Student's guide to the examination of the pulse, and use of the sphygmograph / by Byrom Bramwell.

STUDENT'S GUIDE

TO THE

EXAMINATION OF THE PULSE,

AND

USE OF THE SPHYGMOGRAPH.

BY

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THE EXAMINATION OF THE PULSE,[1]

INCLUDING

A Description of the Sphygmograph.

At each contraction of the left ventricle, some five or six ounces of blood are suddenly propelled into the aorta, and a blood-wave is generated, which is rapidly propagated through the arterial system. The temporary distension of the arterial walls which is caused by this blood-wave, can be felt by the finger or measured by the sphygmograph, and is termed the pulse.

The exact character of the arterial expansion, *i.e.* of the pulse, varies in different cases, and depends partly upon the mode of contraction of the left ventricle, and the amount of blood which it propels into the aorta, and partly upon the condition of the arterial system. It is evident therefore that in the examination of the pulse we have an important means of investigating *the condition of the heart* and of *the arterial system;* and since the condition of the arterial system is to a large extent regulated by the vaso-motor nerve apparatus, we are enabled, by the observation of the pulse, to obtain in many cases valuable information as to the *condition of the nerve tone* (*i.e.*, the general tone of the system); the frequency and strength of the pulse being chiefly valuable in this respect.

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[1] Being a lecture delivered in the Extra-Academical School of Medicine, Edinburgh.

The radial is the artery which is usually examined, and in speaking of *the pulse* the radial pulse is meant; but, in cases of cardiac and arterial disease, the condition of other vessels, the carotid, temporal, brachial, etc., should be noted.

MODE OF OBSERVING THE PULSE.

We observe the characters of the radial pulse by means of—

(1) the finger (*palpation*);
(2) the eye (*inspection*); and
(3) the sphygmograph.

PALPATION, OR THE EXAMINATION OF THE PULSE BY THE FINGER.

The correct observation of the exact characters of the pulse by the finger is a matter of extreme difficulty, and is only acquired by long practice. Since, however, it is *the* method which is universally available, the student should spare no pains to make himself master of it. Two or three fingers should be applied over the artery where it becomes superficial at the lower end of the radius, and the condition of the pulse noted as regards—

(*a*) its frequency;
(*b*) its rhythm;
(*c*) its volume;
(*d*) its compressibility or tension;
(*e*) the special characters of each pulse wave (celerity, dicrotism, etc.); the condition of the vessel (in respect to its fulness) during the diastole of the ventricle, *i.e.*, between the beats; and
(*f*) the condition of the arterial coats.
(*g*) In cases of suspected aneurism or intra-thoracic tumour a comparison of the two radial pulses should be made.

INSPECTION OF THE PULSE.

In well-nourished individuals the radial pulse is hardly, if at all, visible when the circulation is tranquil; but in emaciated subjects, and during cardiac excitement, its pulsation can often be distinctly seen. Pulsation is very visible in certain cases of disease, notably in conditions of high tension; in atheroma, where the artery stands out as a rigid, tortuous cord; and in aortic regurgitation, where the pulsation is visible, jerking, and collapsing, and the artery tortuous (the locomotive pulse).

In conditions of vaso-motor relaxation with excited action of the heart, the pulse in the peripheral vessels (the radial for example) may present the visible, jerking, collapsing character of aortic regurgitation; but the marked (visible, jerking, collapsing) pulsation in the large vessels, *e.g.*, the carotids, which is so characteristic of aortic regurgitation, is not observed.

THE EXAMINATION OF THE PULSE BY MEANS OF THE SPHYGMOGRAPH.

The exact characters of the pulse, *i.e.*, of its individual waves, and of their relationship to one another, are graphically demonstrated by means of the sphygmograph. The instrument is of great use as an indicator of the manner in which the circulation is being carried on, and of the general condition of the vascular system; it sometimes gives important diagnostic evidence, as in the earlier stages of chronic Bright's disease (especially the cirrhotic kidney), and in some aneurisms; but it is comparatively useless and superfluous as a means of diagnosing individual cardiac affections. But although its (direct) diagnostic value is limited, it sometimes enables us to form a more correct opinion than we could otherwise of the severity of a lesion or case; in pneumonia, and typhoid fever for example, it may afford most useful prognostic information, and the same may be said of many cardiac affections.

FORMS OF SPHYGMOGRAPH.

Mahomed's modification of Marey's[1] Sphygmograph, and Dudgeon's[2] Sphygmograph, are the best forms. (Dr W. J. Fleming of Glasgow introduced a 'simple form of transmission sphygmograph' some years ago. I have not had an opportunity of using this instrument, which is described and figured in the *Journal of Anatomy and Physiology*, vol. xii. p. 144).

For home practice or hospital work I prefer Mahomed's modification of Marey's instrument, as I think it permits of more accurate adjustment of the pressure; but for general practice Dudgeon's instrument is undoubtedly most convenient; it is extremely portable, easily applied in any position of the patient, and is only one-third the cost of the larger instrument. With it excellent tracings may be obtained, and its inventor claims that it gives a more accurate and natural representation of the up-stroke than can be obtained by Marey's instrument.[3]

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[1] This instrument is made by Krohne & Sesemann, 8 Duke Street, Manchester Square, London.

[2] Dr Dudgeon's instrument is made by Mr J. Ganter, and may be obtained through any instrument-maker.

[3] Dr Dudgeon claims the following advantages for his instrument :—

1. It magnifies the movements of the artery in a uniform degree, viz. 50 times.

2. The pressure of the spring can be regulated from 1 to 5 ounces.

3. It requires no wrist-rest, and may be used with equal facility whether the patient is standing, sitting, or lying.

4. With it a tracing of the pulse can be made almost as quickly as the pulse can be felt with the finger.

5. Its sensitiveness is so great that it records the slightest deviation in form or character of every beat.

6. Its construction is so simple, that if accidentally broken any watchmaker can repair it.

7. It is so small (2½ by 2 inches), and it is so light (4 oz.), that it can easily be carried in the pocket.

8. It is only one-third of the price of the imperfect and cumbrous instruments hitherto offered to the profession.

DESCRIPTION OF THE SPHYGMOGRAPH.

Mahomed's modification of Marey's Sphygmograph consists of—

1. A steel spring, A (see figs. 1, 2, and 3), which rests on the artery, and which moves up and down with each movement of the vessel.

One end of the spring, A′ (see figs. 2 and 3), is so attached by a hinge to the framework of the instrument that vertical (up and down) movement is alone permitted. To the under surface of the free end of the spring an ivory pad, A″, is fixed. The ivory pad rests on the artery.

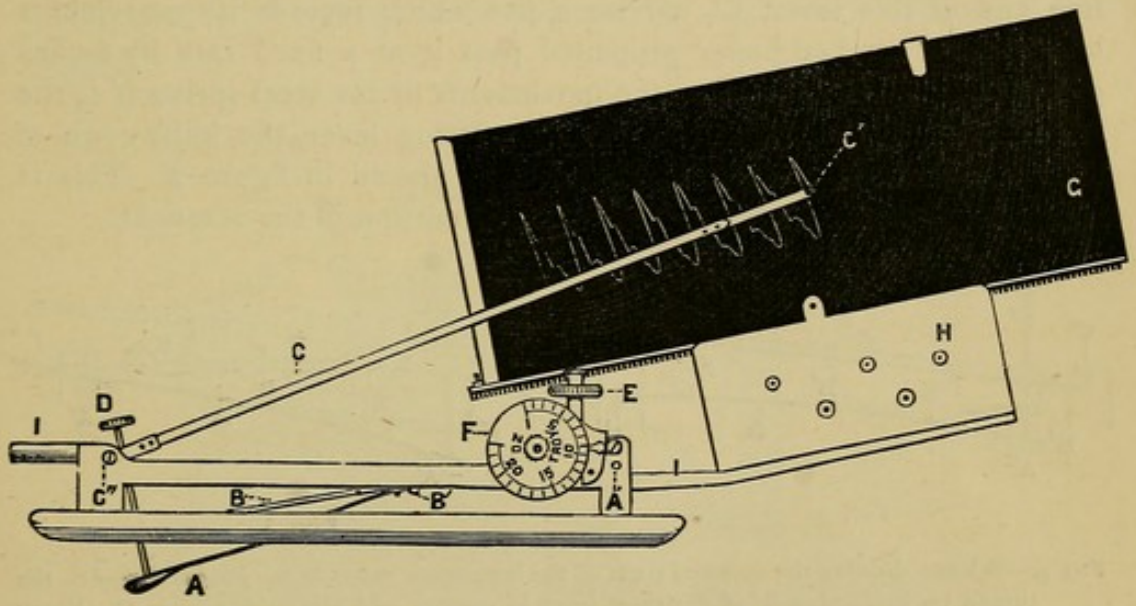

FIG. 1.—*Mahomed's Modification of Marey's Sphygmograph.*

A, points to the steel spring; A′, to its point of attachment to the framework; B, the first lever; B′, its point of attachment to the steel spring A; C, the writing lever; C′, its free end, which carries a pen; C″, its point of attachment to the framework; D, the screw for bringing the turned up free end of lever B in contact with lever C; E, the screw for increasing the pressure; F, the dial on which the amount of pressure is indicated; G, the slide carrying the strip of smoked paper; H, the clockwork, which is wound up by a screw on the opposite side to that shown in the figure; I, I, parts of the instrument over which the straps, which fix it to the arm, are passed.

2. An arrangement of levers, by means of which the movements of the steel spring (*i.e.*, of the artery) are magnified and recorded on a piece of smoked paper.

There are two levers. The lower one, B, is so hinged by its proximal end, B′, to the middle of the steel spring, that up and down movement is alone permitted. The free end of this lever (B″, figs. 2 and 3) is

turned up at a right angle, and ends in a rounded or knife-shaped edge. Through the free extremity of this lever, B (just before it terminates in the turned up end, B″), a screw D is passed. The point of the screw is always (by the force of gravity) in contact with the steel spring, *i.e.*, the artery. And since the screw D and the lever B are practically the same, it follows that every movement of the artery is of necessity communicated to the turned up extremity B″, of the lever B. The object of the screw D is to raise or lower the turned up edge of the lever B, so that it may, whatever the position of the steel spring, be in proper contact with the writing lever C, in other words, in order that it may always communicate the movements of the steel spring, *i.e.*, of the artery, to the writing lever C.

The second lever, C, is so fixed at its distal end, C″, to the framework of the instrument, that vertical movement is alone possible. The free end of this lever, C′, carries a pen which records its movements on a strip of smoked paper propelled past it at a fixed rate by means of clockwork. In order that the movements of the steel spring (*i e.*, the artery) may be communicated to the writing lever, the knife-edge of lever B must be in contact with lever C, as shown in figure 2. This is effected, as has been already stated, by alteration of the screw D.

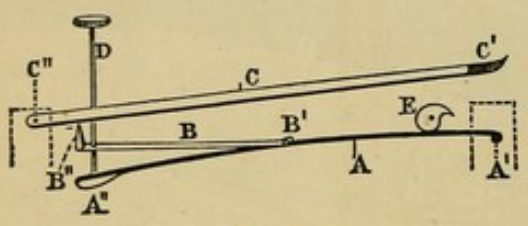

FIG. 2.

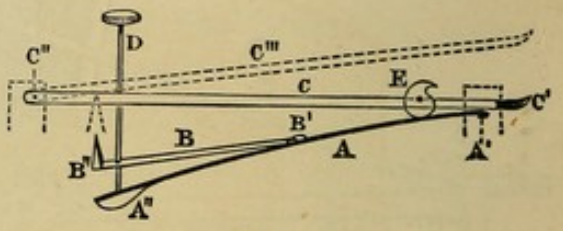

FIG. 3.

FIG. 2.—Scheme showing the essential parts of the instrument *when in working order—i.e.*, the turned up knife-edge B″ of the short lever in contact with the writing lever C. Every movement of the steel spring, at A″, *i.e.*, of the artery, will, when the knife-edge, B″, is in this position, be communicated to the writing lever. The letters have the same significance as in Fig. 1.

N.B.—(The framework of the instrument has been removed).

FIG. 3.—Scheme showing the essential parts of the instrument *after increase of the pressure.* The knife-edged B″ is no longer in contact with the writing lever, and the movements of the steel spring A″, *i e.*, of the artery, are no longer communicated to it. In order to put the instrument into working order the knife-edge B″ must be raised to the position indicated by the dotted lines. This is effected by means of the screw D.

3. An arrangement by which the amount of pressure exerted by the steel spring on the artery can be regulated and measured. This is a most important part of the instrument, and is the modification made by Mahomed. It consists of an eccentric, E, by depression of which, as shown in fig. 3, a definite degree of pressure can be made upon

the steel spring. The amount of pressure exerted is shown on a dial (F, in fig. 1) in ounces troy. The eccentric is depressed by turning the screw E (see figs. 1, 2, and 3).

4. A clockwork, H, which propels at a fixed rate a slide G, to which a strip of smoked paper is attached.

5. A framework to which the various parts of the instrument are fixed, and by means of which the instrument is fastened to the arm by straps (K, K, Fig. 6).

Dudgeon's Pocket Sphygmograph (see fig. 4) consists of:—

1. A steel spring, A (see fig. 5), which rests upon the artery, and moves up and down with each movement of the vessel.

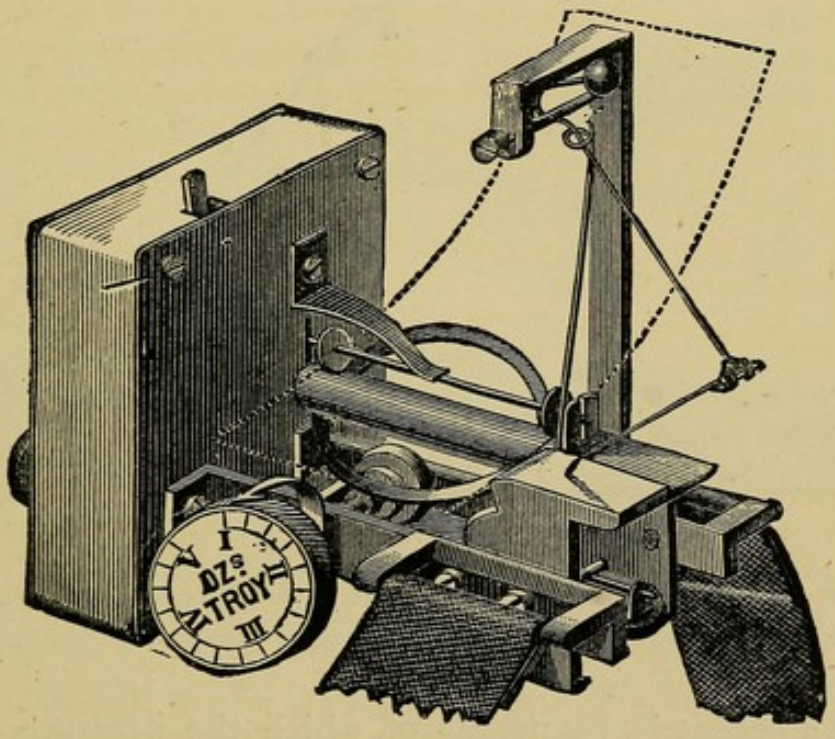

FIG. 4.—Dudgeon's Sphygmograph.

One end of the spring (a) is firmly attached to the framework of the instrument, the other (C) is turned up at a right angle. To the under extremity of the steel spring a button, B, which rests on the artery, is fixed; and to the turned up extremity C a short rod (D) is firmly attached.

2. An arrangement of levers by means of which the movements of the steel spring, *i.e.*, of the artery, are

magnified and recorded on a strip of smoked paper propelled by clockwork.

At right angles to D, and connected with it by the axle E, rises the upright stem F. Every upward movement of the steel spring causes the upright F to move forwards. At the top of F is a loop in which a rod K lies. This rod is connected at the axle H with a bent rod having a counterpoise I. When the upright F makes a forward movement, the oblique rod K also swings forwards by the weight of its counterpoise.

To the lower end of K the needle L is attached by the hinge M, and its point describes on the smoked paper, which is propelled by the clockwork machinery at a uniform velocity, a graphic representation of the movements communicated to it.

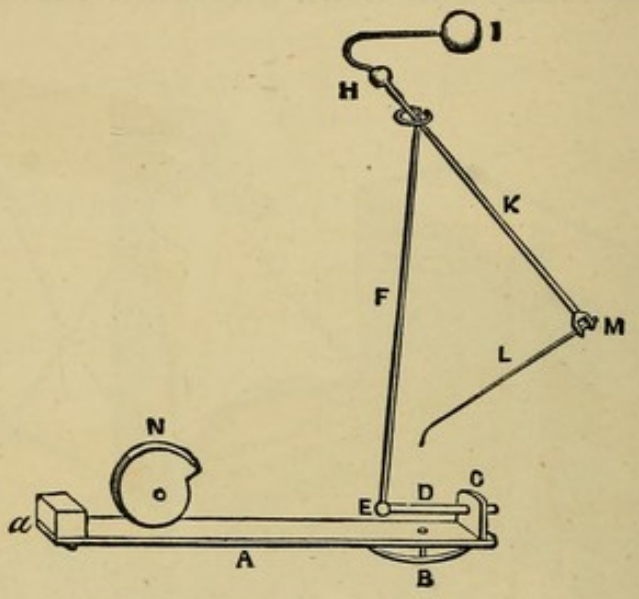

FIG. 5.—Scheme showing the different parts of Dudgeon's Sphygmograph. The description of the figure is given in the text.

3. An eccentric, by means of which the pressure can be increased from one to five ounces.

4. A clockwork, by means of which a strip of smoked paper is propelled at a fixed rate under the writing lever.

5. A framework, by means of which the instrument can be attached to the arm.[1]

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[1] For further particulars respecting Dr Dudgeon's instrument, see his book, 'The Sphygmograph.'

DIRECTIONS FOR TAKING A SPHYGMOGRAPHIC TRACING WITH MAREY'S INSTRUMENT.[1]

1. *Place the Patient in proper position.*—He should be seated by the side of a low table, his arm resting on the pad (a double inclined plane), as represented in fig. 6, the fingers semiflexed into the palm.

If the fingers are quite extended the artery is too much stretched, and jerking movements of the tendons, which interfere with the tracing, are apt to occur. If the fingers are quite flexed, the rigidity of the tendons prevents the perfect application of the instrument.

The position should be as easy as possible, for it is essential that the arm be kept at perfect rest. The shirt sleeve should be turned up; and it must be loose, lest it interfere with the circulation through the arm.

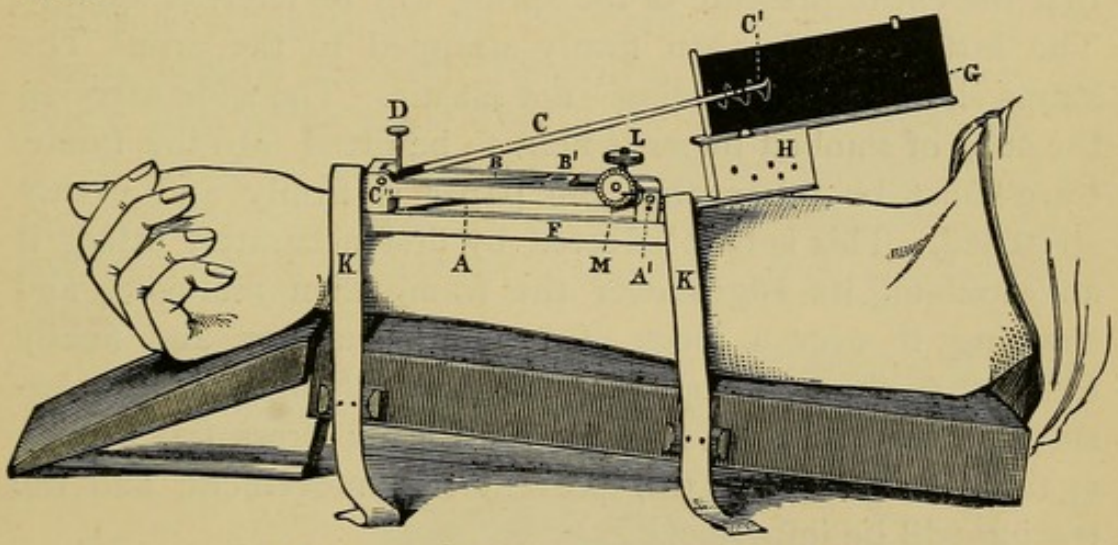

FIG. 6.—Marey's Sphygmograph applied to the wrist.

2. *Mark the exact position of the artery with ink or pencil.*—The ink line should be prolonged to the ball of the thumb, *i.e.*, below the instrument when *in situ*. By this means we can, without removing the sphygmograph, ascertain if the ivory pad is still in proper position, *i.e.*, exactly over the artery.

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[1] For further information on the subject the student is referred to Dr Mahomed's papers in the *Medical Times and Gazette* (January 20th, 1872, and following numbers), and in *Gant's Surgery* (vol. i., page 52), to which I am indebted for much of my information on the subject, and from which some of the following rules for the application of the instrument are derived.

Before applying the sphygmograph, the harmless nature of the procedure must be explained to the patient, for it is of the greatest importance to avoid anything which will excite or disturb the action of the heart. Some persons become considerably agitated, thinking that an operation is about to be performed. (One of my patients left the hospital rather than have the instrument applied.) In such cases a preliminary application to a fellow-patient or nurse is advisable.

3. *Apply the instrument*, having previously screwed up the clockwork and placed the pressure at zero.—The ivory pad must be *accurately* applied over the *very centre* of that part of the artery which lies at the inner side of the styloid process of the radius. By compressing the vessel at this spot, where it is superficial and rests upon bone, we can be quite certain that the entire pressure of the spring will be exerted upon it. The instrument is then firmly strapped to the arm. The straps should be unyielding—not elastic. The slide carrying the strip of smoked paper is next to be fitted into the frame. Care must be taken that the paper is firmly and evenly stretched. This is best effected by first accurately fitting it and doubling its edges over the frame, then removing and smoking it over a piece of burning camphor, and finally fitting it to the frame again. The paper should not be over-smoked, and the point of the pen must not press too heavily against it, or friction will prevent free movement, and the tracing will be imperfect.

4. *Adjust the pen.*—The point of the pen is then (by means of the screw D, see fig. 3) to be brought level with the centre of the strip of smoked paper, as shown in figs. 2 and 6.

5. *Regulate the pressure* by means of the screw L, until the maximum amount of movement of the writing lever is obtained.[1] There is a certain pressure, depending upon the amount of expansion which is going on in the artery, at which the tracing is best marked. If the pressure is too

[1] The student must not forget that every alteration of the pressure, *i.e.*, every alteration in the position of the steel spring necessitates a fresh adjustment of the writing lever by means of the screw D.

little, *i.e.*, if the steel spring just touches the artery in its expanded state, the rise of the lever will be insignificant; if, on the other hand, the pressure be too great, the artery cannot expand to its full amount, and the tracing will be imperfect, —the up stroke shortened (see fig. 7), and the perfect development of the secondary waves of the tracing interfered with.

FIG. 7 — *Tracing taken under too great pressure.*
The up-stroke is cut short, and the perfect development of the tracing prevented.

If the tracing is satisfactory, the name of the patient, the date, the nature of the disease, and the amount of pressure which is required for (*a*) the perfect development of the tracing, and (*b*) the complete obliteration of the pulse in the artery (the latter being the guage of the strength of the pulse) should be inscribed upon the slip of smoked paper by means of a needle or other fine-pointed instrument, and the tracing rendered permanent by dipping it in a rapidly drying varnish.[1]

Character of a good tracing.—In a good tracing the apex (*b*, fig. 8) is pointed; and the best tracing is that in which the

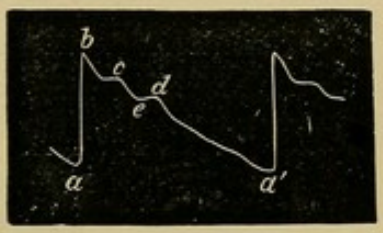

FIG. 8.—*Normal Pulse Tracing.*

up-stroke is tallest and the apex most pointed. It occasionally happens that the apex is rounded, as in some aneurisms

[1] The varnish recommended by Mahomed is made by macerating an ounce of gum benzoin in five ounces of rectified spirit; the mixture, which should be frequently agitated, is allowed to stand for two days, and the clear liquor then poured off from the insoluble constituents of the gum. Dr Dudgeon uses the crystal varnish of photographers, or a varnish consisting of one ounce of gum Damar and six ounces of rectified Benzoline.

(see figs. 9 and 10), and in a few cases of aortic stenosis (see fig. 11); but this is so extremely rare, that a tracing in which the apices are rounded should always be regarded as imperfect, unless it has been verified by repeated and careful readjustments of the instrument, and by repeated alterations of the pressure.[1]

FIG. 9.—(Left radial). Pressure, 2¾ oz.

FIG. 9.—*Aneurism of Left Axillary Artery* (*left radial tracing*).—L. G., æt. 63, admitted to the Newcastle Infirmary under Dr Byrom Bramwell, 7th March 1878, with a large aneurism of the left axillary artery. The apex is rounded; all the curves are obliterated.

FIG. 10.—Pressure, 3 oz.

FIG. 10.—*Aneurism of Left Subclavian* (left radial tracing).—J. M., æt. 50, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 5th September 1878: all the waves in the left tracing are obliterated.

FIG. 11.—Pressure, 1½ oz.

FIG. 11.—*Aortic Stenosis.*—J. B., æt. 51, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 29th November 1878, suffering from anæmia and dropsy. There was a well-marked aortic systolic murmur; the left ventricle was not hypertrophied. The pulse tracing seems to show that the murmur was organic.

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[1] In most cases of aortic stenosis a pointed apex can be obtained by careful adjustment and regulation of the pressure. (See figs. 12 and 13.)

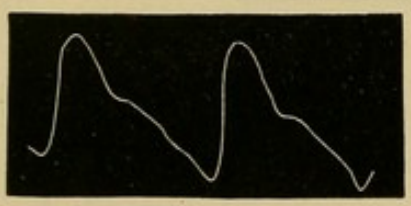

FIG. 12.—Pressure, 1½ oz.

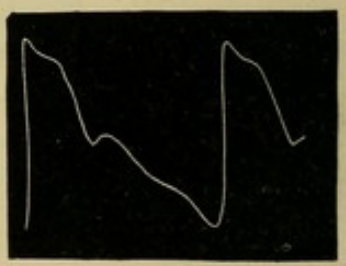

FIG. 13.—Pressure, 2¾ oz.

FIG. 12.—*Aortic Stenosis and Dilated Aorta.*—J. C., puddler, æt. 25, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 20th February 1879. The patient had been under observation for four years previously. Marked thrill and loud systolic murmur over base of heart and over aortic region. Heart moderately hypertrophied. Pressure = 1½ oz.

FIG. 13.—Tracing taken from the same patient with a slightly increased pressure; the apex is now pointed.

Dr Galabin points out that 'if the tracing of any pulse taken at a low pressure show a very marked primary summit, whose proportionate magnitude is modified by increase of pressure, then the tracing taken at the higher pressure more closely represents the pulse-wave. The form of trace at the lower pressure may, however, have much significance, and in these cases the whole of the information to be derived from the sphygmograph cannot be compressed into one curve, but requires at least two for its expression, namely, that trace which has the greatest amplitude, and another taken at a higher pressure.'[1]

The chief points to be attended to therefore in order to get a perfect tracing are:

(1) The accurate adjustment of the instrument, and

(2) The proper regulation of the pressure.

The following tracings, which were taken consecutively from the same pulse, illustrate the effects of different degrees of pressure.

Speaking generally it may be said that pulses of high tension require a considerable amount, and pulses of low tension a small amount of pressure for their perfect development. But to this general rule there are some exceptions. In atheroma, for example, the amount of pressure required to obliterate the pulse is usually considerable, the arteries are abnormally full, but the condition is not necessarily one of high tension.[2] In cases of atheroma the development of a pointed apex is often interfered with unless a small amount of pressure be employed.

In comparing the tracings from different arteries,—the two, radials, for example,—a procedure which is desirable in all

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[1] *Journal of Anatomy and Physiology*, vol. x. p. 306.

[2] In the first edition of this lecture I stated that the pulse in atheroma is one of low tension. This is not always the case. I should have said, that for the perfect development of the trace, a low pressure is (as a rule) required; and that the amount of pressure required to extinguish the pulse in atheroma is not a criterion of the blood pressure, *i.e.*, the tension of the pulse, for where the vessel is rigid a considerable amount of pressure is required to overcome the resistance of the arterial wall, and it is only after the rigidity of the arterial wall is overcome that the pressure is fully exerted upon the arterial contents.

cases of supposed aneurism or solid intra-thoracic growth, the best obtainable tracing from each pulse should, in the *first* place, be taken, all the conditions (with the exception of the pressure) such as the position of the patient, the tightness with which the instrument is strapped to the wrist, etc., being so far as is possible the same; and in this connection it is very important to remember that when the heart's action becomes excited as the result of emotional or other causes, the character of the tracing may be materially modified, as is shown in figs. 14, 15, 16, and 17. *Tracings should, therefore, always be taken if possible during tranquil action of the heart* In the *second* place, two tracings (one from either radial should be taken with *all* the conditions, including the amount of spring pressure the same.

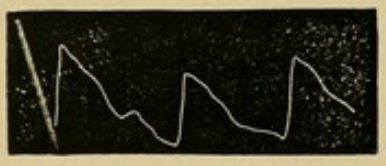

FIG. 14.—Pressure, 2½ oz.

FIG. 15.—Pressure, 2½ oz.

FIGS. 14 and 15.—*Alterations in the Pulse-tracing as the result of Cardiac Excitement.*—A. M., æt 48, admitted to the Newcastle Infirmary under Dr Byrom Bramwell, sufferin from obscure spinal symptoms. The heart became excited, and the tracing shown Fig. 15 was taken immediately after that shown in Fig. 14, the instrument in t meantime remaining *in situ*. The spring pressure was the same in each case.

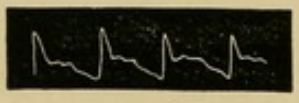

FIG. 16.—Pressure, 3 oz.

FIG. 17.—Pressure, 3 oz.

FIGS. 16 and 17.—*Alterations in the Pulse-tracing which result from Cardiac Excitement.* Figs. 16 and 17 show two tracings from a case of chlorosis. Case: E. F., æt 19, admitt to Newcastle-on-Tyne Infirmary under Dr Byrom Bramwell, 3d March 1878. T tracing shown in Fig 16 was taken on 8th March; the tracing shown in Fig. 17 was m three minutes later, the instrument having remained *in situ;* the heart had bec excited. Pressure in both cases = 3 oz.

DIRECTIONS FOR THE APPLICATION OF DUDGEON'S SPHYGMOGRAPH.

Dr Dudgeon gives the following directions for the application of his instrument :—

'1. Wind up the clockwork, used to drive the smoked paper along, by means of the milled button at the back of the clockwork box.

'2. Insert one end of the smoked paper (smoked side uppermost) on the right-hand side of the instrument, between the roller and small wheels.

'3. Make the patient hold out either hand open and in an easy position, the fingers pointing towards you (see fig. 18), and direct him not to move the wrist or fingers.

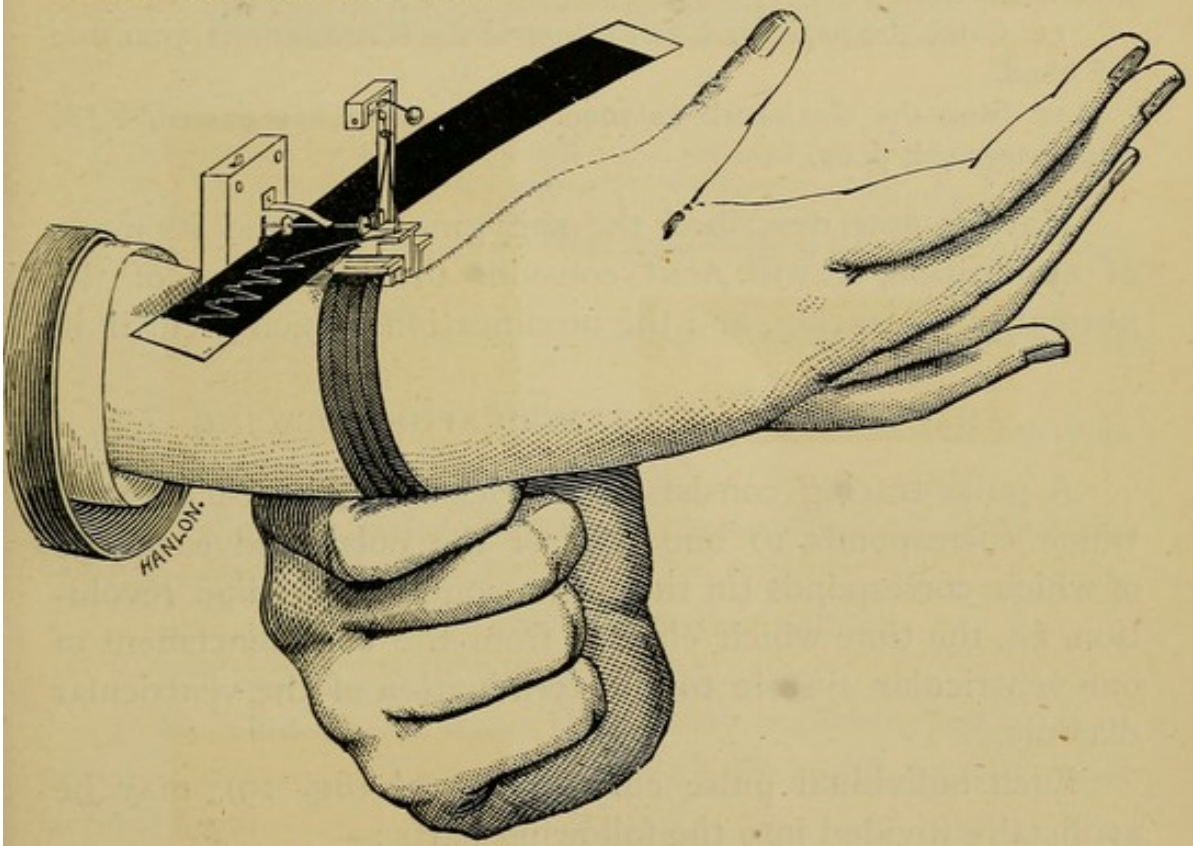

FIG. 18.—*Mode of applying Dudgeon's Sphygmograph.*

'4. Ascertain the precise spot where the radial artery beats at the wrist, close behind the eminence of the os trapezium.

'5. Slip the band, the free end of which has been drawn through the clamp, over the patient's hand.

'6. Apply pressure to the spring by turning the spring-regulator so that the number of ounces, or portions of ounces, you wish is pointed to by the indicator. The pressure may be altered at will when the instrument is fixed on the arm.

'7. Place the bulging button of the spring exactly over the artery, its long axis parallel to the course of the artery, the box containing the clockwork resting lightly on the forearm above.

'8. Retaining the instrument in its place with the left hand, draw the band through the clamp with the thumb and forefinger of the right hand, holding back the clamp with the other fingers of that hand; when the requisite tightness has been obtained, which will be known by the point of the needle working freely over the centre of the smoked paper, screw up the clamp with the left hand, so as to fix the instrument.

'9. Set the smoked paper in motion by pushing towards the right the small handle on the top of the clockwork box.

'10. Let the paper run through, and do not touch the instrument or the patient, unless to support his hand in your own right hand, to secure perfect steadiness.

'11. Catch the paper as it passes out of the instrument in your own left hand.

'12. Stop the clockwork as soon as the paper has passed.'—*The Sphygmograph*, p. 67.

Having now described the sphygmograph and its mode of application, I will next consider the characters of the normal pulse tracing, and the modifications which occur in it.

ANALYSIS OF A SPHYGMOGRAPHIC TRACING.

A pulse tracing consists of a series of curves, each one of which corresponds to one beat of the pulse, and each one of which corresponds (in time) to a complete cardiac revolution, *i.e.*, the time which elapses from the commencement of one ventricular systole to the termination of the ventricular diastole.

Each individual pulse curve, *a* to *a'* (fig. 19), may be artificially divided into the following parts:—

1. A line of ascent (*a* to *b*).
2. An apex (*b*).
3. A line of descent (*b* to *a'*).

This division is convenient for descriptive purposes, but a more natural division is that which separates each pulse wave into two portions (1 and 2, fig. 19) corresponding in time to the systole and diastole of the left ventricle respectively.

I will now describe each of these different parts.

The line of ascent or up-stroke[1] (*a* to *b* fig. 19) represents the sudden distension 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 partly due to the inertia of the instrument; and, in some cases, as in atheroma (where the vessels are extremely rigid), to impulse or shock.

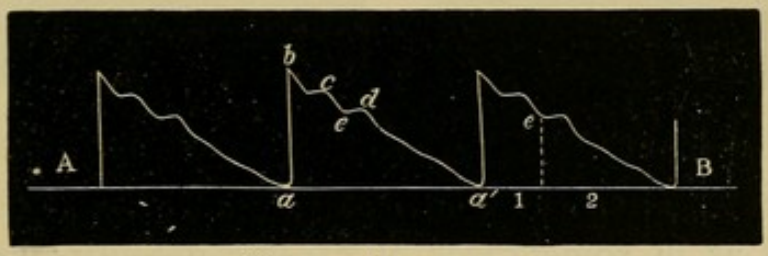

FIG. 19.—Pressure, 3 oz

FIG. 19.—*Sphygmographic Tracing of Normal Pulse.*— Male, æt. 25, admitted to the Newcastle Infirmary under the care of Dr Byrom Bramwell, suffering from psoriasis.

(1.) Line of ascent, up-stroke or percussion stroke = *a* to *b*.

(2.) Apex = *b*.

(3.) Line of descent = *b* to *a'*; *d* = aortic or dicrotic wave; *e* = aortic notch; *c* = tidal wave.

A B = base or respiratory line.

1 = Systolic portion of the tracing, *i.e.*, with reference to the systole and diastole of the ventricle, not of the artery.

2 = Diastolic portion of the tracing.

(*Note.*—Dr Mahomed tells me that he thinks the tidal wave in this tracing is rather too sustained, considering the amount of pressure, viz., 3 oz.)

The *direction* of the up-stroke (whether vertical or oblique) depends (chiefly) upon:—

1. The suddenness of the ventricular systole.
2. The condition of the aortic segments.
3. In some degree upon the facility with which the blood wave is propagated from the base of the aorta to the radial artery, and
4. The condition of the arterial (radial) coats.

In the normal tracing the up-stroke 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

[1] It is better, I think, not to use the term *percussion stroke*, proposed by Mahomed, for unless the arteries are rendered extremely rigid by atheroma, percussion or shock probably takes little or no part in the production of the up-stroke.

health, as it is, for example, in some cases of aortic regurgitation, and in conditions of cardiac excitement, the up-stroke is quite vertical, or (in tracings taken with Marey's instrument) it may even slope backwards. (See figs. 20 and 21.)[1]

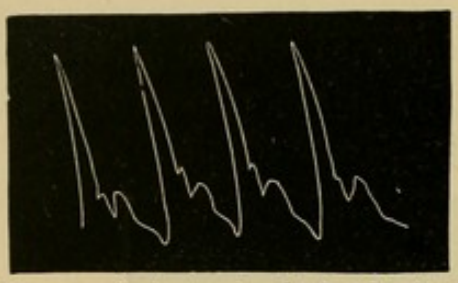

FIG. 20.—*Pulse-tracing during Cardiac Excitement.*

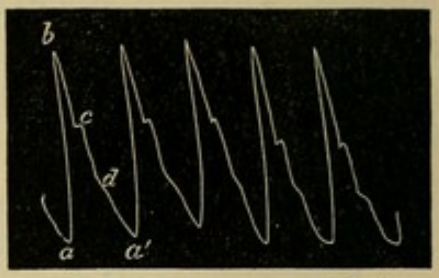

FIG 21.—*Aortic regurgitation.*

Vice versâ when the ventricular contraction is slow and hesitating, as in some cases of cardiac debility; when the aortic cusps are rigid; when the arteries are obstructed either by internal or external causes, such as the pressure of a tumour, the presence of an atheromatous patch (at the orifice of the innominate in the case of the right radial for example); or, when a globular aneurismal dilatation is situated between the heart and the vessel (*i.e.*, the radial), the up-stroke may be oblique. (See figs. 22 and 23.)

FIG. 22.— Pressure 1½ oz.

FIG. 22.—*Aortic Stenosis.*—J. B., æt. 51, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 29th November 1878, suffering from anæmia and dropsy. There was a well-marked aortic systolic murmur; the left ventricle was not hypertrophied. The pulse-tracing seems to show that the murmur was organic, and not hæmic.

FIG 23 —(Left radial) Pressure 2¼ oz.

FIG. 23 —*Aneurism of Left Subclavian.*—J. M., æt 50, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 5th September 1878; all the waves in the tracing are obliterated.

[1] In cases of hypertrophy of the left ventricle with high arterial pressure, the ventricular contraction may be laboured and prolonged in order to overcome the obstruction, but the commencement of the contraction is sudden, and the up-stroke vertical.

The *height* of the up-stroke represents the degree of distension of the vessel, and depends upon—

1. The force and (to a less degree) the suddenness with which the aortic cusps are raised, *i.e.*, upon the force (and suddenness) of the contraction of the left ventricle less the resistance offered by the aortic valve-cusps.

2. The extensibility of the arterial coats, which in its turn depends upon the condition of the arterial tunics (their elasticity or rigidity), and the state of the vaso-motor system.

3. The amount of compression which is applied to the artery, *i.e.*, the spring pressure of the sphygmograph.

The up-stroke is tall in simple nervous palpitation, and in cases in which a large amount of blood is suddenly thrown into the arterial system by a hypertrophied left ventricle.

In free mitral regurgitation the left ventricle may be considerably hypertrophied, but the up-stroke is short, for in such cases the large leak at the mitral orifice prevents the distension of the arterial system. So, too, in Bright's disease and atheroma, a powerful (hypertrophied) left ventricle may fail to produce a tall up-stroke, owing to the unyielding condition of the arterial wall.

Vice versâ, the up-stroke is *short* where the left ventricle is weak or acting feebly, and in all conditions in which a small amount of blood is being pumped into the arterial system, as in aortic stenosis and mitral lesions.

The apex of the tracing, or the *primary ventricular wave*[1] (*b* in fig. 19) as it may be termed, is, in the great majority of tracings,—both normal and pathological—pointed; in fact, as I have previously remarked, a rounded apex is so extremely rare, that any tracing in which the apices are rounded should be regarded with suspicion, and should never be accepted as the best attainable (most perfect) tracing without careful readjustment of the instrument and alteration of the pressure.

[1] The term '*percussion wave*' is applied to the apex by Mahomed, but for the reasons already given (see foot-note, page 19) I have not used it in this edition.

A rounded apex does, however, occasionally occur. It is met with in some aneurisms; the angles of the pulse curve are, as it were, flattened out (obliterated) either in the passage of the blood wave through the sac of the aneurism, or by alterations at the mouths of the vessels arising from the sac, or as the result of the pressure of the aneurismal sac on the vessels arising from it. A rounded apex is also seen in some cases of aortic stenosis; but in these cases, and in cases of atheroma, a pointed apex can, I think, usually be obtained by accurate adjustment of the instrument and careful regulation of the pressure.

The line of descent.—In the normal pulse tracing the line of descent (*b* to *a'* in fig. 19) is gradual,[1] and is interrupted by one or more secondary waves, the most important of which is the dicrotic.

The *direction* of the line of descent depends upon—(*a*) the facility with which the blood passes out of the arteries; (*b*) the rapidity of the heart's action; and (to a much less extent) (*c*) the condition of the arterial coats. In the normal condition of things the blood takes some time to flow from the arterial system into the capillaries, the recoil of the arteries is gradual, and the line of descent is sloping.

When the outflow from the arterial system is more difficult than in health, as for example, in the cirrhotic form of Bright's disease, the line of descent is still more gradual. *Vice versâ*, when the outflow from the arterial system is very rapid in consequence of a dilated condition of the small arteries and capillaries; and in aortic regurgitation, in which the arteries suddenly collapse in consequence of the back flow through the aortic valves, the line of descent becomes more and more vertical in proportion to the freeness of the outflow and the rapidity of the action of the heart.

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[1] The line of descent is sloping, because the recoil of the artery is gradual. In this respect there is, therefore, a marked difference between the up-stroke and the line of descent.

The *dicrotic* wave, or the *aortic systolic wave*, as it may be called, is usually present in a normal pulse tracing, and corresponds to that period of the cardiac cycle which immediately follows the closure of the aortic valve cusps: while the point of the tracing which immediately precedes it (*e* in fig. 19), and which is generally, but not universally,[1] 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,—a condition which occurs in robust health,—the dicrotic wave is very feebly marked or altogether absent. (See fig. 24.)

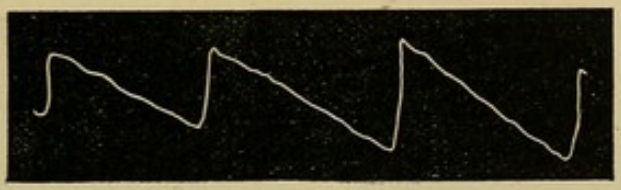

FIG. 24.—*Pulse tracing of good tension* (*pressure* 3 *oz.*) The pulse is slightly irregular, but otherwise normal; the dicrotic wave is scarcely perceptible.

The exact cause of the dicrotic wave has given rise to much debate; but most physiologists are agreed that it is in great part due to a recoil current from the closed aortic valve,—an opinion which is confirmed by clinical observation. Dr Galabin, while agreeing that the great cause of the dicrotic wave is a recoil current from the aortic valves, thinks that its production is aided by the inertia of the fluid.[2]

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[1] *A Text Book of Physiology*, 4to edition, p. 172.

[2] 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 very generally accepted as depending upon the aortic valves. For let us consider a section of artery close to the valves. When the influx from the heart suddenly ceases at the end of systole, the fluid for an instant continues to flow away out of the section on account of its acquired velocity, and the pressure in the section, therefore, 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 valves, causes a second increase of pressure and second expansion. This is propagated as the dicrotic wave into the periphery, and may itself again call out a second similar oscillation or tricrotic wave, which is not unfrequently seen in the pulse. 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. If the fluid in the tubes be air instead of blood or water, its momentum is so small that its velocity is checked instantly at the end of systole,

Dr Roy differs from this opinion. As the result of careful experiment he suggests that the secondary waves which appear in the unopened artery under normal blood-pressure, are the result of an active vermicular contraction of the muscular coat of the arteries. He completely rejects the theories which would ascribe these undulations to reflected, opening and closing waves, etc., or in fact to secondary waves at all, of whatever character. The tracings which he obtained from the opened artery of the rabbit, under a normal blood-pressure, never showed 'the slightest trace of secondary waves superposed on the primary or pulse wave,' although the instrument he used was quite delicate enough to record them did they really exist.—*Michael Foster's Journal of Physiology*, 1879-80, page 76.

The dicrotic wave is absent or feebly marked in free aortic regurgitation. (See figs. 25 and 26.)

FIG. 25.—Pressure, 2½ oz.

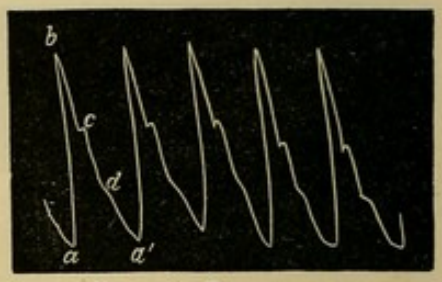

FIG. 26.—Pressure, 3 oz

FIG. 25.—*Aortic Regurgitation.*—Case: G. A., æt. 56, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 21st February 1878, suffering from shortness of breath and swelling of feet. Had been ill for three months. The face was pale and anxious, lips slightly dusky. Double aortic murmur; heart considerably hypertrophied; apex between 6th and 7th ribs, 3 inches below and 2 inches outside left nipple. Considerable hypertrophy and engorgement of right heart. Died 5th March 1878. Aorta very atheromatous; aortic valves very incompetent; segments shrunken, turned in towards the ventricle; coronary arteries much obstructed; cardiac walls fatty; left ventricle dilated; pericardium adherent. The arteries were practically empty during the ventricular diastole. *a-b* = percussion stroke; *b* = apex; *c* = tidal wave; *d* indicates the position of the aortic wave, which is absent in this tracing.

FIG. 26.—*Aortic Regurgitation.*—Taken from same patient as No. 25, after administration of digitalis. The letters have the same significance as in Fig. 25.

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and there is no perceptible dicrotic wave. If, on the contrary, mercury be taken, both the dicrotic and succeeding waves become enormous, on account of the great momentum of the fluid, as was shown by Marey. The fluid remaining the same, the oscillation will be more ample the greater the initial velocity, and the more slowly that velocity is checked. Thus dicrotism is promoted by a sudden action of the heart, and also by distensibility of arteries, by lowness of arterial pressure, and by freedom of outflow. I think that in considering this origin of the dicrotic wave, sufficient attention has not generally been paid to the important part played in it by the inertia of the fluid, and to the fact that the aortic valves, although extremely important, are not absolutely essential.'—*Journal of Anatomy and Physiology*, vol. x. p. 303.

It is also faintly 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.

It is sometimes greatly exaggerated, and the pulse is then said to be *dicrotic* (see figs. 27, 28, and 29).

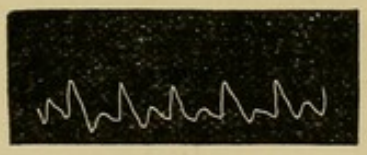

FIG. 27.—Pressure, 3 oz.

FIG. 27.—*Dicrotism.*—A. H., æt. 32, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 21st March 1878, with an enormous scrofulous kidney. There were occasional rigors. This tracing was made during a rigor, the temperature being 100° F.

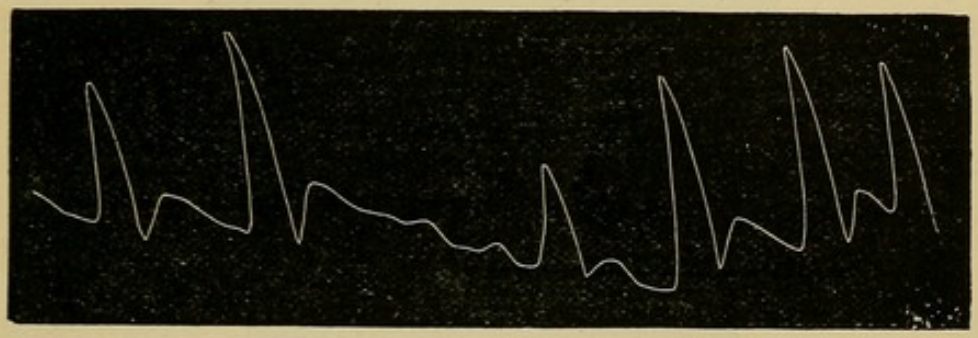

FIG 28.—*Irregular and Dicrotic Pulse in Pneumonia.*—J. R., æt. 68, admitted to the Newcastle Infirmary under Dr Byrom Bramwell, 28th December 1878. Died 31st December. Croupous pneumonia, limited to the upper lobe of the right lung. No cardiac affection.

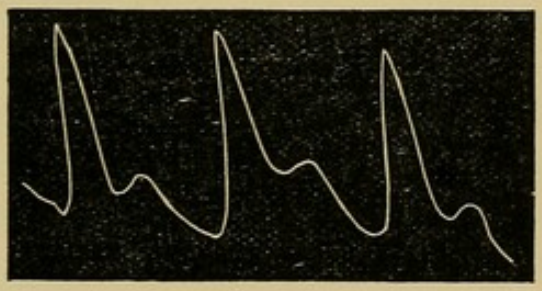

FIG. 29.—*Dicrotic Pulse from a case of Rheumatic Fever.*

Degrees of dicrotism, and their significance.—Various degrees of dicrotism occur, 'to which terms have been applied indicating the relation of what is known as the "dicrotic notch" to the respiratory line of the tracing' (Mahomed).[1]

[1] *Gant's Surgery*, vol. i. p. 56.

They may be said to represent the relative condition of the artery at the point where the sphygmograph is applied, as regards its fulness or state of distension at the commencement, and at the termination of the ventricular systole respectively.

(*a*) When the dicrotic wave is well marked, but the aortic notch C is above the base line A B (see fig. 30), the pulse is called *dicrotic*. In this condition the artery is more distended at the end of the ventricular systole than it is at the commencement.

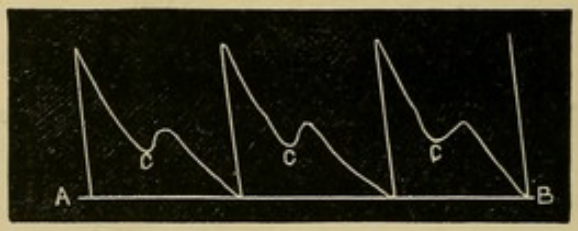

FIG. 30.—*Dicrotic Pulse.* (*After Mahomed.*) A B, = base line; C, = aortic notch.

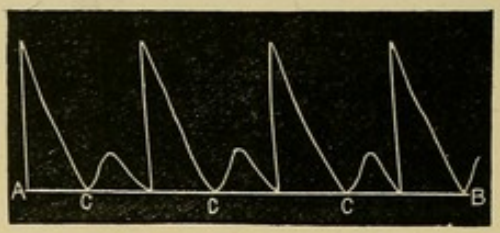

FIG. 31.—*Fully Dicrotic Pulse.* (*After Mahomed.*) A B, = base line; C, = aortic notch.

(*b*) When the aortic notch C reaches the level of the respiratory or base line A B, the pulse is called *fully dicrotic*. In this condition the artery is *apparently* no more distended at the end of the ventricular systole than it is at its commencement, and it is *apparently* less distended than it is during the ventricular diastole[1] (see fig. 31).

(*c*) When the aortic notch C sinks below the level of the

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[1] This does not of course imply that the arterial system, *as a whole*, is more empty at the end of the ventricular systole than it is during the ventricular diastole, but simply that the vessel at the point where the observation is taken, *apparently* presents such a condition; I say apparently, for the depression of the curve at the end of the ventricular systole is doubtless in part due to the sudden fall of the lever itself.

respiratory line A B, the pulse is called *hyperdicrotic.* (See fig. 32). In this condition the artery (at the point of observation) is *apparently* less distended at the termination of the ventricular systole than it is at its commencement.

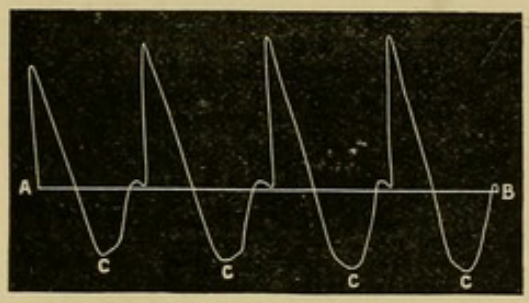

FIG. 32—*Hyperdicrotic Pulse.* (*After Mahomed.*) A B, = base line; C, = aortic notch.

When dicrotism is well marked, *i.e.*, when the pulse is fully dicrotic, each cardiac cycle is (apparently) attended by two pulse beats, hence the term dicrotic, or double pulse.

The conditions which favour dicrotism are:—

1. A low condition of arterial tension.
2. Freedom of outflow from the arterial system, *i.e.*, through the capillaries.

(Both of these conditions are usually due to one and the same cause, viz., loss of vaso-motor tone).

3. A sudden sharp ventricular systole.
4. Elasticity of the arterial walls.

According to Dr Burdon Sanderson, 'dicrotism is characteristic of that condition of the circulation in which the arterial pressure is diminished, while the venous is increased. It denotes that the capillary current, instead of being constant in its rate of movement, is markedly accelerated during diastole, and retarded during the diastolic interval.'

Dr Roy thinks that the dicrotic wave of fever, which is associated with reduced blood-pressure, is due to a secondary and most probably reflected wave.

He found that any considerable reduction of the medium blood-pressure, from whatever cause, leads to the appearance of a dicrotic pulse-wave in tracings both from the opened and unopened artery. 'This form of dicrotism,' he says, 'must not be confounded, as is often

done, with those undulations more or less marked, which give to the pulse curve in health its characteristic outline. The dicrotism from reduction of blood-pressure, as it is seen in tracings from the new opened artery, is characterised by the fact, that it does not disappear when the extra-arterial pressure is raised nearly as high as the blood-pressure, showing that it is really due to a secondary and most probably reflected wave.—*Michael Foster's Journal of Physiology*, 1879, 1880, page 80.

Clinically, the pulse is dicrotic in cases in which the nerve tone (vaso-motor system) is feeble. Many persons who apparently enjoy good health have dicrotic pulses. Such persons are easily 'knocked up,' are unable to undergo any severe and prolonged strain, and are most unfavourable subjects for an attack of continued fever; in them a severe pneumonia, or an attack of typhus, would almost certainly be fatal. A comparatively slight elevation of temperature in such persons causes the pulse to become *fully dicrotic* or even *hyperdicrotic*. (See figs. 33, 34, and 35.)

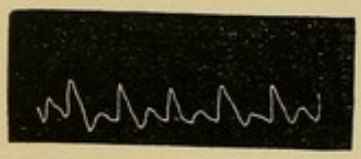

FIG. 33.—Pressure, 3 oz.

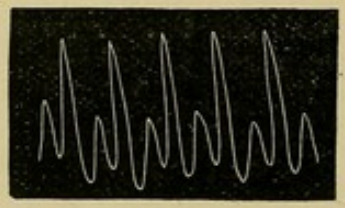

FIG. 34.—Pressure, 3 oz.

FIG. 35.—Pressure, 4 oz.

FIG. 33.—*Dicrotism.*—A. H., æt. 32, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 21st March 1878, with an enormous scrofulous kidney. There were occasional rigors. This tracing was made during a rigor, the temperature being 100° F. A draught of hot milk was administered, and the tracing, shown in Fig. 34, was taken. Tracing Fig. 35 a few minutes later.

FIG. 34.—*Hyperdicrotism.*

FIG. 35.—*Hyperdicrotism.*

The great clinical condition with which dicrotism is associated is pyrexia. Where a considerable elevation of temperature (102°-104° Fahr.) continues for some time, as in enteric and typhus fever for example, the pulse usually becomes dicrotic. A dicrotic pulse under such circumstances indicates the free use of stimulants.

Hyperdicrotism is (as a rule) only seen in cases of high fever with great exhaustion, but in debilitated subjects it may, as I have previously remarked, be produced by slight

elevations of temperature. In the hyper-dicrotic pulse the second (dicrotic) beat is cut short by the up-stroke of the ventricular systole of the next beat.

If the rapidity of hyper-dicrotic pulse is increased the second beat is lost altogether, and the pulse is then said to be *monocrotic.* (See fig. 36.)

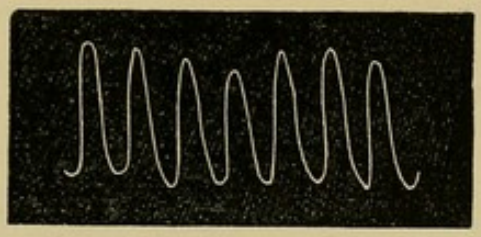

FIG. 36.—*Monocrotic Pulse.* (*After Riegel.*)

The *predicrotic, true tidal,* or *second ventricular systolic wave.*

Between the apex of the tracing and the aortic notch a second wave (see fig. 19) is often, but not always, seen. Dr Mahomed thinks that it corresponds to the steady onward passage of the blood which results from the ventricular systole, hence he has termed it the *true tidal* wave. It is sometimes called the *predicrotic* wave, because of its relationship to the dicrotic wave. It may, I think, with advantage be termed the *second ventricular systolic wave,* for it occurs after the apex or first ventricular systolic wave, and during the systole of the ventricle.

The essential condition, which favours the production of the second ventricular systolic wave, is increase of the arterial tension during the ventricular systole. This condition (increased arterial tension) is usually due to difficulty of arterial outflow as in Bright's disease (see figs. 37 and 38), and in atheroma (see fig. 39); but it may also result from an excessive amount of blood being propelled into the arterial system at each ventricular systole. An excellent clinical example of the latter condition is seen in aortic regurgitation, in which a powerful and dilated ventricle propels a large quantity of blood into the arterial system, producing high tension during systole, with a well-marked *predicrotic wave,* but in which the

arterial pressure during the ventricular diastole is extremely feeble.[1]

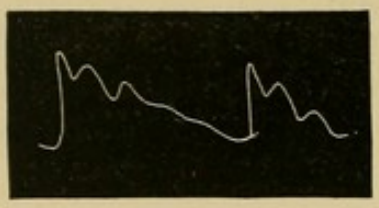

FIG. 37.—Pressure, 5 oz.

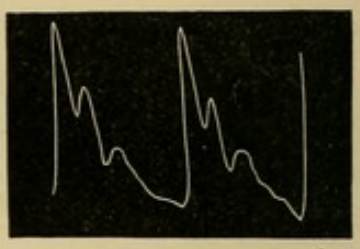

FIG. 38.—Pressure, 4 oz.

FIG. 37.—*Chronic Bright's Disease.*—D. G., æt. 40, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 5th September 1878, suffering from renal dropsy (large white kidney). The pulse is one of high tension; the tidal wave is strongly marked.

FIG. 38 —*Acute Bright's Disease.*—Tidal wave strongly marked, from a patient admitted to the Newcastle Infirmary under the care of Dr Drummond.

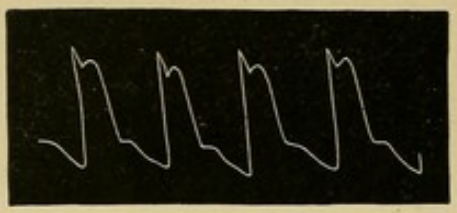

FIG. 39.—*Atheroma and Aneurism of Aortic Arch.*—J. D., æt. 52, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 21st February 1878, suffering from aneurism of the ascending portion of the aortic arch and atheroma. The tidal wave is very strongly marked. There was no perceptible difference between the two pulses.

Vice versâ, the second ventricular systolic wave is absent in those cases in which the arterial tension during the ventricular systole is low. Now, low tension during the ventricular systole may be due either to an insufficient amount of blood being pumped into the arterial system during the ventricular contraction, a condition which is seen in cases of cardiac weakness, mitral disease, etc.; or, it may result from an abnormally free outflow from the arterial system during the ventricular systole, a condition which is

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[1] Dr Galabin (formerly) supposed that the separation of the primary, or so called 'percussion' and tidal waves did not really exist in the artery, but was produced in the trace by the velocity acquired by the sphygmograph in the sudden primary up-stroke. Further observation, he states, 'has convinced him that, although this explanation applies to many cases, it yet does not express the whole truth, and that in some instances at least there is a real first secondary wave or oscillatory expansion in the artery, *i.e.*, the tidal or predicrotic wave.'—*Journal of Anatomy and Physiology*, vol. x. p. 299.

due to a dilated condition of the small arteries and (?) capillaries. In such cases the pulse rapidly falls away *during the ventricular systole*, in other words, after the first distension of the arterial wall there is a quick and rapid collapse, which is only arrested by the occurrence of the dicrotic wave; and this is, as we have seen, chiefly a recoil wave from the closed aortic valves. A rapid collapse of this description is best marked in the dicrotic and hyperdicrotic pulse of fever, and in conditions of vaso-motor debility and relaxed vessels. Hence it will be easily understood why in these cases the second ventricular systolic wave is not present.

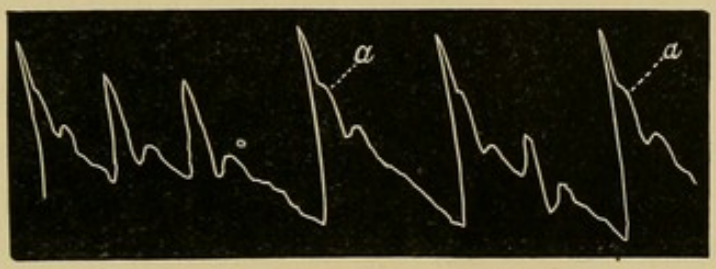

FIG. 40.—*Irregular Pulse in mitral regurgitation, with hypertrophy of the Left Ventricle.*—In the tallest curves the tidal wave, *a*, is well marked, while it is absent in the smaller ones. The letters *a*, *a*, point to the predicrotic wave.

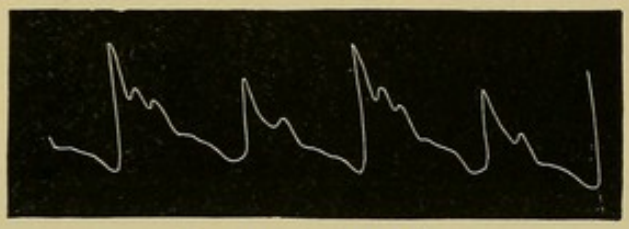

FIG. 41.—Pressure 4¼ oz.

FIG. 41.—*General Atheroma.*—J. D., æt. 60, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 21st October 1878. The radials were very tortuous and rigid; there was no valvular lesion. The tidal wave only occurs every alternate beat.

It not unfrequently happens that the predicrotic wave is present in some pulse curves of a tracing, but absent in others. This condition, which depends of course upon the fact that the arterial pressure during the ventricular systole is greater during some pulsations than during others, is especially frequent in mitral stenosis, in which condition a varying amount of blood is apt to be discharged into the cavity of the left ventricle, and thence into the arterial system. (See fig. 40.) Occasionally the predicrotic wave occurs every alternate beat, as shown in fig. 41; in that case the irregularity was probably due to nervous causes.

Other secondary waves sometimes occur in the lower part of the line of descent. Their exact cause is obscure, but so far as is at present known, they are of little practical importance.

These waves are probably as a rule due to the inertia of the instrument. Occasionally a small wave is seen to occur immediately before the up-stroke, *i.e.*, immediately before the contraction of the ventricle. Possibly it may be due to the contraction of the left auricle.

Respiratory or base line.—In a normal tracing the lowest points of the up-strokes of succeeding pulse waves are on the same horizontal plane (see fig. 19), and a line drawn through the bases of the up-strokes is called the *base* or *respiratory* line. The latter term (respiratory line) is applied to the base line because inspiration and expiration exert, sometimes even in health, but notably in some cases of disease, a marked influence upon it. During a full and sudden inspiration the arterial tension is lowered, and the base line falls; during expiration, on the contrary, the arterial tension is increased, and the base line rises. In cases of spasmodic asthma and severe dyspnœa the base line may be very uneven. (See figs. 42 and 43.)

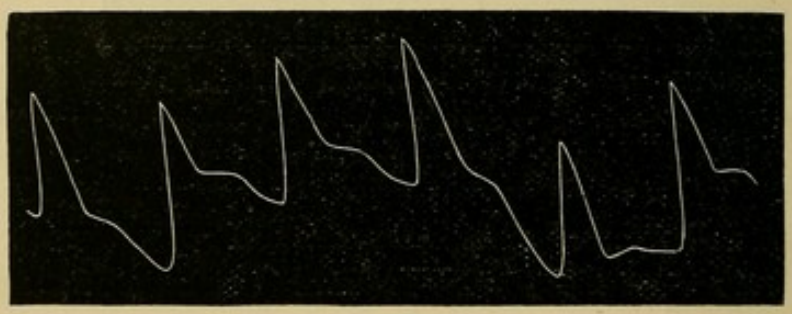

FIG. 42.—Pressure, 3¾ oz.

FIG. 42.—*Uneven Respiratory Line.*—J. R., æt. 31, admitted to Newcastle Infirmary 26th December 1878, under Dr Byrom Bramwell, suffering from acute bronchitis.

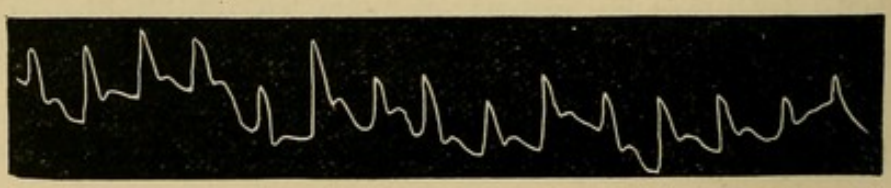

FIG. 43.—*Uneven respiratory line (from a Case of Spasmodic Asthma)* The tracing was taken during the paroxysm.

In the normal pulse tracing, during ordinary (tranquil) respiration, each consecutive pulse curve is an exact repetition of the preceding one; in other words, the pulse is regular in time, the different pulse waves are equal in volume, and the individual character of succeeding pulse curves, in respect to the different parts of which they are composed, is the same.

Having analysed the normal pulse tracing, and described the more important modifications which it undergoes, I will next proceed to consider the character of the pulse in health and disease.

THE FREQUENCY OF THE PULSE.

The frequency of the pulse is determined by counting the number of pulsations which occur in a minute. To insure accuracy, the pulse should be counted for a whole minute, and not for a quarter of a minute, as is sometimes done. In some cases in which the pulse at the wrist is very feeble, the frequency of the cardiac contractions can be best determined by auscultation over the præcordia.

The exact frequency of the pulse can also be determined by means of the sphygmograph. Mahomed's modification of Marey's instrument is so constructed that four inches of the slide are propelled past the point of the writing lever in ten seconds. In order, therefore, to ascertain the frequency of the pulse per minute, the number of pulse waves in four inches of the tracing must be multiplied by six. Dudgeon's instrument is also constructed so that six times the number of pulsations traced on the slip of paper give the number of beats per minute.[1]

In counting a very rapid pulse, Dr Abbot describes, in the *New York Medical Record* for August 12, 1882 (quoted in the *Medical Times and Gazette*, September 30, 1882), a method which he adopted for counting the heart's action during some experiments he performed with alcohol on birds. He found that he was unable to count by the usual mode when the cardiac contractions exceeded 240 per minute, whereas, by the method he now describes, he easily counted 280. 'During a definite

[1] These measurements only hold good so long as the slide is travelling at full speed. To insure accuracy, therefore, the clock-work should be fully wound up before the tracing is taken.

part of a minute, usually one-fourth, dots were made with a lead pencil upon a sheet of paper, *synchronous with the heart's beats*, as heard over the cardiac region. The dots were then counted. A pulse of *four hundred* could be taken in this way, provided each pulsation were distinct enough to be discriminated by the ear. The indistinctness of the separate pulsations alone fixes the limits to the use of this method, as the human hand is capable of marking intelligently and with accuracy at the rate of 450 dots per minute, for thirty seconds, which rate is probably beyond not only that of the human heart, but also of the pulse of any of the lower animals available for experiment. I have had a sufficient experience with this method,' he says, 'to know that it is of practical value, especially with children. All movements, whether of the body or not, that can be seen, felt, or heard, can be thus counted up to 400 or 500 per minute, provided that they are sufficiently distinct to be discriminated.'

Frequency in health.—The normal frequency varies in different individuals, and in the same individual under different circumstances. The average normal rate in the adult male in a state of rest is 72, but there are many exceptions. In some persons the pulse rate is habitually as high as 100, in others as low as 50. In practice, therefore, such idiosyncracies must be kept in view. The pulse is quicker in children than in adults, but it quickens slightly again in old age; it is quicker in women than in men. The pulse rate is increased by active exercise (bodily or mental). It varies, too, with the position of the body, being quicker in the standing than in the sitting, and in the sitting than in the recumbent position. Its frequency also varies with the time of day, being lower in the early morning hours. It is decreased during sleep and increased after a meal. It varies with the temperature of the body, and is to a slight extent influenced by the atmospheric pressure. The frequency of the pulse is also profoundly influenced by emotional disturbances, and by the mental condition, hence it is often difficult to get a proper estimate of the pulse-rate in children and nervous persons, the mere presence of the doctor being sufficient to increase the frequency by 10, 20, 30, or even 40 beats. Due allowance must of course be made for this and other disturbing causes. It is often a good plan to count the pulse at the

end of the visit, or at all events to allow sufficient time for any temporary alteration in the pulse-rate to have disappeared.

TABLE OF THE AVERAGE PULSE RATE AT DIFFERENT AGES.

Fœtus in utero,	about 140
Child newly born,	140—135
„ 1st year,	120—110
„ 2d year,	105—100
„ 3d year,	100— 85
7th—14th year,	85— 80
14th—20th year,	80— 72
21st—60th year,	70— 75
Old age,	75— 80

Alterations in the pulse-rate, which occur in disease.

The pulse-rate may be either increased or diminished by disease.

The pulse-rate is increased in :—

1. *Pyrexia* (increased temperature).—As a general rule the amount of increase varies with the height of the temperature. According to Dr Aitken, an increase of temperature of one degree above 98° Fahr. corresponds with an increase of ten beats of the pulse per minute, as shown in the following table :—

Temp. Fahr.	Pulse-rate.	Temp. Fahr.	Pulse-rate.
98°	60	103°	110
99°	70	104°	120
100°	80	105°	130
101°	90	106°	140
102°	100		

Exceptions.—In some cases of typhoid, especially in its earlier stages, and in meningitis, the pulse may be slower than natural. At the commencement, too, of some cases of pericarditis the frequency of the pulse is diminished—(Stokes).

2. Conditions associated with extreme debility. This is chiefly the case where there has been some previous elevation of temperature, or where the nerve irritability of the heart is increased.

3. Cases in which the vagus is paralysed or the cervical sympathetic irritated. In these cases the nerve balance of the heart is deranged, and the pulse-rate increased. In a few cases (as for instance, in exophthalmic goitre in which the sympathetic is irritated, and in the later stages of basilar meningitis, in which there is probably paralysis of the vagus), the nerve derangement depends upon organic disease; but in the large majority of cases, as in hysteria, in which affection the pulse frequency may be enormously increased, the condition is a functional one.

4. In some cases of organic cardiac disease, especially mitral regurgitation (see fig. 44), and (to a less extent) in aortic regurgitation (see fig. 45).

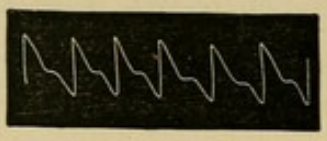

FIG. 44.—Pressure, 3 oz.

FIG. 44.—*Mitral Regurgitation.*—M A. C., æt. 16, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 24th January 1878, suffering from cough and shortness of breath, dating from an attack of rheumatic fever two months previously. Heart's action very rapid (120-130). The first sound appeared to be reduplicated; a systolic murmur was audible at the apex when the heart became slower.

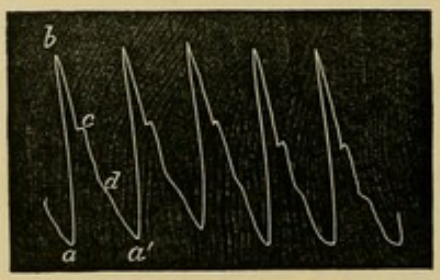

FIG. 45.—*Pulse tracing from a Case of Aortic Regurgitation.*

The Clinical Significance of Increased Frequency of the Pulse.

Increased frequency of the pulse, when not merely temporary, is very suggestive of pyrexia; it is, however, *per se*, an unreliable sign of fever, for, on the one hand, fever may be present without any increase (a diminished pulse-rate being, as I have already pointed out, sometimes met with, as in the earlier stages of typhoid and in meningitis); and, on the other, increased frequency of the pulse may occur without

fever, or even with a low temperature, as in pro-agonistic collapse. In order to ascertain the cause of the increased frequency of the pulse, the first step is to take the temperature. If there is no pyrexia, the causes mentioned under heads 2, 3, 4 must be looked for.

A slow pulse may be due to—

1. *Functional derangement of the heart.*—This is probably the cause of the slow pulse which occurs in jaundice, in some cases of gout, etc.

Non-febrile jaundice usually produces a retarded action of the heart and diminished arterial tension. The pulse may fall to 50, 40, or even 20 beats in the minute, and it may also be irregular.

Dr Wichham Legg and others believe, that the slowness of the pulse is produced by the presence of unchanged biliary acids in the blood.

Dr Murchison has seen a slow pulse (36-60 per minute) in cases of hepatic disease in which there was no jaundice.

2. *Organic lesions of the heart,* such as fatty degeneration of the left ventricle and aortic stenosis. In the former case (weakness of the ventricular wall) a slow pulse is perhaps exceptional, and is only seen when the patient is at rest. Any exertion which throws a strain on the beat, is attended in these cases with a quick, rather than with a slow, pulse. In the latter case (aortic stenosis) the left ventricle has difficulty in emptying itself, its contraction is prolonged and somewhat laboured, and the frequency of the pulse is diminished.

3. *Lesions of the nervous system,* in which the cardio-inhibitory centre in the medulla on the branches of the vagus which pass from that centre to the heart are irritated (stimulated). In some cases the condition is a temporary (functional) one, as for example, in those cases in which a slow pulse is associated with a neuralgic headache (megrim); in others, as for example meningitis (in the earlier stages of which the pulse may be abnormally slow), the lesion is organic.

4. *The rapid defervescence of fever;* the pulse-rate may in these cases rapidly fall from a high rate to a point much below the normal.

5. *Reflex stimulation of the cardio-inhibitory centre in the medulla.*—The reflex impulse may in all probability be generated by powerful stimulation of any peripheral nerve; but it most frequently arises in the alimentary tract.

The slow pulse which is so frequently seen in the earlier stages of typhoid, is probably due to reflex inhibition of the heart—the local lesion of the intestine stimulates the mesenteric nerves, producing an impulse which travels to the medulla, and is reflected down the vagus to the heart.

The Clinical Significance of a Slow Pulse.

A slow pulse *per se* (*i.e.*, without any associated signs or symptoms) is of little practical importance, the most frequent cause (provided that it does not depend upon idiosyncracy) is a temporary functional derangement of the heart. The possibility of the condition being due to irritation of the vagi must be remembered, and the symptoms of disease at the base of the brain or in the course of the vagus looked for. In other cases (*i.e.*, where there are associated signs and symptoms) the clinical significance of a slow pulse entirely depends upon the cause of the condition, and the prognosis must be guided accordingly.

Variability of the pulse-rate.

The pulse-rate is in some cases liable to very marked fluctuations. In the convalescent stage of fevers, for example, and in conditions of debility, the pulse-rate is very variable; these changes are readily produced by any trivial movement or mental excitement. I have noticed the same liability to rapid changes in the pulse-rate in the earlier stages of some cerebral cases. Extreme variability of the pulse-rate has also been noticed after concussion of the spine.[1]

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[1] Dr Guinoiseau, quoted in the *Medical Times and Gazette*, relates in the *Bulletin de Thérapeutique*, February 28th 1882, the case of a man who had received a concussion of the spine from a fall from a carriage on May 9th, 1881. He recovered, and was able to resume his occupation, which was laborious, but a peculiarity in his pulse remained. Examined on October 8th, it was found that his pulse was 49 when recumbent, 73 when seated, and 109 when standing; and on November 1st the pulse-rate in these positions was respectively 45, 57, and 77.

THE RHYTHM OF THE PULSE.

The normal pulse (during tranquil respiration) is perfectly regular (the individual pulse waves being of the same duration and volume, and presenting the same features as regards individual curves); but departures from the normal are common, indeed in some persons who enjoy perfect health, the pulse is habitually irregular. Such idiosyncracies are more common in old than in young people.

The alterations in rhythm which occur in disease can of course be observed by the finger, but are best studied by means of the sphygmograph. They may consist of alterations in time, alterations in volume, or differences in the sphymographic characters of the individual pulse curves.

Time irregularities.—All degrees of time irregularity are met with. In some, the alteration is only occasional, occurring every ten, twenty, or thirty beats; in others, the normal rhythm of the pulse is very much altered or entirely lost. (See fig. 46.)

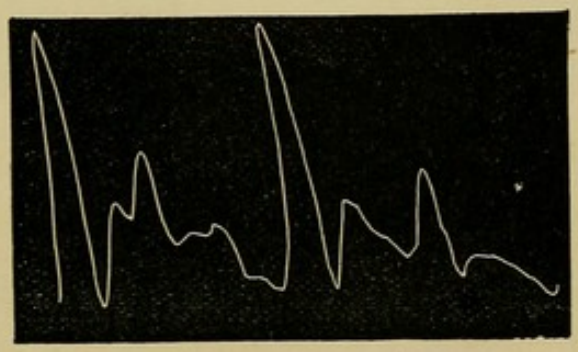

FIG. 46.—*Extreme irregularity of the pulse from a Case of Pneumonia.* Five pulse waves are shown in the tracing, which was taken twenty-four hours before death.

The irregularity sometimes consists in the omission of a beat (see figs. 47 and 48); the pulse is then said to be '*intermittent.*' Intermission of the pulse may be due either to arrest of the contraction of the left ventricle—a condition which is not uncommon as the result of simple nervous derangement, and is then of comparatively little importance; or, it may be owing to the fact that some of the ventricular

contractions fail (are too feeble) to raise the aortic valves and send into the arteries a pulse wave of sufficient strength to be felt at the wrist.

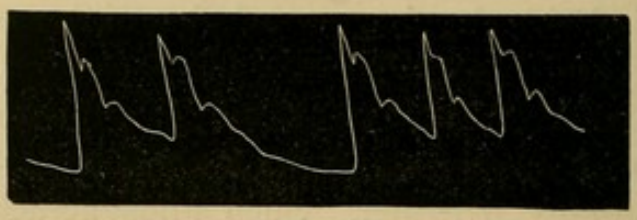

FIG. 47 —*Intermittent Pulse.*

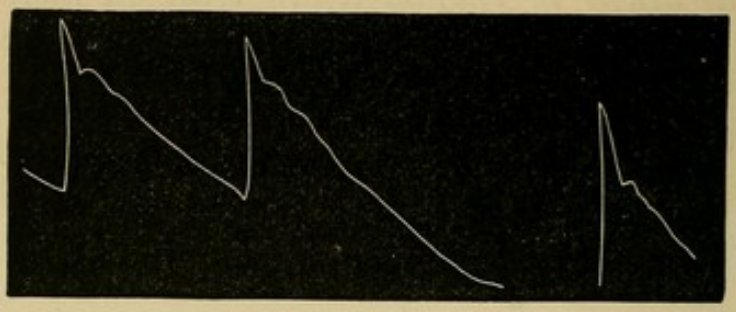

FIG. 48.—Pressure, 4 oz.

FIG. 48.—*Intermittent Pulse.*—J. B , æt. 38, admitted to the Newcastle Infirmary under Dr Byrom Bramwell, 11th July 1878, suffering from mitral regurgitation. The heart was much hypertrophied. Tracing made 6th January, when patient was much improved and attending as an out-patient.

This latter condition, which is a very serious indication of heart-failure, is chiefly met with in those cases in which the heart-walls are degenerated (dilatation, fatty and fibroid degeneration), but is also seen in some mitral lesions.

M Marey, quoted by Mahomed, *Guy's Hospital Reports*, 1879, p.397, gives the following explanation of this condition :—'In a heart in which mitral regurgitation occurs, blood is forced out of the ventricle in two directions during systole ; one portion passes through the aortic orifice, the other is driven backward into the auricle. Now, when the heart is dilated, it sometimes occurs that the ventrical contraction is not of sufficient strength to overcome the arterial resistance and raise the aortic valves ; it finds it easier to force all the blood backwards through the incompetent mitral, which thus plays the part of a safety valve. Meantime this intermission, which is caused in the pulse, allows time for more blood to flow out of the arterial system through the capillaries, and when the next contraction of the heart occurs, it finds the arterial pressure considerably decreased, and it is now able to open the valves and cause another pulse-wave to pass through the arteries.'

In some cases the irregularities occur at fixed intervals, *i.e.*, every two, three, or four beats. One of the most interesting of these is the *pulsus bigeminus* of Traube, in which the pulse waves run in pairs. (See figs. 49 and 50.) In other cases three pulsations occur together in a group, constituting the so called *pulsus trigeminus.* (See fig. 51.)

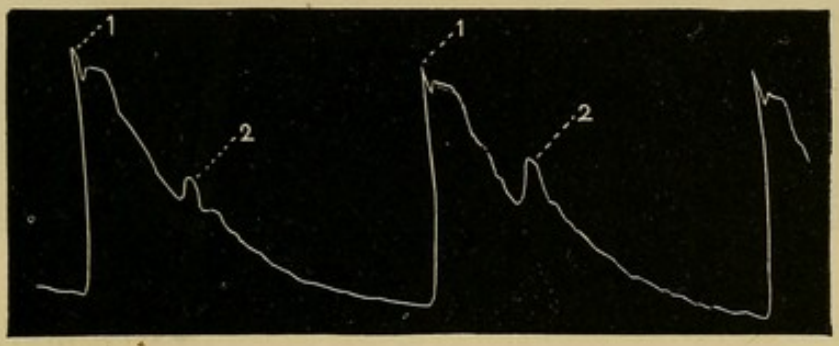

FIG. 49.—*Pulsus bigeminus*

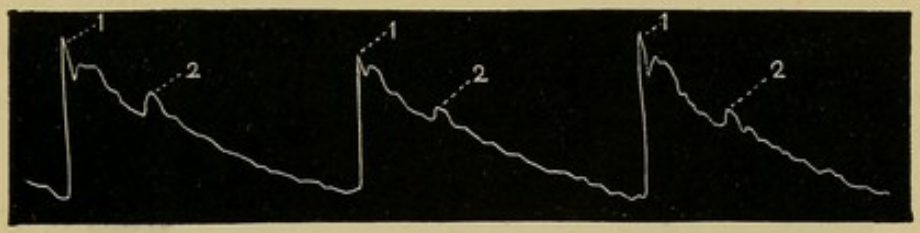

FIG. 50.—*Pulsus bigeminus.*

FIG. 51.—*Pulsus trigeminus*

Irregularity in volume.—Irregularities in volume (see fig. 52) depend upon the fact that unequal quantities of blood are discharged into the arterial system at different contractions of the left ventricle. The condition is usually associated with irregularity in time, for when the time between the ventricular contraction varies, the amount of

blood which the left ventricle has to discharge will be apt to vary too.

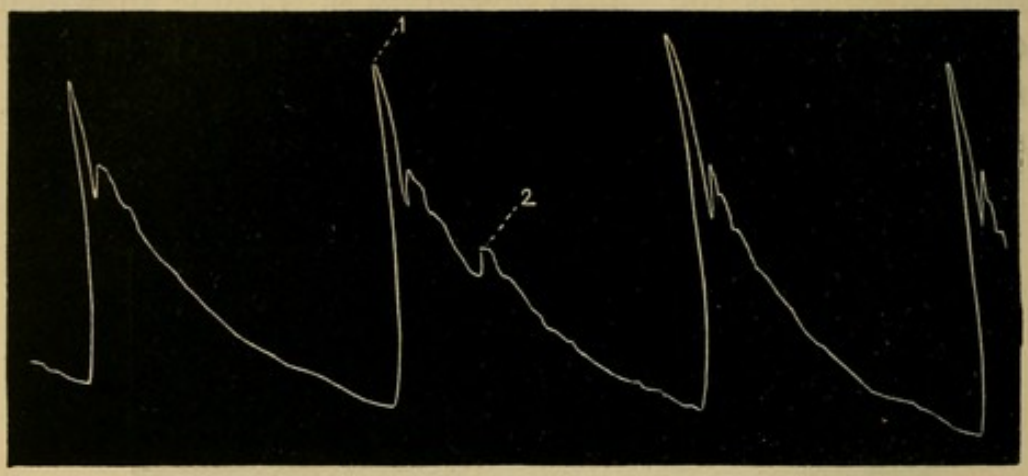

FIG. 52.

Differences in the sphygmographic characters of individual pulse waves depend upon differences in arterial tension, which in their turn may be due either to—

1. Different quantities of blood being propelled during successive contractions of the left ventricle into the arterial system, the causes of which condition I have already considered.

FIG 53.—Pressure, 3¾ oz.

FIG. 53.—*Uneven Respiratory Line.*—J. R., æt. 31, admitted to Newcastle Infirmary 26th December 1878, under Dr Byrom Bramwell, suffering from acute bronchitis.

2. The effects of respiration. During tranquil respiration the *respiratory* or *base line* is, as I have previously stated, a straight line. During deep inspiration and expiration, even in health, and in many pathological conditions in which the respiratory movements are profoundly affected, the 'base line' becomes very uneven (see figs. 53 and 54), and the sphygmographic character of successive pulse waves is

different. The frequency of the pulse and the arterial tension are increased by expiration, lowered by inspiration.[1]

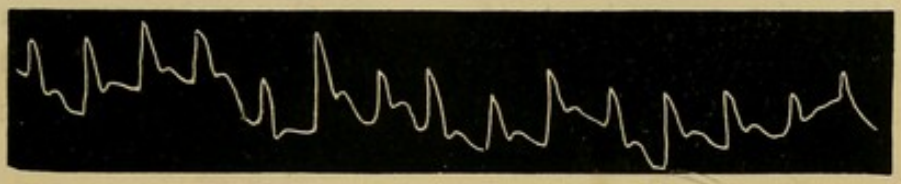

FIG. 54.—*Uneven Respiratory line (from a Case of Spasmodic Asthma).* The tracing was taken during the paroxysm

In extreme cases the pulse-wave may be entirely absent during inspiration. This is the *pulsus paradoxicus* of Kussmaul. The most striking examples of this condition are seen where fibrous adhesions pass between the thoracic parietes and the roots of the aorta and great vessels. During a full inspiration these fibrous bands are stretched, the vessels are constricted, and the pulse-wave is unable to reach the wrist.

The pulsus paradoxicus has also been noted in cases of pericarditis without constricting adhesions; also in cases of stenosis of the air-passages.

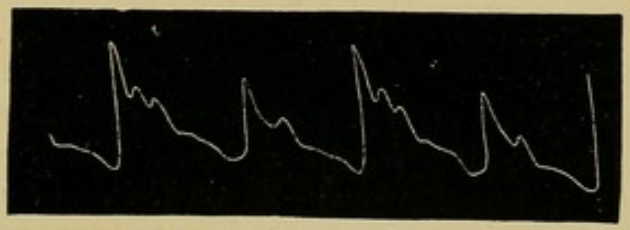

FIG. 55 —Pressure 4¼ oz.

FIG. 55.—*General Atheroma.*—J. D., æt. 60, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 21st October 1878. The radials were very tortuous and rigid; there was no valvular lesion. The tidal wave only occurs every alternate beat.

3. Differences in the resistance which the blood stream meets with in the peripheral vessels. This cause is probably rare, but it is difficult to account for such a pulse tracing as the following (see fig. 55), unless we suppose either a

[1] At the beginning of inspiration the arterial pressure falls; it soon, however, begins to rise, but does not reach the maximum until some time after expiration has begun. The fall continues during the remainder of expiration, and passes on into the succeeding inspiration (Foster, page 344). In speaking of the cause of the respiratory undulations, Foster says, 'We may conclude then, that the respiratory undulations of blood-pressure are of complex origin, being partly the mechanical results of the thoracic movements, possibly also produced by the alternate expansion and collapse of the pulmonary alveoli, but probably, in addition, brought about by a rhythmical variation of the vascular peripheral resistance, the result of a rhythmical activity of the vaso-motor centre.'

rhythmical difference in the strength of the successive cardiac contractions, or a rhythmical difference in the peripheral resistance; in both cases the primary cause is evidently nervous, and I see no reason why a rhythmical alteration in the peripheral resistance due to vaso-motor causes might not occur.

The clinical significance of inequalities of rhythm depends entirely upon the cause of the condition. Occurring *per se*, without any associated signs and symptoms of disease, the condition is of no practical importance. When there are other signs and symptoms the prognosis depends entirely upon their cause.

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

1. Functional derangements of the heart, such as are produced by hysterical conditions, venereal excess, gout, tobacco, tea, etc.

Physiologists have shown that when the excised heart is fed with rabbit's serum its action is apt to become intermittent. This intermittence is possibly due to the chemical action of the serum. 'Various chemical substances in the blood (natural or morbid),' says Michael Foster, 'may thus affect the heart's beat, by acting on its muscular fibres, its reflex or automatic ganglia, or its intrinsic inhibitory apparatus.'[1]

2. Mitral lesions, both stenosis and regurgitation, especially after compensation has failed. (See figs. 56, 57, 58 and 59.)

FIG. 56 —Pressure 3¾ oz.

FIG. 56.—*Irregularity of the Pulse.*—W. M., æt. 50, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 30th November 1878, suffering from the usual symptoms of mitral disease. The heart's action was extremely irregular. The left ventricle much hypertrophied. There was no rheumatic history. The symptoms were of two months' duration.

[1] *A Text-Book of Physiology*, p. 178.

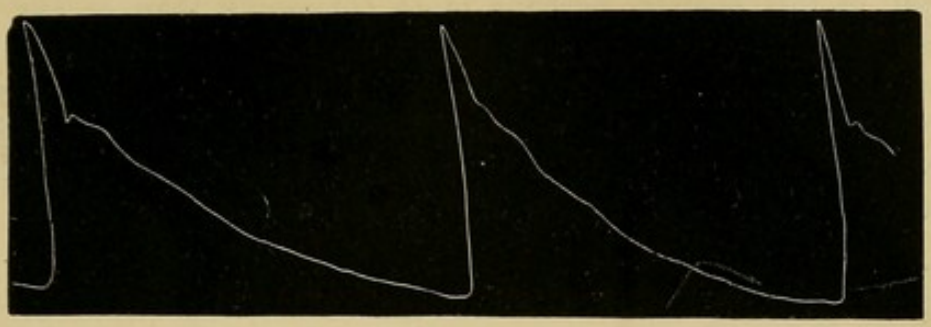

FIG. 57.

FIGS. 56 and 57 were two consecutive tracings taken on 10th December, after the patient had improved under digitalis. The intermittent action of the heart is well shown in Fig. 57.

FIG. 58.—Pressure, 4 oz.

FIG. 58.—*Mitral Regurgitation.*—S. B., æt. 58, admitted to the Newcastle Infirmary under Dr Byrom Bramwell, 25th February 1878, suffering from cardiac dropsy. There was a well-marked mitral systolic murmur, which disappeared under treatment. The heart was considerally enlarged (hypertrophied and dilated).

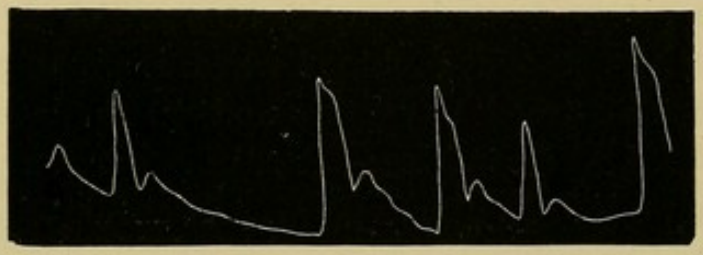

FIG. 59.—Pressure, 3 oz.

FIG 59.—*Irregular and Intermittent Pulse.*—O. M., æt. 40, admitted to the Newcastle Infirmary under Dr Byrom Bramwell, suffering from cardiac dropsy. The heart was very much enlarged; apex beat 4½ inches below and 3 inches outside left nipple; systolic mitral murmur.

There has been a good deal of debate with regard to the rhythm of the pulse in mitral stenosis. The majority of observers are, however, agreed—and with their opinion I entirely concur—that the pulse is irregular. Dr Sansom—than whom no better authority could be quoted—says, 'We may conclude, therefore, that a pulse tracing which shows irregularity in the diastolic periods, sometimes missed pulsations, and, as described by Dr B. Foster, the occasional appearance of a small abortive pulsation in the line of descent, is very strong evidence of mitral stenosis.'—P. 269. Dr Mahomed, who was, I believe, the first to describe this peculiar rhythm as characteristic of mitral stenosis, has since somewhat modified his views. He says, 'Although it is very commonly

present in this disease, perhaps more commonly than in any other valvular lesion, nevertheless it is not so much a sign of stenosis of the valves as of dilatation of the ventricle. It is true, that in the typical cases of mitral stenosis the ventricle is not dilated, but I am unable to say whether this irregularity only occurs in cases in which dilatation exists.'—*Guy's Hospital Reports*, 1879, p. 401.

3. Degenerations of the cardiac walls. In cases of fatty and fibroid degeneration, and, in fact, in all conditions in which there is much degeneration of the left ventricle, the heart's action may be very irregular.

4. Some affections of the central nervous system, such as meningitis, in which condition, alterations in the condition of the intra-cranial circulation, such as result from sudden changes in position, etc., may cause alterations in the rhythm as well as in the rate of the heart's contractions.

THE VOLUME OF THE PULSE.

The *volume* of the pulse, which depends upon—(1) the size of the artery (radial, carotid, etc.) which is being examined; (2) the amount of blood which is propelled into the artery at each ventricular systole; and (3) the tonicity of the arterial wall, *i.e.*, the condition of the vaso-motor apparatus,—may be appreciated by the finger, but is accurately measured by means of the sphygmograph.

In *health* the volume of the pulse varies from time to time, and is of course different in different individuals. There are also many modifications in disease.

A *large* pulse, *i.e.*, a pulse of large volume, is seen in the following conditions:—

1. In many cases of fever during the earlier[1] periods of the attack, when the heart is acting powerfully, and propelling a large quantity of blood into vessels, the tonicity of which is already somewhat relaxed.[2]

---

[1] During the stage of rigor the pulse is small.

[2] Towards the termination of cases of fever, *i.e.*, after the condition has continued for some time, the pulse vessels become still more relaxed, and the pulse becomes small. It is often under these circumstances dicrotic.

2. In atheroma, when the elasticity of the arterial walls is impaired and the vessels are dilated.

3. In some cases of hypertrophy, and in some cases of simple cardiac excitement. (See figs. 60 and 61.)

4. In aortic regurgitation the systolic portion of the tracing is of large volume, but the diastolic extremely small.

FIG. 60.—Pressure, 2½ oz.

FIG. 60.—*Alterations in the Pulse-tracings as the result of Cardiac Excitement.*—A M., æt. 48, admitted to the Newcastle Infirmary under Dr Byrom Bramwell, suffering from obscure spinal symptoms. The heart became excited, and the tracing shown in Fig. 60 was taken, immediately after that shown in Fig. 14, the instrument in the meantime remaining *in situ*. The spring pressure was the same in each case.

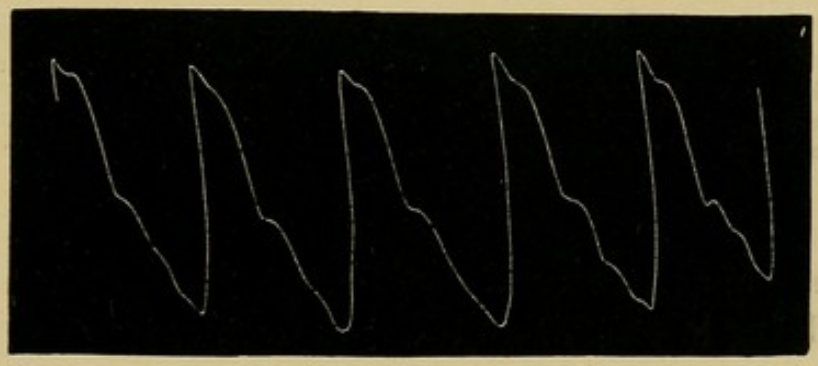

FIG. 61.—Pressure, 3¾ oz.

FIG. 61.—*Hypertrophy of Left Ventricle.*—A T, æt. 54, admitted to Newcastle Infirmary under Dr Byrom Bramwell, suffering from cirrhotic kidney.

A *small* pulse is met with:

1. In those conditions in which the amount of blood discharged by the left ventricle is below the normal amount. Under this head are comprised:—

(*a*) Cases (such as inanition) in which the total amount of blood in the body is reduced in quantity. (See fig. 62.)

(*b*) Cases of mitral disease, both stenosis and regurgitation. (See fig. 63.) In the former (mitral stenosis) the left ventricle does not receive the usual (normal) amount of blood from the auricle; in the latter case, some of the blood which ought to be discharged into the aorta flows back into the auricle through the incompetent valve.

FIG. 62.—Pressure, 2 oz.

FIG. 62.—*Small Weak Pulse.*—J. M., æt. 18, a soldier, admitted to the Newcastle Infirmary under Dr Byrom Bramwell, 23d January 1879, suffering from abscess of the liver and pericarditis The heart was displaced upwards and to the left.

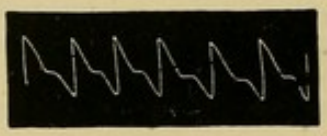

FIG. 63.—Pressure, 3 oz.

FIG. 63.—*Mitral Regurgitation.*—M. A. C., æt. 16, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 24th January 1878, suffering from cough and shortness of breath, dating from an attack of rheumatic fever two months previously. Heart's action very rapid (120-130). The first sound appeared to be reduplicated; a systolic murmur at the apex, audible when the heart became slower.

(*c*) Cases of aortic stenosis (see fig. 64); the size of the pulse being in proportion to the narrowing of the orifice.

FIG. 64.—Pressure, 1½ oz.

FIG 64.—*Aortic Stenosis.*—J. B., æt. 51, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 29th November 1878, suffering from anæmia and dropsy. There was a well-marked aortic systolic murmur; the left ventricle was not hypertrophied. The pulse-tracing seems to show that the murmur was organic.

(*d*) Conditions of cardiac debility, both temporary and permanent. (See fig. 62.) Conditions of collapse are good examples of the former; the pulse in collapse being small and thready, while fatty and fibroid degeneration of the left ventricle, with dilatation, are types of the latter.

2 In those cases in which the vessels are unduly contracted, as in peritonitis, the cirrhotic form of kidney disease, and the cold stage of fevers, *i.e.*, during the rigor.

THE COMPRESSIBILITY OR STRENGTH OF THE PULSE is a point of great practical importance, for in many cases it indicates the condition of the vaso-motor nerve apparatus, and hence of the general tone of the system.

The strength of the pulse is measured by the finger or by means of the sphygmograph, the amount of pressure required to obliterate the pulse-wave being (provided the arterial walls are healthy) the true indication of the pulse-strength.[1]

Dr Mahomed guages high tension in the following manner:—'A line must be drawn from the apex of the up-stroke to the bottom of the notch preceding the dicrotic wave (fig. 65, A B). No part of the tracing should rise above this line;

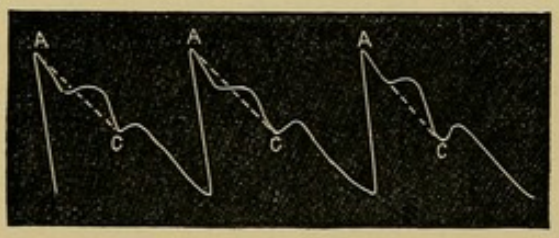

FIG. 65. (*After Mahomed.*)

FIG. 65.—*Mahomed's method of guaging high tension.* The tidal wave in A rises above the dotted line drawn from the apex A, to the aortic notch C.

if it does, then the pulse is one of high pressure. The height of this notch is another good gauge of pressure; the higher it is from the base line of the tracing the higher is the pressure, the nearer it approaches the line the lower is the pressure.

[1] When the arterial walls are inelastic and rigid, as in cases of atheroma, the amount of pressure required to obliterate the pulse is not a true criterion of its strength, for a certain (often a considerable) amount of pressure must be applied before any influence is exerted upon the blood wave itself.

Lastly, the duration of systole compared to that of diastole may perhaps be reckoned an important sign.' Dr Mahomed believes 'that there is a normal length of systole for a pulse of a given frequency, and that the length of the systole is increased if the arterial pressure is increased.'[1]

I am in the habit of considering a pulse of high tension as synonymous with a strong pulse, and *vice versâ* a pulse of low tension with a weak pulse.[2]

In vigorous health the strength or tension of the pulse is considerable, but there are great differences, even in persons who present all the external appearances of good health; while alterations in the tension of the pulse are common in disease.

A pulse of *high tension* may be either large or small, the pulse of chronic Bright's disease (especially the cirrhotic form), in which the heart is notably hypertrophied, is illustrative of the former; the pulse of peritonitis is an excellent example of the latter.

A *weak* pulse, or pulse of *low tension*, is also very common in disease, and is also of great importance. A pulse of low tension is usually associated with feeble action of the heart and a relaxed condition of the blood vessels. It may be either small or large; the former—a small pulse of low tension—is seen in mitral lesions and towards the terminal period of fever; the latter—a large pulse of low tension—is also seen in some cases of fever (as for instance in certain stages of rheumatic fever), and in many persons who enjoy apparent good health, but in whom the vaso-motor tone is below par.

As a general rule, a slow pulse is a pulse of higher tension than a quick pulse, and *vice versâ*, but such a relationship is by no means constant or necessary.

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[1] *Guy's Hospital Reports*, 1879, p. 371.

[2] I, therefore, differ from Dr Mahomed, who considers that hardness or incompressibility is the least constant character of the high pressure pulse. 'It is not unfrequent,' he says, 'to find *overfull* vessels associated with a weak or failing heart, the pulse is then often small and feeble, it is very easily compressed, and is described as a small weak pulse, which is thought usually to require stimulants; the reverse, however, is the case; bleeding or purging will be well borne by such patients, and the result will be most satisfactory.'

The clinical significance of the tension of the pulse.—The tension of the pulse is of great importance, both for diagnosis, prognosis, and treatment. Dr Mahomed, for example, has shown that in many cases of chronic Bright's disease there is in all probability a stage of the disease in which the urine is healthy (free from albumen, casts, etc.), but in which there is persistent high arterial tension, and further, that by reducing this condition of high tension the subsequent structural affection of the kidney may be prevented. So, too, a *hard* pulse associated with cardiac pain (angina pectoris) indicates a serious condition, and urgently calls for treatment.[1]

On the other hand, a pulse of low tension is no less important both for prognosis and treatment. Persons whose vaso-motor tone is below par, *i.e.*, who have weak pulses, bear severe (especially acute) disease badly; while a weak, and especially a dicrotic pulse in a case of fever, indicates the free use of stimulants.

THE SPHYGMOGRAPHIC CHARACTERS OF THE INDIVIDUAL PULSE-WAVES.—Most of these points, which are of considerable practical importance, such as dicrotism, have been already fully considered.

The relative duration of those portions of the tracing which correspond to the ventricular systole and diastole.

Speaking generally, it may be said that in the normal condition the ventricular systole occupies about $\frac{4}{10}$ of the entire cardiac revolution.[2] This relationship is altered in certain

---

[1] Dr Mahomed, and also Dr Sansom, think that cases of angina pectoris may be divided into two great classes by means of the pulse, and that 'these two classes have a totally different pathology, prognosis, and treatment.' If the pulse be normal, the case is probably one of local arteritis, due to local strain, syphilis, or some such condition; if, on the other hand, the pulse is hard and long, the case is one of general arterial disease, with an extensively diseased aorta, with hypertrophy of the heart, and probably thickening of the smaller arteries, in short, Dr Mahomed would consider such a case as a case of chronic Bright's disease with aortitis deformans.

[2] The measurement given in the text is not strictly accurate. Dr Gibson, as the result of careful cardiographic observations, gives the 'average absolute duration of each phase of the entire cardiac cycle,' as follows :—

Auricular Systole.	Ventricular Systole.	Ventricular Diastole.	Entire Cycle.
·112 sec.	·368 sec.	·578 sec.	1·057 sec.

—*Journal of Anatomy and Physiology*, vol. xiv. p. 237.

cases of disease. When the heart is acting very rapidly, the diastole is relatively more reduced than the systole, and the proportional duration of the systole to the diastolic portion of the tracing is increased. Increased temperature of the blood, has, as Dr Paul Chapman[1] has shown, a distinct influence in shortening the duration of the ventricular systole; and the same authority has observed, on coming out of a Turkish bath, 'that although the pulse-frequency may actually increase, the systole lengthens as the patient cools.' So, too, in aortic regurgitation, the systolic portion of the tracing is usually longer than the diastolic.

The relative condition of the vessel (as regards its fulness) during systole and diastole.

In some cases the systolic portion of the tracing is relatively very much larger than the diastolic. A good example of this condition is seen in aortic regurgitation (see fig. 66), in which the artery is fully distended during systole, but comparatively very empty during diastole.

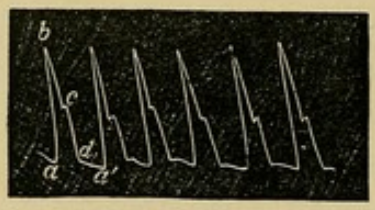

FIG 66.—Pressure, 2½ oz.

FIG. 66.—*Aortic Regurgitation* - Case: G. A., æt 56, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 21st February 1878, suffering from shortness of breath and swelling of feet. Had been ill for three months. The face was pale and anxious, lips slightly dusky. Double aortic murmur; heart considerably hypertrophied; apex between 6th and 7th ribs, 3 inches below and 2 inches outside left nipple. Considerable hypertrophy and engorgement of right heart. Died 5th March 1878. Aorta very atheromatous; aortic valves very incompetent; segments shrunken, turned in towards the ventricle; coronary arteries much obstructed; cardiac walls fatty; left ventricle dilated; pericardium adherent. The arteries were practically empty during the ventricular diastole. *a-b*=percussion stroke; *b*=apex; *c*=tidal wave; *d* indicates the position of the aortic wave, which is absent in this tracing.

An empty condition of the arterial system during the ventricular diastole is often associated with a failing heart, and is a serious indication (see figs. 67 and 68).

When the pulse is *fully dicrotic*, the artery at the point of observation is *apparently*[2] as empty at the end as it is at the

[1] *British Medical Journal*, August 19, 1882, p. 300. [2] See foot note on page 26.

beginning of the ventricular systole, and is apparently more empty at the end of the ventricular systole than it is during the ventricular diastole. Again, in hyperdicrotism the artery at the point of observation is *apparently* still more empty at the end of the ventricular systole.

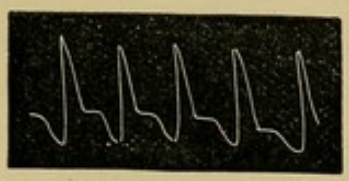

FIG. 67.—Pressure, 2 oz.

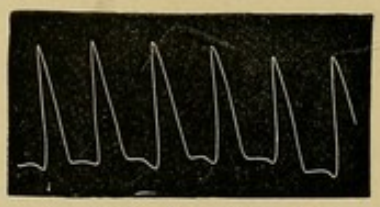

FIG. 68 —Pressure, 2⅛ oz.

FIG. 67 —*Weak Pulse.*—R. R., æt. 17, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 21st February 1878, suffering from idiopathic anæmia. Died 12th April. There was a systolic (anæmic) mitral murmur. The pulse-tracing resembles that of mitral regurgitation (see Fig. 14). Tracing made 23d February.

FIG. 68.—*Progressive Pernicious Anæmia.*—Same patient (see Fig. 15). Tracing taken 19th March. The artery is almost empty during diastole.

In other cases on the contrary (in chronic Bright's disease, for example), the diastolic portion of the tracing is unduly sustained. A *persistent* pulse is usually a pulse of high tension, and *vice versâ* a pulse which rapidly falls away under the finger is a pulse of low tension.

THE CONDITION OF THE ARTERIAL COATS.

The condition of the arterial coats with regard to the presence or absence of atheroma, is a point of great practical importance, for atheroma of the superficial arteries—the radial, temporal, etc.,—is almost invariably associated with similar disease of the aorta, and very often with disease of the cerebral vessels. The presence or absence of atheroma may be of diagnostic importance, as for example, in a case in which the diagnosis lay between an aneurism and a solid intrathoracic tumour.

Atheroma in the superficial vessels is indicated by:—

(1) Rigidity of the arterial coats; the vessel generally 'stands out like a cord,' is markedly visible and tortuous, while its coats feel thick under the finger. To determine whether the arterial coats are actually thickened or not, the artery should be firmly compressed and emptied by one

finger, while the other finger searches for the vessel below the compressed point. When the vessel can be distinctly felt, even though empty of its contents, its walls are obviously thickened and diseased. This is an important point, for all arteries which are firm and hard, and which stand out like cords, are not thickened.

(2) The presence of a well marked tidal wave, the tension of the pulse being high (*i.e.*, the pulse with difficulty obliterated) but a low pressure being usually required for the production of the best tracing (*i.e.*, the first ventricular wave being very easily extinguished).

THE COMPARISON OF THE TWO RADIAL PULSES is useful in the diagnosis of some cases of aneurism and intra-thoracic tumour. The two pulses must be compared as regards; (*a*) their synchronism as to time; (*b*) the character of their respective pulse waves.

Differences in time between the two pulses are most easily appreciated by the fingers. In health the two radial pulses are of course synchronous, but in some conditions of disease, as for example, where the sac of an aneurism is situated in the course of the circulation, the pulse wave is retarded, and is consequently delayed at one wrist.

Differences in the character of the pulse waves in the two wrists may also be detected by the finger, but are much more accurately observed by means of the sphygmograph. The precautions which should be taken in comparing the two pulses have been already described. (See page 15.)

Differences in the two pulses may be due to:—

1. Irregular distribution, such as high division of the radial artery. In such cases there is a notable difference in the size of the vessel in the two wrists, or indeed it may apparently be altogether absent on one side. The condition is easily recognised by observing the condition of the brachial arteries on the two sides, and by feeling for the position of the abnormal vessel.

2. The presence of an aneurismal sac in the course of the circulation on one side. The alterations produced by the

passage of the blood wave through a globular elastic aneurismal sac, consist in retardation of the pulse-wave and flattening of its curves. In well marked cases the up-stroke is sloping, the apex rounded, and the secondary curves entirely obliterated. (See figs. 69 and 70, which represent the pulse tracings on the two sides, from a case in which a large aneurism involved the axillary artery on the left side of the body.)

FIG. 69.—Pressure, 3 oz.

FIG 69.—*Aneurism of Left Axillary Artery* (*left radial tracing*).—L. G., æt 63, admitted to the Newcastle Infirmary under Dr Byrom Bramwell, 7th March 1878, with a large aneurism of the left axillary artery The apex is rounded; all the curves are obliterated.

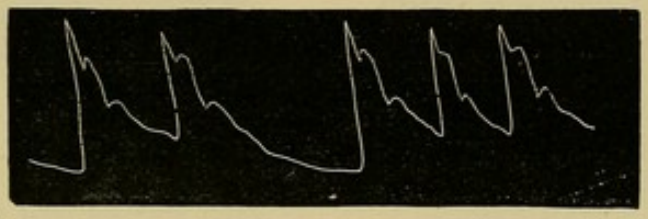

FIG. 70.—Pressure, 3 oz.

FIG 70.—*Aneurism of Left Axillary Artery* (*right radial*) —Right radial tracing for the same patient. The pulse is intermittent, but all the curves are well marked.

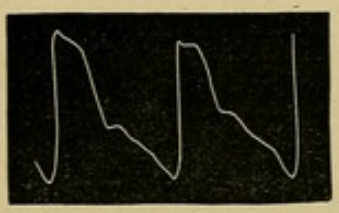

FIG. 71.—(Right radial.) Pressure, ½ oz.

FIG. 72.—(Left radial). Pressure, ¾ oz.

FIGS. 71 and 72.—*Aneurism of Ascending Portion of Aortic Arch* —J. D , æt. 50, admitted to Newcastle Infirmary under Dr Byrom Bramwell, suffering from a large aneurism of the ascending thoracic aorta and atheroma. There is no important difference between the two pulses.

Differences in the pulse tracings from the two wrists are not of course observed in all aneurisms. When the aneurism involves the aortic arch below the origin of the innominate the pulse-wave in the two wrists is the same, though the sphygmographic tracing on each side may be modified [1] (each

[1] The alteration in these cases is seldom so great as in aneurism more peripherally seated (aneurisms of the innominate or subclavian for example).

pulse-wave being affected, *quoad* its curves, in alike degree). Figs. 71 and 72 illustrate this point.

Again, it may so happen that an aneurism is situated on the vessel of each side, or that an aneurism is so filled up with clot that the pulse-wave is very little if at all affected in its passage through it. Such was the fact in a remarkable case of multiple-aneurism which I have recorded in the *Edinburgh Medical Journal* for June 1878, p. 1076. The pulse-tracings from the two radials were in that case almost identically the same. (See figs. 73 and 74.)

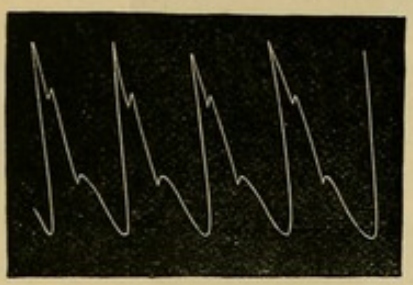

FIG. 73.—(Right radial.) Pressure, 3 oz

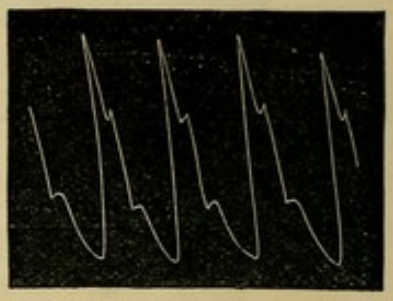

FIG. 74.—(Left radial). Pressure, 3 oz.

FIGS. 73 and 74 —*Case of Multiple-Aneurisms.*—M. F., 64, admitted to the Newcastle Infirmary under Dr Byrom Bramwell, 18th February 1878, suffering from symptoms of intra-thoracic pressure. Died 10th March. *Post-mortem* showed dilatation and small aneurism of aortic arch. Fusiform aneurisms of the innominate, left common carotid arteries, and left subclavian, just above their origins. The aneurisms were filled with firm clots, through which a straight narrow channel for the blood remained.

When the aneurismal sac involves the transverse portion of the aortic arch between the innominate and the left subclavian (the circulation through the innominate being interfered with), the right radial pulse is normal, but the left may be modified.

When the aneurism involves the innominate, the right subclavian, or right axillary artery, the right radial pulse presents the aneurismal characters, while the left is normal. *Vice versâ*, when the left subclavian, or left axillary artery is affected, the left radial pulse will be modified, but the right normal.

The alteration in the two radial pulses may be of considerable diagnostic importance. Figs. 75 and 76 represent, for instance, the right and left pulse-tracings for a patient who was admitted to the Newcastle-on-Tyne Infirmary, under

my care, suffering from dyspepsia. There was no complaint of any thoracic trouble. On taking a tracing of the left radial (I was at that time working at the sphygmograph, and taking tracings of every case admitted to hospital) I was, of course, at once struck with its aneurismal character, and on careful physical examination found decided dulness, faint pulsation, and marked accentuation of the cardiac sounds over the chest at a point corresponding to the origin of the left subclavian artery.

FIG. 75.—(Left radial.) Pressure, 3 oz.

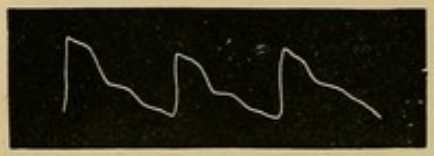

FIG 76.—(Right radial.) Pressure 3½ oz.

FIGS. 75 and 76.—*Aneurism of Left Subclavian.*—J. M., æt. 50, admitted to Newcastle Infirmary under Dr Byrom Bramwell, 5th September 1878; all the waves in the left tracing are obliterated.

FIG. 77.—(Right radial.) Pressure, 2½ oz.

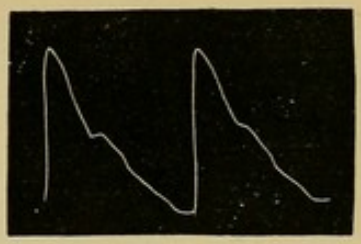

FIG. 78.—(Left radial) Pressure, 2½ oz.

FIGS 77 and 78.—Difference in the radial pulses, the result of pressure by an intra-thoracic tumour on the innominate artery. J. F., æt 50, admitted to the Newcastle Infirmary under Dr Byrom Bramwell, 24th January 1878

3. The pressure of a tumour (solid or aneurismal) on the vessels of one side (on the innominate, or left subclavian for example). (See figs. 77 and 78.)

4. Differences in the calibre of the vessels on the two sides (innominate and left subclavian) such as are produced by obliquity of origin due to disease (aneurismal dilatation of the aortic arch, etc.), or to the presence of an atheromatous patch at the mouth of the vessel or in its course.

5. Local disease in one radial artery.

Asynchronism of the radial pulse with the cardiac contractions has been already alluded to. (See page 39.)

INDEX.

THE DISEASES OF THE SPINAL CORD.

BY

BYROM BRAMWELL, M.D., F.R.C.P. EDIN.

OPINIONS OF THE PRESS.

From THE BRITISH MEDICAL JOURNAL, May 20, 1882.

'The work before us, just published, brings before its readers a complete *resumé* of all the recent investigations on the subject both at home and abroad. . . . It professes to place before the student and the profession all that is known of the subject it undertakes to elucidate. Considering the enormous difficulties to be encountered in the task, this has been carried out in a masterly manner, and evidently by one who has thoroughly and practically acquainted himself with all the details of the inquiry. . . . Next is discussed the pathological condition of the cord, and this is done in an equally clear and convincing manner. Dr Bramwell, while continuing to employ the diagrammatic method to explain his views, supplements it by what constitutes one of the chief attractive features of his book. This consists of a series of exquisitely artistic chromo-lithographs of original sections of the cord in health and disease, prepared and drawn by himself. We have never seen anything more beautifully displayed, and we congratulate the author on the successful manner in which they have been executed. . . . The work, as a whole, we can strongly recommend to our readers. It is evidently the outcome of experience, labour, and thought. Although simple, clear, and concise, it brings before the profession a complete and exhaustive statement of one of the most difficult and complex problems in the whole range of medicine.'

From the CENTRALBLATT F. NERVENHEILKUNDE, PSYCHIATRIE, UND GERICHTLICHE PSYCHOPATHOLOGIE, Leipzig, June 1, 1882.

'The first impression which one receives on opening this book is that it is rather an atlas than a description of the diseases of the spinal cord, so numerous are the illustrations, both systematic and microscopical, which meet the eye on all sides. In this respect the work, which is really a text-book, and intended for students and practitioners of medicine, stands alone of its kind, and we cannot but recognise the great advantage in the mode of representation which carries to the brain a number of clear impressions through the eye, as compared with a dry, bare, verbal description which obliges the reader to call in every moment the help of his imagination in order to sketch a

picture of the subject under discussion. It is very much to be wished that many may follow Bramwell's example in this respect, for it would be a great advantage to the student. . . . The pathological anatomy of these conditions, as also the symptoms which immediately result, are here discussed, in the same concise but most complete manner, in forty pages, containing fifty illustrations, which are for the most part chromo-lithographs. The most weighty chapter in the book is, nevertheless, the third, in which the author describes his method of investigating individual cases. . . . Space does not allow us to enter into a short description, especially as the author has himself studied a praiseworthy and pregnant brevity which would render it very difficult to give an abstract of the chapter. It will suffice to observe that whoever has made himself fully acquainted with it will very seldom meet with difficulty in the diagnosis of a concrete case of disease of the spinal cord. . . . Our whole opinion of Bramwell's work is, that although it contains no new discoveries of a pathological, diagnostic, or therapeutical nature, it is, on account of the peculiar manner in which the subject is treated, an original work. We are not acquainted with any work in the literature of spinal cord disease in which the correlation of normal and pathological anatomy and physiology, as well as the clinical pictures which depend thereon, is carried through in such a practical and complete manner. We have not to do, here, with dry bones, but with real flesh and blood. . . . The book deserves, more than many others, to be made universally accessible to the German medical public by means of a good translation.' *—(Signed) Julius Althaus (London).

From THE MEDICAL NEWS, June 9, 1882.

'We shall be guilty of no extravagance in describing this work as one of the most important books recently published. . . . We cannot too strongly recommend this work to students particularly. The points of an unsettled nature dwelt on by its author are so treated that no dogmatic statements are put forth as truths to be accepted, and, on the other hand, all needful information on open subjects is afforded. In publishing the volume Dr Bramwell has enhanced his own reputation as a scientific physician, and has undoubtedly, also, rendered the study of a most difficult subject henceforward both deeply interesting and much more simple than hitherto.'

From the JOURNAL OF MENTAL SCIENCE, July 1882.

'His chromo-lithographs are the best we have seen in any book on the diseases of the nervous system. . . . It is not a slavish compilation, in which all kinds of facts, reliable and unreliable, are heaped together without any regard to their value. On the contrary, there are constant evidences that Dr Bramwell's reading has been tested by experience at the bedside and in the pathological room. . . . We have read the book through with great care, parts of it twice over, and we can recommend it in every respect. To the beginner anxious to know something of the subject it is a trustworthy and complete text-book, and the more experienced physician will find in it much original work admirably recorded.'

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* Before this review appeared, Messrs Tœplitz & Deuticke of Vienna had, at the suggestion of Dr N. Weiss, requested the author's permission to bring out a German edition, which appeared in October 1882.* A French edition has also been recently issued.

From THE GLASGOW MEDICAL JOURNAL, July 1882.

'One special feature of the work is the wealth of illustration in which it abounds, and which certainly adds very greatly to its value. . . . To students wishing to begin the study of diseases of the spinal cord, or to medical practitioners who feel that their knowledge is not quite up to date, we can heartily recommend the book as a careful and exhaustive epitome of all that is known about this subject.'

From BRAIN: A Manual of Neurology, July 1882.

'The work before us is a gratifying evidence of the increasing attention paid to the study of nervous diseases, and an evidence, let us hope, of the increasing demand on the part of students and practitioners for the most recent information respecting them. . . . The third chapter is the most valuable in the book to the clinical student. After giving an admirable outline of the method of case-taking, the author proceeds to give specific instructions regarding the precautions which must be applied in order to give scientific accuracy to the clinical investigation. . . . This chapter is brought to a close by a general sketch of the diagnosis, prognosis, and treatment, which shows that the author is as familiar with his subject at the bedside as he is in the pathological room and physiological laboratory. . . . These illustrations are indeed works of art. The representations of sections stained by osmic acid are quite unique of their kind, and no one who has not seen the original sections, as the present writer has had an opportunity of doing, can understand the extreme faithfulness with which they represent the originals. This book must greatly enhance the already well-earned reputation of Dr Bramwell as a neurologist, and it cannot fail to prove an acceptable and safe guide to all those who wish to obtain a scientific knowledge of the diseases of which it treats.'—(Signed) James Ross.

From the LIVERPOOL MEDICO-CHIRURGICAL JOURNAL, July 1882.

'This is a most useful book. It contains, in a very condensed but nevertheless clear and well arranged form, a summary of our present knowledge of the functions and diseases of the spinal cord. A great deal of our knowledge in this department of medicine is, as every one knows, of very recent date, and is not easily accessible to the English reader, except in translations of bulky foreign works. Now Dr Bramwell in his book supplies the student with this knowledge in its most recent and completest form, and yet so condensed in bulk, and so clear and intelligible in form and expression, that nothing better could be desired. . . . The section on "the Diagnosis" at the close of this portion of the work is perhaps the most original in the book. . . . The last portion of the book is a systematic description of the various diseases of the cord and its membranes, not differing materially from what may be found in some of the most recent English text-books on medicine. Its superiority to them consists in the shortness and clearness of the descriptions, the large number of woodcut illustrations of the various diseases, and a series of exceedingly useful tables of differential diagnosis. . . . We have very great satisfaction in heartily recommending this book to both the teaching and the learning sections of the profession. The teacher will obtain in the many ingenious diagrams a ready means of making clear to students in the class-room the most difficult points in special pathology; and the medical student and practitioner will find it a much more clear and useful hand-book than the two bulky volumes of *Ziemssen's Cyclopædia* which are devoted to this subject.'

From the MEDICAL PRESS AND CIRCULAR, September 27, 1882.

'This clear, comprehensive, and profusely illustrated work is composed of part of the course of lectures on medicine which is delivered by Dr Byrom Bramwell in the Edinburgh Extra-Academical School. . . . It will therefore be of service to that body of medical practitioners—an ever increasing body in this country—who desire to keep abreast of medical science, as well as to the student, for whose use it is perhaps more especially designed, and we are much mistaken if certain of the drawings which it contains do not pass into the mental portfolios, if we may so say, of many members of the medical profession, to be often referred to by the sick-bed or in the consulting room, for the interpretation of obscure symptoms. . . . So excellent as a whole is Dr Bramwell's work, that it is almost impossible to criticise it without appearing to be captious. It is entitled to a cordial reception as a lucid, concise, and thoughtful text-book of diseases of the spinal cord.'

From the LONDON MEDICAL RECORD, October 15, 1882.

Review signed 'Julius Althaus, M.D.,' very similar to that in the 'Centralblatt F. Nervenheilkunde, Psychiatrie, und Gerichtliche Psychopathologie,'—quoted above.

From the LANCET, November 4, 1882.

'The mode in which the Author thus introduces us to the anatomy, and proceeds in the same way to the physiology and pathology of the spinal cord through the medium of a single segment of it, is philosophical, and tends much to clearness of illustration. . . . Dr Bramwell enters very fully, and with much lucidity of description, upon the important subject of case-taking. . . . A couple of pages are well devoted to the subject of "pain referred to the spinal column," a point which we believe is constantly giving rise to mistakes. The question of the diagnosis in diseases of the spinal cord receives, as it deserves, full attention, and many valuable points are contained in the portion of the work relating to this subject. Some good remarks also occur in reference to prognosis, a subject upon which, unfortunately in the nature of things, there is not very much to be said of a satisfactory character. The same remark may perhaps be applied to the question of treatment. What little there is to be said upon this matter is sensibly put, and exaggerated estimates of the power of therapeutics in chronic disease of the nervous substance are properly avoided. . . . The sketch diagrams, and the concise tables of differential diagnosis, constitute a special and acceptable feature of the work. No doubt the work is not exhaustive as a treatise upon diseases of the spinal cord —indeed, for this would not many volumes be required?—but by a sort of rough analogy, we may liken it to an excellent guide-book amply furnished with charts and tables of ready reference, and sketch maps of the routes by the aid of which the traveller, be he ever so little acquainted with the country, will find his journey immensely facilitated, and will readily learn in what directions he may best concentrate his attention and powers of observation.'

From THE (AMERICAN) JOURNAL OF NERVOUS AND MENTAL DISEASE, October 1882.

'Time was, and that not a decade past, when the practitioner turned to a treatise on general medicine for information regarding the diseases of the spinal cord; later he referred to special treatise on diseases of the nervous system in general; to-day we are offered a comprehensive work of nearly three hundred pages, whose sole object is to explicate the diseases of the spinal cord, including in its scope a full anatomical and physiological *résumé* of the subject.

'The little treatise of Gowers led us up to this larger work, and, so to speak, whetted our appetite for more. This desire has now been most gratefully appeased by Dr Bramwell.

'The book is one of those sumptuous efforts that appeal to the eye preliminary to an appeal to the understanding. Indeed, merely to carelessly turn over the pages, from illustration to illustration, beautifully executed in carmine and bronze colours, is enough to excite the keen interest of the student of diseases of the nervous system to a perusal of the text. How copious the illustrations, may be inferred at once when we say that in a work of 289 pages there are 286 illustrations, of which forty-four are chromo-lithographs, ten are lithographs, and the remainder are excellent cuts of sections of diagrammatic representations. The chromo-lithographs, with two exceptions (figs. 56 and 151, copied from Charcot), are drawn by the author himself, first with the camera lucida and then in lithographic chalk. For this original work he deserves much credit. . . .

'On turning to the text our first favourable impressions are subjected to no disappointments. At every step we find evidence of painstaking diligence, of careful condensation, and scientific accuracy. We recognise the traces of the familiar teachings of Charcot and Erb, "to whose writings," the author gracefully says in his dedication, "I am largely indebted for my knowledge of diseases of the spinal cord." But the author's work shows no signs of being a mere compilation; his material has been well disgested. Chapters I. and II., devoted respectively to the anatomy and physiology of the spinal segment, and to the pathology of the spinal segment, are the most interesting chapters in the book. The diagrams illustrating the pathways of the varieties of physiological activities are particularly good; they display the ingenuity of a teacher who has a ready facility in materialising, so to speak, his ideas on blackboard or paper. . . .

'We close the book with a feeling of respect for Dr Bramwell's honest work. His book will always possess a high order of merit with those who conscientiously desire to understand the diseases of the spinal cord.'

From the NEW YORK MEDICAL JOURNAL, March 10, 1883.

'For the student of neurology, and, as well, for the practitioner who has not yet reached an "expert" eminence from which he can look down upon all text-books, Dr Bramwell's present treatise is, beyond comparison, the best work of its kind which has thus far come to our notice. Beginning with the anatomy and physiology of the cord, the reader is gradually led to foresee the symptomatology of lesions of different tracts, and, if he possess ordinary intelligence, will find himself a tolerable diagnostician even before he reaches the specific description of classified spinal diseases.

'A prominent feature of the author's teaching lies in considering the spinal

cord as a series of segments, each of which, with its anterior and posterior pairs of nerve-roots, may be viewed as "a distinct spinal cord for a definite area of the body." The relations of these segments to each other and the functions of their several parts are described in accordance with the most recent state of our knowledge, and ingeniously demonstrated by means of diagrams, in the use of which Dr Bramwell is peculiarly happy. . . . .

'The most practically valuable chapter is that which describes the clinical examination of patients with spinal disease, fully describing the diagnostic methods of investigating the motor, sensory, and reflex conditions, and the pathognomonic importance of the various collateral disturbance—trophic, gastric, articular etc. The explanation of the mechanism of the vesical reflex and the differential diagnosis of different forms of incontinence or retention of urine merit especial commendation.'