six, or up to twenty beats, or the number of intervening pulsations may vary. The interval produced by the missing beat is not usually quite equal to two beats, and the succeeding beat feels stronger from the pressure in the arteries having run down. (Fig. 35.) The heart-beat, again, which immediately follows the intermission is usually more powerful than the others.

The intermittent pulse may be habitual and

constant, and, in this case, is more likely to be at definite intervals; or it may be occasional only, under the influence of some disturbing cause, of which flatulent indigestion is the most common.

A regular intermission may be disturbed by varying circumstances —exercise, excitement, and the like —when, as a rule, it occurs less frequently, and the pulse may be for a time quite regular; pyrexia almost always suspends the intermission while it lasts. Occasionally nervousness or fatigue will render the intermissions more frequent.

In some persons the pulse is intermittent after each meal; in others, tea, coffee, or tobacco is the special cause of the intermission. Intermittent pulse is very common in chronic gout, either as an effect of high tension, or as the result of the action of the gouty poison on the heart directly, or on the nervous system, or as a concomitant of dyspepsia. Arterial tension may be excluded, since intermission is not common in other affections, such as chronic Bright's disease, attended with this condition of the circulation. It has been found to be established as a result of shock, mental or bodily. It may, again, be associated with nervousness and hypochondriasis, coming and going with the accession of one or other.

Fig. 35.—Intermittent Pulse.

An intermittent pulse may be

among the signs of fatty degeneration of the heart, but it will not stand alone. In case of doubt, the patient should be made to walk briskly for a minute or two, when the really weak heart will falter; while if the heart is healthy, the intermission will usually disappear.

The patient may or may not be conscious of the intermittent action of the heart. He is more likely to feel it when it is symptomatic of some functional derangement than when it is habitual, and he may have merely a vague sense of discomfort in the cardiac region, or he may be conscious of the stoppage of the heart and of a disagreeable sinking sensation attending it, or he may feel the bump of the stronger beat which follows the intermission.

On examining the heart it is usually found that the cause of the intermission of the pulse is not the actual omission of a heart-beat, but the occurrence of a hurried and very imperfect contraction which rapidly follows the last of the series of normal beats. It can often be felt by the hand, not, however, as a true impulse but as a kind of vibration; usually only its first sound is heard, though at times a feeble pulmonary second sound is also audible. While it is the rule that there is this feeble interposed beat, instances occur in which nothing of the kind can be heard or felt, and in which, therefore, the heart appears to remain quite passive.

The constant intermittent pulse appears to have no significance either in relation to the heart, or to the nervous system, or to the vital power generally. It is common in men who retain vigorous health to a good old age, and I have met with it at the age of eighty, when it had been known to exist for forty years. Dr. B. W. Richardson, however, states that acute disease is not well borne when the pulse is intermittent.

A practical question not uncommonly arises as to whether it is safe to administer chloroform when the pulse is intermittent. I do not hesitate to authorise it when consulted on the subject, on condition that the pulse be carefully watched. The rule is that the chloroform suspends the intermission ; should it have a contrary effect from the first, the pulse becoming weak and irregular, fatty degeneration of the heart may be suspected, and the administration should be at once stopped.

It is unnecessary to say that no treatment of intermittent pulse, as such, should be attempted. When it is traceable to tobacco, tea, or coffee, these should be relinquished ; and when it is symptomatic of indigestion, or other cause of reflex disturbance of the heart, appropriate remedies should be applied. If the nervous system is depressed, influences tending to this should be removed, and such tonics as strychnine and arsenic or zinc may be given, or occasional bromides.

Irregular pulse.—This term conveys its own meaning ; the beats follow each other at irregular intervals, and are unequal in force. In degree, irregularity of the pulse varies greatly, and it is not impossible that different varieties may have a different significance. Slight irregularity needs no further notice ; but when it is extreme there may be a rapid succession of small, weak beats, and then a few large and distinct; or there may be no method whatever in the irregularity, no two beats being alike, either in time or force.

Irregularity, like intermission, may be either habitual or occasional. When occasional, it may be induced by reflex disturbance of the cardiac rhythm, by gastric derangement, with or without flatulence, by flatulent distension of the colon, or by other functional affections. When there is distension, either of the

stomach or colon, it may be mechanical embarrassment of the heart by upward displacement of the diaphragm which sets up the irregular action. Tobacco is a very common cause of irregular and weak pulse, especially the stronger forms and such as are rich in nicotine. In one case in my experience, irregular action of the heart, attended with unusual discomfort, was traced to a particular and very fine brand of cigars. Tea in this respect is to women as tobacco to men. Irregularity of pulse and palpitation mostly go together as symptoms of reflex disturbances.

The regularity of the pulse may be disturbed either temporarily or permanently by affections of the respiratory organs. The pressure in the large arteries near the chest rises and falls with each respiration, and the influence on the circulation thus manifested as a normal phenomenon, is sufficient to affect the regularity of the action of the heart when exaggerated in any way. A deep inspiration, and still more, to hold the breath either at the end of inspiration or expiration, will affect the pulse, as has been shown in a previous chapter.

The respiratory variations in the blood-pressure are variously explained. Professor Burdon Sanderson attributes them, in his "Croonian Lectures," to the effect on the respiratory centre of varying aëration of the blood; Marey, to the varying pressure upon the aorta. But it seems to me that the respiratory variations of pressure will tell most on the thin-walled auricles; and that while in the right inspiration helps to draw blood from the vena cava, in the left it will influence the amount of blood passed on into the ventricle. Respiratory effort when the air-passages are obstructed will intensify such effects, and these will be still greater when the lungs themselves are affected, as in bronchitis, and obstruction in the pulmonary circulation is added to obstruction in the minute bronchial tubes. The frequency of the pulse and other changes

in the circulation due to pyrexia, disguise, more or less, the irregularity of the pulse in acute bronchitis, but this is often distinct in chronic bronchitis and emphysema; and it may be noted on careful observation that the effect on the pulse is most marked at the end of inspiration and expiration, and is thus coincident with the changes of pressure in the chest.

Habitual irregularity of pulse is a common result of mitral insufficiency, and among valvular affections of the heart is the characteristic pulse of this form of disease. It is so frequent in mitral regurgitation, and so rare in other forms of valvular disease, that it can scarcely be put down to any secondary alterations of the cavities or walls of the heart; still less can it be attributed to nervous influence. It appears to me that it is explained by mechanical or hydrostatic conditions, peculiar to insufficiency of the mitral valve, which intensify the effect of the varying pressure upon the heart in respiration. The heart lying in the mediastinum between the lungs is habitually exposed to negative pressure; the lungs, were the pleural cavities opened, would collapse, and this tendency to collapse, which is only prevented by their being lodged in closed cavities, will exercise traction on all the walls, the costal parietes, diaphragm and mediastinum; the lungs, therefore, do not press upon the pericardium, but drag upon it and upon the great vessels, and the suction-action thus caused is a recognised aid in promoting the flow of blood in the vena cava into the right auricle. When from damage to the valve there is reflux of blood from the left ventricle into the auricle, the auricle is dilated and, although there is some hypertrophy of its muscular walls, these are still so thin and flexible that they are incapable of resisting variations of external pressure, the influence of which will be all the greater in consequence of the dilatation. Now, in mitral

regurgitation, the blood is driven partly onwards into the aorta, partly back again into the auricle, and the proportion which passes onwards or backwards will depend on the degree of resistance met with in one or other direction: if the pressure in the arterial system is low, and that in the pulmonary veins and left auricle great, there will be little reflux into the auricle, however slight the obstacle presented by the damaged valve; if, on the other hand, the systemic arterial tension is high, while the pressure in the auricle is low, the regurgitation will be considerable. Whatever, then, increases or diminishes the blood pressure in the auricle, will have a corresponding effect on the amount of regurgitation. Now, the dilated left auricle will be supported by a certain amount of pressure during expiration which will tend to resist distension by the regurgitant blood; but at the end of expiration the conditions are suddenly reversed, and it will be exposed to the negative pressure or suction-action which fills the chest with air, so that the reflux is favoured. The sum total of the resistance to the ventricular systole is thus diminished at this moment, and the systole is consequently very rapid, at the same time that less of the blood goes forward into the aorta. At the end of inspiration opposite forces come into play, and the repetition every two or three beats of these disturbing influences quite accounts for the irregularity of the heart's action. In an early stage the irregularity can be seen to occur coincidentally with the end of inspiration and expiration. It is the incompetence of the mitral valve which exposes the ventricles to the respiratory pressure variations. In mitral stenosis, although the auricle is dilated and subjected to the varying pressure of the respiratory movements, the narrowed orifice prevents this from taking effect on the ventricle.

Dilatation of the heart is frequently but not constantly attended with irregularity of the pulse; when it is present its influence and significance are not very clear, and the prognosis is determined by other considerations.

Extreme and habitual irregularity of the pulse may be present which cannot be traced to any other cause, and must, therefore, be attributed to the nervous system. Irregularity of pulse of nervous origin is illustrated by the disturbance of the cardiac rhythm in dyspepsia and by tobacco, already mentioned; but it may be present in an extreme degree independently of any recognisable influence, and may be habitual. There need be no affection of the general health, or impairment of vigour or endurance. In one of the worst cases I have ever seen, the patient, who was for some time under my observation, was, long after the age of sixty, in the habit of addressing public meetings. The trace exhibited was taken recently upon a gentleman, now aged seventy, who consulted me twenty years ago on account of irregular action of the heart, and has had it ever since. (Fig. 36.) I

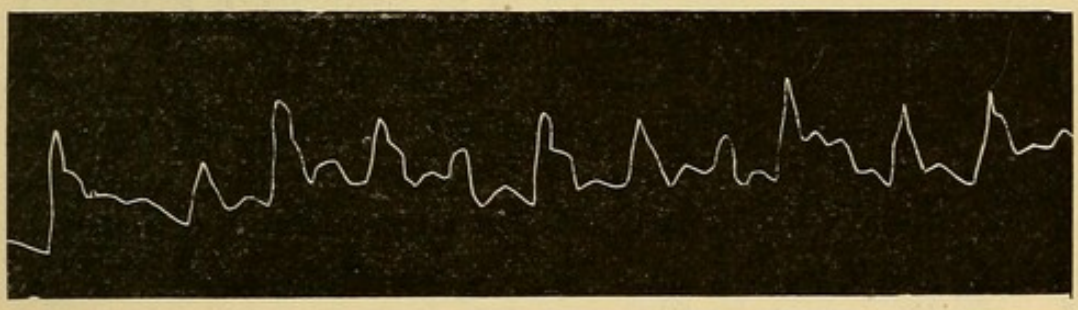


Fig. 36.—Irregular Pulse.

am unable to fix any prognostic value on irregularity of the pulse as such. Speaking generally, however, irregularity is much more serious than intermission.

CHAPTER VIII.

THE PULSE AS INFLUENCED BY VARIATIONS IN ARTERIO-CAPILLARY RESISTANCE—LOW ARTERIAL TENSION.

THE effects on the pulse of variations in the force of the heart's action do not require separate discussion, and we now proceed to the consideration of those characters of the pulse which are due to changes in the vessels.

Of these, the most important are those produced by contraction and relaxation of the arterioles and capillaries, which, as has been already said, give rise to the variations in the arterial tension, or pressure within the vessels, by hindering or facilitating the passage of the blood through the capillaries.

Seat of arterio-capillary resistance.—A preliminary discussion must be undertaken here: namely, whether the freedom of flow through the peripheral vessels is determined entirely by changes in the size of the arterioles, or is influenced primarily by the degree of obstruction in the capillaries: whether, in fact, the stop-cock action of the arterioles—as the contraction and relaxation of the arteries was happily named by Dr. Geo. Johnson—is the sole or main agency in raising and lowering the arterial tension, or is secondary to changes in the flow of blood through the capillary network.

Arteriole contraction and relaxation.—It must be admitted at once that the muscular walls of the minute arteries respond more promptly and energetically to nervous stimuli than the capillaries; and in emotional and reflex influence upon the peripheral circulation there can be little doubt that spasm or relaxation of the arterioles is the

mechanism employed. When, for example, there is from nervousness sudden and fugitive high arterial tension, it is in part due to tightening up of the minute arteries, and not solely to the hurried and forcible action of the heart; and in blushing, it is relaxation of the arterioles of the affected region which allows the skin of the face and neck to be flooded with blood. Such relaxation may be partial, as is frequently seen on exposing the chest in young women for the purpose of stethoscopic examination, when it is found to be covered with large bright red blotches.

Chief resistance in capillaries. — But while the arterioles are competent to influence the supply of blood to different parts and organs of the body, and undoubtedly play an important part in regulating this, in doing which they will produce corresponding effects on the arterial tension by opposing or facilitating the flow into the capillaries, it is probable that the capillaries themselves are the seat of the principal obstruction to the onward movement of the blood, and of those variations in the degree of obstruction which are most influential in modifying the blood pressure. It is indeed certain that it is in the capillary network that the normal physiological resistance in the peripheral circulation takes place, and it is only here that the resulting pressure could have the effect which it subserves, of promoting the transudation through the capillary walls of nutrient material for the use of the tissues. Up to the very edge of this network the blood pressure in the arteries and arterioles is maintained; beyond it there is only just sufficient to carry the blood back to the heart in the veins. We might reasonably expect, then, that where the resistance which gives rise to the pressure in the arterial system is originally situated, there would arise those differences in the degree of

resistance which affect arterial tension. Evidences in favour of this view are not wanting.

The relaxation and contraction of the arterioles are reflex, or, at any rate, take place in response to stimulation of their muscular walls by the vaso-motor sympathetic nerves, but experiments with a variety of drugs have shown that variations in the rate of flow through the capillaries and in the arterial tension can be induced when the spinal cord and sympathetic ganglia and nerves are destroyed—*i.e.* when the vaso-motor nervous apparatus is abolished. Drs. Ringer and Sainsbury have described such experiments made with the digitalis group of remedies (Med. Chir. Trans., vol. lxvii.), and corresponding results have been obtained with amyl nitrite and other relaxants of the peripheral vessels by Dr. Lauder Brunton. Important evidences, again, are derived from an examination of the minute arterioles in the brain after death, resulting from contracted granular disease of the kidney. When there have been uræmic convulsions, capillary hæmorrhages are almost always present in various parts of the cortex, so much so that the late Dr. Mahomed attributed the convulsion to capillary rupture. If the capillaries were protected by contraction of the arterioles, such rupture could scarcely occur. Again, these arterioles are themselves at certain points dilated into miliary aneurisms and liable to rupture: not where they are given off from larger branches, but just where they break up into the capillary network, which shows the obstruction to have been beyond, and not in, the arterioles; while not only are the perivascular spaces round them large, as if from distension, but a ring of the surrounding brain substance is pale, as if from pressure. Another reason is that although the medium-sized arteries (such as the radial) and the small arteries (such as the digital) are usually contracted and small

in high tension, this is not always the case, and it seems improbable that in vessels anatomically continuous and physiologically correlated there should be contraction in the minute arterioles without a corresponding condition of the arteries leading to them. We find, indeed, in cases of old standing high tension, the worn-out arteries large, as well as thick and degenerated, as if they had been unable to resist the distending pressure of the blood within them.

Capillary contraction.—Taking it as established that the capillary network is the seat of the varying resistance which affects the blood pressure in the arteries, a further question arises whether such variations are the result of relaxation and contraction of the capillaries, or of varying cohesion between the blood and capillary walls. Much is to be said for this latter view. Capillary attraction is capable of facilitating or opposing the transit of liquids according to the affinity between them and the walls of the channels; and capillary force is of itself the agent of the circulation of the sap in vegetables, carrying it to the summit of trees, and back again. There is apparently also ocular demonstration of increased cohesion of the white corpuscles to the capillary walls in inflammation. While, however, the affinity between the blood and tissues probably has a share, and sometimes an important one, in modifying the rate of the capillary circulation, the contractility of the capillary walls, which has been demonstrated by Roy and Graham Brown, is no doubt the great agent in the production of the varying resistance which influences the arterial tension. Changes in the blood, which are too slight to affect the cohesion between the blood and the capillary walls, affect powerfully the arterial tension, and the variations in the volume of a member, as demonstrated by the apparatus of Mosso and François Franck, indicate variations in the

capacity of the capillaries, best explained by their contraction and dilatation.

Although it appears from the preceding considerations that the starting-point of the physiological resistance in the peripheral circulation and of the variations in this resistance is the capillary network, the arterioles are not without an important share in the process. The contraction of the capillaries is continued backwards along the arterioles to arteries of the size of the radial, and the narrowing of the afferent channels thus produced, at the same time contributes to the production of the arterial tension, and protects the capillaries from the afflux and pressure of blood; in like manner when the capillaries are relaxed, the arterioles and arteries are large. The arteries and capillaries, in fact, form part of one system, and the expression arterio-capillary resistance is more exact than when an obstruction is qualified as either arterial or capillary alone.

Low arterial tension.—The capacity of the arterial system, as has already been stated, increases with the subdivision of the arteries, and the capillary channels are collectively much larger than the arterioles which supply them; it is conceivable, then, that the outflow might be so free, in spite of the friction between the blood and the walls of the containing vessels, that it would pass onward into the veins as fast as it was injected by the heart into the aorta. Of course the same amount of blood does pass through the capillary network generally at each pulsation as is propelled by the corresponding ventricular systole, but the systole is effected in one-third of the time occupied by the entire cardiac revolution, so that the blood is three times as long in escaping by the capillaries, and there are accumulated in the arteries, distending them and bringing into play the elasticity of their coats, a considerable number of charges of the ventricle. In

proportion as the flow through the capillaries is free, the number of heart-beats stored up in the arteries will be diminished, the mean of continual blood pressure within them and the degree of tension of their coats will be lowered, and, most important of all, the smaller will be the amount of nutrient material passing through the capillary wall for the use of the structures. Low arterial tension, which is now to be considered, then, implies a diminished arterial reserve and a lessened supply of nourishment to the tissues.

Venous pulsation.—It has just been remarked that it is conceivably possible for the blood to pass so readily through the capillaries that it would issue into the veins as fast as it was propelled into the arteries, but the actual realisation of this possibility is incompatible with life, as the functional activity of the nerve centres is dependent upon a continuous flow of blood through them, and under the circumstances supposed it would be intermittent. The resistance, however, in the arterioles and capillaries may be so slight that the blood has still a pulsatile movement when it reaches the veins. This is best seen in the veins of the dorsum of the hand, the fore-arm being held horizontally and the wrist dropped ; a long film of sealing-wax resting on the vein will render the gentle rise and fall more visible. Such venous pulsation in the veins of the back of the hand can often be demonstrated in aortic regurgitation, but here it is not so much the diminished resistance in the capillaries as the exaggeration of the pulsatile variations of pressure in the arteries which brings it about. In order to see pulsation in the veins, however, the capillaries must be relaxed by putting the hand in hot water, or there must be pyrexial relaxation of the peripheral vessels, as, for example, in acute rheumatism, especially when attended with pericarditis, which often renders it very

evident. Occasionally venous pulsation is met with when there is no heart disease, or any other cause than abolition of the normal resistance in the capillaries. I witnessed it in a gentleman who was gradually sinking from the effects of alcohol, without any of the usual alcoholic disease of the liver and kidneys. He took less and less food, and came to live on alcohol, and finally had slight pyrexia and occasional attacks of hæmoptysis. The arteries were large, thin, and soft, and for some weeks pulsation in the veins of the dorsum of the hand could be rendered visible at any time by dropping the wrist so as to allow the veins to fill.

Characters of low-tension pulse.—The essential characters of the low-tension pulse are, that the artery is so readily effaced by moderate pressure that it cannot be felt at all between the beats. It seems to start into existence with each

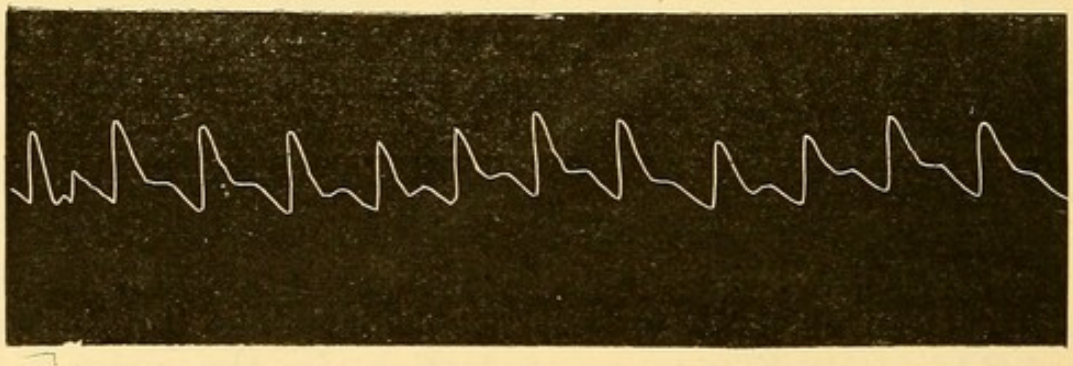


Fig. 37.—Normal low-tension Pulse.

pulsation, and to disappear as the wave passes. The pulse is sudden in its ictus, is brief in its duration, seeming to pass quickly under the fingers (celer), and its subsidence is rapid, and is broken by a dicrotic rebound, easily recognisable when sought for in the way described in an earlier part of this book.

The sphygmographic trace will have a perpendicular upstroke, a sharp top, and a steep fall, with a deep notch and dicrotic rise. (Fig. 37.)

Varieties of low-tension pulse.—There are many varieties of low-tension pulse, according to the frequency and force of the heart-beat.

When the heart is acting forcibly the pulse is large, sudden, and vehement (full and bounding), the size of the dilated artery and the shortness of the wave intensifying the impression of force conveyed to the fingers. The force and frequency of the systolic discharge of blood into the aorta may be such as to maintain a degree of fulness of the arterial system in spite of the free outflow by the capillaries, and the radial can then be felt between the beats when only

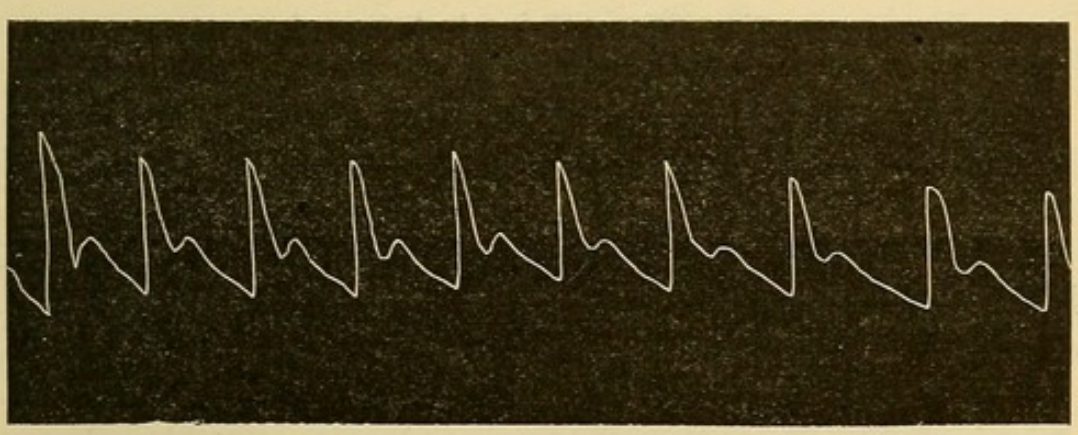


Fig. 38.—Low Tension, with forcible Action of the Heart.

moderate pressure is employed. It can, however, be flattened without difficulty. Dicrotism is, of course, distinct.

The sphygmogram will have an amplitude corresponding to the increased size of the artery, and will require a certain degree of pressure for the development of the trace. (Fig. 38.)

When the heart acts feebly, or sends out a diminished amount of blood at each systole, the diminished amount of blood in the arterial system allows the arteries to contract, and the pulse will be small and very easily suppressed; the ictus, again, will lose in sharpness, so that the fingers must be

applied very lightly. Carried to an extreme, this association of weak heart and relaxed arterioles and capillaries gives the running pulse. (Fig. 39.)

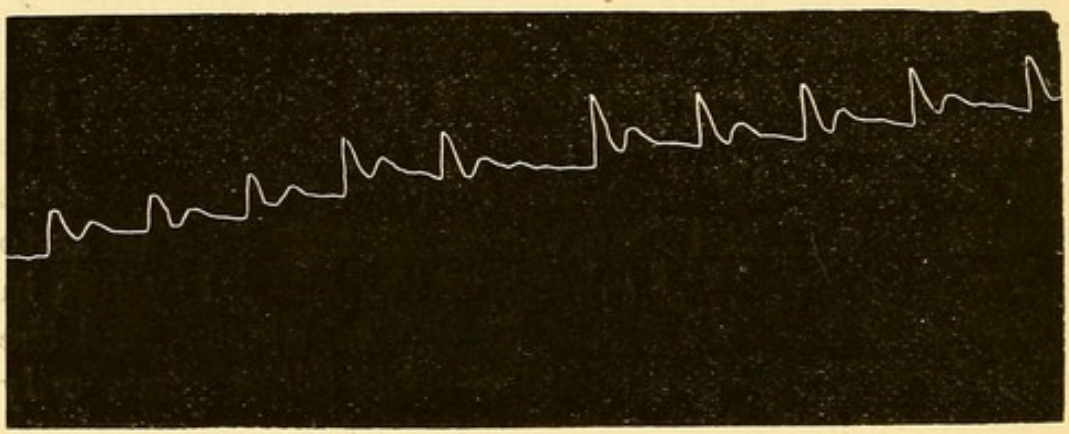


Fig. 39.—Low Tension, with feeble Action of the Heart.

Names have been given to pulses of low tension according to the position of the dicrotic notch and wave.

The dicrotic notch may descend below the base line of the trace, when the pulse is said to be "hyperdicrotic." This, however, is merely a question of the

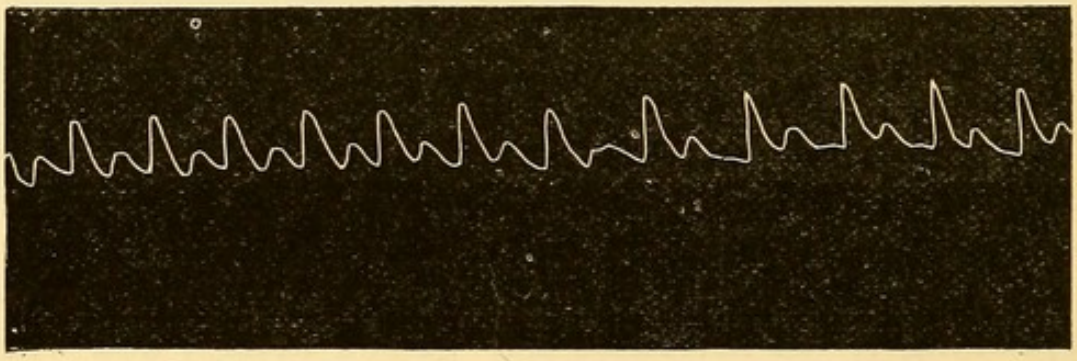


Fig. 40.—Hyperdicrotic Pulse.

degree of pressure on the artery by the spring of the sphygmograph. (Fig. 40.)

The name "anacrotic" pulse is given when the dicrotic wave seems to come in the upstroke of the next beat. This is simply a question of frequency; the dicrotic wave occurs at a definite interval after

the primary wave, and when the pulse is extremely frequent the lever has not time to fall after the dicrotic rise before the next rise is due.

Causes of low tension.—As with the time of the heart so with the tone of the arteries: there are variations on each side of the normal average without apparent effect on the health and vigour, and a low-tension pulse may be congenital, or it may run in a family. It is sometimes important to bear this in mind; absence of a proper degree of tension is one of the signs of fatty degeneration of the heart, and when present after middle age, together with symptoms of cardiac debility, it might lead to an erroneous diagnosis of this disease. A medical man who knew the family pulse to be soft would be in no danger of making this mistake.

Obesity is usually associated with low-pulse tension, the arteries also being small and the action of the heart weak. It is not unlikely that the languid movement of the blood indicated by these conditions may favour the deposition of fat.

Warmth, especially combined with moisture, relaxes the arterioles and capillaries, and lowers the arterial tension; a hot bath will do this very effectually.

Food, particularly when taken warm; hot drinks, sustained exertion, fatigue, and exhaustion, bodily or mental, are other physiological causes of relaxation of the peripheral vessels.

As regards the effect of a meal in lowering the pulse tension at the wrist, it might be attributed to the large diversion of blood to the abdominal viscera during digestion, but this will be compensated in some degree by the increased volume of the blood by rapid absorption from the gastro-intestinal mucous surfaces. It is, moreover, evident from the character of the pulse that the arteries are relaxed and large, and not simply unfilled. We see, too, in the flushed

face and red nose of certain forms of dyspepsia, especially in women and young girls, a local exaggeration of the general arterial relaxation.

Anxiety, worry, and the depressing emotions; inadequate food or deficiency in the nitrogenised constituents of food, occasionally excessive indulgence in alcohol, and various unfavourable hygienic influences, may give rise to low arterial tension.

Debility of certain kinds is attended with low blood pressure, but anæmia, especially when associated with chlorosis, often has a high-tension pulse.

Certain states of the nervous system are associated with low-pulse tension. Sometimes it is the affection of the nervous system which causes the low tension, sometimes it is absence of due intra-arterial pressure, which gives rise to the morbid condition of the nerve centres. This subject will be discussed later.

The most common cause of relaxation of the arterioles and capillaries and of low tension in the pulse, however, is pyrexia.

Effects of low arterial tension.—Deficient resistance in the peripheral circulation, and consequent abnormally low pressure, are not likely to affect injuriously the heart or arteries directly, and no morbid change in either has been traced to low-pulse tension. The nutrition of the tissues generally will not, however, be maintained at a high point, and the heart will share in the imperfect renewal of structures, especially as the blood pressure in the coronary arteries will be low, and the movement of blood in the walls of the heart languid. Moreover, the heart is not called upon to exercise full normal energy; and just as over-work in consequence of high arterial tension gives rise to hypertrophy, under-work will tend to atrophy. It is possible, then, that low-pulse tension may predispose to cardiac degeneration.

Symptoms.—The symptoms associated with a pulse of low tension are extremely varied, and they are, for the most part, not the result of the weak pulse, but concomitant effects of an underlying cause. Many of them are equally common when the pulse tension is high, and the question is not what symptoms arise out of low or high pressure in the arteries, but, given certain symptoms, what is the state of arterial tension, since this is an important guide in the treatment.

It has appeared to me that undue relaxation of the small arteries is sometimes a cause of weakness and depression by permitting undue loss of heat. It is the duty, so to speak, of the arterioles to shut off the blood from the surface of the body on exposure to cold, and thus to protect it from being cooled down. When this function is imperfectly performed the skin and the extremities may be warm in spite of very low external temperature, but the body must lose heat rapidly from exposure to cold of successive portions of blood distributed freely to the skin, and either the temperature of the body generally will fall, or increased oxidation and tissue change will be required in order to keep it at the normal level. In either case the tax on the system will be heavy, and only a vigorous constitution can support it with impunity. A sufferer from the depression produced in the way just described will often exhibit his warm hands and boast of his warm feet as proofs of his excellent circulation. Some years ago I had under my care for several successive winters a melancholy giant, one of the tallest men I ever saw, proportionately stout and well built. During the summer he was well, and was capable of considerable and sustained exertion; but in cold weather he was depressed, miserable, incapable of giving his attention to his official duties, and continually under the

fancied necessity of resorting to stimulants. At the same time, he was unconscious of the external cold, and did not take cold; he never wore an overcoat, and his hands were always comfortably warm. He could not understand that the very warmth was a cause and a mark of weakness, and refused to seek protection from cold which he did not feel to need. Under the influence of depression and want of energy he resigned a valuable appointment, and incurred the evil consequences of entire want of occupation, and is now, a confirmed hypochondriac and the subject of the curious dread of open spaces which has been called agoraphobia. Cases of this kind are by no means uncommon.

Abnormally low pulse-tension may be associated with a great variety of functional derangements, as well as of symptoms; dyspepsia, constipation, sleeplessness, headache, and a multiplicity of pains and sensations in the head, or about the heart, or in the back; and when flatulent dyspepsia and constipation are present it is sometimes a defensible hypothesis that the depression and other nervous symptoms may be due to the gastro-hepatic or intestinal derangement, and the rectification of all recognised departures from functional efficiency and regularity would be one of the first objects of treatment. It is worthy of note, however, that when the pulse tension is low the patient often feels better while the bowels are confined, and depressed and faint for some time after any action, either spontaneous or however induced. Such patients bear purgatives of all kinds badly, especially when mercury in any form enters into their composition.

Low arterial tension in diseases usually attended with high tension is prognostic of evil. This is especially the case in kidney disease, as will be seen by examples related when the subject of Bright's

Disease and associated circulatory conditions are considered later.

Treatment.—In speaking of treatment, it is scarcely necessary to say that it is not treatment of low tension as such, but of cases in which low arterial tension is a prominent symptom. The first point to be considered will be whether the imperfect resistance in the arterio-capillary system of vessels is due to the state of the blood and tissues, or to deranged nervous influence. It is not easy to establish such a distinction, for, under the influence of mental shock, or grief, or anxiety, anæmia may supervene with extraordinary rapidity; and, on the other hand, deterioration of the blood and tissues may re-act upon the nervous system. Iron, the mineral acids, arsenic, phosphorus, nux vomica, or strychnine, quinine, bark, are among the medicaments most generally useful; digitalis, again, the special tonic of the heart and arterioles, may be of service.

The food will, of course, be simple, nourishing, and digestible. Alcohol will be given with caution at meal-times only, and in the form of red wine or beer.

Change is often of the greatest service, the most powerful climatic influence being sea or mountain air, one or other being selected, according to the previous experience of the patient.

While absence of resistance in the peripheral vessels is the normal cause of low tension in the arteries, it is obvious that, since the blood pressure is ultimately due to and dependent upon the ventricular systole, the tension must be low when the propulsion of blood into the arterial system is feeble or deficient in amount. In the latter case, however, the arteries contract upon their contents, still remaining full between the beats, and the pulse becomes small without necessarily being short. Usually, relaxed arteries

and capillaries and weak action of the heart go together, or the circulation would come to a standstill. There are, however, circumstances in which a low tension pulse, due to the heart, is worthy of special note. This is in association with approximation of the first and second heart sounds already considered in chapter iv., to which attention was originally called in a paper in the *Practitioner*.

CHAPTER IX.

HIGH ARTERIAL TENSION.

UNDULY high pressure in the arterial system or high tension of the pulse is a condition worthy of careful attention and study. It explains many of the forms of failing health at and after middle age, and is often the means of shortening life through lesions of the brain and heart. It points out tendencies which later result in serious illness or fatal disease, and its recognition often directs us to measures by which ailments may be relieved, and enables us to foresee and sometimes to avert premature death.

Characteristics of high tension pulse.—High arterial tension is not to be measured by a certain number of grammes or ounces of pressure employed to elicit a characteristic sphygmographic trace; it is a relative, not an absolute term. Ultimately, the measure of the tension in the arteries is the force of the systole or the heart, but modifying influences of extreme importance are introduced by the peripheral circulation. Under normal conditions the relation between the force of the heart and the outflow by the capillaries is such that the artery gradually subsides under the pressure of the fingers in the intervals between the pulse-waves; and the chief characteristic of unduly high tension is that the vessel remains full between the beats. For our present purpose, then, it may be taken that high tension exists whenever the artery is full between the beats, so that it can be rolled under the fingers like a tendon in the wrist. To appreciate this condition, three fingers should be placed on the vessel, when it will be found to stand out not only during the wave of the pulse, but in the intervals; and, as has just been said, it can be

rolled transversely under the fingers, and can often be followed for some distance up the fore-arm, feeling almost like the vas deferens. This having been recognised, other points must then be ascertained.

The force of the pulse beat and the degree of actual pressure in the blood column may vary. This will be approximately estimated by the pressure of the fingers required to flatten the artery and arrest the wave—one, two, and all three fingers being employed, and the pressure being varied several times. Very frequently the force needed is unexpectedly great, and a pulse which at first seems to be weak may really be extremely powerful.

Not unfrequently, especially when the skin is thin, the artery can be seen to form a distinct cord-like projection along the line of its course, but no pulsation will be visible in it, unless it is thrown into a curve, when this will be seen to be accentuated at each beat.

The artery may be either large or small; sometimes it is distended and dilated to its full capacity, but so long as its coats are sound and not worn out, it will usually be contracted and small. The pulsation is not very marked, and to the fingers lightly applied seems weak, since, as has already been stated, when the mean blood pressure is high, the fluctuations are comparatively small. The onset of the wave is gradual; it is felt for an appreciable and relatively long period under the fingers, and it subsides slowly. The sphygmographic trace will have an upstroke with a faint inclination forwards, a round or flat summit, and a gradual decline without dicrotic notch or wave. (Fig. 41.) While there is no apparent vehemence of the beat, when the strength of the pulsation comes to be tested by an attempt to arrest the wave it is found to have an unexpected degree of force, and very often the greater the pressure of the fingers the

stronger it seems to become. This is especially the case when the artery is much contracted and the pulse

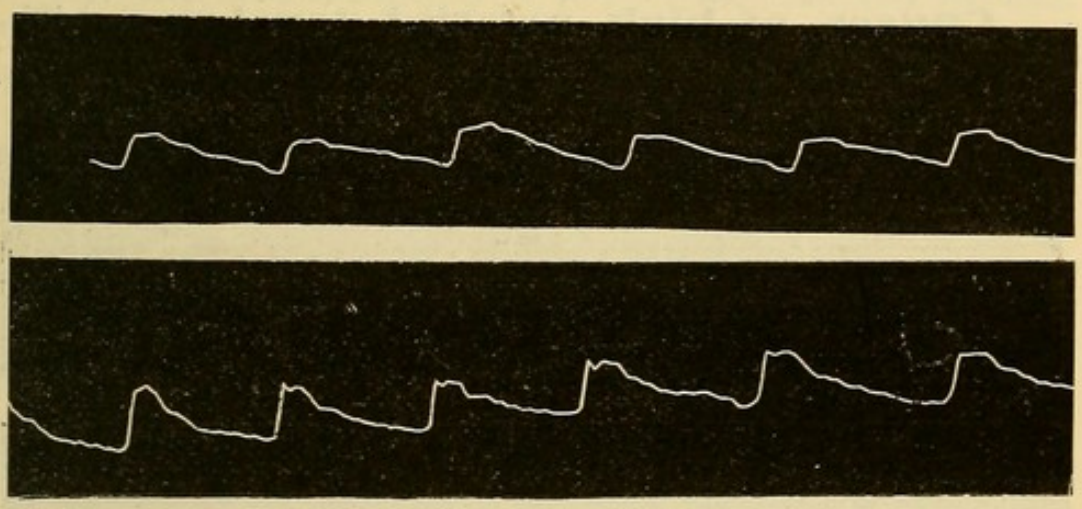


Fig. 41.—High Tension Pulses.

therefore small, the pulse under these conditions often being supposed to be weak from the inconspicuous character of the pulsatile movement.

Virtual tension.—An important deviation from the form of pulse just described may, however, be met with when the essential cause of high arterial tension—obstruction in the peripheral circulation—exists. This is usually at a late stage in the history of the case, when the arteries are worn out and dilated by old-standing high pressure of the blood within them ; when, also, the heart has yielded to the resistance by which it has been opposed and dilatation of the left ventricle has taken place. The artery then is large and full between the beats, but when moderate pressure is employed it allows itself to be flattened, and the pulse is sudden in onset and as sudden in its ending, the pressure in the vessel is abruptly raised, remains high for a brief period, and then falls abruptly. The ventricle, in fact, cannot go through with its systole in the face of the resistance in front. This may be called the pulse of *virtual* as distinguished from actual tension ; the peripheral

condition for the production of tension exists, but the sustained central force required for "actual" tension is wanting. (Fig. 42.)

Causes of high arterial tension. — The causes of high arterial tension are many and various.

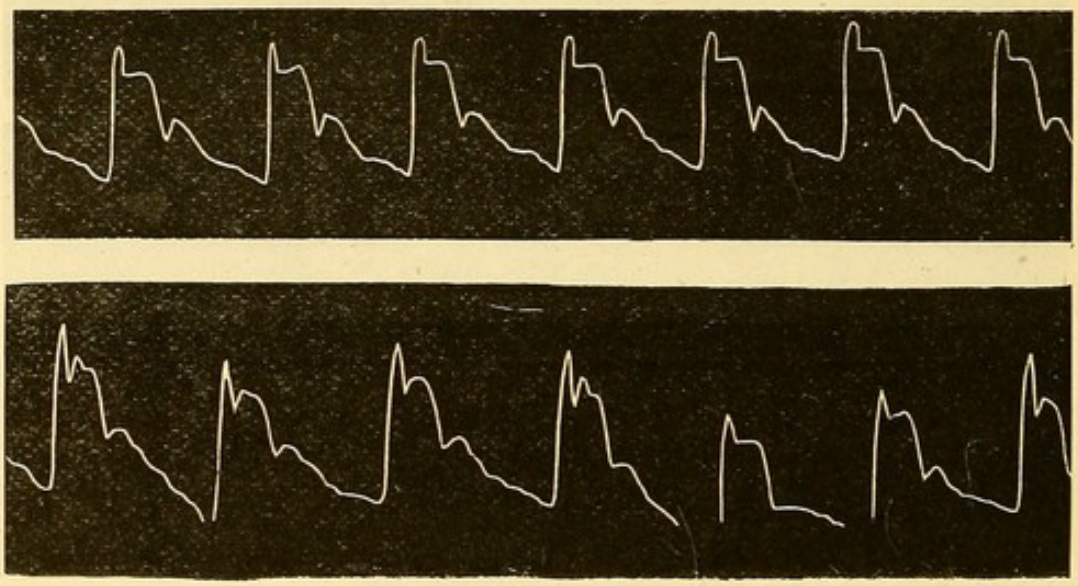


Fig. 42.—Pulses of Virtual Tension.

1. *Increase in the volume of blood.*—It is easily understood that when the amount of blood in the body is increased, the vessels generally must be fuller and the fluid pressure in them greater. There is a temporary increase in the volume of the blood after each meal, as the products of digestion are absorbed, but the effect of this on the tension in the arteries is neutralised by the relaxation of their muscular walls, and the freedom of the outflow through the capillaries which attends digestion. The tension of the pulse is thus usually lowered after meals. After a very heavy meal, however, the amount of matter taken up into the blood may be so large as to give rise to a general vascular turgescence, and, the predisposition existing, this may determine the occurrence of cerebral hæmorrhage. Apoplectic attacks do not come on after a copious repast so frequently as is popularly supposed,

but instances are met with, especially where stimulants have been taken freely.

A constant repletion of the entire vascular system is present in the condition called plethora. This, of itself, would give rise to high pressure in the arteries; but in plethora elimination is rarely efficient, and the blood is charged with waste products, which provoke resistance in the capillaries, so that an additional cause of arterial tension is present.

Another instance in which an increase in the volume of the blood contributes to the production of high tension is afforded by the early stage of acute desquamative nephritis, where the retained urinary water at the same time dilutes the blood and augments its volume.

2. *Frequent and powerful action of the heart.*—Whenever, from any cause, the heart begins to beat more rapidly and to pump more blood into the aorta in a given time, the outflow by the capillaries remaining the same, there must be a rise of pressure in the arteries. This occurs in excitement and on exertion; but high tension produced in this way is usually fugitive. The first effect is to transfer blood from the veins and abdominal venous reservoirs to the arterial system; but when the surplus in the veins is exhausted, the ventricle does not receive a full supply, and, although it may act frequently and vigorously, the total amount of blood propelled into the arterial system will not be increased. The increase of pressure may, however, last long enough to do mischief.

3. *Arteriole contraction.*—The most simple example of this is the increased arterial tension which is produced by external cold. This is the result of the physiological contraction of the cutaneous vessels, which excludes the blood from the surface, and so prevents undue loss of heat. But, although a normal process, the increase of intra-arterial pressure is often

the exciting cause of cerebral hæmorrhage when the predisposition exists in atheroma of the cerebral arteries. Every winter the first spell of cold weather is attended with a number of cases of apoplexy, as is each succeeding one.

The cramp which proves fatal to swimmers is almost certainly a general arterial spasm provoked by the chill of immersion, the resistance to the circulation being aided by the pressure of the water, while the heart is usually also weakened by exertion. It is not only after prolonged exertion in swimming that the so-called cramp occurs. I have known one instance in which a vigorous young man plunged into a pool, and was seen by his companion to go straight to the bottom and lie there dead.

One of the factors in the causation of angina pectoris, or, at any rate, in one form of this affection, appears to be general arteriole spasm. Why such spasm should be associated with a fatty condition of the heart-walls and atheroma of the coronary arteries —the most common morbid change found after death from angina—is not clear; and it may be that a merely physiological contraction of the arteries, such as is constantly taking place in health without appreciable effect, is sufficient to arrest the systole of the ventricle. This will be effected all the more readily when the aorta has lost its elasticity from disease, so that it cannot dilate to receive the blood. The term "angina vaso-motoria" has been employed to designate angina in which the arterial spasm is a prominent feature. It is, however, possible that in some cases of angina the initial phenomenon is failure of the cardiac systole, and that the small size of the arteries is the result of their contracting down upon their diminishing contents when blood is no longer propelled into the aorta, as they do after death for want of distending force within them.

It would be interesting to know whether the pseudo-angina of young adults is entirely of vaso-motor origin.

The prominent phenomenon of rigor is general arterial spasm; and in the cold stage of malarial fevers this may be carried to such a degree as to bring the heart to a standstill by the resistance produced. It is from this cause that the cold stage of malignant intermittent or remittent fevers is attended with danger of fatal syncope or serious nervous complications.

Cases are sometimes met with, independently of malarial poisoning, in which general arterial spasm becomes a source of danger, as in some forms of angina pectoris; and I may relate an illustration of another kind. A lady still living, aged seventy-five or upwards, the subject of chronic arterial tension, caught a chill, and the arteries, which were usually large, were found to be tightened up, small, and incompressible, while there were severe occipital headache and a sense of oppression in the chest. The urine, previously normal, became, temporarily, extremely copious and pale, had a specific gravity of only 1·006 or 1·008, and contained a small proportion of albumen. On another occasion a similar attack was accompanied by slight left hemiplegia, with marked impairment of articulation; and, in my absence, two distinguished colleagues, not aware of the previous history, came to a diagnosis of advanced Bright's Disease, and gave a most unfavourable prognosis. On both occasions a dose of calomel put an end to the serious symptoms almost at once—the hemiplegia, of course, excepted, which only disappeared very gradually.

In hysteria, arteriole spasm is a highly characteristic feature, especially during a hysterical fit. The copious limpid, watery urine is, no doubt, an effect of the high arterial tension so induced. Nervous

excitement of certain kinds is attended with contraction of the arteries; and this is the explanation of the diuresis of nervousness.

In migraine, again, there is general arteriole spasm, and the attack itself has been attributed to contraction of the cerebral arteries.

The early stage of meningitis is attended with arterial tension from contraction of the arterial walls, and in some cases of cerebral tumour there is persistent contraction of the arteries with reduplication of the second sound of the heart.

In a case of severe neuralgic pain along the sciatic, which was ultimately found to be due to a malignant growth in the spinal canal, which I once saw, there was unilateral arterial spasm, giving rise to a perceptible difference in the pulse of the two sides, which resisted the influence of nitrite of amyl. One side only of the face flushed.

4. *Resistance in the capillaries.*—This is the most frequent and important of the causes of arterial tension. The grounds on which the resistance is localised in the capillary network, as distinguished from the arterioles, have already been discussed, and the cause only of this resistance has now to be considered. This can scarcely be other than some substance present in the blood which acts directly upon the capillary walls, either provoking contraction or affecting the cohesion of the blood and the capillary membrane. The former is the more probable mode in which the obstruction is produced, but in inflammation there is marked cohesiveness between the capillary walls and the blood elements. That certain substances, present in the blood in very minute proportions, give rise to obstruction in the capillaries is clearly demonstrated by Drs. Ringer's and Sainsbury's experiments with digitalin, ergotin, etc., which show also that the effect is independent of reflex nervous influence,

since it occurs when all the nerves are divided. The special material which plays this part is almost certainly nitrogenised waste which has not undergone the complete oxidation necessary for elimination. This is difficult to prove; but carbonic acid retained in the blood gives rise to resistance in the systemic capillaries and to extremely high blood-pressure. The first effect of suffocation is not obstruction to the transmission of blood through the lungs, but resistance to the passage of the blood charged with carbonic acid through the capillaries. And it is not unreasonable to conclude that retained excretory matters of another kind will have a similar effect. The diseases, gout and renal disease, in which high arterial tension is most marked, are exactly those in which there is the greatest certainty of the existence in the blood of the products of imperfect metabolism. The effects of treatment, again, almost amount to a demonstration, eliminants being the great means of removing the resistance in the capillaries and lowering the tension.

High arterial tension produced by arterio-capillary obstruction occurs under the following conditions:

1. *Age.*—There is a tendency to the development of resistance in the peripheral circulation and of arterial tension with advancing years; it is one of the ways in which the tissues show that they are growing old. This is most marked when high tension exists from other causes.

2. *Heredity.*—Inherited tendency must in many cases be assumed as the only explanation of undue tension in the arterial system. I have frequently found it in young students and school-boys, and sometimes in young children, quite independent of gout or gouty family history, and not traceable to habits or mode of life. No condition, indeed, runs more strongly in families than high arterial tension, and it is the explanation of a family liability to apoplexy and

paralysis, or to death from heart disease. It is not an uncommon thing for all the males of a family to die off about or before the age of sixty or sixty-five from consequences of arterial tension of one kind or another, while the female members, although presenting signs of extreme pressure within the arterial system, survive to a greater age—women not being exposed in the same degree as men to the influences, dietetic and other, which intensify arterial tension and precipitate its fatal effect. In my own experience I have had a school-boy suffering from headache and loss of the power of application to work, with a high-tension pulse, whose brother had hæmorrhoids at a very early age, and his father dilated heart and degenerate tortuous arteries; while uncles had died of apoplexy and heart disease. There was no gout, as such, in the family; the boys were active and athletic, the adults strictly temperate.

3. *Renal disease.*—Renal disease of whatever kind, except acute suppurative pyelitis and nephritis and perhaps tuberculosis and amyloid degeneration, is attended with high arterial tension, due to the imperfect elimination of urinary constituents. So characteristic of disease of the kidney is the pulse of high tension, that it has been named the "renal pulse;" but the term is extremely objectionable, for, although such a pulse is often at once suggestive of disease of the kidneys, and may facilitate the diagnosis, it is very common when there is no renal change; and, on the other hand, it may be absent, temporarily or permanently, when advanced disease of the kidneys exists. If tension be permanently wanting, however, when the kidneys are diseased, it may be a prognostic sign of the worst augury.

4. *Gout.*—Gout, again, is so constantly accompanied by high pulse tension that the term "gouty pulse" has passed into currency. It is, of course, open to the

same objections as the name "renal pulse." Arterial tension is present in both acute and in chronic gout; and the name "suppressed gout," conveniently vague and open as it is to abuse, might perhaps serve some useful purpose if it were employed to designate such states of impaired health in middle and advanced life as are characterised by the presence of unduly high arterial tension.

The class would correspond very closely with the conditions described by the late Dr. Murchison in his work on functional derangement of the liver, the symptoms being attributed to lithæmia. In gout the form of nitrogenised waste is uric acid; in some of the states comprehended under the head of suppressed gout the oxidation of nitrogenised matter has probably stopped short of the stage at which uric acid is formed, and the compounds are even more injurious in their effects on the system.

5. *Diabetes.*—In connection with gout may be mentioned diabetes, one form of which is accompanied by high pulse tension, and is closely associated with gout.

It is well known that diabetes is in early life a deadly disease very rarely yielding to treatment, and is from its onset attended with progressive loss of strength and flesh, while after middle age, especially in stout people, it may exist for years without making any obvious impression on the general health and vigour. In a patient recently under observation, sugar had been known to be present in the urine for twenty years. The sugar, again, may, under treatment, completely and permanently disappear from the urine. So marked is the difference between the symptoms and effects associated with the presence of sugar in the urine before and after middle age, that many observers refuse to the glycosuria of elderly persons the name diabetes. Sugar, however, may

be excreted in large amount and without intermission, and the condition is quite distinct from the temporary glycosuria of over-feeding, or indigestion, or of nervous attacks. For the present, therefore, it will be sufficient if we speak of two forms of diabetes.

Between these two forms there is, speaking generally, a remarkable difference as regards the pulse. In the diabetes of the young the pulse is small and of low tension; in the diabetes of advanced life it is large and of high tension.

As has already been said, late diabetes is closely associated with the gouty diathesis. It may supervene in an individual who has had repeated attacks of gout, or who suffers from chronic gout, and when this takes place the patient often feels relieved, and has not only less gouty pain, but is less depressed, and has less indigestion and flatulence. There may, however, have been no overt gout. The state of the arteries, whether gout has been manifest or not, gives evidence of protracted arterial tension; they are large, full between the beats, not very easily compressible, and their walls are thick and dense. Until the heart has begun to suffer from the effects of the peripheral resistance, the pulse wave is forcible and sustained. As the heart becomes worn out the beat becomes sudden and short. In this condition of the circulation it appears to me to be dangerous to insist on a strictly nitrogenous diet, and I have not found it necessary. Taking the high tension as an indication for treatment, a pill containing calomel or other form of mercury with one of the vegetable aperients, is given every night or every second night for a time, and salicylate and carbonate of soda with quinine or gentian three times a day, or the alkaline carbonates without the salicylates will often be sufficient. Usually the sugar disappears from the urine, sometimes very quickly, and the urine may be kept free from it almost

indefinitely by measures which prevent the recurrence of high tension. When the heart is worn out and the pulse tension is virtual and not real, the good effects are more difficult of realisation. These high tension diabetics are the cases which are cured at Carlsbad, Marienbad, and Vichy. It is interesting to note that the sugar often entirely disappears from the urine during an acute febrile attack of any kind, such as pneumonia or bronchitis, to which patients of this class are very liable.

In the more serious diabetes of the young, the pulse, as has been said, is small and short—that is, the tension is low. Exceptions, however, occur in which the artery is full between the beats, and can be rolled under the fingers, but no case has come under my observation in which the artery has been large as well as tense. When tension has been at all noteworthy, the loss of strength has, in my experience, been much less marked, and the disease has remained apparently stationary for a long time. Conversely, diabetes may be met with in elderly people associated with low arterial tension, and when this is the case the prognosis is serious.

6. *Lead-poisoning.*—Lead-poisoning is another cause of high arterial tension, and it is noteworthy that it frequently gives rise to gout and kidney disease, the conditions already spoken of attended with excessive intra-arterial pressure. Probably the formation of compounds of organic matter with lead salts, albuminates of lead too stable to undergo readily dissociation and oxidation, is the cause of accumulation of imperfectly oxidised products in the blood.

7. *Pregnancy.*—Pregnancy is invariably accompanied by increase of tension in the arteries. Whether this arises from a general augmentation of the volume of the blood, or from the presence in the blood of effete matters derived from the fœtus, is perhaps not

altogether settled. Drs. Galabin and Mahomed have carefully investigated the rise of the pulse-tension in pregnancy. It is worthy of note that Bright's Disease may be established by pregnancy as well as by lead-poisoning.

8. *Anæmia.*—It is not easy to understand how anæmia can give rise to high tension in the pulse. One would have expected the watery blood to pass readily through the capillaries and the vis a tergo supplied by the heart to be deficient; but it is a matter of daily observation that the artery is full between the beats, and that the pulse, if more abrupt than in renal disease, is long. The occurrence of dilatation of the left ventricle and mitral regurgitation, which is very common as an effect of anæmia, is at once understood when the resistance in the peripheral circulation is taken into account; it is not merely the innutrition of the walls of the heart, due to anæmia, which causes them to give way, but the increased work thrown upon the left ventricle by this resistance. It has been conjectured that the defective oxygen-carrying power of the corpuscles may cause oxidation to be imperfect, and so lead to the formation of substances which are not readily eliminated, and which provoke resistance in the arterioles and capillaries; but the instability of the tension is suggestive of the intervention of nervous influences, and there are other peculiarities which lend support to the hypothesis that the arterial contraction and powerful heart-action may be vaso-motor phenomena.

While high tension is the rule among anæmic patients seen in the consulting-room and in the London practice generally, exceptions are met with, and in agricultural districts, where little animal food is consumed, low vascular tension is very common.

It has been an object of attention with me to make

out whether any constant difference of another kind attends this difference in the state of the circulation. In particular it seemed probable that there might be some influence on the production of hæmic murmurs which might throw light on their causation, especially from the point of view of Marey's theory, which refers all cardiac murmurs to low pressure beyond the point at which sonorous vibration is excited. Up to the present, however, I have been unable to recognise any distinction between anæmia with high tension and anæmia with low tension, except the difference in the circulatory conditions.

9. *Emphysema.* — In cases of emphysema and chronic bronchitis, and sometimes even in phthisis, the systemic arteries present the signs of increased tension; in emphysema they are specially marked. This may be attributed to general fibrotic change in the tissues as well as in the lungs; but this is not the whole explanation: imperfect aëration of the blood has a share in provoking the resistance, as is shown by its varying degree in the early stages of the affection of the lungs. Mitral stenosis may here be mentioned as associated with arterial tension, without discussing the relation between the two. With scarcely an exception the radial artery is full between the beats in mitral stenosis.

The etiology of high arterial tension will require very few words. The remote causes are just those which conduce to the imperfect oxidation and elimination of nitrogenised waste.

1. *Food.*—A high proportion of animal food, and especially of the butcher's meat, stands first on the list. There is no such great difference between fowls of all kinds or game and red meat as is popularly supposed, but perhaps meats contain more extractive matters. Soups, beef-tea, and animal juices, meat extracts, and the like, however valuable, contain a maximum of

potential waste in comparison with matters available for tissue nutrition.

I have been greatly struck with the frequency and degree of high arterial tension met with in Englishmen returning from India and other hot climates, but especially from the West Indies. My preconceived idea was that the external heat and free perspiration would produce general vascular relaxation; but observation has shown the exact contrary of this to be the usual result. The explanation, apparently, is that the Englishman carries his meat-eating habits with him to hot climates, and there being here comparatively little need for combustion in order to maintain the temperature of the body, the nitrogenised food is imperfectly burnt off and eliminated.

2. *Alcoholic drinks.*—Any form of alcoholic fluid in excess, spirits, wine, or beer, will interfere with the normal metabolic processes and lead to the retention of impurities within the system. The stronger wines such as port and sherry, in the preparation of which the fermentation has been prematurely arrested by the addition of spirits, and beer, which contains glucoid matters, have this effect even in moderate quantities.

3. *Sedentary habits.*—An inadequate amount of exercise in the open air, especially when a great part of the day is passed in offices lighted by gas and imperfectly ventilated, intensifies greatly the effects of excessive food and alcoholic drink, and will of itself conduce to imperfect oxidation. A walk through the streets to and from business is a very inadequate means of counteracting the effects of confinement in an impure atmosphere all day, and the same may be said of occasional violent exercise, which, indeed, may indirectly promote the accumulation of nitrogenised matter in the system by creating an appetite.

4. *Constipation.*—This is a very important cause

of high arterial tension, and it acts in at least two ways. The undue retention of fæcal matters in the large intestine leads to resorption of the fluid parts, and these constitute impurities in the blood likely of themselves to provoke resistance in the capillaries and calculated also to interfere with digestion and with metabolism in the liver and tissues generally, which would add to the impurities. The foul tongue and offensive breath and sallow complexion attending habitual constipation are sufficiently suggestive. But constipation has a direct influence on arterial tension, probably through the effect on the abdominal circulation, and on the amount of blood in the large abdominal veins. This is patent to everyday observation if the pulse is examined before and after defæcation. Many weakly persons are greatly depressed after even an ordinary evacuation, and come to dread it, and syncope is not uncommon after an unusually large motion. It is easily understood, then, how constipation becomes a source of danger and injury. It promotes high arterial tension both directly and indirectly, and a further danger arises from straining at stool, which not unfrequently determines the rupture of a cerebral vessel, or breaks down the modus vivendi of a weak heart. Constipation is the special danger of old age, and the indirect cause of death to numberless old people. It is not inconsistent with a daily action of the bowels, the relief being incomplete; so that gradual accumulation of fæcal matters takes place, and so-called diarrhœa in old people is very often a symptom of such accumulation. The presence of scybala gives rise to frequent calls to the stool, and all that escapes is a little liquid consisting of secretion from the mucous membrane of the rectum stained by débris washed from the surface of the hard masses.

Pathological effects of high tension.—The pathological changes resulting from unduly high

tension must now be traced in the arterioles, arteries, and heart.

Hypertrophy of muscular coat of arteries and fibroid change.—The capillaries being the seat of the obstruction, the minute arterioles, either because they contract under the same influence as the capillaries, or contracting to resist the distending influence of the high blood-pressure within them, are in a constant state of excessive functional muscular activity, and the result is hypertrophy of their muscular tunic. In course of time change of a fibroid character supervenes. The same process goes on in arteries of a larger size. Whenever the middle coat contains any considerable proportion of muscular fibres, these will undergo hypertrophy in the first place and fibrosis later.

Rupture of vessels.—The most common and serious effect on arterioles of the smallest size is cerebral hæmorrhage from rupture of terminal branches of cerebral arteries. This takes place almost invariably in vessels distributed to the white substance, which are extremely few in comparison to those of the grey material; to speak more exactly, the vessels ruptured are usually small arteries traversing white substance on their way to the central grey masses, and miliary aneurisms have been described by Charcot and Bouchard as preceding the rupture.

Rupture of minute vessels may occur elsewhere than in the brain. Retinal hæmorrhages, for example, are not uncommon, attended with more or less injury to vision; sometimes with complete loss of sight. Epistaxis, again, is frequent and is usually salutary, giving the relief which venesection would afford. In rare instances there is copious hæmorrhage from the lungs or bowels.

In the arterioles generally the gradual substitution of fibroid for muscular tissue will prevent these vessels

from taking the part in the regulation of the supply of blood to different organs and structures which belongs to them. The consequences of this are not immediately conspicuous, especially in apparent health, but the loss of this function must impair very seriously the adjustment of the circulation to varying conditions, and must exert an unfavourable influence on the course of disease, and on the processes which lead to recovery. Any marked degeneration in the vessels is recognised as an unfavourable element in prognosis either in acute or chronic disease, and it has been well said that a man's age is that of his arteries.

Atheroma and degeneration of small arteries.—But fibroid change is not the only form of arterial degeneration to which high tension contributes. Pressure within the vessels takes effect on the vasa vasorum in their walls, and tends to interfere with the flow of blood along them, and thus to cut off the supply of nutriment from the coats of the arteries themselves. This will give rise to a change of a strictly degenerative character. Fatty and cretaceous deposits are formed in their walls, and the vessels accessible to the fingers in advanced cases become converted into inelastic tubes, presenting bulgings and tortuosities and often calcareous indurations.

Atheroma of aorta and its consequences.—In the large arteries high blood-pressure is one of the chief causes of atheroma. The constant stress on their walls sets up a chronic inflammatory process, attended with exudation into or beneath the intima, which gives rise to the opaque raised patches of the early stage of atheroma. Degenerative changes follow; the lining membrane of the vessel gives way, and the exuded matter is gradually carried off by the blood; or a calcareous plate is formed in the patch, or other changes take place. At the same time the nutrition of the middle coat suffers from the stretching and

pressure to which it is subjected, and the general result is that the elasticity of the great vessels is lost, the inner surface is uneven and opaque, the walls are thinned, and general or, more rarely, aneurismal dilatation is produced.

The loss of elasticity in the aorta necessarily abolishes the characteristic effect of this elasticity on the pulse wave. In proportion as it becomes a rigid, undistensible tube it ceases to convert the intermittent propulsion of blood by the heart into a continuous stream, and the pulse becomes sudden in its

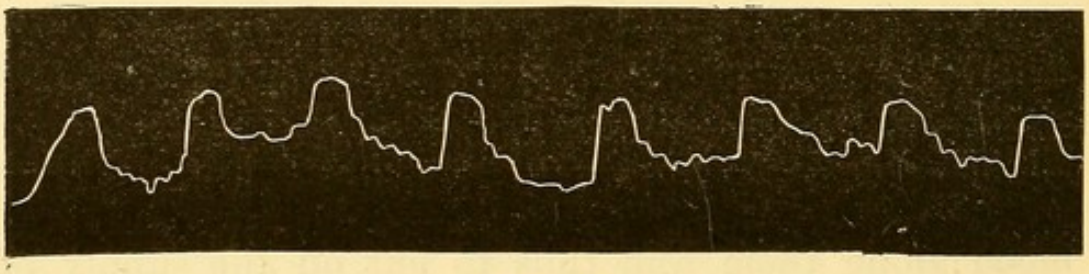


Fig. 43.

onset, and full, the trace resembling more or less that of the left ventricle. This is the well-known senile pulse. (Fig. 43.)

A secondary result of degeneration in the aorta not uncommonly met with is an extension of the dilatation to the ostium, which becomes so stretched that the valves are not large enough to meet and close it. In this way arises one form of aortic incompetence or regurgitation, which comes on for the most part at or after middle age, and is distinguished from regurgitation due to actual valvular disease by the imperfect development of the collapsing pulse and of the carotid delay, and by the persistence of the accentuated aortic second sound, which had preceded the appearance of the murmur. Any hypertrophy of the heart which may exist will have been produced by the protracted arterial tension, and not by the regurgitation, which supervenes at a time of life when the heart is no longer

capable of compensatory increase of muscular fibres and contractile powers.

Another secondary effect of aortic atheroma is narrowing of the orifices of the coronary arteries. This, with extension of atheromatous disease into these arteries from the aorta, is the most common cause of fatty degeneration of the heart, which must thus be set down as one of the consequences of high arterial tension.

Disease of valves of heart.—Valvular disease, properly speaking, is also set up by high arterial tension. The greater the difficulty of driving the blood through the arterioles and capillaries, the greater the pressure in the arterial system and the greater the strain upon the aortic valves in sustaining the column of blood in the aorta. This persistent strain gives rise to chronic valvulitis, which results in thickening and contraction of the cusps with incompetence or stenosis. A systolic murmur is frequently caused by roughening and rigidity or irregularity of the valves without actual obstruction, and the loudest cardiac murmurs ever heard are those so produced. As there is no interference with the mechanism of the heart, such murmurs have no importance, except that they show that changes have set in of a progressive character, which, moreover, may possibly implicate the orifices of the coronary arteries.

The mitral valve suffers in like manner, for, though it has not to sustain the column of blood in the aorta during the diastole, it has to act as the fulcrum during systole, and the greater the pressure in the arterial system the greater the strain upon the mitral valve, while the contents of the ventricle are propelled into the aorta, overcoming this resistance. The consequence of mitral valvulitis thus induced is usually insufficiency—never, so far as my experience goes, stenosis, except, perhaps, to a slight degree, when

calcareous deposit has rendered the valves rigid and unyielding.

Regurgitation through the mitral orifice may also result from dilatation of the ventricle, which may implicate the auriculo-ventricular opening, and make it so large that the flaps fail to occlude it.

Hypertrophy of heart-walls.—On the heart-walls the first and most constant effect is the production of hypertrophy. When the blood no longer passes with normal freedom through the arterioles and capillaries, increased propulsive force is required of the ventricle, and this results in a true increase of the muscular fibres in its walls. Some fibroid tissue is associated with this, and, sooner or later, as the vigour of the nutritive processes declines with advancing years, fibroid or fatty degeneration affects the enlarged heart, and there is a gradual diminution in its efficiency, with or without dilatation.

Dilatation of the heart.—Dilatation is another common result, either preceding hypertrophy, or associated with it from the beginning, or supervening at a later period. It is usually gradual in its development, but acute dilatation of the heart is a more common occurrence than is generally supposed, and, when it is induced by effort, antecedent high arterial tension is, according to my experience, a constant predisposing factor. The left ventricle is unable to overcome the obstruction in the peripheral circulation, and gives way under the strain.

The effects on the heart and vascular system are direct and easily understood. Other conditions which in my experience have usually been associated with high arterial tension, and which I have therefore come to regard as consequences, may be mentioned, although the mode of causation is not clear.

Glaucoma.—One of these is glaucoma, the characteristic feature of which is intra-ocular tension, under

which the globe of the eye becomes bullet-hard and the optic disc cupped. The circulatory conditions in the eye are peculiar. The retinal artery and vein occupy the axis of the optic nerve, but the arteries which supply the vascular tunic and structures—the choroid, the ciliary processes and iris—and the veins which carry off the blood perforate the sclerotic at various points, and there is a remarkable convergence of the venous twigs in a whorl to the emissory veins, which is suggestive of an arrangement for maintaining within the eyeball the tension which enables it to keep its globular form by obstruction in some degree to the outflow of blood. Such an effect is indeed mechanically inevitable; and it is clear that undue internal pressure compressing and flattening, so to speak, the choroid against the sclerotic would have the effect of greatly obstructing the current in the venous whorls, and the exit by the axial veins. The penetration of blood along the various ciliary arteries will not be obstructed in the same degree: high arterial tension, then, must tend to raise the pressure in the vitreous chamber, and may carry it to a point at which it effectually obstructs the filtration angle between the iris and cornea by carrying forwards the lens and ciliary processes, thus realising the conditions which Mr. Priestley Smith has shown in his lucid and instructive lectures before the College of Surgeons to be the immediate cause of glaucoma.

Cheyne-Stokes' respiration.—The remarkable modification of the respiratory rhythm, first noticed and recorded by the two eminent Dublin physicians, whose names Trousseau linked to give a designation to the phenomenon, has, in my experience, been so constantly associated with high arterial tension, that I feel justified in looking upon it as an effect of this condition of the circulation, not indeed, perhaps, simple and direct, but in the sense that high pressure

in the arterial system is, if not a necessary, yet the most constant recognisable factor.

By Cheyne-Stokes' breathing is meant a cycle repeated with remarkable regularity, consisting of an absolute pause and cessation of the respiratory movements, averaging, according to my observation, about twenty seconds in duration, followed by a resumption of breathing at first so slight as scarcely to be perceptible, but gradually increasing in depth till the inspiration and expiration far exceed the normal, after which is a diminution, equally gradual with the rise, down to an absolute cessation. The number of respirations is usually twenty to thirty, and the time occupied by an entire cycle sixty to eighty seconds, during forty to sixty of which there is breathing, and for twenty a pause.

Cheyne-Stokes' respiration proper must not be confounded with the irregular suspension of breathing, seen in the later stages of meningitis and other affections of the brain, in which there is neither the uniformity of the cycle nor the gradual rise and fall, but sudden or gradual arrest, and sudden return of the respiratory movements. This may be called cerebral Cheyne-Stokes' breathing, but only for the purpose of marking its total difference from the true Cheyne-Stokes' rhythm.

On the other hand, in the sleep of infants, something like Cheyne-Stokes' breathing is often observable, and in the snore of an old man there is sometimes a gradual diminution of intensity to a full stop and pause of surprising duration, but the breathing is resumed with a snort, and is compensatingly vigorous for a time; this sort of cycle, however, recurs again and again.

A fact of great interest is that there is no evidence of imperfect aëration of the blood, such as lividity of the lips, and, indeed, the respiratory movements

are about equal to normal, though distributed in cycles.

Usually, again, the heart takes no notice of the alternations between breathing and pause, but sometimes the beats slacken in frequency and force towards the end of the pause, and the respiratory curve will be distinct in the sphygmographic trace at the maximum of the period of breathing.

The patient may be conscious or unconscious, waking or sleeping. When sleeping or unconscious there are often twitchings of the face and jerking of the limbs towards the end of the pause, as a sort of preliminary to the resumption of breathing. Not unfrequently the Cheyne-Stokes' rhythm will be present during sleep and absent in the waking state. I have been told by an intelligent nurse, in one case in which it was marked and constant while the patient was awake, that the breathing was regular and natural during sleep. When the patient is conscious, there is no respiratory distress, no complaint of shortness of breath. He will be unable to talk during the height of the breathing period, but he employs the interval for the purpose, and will thus seem to talk himself out of breath.

The grounds upon which I have been led to refer Cheyne-Stokes' breathing to high arterial tension are as follows :—

In the first place, Cheyne-Stokes' breathing is most frequently met with in association with uræmic symptoms, especially uræmic coma, and it is in kidney disease that the injurious effects of high tension are most common and most marked. The occurrence of uræmic phenomena and Cheyne-Stokes' respiration under like conditions is a reason for attributing both to the same cause.

It is common, again, when dilatation of the left ventricle is giving rise to the symptoms which lead

up to a fatal termination, and it need scarcely be repeated that this condition of the heart is for the most part due to resistance in the peripheral circulation.

Dilatation of the aorta has been assigned as the special cause of Cheyne-Stokes' respiration, and, in effect, it is a very common antecedent. But were it invariably present, it is itself, like dilatation of the left ventricle, an effect of protracted high pressure in the arterial system, and is open to the representation of being simply concurrent with the respiratory cycle, and not causative of it.

When the aorta or ventricle is dilated, Cheyne-Stokes' respiration is often present during sleep long before other symptoms become urgent, or when the only other symptoms are anginoid attacks and breathlessness on exertion.

But it is not the case that the aorta is always dilated. The fact, however, that such a view has been propounded with any degree of evidence in support corroborates my experience of the association of the Cheyne-Stokes' phenomenon and high tension.

When the aorta is dilated the ostium may partake in the overstretching so that the valves fail to close it, and regurgitation may take place. I cannot find a note of the occurrence of Cheyne-Stokes' breathing in either aortic regurgitation or aortic obstruction due to primary disease of the valves, but I have an impression that I have met with it in aortic obstruction.

When high tension is habitually present the supervention of Cheyne-Stokes' breathing may be determined by some complication. In one very interesting case it followed an attack of left hemiplegia due to cerebral hæmorrhage. This was one of the cases in which the patient employed the pause for the purpose of talking, and had to stop while the breathing was going on. In eating, also, he took advantage of the

pause to masticate and swallow. He was unconscious of any distress or even inconvenience, and did not notice that he had to wait till the deep breathing had subsided to speak or swallow. He lived for many years, remaining badly paralysed. The breathing very gradually returned to its normal regularity, the Cheyne-Stokes' cycle lasting altogether several weeks. In two other instances the exciting cause was constipation. One patient was a vigorous old gentleman over eighty years of age, who had habitually a large tense pulse and thickened arteries and powerful heart. When I was called to see him he had slight pyrexia, with a foul tongue and loss of appetite, accompanied by great prostration, confusion of the mental faculties, and a degree of stupor approaching unconsciousness. The Cheyne-Stokes' breathing was perfectly characteristic. Under the use of aperients, which dislodged an extraordinary amount of fæcal matters, all the symptoms disappeared.

In another case the cause of the obstruction was malignant disease of the sigmoid flexure ; the patient was all but unconscious, and was supposed to be dying, but the obstruction was overcome for the time being, and he lived for many months.

The significance of the association of Cheyne-Stokes' respiration with the conditions enumerated is accentuated by its absence when there is mental confusion and loss of consciousness or respiratory distress from other causes not attended with high arterial tension. I have only once seen even an approach to the characteristic cycle in fever, and this was in a case of enteric fever of altogether exceptional character attended with extraordinary excitement from the first and a high tension pulse throughout, notwithstanding temperature of 103° F. For a short time there was the characteristic rise and fall, but the pause was very brief. In the case related on pages

174-5, in which Cheyne-Stokes' breathing was suspended on the supervention of pulmonary apoplexy, it cannot be said with confidence whether it was the respiratory embarrassment or the pyrexia which produced the effect.

Again, we do not see Cheyne-Stokes' breathing in mitral disease.

Two observations which appear to me to have crucial importance with respect to any hypothesis as to the causation of the Cheyne-Stokes' rhythm have come in my way. One was in a case of severe cerebral hæmorrhage with left hemiplegia. When I was called to the patient, some hours after the attack, I found him lying on his back, with flushed, almost purple, face, full and bounding pulse, and stertorous breathing. The congested countenance was obviously due to imperfect aëration of the blood, and this, again, to the falling back of the tongue and to paralysis of the soft palate and parts about the pharynx, which interfered with respiration and gave rise to the loud stertor. The stertor and obstruction to breathing were instantly relieved by turning the patient well on to the paralysed side, as suggested by Dr. Bowles; the face became natural in appearance, and the pulse quiet. But, as the respiratory distress was removed, Cheyne-Stokes' breathing set in, and continued till the patient's death.

In the other case the patient, who was suffering from atheromatous degeneration of the aorta implicating the valves, exhibited the Cheyne-Stokes' respiratory cycle for some months while still going to the City for a short time daily. I saw him under a variety of circumstances and at different periods of the day, sometimes after he had walked a mile and a half to my consulting-room, sometimes in his own house, and this kind of breathing was always present. After a time thrombosis in the veins of the leg took

place, and while in bed on account of this the breathing was still of the same type. It may be inferred, therefore, that it was constant during the waking hours. A portion of clot became detached, and was carried into the pulmonary artery; the infarct was large, and gave rise to hæmoptysis and to consolidation of a considerable portion of lung attended with pyrexia. As was to be expected, the respirations became more frequent; but they also became perfectly regular, and, during the stress of the disturbance caused by the pulmonary embolism, the Cheyne-Stokes' rhythm entirely ceased. As the disturbance subsided and the acute symptoms disappeared, however, it returned, and was re-established in its typical character. The night nurse, however, stated that the breathing was regular during sleep. Another pulmonary embolism occurred, and again the Cheyne-Stokes' cycle was exchanged for accelerated regular respiration.

These observations appear to me to be adverse to any hypothesis with regard to the respiratory centre, whether of exalted or diminished sensibility, and to point to a loss of the normal adjustment between the systemic and pulmonary circulations.

Prognosis. — The prognostic significance of Cheyne-Stokes' breathing is always grave, since it marks a serious want of accord between the systemic and pulmonary circulation, and the cause of such interference must be attended with danger. It must not, however, be looked upon as of necessarily fatal import. In many cases in which it is associated with uræmia, there is not only recovery from the uræmic condition, but cure of the kidney disease, or, at any rate, such alleviation as allows the patient to live for years. The same may be said when it is an incident of high arterial tension from other causes than renal disease, if the heart or aorta is not irreparably

damaged. Cases have been mentioned in which the patient recovered from attacks of various kinds, attended with well-marked Cheyne-Stokes' breathing, and lived for some time. The case may also be again referred to of the patient who attended to his business in the City for some time while exhibiting this phenomenon, and ultimately died from thrombosis of the femoral veins and pulmonary embolism (*see* pages 174-5). While the immunity from other symptoms of a serious character was more complete than in any other case, he is not the only patient who has come to my consulting-room breathing in the same way.

Symptoms attending high arterial tension.—Numerous symptoms are associated with high arterial tension, but it is not easy to say with regard to all of them whether they are due to the state of the circulation, or to the impurity present in the blood, which is the cause of the resistance in the arterio-capillary network and high pressure in the arteries. Doubtless the primary cause is the blood-contamination; and certain substances, the result of incomplete blood and tissue metabolism, or of imperfect digestion and assimilation of food, are capable of giving rise to headache, of depressing the spirits, and clouding the mind. But it seems clear that circulatory conditions are not unfrequently an intermediate cause, and it is certain that the pulse affords a clue to treatment.

Among the more frequent and important of these symptoms are headache, sleeplessness, breathlessness, depression; loss of energy, resolution, memory, and nerve; giddiness, a sense of fulness in the head, pain and oppression in the chest, and neuralgia.

Headache may vary in seat, character, and duration. It may be frontal, occipital, or vertical. It is sometimes a morning headache, which disappears after the bath and breakfast; at others it comes on after mental work or towards the end of the day.

Headache is very common in the subject of high arterial tension; but high tension has no specific form of headache.

Migraine, or sick-headache, again, is, according to my experience, almost always associated with high tension, not only during the attacks, but as an habitual condition; and the liability runs in high-tension families.

Dr. Haig has endeavoured to show that the paroxysms are associated with the elimination of a large amount of uric acid; and in many cases there is habitual precipitation of uric acid in excess of the normal quantity. Sick-headaches, however, often cease as old age comes on, while the liability to the formation of uric acid continues; and I have seen cases in which migraine lapsed with age, when the formation and excretions of uric acid continued to be extremely great.

Neuralgia, not of migraine character, is one of the less common effects of high tension, and it is only mentioned because we should not under ordinary circumstances think of resorting to the treatment suggested. I have, however, seen neuralgia cured by a dose of calomel when all other remedies, including change of air, had failed to give relief. The patients were pale and weakly ladies, and were not suffering from constipation. Calomel, therefore, appeared to be contra-indicated, and was only at length given because of the high arterial tension which had been noted throughout.

Depression of spirits, loss of the power of concentrating the attention, impairment of the memory, painful irresolution, irritability of temper, and loss of nerve, are other symptoms commonly met with in association with high arterial tension.

A member of the profession, aged about 54, who had retired to a country life, came to me complaining

of these symptoms. He was a man of active habits, spending much time in the saddle, temperate in eating and drinking, and had the look of perfect health and vigour; but he wept before me when relating his trouble, and said that when out riding he was sometimes taken with sudden nervousness and timidity, and was compelled to make some excuse for returning home. His tongue was clean, his bowels regular, his urine normal, and his sleep good. Nothing wrong could be discovered about him, except very high arterial tension, and he was gradually restored to cheerfulness and energy as the tension was reduced.

In another case the patient would be seized in the street with sudden faintness and deadly apprehension. The attacks ceased with the disappearance of high tension from the pulse.

Breathlessness on exertion, as severe as in advanced heart disease, may be simply the result of high tension; the resistance in the peripheral vessels may have an effect on the circulation equivalent to that of valvular disease or dilatation of the heart. I have seen several cases in which the patient has been compelled to stop and sit down or support himself by railings, gasping for breath, two or three times in the course of a few hundred yards of level walking at a slow pace, no cause for this being recognisable in the heart, and complete and permanent relief being afforded when extremely high tension was reduced.

In one case, complicated by pulmonary emphysema, there was not only extreme shortness of breath, but lividity of countenance, considerable œdema of the legs, and a large amount of albumen in the urine. Under treatment suggested by the high arterial tension, which was present in a marked degree, the dropsy and albuminuria disappeared at once. This patient was seen three times at intervals of two or

three years with the same train of symptoms. The emphysema no doubt played an important part in the production of the dropsy and albuminuria, but these conditions were relieved too rapidly to be due to it alone.

Sleeplessness, as a result of high arterial tension, will be considered later in a chapter on the pulse and the nervous system, as will also sudden loss of consciousness and convulsions, which may also be due to this condition.

Treatment.—We need occupy ourselves only with the treatment of persistent high tension, the injurious effects of which have just been set forth. The main cause being the presence in the blood of imperfectly oxidised nitrogenised wastes, the object to be aimed at must obviously be to keep the blood free from such impurities.

Exercise and fresh air are of primary importance for this purpose, and a persistent neglect of these essentials to health will defeat any attempt to rectify permanently a tendency to high pressure in the arterial system. From this point of view, a morning gallop is invaluable to men whose occupations are sedentary.

Diet.—One great source of nitrogenised waste is the consumption of an undue amount of nitrogenised food, and in all cases of abnormal tension in the arteries, the amount of highly nitrogenised articles of diet should be limited to a minimum compatible with the health and vigour of the individual. It is usually sufficient if animal food is taken once a day; fowl and game being included under this head as well as beef and mutton and the like.

In the estimation of the public there is a broad line of demarcation between butcher's meat and poultry, and a patient will often suppose that he is limiting his animal food if he takes mutton or beef

in the middle of the day and chicken at breakfast and game at dinner; but chemically there is no great difference. In Germany gout has actually been called the chicken disease, on the absurd hypothesis that since the urinary excretion of birds is uric acid their flesh will yield it when eaten. Fish, milk, and cheese are, of course, also animal food, and it would not be impossible to take an excess of nitrogenised matter in this form. The most important chemical difference between butcher's meats and other animal food consists in the presence of a greater amount of extractive matters so called, to which they owe their flavour, and since soups contain these matters in large proportion, they are an article of diet which must be very sparingly employed when the tendency to high tension exists. A more important difference than the chemical one is the difference of digestibility and assimilability, and this will in one case tell in one direction, in another direction in another case.

In many instances the reduction in the amount of nitrogenised food has little effect on the presence of imperfectly oxidised matters in the blood. The metabolic processes are imperfectly carried out, and be the amount of nitrogenous foods assimilated little or much the oxidation is incomplete.

Alcoholic drinks, if consumed at all, should be taken in very moderate quantity.

It must be borne in mind that in some cases high-pressure in the arteries may be simply one result of a general superabundance of fluid in the blood and tissues, and it may be necessary to reduce the volume of blood by restricting the amount of liquid drunk. Few people are aware how much they drink in the course of the day, and when told to measure it, patients are often astonished at the quantity. When the drink is limited the restriction should apply especially to meal times. Between meals the effect of

drinking water is very different from when it is taken with food.

Water in considerable quantity taken on an empty stomach, flushes the secreting glands and washes out the tissues and is thus a valuable means of eliminating impurities. A good time for taking it is night and morning, and the effects are greater when the water is hot. At night hot water stimulates the stomach to contract, gases are expelled and undigested contents swept on into the duodenum and usually there is a general relaxation of the arterioles; in this way it often conduces to sleep; in the morning it is rapidly absorbed and has a greater effect on the glands and tissues.

It is quite as much by the amount of water taken and the conditions under which it is taken, as by any special constituents of the various mineral waters, that most of the summer and autumn resorts in Germany and France for baths and drinking waters prove beneficial. A course of treatment, however, at one or other of these places is a most valuable resource in reducing high arterial tension. Early rising is enforced, and repeated doses of a weak saline solution taken hot on an empty stomach with intervening walks of ten or fifteen minutes, are admirably calculated to promote blood and tissue metabolism, and to carry off the waste products. The diet, moreover, is regulated, and although some of the restrictions in fashion at different baths are irrational and absurd, the establishment of a sort of superstition on the subject of food and drink has its uses. While it is to the complete reversing of habits and the action of copious draughts of water that the chief benefit is to be attributed, there is no doubt that some of the saline constituents are of greater service in particular cases than others, and judgment is to be exercised in sending patients to different baths.

The lesson obtained from the good effect of baths and watering-places may be utilised for patients who cannot resort to them, and who are the immense majority, by ordering a three weeks' course of Carlsbad salts, or sulphate of soda, or phosphate of soda in small doses, ʒi to ʒii, taken in a copious draught of hot water every morning on rising and while dressing. There is sometimes an advantage in employing a weak infusion of taraxacum as the vehicle, or in adding the succus taraxaci to the saline.

In many cases the Turkish bath is of great service, but if made to take the place of exercise it will in the long run be injurious.

The greatest possible service may be rendered by medicinal remedies at all stages of high tension. An attack of apoplexy may be staved off by a timely dose of calomel, and by the same means a labouring heart, unable to cope with the resistance in the arterioles and capillaries, may be at once relieved. The great remedy for mischief of any kind impending as a result of high blood-pressure is a mercurial purge. The effect of mercury employed as an aperient upon abnormal tension in the arteries is matter of observation. The method by which the effect is produced is a question of hypothesis, but there can be no doubt that it is by elimination, and there need be little hesitation in concluding that the seat of the accelerated metabolism —of which the elimination is a resultant—is the liver. Such, at any rate, is the working hypothesis by which I am guided. It may be added, perhaps, that I entered upon the independent study of medicine fully impressed with the view of teachers held in high respect and confidence, who considered that the action of mercury on the liver had been entirely disproved, and that mercury, indeed, had practically no useful place in medicine, and that it has been from my experience of its effects on blood-pressure that I have come to value it

as one of our most important remedies. Full doses of calomel being reserved for emergencies, the less serious symptoms may be met by the administration of a single grain of pil. hydrarg. with ipecac. and rhubarb or colocynth twice or three times a week, with which may be combined from time to time a three weeks' course of mild salines. To intermediate degrees of urgency may be adapted suitable doses and combinations.

Next to mercury as eliminants will come potash and its salts, liquor potassæ having a greater metabolic influence than the salts ; the carbonate a greater effect of this kind probably than the citrate, and the citrate and acetate, which undergo decomposition into carbonate, a greater effect than the phosphate, or nitrate, or sulphate. As diuretics these different salts are much on the same footing, except in so far as this action is influenced by varying degrees of solubility. The effect of phosphate of potash in preventing the splitting-up of the quadrurates, which constitute the common urinary deposit, with separation of uric acid in the crystalline form is, however, worthy of special note. The demonstration of this by Sir William Roberts is one of the neatest and most interesting bits of recent work with which I am acquainted.

The soda salts have some eliminant influence, but it is not to be compared with that of potash. The idea that potash and soda compounds can be employed indifferently is as erroneous as it is prevalent. The two bases have totally different relations with the organic substances entering into the structure of the body, and have, indeed, nothing in common except their alkalinity.

Prognosis.—At the very outset of the employment of the sphygmograph for clinical purposes, Dr. Burdon Sanderson pointed out the prognostic significance of high arterial tension, and the importance of

this can scarcely be exaggerated. Years beforehand it can be foreseen that certain persons will at a given age be in danger of an attack of apoplexy or will suffer from dilatation or other disease of the heart. These events are simply the developments of the effects of unduly high pressure in the arterial system, and are foretold by the tense radials and tortuous temporals.

It must not be at once concluded that everyone who presents these marks of high tension will necessarily be cut off prematurely by cerebral hæmorrhage, or heart disease, or crippled by paralysis. There are individuals of so tough a fibre and of such vital tenacity that the teachings of average experience do not apply to them, and the heart and vessels do not suffer appreciably from over-strain, which would be destructive of more cheaply organised structures.

Again, degeneration in the arteries and failing energy in the heart may proceed with such even steps that the heart does not rupture the vessels nor the vessels ruin the heart. More than once I have seen patients in whom the tension was dangerously high, and in whom it seemed that something must give way, outlive the dangers to which they were exposed from high blood-pressure in the arteries, and, after slow and gradual failure of mental and bodily vigour extending over many years, ultimately die of senile gangrene or thrombosis of cerebral vessels. Allowance, again, must be made for the effects of change of *régime* and mode of life adopted voluntarily, as when a man retires from business, or enforced by illness. How often does an attack of hemiplegia lead to a prolongation of life?

While, therefore, abnormally high arterial tension is a sufficient ground for apprehension, it is only one factor in the prognosis, and must serve as a starting-point for investigation.

The first point to be ascertained will obviously be the amount of injury already sustained by the heart and vessels. The radial artery will be carefully explored, by the methods already described, as to its diameter and capacity of contraction, as to the thickness and elasticity of its coats, as to the existence of indurations, bulgings, tortuosities in its course, and as to the character of the pulse wave. The aorta will be examined as to any dilatation indicated by extension of dullness beyond the right border of the sternum or by pulsation perceptible on pressing the finger into the intercostal spaces here; the aortic second sound will be the subject of very careful scrutiny with regard to the degree of accentuation it may present and to its audibility at and beyond the apex of the heart, especially with regard to its being heard lower down along the right edge of the sternum than normal, and to the left of the manubrium. The character as well as the loudness of this sound will be noted, a low-pitched and ringing second sound indicating dilatation and degeneration of the root of the aorta.

One of the most important inquiries will be as to the family longevity and the modes of death which have prevailed. In one family apoplexy will predominate, in another heart disease; the latter reveals the more serious tendency.

If brothers or sisters have died at a comparatively early age from cardio-vascular disease this will be much more significant than the age at death of parents or grandparents, and may entirely neutralise inferences from their longevity. It is not at all uncommon in high tension families for successive generations to become shorter-lived, with or without the development of tendencies to diabetes or kidney disease.

The medical history of the individual will have to be taken into account, *e.g.* attacks of acute and

subacute gout from which he may have suffered, liability to functional derangements of the kind ascribed to suppressed gout, the character and quantity of the urine, especially its specific gravity. An estimate also must be formed of his vital tenacity, of the integrity of his structures, and the quality of his blood, from his general appearance and complexion and from the condition of his skin. Conclusions from a healthy and hearty look must be subject to the results of the examination of the heart and vessels; when the vessels are degenerated and the heart is sound, rude health becomes a source of danger.

The habits, dietetic and other, and the mode of life generally must also be taken into consideration. They may be responsible for the high tension in a greater or less degree, and the prognosis may turn on the power or willingness of the patient to modify his mode of life.

These will be the principal elements in a forecast of the probable length of life and cause of death in the subjects of high arterial tension, which, applied with judgment at different ages and in the two sexes, will be of great service.

CHAPTER X.

THE PULSE IN ACUTE DISEASE.

It has already been said that almost all deviations from a normal state of health are attended by increased frequency of the pulse, and this increase of frequency is especially marked in febrile disease. Elevation of the temperature is, indeed, almost as common a result of disease as increased frequency in the action of the heart; and it might be supposed, on a superficial review, that the one depended on the other. This, however, would be very far from the truth; and even in diseases in which pyrexia is the most prominent and characteristic phenomenon, there is no constant relation between the temperature and the pulse. A degree of frequency, which in one kind of fever would have no particular importance, would in another be prognostic of a fatal result.

With increased frequency in the action of the heart, there is in most febrile conditions relaxation of the arterial walls, and arterial relaxation is the condition of the vessels characteristic of pyrexia. The pulse of fever, as such, then, is frequent, large and short; usually, also, markedly dicrotic, since the cardiac systole is mostly sharp. The vehemence of the beats will vary greatly, according to the kind of fever and at different stages, as the action of the heart becomes weaker from the exhaustion due to continued pyrexia.

The sounds of the heart are louder, and the first is somewhat shorter; but there is no modification, and it is interesting to note, as has already been pointed out, that the systolic and diastolic intervals

retain very nearly the relation of health, both being shortened.

The pulse in intermittent fever.—The pulse in intermittent fever is of great interest, first because in twenty-four hours we have almost all the varieties producible by varying states of contraction and relaxation of the arterioles; and, again, because it was in malarial fevers of different types that the pulse was studied by the ancients. It was out of this study and observation that the doctrine of crisis arose, critical epistaxis or diarrhœa being common in case of recovery, and hebetude a common precursor of a fatal termination. Paludal diseases were infinitely more common in early times than they are now in civilised countries and temperate climates, and the forms which they assumed were more varied and malignant on the shores of the Mediterranean than were ever known in Great Britain. It will be remembered that the Asiatic and African shores, which still furnish illustrations of malignant intermittent fevers, were the seat of early civilisation, and were very thickly populated.

In the first or cold stage of a paroxysm of intermittent fever with rigor, when the internal temperature is high—104° or 105°, or even higher—while the surface is cold and pale, with intense subjective feeling of chill and violent shivering, the pulse is frequent, but small and long, the cutaneous arterioles being tightened up so as almost to exclude the blood from the surface of the body. If the same amount of blood were discharged from the left ventricle with each systole as in health, it would follow that there must be an enormous increase in the movement of blood through the muscles and internal organs. But although the pulse has the characters of high tension, the actual pressure within the vessel is not very great, and the wave can be extinguished without

much difficulty. The heart is affected as well as the arteries; it is not simply overpowered by the increased peripheral resistance; its action is not that of violent effort to overcome the obstruction, but

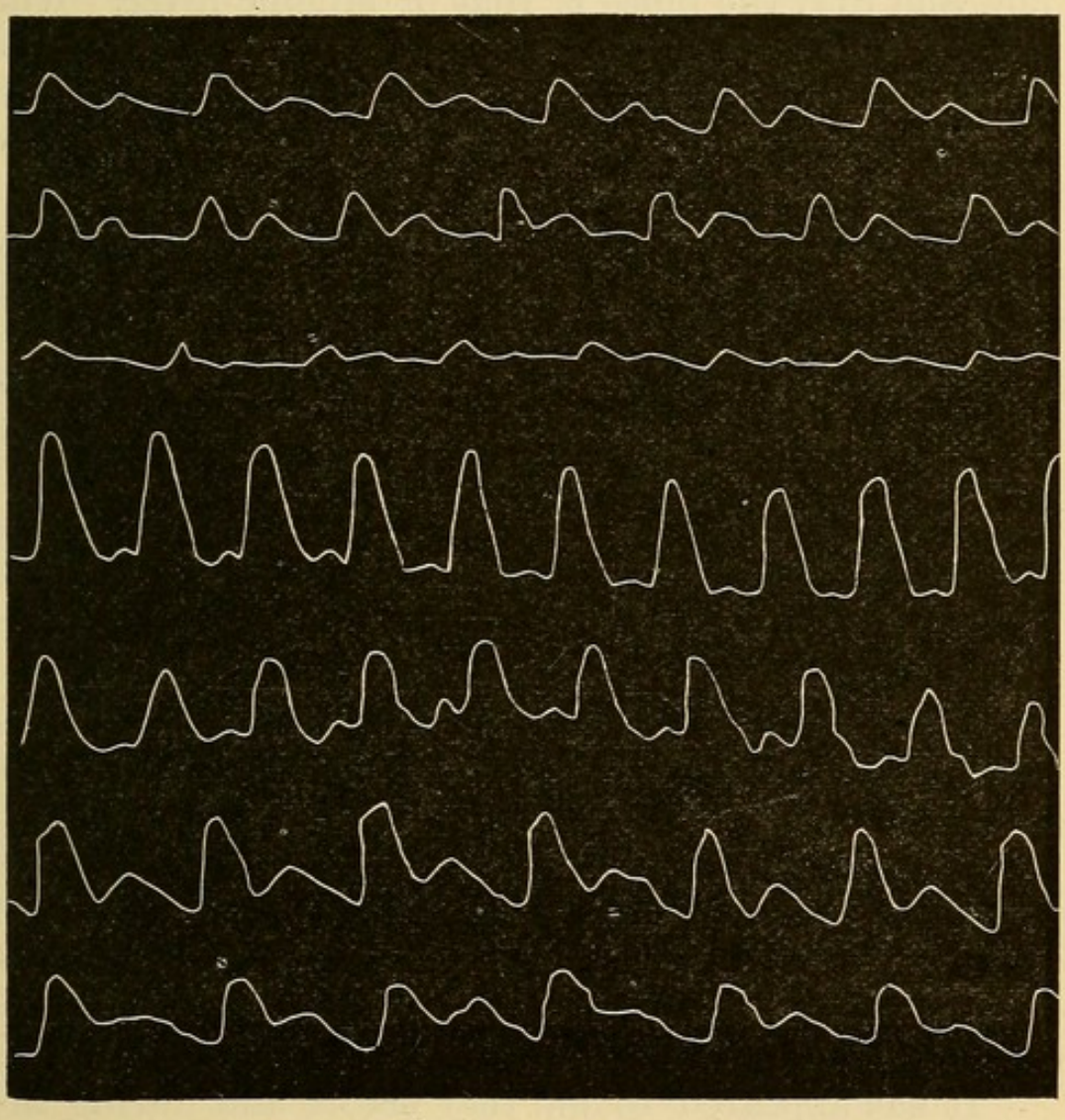


Fig. 44.—Pulse in Intermittent Fever. (From Marey.)

is often irregular, sometimes intermittent, with apparently an imperfect diastole, so that the ventricle has little blood to drive on into the arteries.

That the heart suffers from the direct depressing and almost paralysing influence of the poison is still more evident when the algid stage of pernicious fever is considered. The pulse is infrequent, small, and

scarcely perceptible; the lips and tongue are pale; the extremities, the face, and even the breath, are cold, as in the collapse of cholera.

In the second stage, in which the skin is flushed, hot, and dry, the pulse is that of sthenic fever. The heart's action is more frequent and powerful, the arterioles and capillaries are relaxed, but not to an extreme degree. The pulse is frequent, large, vehement, not very short, the trace being round-topped; the artery full between the beats and not very compressible; dicrotism is not very marked. It is the typical full and bounding pulse.

In the sweating stage the relaxation of the arterioles is more complete, but the action of the heart is also languid. The pulse remains frequent and large, but is soft and weak, the artery being less full between the beats and more compressible. There is little dicrotism—rarely any perceptible to the finger for lack of vigour in the cardiac systole. (Fig. 44.)

The pulse in the eruptive and continued fevers.—In small-pox there is nothing characteristic in the pulse. Its frequency is such as might accompany the high temperature, and there is nothing in the early stage of even a severe attack of confluent small-pox either in the rate or character of the pulse to foretell the danger which will arise in the course of the disease.

It has appeared to me that before and during the coming out of the eruption the arteries are not so much relaxed as in other febrile conditions, but that there is a degree of fulness between the beats and an absence of dicrotism. Later the congestion and infiltration of the skin and the presence of vesicles interfere with a proper appreciation of the characters of the pulse, but it feels short.

In the malignant hæmorrhagic form of small-pox the pulse is frequent, small, and weak from a very early stage.

In the fatal convulsions which occasionally attend the onset of variola I have found the pulse frequent, large, and short, as in sharp sthenic pyrexia.

The pulse of measles calls for little remark. In frequency it has the average relation with the temperature; the relaxation of the arteries is only moderate, so that the vessel is distinctly felt between the beats, and there is little or no dicrotism.

Scarlet-fever has a very remarkable pulse.

To begin with, the frequency is very great, and is altogether disproportionate to the degree of fever as indicated by the thermometer. In children a pulse of 160, 180, or even 200 is not uncommon, and in adults it may be 120 to 140, and this when the temperature is not higher than 102° F.; although with an extremely frequent pulse it will more frequently be 104° F. or upwards. When the frequency is inordinate it is indeed much better that the temperature should be high. The worst cases of scarlet-fever are those which set in with extreme frequency of pulse and low temperature. The pulse, again, is extremely small, instead of large, as is the rule in febrile conditions, the arterioles being contracted instead of relaxed, and this is more remarkable even than the frequency. It may be compressible easily, or with difficulty, but usually the vessel can be felt between the beats; the individual pulsations are necessarily short when they follow each other with such rapidity.

In typhus fever the pulse is not frequent, being usually below 100; it is large and peculiarly soft, as if the muscular coat of the vessel were paralysed, there being also a want of ictus or sharpness of stroke, evincing a languid action of the heart which helps to give the pulse its soft feel; it is, however, also very compressible. Dicrotism is present, but is not so easily recognised by the finger as in enteric fever or pneumonia. Towards the end of a fatal case, although

the pulse becomes more compressible, it retains the character described, and is not so profoundly modified as in enteric fever.

It is worthy of remark that while the nervous and muscular prostration and the dry brown tongue of the later stage of typhus seem to demand stimulants, it has been found that they can be withheld with safety —perhaps even with advantage.

The tendency to death is, in fact, by coma, and not by asthenia; by oppression of the nervous system, and not by wearing-out of the power of the heart; and when the crisis of the disease is past, recovery of strength and return to a normal state of the pulse are very rapid.

In enteric fever the pulse undergoes considerable modifications in the course of the disease, by reason of the diminution in the volume of the blood which takes place, and of the exhaustion of the heart and the granular degeneration of its walls produced by the protracted high temperature. In the early part of the disease the characters are simply those of the degree of pyrexia. The pulse gradually increases in frequency, during the first four or six days rising to 100, or in severe cases to 120; the arterial walls are relaxed, but not to an extreme degree, so that the vessel is only moderately large; the beat is sharp and short, and dicrotism is usually well marked and easily distinguished by the finger. During the second week there is not much change, except that usually dicrotism becomes less distinctly perceptible to the finger. After this, from failing power in the heart and by diminution in the amount of blood, the pulse becomes more frequent, smaller, weaker, and more compressible. When at any period of the disease, but especially later, the degree of cardiac asthenia becomes extreme, the beats of the pulse become indistinct, the pulsations with their dicrotic waves run one into another,

and the line of a sphygmographic trace is a mere undulation. This is called a running pulse, and is attended with enfeeblement and ultimately extinction of the first sound of the heart, the only audible evidence of the action of the heart being a weak second sound. Stimulants are necessary as the heart runs down, and

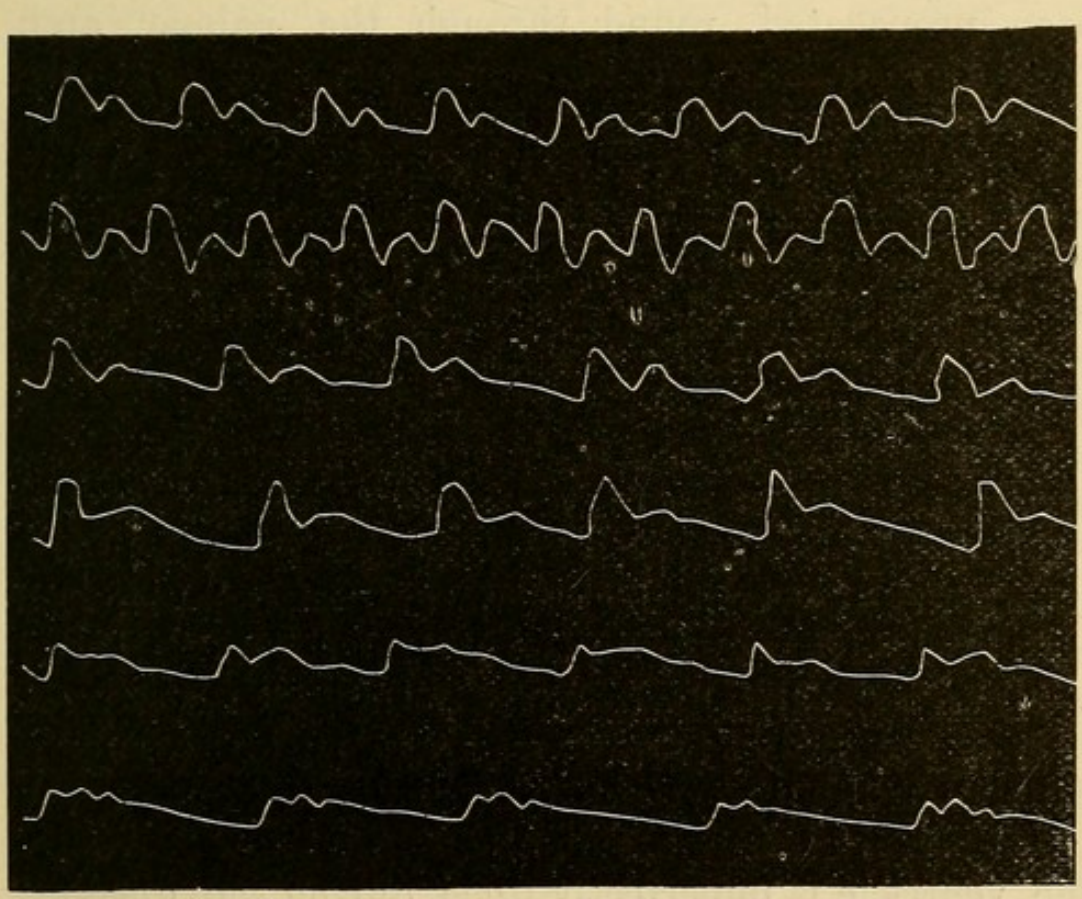


Fig. 45.—The Pulse in Typhoid Fever. (From Marey.)

the pulse is steadied and the temperature lowered by alcohol. Digitalis has sometimes seemed to be of real service. (Fig. 45.)

Relapsing fever in most of its features corresponds with the description of synochus by older writers. The pulse is frequent, forcible, and large, without being very short up to the time of the crisis; after which it is infrequent, soft, and short.

The pulse in pneumonia, etc.—Pneumonia has a frequent, large, vehement pulse with well marked

dicrotism. It is not very compressible, nor is the wave strikingly short, the dicrotism being the result rather of the sharp propulsion of blood by the heart, than of the diminished resistance in the peripheral vessels. Not unfrequently, when the radial is completely closed by the pressure of the finger, pulsation can be felt on its distal side, which has been propagated round through the palmar arch from the ulnar artery in consequence of the relaxation of the arterial walls. This is the pulse characteristic of the early stage, and it often persists up to the crisis; but at times, local conditions affect the circulation, and bring about profound and significant modifications. The obstruction to the pulmonary circulation, either from the extent of lung implicated, or more frequently from attendant engorgement of parts of the lung not distinctly implicated in the inflammatory process, is such as to embarrass the right ventricle. It is unable to propel the blood through the lungs as fast as it arrives by the venæ cavæ, and there result distension of the right auricle and veins and dilatation of the ventricle. The ventricle, indeed, becomes in some degree paralysed by the over-distension. Under these conditions, the amount of blood reaching the left side of the heart is inadequate, the ventricle is imperfectly filled during the diastole, and, having little blood to propel into the aorta, however forcible the contraction may be, there can be little increase of pressure in the arterial system, and little effect on the pulse. We have, then, instead of the large, vehement pulse, a small artery and weak beat, and this while the action of the heart is found on examination to be unusually forcible. The contrast between the violent action of the heart and the weak pulse which results is very significant. The face, again, instead of being flushed, is pale and haggard and often livid. It is in these circumstances that

venesection is of such remarkable service, relieving the over-distended heart, and the engorged lungs, and enabling the right ventricle again to get control over the pulmonary circulation.

In acute bronchitis the pulse varies greatly. It is essentially that of catarrhal fever—*i.e.* in which the relaxation of the arteries is more marked than the increased action of the heart,—modified more or less by the interference with the transit of blood through the lungs. Sometimes no effect of obstruction in the pulmonary circulation is perceptible; at others, the interference with the supply of blood to the left ventricle is such as to render the pulse small, and unequal in force and irregular in time. When the aëration of the blood is imperfect, contraction of the arterioles is induced, and contributes, with the defective filling of the ventricle, to render the pulse small.

In erysipelas the pulse is large and soft, markedly dicrotous, rather from diminished resistance than from sharpness of propulsion. Any vehemence which it may have at first is soon lost, and it tends to become undulating.

Diphtheria may begin insidiously, or with sharp fever. There is nothing remarkable in the pulse in the early stages, except that it is weak and, for pyrexia, small. The weakness of the pulse is not marked at first in cases in which the fever runs high, but becomes so in a few days, and is observed throughout. In the insidious form of the attack at an advanced period of the disease—sometimes after the membrane has cleared away from the fauces and the local affection seems to be cured,—a peculiar shortness, indicative of cardiac asthenia, is often the precursor of a fatal termination. The beat is, perhaps, rendered more distinct from being so extremely brief and unsustained, so that its essential weakness is disguised. The modification of the heart-sounds accompanying

this pulse is even more noteworthy: the systole is extremely short, and the second sound follows the first at so brief an interval as to seem almost to come on the top of it, the rhythm being altered from the normal triple time, which is maintained, or nearly so, in pyrexia, to common time "ONE, TWO, three, four," or even to "ONE, TWO, three, four, five, six," in which "ONE" and "TWO" represent the sounds, and the small letters the counted intervals.

In septicæmia there is the greatest possible range of diversity in the pulse. Speaking generally, the characteristic tendency is to frequency and sharpness of beat with a small artery. In severe puerperal septicæmia the frequency may be extreme—140 to 160 or even 200 per minute—the artery being small and full between the beats but compressible, the heart-sounds short and equidistant, and reminding one of the "tick-tack" of the fœtal heart. This is indicative of shock which may be fatal without rally, or may usher in other effects of blood-poisoning. From this extreme, gradations may be met with to a pulse differing little from that of ordinary pyrexia.

In pyæmia, which is a particular kind of septicæmia, evidences of shock are present at the onset, and throughout the pulse has a frequency and sharpness which do not belong to pyrexia as such. The vehemence of the beat and the feel of the artery will vary according to the phase of fever (rigor, heat, or sweating) which may be present, and it will be remembered that the irregular recurrence of paroxysms and remissions of fever is a distinguishing feature of pyæmia.

Inflammation of serous membranes, while raising the temperature, appears to give rise to contraction of the arterioles, instead of to the dilatation which accompanies pyrexia due to most other causes. This is most conspicuous in peritonitis, in which the pulse

is frequent, small, long and hard, the artery being full between the beats and not easily compressible. There may, however, be evidence of shock from the very first in severe peritonitis, so that the temperature is little, if at all, raised; or it may even be depressed, and when such is the case the pulse is not only extremely small, but weak and compressible, the heart is affected as well as the vessels, and it is either tightened up, like the arteries, so that its diastole is imperfect and the ventricles admit only an inadequate charge of blood, or its systole is feeble. Sometimes the terms "wiry" and "thready" have been applied to the pulse of peritonitis and abdominal shock, according as pressure within the arteries is present or absent. This thready character of the pulse is common, as peritonitis tends to a fatal termination, and all observers are familiar with the pallor of the face, the pinched features and sunken eyes, and the cold extremities of abdominal collapse, which are effects of imperfect filling of the arterioles and capillaries, intelligence remaining perfect to the last. It has been supposed that the mesenteric and other veins of the abdomen are paralysed and dilated in such circumstances, and, as is well known, these veins will hold the blood of the entire body, so that, stagnating and accumulating here, very little is carried to the heart, which is thus deprived of blood. It has been said that the sufferer is bled into his abdominal veins.

In acute pleurisy, again, there is a degree of tightening up of the arteries, but it is much less marked than in peritonitis; the pulse is full between the beats and longer than in fever of the same height from other causes. Possibly the arterial contraction is less marked in purulent pleuritis.

Gout, as has been already said, is one of the causes of high tension in the pulse. This statement applies strictly only to the intervals between the acute attacks,

for during a paroxysm the pyrexia relaxes in some degree the peripheral vessels and diminishes the tension. The pulse, however, has a certain vehemence, and, though sudden and comparatively short, is not readily compressible, nor does the artery efface itself altogether between the beats. At the onset of a paroxysm before redness and swelling of the affected part have set in, when the pain is at its worst and is of a shooting neuralgic character, the arteries are more than ever tightened up.

In acute and subacute rheumatism the pulse presents great differences in different cases, and in the same case at different times. The tension is never really high and the beats are mostly short, but sometimes the artery remains full between the beats and can be rolled under the fingers, and at others allows itself to be flattened by very slight pressure. The degree of acuteness of the articular inflammation will influence the frequency and vehemence of the pulse, and the copious perspirations which are characteristic of rheumatic fever will both relax the peripheral vessels and diminish the vigour of the heart's action; but it is not always easy to connect the pulse found at a given moment with the symptoms then present, or to explain all its variations by the conflicting tendencies which may be traceable in the course of the attack.

CHAPTER XI.

THE PULSE IN VALVULAR DISEASE OF THE HEART.

HEART disease produces the multifarious distressing symptoms which attend it by its effects upon the circulation, and these effects ought to be more or less manifest in the pulse. Such is, in fact, the case, and especially in valvular disease, which will first be considered ; but the compensatory changes in the heart, which tend to neutralise the derangement of the circulation produced by the damage to the valves, will tend also to modify the influence of the valvular lesion on the pulse. This, however, does not impair the diagnostic and prognostic importance of the pulse, but, on the contrary, heightens it, since the pulse comes to represent a resultant of opposing influences and forces.

The pulse in aortic stenosis.—The frequency of the pulse is little affected by this condition, and while the muscular walls of the heart remain sound it is quite regular ; but narrowing of the aortic orifice must have the effect of intercepting the sudden pressure brought to bear on the column of blood in the aorta at the beginning of the ventricular systole and of increasing the time required for the discharge of the contents of the ventricle. The corresponding modification of the pulse will be a loss of suddenness in the ictus and a longer duration of the beat ; the dicrotic wave also will be annulled, since the conditions of its production—rapid action of the ventricle and great fluctuations of the blood pressure—are absent. The ordinary characters of high tension in the pulse are thus reproduced as a result of obstruction and delay in the delivery of the blood by the heart. The

obstruction at the orifice is met and compensated by hypertrophy of the left ventricle; but this does not neutralise altogether the influence upon the character of the pulse. Another element of similarity to the pulse of high tension also comes in, namely, the small size of the artery. It is not very clear why, with normal resistance in front, the arteries generally should not be kept at their usual size by the pressure from the heart, which will be maintained at a normal

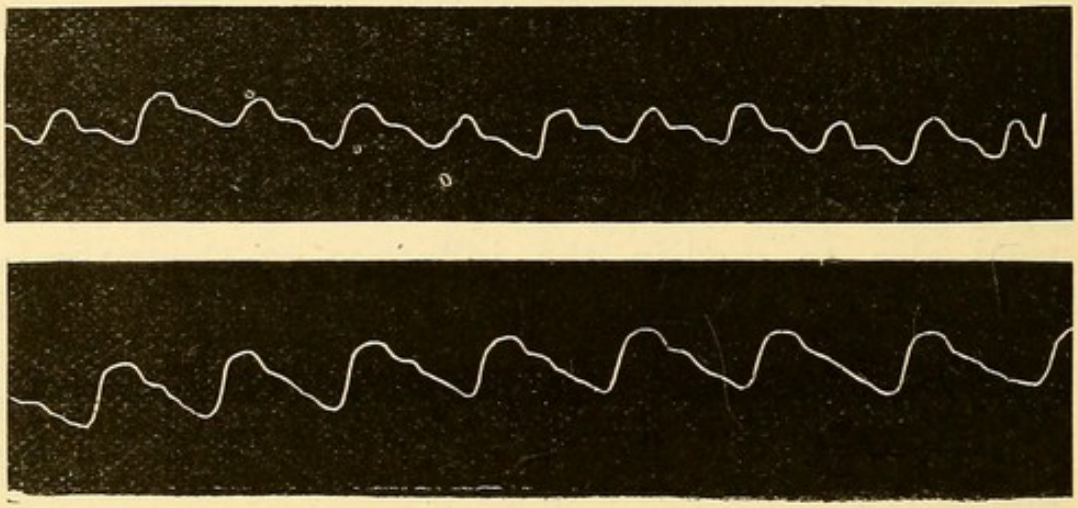


Fig. 46.—Pulse in Aortic Stenosis.

point by the compensatory hypertrophy, merely because the pressure takes effect more gradually; but the entire arterial system seems to contract down upon the diminished blood-stream, and the small diameter of the radial and other arteries is a constant phenomenon. The pulse, then, of aortic stenosis has the following characters:—The artery is small and full between the beats, but not, as a rule, really tense; the wave has no ictus, properly speaking, but lifts the finger gradually—it is persistent and subsides slowly; the trace has a sloping upstroke, little altitude, a rounded or flat top and a gradual descent. (Fig. 46.)

Sometimes the wave is reinforced towards its end and the finger is conscious of a second beat: this constitutes the pulsus bisferiens, traces of which are

here given, from which the difference between the double beat thus produced and that of dicrotism will be recognised at once. (Fig. 47.) A further difference is that the pulsus bisferiens is brought out by firm pressure while dicrotism is best felt when the fingers are very lightly placed on the artery and is

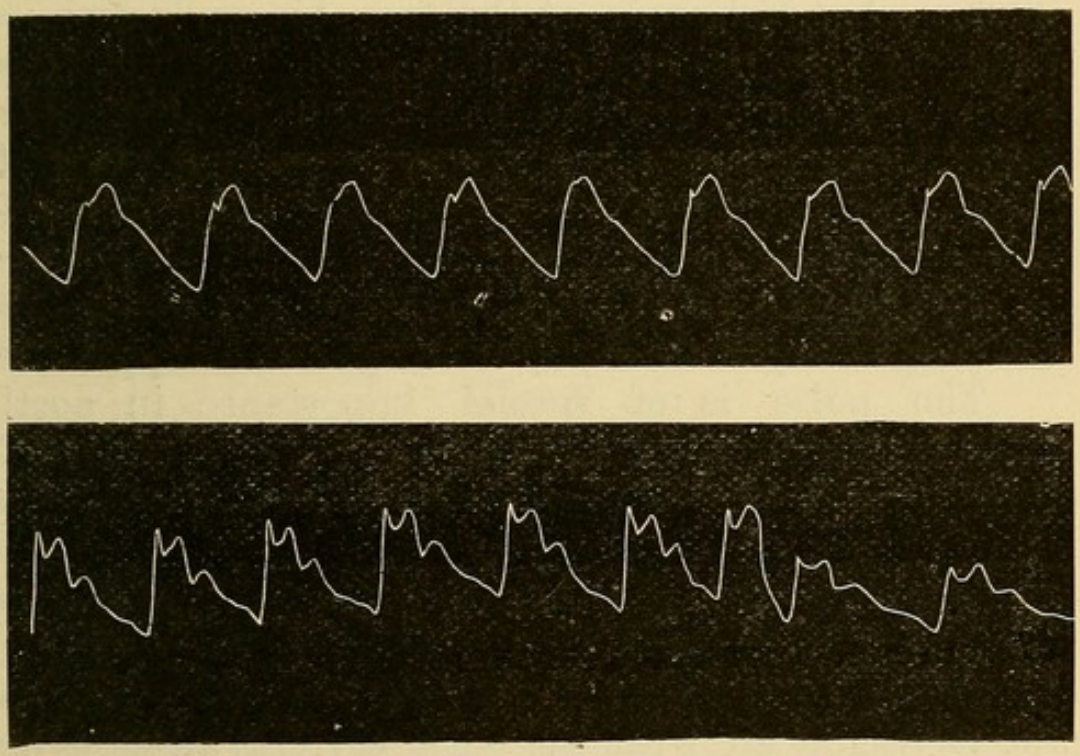


Fig. 47.—Pulsus Bisferiens.

extinguished by pressure. The pulsus bisferiens is interesting in connection with the view propounded by Professor d'Espine, of Geneva, that the ventricular systole is *à deux temps*, *i.e.* is compounded of two distinct efforts fused together, which are not separately recognisable in normal action, but become distinct in certain forms of disease.

The tracing was taken from a case under observation since March, 1884—that of a lady, aged 41, who for nearly three years had been subject to attacks of faintness coming on about one a.m., in which she was cold and absolutely powerless, without, however, losing consciousness; the faintness was followed by

palpitation. The left pulse was very small and long; the right, from an abnormal superficial distribution, was visible and gave a higher trace than would otherwise have been obtainable. She was subject also to menorrhagia and neuralgia. A very loud, coarse, low-pitched systolic murmur was heard over the aorta and its branches and again at the apex by conduction; a faint, short, diastolic murmur was also heard to the left of the sternum, but there was no visible carotid pulsation or other evidence of aortic regurgitation sufficient to affect the pulse or circulation, and the aortic second sound was audible in the neck. There was a moderate degree of hypertrophy of the left ventricle carrying the apex one inch to the left of the nipple line.

The pulse is of special importance in aortic stenosis, since upon it the diagnosis may almost be said to turn. A systolic aortic murmur is one of the most common of physical signs; actual obstruction at the aortic orifice comparatively rare. A slight roughness or rigidity of the valves, a tag of fibrin, or other deposit, not affecting their functional efficiency or interfering at all with the blood-current, a fenestrum in the thin crescent near the free edge of a valve, will cause loud murmurs, and a murmur may be produced at the aortic as well as at the pulmonary orifice in anæmia. There is nothing in the character of a systolic aortic murmur to tell us whether it is indicative of serious obstruction, or is produced by a mere roughness, or some other of the conditions just enumerated, and it is only by symptoms, when present, or, in their absence, by the changes in the heart, and by the character of the pulse taken together, that the distinction is made between dangerous disease and insignificant derangement.

The pulse may be made to throw further light on the degree of obstruction attending an aortic murmur by causing the patient to make some slight but

sudden exertion. Compensatory hypertrophy makes the heart perfectly equal to all ordinary calls upon it, and to the increased demands of exertion, provided this is not begun suddenly; but it does not accommodate itself quickly to the increasing rapidity of arrival of blood in the right side of the heart, caused by the compression of the veins in muscular exercise. The pulse then becomes unequal, short and irregular, and, excluding other causes of cardiac weakness, anæmia, sedentary habits, etc., a faltering pulse, say on going up a single flight of stairs, will indicate mechanical interference with the transit of blood through the heart, while if the pulse responds simply by increased frequency and force, the obstruction cannot be great.

It does not come within the scope of this book to describe the murmur of aortic stenosis, or the physical signs, either of compensatory hypertrophy, or of the changes which attend the later effects, which lead to a fatal termination; but there is one modification of the heart-sounds which should be noted, since it belongs, like the characteristic pulse, to the stenosis as such. This is the muffling of the aortic second sound. The propulsion of the blood into the aorta is gradual, the pressure in the arterial system is not high, the recoil, therefore, of the valves, even were they not thickened, as is probably the case, will not be violent.

The pulse of aortic regurgitation. — This is the well-known "collapsing pulse" of Corrigan, called also sometimes the "water-hammer pulse," which is visible in all the superficial arteries, and is especially conspicuous in the carotids. A diagnosis of aortic insufficiency may indeed be made from a glance at the neck, or from a moment's examination of the radial pulse, or by watching the movements of the foot when one leg is crossed over the other knee. The throb of

the carotids is visible up to the ear, and the beating of the temporal, facial and subclavian arteries at once attracts attention, while, if the elbow is bent, the sinuous brachial artery becomes extremely conspicuous.

Insufficiency of the aortic valves, however, is produced in two quite different ways—by damage to the valvular cusps themselves, which renders them incompetent to close the orifice, and by stretching of the orifice, so that it is too large to be closed even by valves of normal size. In the latter case the enlargement of the orifice is part of a general dilatation of the root of the aorta, due to atheroma or arteritis deformans; the valves may be little changed and will then stretch across the mouth of the aorta, leaving a small central aperture where they fail to meet, or they may be implicated in the degenerative process, when they will be rigid, more or less contracted, and perhaps calcareous. Valvular lesions, properly speaking, thickening, adhesion and contraction, and occasionally ulceration or destruction, are, for the most part, the result of rheumatic, more rarely of gouty, inflammation, and it is as a result of such lesions that the true collapsing pulse is met with. The description to be given in the first instance of the pulse of aortic incompetence will apply only to this form of disease; the modifications presented when the incompetence is produced by aortitis deformans or atheroma will be reserved for separate consideration.

As in aortic stenosis, the pulse remains regular until the heart begins to fail, and then the first departure from the normal rhythm consists in an occasional falter, a hurried, ineffectual contraction of the heart with a weak beat at the wrist, or an intermission due to the wave not reaching the radial artery.

The most striking features of this pulse are the

sudden and complete collapse, or emptying of the artery between the beats, and the extremely sudden, vehement, and short pulsation. This comes from the fact that the aortic valves being insufficient, the fulcrum which sustains the column of blood in the aorta, and therefore the blood pressure in the arteries, is, *pro tanto*, wanting, and the blood drops back out of the radial and other arteries. It must be remembered that for the collapsing character of the pulse to be fully developed the hand must be raised; if the patient is in bed this will be done in the mere act of feeling the pulse, but if the patient is sitting or standing, the wrist will be below the level of the shoulder, and gravity will keep the vessel more or less continuously full, disguising the collapse. But in a well-marked case even in the dependent position, the sudden and collapsing character of the pulse will be recognisable; the column of blood from the shoulder to the wrist has not weight enough to simulate the normal blood-pressure in the intervals, but the collapse becomes much more conspicuous when the hand is raised, so that the blood, no longer sustained by the aortic valves, drops back out of the artery. When, therefore, the prognostic import of the pulse is sought in aortic regurgitation, it should be carefully examined in all positions of the hand and arm.

There is one other important feature of the pulse of aortic regurgitation—the size of the artery. The compensation of aortic regurgitation consists in dilatation and hypertrophy of the left ventricle, and the dilatation—which in other forms of heart-disease is at once a sign, a cause, and a consequence of heart-weakness and failure—is here an essential factor in the compensation. If a certain proportion of the blood propelled into the aorta regurgitates into the ventricle, there must—if the circulation is to be maintained at the normal rate of flow—be a larger

amount than normal injected at each systole: that is, the capacity of the left ventricle must be increased—or, in other words, it must be dilated. A consequence of this will be that a larger amount of blood is launched at each systole into the aorta; and, although a certain proportion of it immediately flows back into the ventricle, room must be made for it in the arterial system for the moment, and so the arteries generally are large.

Still another effect is loss of time between the heart and the wrist. It has already been said that when the general arterial tension is low the wave takes a longer time to reach the periphery, and when the aortic valves are incompetent, this delay reaches its maximum. Accordingly, the loss of time is such that very often, when one hand is placed over the apex of the heart and the other on the pulse, the two beats are felt to alternate at equal intervals, and the loss of time may be carried so far that the pulse at the wrist seems to come before the cardiac impulse.

The loss of time is manifest even in the carotids, and the apex beat, the carotid pulse, and the radial pulse follow each other in triple time—"one, two, three; one, two, three." This delay has not received adequate attention, and the only observer who has fully brought out its significance and importance is Prof. Raymond Tripier, of Lyons, whose papers on the "Retard Carotidien" are of great interest and value.

We can now give a complete technical description of the pulse of aortic regurgitation. The artery is large, but between the beats quite empty; the pulsation is extremely sudden and vehement, and often communicates a vibratory sensation to the fingers; its duration is very brief, and its cessation peculiarly abrupt, giving it the collapsing feel. Dicrotism is not altogether absent, but for want of the fulcrum formed by the valves, it is much less marked than might be

expected from the violence of the fluctuations of pressure and the rapidity of the systole.

It has already been said that this is the visible pulse *par excellence*, and it is not only visible but also audible. If the wrist is placed against the ear, the blood can be heard to come into the radial with a rush, or with a sound like the cut of a whip.

Mention may also be made of the capillary pulsation, the pulsatile reddening of a patch of congestion induced by rubbing the skin.

Several traces are here shown; they vary considerably in appearance, but there will be seen to be common to all of them a sharp and long upstroke, usually ending in a hook at the summit, due to jerking up of the lever, a great altitude, and a very abrupt descent, with a slight dicrotic rebound, usually from the base line. In the last, good compensation has been established. (Figs. 48, 49.)

Here, as in aortic obstruction, the pulse enters into the diagnosis and has very great weight in the prognosis. A diastolic, or a double aortic murmur, will be present whenever there is incompetence of the semilunar valves, whether the leakage is slight or considerable, and we cannot depend on the murmur to tell us whether it is merely a thin vein of blood or a large stream which pours back into the ventricle. For this information, upon which the prognosis largely turns, we must look to the effects upon the heart, the amount of dilatation and hypertrophy, but especially to the character of the pulse. However loud the diastolic murmur, or wherever heard, unless there is a visible carotid and radial pulse, the regurgitation will not be large, it being understood, of course, that we are speaking of a period of the disease in which the patient is in apparent health, and not of a late stage when the heart is failing. But we do not rely on a single first-sight indication; all the characters of

the pulse must be separately considered, the size of the artery, the suddenness and vehemence with which the pulsation strikes the finger, the suddenness and

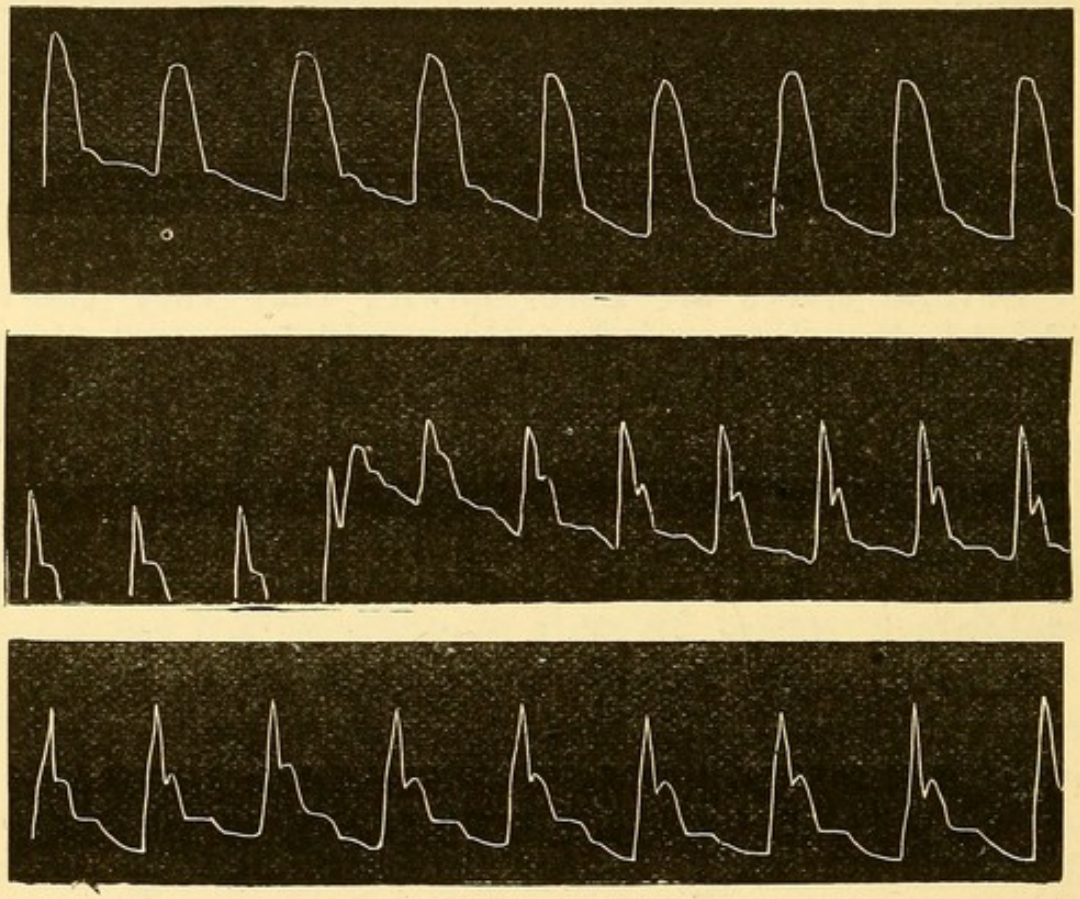


Fig. 48.--Aortic Regurgitation.

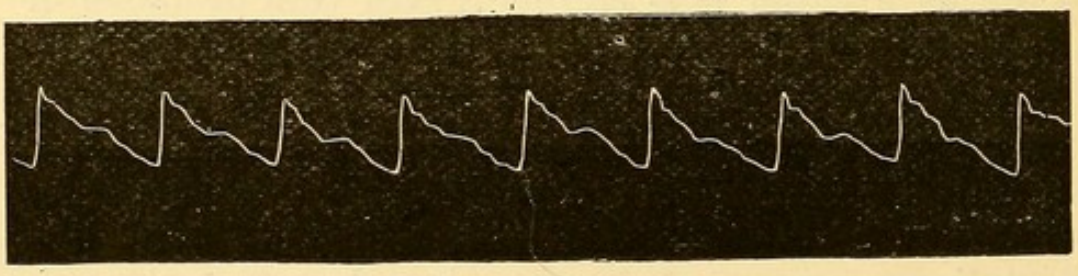


Fig. 49.—Aortic Regurgitation in which Compensation has been established.

completeness of the collapse. Especially must the state of the artery between the beats be ascertained when the hand is held up above the level of the shoulder or head. If the valves are seriously damaged, so that the regurgitation is free, the blood

drops out of the artery immediately the systole is completed, and we have, fully developed, those characters which have given to the collapsing pulse the names "water-hammer" and "whipping;" and when the wrist is applied to the ear the rush of blood, or rather the sudden stretching of the artery, is distinctly heard. If, on the other hand, the reflux is inconsiderable, the valves may sustain the column of blood for an appreciable period; and, although the pulse may be sudden, the artery is emptied gradually only, and can be felt to contain blood for a time after the beat, or even throughout the interval between the beats.

Nothing can be more simple and easy than the appreciation of the differences here pointed out, or more trustworthy than the indications they furnish, when taken together with other indications obtained from the state of the heart and the general condition of the patient. Here, as when treating of aortic stenosis, while the physical signs of hypertrophy and dilatation cannot be described, nor their significance discussed, the modification of the aortic second sound comes within the scope of this book. The valves enter into the causation of this sound by their sudden tension under the pressure of the blood in the aorta. If, therefore, they are practically non-existent, there can be no aortic second sound; and, in proportion as they are incapable of opposing the reflux of blood into the ventricle, they will be unable to give rise to the tension-vibration of the valvular cusps and aortic wall, which produce this sound. The aortic second sound thus becomes an important criterion of the amount of regurgitation. Its absence is among the indications of serious incompetence; and, on the other hand, when it is distinct the regurgitation cannot be considerable. In order that the pulmonary second sound may not be taken for the aortic, the stethoscope must be applied over the carotids.

In cases of extreme aortic insufficiency the murmur is often short, smooth, and almost noiseless, so that it might escape the ear unless the attention has been awakened by the pulse; and when the patient is in bed suffering from some acute disease, such as rheumatic fever, or congestion of the lungs, the diastolic murmur may be inaudible. It has several times occurred to me in such circumstances to predict the appearance of a murmur as the patient recovered, the pulse indicating regurgitation, of which the most careful examination failed to detect the usual auscultatory evidence. It must be borne in mind that pyrexia will exaggerate to an extraordinary degree the collapsing character of the pulse by relaxing the peripheral vessels; and other causes of arterio-capillary relaxation will have the same effect. It must, again, especially be borne in mind that stenosis of the aortic orifice will modify and disguise the effects on the pulse of incompetence of the valves.

The pulse of aortic incompetence from aortitis deformans.—In considering the departures from the collapsing type of pulse as fully developed in valvular disease proper, it has first to be borne in mind that regurgitation through the aortic valves resulting from dilatation of the orifice in aortitis deformans is usually a late event in the course of the disease. There will be associated with the regurgitation loss of the elasticity of the coats of the aorta; this will, indeed, have preceded it in most cases by some considerable time. The pulse then will have the character produced by rigidity of the great vessels —those, namely, already described under the head of senile pulse. The artery will be large, its walls probably thickened and hard, and it will be full between the beats; it will, however, be compressible, and the pulse-wave will arrive and depart suddenly. These features belong to the pulse independently of the regurgitation,

and almost the only modification which this introduces will be that the artery will be more easily flattened between the beats, which will give the beats a sharper feel. (Fig. 50.)

The most striking departure from the type of pulse of incompetence from valve disease is that there is no real collapse or emptying of the artery between the beats, even when the hand is raised; the regurgitation through the valve is, in fact, comparatively small, as has been already pointed out. Any considerable insufficiency of the valves produced by

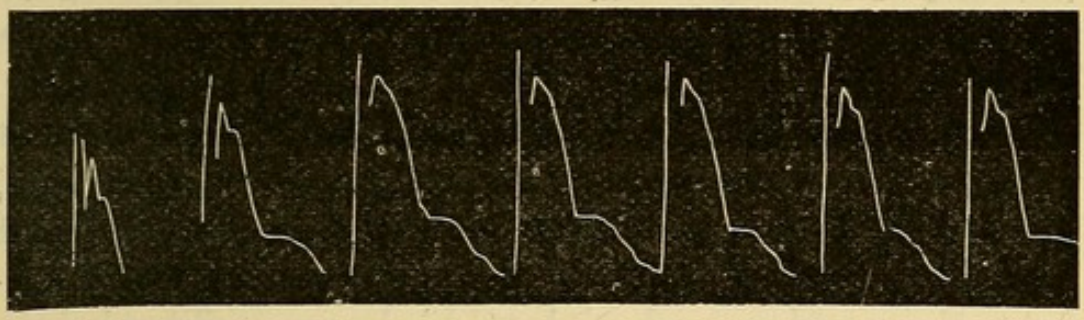


Fig. 50.—Aortic Regurgitation from Atheroma.

atheromatous disease of the aorta would be incompatible with life; the heart would be incapable of responding to the need for compensatory increase of vigour and hypertrophy. This would be rendered impossible by the loss of elasticity in the aorta, and consequent imperfect blood-pressure in the coronary arteries, the orifices of these vessels also being liable to be implicated in the disease and more or less obstructed, were it not also the case that the degenerative change under consideration usually occurs at a time of life when there is not sufficient energy in the nutritive processes for the production of hypertrophy.

Another point of difference between the pulse of aortitis deformans with regurgitation and that of regurgitation from valvular disease is that the loss of time between the heart and the vessels is much less. This is specially noteworthy in the carotids, and the

absence of *retard carotidien* has been pointed out by Professor Tripier as a clinical distinction between the two forms of incompetence.

In the cardiac physical signs there is also an important difference which must be pointed out. It is that, whereas in valvular disease the aortic second sound is impaired or lost, in aortitis this sound, often accentuated and ringing, is distinctly heard heading, so to speak, the diastolic murmur.

The prognosis in aortic insufficiency due to dilatation of the orifice is not dependent simply upon the amount of regurgitation. It is always serious because of the probability that the coronary arteries may be implicated in the thickening and degeneration.

The pulse in combined aortic stenosis and incompetence.—As has just been said, narrowing of the aortic orifice will neutralise some of the effects of incompetence of the valves; it will intercept the sudden and violent discharge of blood into the arterial system by the ventricular systole, and it will diminish the freedom of the regurgitation during diastole. It will thus tend to destroy all the special characters of the pulse, its sharp and powerful ictus, its instant collapse on the passing of the wave, and to interfere with the large size of the radial artery; as regards the indications of the pulse, therefore, it may completely disguise the serious character of the valvular lesion.

Since stenosis and insufficiency may be combined in various degrees, no general description of the pulse would be applicable to all cases: it will be regular, will have more or less of the collapsing character, and will be more or less visible; the appreciation of its diagnostic and prognostic significance must be left to the individual observer.

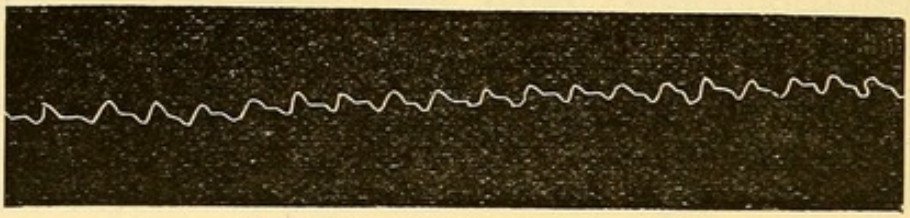
Obstruction and regurgitation are together more serious than obstruction; stenosis may add to or take

from the danger attending incompetence. On the whole, probably, combined obstruction and regurgitation will have a greater tendency to shorten life than regurgitation alone ; but I have more than once seen the supervention of stenosis stave off the fatal effects of extreme regurgitation, and even lead to apparent recovery.

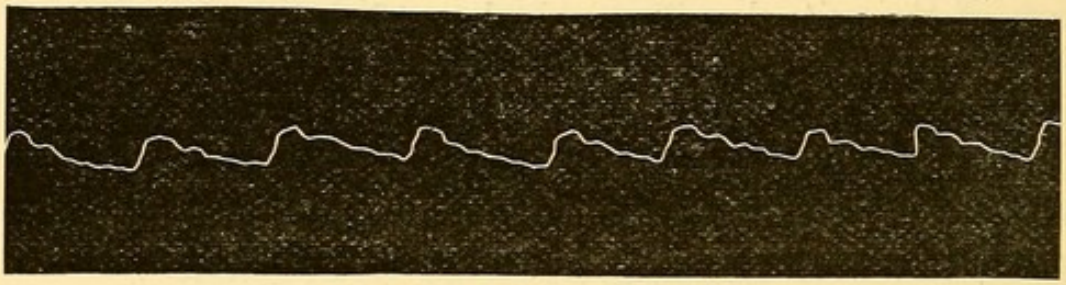
The pulse of mitral stenosis.—Great difference of opinion has existed as to the pulse in constriction of the mitral orifice. It has often been described as irregular, sometimes, as by Dr. George Balfour, as exhibiting the extreme of irregularity. This is altogether contrary to my experience, according to which, in simple uncomplicated mitral stenosis, the pulse remains regular up to an advanced stage of the disease, frequently even when the effects on the circulation have reached a serious point. It is true that when the pulmonary circulation is almost brought to a standstill, the right heart distended, and the tricuspid valve no longer prevents regurgitation into the systemic veins, the pulse may be extremely irregular ; but even in these circumstances the heart will usually be found to be beating regularly, and the irregularity of the pulse is the result of beats not reaching the wrists.

The pulse, then, of mitral stenosis is regular ; its other characters are that it is small, long, and extinguishable by moderate pressure ; it could not justly be termed a weak pulse. A constant feature is that the artery is full between the beats, and the pulse is exactly such as would be described as essentially one of tension, in which the arterioles are contracted, but the vis a tergo from the heart necessary to the production of high pressure in the arteries is only moderate. The explanation of this condition of the arterial side of the circulation is not very apparent ; it may be that the vessels adapt themselves to an inadequate supply of blood by contracting down upon their contents ; or

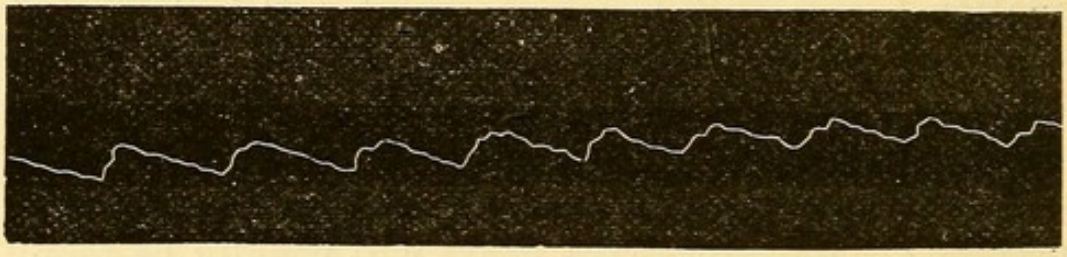
that a tendency to backward pressure in the veins makes itself felt in the capillaries and arterioles. This last supposition, however, is scarcely tenable, and



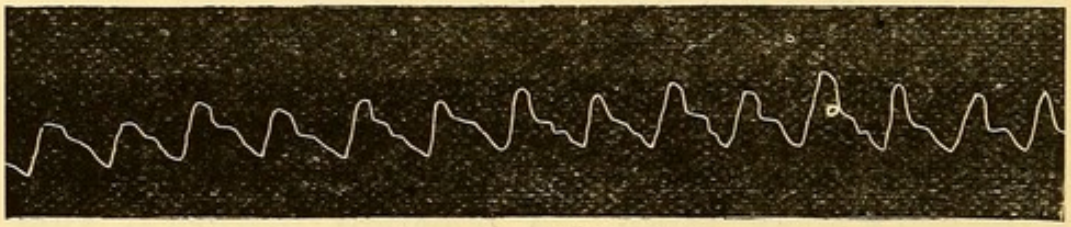
Mitral Constriction (extreme).



Mitral Constriction (Pulse 60).



Mitral Constriction.



Mitral Obstruction and partial Aortic Regurgitation.

Fig. 51.—Pulse of Mitral Stenosis. (From Hayden.)

there is possibly some intervention of the nervous system, the arterial walls being stimulated to contraction by a reflex action through the sympathetic vaso-motor.

The tracings in Fig. 51 are taken from Hayden's work on heart disease rather than from cases of my own, and they correspond with the description just given. All are seen to be regular.

In the final stages of mitral stenosis, when stasis of the venous circulation is setting in and severe symptoms are being developed, the pulse, as has been said, may become irregular. The irregularity first shows itself as inequality in the force of different beats due to imperfect filling of the ventricle; the weaker beats also travel less rapidly, and thus the time as well as the strength of the pulse becomes irregular. Then a certain number of the beats fail to reach the wrist at all, and in this way extreme irregularity is produced, and there is an entire loss of correspondence between the heart and the pulse, so that it seems difficult to believe, when listening to the heart and feeling the pulse at the same time, that the one is at all dependent on the other.

The effects here described are not difficult to understand. When narrowing of the mitral orifice is carried to any considerable degree, the filling of the ventricle in its diastole, which in a normal state is almost instantaneous, must be greatly retarded, and it is only by high pressure in the pulmonary veins that anything like an adequate charge of blood is forced through the constricted communication between the auricle and ventricle in the time during which the diastole lasts. The auricle helps in this for a time, especially when hypertrophied; but, sooner or later, it becomes dilated, and is often distended into a large passive sac or reservoir; and even while it retains contractile energy, the fulcrum—which, in the absence of valves, causes the blood to pass onwards into the ventricle—is the resistance opposed by the pressure in the pulmonary veins. The pressure in the pulmonary circulation is, of course, kept up by the hypertrophied right ventricle, and the right ventricle thus virtually comes to the aid of the left by driving the blood with greater velocity through the narrow mitral orifice. When, therefore, any obstacle is

interposed to the free passage of blood through the lungs, as by bronchitis, or pneumonia, or congestion, or pleural effusion, and still more when the propulsive efficiency of the right ventricle is impaired by weakness of its muscular walls or by over-distension, and especially when the tricuspid valve becomes insufficient, either from dilatation of the cavity and orifice or from damage to its flaps or tendinous cords, and the right ventricle loses its fulcrum, the blood is no longer forced through the narrow mitral orifice quickly enough to fill the left ventricle. This being so, although the ventricle may act regularly and forcibly, when it contracts upon an inadequate charge no beat will reach the wrist, and its contraction, not encountering a normal degree of resistance, will be short and hurried. All this being considered, the wonder is, not that the pulse should be irregular, but that it should continue for so long to be regular.

When mitral regurgitation co-exists with obstruction, it is an independent source of irregularity of the pulse; and the same may probably be said of tricuspid regurgitation when it is established in the course of the disease.

A few words may be said with regard to another variety of pulse sometimes met with in mitral stenosis, especially when under treatment by digitalis. This is when there are two beats of the heart to one of the pulse. It has already been described and discussed in an earlier part of this work (chap. vi., page 108), but is of sufficient interest and importance to merit further reference. It does not occur in connection with any other form of valvular disease, nor, so far as I know, in mitral stenosis, except when digitalis is being administered, and then only in a small minority of cases; but in certain cases it can be produced at will by giving this drug.

The pulse is usually quite regular, both as to time

and force, but is infrequent, the number of beats being, perhaps, forty per minute. When the heart is examined, it is found to be beating at exactly twice this rate. The beats, however, are not alike, but run in couples—a strong beat followed at a brief interval by a weaker one, while a longer pause separates the coupled beats from each other. The first only of the couple gives a pulse at the wrist, as a rule. Not only do the two heart-beats differ in strength, but the impulse is felt at a different spot—at the apex with the first, over the right ventricle with the second; and on auscultation the sounds of the first are heard to belong to the left ventricle, those of the second to the right. It is obvious that the two ventricles are acting alternately; not that one is absolutely quiescent during the systole of the other, but the first beat is predominantly that of the left, the second that of the right. On careful examination, however, both sounds of the right ventricle are audible with each systole, but with the second there is only a short, weak, left ventricle first sound and no aortic second sound, the aortic valves not being raised. A more exact statement of the facts than that the ventricles contracted alternately would be that there were two effectual beats of the right heart to one of the left. And it seems clear that an extra systole of the right ventricle may be useful, and indeed necessary, in order to drive an adequate supply of blood into the left ventricle.

The pulse of mitral insufficiency.—This may almost be said to be the one irregular pulse of valvular disease. It cannot be stated with confidence that the regurgitation through the mitral orifice gives rise in all cases to irregularity of the pulse, but it is rare that it goes on to the production of symptoms without this effect. It is an effect which is easily understood, and which might have been anticipated. When there is free reflux into the

auricle, since the blood will move in the direction of least resistance, the proportion of the contents of the ventricle propelled into the aorta will depend on the fluid-pressure maintained in the auricle and pulmonary veins. Now this will vary in inspiration and expiration; and accordingly at one moment more, at another less blood will be injected into the arterial system, and this inequality will be felt in the

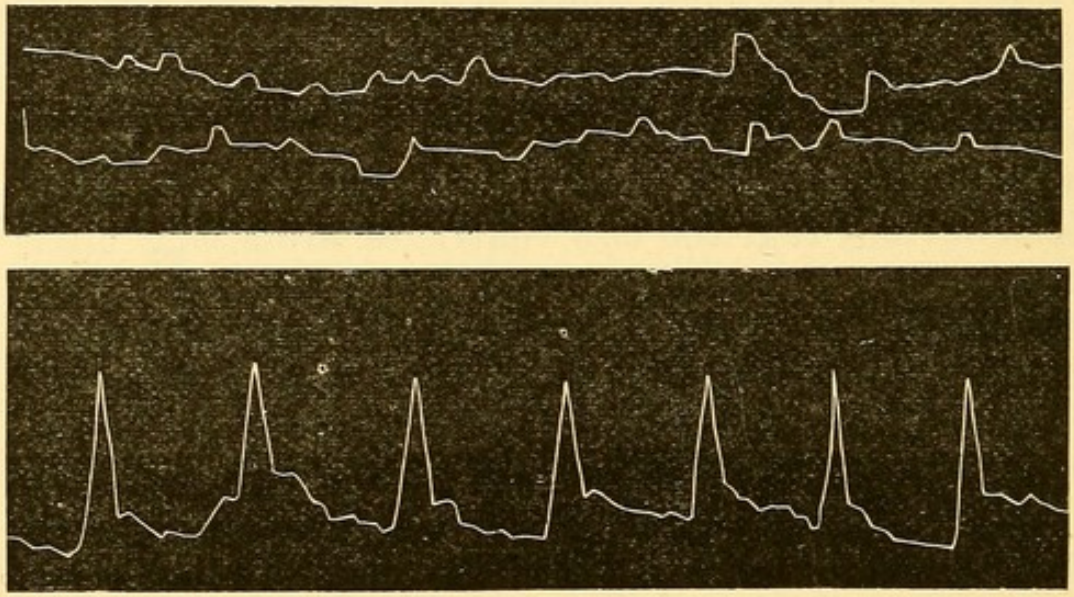


Fig. 52.—Mitral Regurgitation.

pulse. The resistance experienced by the ventricle in its contraction, again, will vary: in inspiration there will be negative pressure in the auricle and diminished resistance to the reflux of blood from the ventricle, in expiration, positive pressure and increased resistance to reflux; and the systole will be short and sharp, or prolonged, accordingly: this is another obvious source of irregularity. The final result is that no two beats of the pulse are alike, either in strength, or duration, or interval.

The pulse, besides being irregular, is easily compressible, short, and unsustained; that is, the tension is low. (Fig. 52.)

As in the case of other valvular diseases, the pulse

enters into the diagnosis and prognosis of mitral insufficiency, but not in the same degree as in aortic insufficiency. A systolic apex murmur conducted to the left and audible in the back may attend either slight or free regurgitation, and of itself gives no information as to the amount of blood which escapes back into the auricle. This, which is a part of the diagnosis and a very important element in the prognosis, is determined upon other considerations, among which is the character of the pulse, together with the hypertrophy and dilatation of the different chambers of the heart and the accentuation of the pulmonary second sound.

CHAPTER XII.

THE PULSE IN STRUCTURAL DISEASE OF THE HEART.

In hypertrophy.—There is no form of pulse which can be said to be characteristic of hypertrophy of the heart. This condition has its origin in some kind of over-work of the heart, occasionally in excessive muscular exercise or sustained effort, but usually in some obstacle in the systemic or pulmonary circulation which demands additional contractile energy on the part of the corresponding ventricle. In the pulmonary circulation the chief causes of obstruction are bronchitis and emphysema and disease in the left side of the heart. In the systemic circulation, with which we are concerned when speaking of the pulse, the obstruction may be at the aortic orifice, very rarely in the course of great vessels—most commonly at the periphery in the capillaries and arterioles. When, therefore, the heart is hypertrophied the pulse will be that of the condition which has given rise to the hypertrophy, modified by the increased contractile energy of the heart.

If the hypertrophy is caused by aortic stenosis, the pulse will be regular, small, long, and firm under compression; if by aortic insufficiency, large and short, and more or less collapsing.

If, as sometimes, though rarely, happens, there is no assignable cause for great hypertrophy, except adhesion of the pericardium, the pulse will present nothing characteristic.

When the case is one of peripheral obstruction and high arterial tension the artery may be small or large, its coats normal or more commonly thickened; but it will be full between the beats, and can be rolled

under the finger, and, until the heart and vessels have suffered serious damage and degeneration, the pulse-wave will be deliberate, sustained, and not easily arrested by pressure.

When the arteries have lost their elasticity, are thick, leathery to the feel, presenting bulgings and irregularities in their walls and tortuosity in their course, while the heart, if not dilated, has become fibroid, the pulse, while the vessel is full between the beats and capable of being traced up the fore-arm nearly to the elbow, will be abrupt both in its onset and cessation.

The pulse of dilatation of the heart.—Dilatation of the left ventricle, which will alone be here considered, may, like hypertrophy, have various causes. Among them are aortic and mitral insufficiency, but these have been sufficiently dealt with elsewhere.

Dilatation, being at the same time an effect and evidence of cardiac weakness, the pulse will generally be weak, but it may strike the finger sharply and give a deceptive impression of vigour; there is, again, a general tendency to irregularity in the pulse of dilatation.

One cause of this condition is premature exertion after acute disease. In acute rheumatism the muscular substance of the heart rapidly loses tone, and probably in many cases there is more or less myocarditis; if the patient is allowed to sit up and walk about too soon dilatation may easily be established. This is particularly liable to occur after pericarditis, when a certain thickness of the muscular fibres is paralysed by the inflammation of the serous covering and infiltrated with the inflammatory products.

In enteric fever the heart is not only exhausted and its nutrition impaired by the protracted pyrexia, but granular degeneration takes place, and many

instances of sudden death during convalescence have occurred simply from the patient sitting up in bed. It will easily be understood that dilatation may follow premature exertion under such circumstances.

Acute rheumatism and typhoid fever are the diseases which are most frequently followed by this form of heart damage, but it may be induced after any acute febrile affection in poor constitutions or badly-nourished subjects.

The pulse of dilatation produced in the way described is usually weak, short and irregular, unless in the course of time compensatory hypertrophy has been brought about by care and an improved state of health : the artery may be small or large. The pulse is perhaps most likely to be irregular after rheumatic fever.

When dilatation of the heart takes place under other circumstances, antecedent high arterial tension usually plays a part in its production. This is the case, for example, in the dilatation which sometimes occurs at the beginning of acute renal dropsy. Slight dilatation is not uncommon in the first few days of this affection, and when sufferers go about their work for some days before seeking admission into hospital it may reach a considerable degree. Here the factors in the causation are resistance in the capillaries and arterioles, due, as has been before stated, to retained nitrogenised matters, increase of the volume of the blood by water which ought to have escaped in the urine, and impaired nutrition of the walls of the heart.

In acute dilatation, again, from violent exertion or from exertion which, without being excessive in point of violence, is unduly protracted, and, perhaps, repeated, as in training for races or other athletics, in all the cases which have come under my observation there has been undue resistance in the peripheral

circulation whether the patient has been old or young. After middle age a single imprudence, such as hurrying to catch a train, climbing too steep and too long a hill, even running upstairs may break down and dilate the left ventricle; this is much more likely to occur when a weight of some kind, such as a bag, is carried, and in my experience the factor of high tension has very rarely been absent. In the young, brief exertion, however violent, does not produce a permanent dilatation, as the heart speedily recovers itself after being over-distended; but this effect may follow effort carried to the point of exhaustion and especially when renewed day after day.

In anæmia, which, as has already been stated, is often attended with high pulse-tension, dilatation of the heart is not uncommon, and it may take place suddenly or gradually. The breathlessness attending this state of the blood may be due not merely to the defective carriers of oxygen to the nerve-centres, but also to this state of the heart.

It may be worth mentioning that I have twice seen acute dilatation of the left ventricle after mumps in young men, without apparent cause, except that there was marked arterial tension; in one it was renewed and kept up by comparatively slight exertion, and threatened to be very serious.

Dilatation of the heart is produced suddenly more frequently than is generally supposed, but it usually comes on gradually in the subjects of chronic arterial tension. Very often there is antecedent hypertrophy, which after a time fails to cope with the resistance in the arterio-capillary network, as fibroid change or fatty degeneration invades the muscular fibres of the cardiac walls. In other cases the heart is from the first incapable of developing compensatory hypertrophy as obstruction in the peripheral circulation increases, and primary dilatation is the result.

Occasionally, when dilatation has been thus established in the first instance, improvement in the health and changed conditions of life, aided perhaps by treatment, may bring about secondary hypertrophy.

It will be seen from these considerations that the pulse of dilatation will vary greatly, not only according as the dilatation is slight or great, but according as it is the simple, direct effect of weakness or degeneration in the muscular walls of the heart, or is superinduced upon antecedent hypertrophy, or is more or less neutralised by subsequent compensatory hypertrophy. Since high arterial tension plays so large a part in the production of the cardiac change, the artery will almost always be full between the beats; but, in proportion as the dilatation has impaired the propulsive power of the heart, it will yield to the pressure of the finger, and that feature of genuine high tension—the feeling that the greater the compression, the stronger the pulse—will be absent. Other effects of the dilatation will be that the systolic pressure in the arteries is not sustained, the pulse-wave begins and ends more or less abruptly—the tension is virtual not actual. Added to these characters may be some degree of inequality in the force of individual beats, and in many cases irregularity as regards time. Another feature will be, that while the pulse may be regular, strong, and sustained, and apparently in all respects a good pulse in repose, slight exertion will develop in a marked degree the characteristic evidences in the pulse of the cardiac weakness—frequency, irregularity, shortness, and compressibility both during and between the beats.

Fig. 53 is a trace from a case in which acute dilatation with hæmoptysis had occurred from overexertion; a mitral systolic murmur remained; the artery was not large.

In the case from which the trace in Fig. 54 was

taken here were breathlessness, difficulty of lying down, and enlargement of the liver, all of which were

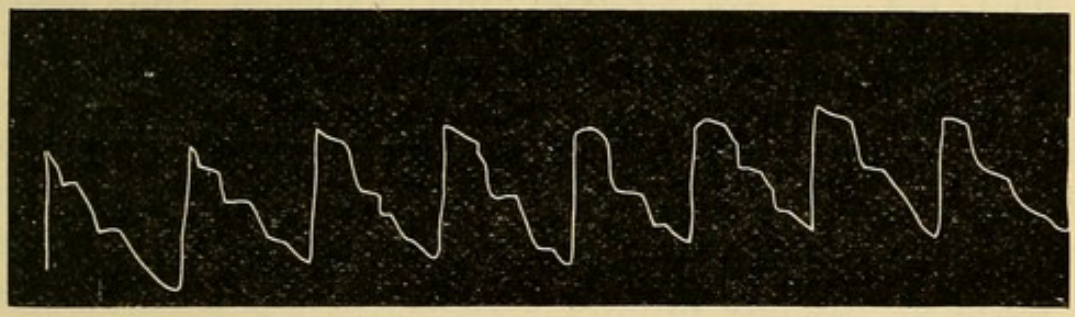


Fig. 53.

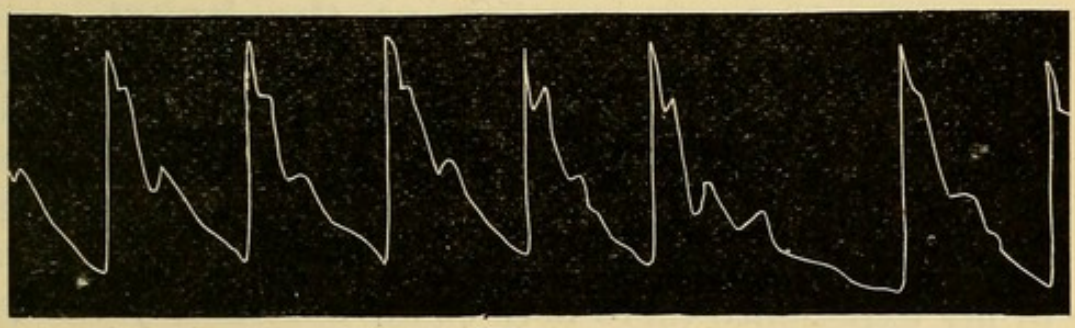


Fig. 54.

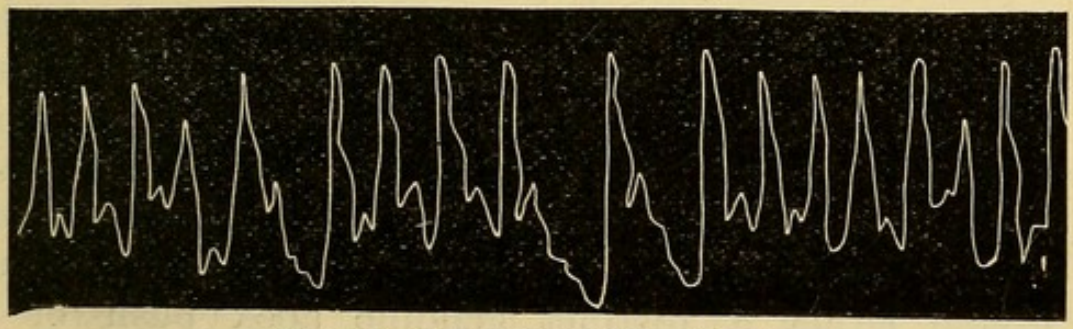


Fig. 55.—Dilatation of the Heart.

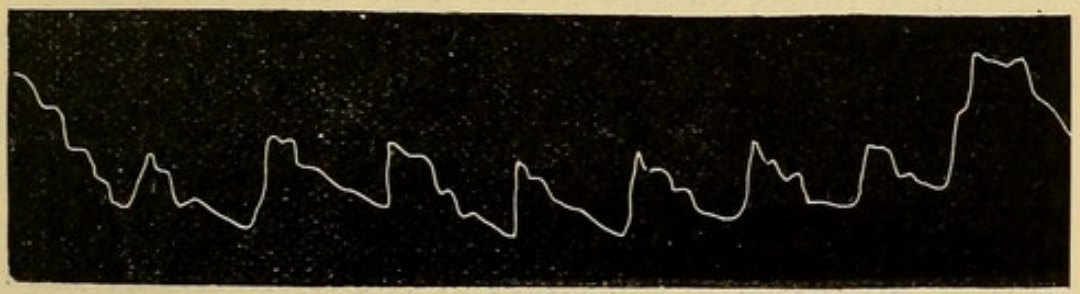


Fig. 56.—Same Case (Fig. 55) after Treatment. Pulse of Dilatation of the Heart.

relieved by treatment. The patient whose pulse is represented by the trace in Fig. 55 was liable to stagger and even fall on rising from a semi-recumbent

position, with momentary loss of consciousness, and on slight exertion he would suddenly lose all power. The artery was always full between the beats, notwithstanding the appearance of the trace. He improved under treatment, could walk eight miles, was more cheerful, and could lie on his side; the pulse then gave the second trace. (Fig. 56.) Later, over-exertion aggravated the dilatation, and thrombosis of the left middle cerebral artery occurred, the case ending fatally.

In fatty degeneration of the heart. — A very infrequent pulse has been said to be highly characteristic of fatty change in the muscular substance of the heart. There is, of course, foundation for this statement, and some justification for the expression, "the slow pulse of fatty degeneration"; but in my experience there may be advanced fatty change without marked slowing of the heart's action, and on the other hand extreme infrequency of the pulse without disease of this character in the heart. The pulse-rate varies in fatty heart, and the rhythm may be regular or irregular, the arteries may be soft and apparently healthy, or they may be in a state of advanced degeneration. A fatty heart is incapable of giving either a distinct genuine apex push or a sustained pulse, except in the rare case of degeneration affecting the right ventricle only. The pulse, therefore, which would tend to confirm a suspicion of fatty degeneration of the heart would be weak, short, and compressible, and the significance of such a pulse would be greater were it infrequent. The great criterion, however, is the effect of slight exertion; if the heart is only functionally weak it will respond, and the pulse will improve; if, on the other hand, it is in an advanced stage of fatty change, it cannot accommodate itself to the least increase of work, and its action becomes irregular and faltering.

CHAPTER XIII.

THE PULSE IN ANEURISM.

DIFFERENCE between the radial pulse of the two sides has long been recognised as one of the most important signs of thoracic aneurism, and here it must be stated that the sphygmograph is of very great service in bringing out and defining the specific difference produced by aneurism between the right and left pulses, and that its indications precede in point of time, and are more delicate and, perhaps, more trustworthy than, those recognisable by the educated finger, although they are very rarely belied. Inequality in the pulse of the carotids and of their branches will often corroborate and supplement, and occasionally correct the inferences derived from differences between the radial pulse. The femoral pulse, again, may afford valuable information with regard to aneurism of the abdominal artery and its branches.

The radial pulse may be different on the two sides from other causes than aneurism, and these must be specified.

Difference in the size of the two arteries at the point where the pulse is examined is the most common. Sometimes it is that the radial artery of one fore-arm is smaller than the other in its whole length and the ulnar larger by way of compensation; at others the radial artery turns round prematurely to the dorsal aspect of the limb, and is represented at the wrist by a branch, the superficialis volæ. An apparent difference may result in another way from the artery being more superficial on one side than on the other, or from the presence of a curve in the vessel in one wrist and not in the other.

A real difference in the pulse of the two sides, simulating more closely than any of the above the effect of aneurism, may be produced by the pressure of a tumour upon any part of the arterial channel, of which the radial is a branch—subclavian, axillary, or brachial.

The modification of the pulse caused by aneurism is not merely a change in the diameter of the vessel or in the strength of the beat—there is an alteration in the character of the pulse-wave, as will be described shortly ; but, when the issue is so grave as that involved in a diagnosis of aneurism, all possible sources of error must be considered and excluded. When, therefore, a suspicion is excited by difference of the pulse on the two sides, the causes of such difference above enumerated must be borne in mind, as must also the possibility of pressure upon one or other artery due to the position of the limb. Such a position, for example, as will stretch the subclavian over the first rib or will compress the axillary against the body or across the arm of a chair or partially obliterate the brachial by flexion of the elbow.

A passing remark may here be permitted. It ought to be a routine practice to examine the pulse at both wrists, not only because a careful investigation may be suggested by which an aneurism of the aorta or intrathoracic tumour may now and then be detected, but for the purpose of acquiring familiarity with such differences between the two sides as are common and devoid of significance. Not unfrequently it is the pulse of one side only which can be depended upon as indicating the state of the patient, and if, in the course of acute illness, one of the two different pulses is felt one day and the other on another, without knowledge of the difference between them, the most erroneous conclusions may be drawn.

Coming back now to the pulse of aneurism, it must first be observed that, in order to give rise to

difference in the two radial pulses, a thoracic aneurism must be so situate as to affect differently the two subclavians. An aneurism at the root of the aorta, or in the ascending aorta, or even in the first part of the arch, need not cause any difference between the pulse on the two sides. Aneurism of the first part of the arch will have this effect when the innominate is directly or indirectly implicated, as will aneurism of the innominate artery itself, and in such cases the chief modification will be in the right pulse. It is, however, in aneurism of the transverse and descending arch that a difference is most frequently felt between the two pulses, that of the left side being affected, and it often constitutes an important, sometimes almost a determining element in the diagnosis. There is a great difference between aneurism of the ascending aorta and of the arch, both as to the effects which they produce and as to the indications by means of which they are recognised. I have long been in the habit of calling the former the "aneurism of physical signs," the latter the "aneurism of symptoms." The ascending aorta is freely movable, and does, in fact, move up and down—or, to speak more exactly, down and up—with each beat of the heart; it probably expands and contracts also more than any other part of the vessel. Accordingly, provision exists for its free play; it is not closely attached to surrounding parts, and is not in close relation with the nearest important structures, the root of the lung and the vena cava superior and the pneumogastric nerve. In this way it comes to pass that an aneurism of the ascending aorta may attain a considerable size before pressure-effects, or, in other words, symptoms, are produced. On the other hand, this part of the aorta is near the surface of the chest, an expansion of the vessel here very often takes a superficial direction upwards and outwards, and in whatever direction it

takes place can scarcely fail to produce actual contact with the chest-wall; the aneurism, therefore, reveals itself by dullness, pulsation and characteristic auscultation signs.

The aortic arch, especially the transverse and descending part, presents a complete contrast of anatomical conditions to those just enumerated. It has little freedom of movement, is closely related with important parts—the trachea and œsophagus, the root of the left lung, the innominate veins—while the cardiac plexus of nerves, and the branches of the pneumogastric and sympathetic, which form it, may almost be said to ramify upon it, and the left recurrent laryngeal nerve actually winds round it. Again, crossing as it does almost directly backwards, it recedes from the surface of the chest. Pressure-effects or symptoms—pain, paralysis of the laryngeal muscles, dyspnœa, blocking of the great veins, etc.—thus appear at an early period of the disease, before the aneurism has attained a size which is appreciable by percussion, or has reached the surface so as to communicate pulsatile movement. It will be evident that a characteristic modification of the left radial or carotid pulse will be a critical indication in doubtful cases when symptoms are present which may or may not be due to aneurism.

An aneurism may interfere with the flow of blood through one of the main branches given off from the aortic arch in four ways:—

1. By the interposition of a sac which expands under the systolic increase of pressure and receives part of the blood injected by the heart, diverting it for the moment from the current in the aorta, and delivering it gradually afterwards so as to render the stream more continuous.

2. By the branch or branches being given off from the aneurismal sac itself.

3. By pressure upon the vessel by the sac; not unfrequently one of the branches of the aorta will run for a longer or shorter distance in the wall of the aneurism.

4. By simple narrowing of the mouth of the branch at its orifice.

The general tendency is the same in all: namely, to intercept the systolic ictus and to retard and smooth down the pulse-wave. The degree of modification varies greatly; in some cases this is carried so far that the flow of blood in the radial and other small branches becomes almost equably continuous, the variations of pressure which give the pulse being obliterated, and the beat being scarcely perceptible, while the artery is continually full.

The interposition of the aneurismal sac, acting the part or imitating the effect of a blacksmith's bellows, by receiving a considerable proportion of the blood propelled into the aorta by the ventricular systole, and thus annulling or diverting the pressure-wave normally transmitted onwards to the remotest branches, the special and characteristic effect of which is supposed to be the modification of the pulse just described, is, according to my experience, less frequently its cause than one or other of the remaining conditions enumerated, the origin of a branch from the sac, and obstruction by pressure or narrowing of the orifice. When the artery comes off from the aneurism itself or is implicated in the wall of the sac it may be completely obliterated, so that the circulation in the parts which it supplies is entirely collateral.

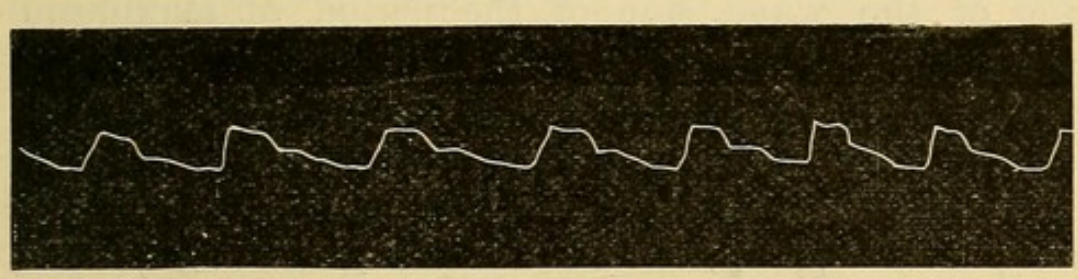
Sometimes an effect upon the pulse is attributed directly to the aneurism as an elastic reservoir, which is produced indirectly by interference with the full and free flow of blood through a branch. A remarkable case came under my notice some years since, in which, without aneurism, a simple narrowing of the

mouth of the innominate and subclavian arteries, with enlargement of these vessels near their origin from the aorta, almost completely annulled pulsation in both radials. The patient was admitted into St. Mary's Hospital suffering from cirrhosis of the liver, and it was at once observed that he had no pulse in either wrist, except under excitement or on exertion, when it could just be detected, and scarcely perceptible pulsation in the brachials or carotids. At the same time, the arteries were full and the circulation in the hands good. There was forcible pulsation in all the arteries of both lower extremities. To add to the difficulties of the case, the absence of the pulse had been first observed after a railway accident many years previously, and had been taken into account in the compensation awarded. No signs or symptoms of aneurism existed, and it turned out, on post-mortem examination, that the cause was simply a constriction at the mouth of each of the branches given off from the arch of the aorta with a small ampulla just above.

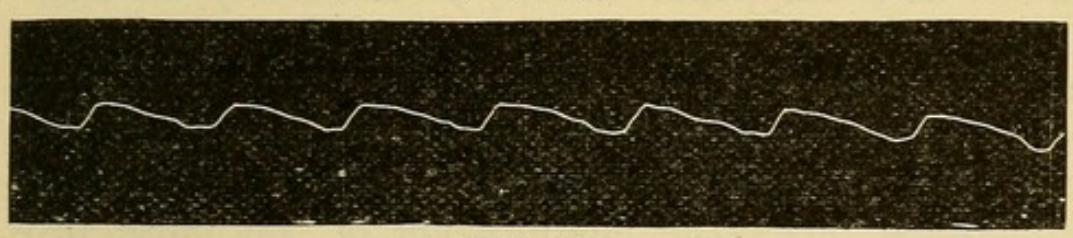
In comparing the pulse of the two sides with a view to the diagnosis of aneurism, the most important point to which attention must be given is whether they are absolutely synchronous or not; the most significant indication of aneurismal interference with the pulse is delay of the beat. To appreciate this the two pulses must, of course, be felt simultaneously; and it is well to test the first impressions by varying the position of the patient's hands, raising and lowering them, and by examining each pulse with both hands, *i.e.* after feeling the right pulse with the left fingers and the left pulse with the right, sitting in front of the patient, to cross the hands either of the patient or of the observer so as to feel each pulse with the corresponding hand, or to stand behind the patient for the same purpose. With the delay on

the affected side there will usually be a smaller artery and a more compressible pulse, and the vessel will be more continuously full between the beats.

Case 1.

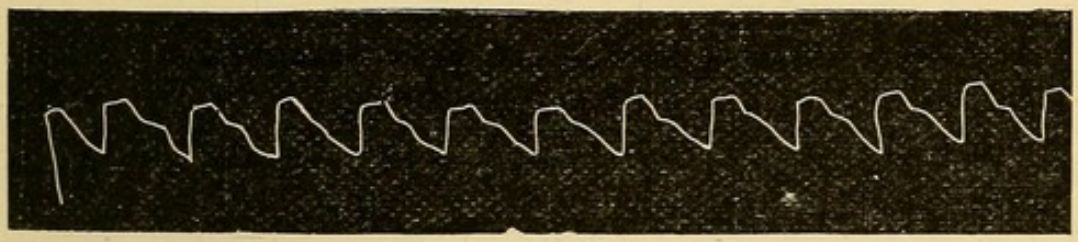


Left Side.

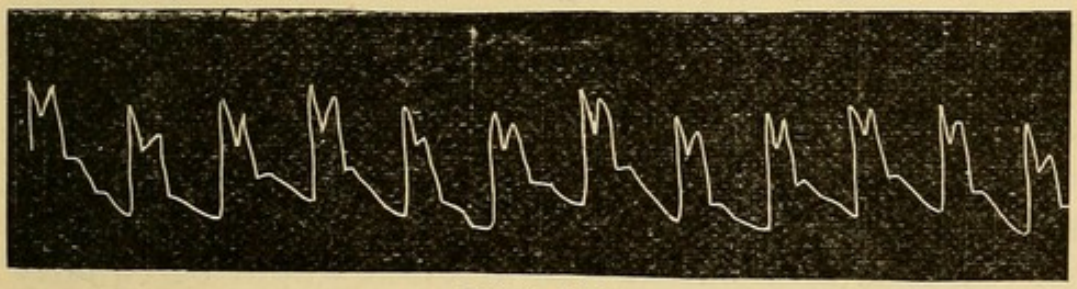


Right Side

Case 2.



Left Side.



Right Side.

Fig. 57.—Pulse in Aneurism.

As has been already said, the sphygmograph is here more definite and delicate in its indications than the pulse as felt in the usual way. (Fig. 57.) The upstroke of the trace is more sloping, the height less, the summit more rounded, and the fall more gradual ;

and the trace shows minute changes in the character of the pulse which are not appreciable by the finger. It shows, moreover, as pointed out by Marey, that the delay of the beat is not any retardation of the beginning of the wave, but of the period of maximum pressure.

Applying these conclusions to the effects on the pulse of aneurisms of different parts of the aorta.

1. *Aneurism of the ascending aorta* will affect all the pulses equally, unless it is large enough or so situate as to implicate directly or indirectly the innominate artery. Occasionally, the right pulse seems to be stronger and better than the left. There being no difference between the pulse of the two sides, and thus no comparison between a normal and a modified pulse, the modification of the pulse produced by aneurism may be scarcely appreciable, even when it is very large, especially if much laminated clot has been deposited. In some cases the wave is more or less smoothed down, as already described.

When aneurism of the ascending aorta involves the innominate, or when the aneurism springs from the first part of the arch, the right radial pulse will be modified, and, as compared with the left, it will probably seem to be slightly retarded and longer, but smaller and more compressible, while the artery will be more continuously full between the beats. An examination of the carotids ought to show a corresponding modification of the right carotid pulse, since the carotid and subclavian arteries would be equally affected by interference with the innominata.

2. *Aneurism of the innominate artery*, whether by extension from the aorta, which is a frequent event, or of independent origin, will usually affect the pulse of the carotid and of the radial, and in much the same degree; but one of the two divisions may be implicated more than the other, and such a

difference might have diagnostic importance as between innominate aneurism and a sac projecting into the neck from the aorta.

Occasionally aneurism of the arteria innominata is simulated by an abnormal origin and course of this artery, which, arising from the aorta farther along the arch, crosses over the root of the neck in front of the trachea; or it may be the subclavian (rarely the carotid) which, springing independently from the arch of the aorta, takes this course. Sometimes the diagnosis between such an abnormality and aneurism is really difficult, and the pulse may afford valuable aid in establishing the distinction. It will not be affected when there is no aneurism.

3. *Aneurism of the transverse part of the arch.*— This may involve the innominate artery, on the one hand, or the left carotid, with or without the subclavian, on the other. Occasionally, though rarely, aneurism of the arch may be large enough to implicate all the three great branches. Either right or left pulse, therefore, may be affected to a greater or less degree, or both. It is when a small aneurism of the third part of the arch or of the descending aorta implicates the left subclavian artery and gives rise to distinct delay in the left pulse, with other of the characteristic modifications enumerated, that inequality of the two radial pulses has its maximum diagnostic importance. There may be no other physical sign of any kind, and the pulse may be the only indication which gives a definite interpretation to a group of symptoms which might otherwise be uncertain and perplexing in their indications.

CHAPTER XIV.

THE PULSE IN KIDNEY DISEASE.

So constant is the association of a pulse of high tension with renal disease, that it has sometimes been called the "renal pulse." This, however, is extremely objectionable, for although almost every form of disease of the kidneys, except suppurative inflammation, is attended with high arterial tension, and although the cord-like artery and persistent wave, when well marked, are extremely suggestive of renal disease, these characters of the pulse are common enough when no affection of the kidneys is present.

The Pulse in Contracted Granular Kidney.

It is in the contracted granular condition of the kidney—the disease to which the name "chronic Bright's" is usually applied—that high arterial tension is most common and best marked : the artery contracted and cord-like, full between the beats, and capable of being rolled under the finger and followed up the fore-arm, while the pulsatile movement is deliberate and inconspicuous, but arrested only by great pressure.

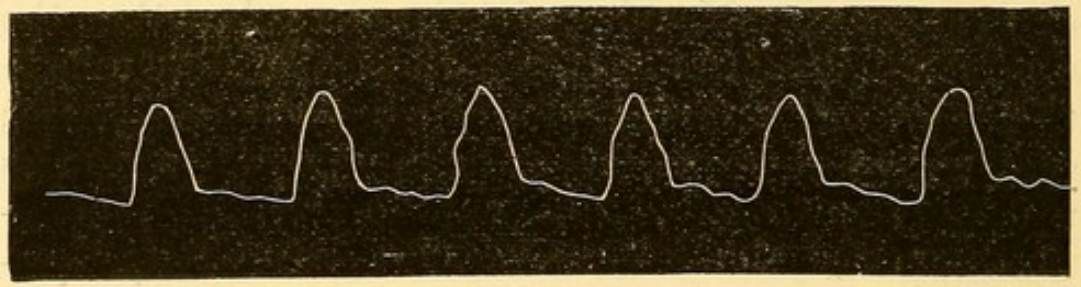
It is with regard to this state of the pulse that so much controversy has arisen. The hard pulse and thickened arteries had been early noted when Dr. George Johnson advanced his well-known theory to explain these facts. This, put briefly, is that impurities, which ought to have been eliminated by the kidneys, being retained in the blood, act as an irritant to the tissues, and provoke a reflex contraction of the minute arterioles, which is protective of the tissues by shutting off, in some measure, the contaminated blood. At the same time, the narrowing

of the arterioles gives rise to obstruction, which dams back the blood in the arterial system and causes the blood pressure to be high. But the distending influence of the increased blood pressure in the arterioles will necessitate increased contraction of the muscular walls of the vessels, and the persistent exercise of contractile energy demanded by the persistence of the cause leads to hypertrophy of the muscular coat of the arteries. Nothing could be more clear than the demonstration of the increase of muscular fibre-cells in the thickened arterioles of chronic Bright's Disease; and except that, in my opinion, the obstruction is primarily in the capillaries, and the arteriole contraction secondary to this, Dr. Johnson's theory commands my entire adhesion.

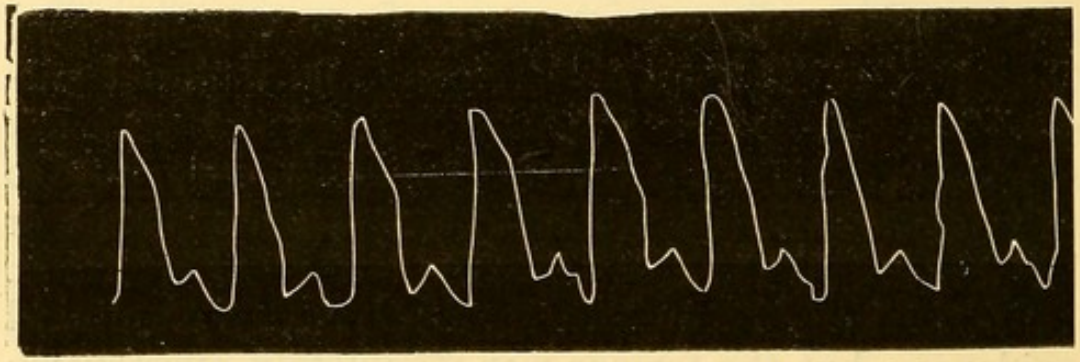
The opposing theory is that of Sir William Gull and Dr. Sutton, according to which the increased thickness of the walls of the vessels is a fibroid substitution and not true hypertrophy of the muscular structure, and the arterial tension is due, not to arteriole contraction but to arterio-capillary fibrosis or hyaline fibroid change in the capillaries and arterioles. It is based partly on microscopical, partly on clinical evidence; but the former was completely overthrown by Dr. Johnson, who showed that the hyaline fibroid appearance was producible by the method of preparation employed. A certain degree of fibroid change is, no doubt, present together with the hypertrophy of the muscular fibres, and late in the disease the muscular fibres undergo more or less degeneration, allowing the fibrosis to predominate; but this, in my judgment, is all that subsequent investigations have established.

The clinical evidence adduced is to the effect that the renal changes in Bright's Disease are not primary, but are led up to by a long train of symptoms due to general degenerative change in the arterioles and capillaries. It is, no doubt, true that before albumen

appears in the urine, and before the change in the kidneys has reached a point which could affect the health, almost before the morbid condition of these organs is recognisable on examination after death, there may be loss of flesh and strength, headache and depression, impairment of appetite and digestion, and other indications of failing health. But evidence of



Tracing taken just before Inhalation of Nitrite of Amyl. Patient had chronic Bright's Disease.

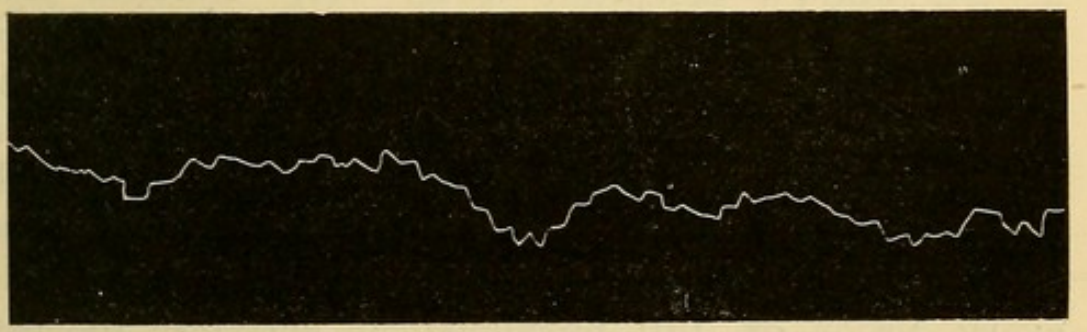


Tracing taken about two minutes after Inhalation of Nitrite of Amyl.

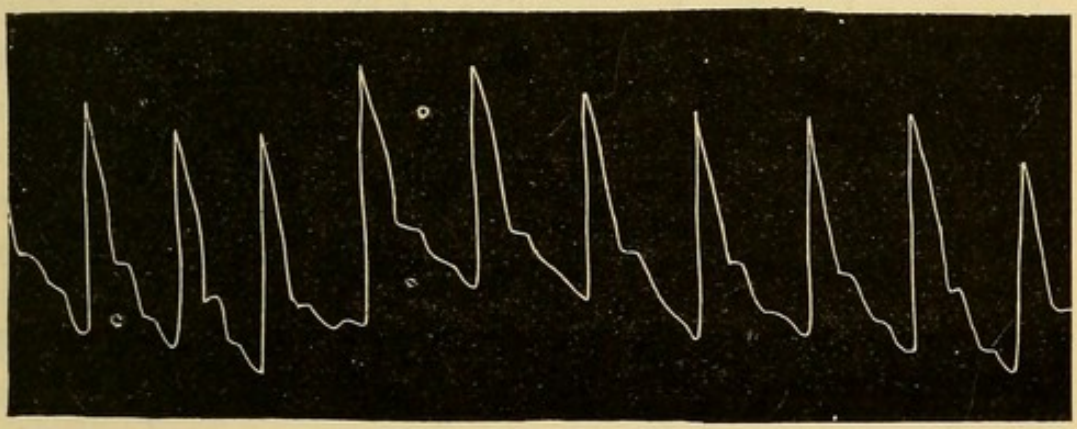
Fig. 58.—Effects of Nitrite of Amyl in Bright's Disease.

this kind is capable of another interpretation, namely, that these symptoms are due, if not to the high arterial tension itself, yet to disturbance of the normal relation between the blood and tissues which provokes resistance to the circulation in the capillaries, and constitutes the real antecedent of the renal disease. When it is considered how minute a proportion of various drugs may affect in one direction or another the freedom of the peripheral flow of blood, it can scarcely be denied that impurities due to imperfect metamorphosis or insufficient elimination may give rise

to obstruction in the capillaries and arterioles. There is, further, the simple experiment to which appeal was made when the question was under discussion before the Medico-Chirurgical Society. If the change which gives rise to resistance in the arterioles and capillaries is degenerative or hyaline fibroid, it must interfere with the contraction and relaxation of which these



Morb. Br. Anasarca, albuminuria. No cardiac murmur. Pulse very small and firm; radial tortuous.



Took calomel gr. v.; has had some diarrhœa since. Feels much better. Urine contains less albumen.

Fig. 59.—Effects of Calomel.

vessels are normally capable. Relaxation, especially, should be slow and imperfect. Let, then, nitrite of amyl be administered to the subject of this supposed fibrosis; if the physiological effects are produced, the muscular fibres of the arterioles and the contractile element in the capillary wall cannot well have undergone structural change or degeneration. This test has been applied and always with the same result: the full effects of the nitrite are manifested. (Fig. 58.)

Other physiological relaxants of the peripheral vessels also have their normal influence ; nitro-glycerine is, indeed, employed therapeutically in kidney disease. A mercurial aperient will lower the arterial tension, as may be seen by the accompanying traces before and after a dose of calomel, sent to me by Dr. Handfield Jones many years ago, when my senior at St. Mary's Hospital. (Fig. 59.) Pyrexia also relaxes the arterioles and capillaries, and it is a common experience that a patient suffering from contracted granular disease of the kidneys, whose pulse exhibits habitually all the characters of high tension, will on the supervention of pyrexia at once have a large, soft, and dicrotous pulse. We have thus every proof of full physiological activity in the muscular coats of the arterioles, and they cannot, therefore, have undergone fibroid change.

The hypothesis of a general hyaline fibroid degeneration, of which the cirrhosis of the kidney is only a part or a consequence, appears to me to have no foundation in fact, and to survive only in virtue of the great and merited reputation of its authors, and of the euphonious terms which they introduced.

It will, then, be taken as proved that the high arterial tension of contracted granular kidney is due to arterio-capillary contraction and not to arterio-capillary fibrosis or degeneration, the contraction being provoked by the presence in the blood of some matter which acts as an irritant. The fact that increased blood pressure often precedes the kidney mischief shows that it is not due purely and simply to deficient renal elimination, but it cannot be doubted that, when disease of the kidneys is established, the retention in the blood of waste products which ought to have passed out of the system by these organs adds to opposition in the capillaries, and becomes an important factor, perhaps the most considerable factor, in

the production of the high arterial tension. Additional certainty is given to this conclusion by the fact that other affections of the kidneys at once give rise to increased blood pressure, and that it is in chronic Bright's Disease that arterial tension reaches its maximum.

Co-operating with the arterio-capillary resistance to produce the renal pulse, so-called, is an increased propulsive power of the heart.

The gradual advance of the renal changes and of the peripheral obstruction to the circulation in chronic Bright's Disease affords time for the heart to accommodate itself to the increased work thrown upon it, and to meet the resistance in the arterio-capillary network by hypertrophy. That the hypertrophy is a real increase of the cardiac muscular fibres and not merely an addition of adventitious fibrous tissue there can be no manner of doubt; it is demonstrated by the microscope and proved by the increase of functional energy. In the late stages of the disease, when the heart is worn out, an excess of fibroid material is present and the proportion at all periods will vary according to individual tendencies and mode of life—it may be larger, for example, in cases of alcoholism—but the characteristic change in the heart is true muscular hypertrophy, the result of excessive functional exercise.

The apex beat is displaced downwards to the sixth or even to the seventh space and carried somewhat outwards; it is a genuine thrust and not a mere shock, and the cardiac impulse generally is powerful. The first sound, as heard at the apex, is dull and prolonged, while over the aortic area it is scarcely, if at all, audible. At an advanced period of the disease, when the heart has begun to suffer from the effects of protracted over-work, and in some cases throughout, the first sound is reduplicated over a larger or smaller area near the apex. The aortic second sound is loud

and accentuated both in the right second space and at and to the left of the apex.

It would almost appear from the considerations stated that the heart and vessels were engaged in a work of mutual destruction, and such is indeed the fact, as is testified by cerebral hæmorrhage and valvular and structural disease of the heart. It is, however, probable that other evils are averted which would prove fatal sooner, and that high arterial tension is really the result of a defensive reaction. Cases will be related later in which kidney disease with low arterial tension has run a rapid course, but for the present we may try to discover in what ways arterial tension may be conservative.

The general condition of the circulation will at any rate have the effect of keeping up a rapid flow of blood through the kidneys ; this must result from the increased driving power of the heart and from the contraction of the minute arterioles and the resistance in the general systemic capillaries. Now, the transmission of an increased amount of blood through the kidneys gives rise to an increased flow of urine which will in some degree compensate for the diminished eliminative action of these organs ; and, in effect, the low specific gravity and diminished urea of the urine in contracted kidney are more or less neutralised by the polyuria.

Another conservative effect of the rapid passage of blood through the kidneys resulting from the high arterial tension and hypertrophied heart will be the diminished tendency to albuminuria. It would seem *à priori* that the increased blood pressure would tend to promote transudation of albumen from the malpighian glomeruli ; but all clinical evidence is against this, and experiment has shown that the condition which determines the passage of albumen from the tufts of capillaries into the tubes is not high or low pressure,

but stasis or slow movement of the blood in them. Variations of pressure influence it only as they tend to produce stasis. Now, it is obvious that the morbid change in the kidney which causes the contraction, whether it is denudation of the tubes or cirrhosis of the intertubular cement, will tend to obstruct the capillaries ramifying between the tubes and to give rise to stasis of blood in the glomeruli. This the high pressure and rapid circulation will tend to obviate, and thus to prevent albuminuria.

The conservative effect of sustained blood pressure in the prevention of albuminuria in chronic Bright's Disease is illustrated when from any cause the propulsive power of the heart is impaired, or when the peripheral resistance is diminished, as by pyrexia. As the heart gets worn out and dilatation or degeneration supervenes upon hypertrophy, in proportion as it fails to maintain the arterial tension at a high point, the amount of albumen increases in the urine, and it is slackening of the circulation and not aggravation of the kidney disease which gives rise to this increase. Again, it is not uncommon for acute dilatation of the heart to occur in course of Bright's Disease in consequence of effort or sustained exertion; or without recognisable event of this kind, the heart may become weak from insufficient food, alcoholic excess, anxiety, or impaired health. The diminished vigour of the circulation is at once attended with increase of albumen in the urine. Pyrexia, again, from whatever cause, will give rise to an aggravation of this albuminuria; the relaxation of the arterioles and capillaries lowers the general arterial tension and thus slackens the current of blood in the renal vessels. The sudden increase of albumen in the urine due to cardiac weakness or to pyrexia is often attributed to intercurrent tubular nephritis.

More striking still is the effect of bronchitis in

increasing the amount of albumen in the urine when the kidneys are diseased, and in precipitating the appearance of dropsy. In addition to the pyrexial influence on the general circulation there is obstruction to the passage of blood through the pulmonary capillaries, which at the same time tends to dam back the blood in the systemic veins and diminishes the supply of blood to the left ventricle.

The characteristic pulse and heart sounds of contracted granular kidney have been fully described, and the modifications of these which attend the downward progress and attest the injurious effects of the disease may now be traced.

Beginning with the pulse, the walls of the arteries become thickened, and when the blood is excluded from the radial by pressure, and the fingers are made to carry the skin to and fro along it, the vessel has a leathery, inelastic feel, and may present indurations, inequalities, and bulgings. It is large, also, as if it had yielded to the distending force of the protracted blood pressure within it. The muscular coat is worn out, and is more or less replaced by fibroid structure. In proportion as the muscular coat of the small arteries has lost its contractile power there will be loss of control over the blood supply to different parts, and in proportion as the arteries generally have lost their elasticity there will be a change in the character of the pulse-wave, which will become more sudden.

In the heart, reduplication of the first sound will become more distinct, and the interval between the first and second sound prolonged. The apex-beat loses in vigour and precision, and becomes diffuse and slapping.

As these changes in the pulse and heart take place, symptoms referable to weakness and disorder of the circulation supervene:—Sleeplessness from loss of

tone and power in the cerebral vessels, which are unable to co-operate in the production of the anæmia of the cortex, essential to sleep, by contracting and shutting off the blood from the brain; breathlessness on exertion and palpitation of the heart; paroxysmal dyspnœa, often extremely severe, occurring chiefly in the night; a form of cardiac asthma; fugitive amaurosis in some very rare instances; œdema of the lower extremities.

It is when the heart is no longer competent to cope with the resistance in the periphery that uræmic phenomena are liable to set in, and it appears to me that disorder in the cerebral circulation, associated with, and in some way resulting from, high arterial tension, constitutes the connecting link between the contamination of the blood and the convulsions, which are the most striking and characteristic effects of uræmia.

When the connection between renal disease and convulsions was ascertained, it was a most natural and plausible idea to attribute the convulsions to the direct action upon the brain of the urinary constituents retained in the blood, and expression was given to this theory in the term "uræmic" applied to the entire group of symptoms. From the first, however, a difficulty was recognised in the capricious incidence of the convulsive attacks. It was seen that they occurred early in some cases when comparatively little poison could have been accumulated, late or not at all in others when the blood must be charged with it: the convulsions appeared, in effect, to have no relation with the amount of urinous impurities present in the system, or with the general deterioration of the blood and tissues. The difficulty was only shifted, not removed, by supposing that the poison which gave rise to convulsions was not urea or uric acid, or any of the normal constituents of urine, but

ammonia, derived from the decomposition of urea or some nitrogenised waste of a lower degree of oxidation than urea or uric acid which was imperfectly oxidised and broken up, because the blood was already charged with products of combustion.

Experimental investigation rendered the idea that uræmic symptoms were due to urea altogether untenable. Urea injected into the blood, in whatever quantity, did not give rise to convulsions, and when convulsions resulted from some experimental interference with the renal excretion there was no abnormal amount of urea in the blood. The same conclusion was reached when the hypothesis that the symptoms were due not to urea, but to the products of its ammoniacal decomposition in the blood or tissues was experimentally tested. First one substance, then another obtained from the urine, has been considered by different experimenters to be the toxic agent to which the convulsions and other uræmic symptoms were attributable. It seemed, indeed, to be almost a *reductio ad absurdum* when the potash salts were found, after most careful and skilful and apparently exhaustive investigation by Feltz and Ritter, to be the most powerfully toxic of the constituents of urine.

If the theory of Traube, which refers the convulsions and other uræmic symptoms to cerebral anæmia, the result of œdema, be slightly modified, it seems to me that all the phenomena fall within the range of its explanation. For the production of the cerebral anæmia, Traube considered a watery state of the blood and hypertrophy of the heart to be necessary, and these conditions are not always present: there may be no hypertrophy of the heart, for example, when convulsions come on early in acute renal dropsy, and there may not be a watery state of the blood in contracted granular kidney with

uræmic symptoms. The œdema of the brain, moreover, has not been demonstrated.

If blood-stasis in the brain, local or general, complete or partial, be the condition to which the symptoms are attributed, we find ourselves on more solid ground.

It has been demonstrated experimentally that convulsions are excited by stasis in the cerebral circulation, in whatever way the stasis is induced: whether by rapid loss of blood, when the vessels will be empty, or by suffocation, when they will be engorged; by ligature of the arteries, or by compression of the veins. The question, then, is, are there conditions in renal disease capable of so influencing the intracranial circulation as to produce local or general, partial or complete, stagnation of the blood in the capillaries, especially of the cerebral cortex? In other words, can high arterial tension have any such effect? The question as thus stated, and the way in which the effect may be produced, will be discussed more fully later; but it may be said here that cases are met with in which convulsions are associated with high arterial tension, no other cause for them being assignable but this state of the circulation. Further, the good effects of venesection in uræmic convulsions, to which all observers bear witness, are scarcely explicable except through its direct influence on the circulation, which is to lower the blood-pressure.

Assuming that the above considerations lend a *primâ facie* support to the hypothesis that uræmic convulsions are produced by blood-stasis in the cerebral convolutions, and that the high arterial tension attending renal disease may give rise to such stasis, it may be pointed out in how many instances the varieties of uræmic phenomena, and their occurrence under various conditions, are capable of explanation.

As has already been said, it is when the arterial tension or resistance to the onward movement of the blood is over-mastering the heart that symptoms supervene. This being so, a local aggravation of the general obstruction, which the heart overcomes with difficulty, will easily give rise to local stagnation of blood.

Bearing this in mind, we may go back to the fact that uræmic convulsions may occur without antecedent hypertrophy of the heart, which has been made an objection to Traube's theory. Instead of being a difficulty, the absence of hypertrophy enters into the explanation of the attack. Convulsions, for example, sometimes come on early in acute desquamative nephritis when there is no cardiac hypertrophy. Now, in this disease, the obstruction in the arterio-capillary circulation comes on so suddenly that the heart, which is enfeebled by the pyrexia and general interference with nutrition, is for a time unable to cope with it efficiently. If time is given hypertrophy will gradually be established; but, the tissues being waterlogged, the blood increased in volume and diluted by water which has not been properly carried off in the scanty urine, the weak and dilated heart is so far overpowered that the stagnation in the cerebral circulation which gives rise to convulsions occurs. In proportion as efficient hypertrophy is established the danger is warded off.

The occurrence, which is not very uncommon, of unilateral uræmic convulsions followed by temporary paralysis is almost inexplicable on the hypothesis that they are due to the action of a poison on the brain, which would have equal access to the two sides. The late Mr. Callender, it is true, found that injuries to the right hemisphere were more frequently followed by convulsions than injuries of the left, but unilateral uræmic convulsions are not confined to the left side. It is not at all difficult, on the

other hand, to understand that stasis might be produced more easily in the cortex of one hemisphere than in the other. When the circulation is being carried on with difficulty, a very slight anatomical difference between the two sides—a smaller jugular foramen, a different disposition of the veins on the surface of the hemisphere, a different curve of the internal carotid—might determine an arrest of the blood-current on one side sooner than on the other. Experimental evidence in favour of the possibility of such an occurrence is not wanting. It has been found by Raymond and G. E. Bernard that if the inferior cervical ganglion of the sympathetic on one side be cut through and the ureters be then tied, the resulting convulsions, instead of being bilateral, affect only the side opposite to that on which the ganglion was injured. That is, the loss of vaso-motor control on one side of the brain determined the production of unilateral convulsions on the side governed by it.

But much smaller areas than an entire hemisphere are often affected in uræmia, and this would increase the difficulty of attributing the symptoms to the action of a poison. There may, for example, be fugitive aphasia, or amblyopia, or hemiopia, which are easily explained by stasis or ischæmia in certain vascular areas, but incomprehensible as results of a poison circulating everywhere through the brain.

The great diversity of the symptoms met with in uræmia, again, is best explained by varying degrees of interference with the intracranial circulation. If a given set of symptoms were present in one case throughout, and another set in another, it would be permissible to suppose that different kinds of urinary impurity predominated in different cases, excitant, convulsant, or narcotic respectively ; but the various symptoms may all succeed each other in the same case, and it is not difficult to trace some kind of

parallelism between the effects of progressive degrees of pressure as observed in meningitis and successive uræmic phenomena. It has seemed to me, from observation of cases of disease of the brain, attended with convulsions and maniacal excitement, that the latter represented a minor degree of the same irritation, which, carried further, gave rise to convulsions.

For irritation may be read blood-stagnation, or stasis, or congestion, or anæmia; and just as I have seen maniacal excitement supervene on the cessation of violent convulsions in a case of interpeduncular tumour, so have I seen maniacal excitement come on when severe uræmic convulsions had been arrested by venesection.

The pulse in acute tubular nephritis.—In acute renal dropsy the tension of the pulse is raised, but the conditions are not such as to favour the production of the extreme blood-pressure found in contracted granular kidney. At the onset of the disease the resistance in the arterioles and capillaries is developed too rapidly for hypertrophy of the heart to keep pace with it, especially as there are the anorexia, the tissue-relaxation, and the impaired nutrition of all the structures attending acute disease with pyrexia. The artery, while full between the beats, is at first, though small, compressible, and the pulse-wave is not sustained. At this period, again, the apex-beat is more or less displaced to the left, diffused, and wanting in push; while the first sound at the apex is either short or reduplicated, the aortic second sound being only slightly accentuated. Sometimes there is reduplication of the second sound. There is, in fact, in a large proportion of cases, a temporary acute dilatation of the left ventricle. Under favourable circumstances and proper treatment the heart rapidly gains strength, as is testified by the increasing vigour and definition of the apex-push, the prolongation of the first and the accentuation of the

second sound, the pulse becoming, *pari passu*, longer and stronger, and the fulness and firmness of the artery between the beats more marked. Reduplication of the first sound often persists for a long time, and this is not an unfavourable sign; persistent reduplication of the second sound is, according to my experience, unfavourable.

The prognosis in acute tubular nephritis turns very much upon the way in which the heart responds to the demand for increased contractile energy, and the pulse develops actual tension. In proportion to the rapidity of the passage of blood through the malpighian tufts will the transudation of albumen be lessened and the excretion of water increased; and, other things being equal, this will depend on the vigour of the propulsion by the heart. When, therefore, the pulse becomes firm and long, and hypertrophy of the left ventricle is developed, the increased vigour of the circulation is not only a sign, but also a cause, of improvement and a direct agent in diminishing both the albuminuria and the dropsy. When, on the other hand, the pulse remains unsustained and compressible, and the signs of cardiac weakness or dilatation persist, the state of the circulation favours the stasis in the obstructed renal capillaries, which diminishes the outflow of water and permits of the transudation of albumen. The sudden increase of urine, which usually marks the beginning of convalescence from the disease under consideration, is often entirely attributable to increased arterial tension, and not uncommonly an increase or reappearance of albumen in the urine on getting out of bed and walking about is thought to indicate a relapse of the renal affection, when it is really due to weakness of the heart, which has been unequal to the increase of work thrown upon it by the exposure and exertion, and has been unable to maintain the arterial pressure

at the point required for keeping the circulation through the kidneys at the proper rate of speed.

The Pulse in Chronic Desquamative Nephritis and Fatty Kidney.

The large white kidney, whether it comes on as a primary disease or represents the effects of acute tubular nephritis, is attended with resistance in the capillaries and arterioles; but the actual degree of tension in the pulse is dependent upon the strength and vigour of the heart, which is very different in different cases. It is very rarely indeed that the artery has the incompressible cord-like feel characteristic of contracted granular kidney, or that the hypertrophy of the heart is carried to the same degree. The pulse is usually small, long, full between the beats, and moderately tense; the left ventricle first sound is short and weak, reduplication of the first sound not being marked, even if present.

Here, again, the state of the circulation is a prognostic factor of enormous import. While the vigour of the heart and the tension of the pulse are well sustained, there may be no dropsy even when the urine is loaded with albumen. When, on the other hand, the heart is weak and the pulse, though full between the beats, is short and compressible, dropsy, if not present, is imminent. In this form of disease, as in contracted kidney, extreme high tension when present will tend to dilate the heart and damage the arteries; but its immediate effects are protective.

Much has been already said as to the apparent usefulness of arterial tension in the various forms of renal disease, and some degree of comprehension of the way in which it operates has perhaps been attained; but the evidence would not be complete without the relation of cases which have come under

my notice, in which absence of tension has coincided with an unfavourable course and issue. It is clear enough why, when the resistance in the capillaries is increased, inadequate propulsive power on the part of the heart is of unfavourable prognostic import; but it is not so clear why absence of peripheral obstruction should be a bad sign: but so it appears to be.

Absence of resistance in the arterioles and capillaries is most uncommon in contracted granular kidney, but a few cases have come under my observation. One was a patient seen in November, 1885, with Dr. Ranking at Tunbridge Wells. His age was 66, and he first consulted Dr. Ranking in July of that year on account of tightness across the chest experienced on walking uphill. He was anæmic, and had no appetite; the bowels were regular. His sleep was broken three or four times every night for micturition, the daily quantity of urine, measured on three successive days, seventy-six, fifty-six, and sixty-six ounces; it was pale, had a specific gravity of 1008, and contained neither albumen nor sugar. The pulse was frequent, 120, soft, short, and compressible; the cardiac apex was in the nipple line, the impulse weak and excited, the first sound short, the aortic second slightly accentuated.

The patient improved under treatment, but again came under Dr. Ranking's care in October with increasing weakness and anæmia, and the urine now contained albumen with hyaline and granular casts, being still copious, pale, and of low specific gravity. When seen by me on November 17th the urine was of the same character, and was considered to indicate definitely the existence of contracted granular disease of the kidneys. The pulse, however, was frequent, short and weak, and had none of the features of high tension, but was, on the contrary, a characteristic low-tension pulse, and the heart-sounds were short

and feeble. The patient sank rapidly, and died in December from asthenia.

In another case the evidence of granular kidney was not so decided, but the patient suffered from gout, and after a time had albuminuria. He always had a large, short, soft pulse, the arteries being free from any evidence of thickening or degeneration, and never full between the beats; the low tension was due to absence of resistance in the capillaries, and not to any weakness of the heart. This patient drank a good deal of whisky, which may have influenced the tension, and contributed to the final result, which was rapid succession of violent, one-sided convulsions, which came on suddenly, and persisted to a fatal termination.

An imperfect development of arterio-capillary resistance and arterial tension is much more common in acute tubular nephritis. It is met with at all ages, but with greater relative frequency in patients of middle age. One of the most striking examples came under observation many years since. The patient was a man of forty-five or thereabouts, stout, fresh-coloured, and healthy-looking, perfectly temperate in his habits, and not very sedentary in his mode of life: he was a foreman at a large place of business. He was admitted into St. Mary's Hospital on account of acute albuminuria, attributed to a chill. At first sight it seemed that the patient had vigour sufficient to enable him to throw off any form of acute illness, but the pulse was found to be singularly deficient in tension, not because of any weakness of the heart, but from absence of resistance in the capillaries. Dropsy was developed rapidly, and the patient died after a long, lingering illness.

In younger patients there is usually recovery from the acute attack, but large white kidney remains. Brief notes may be given of a case recently under

observation. The patient, a carman, aged twenty-seven, of sober habits, and previous good health, was admitted into St. Mary's Hospital on October 29th, 1887, suffering from acute general œdema affecting the scrotum specially, and the hands and arms more than the legs. The urine had a specific gravity of 1030, and was almost entirely converted into a coagulum of albumen by heat and acid. It was straw-coloured, and not smoky, but gave slight evidences of blood-reaction with guaiacum and ozonic ether. The pulse was 60, short and very compressible; the heart's impulse weak, and the sounds short and indistinct. The opinion formed on these grounds was that the case would be tedious; but that the large amount of albumen did not necessarily indicate a specially severe affection of the kidneys, being probably due to the languid movement of blood in the renal capillaries resulting from the slight development of resistance in the arterioles and capillaries generally, and the weak action of the heart. For a fortnight the dropsy increased, and much fluid accumulated in both pleural cavities, and at the end of this time the pulse was still short and compressible. The urine, however, had a specific gravity of 1008, and the coagulated albumen only occupied half its volume. At the end of three weeks a marked improvement had taken place in the pulse; the artery could be felt between the beats and rolled under the finger and was not so compressible. Simultaneously the amount of albumen had fallen so that it was described by the clinical clerk as rather more than a trace, and the dropsy and pleural effusion were much less. The rapid diminution in the proportion of albumen justified the opinion that the excessive amount present at first was attributable rather to the languor of the circulation than to the severity of the local disease.

A week later the patient appeared to be so nearly

well, that he was allowed by the resident to get up for a short time; but there was at once some return of swelling in the legs, the pulse became more compressible, the apex-beat and heart-sounds weaker, and the proportion of albumen rose from one-eighth to one-third. The heart was not equal to the maintenance of the circulation in the erect position, and with the languid movement of the blood, which resulted, came the increased amount of albumen. On the patient's return to complete rest in bed, the albumen promptly diminished. He was now kept in bed, took his food, slept well, remained free from any dropsy, and had only a varying trace of albumen in the urine. Before it was considered safe to let him get up again —six weeks from the previous attempt—he had tonsilitis, with a temperature of 103° F., and, accompanying this, severe hæmoglobinuria, the urine having a dark blood-purple colour, and containing albumen to four-fifths of its bulk, but presenting no blood-corpuscles under the microscope. The blood and albumen disappeared rapidly, and were succeeded by uric acid crystals in large quantity. Or the attack might be described as one of hæmoglobinuria complicated with tonsilitis. It seemed to me, however, that the tonsilitis was primary, and that under the stress of high temperature hæmatolysis occurred. Like the defective reaction of the capillaries and heart, it showed that the patient was made of poor stuff. He remained in hospital some months longer, and, when discharged, was still suffering from albuminuria.

Similar examples might be given in quite young children. Usually the prognosis from this condition of the circulation becomes fairly definite at the end of a fortnight.

In primary large white kidney, the imperfect development of cardio-vascular reaction is almost the

rule, the weakness, however, being more commonly on the side of the heart than of the vessels.

It may be pointed out, in conclusion, that cases of fatal disease of the kidneys without thickened arteries and hypertrophied heart (which are cited for the purpose of proving that, when such changes are found associated with renal disease, they are concurrent with, or antecedent to, and are not consequent upon it) are simply cases of the kind just exemplified, in which the kidney disease has proved fatal early from want of the cardio-vascular changes.

Arterial tension as a guide to treatment in renal disease.—Perhaps the most striking illustration of this is the resort to venesection in uræmic convulsions. Here, no doubt, practical experience was in advance of scientific teaching, demonstrating the good effects of bleeding before the *modus operandi* was understood. It is, indeed, the rule, as yet, in therapeutics that facts are first established by observation, and the explanation is learnt afterwards. This has always been the safest basis for treatment, and the advance of medicine has been retarded and countless lives have been sacrificed through the premature adoption of theories of therapeutic action. The time will come, however, when the processes of disease and the mode of action of remedies will be understood, so that treatment will be guided by knowledge of methods, and not only by observation of results.

It is not necessary to give cases illustrating the good effects of bleeding in uræmia. I have employed it in all forms of renal disease. In acute albuminuria, not only are the immediate effects good as regards the convulsions, but sometimes the kidney affection is at once relieved, and recovery takes place with extraordinary rapidity. In chronic disease of the kidney, curative effects are not to be expected,

but a condition of great danger is often promptly ended for the time being; and in the last extremity, although a fatal issue cannot be prevented, life may be prolonged, and the mode of dying may be changed, a quiet and peaceful asthenia being substituted for excitement, convulsions, and coma.

In chronic Bright's Disease it has already been seen that a fatal result is reached in a large proportion of cases through the injurious effects of high arterial tension on the arteries or upon the heart. It is a cardinal principle of treatment to obviate the tendency to death, and we ought not to wait till a fatal termination is imminent before acting upon it. The tendency being foreseen, and the danger being in some sort measurable by the degree of tension present in a given case, one of the main problems of treatment will be how to keep down the tension at a point which will not work mischief in the circulatory system without doing injury or incurring danger in other ways; and this must be done by removing, as far as this is practicable, the resistance in the capillaries, which is the cause of the tension. If, for example, the tension is lowered simply by reducing the power of the heart, the result will be the disastrous one of opening the door to albuminuria and dropsy. Injury, again, would be produced by any course of treatment which impoverished the blood or impaired the nutrition of the tissues.

All the hygienic measures in common use in the treatment of Bright's Disease comply with the indication laid down, the diminution of obstruction in the capillary circulation. The amount of nitrogenised food is restricted, care being taken that the general nutrition does not suffer, and the simplest forms of such food are recommended. The object of this is not merely to throw less work upon the kidneys, but, since the great cause of resistance in the capillaries is

the presence of nitrogenised waste of some kind in the blood, the limitation of nitrogenous food cuts off the supply of the irritant. On the same ground milk, eggs, and fish are better than flesh, since they contain little of the so-called "extractives," which, while giving flavour to meat and soups, yield a minimum of nutrient material, and probably a maximum of waste products. The various meat extracts consist of little more than the extractive matters.

All the fuel for the generation of heat and mechanical energy should be applied, as far as possible, in the form of fats, starches, and sugars.

Alcoholic drinks are forbidden, or strictly limited, for reasons which need not be here specified.

Fresh air and exercise are ordered as promoting complete oxidation of all forms of food and all products of tissue metamorphosis. Elimination through the skin is also encouraged.

Warm clothing is enjoined, with a pure woollen material of some kind next the skin, for the double purpose of promoting perspiration and reducing to a minimum the physiological arteriole contraction set up by external cold.

A warm, dry, equable climate is recommended on the same grounds.

Turkish baths, again, when well borne, may be of the greatest service by promoting free cutaneous elimination, but they must be taken with caution and in moderation.

All these hygienic measures may be faithfully carried out, and yet the arterial tension may remain at too high a point. The question then arises whether medicinal treatment may not find an opportunity.

The mind naturally turns to the physiological relaxants of the arterioles—nitro-glycerine, amyl nitrite, and the nitrites; and great results have been attributed to their employment. In my experience,

they have not been of any real service. A patient will look better, and sometimes feel better, while taking nitro-glycerine; the pallor is lessened by the free admission of blood to the surface, but the effect is fugitive; it ceases with the withdrawal of the drug, and often wears off while it is being taken. This line of treatment, moreover, has the defect of dealing only with an effect, the arteriole contraction, without removing the cause, the blood contamination.

Nitro-glycerine and the other remedies belonging to the same class may, however, render great service in an emergency—averting, perhaps, convulsions, or relieving the heart when overpowered by the resistance in the peripheral vessels.

In uræmic asthma they are often of signal service, relaxing the spasm in the systemic, and probably also in the pulmonary arterioles.

Great caution must be exercised in first administering nitro-glycerine for the relief of extreme tension, whether due to renal disease or to other causes. Sometimes the effect of the first dose of a single minim of a one per cent. solution is as if a violent blow had been received at the back of the head, and the patient feels stunned and giddy. This is, no doubt, the impact of the blood driven by the hypertrophied heart along the suddenly relaxed arteries.

In all cases of chronic renal disease the influence of constipation in increasing the blood pressure must be borne in mind, and the bowels must be kept not merely regular but open. Very important service may be rendered in this respect. Both the liver and the bowels may be made use of for the purpose of elimination. Some aperient mineral water may be taken from time to time for a week or two in sufficient quantity every morning to produce a single copious loose motion; or Carlsbad or Glauber's salts

may be employed in the same way; or sulphate of magnesia, or of soda, or the two sulphates combined, may be given.

Better than salines, according to my experience, is a mild mercurial pill—a single grain of calomel or blue pill with rhubarb or colocynth and hyoscyamus taken at night, either regularly once or twice a week, or occasionally more frequently for one, two, or three weeks. The pill may or may not be followed by a mild saline draught in the morning. It is often useful to give blue pill and colocynth once or twice a week in connection with the fortnight's course of mineral water or salines spoken of above.

Mercury in all forms has been forbidden in renal disease, but this is to forego a most valuable remedial agency. A so-called "course of mercury" would, no doubt, be injurious, aggravating the anæmia and rapidly producing specific effects on the gums and tissues; but an occasional aperient dose is attended with no such results, and, with comparatively slight effects on the bowels, it makes a more decided impression on the arterial tension than free purgation by other means. In some way or other—probably by an action upon the liver—mercury, as an aperient, has an extraordinary eliminant influence. The best remedy for uræmic vomiting or diarrhœa known to me is calomel in doses of two or three grains or upwards.

With these measures for keeping the blood free from the impurities which provoke resistance in the capillaries and tension in the arteries, will be combined the administration of iron and tonics to prevent anæmia, and it will be found that these are all the better tolerated and assimilated in consequence of the employment of eliminants.

When in contracted granular kidney the heart has at length given out under the stress of protracted

arterial tension and the left ventricle has begun to dilate, a careful study of the different influences co-operating in the production of this result, and an estimate of their relative share in it, will greatly conduce to successful treatment. In one case the main factor will be the peripheral resistance, and the pulse will not only be full between the beats, but firm and incompressible, and the impulse and apex beat of the heart will be powerful ; here free purgation by calomel or blue pill and colocynth, or with salines, will be the most important part of the treatment. In another, over-work or anxiety has impaired the vigour of the heart, and it has yielded under moderate strain ; or anæmia may have been allowed to gain ground ; or the heart, together with the structures generally, may be lowered in tone by intercurrent illness of one kind or another. In proportion as the strength and vigour of the heart are impaired, its impulse will be diffuse and deficient in push, and the pulse, while full between the beats, will be sudden and short, and in the same proportion iron, strychnine, digitalis, and the like will be required. Eliminants, however, and especially aperients, must still be employed boldly, the effects being carefully watched ; greater service is often rendered to an overweighted heart by relieving it of work than by endeavouring to improve its strength.

In large white kidney disease the same principles are applicable. It is when severe cardiac symptoms have arisen which place life in jeopardy that a clear realisation of the circulatory conditions existing at the moment, and of the relative force of the influences which are in conflict, is more necessary than in any other circumstances. The heart does not fail from intrinsic weakness, but is overmatched by the obstruction in the circulation. The fact that it has for a long time coped with the high arterial tension shows that it has been abnormally strong, and unless

it is completely worn out there is a possibility that a diminution in the resistance may enable it to resume its command over the circulation. The great object, then, as in connection with contracted kidney, will be to afford this relief. It is true that the arterial tension does not reach so high a point, and that the strain upon the heart is not so great, but the peripheral resistance is present and plays an important part in producing the symptoms and in determining the course of the disease. The nutrition of the heart is injuriously affected by the watery condition of the blood, which is diluted by the water being retained instead of being given off by the kidneys; the increased volume of this diluted blood also adds to the difficulties of the heart; the resistance in the arterio-capillary network then may easily so far add to the embarrassment of the heart as to retard very seriously the movement of the blood, and with slow movement of the blood exudation of albumen into the renal tubes and of serum into the connective tissue will be promoted; uræmic symptoms, again, are liable to supervene from stasis in the cerebral capillaries. The pulse, then, is a valuable guide in the treatment. Whatever may be the other indications and the other remedial measures, it must be an object to diminish the peripheral resistance and to increase the driving power of the heart, so that the heart may have full control over the circulation and may maintain a steady flow of blood through the capillaries. It is by means of the pulse and heart sounds that the degree in which this object is attained is ascertained, and it adds greatly to the interest of this struggle against the fatal tendency of severe disease of the kidneys to know this guide.

It would simply be a repetition of what has been already said were the treatment of acute renal dropsy to be discussed from the same point of view. But it is worth while pointing out that here treatment will

not merely relieve suffering and prolong life, but will definitively restore health and soundness. Anything, therefore, which gives a more definite aim and direction to our endeavours is of special value, and this is claimed for the indications derived from the pulse and heart. It has been pointed out that at the onset of acute renal dropsy the heart is embarrassed by the sudden increase of resistance in the peripheral circulation, and, being itself enfeebled by the pyrexia and general derangement of the system, is unable to cope with it, so that a degree of dilatation takes place. Both experience and theory (experience especially) show that the recovery of the heart from this condition and the establishment of a certain degree of arterial tension are necessary to the cure of the disease; this appears to be a link in the chain of reaction by which a return to the normal state is brought about. It becomes an object, therefore, to aid in the production of this condition in the circulation.

Uræmic symptoms of the most violent character may occasionally supervene at quite an early period of acute desquamative nephritis. The peripheral resistance so far overcomes the propulsive power of the heart as to give rise to stasis in the cerebral circulation. Venesection is of such striking service in these circumstances, not merely in arresting the convulsions but in its beneficial influence on the disease itself, that it has become a question in my mind whether it ought not to be commonly resorted to as part of the treatment of acute renal dropsy at an early stage.

CHAPTER XV.

INTERMITTENT ALBUMINURIA.

The intermittent albuminuria of young people may not seem to come within the scope of a book on the pulse; but it is, according to my judgment, entirely dependent on the condition of the circulation. The term intermittent albuminuria seems to me to be preferable to the other names which have been employed to designate this affection. To call it "cyclic" is to misstate facts; to speak of it as "functional" raises a controversy as to its character; while to name it the "albuminuria of adolescence" ignores its occurrence at other periods of life.

Dr. George Johnson has, as was to be expected, investigated this interesting subject, and it had attracted the attention of the late Dr. Moxon. Professor Grainger Stewart also has discussed the questions which suggest themselves in connection with the appearance of albumen in the urine of children and adolescents otherwise apparently healthy; while Dr. Clement Dukes has taken advantage of the opportunities afforded by his position as physician to the great school of Rugby for obtaining experience as to the conditions under which the albuminuria occurs, and as to the symptoms and effects on the health and vigour which attend it.

The most striking feature of this form of albuminuria is the extraordinary fluctuation in the amount of albumen present. One day the coagulum may occupy one-third or even half the volume of urine, on the next there may not be a trace; the albumen may be abundant at one period of the

twenty-four hours, and entirely absent at another. The conditions under which these variations occur are of great interest. The morning urine is usually free from albumen, while that passed after breakfast not uncommonly presents the maximum for the day. Later, the amount varies in a manner which appears to be capricious, until the causes are traced in the incidents of the day; albumen, for example, is often absent after the midday dinner of a boy when it has been abundant after breakfast.

Except for the presence of albumen the urine appears to be normal. It is usually clear, and has a good colour, varying in tint and in the presence or absence of lithates, as in health; and there is nothing in its appearance or specific gravity or reaction which enables us to say that a given specimen contains albumen.

From the fact that the albumen often appears after breakfast it has naturally been attributed to malassimilation of the food taken; but this idea is not supported by any peculiarities in the character of the albumen, which, according to the researches of Dr. R. Maguire, consists mainly of globulin and serum-albumen, and not of peptones; and it is rendered untenable by the fact that after a larger meal richer in albuminoids later in the day there may be no albumen in the urine at all; and still more clearly by the fact that the albumen will be absent, or greatly diminished in amount, after breakfast taken in bed. I have found, again, that a cup of hot milk taken before dressing has a marked effect in diminishing the amount of albumen found after breakfast, especially in cold weather.

External cold has naturally a considerable influence on the albuminuria, and Dr. George Johnson has found that albumen is often present in the urine after cold bathing, and probably it is very common after swimming for any length of time; but good

swimmers are rarely under the necessity of consulting a medical man.

The influence of greatest power is undoubtedly exertion, but from this it is difficult to disentangle altogether the influence of position. The difference observed between one day and another and between different times of the same day can usually be traced to the kind and amount of exertion. In one young lady of twelve or thirteen albumen appeared in the urine whenever she was allowed to go up and down stairs, whereas it was absent so long as she was kept on one floor. In some cases, however, violent exertion will be followed by little or no albumen, when an ordinary walk gives rise to a considerable amount. In my opinion it is the combined influence of the erect position, of the exertion of dressing, and of exposure of the surface to the lower temperature of the room after the warmth of bed, which determines the after-breakfast albuminuria, aided probably by the lowered tone and energy of the heart and vascular system after sleep, and perhaps by the rapid repletion after a long fast. When a patient, subject to intermittent albuminuria, and actually suffering from it, is kept in bed, the albumen usually ceases to appear; but when he again gets up it reappears copiously for a time, unless a purgative has been taken. Pyrexia appears to dismiss the albumen, but as the patient is usually put to bed it is not easy to determine how far pyrexial relaxation of the vessels and activity of circulation of themselves produce the result.

Constipation is another influence under which the albumen is extremely liable to appear. This and the effect of an aperient in dismissing the albumen have been well brought out by the experience of Dr. Dukes of Rugby.

The special feature of intermittent albuminuria is the readiness with which albumen appears in the urine,

and the comparative slightness of the cause which is sufficient to induce it. A temporary albuminuria is not uncommon as a result of over-study, with the attendant late hours and privation of fresh-air exercises. Attention has been called to it by Sir Andrew Clark in connection with the laborious and protracted preparation for Civil Service and other examinations, and with the over-strain of the examinations themselves. This is no doubt allied to intermittent albuminuria, but it cannot properly be included under it.

Intermittent albuminuria, while most common in growing boys and adolescents, is met with at all ages and in both sexes, except in infancy and early childhood and old age; I have seen it in a boy of eight, in girls of twelve and thirteen, and in a man of forty-five. It cannot, therefore, be put down to masturbation, and in very few of the cases which have come under my observation has there been any reason to suspect that this vice was practised.

In every case which I have seen there has been a neurotic family history, usually of a marked type. In one case, however, the only neurosis traceable was spasmodic asthma.

The subjects of the affection are not by any means necessarily anæmic, or weak, or languid. On the contrary, they have often a good colour, and are well developed and muscular, and only too full of energy and impetuosity. Very commonly, however, they are easily exhausted, and especially are incapable of sustained mental effort. At school they are subject to headaches when the lessons demand close application, and at college, or when preparing for the examinations which form the portal to the military or civil service, they are liable to break down, or to find that they are unable to concentrate their attention on their work. In many of them there is a great proneness to catarrh, which is often very severe and attended

with great prostration. Dr. Dukes has made the interesting observation that when a boy faints in the class-room or chapel, he is certain to be the subject of intermittent albuminuria.

If we exclude malassimilation, this irregular occurrence of albumen in the urine can only be explained by variations in the blood pressure or circulation; it is, indeed, closely analogous to the fluctuating albuminuria of some forms of heart disease. It is certainly not due to any organic disease of the kidneys, though it may possibly lead to the establishment of organic changes in these organs if it persists for long periods. The continued blood stasis in the malpighian tufts, and filtration of albumen through the walls of these capillaries must in time impair their nutrition and structural integrity. I have not known disease of the kidneys, either acute or chronic, follow upon this intermittent albuminuria; but Dr. Dukes, who sees large numbers of boys at school, and has the opportunity of watching their careers in after life, has met with examples.

But to return to the question of the relation of intermittent albuminuria with varying conditions of the circulation. Malassimilation may be excluded as a cause on the grounds already stated, namely: that albumen is found in the urine most frequently and abundantly after breakfast, a comparatively light meal, and not after dinner, when more nitrogenous and less digestible food is taken; that it does not appear, or is comparatively slight in amount, when breakfast is taken in bed, and is often lessened after breakfast when hot milk is taken before the patient rises. The influence of position and exertion, which is marked and undoubted, is also against malassimilation, and perfectly consistent with variations of circulation, as causing the albuminuria.

The great characteristic of the circulation met

with in association with intermittent albuminuria is its extreme instability. The pulse will be small and weak one day, and large and firm another, or such diversities will be exhibited at different hours on the same day; this, indeed, I should expect would be found to be the rule were the pulse examined at short intervals throughout the day. Sometimes the pulse appears to be destitute of tension, but I have met with no case yet in which the pulse does not possess from time to time, or, indeed, habitually, a certain degree of tension—*i.e.* the peripheral resistance, which is the ultimate cause of tension, is present, keeping the arteries full between the beats; but the energy of the cardiac systole, the *vis a tergo*, which determines the actual degree of blood pressure within the vessels, is deficient, and varies extremely.

In all the cases which I have examined, the heart has presented an interesting peculiarity. The apex beat has been feeble and inconspicuous, and the left ventricle sounds have been short and weak; while the beat of the right ventricle has been powerful, lifting the costal cartilages, and its sounds have been loud. This I have found, not only in boys and girls in whom the apex beat is often undefined, but in young men. The significance of such a disproportion between the action of the two sides of the heart in relation to the albuminuria is not clear; it would seem to indicate some obstruction in the pulmonary circulation and, if this went so far as to interfere with the supply of blood to the left auricle, it might greatly influence the systemic circulation. *Pro tanto* the effects of mitral disease would be imitated, and it is in mitral regurgitation that fluctuating albuminuria is most common.

Treatment.—From the point of view here set forth the main object of treatment is to improve the tone and vigour of the circulation. The patient should live an active outdoor life. I have not often

considered it necessary to advise that a boy should not be sent to a public school, or that while there he should be forbidden to engage in games; I have sometimes permitted even football, if the boy has been otherwise physically qualified for it. Girls should be encouraged to take exercise, such as skipping and lawn-tennis, besides walking, and should be allowed to ride. The effects of confinement to the house, and of such care as would be required for kidney disease, have been in my experience most injurious. When a long voyage has been ordered I have known the albuminuria to persist while at sea and cease with active exercise on shore.

Flannel, or some other material made entirely of wool, should be worn next the skin, summer as well as winter. The diet should be simply that suitable for a growing boy or youth. It is very important that the bowels should act regularly and efficiently, and an occasional or even regular small dose of hydrarg. cum cret. with rhubarb and soda, or of pil. hydrarg. with pil. rhæi co., or colocynth and hyoscyamus is most useful.

The prognosis is good. Most youths grow out of the liability, and when it is perpetuated, or induced by coaching or cramming for an examination, it usually disappears after a time. Young men subject to it, who have to go up for a medical examination previous to admission into one of the public services, should always take an aperient beforehand. The presence of albumen in the urine would probably disqualify a candidate, and a purge prevents its appearance for twenty-four hours or longer.

CHAPTER XVI.

THE PULSE IN AFFECTIONS OF THE NERVOUS SYSTEM.

THE pulse is under the direct and immediate control of the nervous system. The centres for the cardiac and vaso-motor reflexes are situate in the medulla, and receive, on the one hand, impressions from the viscera, the cutaneous surface, and the body generally, which reach them in an upward direction through the spinal cord and sympathetic system, and on the other, impulses which travel downwards from the brain. The heart thus responds to every emotion and sensation, and even thought, as well as to the demands created by muscular exercise, or by the organic processes, such as digestion. In excitement the action of the heart becomes more frequent and violent, producing a corresponding frequency and force in the pulse. This, however, is not all, for the arterioles are almost as susceptible as the heart to nervous influence and there may be local dilatation of these vessels, such as is seen in blushing and in the flushed face of excitement, or a general contraction.

Since, then, the nervous system is the intermediary through which all modifications of the pulse are produced from whatever cause, and whether due to the action of the heart or of the arterioles; since, again, all states of the nervous system are reflected upon the circulation, each emotion being attended with its own reaction upon the heart and arteries, and even sensations producing recognisable effects, it is to be expected that diseases of the nervous centres will be attended with special symptoms manifested through the circulation and by the pulse, and such is the case. It is, however, very difficult to give any such account of the

modifications of the pulse in affections of the nervous system as shall be useful; for, while the slow hesitating pulse is almost as significant as the purposeless vomiting in meningitis, and the contracted arteries almost as characteristic as the retracted abdomen, in respect of these, as of other symptoms, different and even opposite effects may be produced by the same disease, and different diseases may give rise to like effects. Although, therefore, the pulse may form a most important item in the diagnosis of disease of the brain, it is not by any individual symptom, but by the concurrence of symptoms derived from various sources, that a trustworthy opinion is formed.

Meningitis and the pulse.—Perhaps the most remarkable effect upon the pulse produced by cerebral affections is the slow and hesitating pulse of the early stage of meningitis; it is scarcely met with except as a result of serious brain mischief, and is therefore of very great diagnostic importance. The temperature is above the normal point, but instead of increased frequency in the pulse and relaxation of the arterioles, the pulse is less frequent than normal, and the arteries are contracted; the individual beats are long and not quite equal in force or regular in time, and the general effect is to give the pulse a peculiar, deliberate and slightly hesitating character. The second sound of the heart is not unfrequently reduplicated with this kind of pulse.

At this period of the disease, in which the pulse has the character just described, the inflammation has reached the stage at which stasis of the blood is taking place in the capillaries. With the effusion which occurs later and the compression of the brain produced by it, an uncontrolled frequency of the pulse supervenes varying from minute to minute under the influence of slight causes, and it is at the same time devoid of tension.

Cerebritis and tumour.—Cerebritis and cerebral tumour may also slow the pulse, and, without affecting the frequency, a local cerebritis going on to the formation of an abscess may be attended with so much spasm of the peripheral vessels that the surface of the body may be cool and the extremities cold, while the internal temperature is as high as 103° or 104° F. In some cases of tumour of the brain there may be very remarkable reduplication of the second sound of the heart in association with high pulse-tension. In late stages of tumour of the brain the pulse often becomes extremely weak, short, and small, exemplifying an extreme degree of low tension from a combination of relaxed arteries and weak action of the heart.

The pulse in coma.—Coma has no characteristic pulse. The causes of this condition are varied, and the pulse will be that of the particular cause. As regards the circulation, the cerebral hemispheres have in coma ceased to act as controlling or disturbing agents, and it is abandoned to the influence of lower centres or of external impressions. The pulse may be frequent or infrequent, of high or low tension.

Convulsions.—Convulsions react upon the circulation like any other form of violent muscular exertion, increasing the frequency and force of the heart's action and lowering the arterial tension. Convulsions and the pulse, however, will be considered fully from another point of view.

Mania.—In acute mania the pulse is singularly little affected, and it is astonishing to find how little it is accelerated or otherwise influenced by the mental excitement and bodily agitation present in this disease. Whatever the local vascular conditions of the cerebral hemispheres may be in mania, the symptoms are not due to any excitement of the general circulation.

Melancholia.—Melancholia may be associated with different kinds of pulse as regards tension—usually, however, the frequency is diminished; but the relation of melancholia with states of the circulation is reserved for special consideration.

General paralysis in its early stage usually has the arteries contracted and a pulse of tension; later the pulse becomes weak and toneless.

Hysteria.—In hysteria the pulse may have different characters, but it will be mobile and liable to disturbance in sympathy with the emotional mobility of the subject. The hysterical paroxysm is usually attended with frequent and violent action of the heart and throbbing of the vessels in the neck, of which the patient is conscious; but even more constant and characteristic is the arterial spasm, which makes the pulse small and hard, and gives rise to high arterial tension—probably the most important factor in the secretion of the pale and watery urine which accompanies an hysterical attack.

Migraine.—Migraine or sick-headache, again, is attended with contraction of the arterioles, sometimes, if not unilateral, yet predominant on one side; and it has been questioned whether the essential condition and cause of the attack is not spasm of the cerebral arteries.

Lesions in the pons and medulla oblongata, implicating, directly or indirectly by proximity, the great cardiac and vaso-motor centres, will produce very important effects. It is by pressure or other form of disturbance of the cardiac or respiratory centre that tumour or abscess of the middle lobe of the cerebellum so often causes sudden death.

There are not many diseases of the spinal cord which are attended with modifications of the pulse in any degree characteristic or entering prominently into their symptomatology.

In spinal meningitis the pulse is small and tense; and in locomotor ataxy the arterioles are habitually contracted, and are specially tightened up during paroxysms of the lightning pains—perhaps as a reflex effect of the pain.

In cervical pachymeningitis with compression of the cord the pulse becomes infrequent, and, according to Charcot, sudden death from arrest of the heart is not uncommon.

Neuralgia is attended with contraction of the peripheral vessels; and it is not without interest to remark that many of the agents by which attacks are warded off or arrested are such as relax the arterioles.

More important for the purposes of this book are the effects produced on the brain and cord by abnormal conditions of the circulation, since, if these can be identified, it may often happen that the pulse will afford a clue to remedial measures.

The nutrition of the brain, as of all parts of the body, is dependent upon the supply of an adequate amount of healthy blood; but the functional activity and efficiency of the brain are even more dependent upon the blood-supply than its nutrition, and are influenced by it to an extraordinary degree; so that blood which would maintain the structural integrity of the brain might be altogether unfit to minister to its functions. The fœtal brain, for example, grows and develops with the greatest rapidity when supplied only with placental blood, which is very imperfectly aërated. The presence of alcohol, or chloroform, or morphia in the blood, again, does not interfere with the nutrition of the nerve-centres, but it deranges their action; and it cannot be doubted that poisons generated in the system or retained excretory matters have a similar effect. The most striking illustration of disturbance of the cerebral functions by interference with the supply of blood is

the occurrence of convulsions as a late, if not final, symptom attending rapidly fatal hæmorrhage.

The liberation of nerve-force has been represented as an explosive action, and this implies the presence in the nerve-cells of a substance of a high degree of chemical tension, ready, on the application of the proper stimulus, to combine instantly with the oxygen brought by the blood. The formation of such material, its maintenance at a given state of chemical tension, so to speak, which differs in the different centres—in the cortex, in the central ganglia, and in the medulla and cord—is not paralleled by any other nutritive operation. It is only, moreover, by the free access and renewal of aërated blood that the oxygen required for the proper and orderly evolution of nerve-force is supplied.

Sleeplessness and the pulse.—An illustration of the relation between cerebral functions and the circulation is seen in the anæmia of the cortex of the hemispheres during sleep. This bloodlessness is essential to sleep, and if it can be induced, sleep follows. It is not clear, however, whether in natural sleep it is primarily the shutting off of the blood which determines and compels sleep, or the exhaustion of the nerve-cells, and the periodic cessation of activity organised in the experience of the nervous system by the succession of day and night, which disposes to sleep, and provides for it by causing the cortical arterioles to contract so as to produce the anæmia.

Sleeplessness is extremely common, and may be due to a great variety of causes: to excitement during the day or in the evening; to grief or anxiety; to exhaustion or over-work, especially work of an intellectual kind carried on far into the night; to late meals; errors in diet; indigestion and flatulence or other functional derangements; to tea or coffee. It

might be shown with more or less plausibility that some, at any rate, of these disturbing influences act through the circulation; but no useful purpose would be served, and on the recognition and removal or avoidance of the particular influence affecting a given case the sleeplessness will disappear.

Sleeplessness and high pulse-tension.—We are frequently called upon, however, to relieve habitual sleeplessness which cannot be referred to any of the above causes, and in many instances it is associated with abnormal conditions of the circulation. Not to speak of heart disease, all forms of which are prone to disturb or interfere with sleep, or of palpitation, which is apt to come on at night, there are two distinct conditions of the circulation to which sleeplessness is very often attributable. One is the state of high tension, absolute or virtual. This is a very common cause of sleeplessness at all periods of life and in both sexes, especially, perhaps, in women. There may be little beyond the sleeplessness of which the patient complains, or the loss of sleep may merely be one of a long train of symptoms—depression, apprehension, loss of memory, want of energy and the like. The clue to the loss of sleep is afforded by the pulse; the artery at the wrist may be large or small, but it is full between the beats and can be rolled under the finger; the pulse may be long and gradual when the artery is contracted, or somewhat abrupt in its beat, and ending suddenly when the artery is dilated and worn out. It will vary also according to the condition of the heart; usually it is firm and not easily compressible, but it may be weak and yielding under the finger.

It appears to me probable that the cause of the sleeplessness in the class of cases under consideration is the inability of the arterioles of the cerebral cortex to overcome the excessive blood pressure within them,

and to shut off the blood so as to induce the anæmia requisite for sleep. In common with the peripheral arteries generally, their muscular walls are all day putting forth an abnormal amount of contractile force, and at night are unequal to the further task of exercising sufficient additional contraction to narrow the channels to the sleeping-point. In such cases sleep is often at once procured by reducing the general arterial tension, and a mild mercurial aperient is the most effectual hypnotic. The cases are numbered by scores in which I have found this line of treatment efficacious in habitual sleeplessness which had lasted for years, many patients having been rendered independent of sedative draughts to which they had been driven. At first it is not unusual for the patient to suppose that a new and powerful opiate has been given, and the good effects do not wear out by repetition. It is not by unloading the bowels that the aperient acts; it has often to be given to induce sleep when the bowels are acting regularly and freely; and it is to be remarked that it is within an hour or two of the pill having been taken, and many hours before an evacuation results, that the patient sleeps. The general intravascular pressure being lowered, the cortical arterioles are no longer over-distended by the pressure within them, and they can respond to the call for exclusion of the blood from the hemispheres. The desired effect may often be obtained by other means—by a hot bath, by the sitz bath and wet compresses employed in hydropathic establishments, by a hot compress over the epigastrium, by standing in cold water and then rubbing the feet well with a rough towel, and by a variety of other measures which either relax the cutaneous vessels generally, and so lower the arterial tension, or set up a local hyperæmia which diverts the blood from the head. A method of courting sleep which I have known to be adopted by

subjects of high arterial tension is to lie with the back of the neck on a hot water bottle. Sometimes, however, the object of the measures just alluded to is defeated by some collateral effect—as, for example, when a hot bath sets the heart beating violently—and, when successful, such methods have the defect of dealing with the effect and not removing the cause.

Sleeplessness and low tension.—An opposite state of the circulation, extremely low tension, may be the cause of sleeplessness. It is more common after middle life and in men of sedentary occupation, and may be traceable to over-work or anxiety, or simply to close attention to business and neglect of holiday. It is not often that there is excess in eating and drinking; more frequently the patient is distinctly abstemious. Without apparent cause he begins to lose his sleep at night. In the day time, however, he is disposed to sleep, and not unfrequently, unless engaged in actual work, he drops asleep at any moment; not only after dinner, when sleep in such cases is irresistible, but when reading his newspaper after breakfast in the morning. Not unnaturally it is supposed that the after-dinner nap spoils the night's rest, or that the bad night causes the drowsiness during the day; but attempts to rectify this state of things by preventing sleep after dinner and similar measures fail. The sufferer may try to secure a good night by going to bed soon after dinner, but at this time, as at the usual hour for retiring, as soon as he lies down he is wide awake and so remains. After a time it will be discovered that it is position which induces or prevents sleep. In the sitting posture sleep is almost irresistible, in the recumbent position almost impossible. The patient will sometimes get out of bed in the night and sleep in his chair, as we see sufferers from advanced heart disease. Under such circumstances I have found the circulation

unstable; the pulse, during the same interview, at one time short and weak, and at another presenting the characters of virtual tension—that is, full between the beats, but compressible, sudden, and not sustained; and the power of the heart low. There is a want of tone in the muscular tissues of the arterioles, and a want of driving power in the heart, and the distribution of the blood thus becomes unduly influenced by gravity. In the sitting position, when the heart is not stimulated by exercise, nor afflux of blood to the convolutions determined by mental work, the blood is not lifted to the height of the brain with sufficient energy, the contractile tendency of the cortical arteries is not opposed by intravascular pressure, and sleep-producing anæmia of the hemispheres results. On the other hand this same want of tone in the arterioles allows them to be distended by the increased pressure of the blood within them, which attends the horizontal position, and thus sleep is prevented. In such cases change of air will often put an end to the sleeplessness at once, and the appropriate remedies are vascular tonics—iron, acids, strychnine, and digitalis.

Convulsions and the pulse.—We have now to show that conditions of the circulation indicated by the pulse are among the causes of convulsions.

The brain responds by convulsions to a great variety of disturbing influences: to direct irritation of the cortex, as has been demonstrated by experiment, and is illustrated by the effects of spiculæ of bone, tumours, meningeal hæmorrhage, etc.; to irritation of lower centres and tracts, such as the cerebellum and its superior peduncles, or of parts in the interpeduncular space, and to reflex irritation of the most varied kind; to poisons of different sorts, but especially to the poisons of specific fevers, such as smallpox, scarlatina, or measles.

With regard to such causes of convulsions as

concern us from their relation with the pulse, experiment has shown that convulsions may attend exactly opposite states of the circulation. The final phenomena of death from rapid hæmorrhage, when the cerebral vessels are empty and the pressure presumably at a minimum, are convulsions, and convulsions occur in death by asphyxia, when the blood pressure is high. These extremes probably meet in the production of stasis of the cerebral circulation, and it seems to me that such stasis is producible by high pressure in the vascular system, and that we have clinical illustrations of convulsions produced by unduly high arterial tension, on the one hand, and by excessively low arterial pressure on the other.

It must now be pointed out how high blood-pressure may give rise to stasis in the cerebral circulation.

The cranium being a closed cavity, the total volume of its contents, brain-structures and membranes, blood and cerebro-spinal fluid, must be a constant quantity. The effusion of liquid into the ventricles or meninges, often found after death, shows that some kind of compression and shrinkage of the brain-substance is possible; but whether this is at the expense of the blood in its capillaries, or of fluid in the perivascular spaces, or of interstitial fluid, cannot be stated definitely. Since, however, there may be an abnormally great appearance of vascularity in the sulci and white substance at the same time with effusion into the ventricles, it cannot be simply by exclusion of blood that the intraventricular or subarachnoid fluid makes room for itself. But whatever may be the way in which the brain-substance accommodates itself to effused fluid, it is not to be supposed that it is compressed into smaller compass at each pulsation of its vessels. This being so, either the blood must escape by the veins exactly in the same amount and at the

same time as it enters by the arteries and, therefore, more or less, in a pulsatile manner, or there must be some shifting to and fro of cerebro-spinal fluid between the cranium and spinal cavities—that is, the expulsion of a certain amount of fluid from the cranium into the spinal canal with each arterial pulsation and a gradual reflux in the interval. But the spinal canal is also a closed cavity, and although its walls, being only ligamentous between the vertebræ, are not so rigid as the bones of the skull, there is no provision for variations in its capacity, while the arteries of the cord and its meninges will be distended at the same moment as those of the brain, which will tend to resist the passage of fluid from the cranial to the spinal cavity. The to-and-fro movement of cerebro-spinal fluid, then, which has been postulated as an easy explanation of some of the difficulties with regard to the cerebral circulation, cannot be admitted, and it must be the case that exactly as the blood enters the cranium by the carotids and vertebral arteries it leaves it by the internal jugular veins. The movement of blood in the internal jugulars has, in fact, been found to be pulsatile. It has been pointed out in an early part of this book that neither the expansion of the arteries nor the onward movement of the blood is as great as is usually supposed, and there is a provision both in the carotids and vertebrals for moderating the current (which is present in a still more marked degree in animals which feed on the ground); but with all this, the intracranial circulation is carried on under peculiar difficulties, and any interference with the general circulation will tell with special influence upon it.

In the eye the circulation is under much the same conditions as in the brain, so far as the effects of high intravascular pressure are concerned, the sclerotic

being unyielding; and the changes which are known to take place in the retina in renal disease have more than a mere diagnostic or clinical interest. They throw light on changes occurring in the brain, and study and investigation in this direction will be well repaid. We have retinal, as we have cerebral hæmorrhages. It is possible, again, that there are intracranial conditions like in kind to those of the choked disc, if different in degree. These are, in my opinion, attributable, directly or indirectly, to pressure-effects rather than to blood deterioration. Glaucoma, which, in my small experience, has usually been associated with high pulse-tension, and which I believe to be an effect of intravascular pressure, may also have its analogue in the cranium.

Be this as it may, there cannot be any doubt that the intracranial pressure varies with each heart-beat and with all variations of pressure in the arteries or veins. The brain would pulsate if it could, and does pulsate whenever a part of the cranial vault is absent or yielding. The fontanelle of an infant beats with the pulse, rises and falls with the respiratory movements, is tense and prominent when the child cries, hollow when it is asleep, is full and firm when the child is well, depressed when it is weakened, say by diarrhœa; and the same fluctuations can be seen at a trephine opening in the adult skull. The intracranial pressure, in effect, varies with the blood pressure, and the same pressure-fluctuations which give rise to the pulsation and varying tension of the fontanelle will, in a greater or less degree, tend to compress the cortex of the hemispheres against the vault of the cranium. It is conceivable, therefore, that, when the blood pressure is high, the compression might be such as to interfere with the free passage of blood through the *pia mater* of the convolutions, or to compress more or less the veins which run on the

surface of the hemispheres to the longitudinal sinus; it might even arrest momentarily the cortical circulation.

Blood-stasis in this situation from pressure against the cranial vault must indeed occur when the convolutions are flattened by effusion into the ventricles in tubercular meningitis, and it is not unreasonable to suppose that the convulsions and loss of consciousness, which mark the final stage of this disease, coincide with this event.

The explanation of the production of convulsions by high intravascular pressure, then, would be that with it there is increased intracranial pressure, and perhaps effusion of serum. This pressure in the closed cavity of the cranium must bear on the outside of the vessels, and must tend to hinder the flow of blood through the long capillary network spread over the surface of the cortex—obstruction here, as in the capillaries generally, also being an initial factor in the production of the high tension. At some point or other the external pressure goes so far as to give rise to stasis in a capillary area of large or small extent. It may be by flattening the convolutions against the vault of the cranium, or by compressing cortical veins on their way to the sinus, or by interfering with the outflow of venous blood at the base of the brain; but in whatever way it is produced, the occurrence of stasis will be facilitated when the driving power of the heart is no longer equal to cope with the general resistance in the periphery. With the stasis will come convulsions.

A gradual encroachment on the capacity of the cranial cavity would exert a very similar influence on the cortical circulation, by pressure from without, to that which I am assuming might be the result of pressure from within, and the effects of such an occurrence are exemplified in the following case, seen with the late

Mr. Brend of Kensington. The patient, a lady aged about thirty-six, had enjoyed good health all her life and had had children, when she began to be subject to fits. She was seen by various physicians, and different opinions were given as to the character of the attacks; for the most part they were considered to be hysterical. She was subject to headache and confusion of thought, but not more so than is common; there was no vomiting. When I was consulted she looked well, was stout, and had a good colour, and was discharging all her family and social duties, though with effort. Just as she had taken off her dress to facilitate an examination of the chest, an attack came on, which we were so fortunate as to witness. She turned pale and lost consciousness, there was a slight quiver of the muscles of the face and movements of the arms. But the most striking fact was a complete arrest of the heart for a sufficient time to cause us serious anxiety. This prevented us from adopting the opinion previously given that the fits were of no importance, and our prognosis was soon afterwards verified by the patient's death in one of these attacks. The only morbid appearance found was an extraordinary thickening of the frontal bone, which was as dense as ivory, and more than half an inch thick at the lower part, gradually thinning towards the coronal suture. The inner surface was smooth and the dura mater little changed.

The only assignable cause for the attacks was the diminution in the capacity of the cranial cavity.

There are not wanting evidences of the production of momentary arrest of the cerebral circulation by comparatively slight temporary causes. Some years since I met at a scientific congress a distinguished French surgeon still living, who at the age of fifty-five, or thereabouts, was suffering from whooping cough and at every paroxysm dropped down unconscious;

and I have met with a case in which for many years the patient, who was the subject of chronic bronchitis, fell down with momentary loss of consciousness and slight convulsion whenever he coughed, and this too quickly for the production of cyanosis. In these cases the obstruction would take effect by backward pressure through the jugulars. Short of any such result as loss of consciousness, it is not uncommon for the respiratory concussion attending the act of coughing to be felt as severe pain in the head, and it is perhaps worth noting that it is in influenza associated with depression and low blood-pressure that this occurs.

It has already been argued that in uræmia the immediate cause of the convulsions is blood-stasis in the cortex, high blood-pressure being the intermediary process by which stasis is induced. Experimenters have been beaten from one position to another in the endeavour to identify a poison present in urine which, retained in the blood or formed in the blood, in consequence of the retention of urinary excreta, would directly cause convulsions. It remains now to adduce examples of convulsions associated with high pulse-tension without albuminuria or renal disease.

A medical friend called on me one morning ten years since, at the bidding of his wife, as he laughingly told me. He said he had had some sort of fainting attack in the night, but that he was perfectly well. His forehead, however, was covered with minute ecchymoses, and it was obvious that he had had severe convulsions. He was forty-two years of age, had lived freely and carelessly, but was not intemperate; there was a doubtful history of syphilis, and twelve months previously he had had a severe fall on his head. He had very high tension of the pulse. I was summoned to him early next morning and found that he had gone from one convulsion to another for the greater part of the night

of so severe a character that artificial respiration was required at the end of each attack. I had no hesitation in attributing the convulsions, the first and last he has ever had, to the state of the circulation, and the patient was bled to about thirty ounces. He had the last convulsion as the blood was flowing, made a rapid and complete recovery, and has had no attack since.

In another very similar case, also that of a medical man, aged about fifty, seen three years since, I did not see the convulsions, which had been very violent, but the assurance I felt justified in giving, from the history and from the state of the pulse, that no recurrence need be apprehended, if the tension were reduced by appropriate diet and treatment, has been verified up to the present time, and the patient has improved greatly in general health.

A third case, that of a lady aged at that time forty-nine, came under my observation in May, 1878. She was stout, over-fed, and had marked tension of the pulse, which appeared to be taking effect on the left ventricle. She was said to have albuminuria, but I found no albumen in the urine, and the specific gravity was normal. I was consulted on account of severe and repeated convulsive attacks of an epileptic character, which came on two or three times a week, sometimes in series of six or seven. The convulsions speedily ceased under treatment by saline aperients, which lowered the vascular tension, and she remained free from them until 1885, when she had cerebral hæmorrhage and hemiplegia, and she died late in 1886 from another attack attended with convulsions.

In December, 1885, a gentleman, aged sixty-five, working hard as a teacher of music, consulted me on account of convulsions, which had occurred in the previous July before breakfast and on December 1st after dinner. On the latter occasion there had been sudden loss of consciousness and a fall, in which a cut

on the head had been sustained. He looked and felt well, had a fresh colour, the appetite was good and the bowels regular and he slept well. The urine was copious, had a specific gravity 1015, and contained neither albumen nor sugar. The pulse was tense but short, the first sound of the heart short, the aortic second accentuated. The condition of the circulation was thus that of virtual tension. A mild blue and colocynth pill was ordered to be taken once a week and arsenic and nux vomica prescribed as a tonic. In March, while stooping after a heavy meal, he fell forwards and was unconscious for a few seconds, and this is the only attack he has since had.

Other illustrations might be given, but I will only refer to one more, which I related in my address as president of the medical section of the Brighton meeting of the British Medical Association. The patient, aged thirty-seven, came under my observation in November and December, 1885, suffering from headache and oppression, and was found to have an unusual degree of arterial tension ; shortly afterwards I was called to him, in consultation with Dr. Wilbe, when he was almost unconscious, overwhelmed apparently by a sense of pressure in the head and exhibiting violent synchronous contractions of the pectoral and abdominal muscles. The pressure in the arteries was extreme and the incompressibility of the pulse surpassed anything in my experience. The patient was freely bled with good effect, but repeated free calomel purging was needed to bring down the tension and complete the recovery. There was an early stage of contracting kidney in this case, but no such change as to give rise to uræmia.

The convulsions, whether unilateral or general, which sometimes occur months or years after an attack of hemiplegia, have in my experience always been associated with high tension and have often been

prevented from recurring by treatment directed against this. In a brain damaged by hæmorrhage which has left a cicatrix, the circulation is more easily deranged to a point attended with symptoms than before.

It may perhaps be considered as proved that in some way or other such a modification of the cerebral circulation may be produced by high arterial tension as is capable of giving rise to convulsions, and, as I have already said, it is, in my opinion, through the intervention of high arterial tension that uræmic convulsions are brought about. There may be extreme uræmic intoxication, ending fatally by coma, without convulsions; and, on the other hand, convulsions may supervene at a comparatively early period of renal disease, before any great accumulation of urinary impurities can have taken place; further, urea, the ammoniacal products of its decomposition, and the forms of nitrogenised waste, which may be supposed to accumulate in the blood in renal disease, do not excite convulsions when injected into the blood. These facts seem to show that it is not simply and directly by the presence of renal impurities in the blood that the convulsions are excited. On the other hand, again, there are the facts that uræmic convulsions are most common in that form of kidney disease in which high arterial tension is most marked—the contracted granular form—and they do not occur in the absence of, at any rate, a certain degree of tension. But the consideration which carries, perhaps, the greatest weight is the remarkable effect of blood-letting in uræmic convulsions. The status epilepticus is cut short, the coma is abbreviated, and associated symptoms are relieved. These effects, which are remarkably constant, cannot be from elimination of toxic matter; the blood left in the vessels will be both more watery and more impure from resorption of

liquid from the tissues. They can scarcely have any other cause than diminution of the volume of the blood and lowered pressure in the vessels.

In acute renal dropsy with convulsions, a venesection may not only arrest the convulsion but may exercise a favourable influence on the disease, and in chronic Bright's Disease, nearing a fatal termination, bleeding may prolong life and change altogether the mode of dying from convulsions and coma to a quiet asthenia.

Illustrations of the association of convulsions with low arterial tension are less common, but I have seen in a child of twelve, belonging to a family in which the pulse-tension is low, and who has the family pulse, a momentary convulsion during vaccination. There was no nervousness or fear, but, on the contrary, the operation was regarded with interest. The child, however, dropped as if shot, with a slight general convulsion, and then immediately got up again, looking very much astonished, but not otherwise affected. In another low-tension individual, a young man of remarkable courage, the application of a few leeches to the ankle, which had been injured rather severely, was followed by syncope, emergence from which was attended with brief but sharp general convulsions.

In the following case, seen first with Dr. Godson, Mr. White, and Dr. Kane of Norbiton, and later with the last-named gentleman, the associations of convulsions with an extremely low-pulse tension, and their dependence upon a feeble circulation, seemed to be very clear. The patient, a lady aged about twenty-six, florid and healthy, was confined on Aug. 3rd, 1886. Second confinement, previous one normal. Three weeks before labour she had phlebitis of the right saphena vein with great pain, some pyrexia, but little œdema ; it terminated favourably in about

fourteen days, leaving a hardened vein. Labour natural. Some membranes were retained, but gave no sign of their presence till the seventh day, when the discharge became offensive. There was pain over the abdomen and slight rise of temperature. The uterus was washed out with Condy's fluid, and the next morning the pain had disappeared. A rigor followed the injection of the uterus, and the temperature rose to 105° F. The discharge continuing fœtid, injections were repeated, and the retained membranes came away. An offensive discharge persisted for a day or two, after which the lochia became sweet and normal. The milk stopped fourteen days after labour. From the first rigor dates the commencement of the high temperature. Daily it rose to 104°, 105°, and even 106°; but at the same time it invariably fell to normal some time in every twenty-four hours. There was no regularity in the periods of high and low temperature: some days it would rise rapidly to the maximum, and after an hour it would fall as rapidly; on other days it would remain about normal most of the day. The pulse, always weak, was most rapid and irregular, sometimes quite uncountable. Terrible rigors occurred almost daily, generally commencing when the temperature was low, and invariably followed by rapid rise of temperature. The patient suffered comparatively little from the high temperature, had no delirium, and seemed to be more comfortable than when the temperature was normal, though the weather at the time was extremely hot. On the seventeenth day after labour, phlebitis occurred in the right saphena. This phlebitis continued to give trouble, on and off, for another ten days, and there was considerable swelling of the ankle. The rigor and temperature continuing unchanged in spite of all remedies, it was decided on Sept. 15th, forty-three days after labour, to remove her to another

house. There was the more pressing reason for this, as an expert had decided that sewer-gas entered the house. She bore the move well, and for a time seemed to improve. She had some slight shiverings, but no more rigors, and for several days the temperature never rose above 102°. She had numerous small boils. On the sixth day after the move (Sept. 21st) her temperature suddenly rose to 106°, fell again to normal, and in the evening rose again to 104°. She did not seem worse in any other way. About 10 p.m. I was suddenly summoned, the messenger saying she was dying. I found her sitting up in bed with very flushed face and widely dilated pupils; she appeared quite unconscious, with gasping respiration and very rapid and tumultuous action of the heart. I diagnosed cardiac embolism. Soon after my arrival she fell back and passed into a terrible convulsion, face turned to the left, arm drawn up, and legs extended and rigid. This lasted some minutes, and was followed in half an hour by a second, and again before morning by a third. After the convulsions the left arm seemed paralysed, but this passed off before morning. She was very prostrate, and moaned a great deal, and, if moved, screamed out and applied her hand to the right side of her head. No inequality of pupils; passed urine and motions in the bed. Mental condition very excited; at first aphasic and unintelligible; afterwards noisy—singing, crying, and praying. After this she sank into a very depressed condition with constant tendency to failure of the heart. The slightest attempt to sit up caused giddiness and slight convulsion. She had to be constantly watched, and at times even artificial respiration had to be resorted to. Face sunk and whole aspect that of impending death. Temperature 104°; pulse utterly uncountable. Such was her state on Sept. 26th, fifty-four days after labour. The case was

considered hopeless, but it was determined to persevere steadily with large doses of tincture of muriate of iron and digitalis in gradually increasing doses. The result was highly satisfactory. The patient rallied and steadily progressed from this date. Quantities of small boils appeared, and gave her much pain. Delirium continued for some days, and she had some delusions. The slightest attempt to sit up brought on a slight convulsion. The temperature never again rose above 102°, and in a very few days fell to normal, and remained at that. Ten days after the first convulsion she had another severe one, followed by a return of the mental disturbance. She was free from attacks, excepting slight ones, for about a fortnight, when she had another severe attack; then an interval of three weeks, and a series of terribly severe ones lasting six hours. It being thought that the convulsions might be due to an attempt to restore the catamenia, attention was turned in that direction, and after some time the menstrual flow was established, ushered in by several slight convulsions. All this time her progress to health had been continuous, with the exception of a slight pneumonic attack with some fever and cough, which disappeared after she had coughed up a patch of deep rusty mucus. She had numerous slight convulsive attacks, generally on waking up from sleep; but they left no after-effects, and she steadily improved until Jan. 26th, six months from the commencement of her illness, when she was well enough to be moved to Hastings, and underwent the journey without any fatigue or after bad effect. She is now in capital health, but her pulse has a tendency to become irregular, and she has occasional attacks of threatened convulsions. The above account was supplied to me by Dr. Kane in April, 1887. I first saw the case on October 9th, when the pyrexia had been overcome and the convulsions had set in,

and had no hesitation in referring them to the exhausted state of the heart, which could not be felt, and could scarcely be heard, while the pulse was all but imperceptible. The fact that the patient could scarcely be raised in bed without bringing on an attack of convulsions was of decisive significance. There had no doubt been an embolism of some vessel in the right hemisphere which rendered the cerebral circulation more liable to disturbance. This patient has been lost sight of, but at a recent period was still subject to fits.

A gentleman, aged fifty-two, consulted me on account of shortness of breath on going upstairs, and especially on going up the steps from the railway-station. He had become so nervous on the subject, that his heart began to palpitate and his breath to go before he came to the foot of the stairs. He had palpitation also occasionally at night. He looked the picture of health and younger than his years, was stout and of rather high colour, ate and slept well, and had a regular action of the bowels. He rode a tricycle, and had much fresh air and exercise. The pulse was frequent—90 to 108—small, short, and extremely compressible. The heart was partially covered by lung, did not appear to be enlarged, and gave no impulse or apex-beat ; the sounds were short and approximated, the second following the first too quickly. The first sound was audible not only at the apex and right second-space, but also in the neck, where both sounds were remarkably distinct. He spoke of having had a fainting attack ; and, when this was inquired into, it was described as having occurred as follows. It was in August, and he had had a tricycle ride, had washed and changed, when he thought he would like a glass of beer, which he had not tasted for years. Shortly afterwards he felt a peculiar tingling in his feet, and had just time to

lie down when he lost consciousness, and did not come to himself for forty minutes or more. This could not have been a syncopal attack, and, whether convulsions occurred or not, was epileptoid. It was further learnt, on inquiry, that he had had several similar attacks within the last few years, always beginning with tingling in the feet, and attended with complete loss of consciousness lasting, on an average, twenty minutes. He had had no fits as a boy, or until the occurrence of those referred to.

The pulse and epilepsy.—I am not without hope that a careful study of the pulse, and of conditions of the circulation made known through it, may be of service in furnishing indications for the treatment of epilepsy. It is attended with interest, throws light on different forms of epilepsy, and serves as a guide in prognosis. Epilepsy is apparently the result of instability in the highest nerve-centres, the cells of the cerebral cortex permitting of an indiscriminate general or partial discharge upon lower centres of nerve-force, which normally ought to be set free only in definite degree and in well-defined direction, in response to given stimuli. Such instability is obviously a nutritional defect, which may be due to the inherent want of constructive energy in the nerve-cells themselves, or may be the result of blood which cannot furnish the proper pabulum, or of an inadequate supply of blood. Even in the case first supposed, a regular and ample supply of blood, under sufficient pressure to cause a due exudation of nutrient material and of proper composition, will be important, while in the other conditions supposed it will be remedial. But, given more or less of instability, this does not of itself start the convulsive explosion; some exciting cause must be applied. At one time this was supposed to be arterial spasm in certain convolutional areas; but this hypothesis, which

never seemed to me to be tenable, has gone out of fashion. There are, however, facts which seem to show that circulatory conditions have much influence in determining the occurrence of attack. For example, the great frequency with which fits come on in the night, sometimes on first going off to sleep, when the horizontal position and the anæmia of the cortex attending sleep produce great changes in the intracranial circulation; or more frequently towards morning, when the general circulation has slackened down, as it does during sleep. Another favourite time for attacks is soon after rising in the morning, when the erect posture, the exertion of dressing, and exposure of the surface to a lower temperature make great demands on the circulatory mechanism for adjustment to altered conditions.

It is not my intention to discuss the questions which might be raised on the subject here alluded to, but I desire to call attention to a provisional conclusion to which I have been led by my own limited observation. This is, that in essential epilepsy—the epilepsy which comes on about the period of puberty or during adolescence, the epilepsy met with in neurotic families, and in which the nerve-cells may fairly be assumed to be inherently unstable—the arterial tension is low and variable or fluctuating. I am unable to affirm that when the attacks are suspended, as they may be for months, the pulse-tension is improved; but it is worthy of remark that pregnancy is not unfrequently attended with immunity from fits; and, as is well known, one effect of this condition is high tension in the pulse. A low-tension pulse, therefore, has become with me a mark of unfavourable import in epilepsy.

On the other hand, when the pulse-tension is decidedly and constantly above the average, it has seemed to me that the epilepsy has been amenable to

treatment. Epilepsy with high arterial tension often comes on at a later period than the epilepsy which has its source in a radical weakness of the nervous system; and in most of the cases I have met with the first fit has occurred after the age of twenty; and not uncommonly there have been peculiarities, such as a number of attacks close together, with long but irregular intervals, or some well-marked exciting cause. The fits may, however, date from the usual age at which epilepsy sets in. Many years since a surgeon-major in the army brought his son to me on account of epileptic attacks, which had compelled him to leave the navy. He was a fine, strong, healthy-looking lad, and had a large pulse, not easily compressed. Under treatment directed to the reduction of arterial tension which was extremely high for his age, the fits, which had been frequent, ceased to come on. He studied for and got into Sandhurst, obtained a commission in an infantry regiment, and has since served through the Afghan campaign. So far as I know—and I have seen him quite recently—he has only had two attacks since he entered the army: one in Afghanistan, when, together with trying work, he had bad food and water; and another in Ireland, after great fatigue and a bout of dissipation.

Senile epilepsy is, according to my experience, associated with high tension and scarcely ever fails to yield to a regulated diet with a restricted amount of animal food and little stimulant, aperients and other eliminants being given according to the requirements of the particular case.

The introduction of bromides in the treatment of epilepsy has not in my opinion been an unmixed advantage. It is true that in many instances there has been an apparent cure of cases which seemed hopeless, and that in many more the number of fits has been so far reduced as to restore the sufferer to

comparative happiness and to a useful place in society, but on the other hand we have been too easily content with this result of diminishing the number of attacks; the administration of bromides has become a matter of routine, and under the idea of diminishing reflex excitability we have come to neglect the higher aim of increasing the stability of the nerve-cells by improving their nutrition. The epileptic who enjoys a certain degree of immunity from attacks in virtue of large doses of bromide is on a lower platform than one who obtains such immunity by means which raise the tone of his nervous system, and the immunity may be purchased too dearly. Not to speak of the disfigurement produced by the bromide rash, or of the derangement of the digestion, or of the anæmia, which are common results, I have seen patients reduced to a condition scarcely distinguishable from general paralysis by bromides, and all minor degrees of intellectual and nervous debasement, from which condition they have been rescued by discontinuing the drug and adopting a different treatment, the fits in several instances also ceasing. The legitimate use of bromides I conceive to be for the purpose of palliation, of staving off attacks which have become too frequent, and so of gaining time for a study of the peculiarities of the case, its true causation and ultimate pathology, and for the application of remedial measures which shall go to the root of the disease. When the fits are separated by intervals of months, I can see nothing but harm in giving bromides regularly. If, in such a case, an exciting cause can be identified, bromides can be usefully employed to parry its influence and avert the attack; but no such identification is possible if the bromides are being taken constantly. Only by careful inquiry into the antecedent circumstances of each fit can this be done, and attention to the administration of the drug takes the place of attention to other matters. A very

common history is that the absence of attacks begets carelessness in taking the remedy, and to this, or some omission of a dose, is attributed the next attack, upon which it is taken for a time with renewed diligence, only to be again neglected.

It is not pretended that the pulse furnishes the only clue to the rational treatment of epilepsy or that its teachings are applicable to all cases. Other indications are to be sought in deviations from normal functional action of whatever kind.

Closely allied to epilepsy, and occurring under the same conditions as senile epilepsy, is sudden loss of consciousness, in which the patient falls as if shot without any trace of convulsion, recovering again instantly. All that he knows of the attack is that he finds himself on the ground, and he is usually able to get up and walk at once. This I have met with in elderly or old men, the subjects of old-standing tension with dilated, thickened, and degenerate arteries. The patient may look hale and strong and may exhibit both mental and bodily vigour. The attacks can scarcely be due to anything else than a momentary cessation of the circulation in some part of the brain, either in an area where blood-stasis does not give rise to convulsions, or lasting for too short a time for their occurrence. In one case of the kind the patient subject to these attacks suffered from acute dilatation of the heart after imprudent over-exertion, and at a later period, after complete recovery from this, from paraplegia, due to innutrition of the lower end of the spinal cord. In other cases also I have seen paraplegia supervene.

Maniacal delirium.—I have several times seen violent maniacal delirium associated in such a way with convulsions as to suggest the conclusion that a minor degree of the disturbing influence which caused convulsions had given rise to the maniacal excitement;

after bleeding, for example, for uræmic convulsions; before and after, or apparently instead of, uræmic convulsions; in syphilitic disease of the brain. In the following case the connection between the state of tension of the pulse and ungovernable excitement was recognised by several observers over a long period. The patient was under the care of Dr. Ranking, at Tunbridge Wells, and Dr. Marcus Allen, at Brighton, and in town under Dr. Seton and Dr. Coates, with whom I saw her weekly from March to July, 1884, and again in March, 1885. Dr. Ranking, who has kindly supplied me with the particulars, was called to her in May, 1882, when she was convalescing from a third attack of slight left hemiplegia. She was very nervous and apprehensive, and had dilatation of the heart, with an unstable pulse of virtual tension. During the summer she had several anginoid attacks, which were relieved at once by nitro-glycerine, and in the winter attacks of congestion of the lungs, with partial suppression of urine and albuminuria. One day in July, 1883, she became suddenly excited, with delusions, which lasted some time, but went off after the administration of one-hundredth of a grain of nitro-glycerine. Later in the summer she got into an excited, unsettled, suspicious and violent state, which persisted, together with high tension of the pulse. Once she was comatose for twenty-four hours, but recovered after nitro-glycerine, and at once became maniacal. In the winter of 1883-84 she was under the care of Dr. Marcus Allen at Brighton. It was found by observation that her mental condition was always worst when the pulse-tension was high, and that the only way of keeping it down was to cut off all meat and feed the patient chiefly on milk. This was confirmed by our experience when she was brought to town. When the tension was high she was suspicious, abusive, violent and unmanageable. When

it was normal she was cheerful and tractable. Mercurial aperients were constantly needed in order to keep down the tension and regulate the bowels. Ultimately she died with symptoms of meningeal hæmorrhage.

With advancing years there comes a liability to many forms of cerebral affection. Some are examples of structural change which are clearly traceable to interference with the normal blood-supply. Among such changes are local or general softening from thrombosis of individual arteries or veins, or general obstruction to the blood-supply from atheroma, affecting all the cerebral arteries. Very commonly the existence of atheromatous change in the cerebral arteries can be inferred from the evidence of advanced degeneration in the radials. When the fingers are made to carry the skin to and fro along the vessel with varying pressure, it is found to present irregularities, bulgings, and tortuosities, and in its coats can be felt calcareous patches or rings ; or the artery may be converted into a calcareous tube. Such a condition of the arteries at the wrists seldom exists without corresponding alterations in the arteries of the brain. The local arterial change is not, however, the sole factor in the production of the lesions. A state of blood which renders it prone to coagulate or deposit fibrin will predispose to the formation of a thrombus at any point where the walls of the artery are diseased ; and want of propulsive power in the heart, giving rise to languid movement in the blood, may antedate the occurrence of degenerative changes in the nerve-centres attributable to widely distributed disease in the vessels. Over-fatigue, or a slight indisposition, such as an attack of diarrhœa, is thus sometimes the immediate occasion of hemiplegia or aphasia, or other form of paralysis, by so far weakening the action of the heart that an opportunity is given for thrombosis to take place. But an impeded cerebral circulation may,

in course of time, so far modify the nutrition of the brain as to give rise to functional affections without structural lesions; and this, in my opinion, is the mode of causation of some forms of insanity which come on late in life,—such, for example, as senile melancholia. The term "senile" may have two meanings: it may designate an age or be descriptive of a kind of change. The term "senile" is unobjectionable if it is meant to apply simply to the period of life at which this affection is met with; but it is often understood to imply a certain character of change, and its frequent employment to designate degeneration has led to the common idea that "senile" and "degenerative" are convertible terms. This does harm in two ways. Senile changes, if degenerative, are irreversible; and, if this view is taken of dementia or melancholia, there will be no object in careful investigation of associated conditions. No other cause than senile decay will be looked for, and no individual treatment will be adopted, based upon the peculiarities of different cases. Furthermore, the reproach of insanity or of neurotic tendencies may be attached to families, when the derangement of the cerebral functions may be as much a result of vascular disease as cerebral hæmorrhage. A family liability to melancholia may consist in hereditary high tension of the pulse, just as a liability to apoplexy may be due to family gout. Now, the complete recovery witnessed in many cases of melancholia is proof that there cannot have been any structural degeneration, and an example like the following may be worth relating.

Some four years since I was asked to see periodically a gentleman aged about sixty-five, who for four or five years had been under the Commissioners in Lunacy on account of melancholia. He had had delusions as to conspiracies against his life, but such

delusions as remained related chiefly to wilful and malicious injury inflicted upon him and attempts to destroy him in an asylum. He spent nearly all his time in bed, scarcely ever left his room, and never went out of doors. He was well nourished, and, excepting that he was etiolated by his long confinement to the house, looked well; but the pulse was extremely small, soft, and short, and the action of the heart extremely weak. He had eczema, and suffered from constipation and want of appetite, and treatment was prescribed for the relief of these symptoms. His general health improved, but his mental condition remained much the same, and in particular the death of a son made little impression on him. In June, 1886, he suddenly shook off his delusions and became perfectly sane and cheerful. Simultaneously, whether as a cause or as an effect, the pulse improved, but never reached an average volume or tension. In October he remained well, and after examination by Dr. Maudsley was discharged from his lunacy. He transacted business, arranged his affairs, and was himself in every way, exhibiting no irritability or depression, and free from eccentricities or delusions till in February, 1887, after excitement and over-exertion, he unfortunately became aphasic from thrombosis of the vessels supplying the cortical speech centre, which was followed by a relapse into melancholia and speedy death.

Pulse and melancholia.—The connection between melancholia and its allied mental states and conditions of the circulation is more direct and decided than can be traced in epilepsy. An unbroken series of gradations can be traced from the irritability and depression of spirits attending functional disorder of the liver and other ailments up to complete melancholia with delusions. In the case of temporary hepatic derangement, the state of the temper and spirits might be attributable to the retention in the blood of the

impurities which tinge the eyes and complexion, these acting as a poison; or to some reflex influence inhibiting cerebral functions, or deranging the cerebral circulation by setting up contraction of some of the arteries. When, however, the mental depression is more pronounced and persistent, these explanations are found not to apply; there may be, in the first instance, constipation, a furred tongue, sallow complexion, large liver, etc. But when these evidences of deranged function are removed the mental condition does not clear up. If the symptoms, therefore, are due to any somatic cause and are not the outcome of a primary cerebral affection, this cause is something more persistent than the functional derangement or reflex disturbance mentioned. This has appeared to me to be protracted arterial tension, or, if this is not itself the cause, it is at least the index of the state of system on which the mental condition depends.

The method by which high arterial tension may influence the cerebral functions may be conceived to be as follows. The resistance in the peripheral vessels calls for increased contractile force on the part of the left ventricle, and there is a response by a certain degree of hypertrophy. In the course of years, however, the resistance increases, degeneration of the capillaries and thickening of the arterioles being superadded to the original loss of due relation between the blood and tissues, while the heart no longer gains in strength. With, then, the same, or somewhat diminished, driving power, and increased resistance in the periphery, there will be a slower onward movement of the blood. The pulse may be equally strong, may even seem to be more incompressible, but the capillary circulation will be sluggish. This will be the case throughout the system, but it will not give rise to appreciable effects in most of the structures and organs; in the brain, however, as has already been

said, functional activity and efficiency are absolutely dependent upon a due supply, not only of nutrient material but also of oxygen, and this fails when the flow through the capillaries is sluggish.

In a very large proportion of the cases of melancholia coming on late in life, the evidence of persistent high tension of the pulse has been most marked; and, when this has been the case, it has seemed to me that persevering endeavours to diminish the peripheral resistance, and at the same time to strengthen the action of the heart, have been more successful than any other line of treatment. The object is so to relieve the heart that it may no longer be mastered by the obstruction in the general capillary circulation; there will then be a general acceleration of the flow of blood through the tissues, and by the increased supply of blood to the brain, its nutrition and functional efficiency may gradually be restored. The possibility of this result and the time required for its attainment will depend on various conditions. There must be a capability on the part of the heart to resume its control over the circulation; it must not be degenerate or worn out. The state of the cerebral arteries, again, will have an important influence. If they are extensively diseased, the access of blood to the convolutions may be barred, even when the circulation elsewhere is good. Further, the change in the nervous elements must not have gone too far; the longer they have been subjected to the deteriorating influence of imperfect blood-supply, the longer will be the time required for the reversal of these effects. Of these three sets of conditions we can only estimate the first by examination; with regard to the others, the basis of our judgment must be the history. Speaking generally, the more acute the attack and the shorter its duration, the better will be the chances of recovery.

Cases of this kind, many of which have come under my notice, do not lend themselves to narration, especially when seen in consultation only once or twice, and I shall not, therefore, attempt to give examples. I may, however, relate an occurrence with regard to one such—a most distressing case of religious melancholia in a lady of about sixty—with extreme high tension in the pulse. I had explained my views to Dr. Baines, with whom I saw the patient, and had recommended, among other measures, a series of mild calomel purges, when the sister of the patient joined us in order to learn our opinion. Before hearing this, however, she said there was one more fact which she ought to have told us, namely, that their mother, at very nearly the same age, had suffered in exactly the same way. It seemed as if my hypothesis of the relation of the melancholia to the state of the circulation was at once overthrown, and with it my favourable prognosis. "But," she continued, "in those days they gave calomel for everything, and it was prescribed for her, and she got quite well." Our patient also recovered, only, however, to relapse some time later.

Melancholia, associated with low—extremely low—pulse-tension, has, in my experience, usually proved incurable, and has in several instances gone steadily from bad to worse to a fatal termination. The case related a few pages back is the only instance of recovery I have met with.

The pulse and common paraplegia.—The subject may be brought to a conclusion by a brief notice of an affection of the lower end of the spinal cord, especially as it furnishes a sort of parallel to the production of melancholia by derangement of the circulation. The late Dr. Moxon, whose loss those who knew him well will never cease to deplore, pointed out in his brilliant and original "Croonian Lectures" that

common paraplegia, as he called it, was explained by anatomical facts. The spinal cord receives its blood-supply by means of the arteries which reach it along the nerve-roots. These, in consequence of the downward elongation of the spinal canal beyond the cord, get the more oblique and longer from above downwards, and at the cauda equina are many inches in length, so that the arteries of the lumbar enlargement which occupies the lower part of the dorsal division of the spine have to travel upwards for this distance from the foramina in the lumbar and sacral regions. When, then, the circulation becomes languid, the mechanical disadvantages of this arrangement make themselves felt. The blood is not propelled with sufficient force to travel up the long narrow vessels against gravity from the lumbo-sacral foramina to the cord, stagnation occurs, and the nutrition of the cord suffers.

The symptoms attending the early stage of paraplegia, due to failing circulation in the lumbar enlargement of the cord, are very interesting. As the nutrition of the lower end of the cord begins to suffer, there is at first muscular weakness and loss of control over the legs only after a night's rest. The patient has some difficulty in standing and walking steadily when he first gets out of bed, but after he has moved about a little the legs regain power and he can walk perfectly. A similar state of things is observed with regard to the bladder. He cannot pass urine on rising, but when he has had a little walking he empties the bladder easily. Whenever he sits down for any length of time during the day there is more or less impairment of mobility and strength in the lower extremities, which quickly passes off with movement. Sensation is not affected at first, but there may be feelings of numbness. The coming on of the weakness during the night is due to the slackening down of the

circulation, which takes place during sleep, and is paralleled by the morning depression in melancholia and debility.

I have met with this train of symptoms at the two extremes of high and low pressure. When there is high pressure it is that the general resistance in the periphery has overtaxed the powers of the heart, so that the whole circulation is sluggish, and the languid flow is most easily brought to a standstill where the difficulties are greatest. Usually the symptoms come on very gradually, but I have known their onset to be determined by the occurrence of acute dilatation of the heart, and have seen cases in comparatively young men suffering from high arterial tension in which paraplegia came on almost suddenly after prolonged over-work. The patient may have previously suffered from other ill effects of high arterial tension.

When the tension is low and the heart weak, no explanation of the impeded circulation is needed.

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