Shower, Needle, and Douche Baths.—The influence of temperature *per se* is greatly modified by the mechanical agency of percussion and vibration, so that when the water is divided into innumerable fine jets which impinge all over the surface of the body with a certain degree of force, or when it is applied in the form of the travelling douche accompanied by a vibratory form of massage, the vasomotor system is much more powerfully affected than by any form of still bathing, even though the water be charged with carbonic acid or saline matter. The therapeutic advantages of this form of bathing are very considerable, for it can be made to furnish all the tonic effects of cold bathing along with an agreeable temperature that may be adapted to individual requirements. A more intelligent appreciation of the curative value of this and of similar forms of tonic bathing now prevails at many health resorts, where in times gone by a course of bathing often simply meant soaking in hot water with the consequent impairment of the vasomotor mechanism. The massage, douche, and needle bath is the form of bathing best of all adapted to counteract the enervating effects of city life with all its attendant worries and anxieties; and were it established in our large centres it would, I am persuaded, prove to be a boon to many sufferers from nervous exhaustion, and would become even more popular and valuable than the Turkish bath.

Carbonic acid and saline baths also belong to the stimulant form of bathing.

CAUSATION OF THE DOUBLE FIRST CARDIAC SOUND.

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By a double first sound I mean that physical sign which is also known as a reduplicated first sound, and which occurs most typically and most commonly in cases of high arterial tension. This double first sound is sometimes wrongly referred to as a *bruit de galop*, for in the former, the component sounds come closely together, and can be distinctly and undoubtedly placed in their true position, namely, ventricular systole, in the cardiac cycle, whereas there is room for much divergence of opinion concerning the timing of the sounds which form the true *bruit de galop*.

My remarks in this paper will be confined to the double first sound alone, and I shall not at present touch upon the *bruit de galop* at all, although I consider that its explanation is allied to that which I shall suggest to be the cause of the double first sound.

THEORIES AS TO ITS CAUSATION.

My attention has been directed to the first sound more with a view to the elucidation of its physical causation than to the pathological conditions of the body generally in which it occurs. This latter part of the subject has been dealt with recently by Kauffmann.¹ As a result of observations on 34 cases, Kauffmann found that the chief ailments in which a double first sound may occur are chronic Bright's disease; cardiac dilatation, especially the left ventricle; weakness from dyspepsia or from a previous acute illness; displacement of the heart from chronic causes; and occasionally bronchitis and emphysema. I have met with the abnormal sign in Bright's disease (acute and chronic), anæmia, chronic rheumatism with anæmia, and debility from various causes.

As a result of my own experience, I am of opinion that the phenomenon may occur in any case in which the cardiac muscle is actually weak (anæmia, cardiac dilatation, etc.), or only weak relatively to increased arterial tension (Bright's disease). Several theories have been from time to time advanced in explanation of the double first sound of the heart. The most important are referred to fully by Sansom,² but not one of these is to my mind satisfactory. They are as follows:

1. Asynchronous closure of the mitral and tricuspid valves, and from asynchronous action of the ventricles (Potain, Barr).
2. Asynchronous tension of the individual cusps of the mitral and tricuspid valves (Guttmann).
3. Resolution of the first sound into its elements of muscle contraction and valve tension (Hayden).
4. The rapidly following sounds first of auricular then of ventricular systole (Geo. Johnson).
5. A shock caused at the presystolic period of the ventricular action by the auricular systole which suddenly, by *contrecoup* from the apex of the ventricle, makes tense the auricular and ventricular valves (Sansom).

There is very little evidence in support of any one of these theories, and it is in consequence difficult to criticise them. I shall not attempt to do so, but will simply bring forward what I consider to be a more probable cause of the double

first sound, and mention physiological and clinical facts in its support.

A SUGGESTED EXPLANATION.

The double first cardiac sound is caused by failure on the part of the musculi papillares to retain under the intra-ventricular blood pressure the contracted and shortened condition which they assume during the ventricular systole of the cardiac cycle. This failure on the part of the papillary muscles allows the curtains of the auriculo-ventricular valves to rebound, and thereby cause a sound of sudden and forcible tension of the valve curtains and the chordæ tendinæ. The abnormal sound forms the second portion of the double first sound, the first portion being due to the causes which produce the normal first cardiac sound. The failure of the musculi papillares may take place in either or both of the ventricles and affect one or both valves.

PHYSIOLOGICAL EVIDENCE.

I shall, in the first place, refer to the part played by the musculi papillares in aiding the expulsion of blood from the ventricles during their systole. Roy and Adami,³ by means of an ingenious apparatus, took simultaneous tracings of the action of the ventricle wall and of the musculi papillares during the cardiac cycle of a dog. As a result of these observations they divide the ventricular systole into five, and the diastole into three, phases.

PHASES OF VENTRICULAR SYSTOLE.

1. Ventricle wall contracts, but the musculi papillares are at rest.
2. Musculi papillares carry out the first rapid part of their contraction; ventricle wall at rest.
3. Shortening of the musculi papillares still continues, but less rapidly than in Phase 2; ventricle wall contracts further, but not so rapidly as in Phase 1.
4. Musculi papillares and ventricle wall remain contracted, but do not undergo further shortening.
5. Musculi papillares expand rapidly whilst the ventricle wall remains contracted.

PHASES OF VENTRICULAR DIASTOLE.

1. Rapid and uniform expansion of ventricle.
2. Slowing or arrest of expansion if the blood supplying the ventricle be not great in quantity, and all of it has been drawn into the ventricle by the elastic expansion of Phase 1.
3. A wave of blood resulting from contraction of veins and auricles reaches the ventricle, and causes or allows the final expansion of the ventricular wall which precedes systole.

These eight phases then show the parts played by the cardiac muscle during ventricular systole and diastole.

The variation which the intraventricular pressure undergoes during the cardiac cycle was also investigated by Roy and Adami. The conclusions they come to may be stated shortly. "The intraventricular pressure curve is made up of two curves.....which do not bear a constant relation to each other."

The first and larger wave is due to the contraction of the ventricles in Phase 1. At first the rise of pressure in this wave is slow owing to the passive stretching of the musculi papillares, but its completion is rapid. Upon this wave is superposed another of shorter duration (2-3), which indicates the maximum intraventricular pressure. This second short wave is due to the contraction of the musculi papillares. The intraventricular pressure then falls rapidly for a time, owing to the escape of blood into the aorta; but by the final combined contraction of the ventricle and musculi papillares it is maintained at a high level until all the blood is expelled from the ventricle into the aorta.

POSITIONS ASSUMED BY THE AURICULO-VENTRICULAR VALVES DURING THE CARDIAC CYCLE.

During Phase 1 of ventricular systole the curtains of the auriculo-ventricular valves are rendered tense and are bulged upwards towards the auricle. "The edges of the valves may even be pushed away from the point of origin of the papillary muscles by the rise of intraventricular pressure. This must be due to passive stretching of the chordæ tendinæ and the musculi papillares." During Phase 2 the valve curtains are pulled down towards the apex of the ventricle by the musculi papillares diminishing the amount of the bulge into the auricular cavity. During Phases 3 and 4 the relative position of the valve curtains to the apex of the heart remains much the same as in Phase 2, and during the papillary relaxation in Phase 5 the curtains retire towards the auricle. During

¹ *Birm. Med. Rev.*, February, 1894.

² *Diagnosis of Disease of the Heart and Thoracic Aorta*, 1892, p. 209, et seq.

³ *Heart Beat and Pulse Wave*, Practitioner, 1890.

Roy and Adami, *loc. cit.*, pp. 90-91.

the phases of ventricular diastole they are more or less apposed to the ventricle wall by the rush of blood through the valve orifices. "The first sound of the heart is undoubtedly coincident with the systole of the ventricles, though possibly the actual commencement of its becoming audible may be slightly behind the actual beginning of the muscular contraction."⁵ The first sound of the heart then probably takes place in Phase 1. The causes of the normal first sound, which are still uncertain, are immaterial as far as the subject under discussion is concerned.

The points in the above physiology of the heart to which I wish to direct special attention are:

1. The contraction of the muscoli papillares is, in a way, independent of the ventricular systole, and takes place after the commencement of the latter.

2. The intraventricular pressure raised by the systole of the ventricle is further heightened by contraction of the papillary muscles.

3. The first sound of the heart is normally produced in Phase 1 of the ventricular systole.

In the heart in which a double first sound is produced Phase 1 (Fig. 1) takes place normally, producing a first

sound. Phase 2 then comes on, the muscoli papillares contracting and drawing down the edge of the valve curtains towards the ventricular cavity (Fig. 2). By the contraction of the papillary muscles the intraventricular blood pressure is increased, and there is more strain thrown on the muscles, the chordæ tendineæ and valve curtains, than in Phase 1.

It is at this point, in my opinion, that the mechanism of the production of a double first sound comes into play. The muscoli papillares being weakened actually to normal, or relatively to increased intraventricular and arterial pressure, suddenly "fail," and allow the valve curtains to be driven forcibly upwards towards the auricular cavity (Fig. 3). When

the curtains reach the limit of the stretched chordæ tendineæ and muscoli papillares they are suddenly arrested, and an abrupt sound of valve tension is produced, forming the second portion of the double first sound. The intensity of the second part of the double sound thus caused depends upon:

1. The abruptness of the failure of the muscoli papillares. I believe that these muscles when recovering from a pathological state can, and frequently do, fail slowly, and then no sound of sudden tension will be heard. The more abrupt the failure the more abrupt and intense is the resulting sound.

2. Whether the muscoli papillares of one or both ventricles be affected. The second part of the double sound may be formed by the mitral and tricuspid valves. One valve only

may be affected, the muscoli papillares of the other not entering into the causation of the sound, or else both valves may combine to produce the sound of papillary failure.

Both the above factors, then, help to explain the variations in the intensity and loudness of the second part of the double sound which are often observed, and also why at one time the first part of the double sound is louder than the second part, whilst at others the converse holds.

This failure of the muscoli papillares may take place early or late in Phase 2 of the ventricular systole, or in Phases 3 or 4. The distinctness with which the double first sound so produced is heard will depend upon the time of the ventricular systole at which the failure occurs. If we assume, for the sake of further explanation that the normal first and second cardiac sounds can be represented respectively by the phonetic syllables rūbb dūp the double first sound may be compared, when it occurs in Phase 2 with the sound r-rūbb, when in Phase 3 with ru-rūbb, and in Phase 4 with rūb-rūbb. The first sounds of the cardiac cycle heard in these three variations of the double first sound would then be r-rūbb-dūp—rū-rūbb-dūp—rūb-rūb-dūp. This explanation of the double first sound will also hold good if we assume, as some observers do, "that the papillary muscles begin their contraction at the same time as the rest of the ventricular wall."⁶ The fact that the muscoli papillares do contract is the all-important point, the time of contraction being a secondary consideration.

EVIDENCE FROM SPHYGMOGRAPHIC TRACINGS.

If the explanation given be the right one, an examination of sphygmographic tracings obtained from patients in whom the phenomenon can be heard ought to yield some points in its favour.

I have taken pulse-tracings in all the cases which have come under my observation lately with very uniform results. Before describing these, there are one or two points about the normal pulse-tracing to which I wish to direct attention. I again quote from Roy and Adami's⁷ valuable paper on this subject. In their analysis of the various parts of a normal pulse-tracing, special attention is paid to the signification of what used to be called the percussion wave, but which they prefer to call the papillary wave.

There is, on the intraventricular pressure curve, and during that part of it which corresponds to the time when the aortic valve is open, a superposed wave due to the sudden pulling down of the auriculo-ventricular valves by the papillary muscles during the first rapid part of their contraction. It is easy to prove that this superposed wave of the intraventricular pressure curve corresponds exactly in time with the first superposed wave of the pulse curve (Figs. 4 and 6, b to c), one

Fig. 4.—Normal low tension pulse.

Fig. 5.—Low tension pulse with wave of papillary failure.

Fig. 6.—Normal high tension pulse.

Fig. 7.—High tension pulse with wave of papillary failure.

a, b, contraction of ventricles (Phase I); b, c, contraction of papillary muscle, "papillary wave" (Phase II); c', d, wave of papillary failure; c' or c, d, ventricle outflow remainder wave (Phase III); d, e, dicrotic wave (Phase IV).

⁵ Foster, *Textbook of Physiology*, 6th Edition, 1893, p. 233.

⁷ Loc. cit., pp. 415 and 416.

which usually forms the culminating part of the tracing. The rise of pressure due to the contractions of the papillary muscles is synchronous with this first superposed wave of this pulse-tracing; in other words, it is due to the contraction of the papillary muscles. The second wave (Figs. 4 and 5, c, d) between the papillary wave and the dicrotic notch corresponds in time with the outflow from the ventricle due to the continued contraction of the heart wall and papillary muscles after the flaps have been pulled down. This second wave, which is usually known as the tidal or predicrotic wave, may therefore be termed the *outflow remainder* wave. It corresponds in time with the third phase of ventricular systole. After this wave comes the dicrotic notch (Figs. 4 and 6, d, e). The usual explanation of this negative wave is that it is due to the inertia of the blood in the aorta and larger arteries, which has gained a certain velocity during the period of outflow from the ventricle, and which, upon the blood ceasing to leave the ventricle, necessarily causes a negative wave commencing at the root of the aorta and propagated in the same way as the posterior wave. This explanation is, as far as we know, the correct one.

In the normal tracing then that portion of the upstroke above the level of *b* (Figs. 4 and 6), the papillary wave, is caused by the partial contraction (in Phase 2 of ventricular systole) of the papillary muscles. The contraction is completed along with that of the ventricular wall in Phase 3 (Figs. 4 and 6, c, d), causing the expulsion of the remainder of the blood in the ventricular cavities into the aorta, and keeping up the intraventricular blood pressure through Phase 4. If, however, the papillary muscles are unable to maintain through Phases 3 and 4 the contracted condition which has given rise to the papillary wave of the pulse-tracing they will fall more or less abruptly, and with this failure there will be a temporary diminution in the intraventricular pressure. This diminution in the pressure will be recorded on a sphygmograph by an abrupt drop of the lever before the predicrotic wave (Figs. 5 and 7, c-c'). By this abrupt drop the papillary wave is given a sharp pointed appearance, and is about twice its normal height. In some pulse-tracings (Fig. 14) there can be seen on the down stroke of the wave of papillary failure a slight abortive attempt at the formation of a wave which normally would have been the ventricle outflow remainder (predicrotic) wave had not the papillary muscles yielded to the strain. In such cases in which probably the heart muscle is improving, the failure of the valve muscles is not so sudden and complete as in those in which there is no such break in the down stroke of the wave. As a result of the failure of the papillary muscles the ventricle has now to complete the expulsion of the blood from its cavity unaided.

The diminution of the intraventricular blood-pressure caused by the papillary muscle failure temporarily reduces the resistance to the contraction of the ventricle wall. This diminution of intraventricular blood-pressure extends only over the time occupied by the valve curtains in passing from the position they hold during the contraction (Fig. 2), to that occupied after the failure (Fig. 3) of the papillary muscles. When they arrive at this latter point, the intraventricular pressure is raised again by the contraction of the ventricle, which, during the diminution of resistance has been taking place more rapidly and forcibly—with a “rush,” so to speak. The remainder of the blood in the ventricular cavity is then expelled with a jerk, which is recorded on a sphygmograph by a ventricle outflow remainder (predicrotic) wave of exaggerated height, but of fairly normal length (Figs. 8, 10, 11, 12, 13, 15). The alteration in the character of this latter wave is also partly due to there being a larger amount of blood left in the ventricle for expulsion by the final contraction of its wall than there would be did the papillary muscles retain their normal contracted condition.

In the type of double first pulse tracing described above, the failure of the musculi papillares is probably due to actual muscle weakness, the arterial tension remaining normal, or even being subnormal; but when the double first sound is due to abnormally high arterial tension acting on normal or even hypertrophied muscle, the character of the tracing is somewhat different.

Roy and Adami state⁸ that “a rise of pressure in the arteries diminishes the amount of blood thrown out by the contraction of the papillary muscles and increases that thrown out by the heart wall.” There is therefore in a tracing of a high tension pulse a diminution in the height of the papillary wave when the papillary muscles are acting normally (Fig. 6), with an emphasising of the ventricle outflow remainder wave. In a high tension pulse the wave of papillary failure when present is well marked, but not as high

as in low tension pulses. The ventricle outflow remainder (predicrotic) wave has much the same characteristics in both types of pulse, but the height is somewhat greater in high than in low tension.

Fig. 8.—J. J., acute Bright's.

Fig. 9.—J. G., subacute Bright's.

Fig. 10.—M. E. H., chronic Bright's.

Fig. 11.—M. E. H., chronic Bright's.

Fig. 12.—J. H., phthisis.

Fig. 13.—J. T. chronic rheumatism.

Fig. 14.—H. H., anæmia, cardiac dilatation.

Fig. 15.—J. C., rheumatic gout.

The above tracings were all taken by myself and with the same instrument (Dudgeon). They were obtained from patients suffering from acute and chronic Bright's disease,

⁸ Loc. cit., p. 349.

chronic rheumatism, phthisis, and anæmia with cardiac dilatation. I took several tracings from each case day by day, with uniform results. The characteristics of the "double first sound pulse" were always brought out best by obliterating with the finger applied to the radial pulse on the distal side of the sphygmograph any effect which a recurrent pulse from the arteries of the hand might have on the tracing, but an exaggerated ventricle outflow remainder wave was distinct in all those taken in the ordinary way.

PARALYSIS OF THE SIXTH AND SEVENTH NERVES OCCURRING IN A PATIENT WITH WHOOPING-COUGH

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THE case first came under my notice on November 11th, 1895, the patient being a little girl aged 3 years and 4 months. There is no syphilitic or tuberculous history, and the only tendency to nerve disease traceable in the family is in the mother, who periodically complains of headache, probably migrainous. She has given birth to four children. She never had any miscarriages.

Fig. 1.—Showing face at rest.

The patient is the third of the family. When 9 months old she had a slight attack of bronchitis, which soon passed off. Save for this, she has always been healthy. During the second week of September whooping-cough appeared. The paroxysms of cough were not over-frequent during the day, but numerous and severe at night. On one occasion, during a paroxysm of coughing, blood came from the left nostril; this was the only time any visible hæmorrhage occurred.

About the end of the first week in October, the mother noticed the child's left eye squinting, and its face "peculiar," especially when she cried from toothache, due to a few carious teeth.

On November 11th, I was called in; that is, two months after the whooping-cough commenced, and about five weeks after the appearance of the paralysis.

The Facial Paralysis.—The characteristic physiognomy of facial paralysis was well marked. The lower part of the face appeared as if all the furrows were washed out, and it did not

take any part in the movements of the face, even in the movements produced in crying, which deeply furrowed and wrinkled the sound side. The angle of the mouth on the affected (left) side was on a lower level, and somewhat nearer the middle line; this was better marked when the child was sobbing, as the zygomatic muscles of the sound side pulled the mouth towards them. The lower lip and chin also appeared drawn over to the sound side, and the orifice of the mouth oblique. The mother informed me that she put her tongue out nearer the paralysed side of the mouth (she would not protrude her tongue for me). This was explained by the paralysed side of the mouth being drawn over towards the sound side, and the tongue thus appeared to be protruded towards the paralysed side of the mouth.

In the illustrations, in one of which the child was in a comparatively quiet mood (Fig. 1), the deformity produced by the facial paralysis is not great, whereas in the other the child was crying (Fig. 2), and the great contrast between the two sides of the face is well shown. When sleeping, the mother says the contrast between the two sides of the face is not marked. She could not firmly close the paralysed side of the mouth, some saliva more or less constantly dripping from the paralysed angle. Her mother stated that she could not blow straight in front of her, that she could not spit straight in

Fig. 2.—Showing facial paralysis when the child cried.

front of her, and that she chewed all her food with the non-paralysed side of the mouth. The orbicularis palpebrarum and frontalis muscles were very much weakened, though not absolutely paralysed. In ordinary winking the child did not shut the eye completely. In making an effort to shut her eye, she succeeded in closing it. When the eyes were shut forcibly the wrinkles around the paralysed eyelids were not so well marked as those around the non-paralysed eyelids. When she looked upwards the eyebrow on the paralysed side was not raised so high as on the non-paralysed side. Each inspiration during sobbing was accompanied by elevation of the eyebrow on the sound side only. The space between the eyelids on the paralysed side was greater than on the non-paralysed side, as is seen in the illustration. I believe sensation was unimpaired, and that taste, smell, and hearing were unaffected.

Eye Phenomena.—The left external rectus was completely paralysed, so much so, that in the resulting internal