GRADUATION THESIS

for

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"Observations on the Pulse"

1. Analysis of a Sphygmographic Tracing.
2. Rhythm of the Pulse.
3. The Pulse in Valvular disease of the heart.
4. Pulse Tension.

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The Pulse physiologically and clinically considered affords one of the most interesting studies in clinical medicine. A thorough appreciation of its significance in health and disease enables the physician to grasp the many interesting phenomena that occur in its many changing phases, and to draw conclusions from its departures from the normal type, which enable him to diagnose, treat, and observe the effects of treatment in the course of his clinical career. The subject has been elaborately treated in the many learned discourses on Physiology and medicine, and books especially devoted to it in its morbid conditions are numerous.

In selecting such a hackneyed subject for a graduation thesis my object is not to endeavour to rival those excellent treatises, but to grasp for my own edification and instruction one of the salient factors in clinical medicine. Any man may write a book for others to read, but however thorough the book may be it is useless as an educating factor if the reader does not endeavour to grasp and apply its principles, and observe for himself the points which others may indicate. In order to become a competent practical member of the noble and useful profession of medicine, votary of the science, must be not only a speculative absorber of other men's facts and experiences, but must be on the outlook for such
facts clinical and other that may fall to his humble lot. My only apology for taking up such an extensive subject is the perhaps common one, viz:— the absorbing interest the subject of pulse & circulatory system has always had for me. Many of the problems presented by the circulatory system have been exhausted by abler exponents, but as before said each member of the profession must go over the ground for himself before he is in a position to enable him to become a truthful and useful observer. I do not presume to exhaust the subject within the limited space allowed for in a graduation thesis, that would require a few more years of study and observation. I shall confine my attention to such facts as have directly appealed to me in the course of my clinical career.

The following heads will comprise the greater bulk of my thesis.

1. Analysis of a Sphynmographic tracing.

2. The Pulse in valvular disease of the heart.

3. Pulse Rhythm.

4. Pulse tension as a guide in Diagnosis, Proposal and Treatment.
ANALYSIS OF A SPHYGMOMORPHIC TRACING.

Preliminary Considerations

All the tracings shown here have been taken by means of the Duhamel's instrument, the same instrument has been used for all, so that for purposes of comparison the tracings are fairly reliable.

I have tried in several cases to get tracings under different pressures but have concluded that with this form of instrument any attempt to adjust pressure is bound to fail. The artery can be completely stopped by tightening the arm strap, and it alone can be used as a medium for applying pressure.

Under such conditions where the strap is not graduated it is useless to endeavour to graduate the pressure in ounces by means of the spring & pressure lever. So far as my experience in the use of the instrument goes, it is not possible to say accurately the amount of pressure in ounces there may be on the artery; but for comparative purposes it may be employed approximately. An important point in the use of the instrument is to see that it is always used upon the same part of the artery in making a series of observations. In this relationship it is well to bear in mind the anatomical position and variations of the vessel from which one makes observations.

(a) There may be a smaller artery on one side than on the other.
(b) Abnormal distribution of one or other radial may be a cause of difference in the two tracings. The radial of one side may be much smaller on account of a larger ulnar than normal.

(c) The Radial may bend round the Bone at a higher level than normal, and in such a case the vessel will be represented by a small artery - the Superficialis Valve. Of this abnormality I shew several tracings showing that it is possible to get a tracing from the latter vessel which might easily lead to confusion when comparisons are made.

These tracings are a marked difference in the length of the upstroke. It would be hardly possible to mistake such a case for one of aneurism because an examination of the two tracings would show that both are regular in all respects except the difference in the length of the upstroke. One might expect some obstruction of the radial of one side, or an anatomical difference in the distribution of the two vessels. An apparent difference in the tracings from the two vessels may arise when the artery is more superficial
on one side than on the other, or from the presence of a more marked curve on one side. A real difference in the pulses of the two sides simulating more closely than any of the above the effect of aneurism will be produced by the pressure of a tumour of any part of main vessels of the arm, subclavian, Axillary, or Brachial.

Lastly one has to bear in mind the occurrence of any previous injury to the arm or fore-arm. I have met one such case where a badly united fracture of the radius led to an almost complete obliteration of the radial pulse on the left side.

Another important point in the use of the instrument is the management of the clock-work, if it be allowed to run down, the rate of the smoked paper is much slower, and the tracing shows an apparently rapid pulse. I make it a rule never to allow more than 2 papers to pass through without winding up the instrument. I shew 2 tracings here to illustrate this form of error:

[Tracings shown]
The Normal Pulse.

The pulse as represented by a tracing taken by means of the Sphygmograph shows the following characters. It consists of a complete series of curves, each series corresponding to one beat of the heart and to one complete cardiac revolution, i.e., the time which elapses from the commencement of one ventricular systole to one ventricular diastole. Each individual pulse curve may be divided artificially into the following parts:

(a) Line of Ascent.
(b) An Apex.
(c) Line of Descent.

The line of ascent or upstroke represents the sudden distention of the arterial system which is produced by the contraction of the left ventricle at the commencement of the ventricular systole i.e. when the aortic segments are suddenly opened. It is probably also due to a certain extent to the inertia of the instrument; and in some cases where the arterial wall is more rigid than normal, to impulse or shock. The direction of the upstroke may be vertical or oblique and varies according to circumstances.

(a) The suddenness of the ventricular systole influences the direction, as seen in cases of Aortic regurgitation it is vertical, nor-
mally there should be a slight curve upon it.

(b) The condition of the Aortic segments also exercises an influence over the direction.

(c) In some degree the facility with which the blood wave is propagated from the base of the aorta to the radial artery.

(d) The condition of the arterial coats, it is readily seen how this condition may influence the character of the upstroke.

In the normal tracing the upstroke is nearly vertical for the contraction of the ventricle occurs suddenly, and there is no undue resistance in the aortic segments. When the ventricular contraction is more sudden than in health, as in cases of Aortic regurgitation and in cases of Cardiac excitement the upstroke is quite vertical or may even slope backwards.

Vice versa' when the ventricular contraction is slow and hesitating as in cases of cardiac debility, when the aortic cusps are rigid, when they are obstructed either by external or internal causes, such as the pressure of a tumour, the pressure of an atheromatous patch or when a globular aneurismal dilatation is
situated between the heart and the vessel, the upstroke may be oblique.
The height of the upstroke represents the degree of distension and depends upon:

1. The force and (to a less degree) the suddenness with which the aortic cusps are raised, it represents the force and suddenness of the contraction of the left ventricle, less the resistance offered by the aortic valva cusps.

2. The extensibility of the arterial walls which in its turn depends upon the condition of the arterial tissues and the state of the vasomotor system.

3. The amount of pressure which is applied to the artery namely: pressure exerted by the spring of the instrument, plus that of the band of the instrument.

The apex of the tracing or the primary ventricular wave, (Percussion wave of Mahomed) is in the great majority of cases pointed, according to Bramwell, it is always pointed. A rounded apex is one of aneurysm atheroma or Aortic Stenosis.

The line of descent, in the normal pulse tracing it is gradual and is interrupted by a number of secondary wavelets, each of which bears a significance in the interpretation. The most important is the dicrotic, and around it much discussion has arisen.

The direction of the line of descent depends upon:

(a) The facility with which the blood passes out of the arteries, most interesting in relation to pulse
tension.

(b) The rapidity of the heart's action.

(c) The condition of the arterial coat.

The dicrotic wave, or the Aortic Systolic wave is always present in a normal tracing and corresponds to the portion of the cardiac cycle that immediately follows the closure of the Aortic valve cusps, while the part of the tracing which immediately follows it and which is generally believed to correspond in time to the closure of the aortic segments, is termed the aortic notch.

Where the vaso-motor tone is very good, and the arterial tension high, as it is in some healthy persons, the dicrotic wave is very feebly marked or altogether absent.

As a general rule the secondary elevations appear only on the descending limb of the primary wave, as in most of the curves given, and the whole curve is then spoken of as "Kata-crotic", sometimes however, the first elevation or crest is not the highest, but appears on the ascending portion of the main curve, such a curve is spoken of as "Anacrotic" such tracings may be obtained from atheromatous patients.
Taking the secondary elevations in order of their importance we come to a consideration of the dicrotic. It is the most frequently present in a tracing, and is more or less distinctly visible in all sphygmo-grams, and may be seen in those of the Aorta as well as of other arteries. Sometimes it is so slight as to be hardly observable, at others it may give rise to the appearance of a double pulse and the pulse is then described as dicrotic. Hence it has been called the dicrotic wave, the notch preceding the elevation being spoken of as the dicrotic notch. The dicrotic wave is present in a tracing obtained by opening a vessel in any animal and allowing the blood to record a wave against a moving sheet of paper.

A great deal of discussion has been waged around the cause or causes of the dicrotic wave, and it is hardly my intention to enter into a detailed account of the theories advanced by scientists, to prove their interpretations, I shall briefly indicate the most commonly accepted views.

According to Michael Foster, the explanation is as follows:

"When a rapid flow of fluid is suddenly stopped, a negative pressure makes its appearance behind the column of fluid, in a rigid tube this leads simply to a reflux of the fluid, in an elastic tube like the aorta its effects are complicated by a second factor..."
in the elastic reaction and inertia of the walls of the tube. Upon the sudden cessation of the flow the expansion of the aorta ceases, the vessel begins to shrink, and the lever placed on it falls from "A" onwards in the pulse curve.

This shrinkage is in part due to the elastic reaction of the walls of the aorta, but is increased by the suction action of the negative pressure spoken of above. In this shrinkage the Aorta overshoots the mark through the inertia of its walls, it is carried beyond its natural calibre, i.e. the diameter it would assume if outside and inside pressure was equal, it shrinks too much and consequently begins to expand again.

This secondary expansion causes the secondary rise of the lever up to C, that is the dicrotic rise. In thus expanding again the aorta tends to draw back again the column of blood towards the heart, which by loss of momentum had come to rest, or indeed under the negative pressure was already undergoing a reflux. In this secondary expansion moreover, the aorta is by the inertia of its walls, aided by that of the blood again carried so to speak beyond the mark, so that no sooner has it again expanded and fil-
led with fluid to a certain extent, then it again begins to shrink as from C onwards, and this shrinkage may again be followed by an expansion and a shrinking given rise to a post-dicrotic wave or post-dicrotic waves.

In the above explanation no mention has been made of the semi-lunar valves, the closure of the valves has to be taken rather as an effect than a cause of the dicrotic wave.

The other view advanced in explanation of the dicrotic wave is as follows:—

The column of blood comes to rest in consequence of the great pressure in front, i.e. the arterial pressure exercised by the coats of the vessels, the aorta recoils upon the column of blood within, and tends to force it back towards the heart, the reflux into the ventricle is prevented by the semi-lunar valves which close and effectually stop the backward flow, moreover the column of blood receives a fresh impetus from the impact against the valves, and a new wave of expansion is originated which reinforcing the natural tendency of the arterial elastic walls to expand again after their primary shrinkage produces the Dicrotic wave "C".

Dr. Galabin explains the mode of production of the dicrotic wave in the following manner:—

"The first cause of the dicrotic wave is that which has been generally accepted as depending upon
the aortic valves, for let us consider a section of
the artery close to the valves, when the influx from
the heart suddenly ceases at the end of systole, the
fluid for a moment continues to flow away out of the
section on account of its acquired velocity, and the
pressure of the section rapidly falls and the artery
contracts. As soon as the velocity of the fluid is
checked by the pressure in front, a reflux takes place
which being stopped by the semi-lunar valves causes a
second increase of pressure and a second expansion.
This is propagated as the dicrotic wave into the peri-
phery and may itself call out a second similar
oscillation or tricrotic wave which is not frequently
seen in the pulse".

Dr. Galabin's theory seems to combine both the
above stated explanation.

Even in the total absence of aortic valves the reflux
meeting with the current entering the ventricle may
cause a second increase of pressure or dicrotic wave;
although this will be much less than in the former
case.

In criticising the theories advanced, one is greatly
hampered by the difficulty of doing experimental work
which I think would help to clear up some of the
points in doubt. Considered from a clinical stand-
point one is inclined to adopt the theory which attri-
butes the dicrotic wave to the impact upon the semi-
lunar valves. In advanced aortic incompetence the dicrotic wave is only feebly marked, but I have seen few specimens that do not show a feebly marked notch, in such cases one cannot take for granted that the valves are totally inefficient, and if they act at all then the dicrotic wave present may I think fairly be attributed to the action of the damaged valves. If one could be certain in a particular case that the valves were totally destroyed, and on examination a dicrotic wave was present, then the theory would have to be abandoned and one of the others adopted. Experimental work in this direction is badly required. An experiment to clear up the matter would be rather a difficult one, the valves would require to be destroyed in a similar manner to that employed in determining the factors giving rise to the second sound of the heart, then it would be necessary to obtain tracings from the arteries, this could be done by opening a vessel and allowing the artery to cord itself upon a moving sheet of paper. On the other hand the explanations of Drs. Foster & Galabin are so well put, and appear so strongly to the mechanical instincts that one cannot totally discard them, particularly as later on I shall endeavour to explain some morbid conditions of the pulse on a somewhat similar basis. Dicrotism is prompted by a sudden action of the heart, and also by the distensibility of the arteries, by low-
ness of arterial tension and by freedom of outflow.

D'. Galabin thinks that in considering the origin of the dicrotic wave, sufficient attention has not been paid to the important part played by the inertia of the fluid, and to the fact that the aortic valves although extremely important are not absolutely essential.

It appears that there are three main factors in the production of the dicrotic wave, namely:

1. Inertia of the fluid.
2. The elastic condition of the arterial walls.
3. The action of the Aortic valves.

That all 3 factors play an important part in its production is supported by the following considerations. Sudden contraction of the heart leads to a pronounced action of the inertia of the fluid, and a marked dicrotic wave, especially in a low tension pulse. The elasticity of the arterial walls comes into play especially in pulses of low tension. In a high tension pulse especially in renal disease, but also in other conditions affecting the arterial coats, where there is a thickening of the media and an increase in the constraining power of the wall, and where we may reasonably assume that the elasticity though greater is less perfect in quality, i.e., does not so readily respond to alterations of pressure within the vessel, we find the dicrotic wave is less perfectly marked.
Note also that in atheroma where the elasticity is less perfect the dicrotic wave is feebly marked. Taking the aortic valves as a factor in the production of the dicrotic wave, we have only to refer to cases of aortic regurgitation to clearly demonstrate the important part played by those factors in its production. In those cases we have the other 2 factors acting freely, yet the dicrotic wave is only feebly marked, the inertia of the fluid is greater on account of the more vigorous action of the left ventricle, and the elasticity of the vessel walls is not impaired at least in the early stages.

Dicrotism considered from a clinical point of view

The Dicrotic wave is absent or feebly marked in free aortic regurgitation.

It is also feebly marked in some pulses of high tension, and in some cases in which the elasticity of the arteries is much impaired as in advanced atheroma. Degrees of dicrotism and their significance.

Various degrees are described indicating the relation of what is known as the "dicrotic notch" to the respiratory line of the tracing.

(a) When the dicrotic wave is well marked, but the aortic notch "C" is above the base line the pulse is called dicrotic.
(b) When the e^=^fe^e^ notch "C" reaches the level of the base line A.B., the pulse is called fully dicrotic. In this condition the artery is apparently no more distended at the end of ventricular systole than it is at its commencement, and it is apparently less distended than it is during the ventricular systole.

(c) When the aortic notch "C" sinks below the level of the respiratory line A.B., the pulse is called Hyperdicrotic.
The Predicrotic, true tidal wave.

The next of the interruptions in the line of descent is the Predicrotic wave. It occurs between the apex and the dicrotic wave. It occurs during the contraction of the ventricle and is best marked in a hard pulse. The essential factor which favours the production of the tidal wave is increase of arterial tension during the ventricular systole. It is best seen in aortic regurgitation where a powerfully acting ventricle forces a large quantity of blood into the arterial system at each ventricular systole producing high tension during systole with a well marked predicrotic wave, but in which the arterial tension during diastole is extremely feeble.

It is feebly marked or absent in cases of low arterial tension.

The explanation generally accepted of the predicrotic wave is, that it is due to the momentum acquired by the lever of the instrument bringing it down on the artery before it has ceased to expand.

The other waves which are seen on the line of descent are clinically of no importance and therefore do not deserve much consideration. Foster in his text book of Physiology refers to them, and says that with more perfect forms of instruments we may be able to get
some important indications from their occurrence.

Effect of Respiration on the Pulse.

This is a subject possessing great interest when we consider the intimate relationship that exists between the Respiratory and circulatory system and how they are dependent upon one another. We know the great part played by the elasticity of the lung in promoting the flow of blood into the right side of the heart, and the amount of resistance the lung can offer to the right ventricle when it is diseased.

I think a brief consideration of the physiological relationship existing between the two systems will be of service when we come to consider morbid conditions of the circulatory system dependent upon the respiratory system. This will necessitate a description of the anatomical relations of the heart to the lung, and the consideration of the nerve mechanism of respiration and circulation. But these details I shall not enter into here. I shall endeavour to demonstrate by means of a series of sphygmographic tracings the effect of the different stages of respiration and shew what effect these have when forced inspiratory & expiratory efforts are made.

The following tracings illustrate some of these changes in a pulse of normal high tension.

Breath held at the end of inspiration; Note change in character of 1 wave.
Effect of deep inspiration quickening of the heart's contractions.

A marked change occurs in the pulse wave, it becomes distinctly dicrotous.

Shows a still more marked change.
The series of tracings show the influence of respiration on a healthy pulse, and are, I think sufficiently varied to enable us to understand the great influence the action of the lungs must exercise in cases of cardiac disease, a badly balanced heart may at any time become thoroughly disordered by inflammatory mischief in the lungs.

The base line in normal conditions is a straight one. During deep inspiration and expiration even in normal cases various alterations may occur in presumably healthy individuals. The pulse tracing of individual beats may be made to alter according to the particular phase of respiration going on at the time, a high tension pulse may become dicrotic in some cases when ordinary breathing is departed from.

These many interesting conditions are not at present under consideration, though they may serve to throw light on the great influence the respiratory function has over the circulatory system. I want to refer to cases especially where the base line assumes an undulating appearance instead of the straight line of normal conditions. Such cases may be met with in acute Pulmonary affections and in asthma and allied diseases. The base line becomes very uneven, and sphygmographic character of successive pulse...
waves is different, expiration increases the pulse tension and lessens the frequency of the pulse, inspiration lowers the tension and increases the frequency. These changes may be well shown when tracings are taken from normal pulses under changed conditions, i.e. forced expiration and inspiration and when force expiratory and inspiratory efforts are made with the glottis closed.

The case quoted below is one of the most marked instances (I have met) of an uneven base line.

Pulse about 80, regular except at height of inspiration when it beats more rapidly, aggravated by deep breathing, tension low.

Sphygmographic tracing, weak upstroke. During ordinary breathing the pulsations are practically normal, dicrotic and predicrotic notches well marked.

When patient breathes deeply the character of the pulse changes, beat quickens, pressure seems to rise, the summit of the wave is rounded, the fall is more rapid and the predicrotic notch is still present, there is a distinct fall between it and the dicrotic; this condition is evident for 4 or 5 beats, then the character changes, the pulse slows down, the upstroke is less marked, the fall rapid and secondary wavelets are introduced. During this phase there seems to be a fall in blood pressure lasting for about 6 beats, then there is another rise with a
quickened beat for 6 or 7 beats, than a fall again —
and soon.

On Auscultation an extra cardiac murmur was heard in
all the areas, but loudest in the Aortic. The
second sound was accentuated. There was no intra
cardiac murmur.
THE RHYTHM OF THE PULSE.

The Rhythm during tranquil breathing is perfectly regular, but departures from the normal are fairly common and afford many interesting problems in the study of the circulating system. In some persons who enjoy perfect health the pulse is habitually irregular. Alterations of rhythm can be readily studied by means of the finger, but for more elaborate observations the use of the sphygmographic is to be recommended. These departures from the normal rhythm may be of several kinds - viz:-

(1) Difference of time.

(2) Alterations of volume.

(3) Differences in the Sphygmographic characters of the individual pulse curves.

Taking these departures in their order, we come first to irregularities of time. All degrees of time irregularities may be met with, and may be in their turn irregular, and regular in the occurrence.

A regular intermission has not the clinical significance of an irregular one. An intermission may occur once in five, ten, or twenty beats of the heart or the pulse may lose its regular rhythm entirely.

The causation of these intermissions may be obscure, but the rationale of their production is simple.
The ventricle may be acting regularly and from some cause or other not determinable, the amount of blood entering the cavity is not sufficient to lead to a contraction of the left ventricle sufficient to raise the Aortic valves and the beat of the pulse is not obtained. The next contraction of the left auricle drives sufficient blood into the ventricle to raise the intra-ventricular tension sufficiently to cause a more vigorous contraction, and the valves are raised sufficiently to cause a beat of the pulse. Such a case in a normal heart is probably of a nervous nature and may be due to one of many derangements of the nervous mechanism. It may be that the cause is to be found in some gastric condition when it will be of a reflex character. Then again in valvular disease particularly stenosis of the mitral valve the blood may regurgitate into the Auricle as well as pass into the aorta, the backward wave is counterbalanced by the next auricular contraction, and the heart continues to beat regularly until another regurgitation wave leads to an intermission.

Feeble action of the ventricle is a frequent cause of intermission, and occurring in cases of fatty degeneration of the heart is a symptom of grave prognostic valve, especially when as it usually does it occurs irregularly.

Intermission of the pulse is a frequent symptom in
persons of a nervous diathesis and occurring in such cases with no other morbid symptom is not of any great importance as a prognostic indication.

It may also occur in persons addicted to the excessive use of tobacco, tea, and other stimulants. Another alteration of the pulse rhythm of much rarer occurrence is that of Pulsus Bigeminus, a condition offering great room for theorising when we come to consider the causation. I do not think it is necessary to enter into a detailed account of the nerve mechanism of the circulatory system, in order to explain the occurrence of this interesting condition, but shall endeavour to explain it as far as possible from a mechanical point of view. I have been fortunate enough to secure one example of this interesting condition of the pulse -

The above tracing was taken from a case of cardiac disease in which there was mitral stenosis, a presystolic murmur and a suspicion of pericardial adhesions. During the first week the patient was treated by Digitalis and to this drug I am inclined to attribute the occurrence of the phenomenon.

The double beat occurred regularly for a number of beats, and then there was an intermission followed by a succession of coupled beats.
The condition generally occurs in mitral disease, so we may assume that a certain amount of regurgitation occurs at each systole of the ventricle, the regurgitation increases the pressure in the left ventricle and tells back on the Pulmonary system, raising the tension of the blood in its system of vessels, a result of this will be increased tension of the blood in the right ventricle and if the tricuspid valve be incompetent, a further regurgitation occurs into the systemic veins. If however the tricuspid valves remain competent, the effect will be to increase the tension in the cavity and lead to a more energetic contraction of the ventricular walls. This in time will lead to a hypertrophy of the walls, and we have the ventricle in a condition to act more energetically when called upon to do so. The result upon the pulmonary system will be one of increased tension or rather, say a positive tension. There is now a column of fluid extending from the tricuspid valve to the left ventricle at a greatly increased tension. The effect of the increased tension will be to lead to an asynchronisation of the right ventricle. The left ventricle contracts and raises the aortic valves sufficiently to cause the first beat of the pulse tracing, a smaller quantity regurgitates through the mitral orifice into the auricle and raising the tension of that cavity tells back upon the already overcharged pulmonary system to such an extent that
the tension of the right ventricle is raised sufficiently to stimulate the nerve mechanism in the endocardium, and the ventricle contracts to enable it to clear out its cavity, the fluid wave is set in motion and the result is that a further supply of blood is forced into the slowly contracting ventricles, a second fluid wave is propagated through the aortic valves and the second rise in the pulse tracing is obtained. This action of the right ventricle I would only call a supplementary one to the left, and I am inclined to think that the left ventricle again contract and aid the expulsion of the blood from its cavity.

Digitalis probably acted in this case by its general action upon the vessels raising the blood tension and increasing the energy of the ventricular contractions, causing both ventricles to act more energetically and altering the rhythm in this way.

When the character of the pulse can be altered by change of position of the patient, the explanation is again probably a mechanical one, the column of blood in the ventricle may have its axis of exit altered to such a degree that the passage through the aortic valves is facilitated and less blood regurgitates into the auricle and the extra pressure in the pulmonary system is
not sufficient to cause the independent action of the right ventricle. In the case quoted above, the pulse changed from the geminal variety to a regular simple beat when the patient stood up. Another variety of altered rhythm is found in the so-called Pulsus Trigeminus.

Occurring with any associated sign or symptom of disease, the condition has no significance, frequently in healthy individuals the heart may miss a beat either from a nervous or mechanical cause of a passing nature.

The chief pathological conditions associated with irregularities in time and volume are:

(a) Functional derangements of the heart such as are common in hysterical conditions, venereal excesses, gout, tobacco, tea, &c.,

(b) Mitral lesions both stenosis and regurgitation especially after compensation has failed. In these forms of mitral lesions we may meet many varieties of altered rhythm, such as Pulsus Bigoninus and trigeminus, intermissions generally irregular in time, and therefore more significant as prognostic agents. Such indicate, especially intermission of the pulse when irregular, a tendency to failure of compensation and serve as warnings in our management of these cases. As above stated digitalis may, when given in mitral cases lead to an alteration in the rhythm, and a gener-
al disturbance of the circulatory system, when such alterations occur we have a clear indication that treatment by the drug ought to be suspended for a time at least, and other efficient agents substituted. Strophanthus in cases of failing compensation may be employed, as it does not affect the pulse tension to so marked a degree as digitalis. Belladonna or other members of the same group may be indicated. As compensation becomes re-established, the alteration of the rhythm may disappear, and the heart settle down to a more normal condition.

c. Degeneration of the cardiac walls, in cases of fatty and fibroid degeneration where there is much wasting of the muscle fibres of the left ventricle, irregularity of the heart beat may occur. Occurring in fatty degeneration of the heart wall, the prognosis is grave so long as the irregularity of the rhythm continues, and careful treatment is indicated until the heart assumes a more normal condition.

d. Some affections of the central nervous system.

The next alteration that may occur is irregularity of volume, and it depends upon the quantity of blood sent into the left ventricle at each contraction of the auricle, when a smaller quantity of blood is supplied to it the alteration will be noticed in the tracing and is also perceptible to the finger. In a normal heart the quantity of blood sent into the aorta ought
to be the same at each systole and the pulse will show regularity of upstroke, and a fully sustained descending wave, with the secondary waves equal in each beat.

The last of the 3 groups of departures from the normal rhythm consists of differences between the individual beats. There may, or may not be detected by the finger, but the sphynograph clearly demonstrates them. They are to be met with in organic changes in the valves, and may offer good aids for prognosis and treatment. One beat may show all the points in a normal tracing, another may have a well marked predicrotic wave, or the dicrotic wave may be well marked, two or three beats occur close together followed by one of a more sustained character. I insert below a tracing which shows some of these characters.
The Pulse in Valvular disease of the heart.

Aortic Stenosis.

In this form of valvular disease the pulse is very characteristic, it is necessary to consider a case that shows the symptoms of stenosis purely, and not as is more frequently the case a combination of stenosis and regurgitation.

The frequency of the pulse is very little affected by the condition, and so long as the muscular walls of the heart retain their efficiency it is regular.

Narrowing of the aortic orifice must interrupt the stream of blood in its passage, and will necessarily lead to a prolongation of the systole. The resulting modification of the pulse will be a loss of suddenness of upstroke, and a prolongation of the beat. The dicrotic wave will be modified, and in marked cases it will be completely obliterated, since the condition of its production - viz: rapid action of the ventricle and great fluctuations of the blood pressure was absent. The pulse of pure aortic stenosis is one of high tension, several factors are at work in the production of this high tension, (a) the obstruction of the orifice causes a stronger action of the ventricle, in order to propel a given amount of blood, through the stenosis orifice, (b) The prolongation of the systole will also aid in maintaining the high tension.
The result of the continued resistance at the orifice will lead primarily to a dilatation of the left ventricle, followed as the condition becomes established by a compensatory hypertrophy of the ventricular walls.

The pulse of aortic stenosis has a strong resemblance to that of high tension, and this is further accentuated by the condition of the blood vessels in well marked cases of stenosis, the lumen of the vessels is smaller.

Boardbent in considering the condition says:-

"It is not 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 point by the compensatory hypertrophy, merely because the pressure takes place 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?"

An explanation of the condition is one of great difficulty involving as it does so great a discussion of the physiological and pathological conditions of the arterio-vascular systems.

An intimate relationship exists between the cardio-motor and vaso-motor system of nerve centres, they work together and regulate the blood stream. Increased intraventricular tension will lead to a corresponding
increase of blood tension; we know that in aortic stenosis the tension in the ventricle is increased, and the flow through the orifice is prolonged, there is hypertrophy of the ventricle and a stronger beat, the blood-vessels require to accommodate themselves to the altered conditions, and this they do probably in the first instance by their nerve mechanism, the lumen is diminished to accommodate the slower flow of blood and the tension is increased, the walls of the vessels probably hypertrophy in order to maintain the continued state of high tension.

Donders has shown that the action of the heart not only causes the difference of pressure necessary to establish a blood current, but also raises the mean blood pressure within the vascular system.

Character of the pulse in Aortic Stenosis.

(a) Artery is small and full between the beats.

(b) The wave has sudden rise and lifts the finger gradually, it is persistent and slowly subsides.

Tracings show a sloping upstroke, little altitude, a rounded or flat top and a gradual descent.

The pulse of Aortic regurgitation

The pulse in Aortic regurgitation is very characteristic. It has been called the "collapsing pulse", "the pulse of Corrigan" from its discoverer. A well marked case shows pulsation in all the superficial arteries, notably the Carotids, Brachials and Radials.
In this relationship it is well to exercise a little caution in the diagnosis of Aortic regurgitation from the observation of well marked pulsation in the Carotid this often is excessive in individuals whose tension is high and who are of a nervous temperament. It is also very noticeable in persons whose muscular development is not great.

The throb of the Carotid is visible up to the ear and the beating of the temporal facial and subclavian arteries at once attract attention.

Method of Production.

In this condition the valvular disease is the chief cause of the production of the typical collapsing pulse the valves are incompetent, and the degree may vary from a very slight to a marked one, the blood is forcibly driven into the aorta by the hypertrophial ventricle, and the sharp upstroke is got in the pulse, in the ordinary way the recoil wave sets in and the column of blood not meeting the due amount of resistance in the sigmoid valves, returns to the ventricle. The backward motion of the column of blood leads to an sudden emptying of the blood vessels and gives the sudden fall in the pulse tracing.

The particular cause of the incompetence of the valves may be one of two principal conditions (a) damage to the valvular cusps themselves rendering them unable to close the orifice properly (b) dilatation of the
orifice or dilatation of the aorta beyond the valves, this variety most frequently occurs in aneurism or atheroma of the Aorta, the valves may themselves be affected by the degenerative change.

Influence of the condition of the heart upon the pulse

The pulse remains regular as long as the heart keeps up its regular action, sooner or later the muscular fibre loses its power of responding to the increased strain upon it, and then the normal rhythm becomes altered. An occasional weak beat is introduced giving a weak pulse beat, or an intermission may occur owing to the impulse now reaching the radial artery.

Striking features of the pulse.

In a well marked case there is a sudden upstroke due to the hypertrophied ventricle.

The most striking feature is the sudden collapse or emptying of the artery between the beats and the sudden and short pulsation.

In prognosis it is well to carefully examine the pulse in the ordinary position, and then gently raise the arm from the side of the body, one is then better able to judge the degree of regurgitation and the condition of the ventricles.

The size of the Artery.

The vessel is generally larger than normal. This will vary with the condition of the ventricle, when it is dilated a larger amount of blood will be required to be emptied at each systole into the vessels and
they will remain with the lumen enlarged.
There is a considerable loss of time between the heart and the wrist, noticeable if one times the pulse wave in its passage from the heart to the wrist.
In mentioning the following cases I hope to be able to demonstrate the various characters of the pulse in Aortic regurgitation and to add a few remarks on the progress of these cases. The first tracing was from a man who arrived at the surgery one evening in a great state of excitement, having hurried to get there in time. His arteries were all throbbing violently, and the radial gave one the impression of a corrigan pulse. Auscultation revealed a double murmur of a doubtful character. The patient died suddenly a few months later. I diagnosed regurgitation.

Case II. A typical case. Wm. J age 35.

History of a severe attack of Rheumatic fever. This case was a most interesting one, and I shall give a few notes taken at the time of examination.
About 2 years before I saw him, patient had a very severe attack of Rheumatic fever and has never properly recovered, has never been able to resume his work. He is able to go about, but requires to exercise great care, as the slightest extra exertion brings on severe palpitation, giddiness, &c. There is well marked carotid pulsation, the superficial temporals, facial &
Lingual were all visibly pulsating. The abdominal aorta also pulsated markedly and patient complained of a dull pain in Epigastrium probably the result of the excessive pulsation.

The pulse was taken under various pressures and the following notes taken:-

22/3/95. Pressure 1 oz.

Sudden upstroke, predicrotic notch well marked, dicrotic also present. Pulse regular about 80 per minute fairly well filled.

22/3/95. Pressure 2 oz.

Predicrotic notch well marked, dicrotic also evident, towards the end of the tracing a tendency to the introduction of secondary waves.

22/3/95. Pressure 3½ oz.

Predicrotic notch more marked, note the sudden fall, dicrotic notch feebly marked.

Patient was in a fair state of health, no great inconvenience was experienced unless when patient exerted himself. Pulse exhibited on palpitation the characters of a water hammer pulse. On percussion the heart was greatly hypertrophic.
A peculiar musical murmur heard in all four cardiac areas; but much more audible in the aortic, it is transmitted up and down the sternum and along the great vessels, also along the Superficial temporal arteries. It is also audible at a distance of two or three feet from the patient. I am inclined to consider this murmur due to the vibration caused by some granulations moved about by the bloodstream. I am aware that newer theories have been introduced to explain these musical sounds, but am inclined to the view that vibrations alone could produce so audible a sound.

31/8/’95.

Note character of Aortic regurgitation. Predicrotic notch fully marked, extent of upstroke frequency of pulse regularity.

The patient was treated by Strophanthus and showed a great improvement for a time, but his digestive organs became deranged and he had to stop the drug for a time. Even in such a marked case the drug seemed to be able to tone up the cardiac muscle considerably.

Case III.
Geo. Danks.

History, never had rheumatic fever. Has been a drinker for some years. Well marked Carotid and Subclavian pulsation. On Auscultation a well marked double murmur was audible. I had great difficulty in deciding the nature of the lesion. There was marked pulsation of the right subclavian artery, and a loud double murmur was heard about $2\frac{1}{2}$ inches from the right edge of the sternum. I diagnosed aneurismal dilatation of the Subclavian (right) artery and stenosis of the aortic valves. Under treatment the condition greatly improved.

The following tracings illustrate the case:—

Tracing taken 14/3/’95.

Pulse 80 per minute.

[Graph showing pulse with predicrotic wave well marked, dicrotic faintly marked.]

Pressure 2 oz. shews feebly marked predicrotic wave dicrotic more evident than in proceeding. 27/3/’95.

Pressure $3\frac{1}{2}$ oz. Note how increase of pressure brings out the predicrotic wave. The dicrotic wave is not quite so well marked - upstroke distinctly slopes forward - pulse regular - pulse 76 per minute. 27/3/’95
Pressure 2 oz.
Predicrotic wave not so well marked, dicrotic very evident.

Pressure 3 oz. Predicrotic more marked, dicrotic well marked. 12/9/95.

Pressure 1½ oz.
Note height of upstroke, pointed apex, feeble predicrotic notch. Great fall between predicrotic & dicrotic notches, dicrotic wave well marked. Artery empty between the beats, pulse very rapid.

Pressure 3 oz.
Position of predicrotic notch on the line of descent altered, much nearer the top, dicrotic still well marked, change probably due to momentum of spring.

Pressure

Predicrotic notch rises above the primary upstroke at (b) dicrotic notch at (d).
The predicrotic notch is very evident, dicrotic also well marked.

Predicrotic notch again well marked.

In the last mentioned case the tracings are typical of Aortic regurgitation, and the influence of increased pressure is to render more marked the predicrotic notch, in none of the tracings is there an entire absence of the dicrotic wave, though it may be presumed that in two of the cases the valves were extensively affected, and the regurgitation complete. The last case derived great benefit from a course of treatment by digitalis Strychnia compensation became more established for a time.

In Aortic regurgitation a consideration of the state of the pulse is of very great service in our diagnosis and prognosis. The question of diagnosis in a well marked case cannot at any time be one of great difficulty, a glance at the superficial arteries will indicate the nature of the lesion, but when we come to consider the prognosis, a careful investigation requires to be made into the condition of the blood-vessels and the heart.
However loud the cardiac murmurs may be we must not allow them to influence our opinion until we have considered the pulse, it is the index from which we read the general condition of the circulation, and its indications are invaluable. We must look to the conditions of the heart, the degree of dilatation and hypertrophy present when we come to consider the amount of power (recovery) we can reasonably expect from the heart walls. In the absence of marked pulsation of the vessels we are able to state that the compensation is being maintained. All the characters of the pulse must be considered separately. These are in order:

1. The size of the artery.
2. The suddenness with which the pulsation strikes the finger indicates the amount of vigour possessed by the left ventricle, and the quantity of blood passing into the aorta at each pulsation.
3. The suddenness of the collapse enables us to estimate approximately the amount of regurgitation and the extent to which the valves are disordered.
4. The state of the artery between the beats, it is well to examine the artery in two positions, one the natural position, and the other when the arm is held above, the level of the heart.

If the valves are seriously damaged so that the regurgitation is free, the blood drops out of the artery im-
mediately the systole is over, and we have fully de-
veloped the collapsing character of the pulse named af-
ter Corrigan, and also called "the water hammer pulse". When the valves are not much damaged, they may be able to sustain the column of blood for a longer or shorter period and the regurgitation is slow; a small quantity of blood will then escape into the ventricle.
Auscultation of the aortic valves will also help us in our prognosis, as will also auscultation of the mitral area where a marked regurgitation murmur may be audible. The absence of modification of the second sound in the aortic is an important factor in aiding our prognosis of any particular case, as it enables us to de-
tect the extent of the damage to the valves.
In all cases of Aortic regurgitation the carotid ar-
teries should be carefully auscultated in order that the possibility of the Pulmonary's second sound being mistaken for the aortic may be avoided, for a similar reason the Pulmonary area ought to be auscultated.
THE PULSE OF MITRAL STENOSIS.

Boardbent:

"The pulse is regular, it is small, long, and in extinguishable by moderate pressure. The artery is full between the beats and the pulse is exactly what would be described as one of tension. The power behind is diminished, so why the tension is raised is a matter of speculation, presumably the amount of blood in the ventricle is diminished and the vessels adapt themselves to the existing state of matters."

In the final stage of mitral stenosis, when stasis of the venous circulation sets in, severe symptoms are developed, the pulse becomes irregular. The mode of production is somewhat as follows:— There is imperfect filling of the ventricle and a consequent diminution of the force of ventricular contraction and a smaller quantity of blood injected into the aorta. This leads to a weakness of the pulse impulse, then the beat is not sufficient to raise the aortic valves and irregularity becomes established. There is an entire loss of correspondence between the heart and the pulse. The filling of the ventricle becomes delayed, and when we consider the circumstances it is a wonder that the regularity is kept up so long. The high tension of the Pulmonary veins plus the auricular hypertrophy which occurs keeps up sufficient pressure to maintain
for a time the filling of the ventricle.

The above state of matters may continue for a varying period, but the time comes when from some cause—(Bronchitis, etc.,) the passage of blood through the lungs is obstructed, and the pressure in the right heart is increased, the patient suffers from backward pressure—the blood is no longer forced through the stenosed orifice to fill the ventricle. This being so though the ventricle may continue to act regularly and forcibly, when it contracts upon an inadequate supply of blood—no beat is received at the wrist, and the ventricular contraction not meeting a sufficient force will be short and hurried.

I shall now proceed to relate particulars of a case of Mitral stenosis with presystolic murmur, and shew a few tracings illustrative of the condition sometimes met with in this form of valvular disease.

The case was under the care of Dr. Morison at the Great Northern Central Hospital. The cardiac area was increased, apex bat in sixth space—when first seen there was a triple rhythm of a galloping type, audible in mitral area. Aortic and pulmonary sounds were both heard, second sound audible in both areas.

The patient was given quick digitalis in 5 minimum dose and on examining the case a week afterwards, the rhythm was found to have altered, and the pulse had assumed the bigeminal type. -46-
The following tracings were taken:

Shows Bigeminal pulse, a coupled beat of the heart and then a longer interval, next a primary systole with a second rise close to it. The pulse was 80 per minute. On auscultation the triple rhythm had disappeared and the double beat could be heard. The patient complained of fluttering at the heart and general discomfort. The digitalis was stopped and tincture of Belladonna substituted, and at the end of a week the pulse and heart were again examined. The heart was found to have resumed its former rhythm and the bigeminal character of the pulse at times was lost.

Pulse regular, rapid, shows above characters, tension fair. This condition was found to alternate with a recurrence of the double beat and bigeminal pulse as shewn below.

The pulse at this time varied, at one time regular, and at another shewing Bigeminal character. A week later tracings were taken again and shewed a varied character. Dr. Morison pointed out that the
rhythm varied with the position of the patient.
On examining the heart when patient stood, the triple rhythm was heard and a pulse tracing taken in that position showed a regular pulse similar to number preceding sheet. Patient was then allowed to sit for a few minutes, and the pulse examined showed a change to the bigeminal type, tracings showed similar to one below.

The condition of the patient was much improved, pulse about 80. At a later period the bigeminal character of the pulse had disappeared. The above case shows features of great interest both as regards its peculiar rhythm and the action of digitalis upon a heart with morbid valves.

The diagnosis was probable, pericardial adhesions with mitral stenosis.

I do not presume to be able to explain this case, and shall content myself with a few suggestions as to probable causes of the above conditions.

Dr. Broadbeat in his book on the pulse refers to this condition as follows:

"It does not occur in any form of valvular disease now as 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 the drug".
Further he describes the conditions as follows:

"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 vary greatly in strength, being sometimes almost as distinct as the first, and the interval between the first and the second may vary somewhat in length".

The heart's action is found to correspond. If a murmur is present usually a mitral systolic will be louder and higher with the first and may be inaudible with the second. I have sometimes heard a mitral murmur with the first, and a tricuspid with the second" (Brondbent).

For continuation of this subject see section on Rhythm of Pulse p. 24.
The Pulse of Mitral Insufficiency.

This is the irregular pulse of valvular disease. It is rare that the regurgitation occurs through the mitral orifice without giving rise to irregularity of the heart's action. Let us consider the condition of matters in a well marked case. When there is free reflux into the auricle, the proportion of the contents propelled into the Aorta will depend upon the amount of pressure maintained by the auricle and Pulmonary veins. In this condition we have circumstances which favour the influence of the lungs over the heart and its rhythm. The pressure exercised upon the auricle by the Pulmonary system of vessels will vary with inspiration and expiration, and the effect will be observed upon the pulse. It can be demonstrated in healthy individuals, the marked influence the Respiratory system exercises over the character of the pulse. The tracings may be made irregular dicrotic and the time may be altered. If this be so in health with a presumably well balanced heart, how much more marked must be the effects upon an ill-balanced faltering heart, hovering on the brink of a complete breakdown, a little extra exertion, a run to catch a train, an attack of Bronchitis, may be sufficient to cause the heart to lose its rhythm and become markedly irregular, in other cases the patient may
suddenly expire.

In inspiration there will be negative pressure within the auricle and diminished resistance to the reflux of blood. In expiration positive pressure and increased resistance to reflux, and the systole will be sharp and short, or prolonged accordingly. This is another obvious cause of irregularity, the result being that no two beats of the heart are alike.

The pulse is markedly irregular, compressible, short & unsustained, the tension is low.

I illustrate this form of valvular disease by quoting from a case I had under treatment for some time.

Geo. Smith, age about 60, no history of rheumatism, heart's action very irregular, dilated, bruit in mitral area, pulse irregular, rapid, compressible.

Tracing shews:-

marked irregularity of the pulse.

Under the influence of digitalis and iron, the action of the heart gradually improved, and the following tracing taken after an interval of about 2 months shews the change.

Pressure 2 oz. Pulse quite regular, tension average artery fairly full between the beats. Comparing the above tracing with tracings taken 2 months previously
one is struck with the change for the better, it demonstrates how completely a deranged heart may recover its lost compensation.
PULSé TENSION AS A GUIDE IN DIAGNOSIS & PROGNOSIS.

The tension of the Pulse in any particular patient offers us one of the most interesting considerations in clinical medicine. In using the term "pulse tension" one is only making use of a local condition, the tension really being that of the blood. By a careful study of the tension in particular individuals, I think we may be able to foretell with a certain degree of accuracy the nature of the diseases to which our patient will be liable, and also the nature of the resistance he is likely to offer to a particular disease when he becomes its victim.

In considering pulse tension it is usual to divide into two great groups all degrees of tension, High & low. It must be clearly borne in mind that there are degrees intermediate, and that the limits of healthy & unhealthy high & low tension are naturally arbitrary. One person may have a high tension pulse and be in perfect health, which in another person might indicate a serious disease. Let us take first the low tension pulse and endeavour to trace its significance in Diagnosis, prognosis and treatment.

Low Arterial Tension

In order to maintain a fixed ratio, the factors of tension must be equal, the heart is the active agent in the production and maintenance of tension aided by the elastic property of the blood vessels.
At the other end we have the outlet in the small arteries and capillaries. Normally as much blood escapes from the arteries & capillaries at each pulsation of the ventricle, but the time taken is three times as great, the systole lasting roughly one third of the complete cardiac resolution. It is theoretically conceivable that the resistance might be so diminished that as much blood could escape from the peripheral end of the vascular system as was pumped in by the ventricle and in the same time, but such a condition would not be compatible with life.

As above stated the heart gets in say three systoles while the first quantity of blood pumped into the arteries is escaping from the capillaries into the veins the reserve beats are stored up in the intermediate vessels and raise the tension.

In proportion, as the flow through the capillaries is free, the number of beats stored up in the arteries will be diminished, the means of continual blood pressure within them, and the degree of tension of their coats will be lowered, and the smaller will be the amount of nutritive material passing through the capillary wall for the use of the structures.

Low blood tension then implies a diminished arterial reserve and a lessened supply of nutriment to the tissues.

The importance of the above statement must be borne
in mind when we are considering any case in which there
is low blood tension. The bearing of such con-
ditions upon our diagnosis and particularly prognosis
is highly important.

The artery is readily compressible and can be
obliterated by moderate pressure. It seems to start
into existence with each beat of the heart and to dis-
appear as the wave passes. The pulse is sudden in
its ictus, brief in its duration, and its subsidence
is rapid and is broken by a dicrotic rebound.
Sphygmographic tracing shews a perpendicular upstroke
a sharp top, a steep fall, and a deep notch and di-
crotic rise.

There are many varieties of low tension pulse.
When the heart is acting forcibly the pulse is large,
sudden and vehement, the size of the artery and the
shortness of the wave intensifying the impression of
force conveyed to the finger. The pulsation of the
heart may be so frequent that the outflow from the
capillaries is not quick enough to allow of the com-
plete collapse of the vessel, and one might be misled
into thinking the pulse one of moderate or even high
tension. The pulse can however be flattened without
difficulty, tracings would shew distinct dicrotism,
and that would of course promptly clear up the case.

In such cases the line of treatment would be to ende-
vour to allay the forcible action of the heart, and at
the same time give drugs that would exercise an influence over the peripheral resistance and raise the blood pressure. The heart may act feebly and send out a diminished amount of blood at each systole, the diminished amount of blood in the arteries will allow the vessel walls to contract down upon the column of blood and the pulse to the finger will feel small and very easily compressed, the ictus loses its sharpness. Such a pulse is indicative of a weak action of heart, and probably a change in the muscular fibres.

For normal low tension pulse (see part on dicrotism)

Low tension with forcible action of heart from a man who had served in the Soudan campaign and had roughed it pretty severely, there were no murmurs at any of the orifices. His heart was very irritable

Low tension pulse.

Low tension pulse, with frequent action of the heart, leading to irregularity (case alcoholic)

My attention has been attracted by the frequency of the occurrence of low tension pulse in cases of Rheumatic fever years after an attack. I show here a
series of tracings taken from several cases of Rheumatic patients - tracing No. 1 from a case convalescent from R. fever. Shews, regularity, faint predicrotic notch, dicrotic better marked - a pulse of low tension.

Tracing No. 2 low tension - pulse regular, a marked systole bruit present.

Tracing No. 3 Had Rheumatic fever twice - last attack 4 years ago - subject to bronchitis, pulse regular, tension low. Tracing shews dicrotism.

Causes of low tension.

Obesity is usually associated with low tension, the action of the heart is weak, and the arteries are small. Warmth especially combined with moisture relaxes the arterioles and capillaries, and lowers arterial tension. Food particularly when taken warm, lowers tension. Sustained exertion, fatigue and exhaustion bodily and mental will all lower arterial tension. A good meal will lower tension because of the supply of blood required for the active processes of digestion, the lowering of tension will after a long time be compensated for by the absorption of material fluid & nutriment from the intestinal canal.
Anxiety and worry and depressing emotions will also lower tension, especially if they be prolonged. Diminished supply of nitrogenous food will lower the blood tension, and, as will be shewn later, excessive supply will lead to increased arterial tension.

Certain states of the nervous system will lower tension; but it is rather a difficult matter to settle whether it is the nervous condition that causes the low tension, or, the low arterial tension which leads to the nervous condition, we know that an inadequate supply of blood and impaired nutrition soon cause various nervous symptoms. The great cause of low tension of a temporary nature is Pyrexia, the tension in all forms of inflammatory mischief except that of the kidney when a local inflammation leads to high tension, and when the condition of high tension is purely due to the local effect altering the eliminating power of the kidneys.

Low tension resulting from Pyrexia is frequently met with in many forms of specific fever, pneumonia &c., and is readily recognised. In extreme cases the tension is so low that marked diicrotism and hyper-dicrotism are observed.

I have frequently observed that in the early stage of inflammatory mischief when the heart was acting vigorously and the peripheral resistance was not unduly lowered, that the Pulse tension was increased.
Soon however, this condition gives place to one of low tension as the pyrexia continues.

Dr. Mahomed has demonstrated by sphygmographic tracings that high tension does exist in these cases.

It generally occurs in the Sthenic type of pyrexia. As Pyrexia continues, the heart begins to lessen in its power and the tension becomes lowered. The ventricle is imperfectly emptied and less blood is forced into the aorta, at the same time the peripheral resistance is lowered and then we have the two main factors necessary for the production of a low tension pulse in full action.

The pulse remains quick, but it is now feeble, soft and often small. The tracings show dicrotism or hyper-dicrotism. It is at this stage that a tendency occurs to Hypostatic congestions in various parts of the body.

I have frequently noticed in cases of rheumatism and patients who have had attacks of Rheumatic fever that the pulse is invariably one of low tension, tracings show more or less marked degrees of dicrotism. The point that is not clear is whether such patients have been subject to a low tension all their lives, or if the lowered tension is merely the result of the altered state of the circulation brought on by the attack of fever. In some cases the tension is altogether independent of any valvular lesion. Individuals of the above type are subject to catarrhal affec-
tions, their skin is cold and clammy and on exertion they freely perspire. They evidently possess a lowered peripheral resistance which may account for the low tension. I think persons who possess low tension are more liable to various cartarrhal conditions and that the Rheumatic diathesis may be placed among the causes of low tension.

Effects of Low Arterial Tension

The morbid appearances resulting from low arterial tension are not at all marked. The diminished peripheral resistance will lead to a diminished action of the heart, and consequent imperfect nutrition of the parts supplied. There is a tendency to degenerative changes in the heart blood vessels.

Symptoms of low arterial tension.

The tissues will be imperfectly supplied with nourishment and the heart shares in this condition. Its action will become languid and the systole feeble. Consequently changes may be detected in the heart sounds - the first sound becomes feeble and ill marked. Many of the symptoms are equally common with those of high tension pulse. The question becomes then not one of diagnosis between high and low tension, but what are the conditions present which give rise to such symptoms, and how ought they to be treated? The state of the tension is of great importance in our diagnosis, prognosis & treatment.
Abnormally low tension may be associated with a great variety of functional derangements as well as of symptoms, dyspepsia, constipation and sleeplessness, head-ache, &c., pains in various regions of the body, neuralgic pains in head, back and legs are frequently associated with a condition of low arterial tension. Many cases occur especially in women where particular and to the patient real pains are experienced, careful clinical examination fails to reveal any morbid conditions of the organs, but the heart's action may be feeble, languid and the tension low, the peripheral circulation is also altered, the surface of the body may be warm and flushed. Such conditions yield to no particular line of treatment that would readily act upon such symptoms if the particular part affected were treated on recognised lines. But if such a patient be treated on lines indicated by the condition of pulse a speedy return to a normal condition will be got. Let your patient have a thorough change, feed judiciously and use the tonics that are known to act directly on the heart and vessels, digitalis, mineral acids, &c., and the symptoms will clear up. Symptoms of all kinds may be present in low tension (blood), and it must not be lost sight of that in many cases of disease, low tension is present and cannot be stated as the cause of symptoms but even in such cases relief may be obtained by combining
with the ordinary treatment, that of low arterial tension.

Treatment.

In speaking of treatment we must bear in mind that it is not the low arterial tension we are called upon to treat, but the causes that give rise to such a condition. In considering causes of imperfect resistance in the arterio capillary system of vessels, we require to distinguish between those causes by the state of the blood, and such cases as can be traced to distinctly nervous causes. Such distinctions are not easily made, for it is an easy matter for shock, nervous depression, &c., to lead to a condition of low arterial tension, and on the other hand for degenerative changes in the blood and tissues to give rise to nervous symptoms amounting to melancholia, and cases are frequently seen where patients are greatly depressed, sleepless, and on the verge of suicidal melancholia, which readily respond to cardiac and vascular stimulants and tonics.

The main lines for treatment are to tone up the heart and vessels and improve the digestive functions.

Digitalis, iron, strychnine, Mineral acids, particularly Nitric and Hydrochloric acids, quinine, and other tonics. The diet must be regulated, simple and digestible articles of diet prescribed, meat in fair proportion, alcohol should be given with caution,
particularly when the habit is one of the exciting
causes of the condition. In cases with an alcoholic
history I find it best to stop alcohol in any form &
to give nux vomica with a mineral acid and a bitter
infusion. Change of scene is often of the greatest
benefit; sea voyages and travelling are of great value.
If the patient is not in circumstances suitable for
prolonged travelling, then I think in these days of
what may be termed hyper-athletism there should be no
difficulty in getting him to exercise at one of the
many games so popular at the present time. Golf I
consider an excellent therapeutic agent for such cases:
The pulse of high tension

This deserves careful study, and is an important
factor in diagnosis and prognosis. It points out
tendencies on the part of individuals, which enable
us to warn patients of the possibility of their oc-
currence.

Characteristics of high tension pulse.

The pulse feels hard to the finger, and we are able
to follow the course of the artery in the arm, it does
not empty rapidly when the systole of the heart is
over, but remains full between the beats, in old per-
sons the hard teusl artery may simulate atheroma of
the arterial system, but can readily be distinguished
from it by pressing from the artery from above and
checking the flow of blood to the part under examina-
tion, it is then found to empty itself below, and the hardness leaves the vessel. The amount of tension may be approximately estimated by gradually increasing the pressure of the finger upon the artery. The artery may be small or large, according, as it is dilated to its full extent or not. The ictus of the wave is gradual and does not rise to any great extent. The tracing will have an upstroke with an inclination forwards and it may be somewhat rounded at the summit. The wave has an unexpected amount of force and if the fingers be firmly pressed it appears to become stronger. The causes of increased arterial tension are numerous and I shall refer to the most frequent.

a. Increase in the volume of blood, it can readily be understood how an increased amount of blood will lead to an increase in the tension of the pulse.

b. Frequent and powerful action of the heart.

c. Renal disease is attended with high arterial tension, and the recognition of the state of the pulse is one of our most important signs in disease of the kidneys, a high tension pulse occurs from many other causes than renal disease, so we must not be too hasty in diagnosing mischief in the kidneys when we get high tension of the pulse.

d. Gout is another of the common cause of high tension.

e. Diabetis occurring after middle age is frequent
ly accompanied by high arterial tension.

f Lead poisoning is another cause of high tension and I have seen several cases where the symptoms could be traced to this agent, albumen was present in small quantities so probably the renal mischief accounted for the increase in tension.

g Pregnancy is a common cause of increased arterial tension, a slight degree may be looked upon as physiological in this condition but it may overstep the limit and assume a pathological character.

h Anaemia is frequently accompanied by high tension though how it occurs in cases where the blood is much below its normal standard is difficult to understand. One would expect to find low tension from the watery condition of the blood allowing a more rapid passage through the vessels at times. The common association of chronic constipation with anaemia may lead to increase of tension from the quantity of effete material in the blood, such matters may have a direct influence on the function of the kidneys and so increase the resistance to the circulation of the blood.

i Conditions of the lungs such as emphysema or chronic bronchitis are frequently associated with high tension.

j Age, as the vessels grow older they lose their elasticity and degenerative changes occur which must have an influence in the production of high tension.
Food, a high proportion of animal food leads to an increase of the tension, and a habitual over-supply of such food must tell on the kidneys and other excretory organs and lead to other morbid changes in the organs whose duty it is to eliminate the excess of nitrogenous matter in the blood. The amount of animal food in a person's diet ought to be regulated carefully in order to avoid this far too frequent cause of high tension and later onset of renal and hepatic disorders.

Abuse of alcoholic drinks, any form of alcohol taken in excess must invariably lead to changes in the organs of digestion and elimination of waste materials. Alcohol arrests the normal processes of elimination and leads to an accumulation of impurities in the system. The effect on the circulatory system is to primarily increase the blood tension.

Sedentary Habits.

Constipation. Has been already referred to under Anaemia.

There are other causes of less frequent occurrence.

Symptoms of high arterial tension.

Among the most frequent are headache, sleeplessness, breathlessness, depression of spirits, loss of energy, memory and nerve power, neuralgic pains in various parts of the body. Headache is a frequent symptom and varies in position, it may be frontal, occipital or vertical, and varies in its intensity as
the tension varies. In cases of persistent head-ache attributable to no other cause it is a good method of treatment to keep the bowels freely acting and attend to dietetic errors. Migraine is constantly attended by high arterial tension and is greatly benefited by treatment aimed at the reduction of the blood tension, the fact of high tension being present is an explanation of the use of the nitrite group of remedies in the management of migraine. Breathlessness or exertion is another symptom of high tension and may be of a severe type, and may suggest cardiac lesions.

High tension may give symptoms which indicate a grave lesion of the organs of excretion. I have seen several cases in which there was a considerable quantity of albumen in the urine. The following case is an interesting example of this class:

Case seen at the Great Northern Hospital under the case of Dr. Morison. A man about 50 came complaining of shortness of breath on exertion and general depression loss of appetite, &c.,

Heart greatly dilated right border extending to about 2½ inches beyond right sternal margin, apex in 6th space.

Auscultation no bruit could be detected, first sound obscure, second accentuated, pulse tension high, artery felt hard and tense to the fingers.

Urine scanty, pale in colour, large deposit of albumen,
on heating, and with cold nitric acid. The patient was treated by saline calhartics, and a mixture containing tincture of Strophanthus and Spirits of Nitrous ether prescribed. The patient gradually improved and in a month's time the condition was observed to be as follows:—Heart diameter greatly diminished, sounds, first much more distinct, second still accentuated, though not so much so, as at first examination, pulse tension greatly lowered, artery empties between the beats, readily compressible. Urine more abundant, merest trace of albumen present, none on heating, slight haze with cold nitric and picric acids. Patient feels very much better, and looks brighter.

The above case illustrates the great importance of the early recognition of high tension as a factor in the production of symptoms closely resembling those of chronic renal mischief, an early diagnosis and prompt treatment enables us in many cases to stave off the onset of the graver form of disease.

I have seen other cases attended by symptoms of impending serious nerve changes relieved by a line of treatment having for its aim the reduction of arterial tension, I may be pardoned for quoting another case which occurred during my dispensary career previous to graduation and which gave me some trouble in understanding the rationale of the case. A man complaining of great depression of spirits, homicidal inclina-
tions and marked irritability was promptly relieved by the free use of calh hartics and the internal administration of Strychline with nitric acid. This I have no doubt was a case of high arterial tension with mental instability and the rapid disappearance of the symptoms was due to the great lowering of blood tension that resulted from the free use of calhartic remedies. I have seen many cases since of a similar nature. A proper appreciation of unduly high arterial tension enables us to point out tendencies which later, result in fatal or serious disease, and its recognition enables us to at all events prolong lives that would have terminated suddenly, or at a much earlier date. As a person advances in years his tissues change, and none more so than the tissues of the arterial system, as the tissues lose their powers of resistance they tend to rupture if the high tension continues; thus there may be rupture of blood-vessels in the brain, the retina and other parts of the body, changes in the Aorta and heart, calcareous degeneration in the walls of the aorta may lead to the formation of an aneurism. Valvular disease of the heart is another of the results of high arterial tension, murmurs may be detected in these cases. Dilatation and hypertrophy of the heart are also consequences of high arterial tension. All these changes occurring from high arterial tension are to a certain extent under our control if we are
fortunate to get a case early enough to direct our attention to the treatment of the high tension, and even when one or more of them are pronounced we can, by treating the tension, delay the progress of the disease. Having diagnosed existing high arterial tension, and having in our minds the consequences that will follow in its wake if allowed to run its course, we can by keeping the tension within normal bounds, guard against the onset of symptoms indicative of the onset of one of the graver lesions.

The following tracings are examples of high tension pulses of varying degrees:-

*The tension is not very high in this tracing, but it varied much at different times, patient was subject to fits of depression and was greatly benefited by our-gatives and nervine tonics.*

Pressure 4½ oz.

Not irregularity of the Pulse, at one point an indicator of a double beat, patient was also subject to depression of spirits, a case of long standing.

Pressure 2 oz.
Pressure 2½ oz.

Pressure 3 oz.

Pressure 3 oz.

The above 4 tracings are taken from a man the subject of profound anemia, skin very pale and a yellowish tint, no albumen in urine. I am inclined to think this was a case of early prenicious anemia, but unfortunately did not see the patient again.

Pressure 3 oz.

Taken from a patient the subject of morphia habit.

Two tracings show high tension due to gout, probably changes in the vessel walls had a good deal to do with the apparent high tension.
Treatment of high arterial tension.
The chief object in treating cases of this description will be to keep the blood free from impurities.
Exercise and fresh air are of great importance, horse exercise, golf, &c., may be recommended.
Diet should be regulated, nitrogenous food should be limited as much as possible, the minimum of such food should be allowed. Alcoholic drinks should be taken sparingly, or not at all. Fluids should be taken in moderate quantities.
The free action of the bowels should be encouraged.

Mercury in one of its many preparations is an excellent drug to employ in these cases. Calomel at intervals is useful. In cases of migraine with high tension Nitrite of Amyl sometimes exercises a beneficial effect, Nitrite of Sodium and Nitroglycerine are of great service. Dilute nitric acid with a bitter tonic often acts well.
In cases of high tension associated with dilatation the use of digitalis is of great service, particularly as it has a diuretic action and thus aids in the reduc-
tion of the tension. Strophanthus may also be of great service particularly in cases where there is albumenuria, it acts by increasing the strength of the cardiac systole and enables the heart to recover its lost compensation.

Purgative salts may be used when we want to reduce the quantity of fluid in the blood.

Other conditions may require treatment. In cases of gout, remedies must be used to relieve symptoms dependent upon the gouty condition.

The heart will require tonics in cases where there is dilatation and when compensation threatens to fail.
Broadbeat on the Pulse –
Byron Bramwell: Diseases of the heart and Thoracic Aorta.
Magge & Pye Smith: Medical.
Guy's Hospital reports.
Lendoes & Stirling Physiology.
Fosters Handbook of Physiology.
Taylor's Medicine.
Kirkes Physiology.
Gibson & Russel's Physical Diagnosis.