

EXPERIMENTAL AND CLINICAL INVESTIGATION OF THE PULSE AND BLOOD PRESSURE CHANGES IN AORTIC INSUFFICIENCY.*

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INTRODUCTION.

Although the collapsing pulse has, since the time of Corrigan, been one of the most characteristic features of aortic insufficiency, and although in its exaggerated form it furnishes the most typical of all pulse tracings, nevertheless one is struck by the frequency with which the sphygmograph produces a seemingly normal tracing in cases of perfectly definite aortic insufficiency where the sensation to the finger is that of a collapsing pulse. Usually it has been considered that such tracings are the result of instrumental defect or imperfect technic in making the tracings, and in a few cases they are ascribed to a simultaneous occurrence of aortic stenosis along with the insufficiency.

The discrepancy between the sphygmographic tracings and the subjective notes is well exemplified in the examination of the records of patients observed in the Johns Hopkins Hospital during the past four years, in which the pulse was described and the description accompanied by a sphygmographic tracing. Of 50 such records of cases of aortic insufficiency, in 58 per cent. the pulse was stated to be collapsing, while in the remaining 42 per cent. it was described as being gradual in its fall or non-collapsing.

The large proportion of cases which do not present a collapsing pulse is a point of great importance, and will be referred to later. The rapidity of the upstroke, which is constantly referred to by authorities as one of the leading features of the pulse, varied so little in the various tracings that it was impossible to utilize it as a standard whereby the two types of pulses could be compared. Advantage was taken, however, of that other characteristic of the sphygmogram, viz.: the low position of the diastolic notch on the catacrotic limb as the criterion for comparison. In all but two cases, in which the pulse was described as non-collapsing, the notch was situated above a line drawn parallel to the abscissa, and midway between it and the apex. In those cases described as collapsing,

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no such definite relation existed, for in those in which the amplitude of the pulse wave was great it was situated below this line, while in those in which the pulse wave was small it assumed a position above it, and the tracing partook of all the characters of a normal pulse.

Examples of these different types of pulse in aortic insufficiency are seen in Figures 1, 2 and 3.

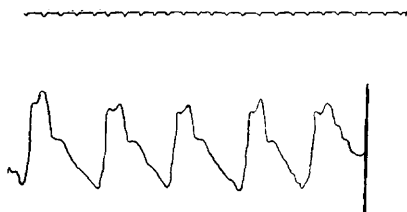


Fig. 1.—Pulse described as non-collapsing. The dicrotic notch is situated above the line midway between apex and trough of the waves. Case of uncomplicated insufficiency. Systolic blood pressure, 160. Diastolic blood pressure, 70.

From the explanation hitherto advanced, that collapse of the pulse in this disease results from regurgitation, it is difficult and indeed impossible to account for the absence of the collapsing pulse in the large percentage of cases mentioned above. The explanation which would first suggest itself would be that the leak was small in degree and insufficient to render the vessels flaccid. The intensity of the diastolic murmur at the base of the heart, or the extent of cardiac hypertrophy and dilatation being no absolute gauge of the amount of blood which regurgitates

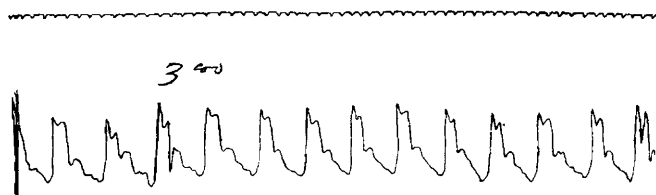


Fig. 2.—Pulse described as collapsing. The dicrotic notch is below the mid-line. Systolic pressure = 150. Diastolic pressure = 60.

into the ventricle, it was only from those cases which came to autopsy that information could be gathered on this point. Of the 21 cases which during life presented evidence of aortic insufficiency without a water-hammer pulse, 6 were examined postmortem. Four had varying degrees of arteriosclerosis. In 3 autopsies, the cusps were not so described that conclusions could be reached as to their actual incompetency. In the other 3, however, the valves were stated to be markedly shrunk and incompetent (without any accompanying stenosis). It is clear, then,

from these facts that distinct inadequacy of the aortic valves can exist without any accompanying water-hammer pulse, and its absence must be due to some other condition of the circulation.

The explanation will be proved experimentally to be that which was surmised by Sansom.¹ He recognized two types of pulse in aortic insufficiency. One in which the rise and fall are sudden, and a second in which there is "a resemblance with the tracings indicating high tension." In describing the latter class, he says: "The summit of the trace is broadened, the tidal wave being sustained, the dicrotic notch, though ill pronounced, is high in the tracing, and the gradually sloping line corresponding to the diastolic period tells of peripheral obstruction." The question, then, naturally arises, When shall a pulse be considered collapsing? Simple as this question at first sight seems, we are confronted at once with some difficulty in answering it. For, according to MacKenzie² and Broadbent,³ the most distinctive feature of a collapsing

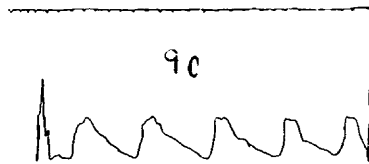


Fig. 3.—Pulse described as collapsing. The amplitude is small and the dicrotic notch is situated above the midline. Apparently a normal pulse tracing. Systolic pressure = 110. Diastolic pressure = 60.

pulse is, in effect, the fact that while in the normal radial pulse the dicrotic notch falls nearer the summit than the trough of the pulse wave (i. e., above the line drawn midway between summit and trough), in the collapsing pulse it is to be found below this line. In other words, the greater portion of the fall in the collapsing pulse occurs before the dicrotic notch, while in the normal pulse it occurs after it. Now, since Huerthle⁴ has proved that the dicrotic notch divides the pulse tracings into two portions, the systolic occurring before the notch and the diastolic occurring after it, it is but a paraphrase to restate the criterion of MacKenzie and Broadbent by saying that *in the normal pulse the greater portion of the fall in the pulse wave is diastolic in time, while in the collapsing pulse of aortic insufficiency the greater portion of the collapse (occurring before the dicrotic notch) is systolic.*

1. Diagnosis of Diseases of Heart, London, 1881, p. 290.

2. Study of Pulse.

3. The Pulse, p. 205.

4. Arch. f. d. ges. Physiol., Bonn, 1891, xlix, 65.

In other words, if MacKenzie and Broadbent are correct, the greater part of the collapse occurs, not at the time when blood should be leaking back into the ventricle, but while it is still flowing from the ventricle into the aorta, during systole. The authors themselves do not attempt to explain this paradox; in fact, they do not even appear to realize its existence.

We are, therefore, confronted with the following problems:

First.—To investigate the substantial truth of the statement of MacKenzie and Broadbent, that the dicrotic notch occupies a low position in the pulse curve of aortic insufficiency. In other words, to determine whether the main fall of pressure is systolic or diastolic in time.

Second.—To determine the condition to which the main fall of pressure in aortic insufficiency is due.

In order to solve these problems, I have made numerous observations on patients in the medical service of Prof. L. F. Barker, in the Johns Hopkins Hospital, Baltimore, and I have performed many experiments on animals in the physiologic laboratory of the Johns Hopkins medical clinic, which is under the charge of Dr. A. D. Hirschfelder.

I would take this opportunity of expressing my warm thanks to Professor Barker for the many privileges which he has in so generous a manner extended to me while attending the medical clinic of the Johns Hopkins Hospital. Through his kindness I was enabled to utilize the clinical material under his charge and at the same time to carry on experimental work in the physiologic laboratory of his clinic.

I am also under a debt of gratitude to Dr. Hirschfelder. The experimental part of this research was carried out under his guidance, and my obligations are heartily acknowledged for the untiring assistance and valuable advice which he has at all times given me.

HISTORICAL SUMMARY.

While to Hodgkin⁵ may be ascribed the distinction of being the first to give a clinical description of this disease, it was reserved for Corrigan⁶ to offer, three years later, in 1832, an explanation of its most prominent features.

He recognized in the pulse only the grosser qualities of throbbing and pulsation. In his original article the following description is given:

“When the semilunar valves . . . become incapable of closing the mouth of the aorta, then, after each contraction of the ventricle, a portion of the blood just sent into the aorta, greater or less according to the

5. London Med. Gaz., 1829, iii, 433.

6. Edinb. Med. and Surg. Jour., 1832, xxxvii, 225.

degree of inadequacy of the valves, returns back into the ventricle. Hence, the ascending aorta and arteries arising from it, pouring back a portion of their contained blood, become, after each contraction of the ventricle, flaccid or lessened in their diameter. While they are in this state the ventricle again contracts and impels quickly into these vessels a quantity of blood which suddenly and greatly dilates them. The diastole of the vessels is thus marked by so sudden and so great an increase of size as to present the visible pulsation which constitutes one of the signs of the disease."

Corrigan was conscious of the fact that the backward current of blood could not sufficiently account for the appearance of the collapse in the arteries of the lower limb, or of the arm when dependent, since the blood would have to flow upward in its return to the aorta, and to render this possible he says: "The elasticity of the brachial arteries on the blood just urged into them forces it back along the retrograde current of the subclavians, no obstacle meeting it in that direction." He considered that the strength of the heart had little to do with the result, and that the explanation lay in the wave of large amplitude which was thrown into the empty artery.

Galabin,⁷ forty years later, laid stress on the quality of extreme suddenness in the commencement of the pulse, which gives to the finger the sensation of a sudden jar or knock. He considered that this is the diagnostic quality of the pulse wave of aortic insufficiency, and pointed out that the amplitude of the wave itself is not characteristic, unless it is accompanied by the sudden jar.

This suddenness he attributed, not to an increased rate of systolic output, but to conditions in the vessel which altered the character of the pulse wave as it travels from the heart to the periphery. He maintained that the collapse did not exist in the larger arteries, such as the aorta and subclavian, but that the transformation which is seen in the wave as it traverses the peripheral vessels, is only an exaggeration of a change in the pulse form which takes place in the healthy adult. He showed that the rate of transmission of the pulse wave is dependent on the tension of the arterial walls, it being greater as the tension rises. Since the tension on the arterial walls is greater at the summit than at the trough of the pulse wave, the apex tends to overtake the base. The result is that the systolic elevation becomes steeper the further the wave travels from the heart. According to Galabin, in the normal pulse this tendency toward increased steepness of the systolic elevation is counter-

7. *Trans. Roy. Med. and Surg. Soc., London*, 1876, lix, 361.

acted by the recoil of the arterial walls, and as the influence which tends to make this upstroke steeper is the difference between the systolic and diastolic pressures, the conditions are established in aortic insufficiency for its production.

However faulty Galabin may have been in his reasoning, there is no doubt that he has the credit of being among the first to emphasize those qualities which we now imply with reference to the waterhammer pulse. These are the sudden, abrupt rise of the pulse curve and its rapid subsidence.

Clifford Albutt⁸ introduces a new factor into the conditions which cooperate in the production of the collapsing or water-hammer pulse. The great changes of pressure which occur between systole and diastole have the effect of lengthening and widening the vessel, so that the walls adapt themselves but loosely to the blood which they contain. The arteries are thrown into curves, which on being straightened by each ventricular systole cause the vessel to be thrown out of its bed "with a visible and palpable jerk." That this contributes to the sensation of a jar or shock is undoubted, but it can not be the sole cause, for the waterhammer pulse can be distinctly seen before the vessels have had time to elongate, as is seen to follow the recent rupture of an aortic cusp.

With the exceptions already stated, our conceptions regarding the collapsing pulse have undergone no fundamental change since the publication of Corrigan's article, and while the blood pressure changes have been extensively studied in experimental aortic insufficiency by Cohnheim,⁹ Romberg and Hasenfeld,¹⁰ Kornfeld¹¹ and Rosenbach,¹² the underlying factors in the production of the collapsing pulse either are not mentioned by them or are assumed to be the direct consequence of regurgitation.

Among clinicians, some of the more recent authorities incline more to the emptiness of the arteries as the essential factor in its production (Gibson¹³), while others maintain that the sudden drop of the pulse wave is due to the loss of support from defective valves (Mackenzie²). The pervading idea is that the collapse occurs in the diastolic phase of the cardiac cycle, and that it is a direct consequence of regurgitation into the ventricle.

That the collapse is not diastolic, but occurs during ventricular sys-

8. *System of Med.*, vi, 937.

9. *Lect. on Clin. Path.*, New Syd. Soc., 1889, i, 49.

10. *Arch. f. Exp. Path. u. Pharmakol.*, Leipz., xxxix, 333.

11. *Ztschr., f. klin. Med.*, Berl., xxix, 91.

12. *Arch. f. Exp. Path. u. Pharmakol.*, Leipz., ix, 1.

13. *Diseases of Heart and Aorta*, 183.

tole, and that it is only a secondary accompaniment of regurgitation, the evidence here advanced will prove.

METHODS OF INVESTIGATION.

The experimental part of this research was performed on dogs. It was found, after the first few experiments were performed, that the most suitable animal was a medium-sized English terrier, if possible over 2 years old. Young dogs were unsatisfactory, in that they did not appear to possess the same resistance as the older dogs and succumbed early in the experiment, owing to the severity of the cutting operation.

Each animal received $\frac{1}{2}$ grain of morphia before being anesthetized with ether. This was administered by means of a tracheal cannula in connection with an ordinary ether bottle. After the thorax was opened, this was attached to an artificial respiration pump, and by the suitable adjustment of valve and T-tubes the lungs could be filled to any desired extent.

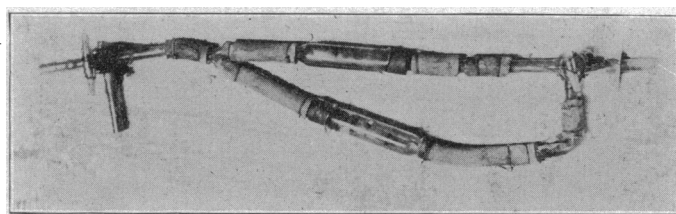


Fig. 4.—Dawson's maximal and minimal valves.

The pulse wave was recorded with a Huerthle membrane manometer, attached to the right carotid artery. Fling of the lever was eliminated by damping the movement with the screw lock of the apparatus.

The systolic and diastolic pressures were measured by the use of the maximal and minimal valves devised by Dawson¹⁴ (Fig. 4). The more minute details are shown in Figure 5.

Each consists of two glass tubes, one enclosed within the other. The orifice of the inner tube, which is situated laterally, is surrounded by a layer of peritoneal membrane, so arranged as to permit the flow of fluid from within outward. When the inner tube A is attached to the artery, and the outer B to a mercury manometer, as in the upper figure, the systolic or maximal pressure only is recorded, the pressure in the outer tube being maintained by the membrane which closes the orifice C of the inner tube, when the pressure falls during diastole. By connecting the

14. Amer. Jour. of Physiol., Boston, 1906, xv, 224.

tube B with the artery and A with the manometer, the valve measures the minimal or diastolic pressure. The systolic rise of pressure now closes the orifice C, while the diastolic fall makes the pressure in the outer tube less than that which is in the inner, and thus causes a flow of fluid from the manometer toward the artery. By a suitable arrangement of stop-cocks (see photograph), the maximal valve is first connected with the artery, and having read off the systolic pressure on the manometer, it is cut off and the minimal or diastolic valve brought into communication with the artery.

In order to render the tracings less complicated, the systolic and diastolic pressures were read off and not recorded on the drum. It is to be noted that these valves do not record the systolic and diastolic pressures

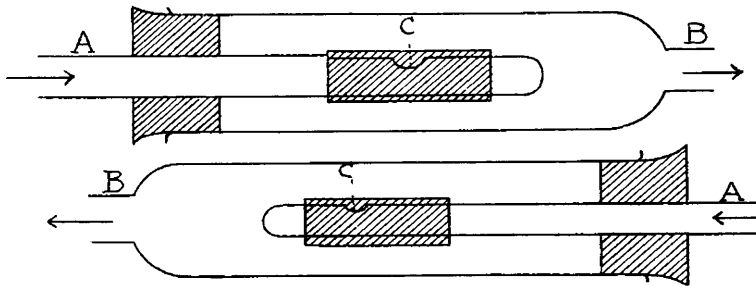


Fig. 5.—Minute details of Dawson's maximal and minimal valves.

of individual pulse waves. They measure the highest and the lowest pressures which have obtained in a given interval of time. If, however, this interval of time is made as short as possible, they will give records, which, for all practical purposes, are the systolic and diastolic pressures of the beats which occurred while the reading was being taken.

The amount of systolic output and the different phases of the cardiac cycle were recorded by the ventricular plethysmograph devised by Henderson¹⁵ (Fig. 6).

It consists essentially of an ordinary soft rubber ball into which a circular window is cut, having a diameter of seven-tenths that of the ball. Over this window a very thin rubber dam is cemented with rubber cement, and after this has dried thoroughly a second circular window is cut into the rubber dam, this window having six-tenths the diameter of the window in the ball. A small hole is then cut into the side of the rubber ball, and through it a small glass tube is inserted and held in place with washers of cork or rubber. The apex of the heart is then

15. Amer. Jour of Phys., 1906, xvi, 325.

inserted into the window in the dam, and the plethysmograph pushed over the heart until the rubber dam fits about the auriculoventricular groove. The fitting should be airtight, but never sufficient to compress the heart. As Henderson states, it is necessary to have several sizes of the plethysmograph, and balls of diameters of 7, 9 and 11 cm., respectively, were used, according to the size of the heart. By its opposite end the plethysmograph communicates by rubber tubing with a large Marcy tambour. The superiority of this instrument over that of Roy and Adami and the older forms lies in the fact that it registers the volume changes of the ventricles only, and, the transmission being by air, the ventricles are subjected to no external pressure which would impede their diastole.

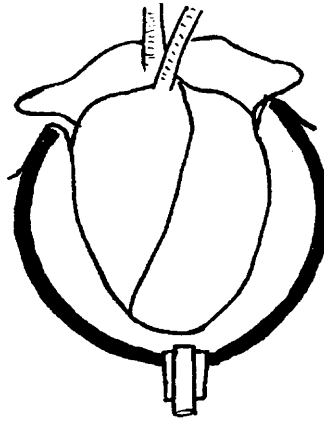


Fig. 6.—Henderson's ventricular plethysmograph in position.

After each experiment, the tambour was calibrated by attaching it to the upper end of a graduated burette filled with water. One cubic centimeter of water was run out at each time, and the excursion of the lever recorded.

After the insertion of the tracheal cannula, the internal mammary arteries were ligatured in order to avoid hemorrhage when the chest was opened. This was done by prolonging the incision for the preliminary tracheotomy to the upper border of the sternum, and separating the suprasternal muscles. The pleuræ were then punctured by the finger and, having dissected the arteries with the finger, a ligature was passed around them with an aneurism needle and then the ligatures tied.

The heart was exposed by reflecting the skin and pectoral muscles by a median incision extending along the whole anterior aspect of the chest. The ribs were cut near their attachments to the cartilages, and the ster-

num and costal cartilages removed. The pericardium was incised and reflected, and the plethysmograph slipped over the ventricles, until the rubber membrane grasped the auriculoventricular groove.

The aortic valves were ruptured by MacCallum's valvulotome.¹⁶ This instrument (Fig. 7) consists of a steel cutting hook which is passed down the left carotid artery. The cutting surface is on the concave

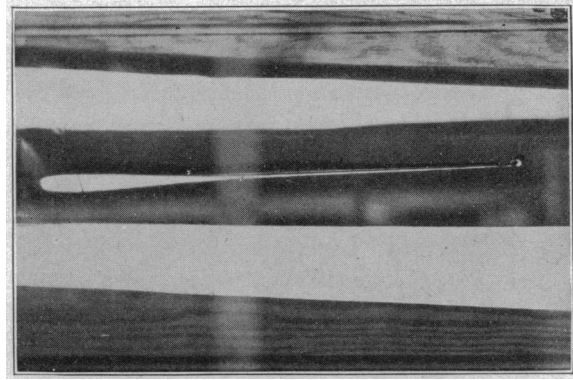


Fig. 7.—MacCallum's valvulotome.

aspect of the hook, which is so curved that it does not injure the vessels when passed down toward the heart. The instrument is pushed down the artery until it enters one of the sinuses of Valsalva, after which it is forced through the base of the cusp. The hook is then pushed inward, and, on being withdrawn, it cuts the cusp in two.

Intraventricular pressure was measured by means of a metal catheter passed down the left carotid artery and connected with a Huerthle manometer (Fig. 8).

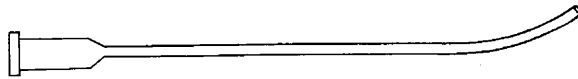


Fig. 8.—Intraventricular sound.

The changes in auricular pressure were recorded by passing a glass cannula into the auricle through a pulmonary vein and connecting this with a Harvard membrane manometer.

For the arterial, intraventricular and auricular pressures, the medium of transmission was a 5 per cent. solution of sodium citrate.

The heart of the animal was examined at the termination of each experiment, and only in those cases in which the valves were torn were

16. Johns Hopkins Hosp. Bull., Balt., No. 185, 1906.

the results put on record. In several cases, it was found that, in addition to the production of insufficiency, the intraventricular septum and the chordæ tendinæ were injured. The results of these experiments were not taken into account. In all, thirty experiments were performed, of which ten were successful, i. e., the aortic valves only being injured. In 9 the myocardium had been more or less damaged. In the remainder no insufficiency was obtained.

Methods of Recording.—Simultaneous tracings were made of the pulse changes, the volume changes of the ventricles, and of the time in fifths of a second with a Jacquet chronograph. In the earlier experiments smoked paper was employed, but in the later ones, by the use of improved writing pens, it was found that black on white tracings were

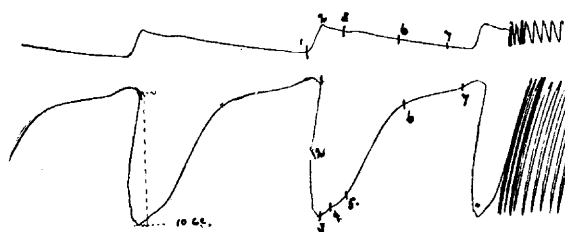


Fig. 9.—Simultaneous tracing of the pressure in the carotid artery (upper curve) and of the volume changes in the ventricle (lower curve): 1. Commencement of ventricular contraction. 2. Summit of primary wave. It is reached about the time that the ventricle has discharged half its contents into the aorta. 3. End of systole. It corresponds to the dirotic notch in the carotid tracing. 3-4. The rapid increase in volume due to the filling of the coronary arteries. 5. End of relaxation or diastole proper. 6-7. Period of diastasis. 7. Commencement of auricular systole. The contraction of the auricle takes place after the ventricles have practically filled. It causes only a slight rise in ventricular volume. The downstroke 1-3 indicates a systolic discharge of 10 c.c.

more satisfactory. In every case, before and after a tracing was taken, the maximum and minimum blood pressures were read off. Typical normal tracings were always obtained before the aortic valves were injured.

THE VENTRICULAR VOLUME CURVES OF THE NORMAL HEART.

The results obtained by Henderson¹⁵ are of such far-reaching importance that they mark a new era in our conceptions regarding the mechanism of the circulation in the normal heart. And, if true of the normal heart, they must modify to a still greater extent our views concerning the changes which occur in the circulation as a result of incompetency of the aortic valves. As one of the main arguments of this research is based on these results, his experiments have been carefully repeated, and the findings are in entire accord with those which he has obtained.

Figure 9 is a simultaneous record of the volume curve and the lateral pressure in the aorta as measured from the right carotid artery. After making allowance for the time taken by the pulse wave to reach the carotid artery, the figures represent simultaneous events in the two tracings. It will be seen that the greater part of the downstroke of the volume curve is practically a straight line, indicating a uniform rate of systolic discharge. Keeping in view the fact that the lever carrying the pen writes in the arc of a circle, the lower part of the systolic downstroke is seen to be more gradual, showing that toward the end of systole the discharge is less rapid; 3 indicates the end of systole and the commencement of ventricular relaxation. This is seen to correspond with the dicrotic notch of the carotid tracing. Immediately following the completion of systole there is a small, rapid rise of pressure from 3 to 4.

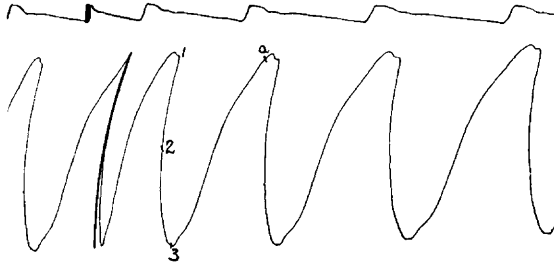


Fig. 10.—Simultaneous tracing of carotid pressure (upper curve) and of volume curve of normal ventricle (lower curve). The heart rate is increased to the optimum rate. The period of diastasis is abolished and the auricular systole (a) follows immediately the ventricular relaxation. The filling is complete. Systolic output, 12 c.c.

Henderson believes that this is due to the rapid entrance of blood into the coronary vessels after they are released from the compression of the contracted ventricles. For an instant after this sudden rise of pressure there is a delay from 4 to 5, followed by a large upstroke *which is almost as rapid as the systolic discharge*. The tracing shows the ventricles have filled at 6 in .43 of a second, early in diastole, and they remain in this quiescent state until the onset of the following auricular systole. At 7 the auricle contracts, and it is seen that it ejects only a fraction of a cubic centimeter of blood into the already filled ventricle.

RELATION OF HEART RATE TO THE AMOUNT OF SYSTOLIC OUTPUT.

While the preceding tracing may be taken as typical of the heart beating at a normal rate, Figure 10 shows the changes which occur when the rate of the heart beat is increased. Tracing No. 25 shows, after

vagal stimulation, the effect of a slowing of the heart. It will be seen that at the higher rate the rate of discharge or refilling has not appreciably varied. The change has involved only a shortening, and in this case abolition of the period of quiescence. On the other hand, after vagal stimulation, the duration of systole and relaxation have remained the same, but slowing has been affected by lengthening the interval of rest. It is evident that an increase of rate up to the point which abolishes the period of quiescence increases the amount of blood thrown out into the aorta, per unit of time, and, other things being equal, it will increase the work done by the heart and also the mean pressure in the arterial system.

If, however, the heart be made to beat at a still higher rate (Fig. 11), the relaxation curve is encroached on by the following systole and, complete filling being prevented, it will involve a diminution in the amount of blood discharged at that systole. The work done by the heart in such

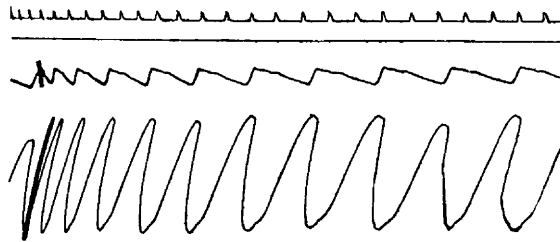


Fig. 11.—Upper curve, carotid pressure. Lower curve, volume curve of ventricle. This shows effect of a very fast heart rate. Not only is there no period of diastasis, but the ventricular filling is interfered with by the following systole. The output at each contraction is diminished. Systolic discharge, 8 c.c. Time in fifths of a second.

a case is not increased, for, although the rate is greater, the amount thrown out at each systole is less, and, therefore, the output per unit of time does not vary.

The conclusions, then, which are to be drawn from the volume curves of the ventricles are:

First.—That the part played by the systole of the auricles is of little importance in the filling of the ventricles. They act more as elastic reservoirs than as muscular force pumps. The force which fills the ventricle is acquired from the potential energy stored in the walls of the auricles from their distension by the venous pressure. The ventricles are not filled by the contraction of the auricles, for they are already full before the onset of auricular systole. Henderson thinks that the wave of increased pressure sent into the ventricles by the auricular systole has as its function only the closure of the auriculoventricular valves, which have previously been floated into position by the inflow during diastole.

Second.—That the rapidity of ventricular relaxation is independent of the rate of the heart, and is an unalterable characteristic for each heart.

Third.—That the cardiac cycle is not diaphasic but triphasic, and “consists of systole, the period of contraction and discharge, diastole the period of relaxation and filling; diastasis, the period of rest.”

EFFECT OF VOLUME CURVES OF VENTRICLES WHEN AORTIC VALVES ARE DAMAGED.

The most satisfactory method of comparing the volume curves before and after insufficiency is to trace the normal on transparent paper and lay this over the abnormal. It is necessary for this purpose that the heart rate be the same in the two tracings and the rate of rotation of the drum

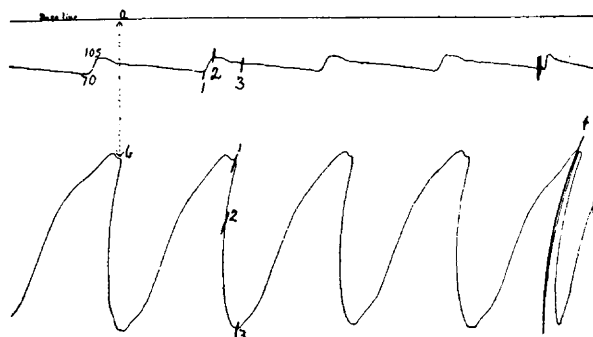


Fig. 12.—Carotid and plethysmographic curves taken immediately before the production of insufficiency. Systolic pressure, 105; diastolic pressure, 70; systolic output, 12.8 cc. The line a-b represents the ventricular tonus.

constant. Figures 12 and 13 are the actual tracings taken before and after the production of insufficiency.

When the tracings are superimposed (Fig. 14) it will be seen, contrary to expectation, that the rapidity of ventricular output is slower in aortic insufficiency than it is normally. This is proved by the fact that the apex of the curve is reached in the normal in .18 second, while in the abnormal it occupies an interval of .22 second.

The amount of ventricular output differs in the two by only $\frac{1}{2}$ c.c. of blood. This is in direct contrast to the views at present held regarding regurgitation. It is thought that the amount of blood which returns to the ventricle when added to that which normally enters from the auricle is sufficient to cause a dilatation of the ventricles. Even with the most extensive damage to the valves, the increased systolic output did not exceed the normal by more than 1 c.c. of blood.

The upstroke, that part of the tracing which represents the cardiac filling, shows that the ventricle fills somewhat faster in the abnormal condition. Its acceleration, however, involves only the commencement of diastole proper, as is demonstrated by the more gradual ascent of the first part of the uprise. After the ventricles have filled to about one-fifth of their capacity, the rate of filling becomes the same, the remaining part of the uprise being gradual. When the heart remains the same, the time lost by the longer systole is compensated for by this increased rate of filling in early diastole.

If, now, the distance between the diastolic positions of the curves and the base line be compared in the two tracings, an indication will be

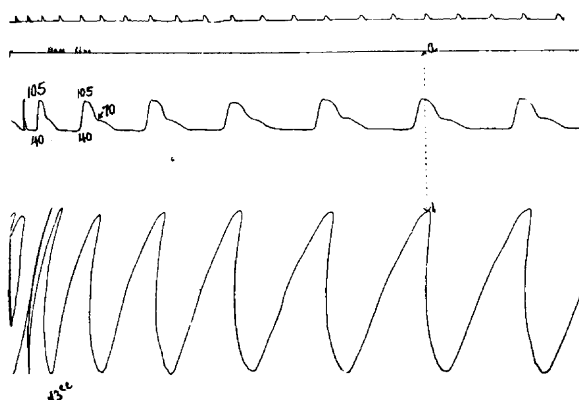


Fig. 13.—Carotid pressure and ventricular volume curve after the production of aortic insufficiency. Systolic pressure, 105; diastolic pressure, 40; systolic output, 13 c.c. The line a-b represents the ventricular tonus. Compare with tracing No. 12, in which it is shorter. This indicates that the ventricle is less distended. Ventricular tone and distension vary inversely with one another.

given of the relative distension in the two conditions. The “base” in this instance being situated above the tracing, it is, of course, evident that the shorter this distance the greater the ventricular distension, and *vice versa*. On the other hand, the further the systolic portions of the curves are from the base line the more completely do the ventricles empty themselves.

It must be recalled that the systolic discharge volume of the normal heart is not necessarily an indication of the amount of ventricular distension or of the actual amount of blood which the ventricle contains. As Henderson¹⁷ has shown, it is about 60 per cent. of the total volume difference between extreme distension as would follow a prolonged vagus

17. Loc. cit., p. 365.

stimulation and extreme contraction, as exhibited in hearts which have stopped beating in systole.

A constant systolic discharge may be obtained in one of three ways.

(1) It may occur with the normal amount of relaxation and the normal degree of ventricular contraction. (2) With an increased relaxation and less complete ventricular contraction, the residual blood being increased. (3) With a diminished relaxation and a more complete systole, in which case the residual blood is diminished.

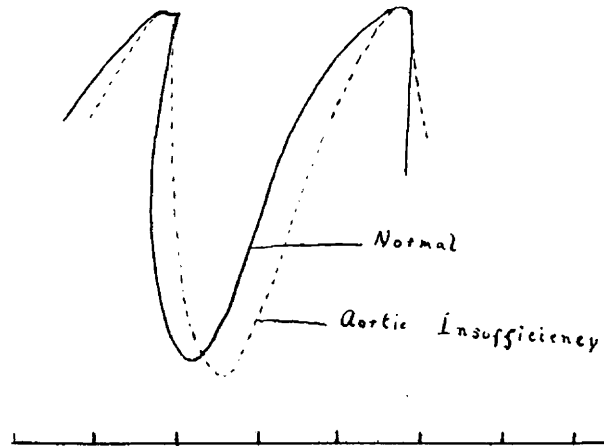


Fig. 14.—Superimposed tracing of the normal ventricle and that of the ventricle after the production of aortic insufficiency. The rate of systolic discharge is slower in abnormal state.

These different states of the ventricle are represented diagrammatically in Figure 15. As to which of these three conditions will obtain will depend entirely on the ventricular tonus. A comparison of this distance in the two tracings (12 and 13) reveals the surprising fact that the production of aortic insufficiency causes the ventricle to contract in Condition 3, viz.: with a diminished relaxation and more complete systole. The amount of residual blood which is known to exist normally is diminished, and, while the ventricular filling is slightly greater, the ventricle at the end of diastole is less distended. This is also shown in Figure 18.

It is impossible, in view of these facts, to accept the statement of Johannsen and Tigerstedt,¹⁸ that in experimental aortic insufficiency the ventricle does not empty itself so completely. This statement was based chiefly on the theoretic grounds that when the ventricular cavity is dilated the blood is less completely expelled during systole. But it has

18. Skand. Arch. f. Physiol., i, 331.

been shown above that the ventricle is less distended after the simple production of aortic insufficiency, and this can only result from an increased ventricular tonus. It will be shown later that if the tone is diminished the ventricle dilates and the amount of residual blood becomes increased. The comparison of the tracings, however, furnishes important information on the quantity of blood which regurgitates. In the animal from which these tracings were taken the posterior cusp was completely divided by MacCallum's valvulotome, and a greater degree of insufficiency thus produced than can probably occur in man as a

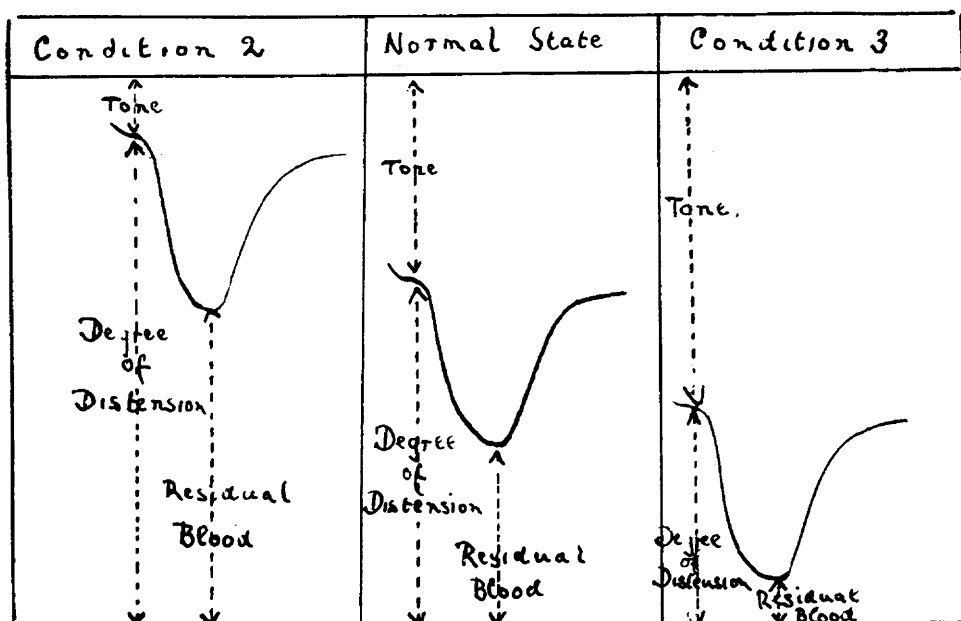


Fig. 15.—Diagram to illustrate the different conditions under which the ventricle with intact valves may contract. The curves represent a plethysmogram, indicating the same amount of systolic discharge in the three conditions. The length of the interrupted lines indicates the degree of tonus, the degree of distension and the amount of residual blood, respectively. In Condition 2 there is an increased relaxation and an increased amount of residual blood, but a diminution of the degree of tonus. In Condition 3 the tonus is increased, while the degree of distension and the amount of residual blood are diminished. Tonus varies inversely with distension and the amount of residual blood.

result of disease. As stated above, the systolic output in the two cases, at the same pulse rate, differed only by $\frac{1}{2}$ c.c. of blood. There was no diminution in the amount of blood delivered by the auricle, as was evidenced by the fact that there was no dilatation of its cavity.

The facts admit of but one interpretation, viz.: that after an extensive valvular lesion the actual amount of blood which regurgitates is fractional in quantity as compared with that which enters naturally from the auricle.

It may, then, be asked why the loss of so small a quantity of blood should give rise to the various phenomena associated with this lesion. The point will at once become clear when one understands that the essential change is a loss of pressure rather than a transference of blood from the aorta to the ventricle. It must, of course, be admitted that some blood must regurgitate, but the conditions under which the regurgitation occurs, are such that it becomes of the smallest possible amount. The aorta and ventricle being in direct communication, the pressures in the two chambers will tend to become equalized, and the curves of intra-ventricular and arterial pressures will show that this does occur. But a transference of pressure does not necessarily presuppose a transference of blood, just as a rise of pressure in the radial artery during the passage of the pulse wave is not due to a corresponding increase in the amount of the radial blood stream.

The tendency toward regurgitation is lessened in the first place by the low pressure which will be shown to obtain in the aorta immediately after the completion of systole. This systolic end pressure or that which is present at the termination of the systolic plateau will be shown, also, to be very different from that which is produced at the summit of the primary wave, and the relation of the two is an indication of the rapidity of capillary flow. It is entirely distinct, although it does not differ, on an average, by more than 15 mm. mercury from the pressure in the aorta at the end of diastole. The difference in pressure between the systolic end pressure and that at the end of diastole is accounted for partly by the loss due to regurgitation and partly by the capillary escape. Under no circumstances, then, can this fall of pressure due to regurgitation exceed or even equal this difference.

But another factor contributes to the prevention of a large amount of regurgitation of blood, viz.: the rapidity of ventricular filling. At the moment of ventricular relaxation, the pressure in the left ventricle is negative, and, as a result, the aortic and auricular blood are rapidly forced toward the ventricle. But since, as has been shown already, in the physiologic condition the ventricle receives venous blood only during the early part of diastole, the outflow from the auricle must take place with extreme rapidity. In the pathologic condition, while the difference between the pressures in the left auricle and the aorta is still great, and would increase the rapidity of stream from the latter, the great differ-

ence in size between the two openings through which the blood enters will render the amount of blood delivered from the aorta insignificant as compared with that which comes from the auricle. The ventricles will have filled, therefore, before an appreciable quantity has returned from the aorta. And since the volume curves show that the ventricular tone is greater, immediately the filling is completed, the ventricles are able to withstand the relatively low pressure which is transmitted from the aorta without becoming further distended. When the intraventricular pressure curves are presented, it will be seen that at the end of ventricular repletion the pressure has already reached that which obtains in the aorta, and during the period of diastasis there is no further increase of intraventricular pressure.

The change in form which results in the volume curve as a consequence of increased tension at once suggests an analogy with the corresponding effect of tension in the contraction of a skeletal muscle. The

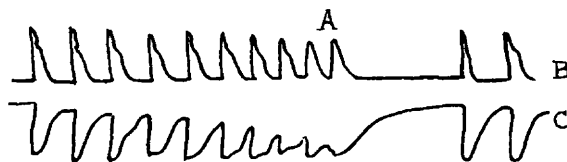


Fig. 16.—Simultaneous isometric and isotonic contractions of the frog's gastrocnemius.

similarity, which was first referred to by Francois Franck¹⁹ between the volume curve and the isotonic muscular contraction and between the intraventricular pressure curve and an isometric contraction in the normal heart, was subjected to experimental test by Henderson.²⁰ By means of an apparatus called a work adder he was able to cause a skeletal muscle (a frog's gastrocnemius) to record simultaneously an isometric and an isotonic curve. In an isometric contraction the muscle is made to contract against a resistance which it is unable to overcome. The resistance commonly employed is a strong spring, and the small excursion induced is greatly magnified by the use of levers. The shortening which results in the muscle being negligible, the curve indicates the changes in tension which the muscle undergoes. An isotonic contraction, on the other hand, is one in which the muscle is, or is not, loaded with a weight which it is able to lift, and the natural shortening is not impeded. The isometric curve is distinguished from the isotonic by the more rapid uprise, indicating that the greatest tension is reached much sooner than

19. *Trav. du lab., de Marey*, 1877, iii, 321.

20. *Loc. cit.*, p. 357.

the greatest shortening, and by an apex which is flat topped instead of rounded. Henderson's simultaneous isometric and isotonic curves (Fig. 16) show in a striking manner the close resemblance which they bear, respectively, to the pressure and volume curves of the normal ventricle.

As a skeletal muscle is influenced in the form and strength of its contraction by changes in the tension which precede or accompany its contraction, so also is the heart when increased tension is thrown on it, as a consequence of abnormal states. Starling²¹ holds that in the normal state the ventricle contracts isotonically, since after overcoming the aortic pressure the muscle fibers can shorten without further hindrance, while in aortic stenosis the ventricle meets with resistance during the whole period of its systole, and, therefore, approximates an isometric contraction. In aortic insufficiency, however, we have a condition in which the increased tension is exerted before the onset of the ventricular contraction and is comparable to the "loaded" isotonic curve. The normal volume curve is similar to the "after-loaded muscular contraction," that is to say,

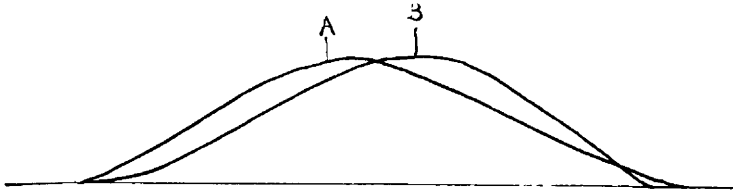


Fig. 17.—A. After-loaded contraction. B. Loaded contraction.

the tension to which the muscle is subjected is that of the load, and it begins to act only after the muscle has already contracted. The ventricles are subjected to no tension before contracting, and, after reaching a certain degree of contraction, the tension remains constant until the end of systole. It is different, however, when the aortic valves are incompetent. Here a considerable tension exists in the ventricular walls as a consequence of the transmission of pressure from the aorta. The condition is the same as that which exists in a loaded muscle preparation, tension is applied to the muscle before it contracts. A comparison of the two volume curves at once shows their resemblance to an after-loaded and a loaded muscle, respectively.

The inversion of the volume curve will render the resemblance still more striking. The above curve (Fig. 17) is based on the work of von Kries²² and shows the effect of applying tension to a muscle prepara-

21. London Lancet, 1897, i, 570.

22. Arch. f. Physiol., Leipz., 1880, p. 370.

tion before contraction. The uprise is more gradual, showing that the rate of contraction is diminished, and the summit of the loaded curve follows that of the after-loaded muscle. It will be recalled that the volume curve of the ventricle in aortic insufficiency differs from the normal in exactly the same respects. It was shown that the rate of ventricular output is slower, and the time taken to effect complete systole delayed.

But the comparison may be extended further. The ventricular contraction of the normal heart is analogous to that in a muscle which contracts and at the summit of its contraction is relieved of its load and allowed to relax without any extending force. The heart in aortic insufficiency, however, is comparable to the muscle which is subjected to ten-

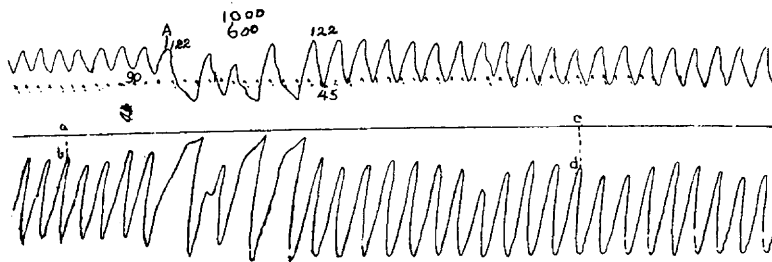


Fig. 18.—Carotid pressure and ventricular volume curve to show the effect of the production of aortic insufficiency. (a) Equals insufficiency produced. The systolic pressure remains the same, 122 mm. of Hg., but the diastolic pressure falls from 90 mm. Hg. to 45 mm. Hg. The volume curve shows that for four beats after the production of the lesion the ventricle becomes considerably dilated, and at this moment regurgitation does probably occur. After a lapse of about two seconds, however, the ventricle recovers itself and the distension becomes less afterward than before. The line c-d is longer than a-b, equals increase in the ventricular tone. The ventricle also empties itself more completely. The apex of the volume curve is further from the base line.

sion during the phases of contraction, elongation and rest. The effect of tension so applied is to increase the rapidity of elongation, and to this action we may attribute the more rapid uprise of its volume curve. The conclusion arrived at by Schenck²³ in relation to the effect of tension on the contraction of skeletal muscle may with equal force be applied to the ventricle: "Tension protracts contraction and hastens relaxation."

EFFECT ON BLOOD PRESSURE OF RUPTURE OF THE AORTIC VALVES.

Cohnheim,⁹ in his classical experiments, found that, although the valves are ruptured and no longer able to effect closure during diastole, "the mean arterial pressure remains at precisely the same level." Rosenbach¹² repeated his experiments and obtained the same results. Since then

23. Arch. f. d. ges. Physiol., Bonn, 1894, lvii, 606.

the investigation has been undertaken by numerous other observers, but their experiments do not, in every case, agree with those of Cohnheim and Rosenbach. While MacCallum¹⁰ found that the blood pressure remains remarkably high, Kornfeld¹¹ noticed that it may either be increased or diminished.

These experimenters, without exception, used the ordinary mercury manometer attached to the carotid artery. As is well known, this instrument does not follow promptly the quick changes of pressure which occur between systole and diastole, and the limited excursion gives only an approximate reading of the mean blood pressure; nor, on account of its relatively sluggish movement, can either the systolic or diastolic pressures be accurately measured. The use of the Huerthle manometer, along with the maximal and minimal valves, gives a ready means whereby both systolic and diastolic pressures can not only be measured accurately, but also recorded.

The following table gives the result of the production of aortic insufficiency on the maximal and minimal blood pressures in ten experiments:

Expt. No.	Before.		After insufficiency.		Lesion.	Result.
	Sys.	Dias.	Sys.	Dias.		
1	125	90	125	50	Post. cusp.	Dog survived shock of lesion.
2	126	80	90	20	Int. cusp.	Dog died.
3	140	120	140	30	Post. cusp large.	Dog survived.
4	115	80	115	28	Ext. cusp.	Dog survived.
5	105	70	75	35	Post. cusp.	Dog died.
6	170	155	100	40	Post. cusp.	Dog died.
7	100	70	100	45	Post. cusp.	Dog survived.
8	155	120	160	80	Ext. cusp.	Dog survived.
9	150	72	150	50	Post. cusp.	Dog survived.
10	120	90	85	10	Post. cusp.	Dog died.

It will be seen that in none is the systolic pressure increased after the valves are rendered incompetent. In those cases in which the animals survived the shock of the lesion, the *systolic pressure remained at exactly the same level*. The diastolic pressure invariably undergoes a diminution which was found not to depend entirely on the amount of damage done to the valves. While the systolic pressure is not affected by the lesion, the mean pressure falls, since, as shown by Dawson,²⁴ it is nearer and follows the diastolic pressure much more closely than the systolic. In all the experiments, therefore, the mean blood pressure fell. It is to be noted that in those animals which succumbed after the production of the insufficiency the systolic pressure also underwent a marked diminution. This merely indicated that the heart was failing and unable to eject the previous quantity of blood. The ventricles under these cir-

24. Ref. Erlanger and Hooker, Johns Hopkins Hosp. Rep., 1904, xii, 150.

cumstances gradually dilated and stopped beating in diastole after the lapse of about ten minutes. And while the fall of systolic pressure resulted from a lessened output the maintenance of the systolic pressure showed that the amount of systolic output in the non-hypertrophied and non-dilated heart did not vary. This was also shown by the volume curves of the ventricles after damage to the aortic valves.

In the meantime it is to be emphasized that the mean blood pressure falls considerably, and this fall is proportional to the fall in the diastolic pressure, the systolic pressure remaining the same as before.

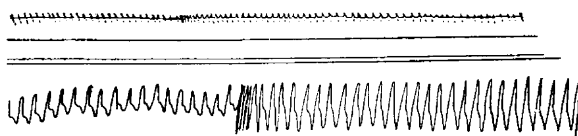


Fig. 19.—Carotid tracing to show the effect of rupture of the aortic valves. Systolic pressure 150 mm. Hg. before and after the lesion. Diastolic pressure falls from 70 to 50 mm. Hg.

It was frequently observed in the earlier experiments that when the valvulotome failed to enter the sinus of Valsalva, but passed directly into the ventricle without damaging a valve, the curve assumed the form seen in undoubted insufficiency. There was an immediate fall in blood pressure, with a great increase in the pulse pressure. Indeed, on two separate occasions the collapsing character of the pulse which resulted was so marked that it was assumed that a valve was cut, and the experiment was proceeded with without again introducing the instrument. When,

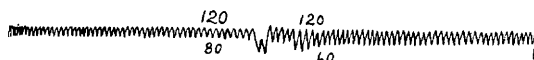


Fig. 20.—Carotid tracing showing the fall in diastolic pressure from irritation of the posterior wall of the ventricle by the valvulotome. There was no insufficiency produced. Systolic pressure before and after, 120 mm. Hg.; diastolic pressure before, 80 mm. Hg.; diastolic pressure after, 60 mm. Hg.; slow drum.

however, the heart was examined postmortem, one was astonished to find that the valves were absolutely intact. In a third experiment it was thought that an insufficiency was obtained, but after the lapse of about an hour the collapsing character of the pulse disappeared and, in view of the previous experience, it was suspected that the instrument had merely entered the ventricle without damaging the valves. It was again introduced and the external cusp torn, which was found on autopsy to be the only valve damaged. The tracings of these experiments are shown in Figures 20 and 21.

In seeking for an explanation of this phenomenon one was at first inclined to believe that it was due to the presence of the instrument in the mouth of the aorta which prevented closure of the valves. This, however, could not have been the cause, since the collapse persisted after the withdrawal of the instrument.

It seems not at all unlikely that the rubbing of the point of the instrument on the posterior wall of the ventricle mechanically stimulated the endings of the depressor nerve and thus brought about the immediate fall in blood pressure. This hypothesis seems justifiable, in view of the fact that Köster and Tschermak²⁵ showed anatomically that the nerve ends partly in the wall of the ventricle and partly in the aorta beyond its mouth. Whatever the explanation is, the observation has an important bearing on the question of the collapsing pulse. It proves that this type of pulse is not peculiar to aortic insufficiency, but can be obtained in any condition in which, with a persistence of ventricular activity, there is a fall in the diastolic blood pressure.

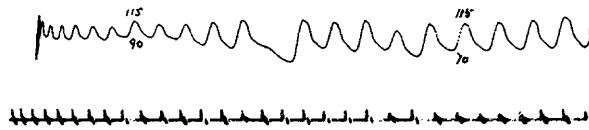


Fig. 21.—Carotid tracing to show also the fall in diastolic pressure which results from passage of the valvulotome into the ventricle without damaging a valve. Diastolic pressure fell from 90 to 70 mm. Hg. No change in systolic pressure.

COMPARISON OF THE NORMAL PULSE WITH THAT OBTAINED AFTER PRODUCTION OF AORTIC INSUFFICIENCY.

Huerthle⁴ has shown, and the plethysmographic tracing confirms it, that the period which elapses between the appearance of the primary and the dicrotic wave is an accurate measure of the outflow from the heart during systole. Of course, it is evident that it is not synchronous with the commencement and termination of systole, since a fraction of a second elapses before the ventricle gathers sufficient strength to open the semilunar valves, and a definite interval also elapses after the end of systole before the appearance of the dicrotic waves. Huerthle found, however, that these intervals are of the same length; therefore, all changes occurring between those points are included in the systolic phase of the pulse curve.

Since the abnormal condition induced by the rupture of the aortic valve does not affect the mechanism of the production of the systolic

25. Arch. f. d. ges. Physiol., Bonn, 1902, xciv, 24.

portion of the pulse curve, that phase can be compared, point by point, in the two pulses. While in the pulse of aortic insufficiency the diastolic portion is the product of an abnormal mechanism, the systolic in both is affected physiologically.

The Amplitude of the Ascending Limb of the Pulse Wave.—The production of aortic insufficiency invariably increases the pulse pressure. The following table shows the increase which took place in six experiments:

Expt. No.	Before insufficiency.			After insufficiency.		
	Sys.	Dias.	Pulse pressure.	Sys.	Dias.	Pulse pressure.
1	125	90	35	125	50	75
2	140	120	20	140	30	110
3	115	80	35	115	28	87
4	100	70	30	100	45	55
5	155	120	35	160	80	80
6	150	72	78	150	50	100

According to Marey,²⁶ it was first demonstrated by Hales, and afterward by Bernard, that when the blood pressure falls, the amplitude of the pulse is increased. Marey himself confirmed their observations, and more recently the investigation has been revised by Huerthle²⁷ with improved methods. He showed that the fall of blood pressure resulting from stimulation of the depressor nerve is accompanied by an increase of the pulse pressure, and conversely that the rise of blood pressure from splanchnic stimulation diminishes the pulse pressure.

Granted, then, a constant systolic output, vasodilation will increase the pulse pressure by lowering, while vasocontraction will diminish it by raising the diastolic blood pressure.

But the pulse pressure is materially influenced by the amount of blood ejected at each ventricular systole and by the relative capacity of the arterial tree. Different amounts of blood, thrown into an arterial system whose capacity does not vary, will produce differences in pulse pressure which will be greater the larger the systolic output. The difference will be affected by an increase of the maximum pressure rather than by a lowering of the mean or diastolic pressure.

If, then, after the production of aortic insufficiency increase of the pulse pressure be accompanied by an increased systolic pressure, the diastolic remaining the same, it must have resulted from an increased systolic output. If the diastolic pressure is lowered and the systolic pressure remains as before, there must have been an attendant vasodila-

26. La Circulation du Sang., p. 269.

27. Pflueger's Arch., 1888, xxxiv, 393.

tion. If, on the other hand, the increased pulse pressure is the result of a lowering of the diastolic and an increase of the systolic pressure, it must have been caused by a true regurgitation. The regurgitated blood will have been added to the normal systolic output, thus raising the maximum pressure, while the diastolic pressure will have been lowered by the loss of the same amount.

The actual conditions which obtain in experimental aortic insufficiency have already been shown (page 123). It was shown that when the animal survives the production of the lesion, the systolic pressure remains at exactly the same level, whereas the diastolic pressure falls in a very marked manner. The pulse pressure has been increased because of this fall, and, as above stated, it can only have resulted from peripheral dilatation.

The Systolic Plateau.—That portion of the pulse curve which extends from the point of maximum pressure to the commencement of the dicrotic notch has been termed by Marey the systolic plateau. This plateau may be horizontal, ascending or, as in the normal pulse, descending.

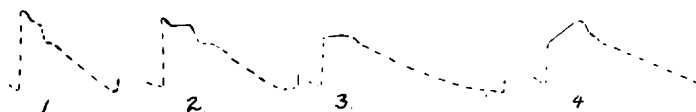


Fig. 22.—After Marey.

When it is horizontal the inflow of blood is exactly equal to the systolic escape from the capillaries. When it ascends, as in the anacrotic pulse, the escape is less than that received into the arterial system, and the pressure continues to rise. When it descends the indication is that the systolic escape by the capillaries is greater than the systolic influx. It naturally follows that the steeper the declivity of the systolic plateau, the greater is the excess of the systolic escape over the systolic inflow. Such, for instance, can always be seen to take place in the normal pulse when the capillary flow is accelerated from dilatation of the peripheral arterioles by the administration of nitrate of amyl.

The comparison of the normal pulse with that obtained after the production of aortic insufficiency shows the much greater systolic fall of the latter. Indeed, the main fall of pressure is composed of this declivity, for the amount of fall which is purely diastolic, viz., from the dicrotic notch to the abscissa, is almost negligible, as compared to that which occurs in systole.

It must be conceded, then, that from the form of the pulse curve alone we have evidence that the essential feature of the collapsing pulse lies in the systolic phase of the cardiac cycle. The pulse differs in no way from that in which, with the maintenance of ventricular activity, there is a relaxation of the peripheral resistance.

EFFECT OF CHANGES IN HEART RATE IN AORTIC INSUFFICIENCY.

From the examination of the volume curves of the normal heart it was shown that an increase of rate up to a certain limit increased the amount of systolic output per unit of time. The action is due to the shortening of the period of diastasis, which allows of an increase of heart rate, without interfering with the ventricular filling. Erlanger²⁸ and Hooker observed that an increase in the quantity of blood thrown out in a given time will increase the mean and therefore the diastolic pressure in the arterial system. In aortic insufficiency an increase in the diastolic blood pressure must increase the pressure which is transmitted to the ventricular walls during diastole. Any increase of rate, however, beyond that which abolishes the period of diastasis occurs at the expense of diastole, in Henderson's sense (i. e., time of filling), for each succeeding systole will take place before the normal relaxation of the ventricle is completed. The blood thrown out during systole must, then, be diminished, and while the heart beats more rapidly, the output in a given time does not vary. The diastolic pressure will, therefore, undergo no further augmentation.

Disregarding for the time being the possible influence which a lengthening of the period of diastole and diastasis may have on regurgitation, it is evident that in so far as aortic pressure is affected by changes in rate a slowing of the pulse within normal limits will not affect the pressure which can be transmitted to the ventricle, whereas an undue lengthening will lower it. Regarded in this light, an unusually slow heart rate is advantageous in aortic insufficiency.

This effect of rate, however, becomes secondary to that which it has on regurgitation and transmission of pressure. It is usually taught that a lengthening of the period of diastole is disadvantageous in that it increases the leak into the ventricle and causes it to dilate. This is clearly based on the conception that an "appreciable" quantity of blood returns to the ventricle. It has been shown in the plethysmographic records that the volume of the blood which regurgitates in experimental insufficiency is negligible, and that the phenomena associated with the

28. Johns Hopkins Hosp. Rep., 1904, xii, 160.

condition are due to the attendant transmission of pressure. An estimation, then, of the rate at which this pressure is transmitted will give the desired information as to whether a lengthening of the period of diastole and diastasis is harmful or advantageous. For, clearly, if the pressure in the ventricle equals that in the aorta at the end of ventricular relaxation, it is immaterial how long the period of diastasis may be, since it will involve no further increase of the intraventricular pressure. If the pressure, on the other hand, is transmitted slowly from the aorta to the ventricle; that is, if the intraventricular pressure rises gradually during the whole period of diastole and diastasis and equals the aortic pressure just before the following systole, then a lengthening of the period of diastasis will have a harmful effect.

In order to determine which of these conditions holds in aortic insufficiency the curve of intraventricular pressure was recorded in three experiments.

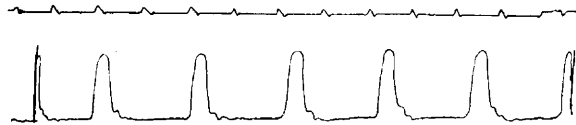


Fig. 23.—Intraventricular pressure tracing from the normal ventricle. Contrast with tracing No. 24.

Figure 24 is the intraventricular pressure curve after the posterior aortic cusp was divided. It shows a somewhat oblique upstroke, which corresponds closely to the protracted volume curve of systolic discharge. The systolic plateau is rounded and merges insensibly into the diastolic downfall. The fall in pressure is at first rapid, but it becomes more gradual as it approaches the abscissa. This latter point is of great importance, for it stands in marked contrast with the abrupt and uniform fall in pressure which is seen in the intraventricular pressure curve of the normal heart, as in Figure 23.

It will be recalled that the plethysmographic records showed that the effect of aortic insufficiency was to increase the tonus of the ventricle, and, as a consequence, its distension during diastole was less than in the normal heart. It was shown also that the increased rapidity of filling took place only during the first fifth ventricular relaxation. This of itself would indicate that at the end of this period, viz., the first fifth of relaxation, a certain amount of aortic pressure had already been transmitted to the ventricle, and its further relaxation was effected with an increasing tension on its walls. As is well known, the pressure in

the normal ventricle falls below atmospheric pressure immediately after the ventricle begins to relax, as a consequence of the suction action exerted by its walls. If, as in aortic insufficiency, a positive pressure is being transmitted at the same time from the aorta, it should follow that this fall in pressure will become less rapid, and the downstroke of the intraventricular curve more inclined. That such is really the case, tracing No. 24 proves.

On the one hand, the relaxation of the ventricle tends to produce a diminution of the pressure within it, while, on the other hand, the free communication with the aorta will permit of the transmission of positive pressure. The excess (algebraically) of the negative over the positive will occasion a fall of the intraventricular pressure curve, which will be slow or rapid according as the excess is small or great. In other words, if the aortic diastolic pressure is high, and the communication between it and the ventricle large, the fall in pressure in the latter, during its relaxation, will be more gradual than if the leak were small or absent. Further, it is evident that the fall will be more rapid at the begin-

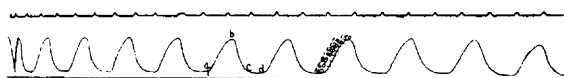


Fig. 24.—Intraventricular pressure curve after production of aortic insufficiency. The upstroke a-b is more gradual, as is also the fall in pressure b-c. The line c-d is horizontal, showing that the intraventricular pressure does not rise during the period of diastasis. The figures represent the pressure in mm. of Hg. at different points of the curve.

ning of the process of equalization than near its termination, since then the negative relaxation pressure in the ventricle is greatest. The rate of fall, therefore, is seen to be more gradual as the downstroke reaches the abscissa, for the negative pressure is being gradually neutralized by the positive.

The most striking feature, however, of the curve of intraventricular pressure lies in the fact that the downstroke does not reach the abscissa at any stage of the diastolic phase. Following the gradual fall of pressure which accompanies the completion of ventricular relaxation, the lever traces a line which runs parallel to the abscissa, and in this instance indicates a pressure of 40 mm. Hg. But the rapidity with which the aortic pressure is transmitted to the ventricle varies directly with the size of the communication. It follows, therefore, that when the insufficiency is extensive, the pressures in the aorta and ventricle become equalized during the period of relaxation, and there is no further transmission in the phase of ventricular diastasis. This proves conclusively

that, under such circumstances, if the tonus of the ventricle is maintained a slowing of the heart rate can not have a harmful influence in aortic insufficiency.

It is possible, however, in those cases in which the opening between the aorta and ventricle is small that the delay in the transmission of aortic pressure which results may cause the pressure in the ventricle to rise during the whole period of diastole. The smallness of the communication probably accounts for the rise of diastolic pressure seen in the tracings of Marey.²⁹

He produced an aortic insufficiency in the horse by the passage of a cardiac sound which merely punctured an aortic cusp. An insufficiency thus produced is much smaller than that which follows section of the cusp. Further, it was noticed in the present series of experiments that an opening in a valve produced by the cardiac sound becomes blocked

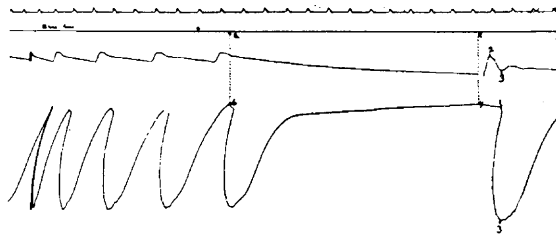


Fig. 25.—Carotid and ventricular volume tracings from the normal heart, showing the effect of vagal stimulation. The difference between the lines a-b and x-y represents the distension of the ventricle due to loss of tone = 1 c.c. of blood. Stimulation of the nerve slows the heart rate by prolonging the period of diastasis. The rapidity of ventricular filling is not affected. The marks on the base line indicate the period of stimulation.

rapidly by the formation of a fibrin clot. In one such experiment a clot had formed in an hour, and many of the features of this condition became distinctly less marked.

A gradual rise of pressure, then, is possible when the communication is small, and only under such circumstances can a slowing of the pulse rate, *per se*, be said to have a harmful influence on the ventricle. A similar conclusion as to the effect of rate is arrived at from the results obtained by stimulation of the peripheral end of the vagus nerve.

EFFECT OF VAGUS STIMULATION.

The effect of the stimulation of this nerve has purposely been omitted from the discussion of the relation of heart rate to aortic in-

29. Loc cit., 674.

sufficiency, since stimulation of the vagus gives rise to two (among several) distinct physiologic changes in the ventricle.

As is well known, when the peripheral end of the vagus is stimulated, in addition to slowing of the ventricular contraction, it diminishes ventricular tone. The two effects can not be disassociated, and, while at first we would appear to have a ready means of studying the effect of rate on regurgitation, we are immediately confronted with the difficulty that arises as a result of the diminution of tone, simultaneously induced.

When the vagus is stimulated the volume curve of the normal heart shows that the ventricle becomes gradually distended (Fig. 25). This follows from the fact that the auricular pressure, although small, is sufficient to overcome the diminished tone of the ventricular walls.

If after aortic insufficiency has been produced the nerve is stimulated with the same strength of current and for the same length of time, any distension which may take place beyond that which occurs normally

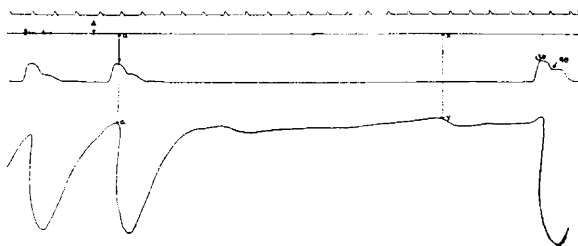


Fig. 26.—Carotid and ventricular volume tracings after the production of aortic insufficiency. The vagus nerve was stimulated at A and the loss of tone causes the ventricle to dilate. The amount of distension produced (difference between a-b and x-y) is the same as in the normal heart—1 c.c. in each. The slowing of the heart rate, therefore, can not allow of regurgitation of blood.

under vagus stimulation will result from regurgitation. In this manner only is it possible to draw any accurate conclusions as to the effect produced by slowing the heart rate experimentally by vagus stimulation.

In order to confirm the findings obtained by the records of the intra-ventricular pressure curves, two such tracings are reproduced. In the normal, Figure 25, the effect is seen to involve only the period of diastasis. The vagus standstill allows the completion of diastole, which is completed in the usual time, in this case .272 of a second. The line of prolonged diastasis inclines gradually upward, and indicates a distension of the ventricle from loss of tone.

Practically the same thing is seen to occur in tracing No. 26, where the aortic valves have been damaged and the distension which results

is no greater than that which occurs in the normal heart, 1 c.c. of blood in each.

Before stimulation the tone of the heart whose valves were damaged was greater than that of the normal, in consequence of the mechanical stimulation of increased intraventricular pressure. During stimulation the tone is reduced in both in the same degree, and a correspondingly equal distension is produced in the two states.

It is interesting to note that even after vagus stimulation the tone of the ventricle, when the aortic valves are damaged, as measured by the distance of the volume curve just before systole from the base line, is equal to that which obtains normally in the heart with intact valves.

This experiment has a twofold bearing. Taken in conjunction with the intraventricular pressure curves, it proves beyond doubt the relative unimportance of heart rate, *per se*, while, on the other hand, it emphasizes the important rôle which the ventricular tonus plays in the prevention of extreme regurgitation.

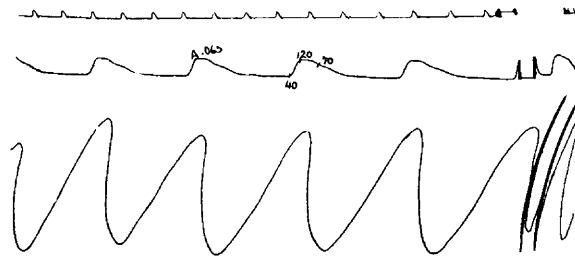


Fig. 27.—Carotid and ventricular volume curve in aortic insufficiency before compression of the abdominal aorta. A, summit of the primary wave. It is reached in .065 of a second after the commencement of systole. Systolic pressure, 120 mm. of Hg.; pressure at beginning of diastole, 70 mm. of Hg.; pressure at the end of diastole, 40 mm. of Hg.; systolic output, 12 c.c. (Compare with tracing No. 28.)

EFFECT OF INCREASING PERIPHERAL RESISTANCE.

The examination of the pulse of aortic insufficiency, and the similarity between the action of a true rupture of the aortic valves and an irritation of the ventricular wall in lowering blood pressure, have suggested the importance of a vaso-dilatation as the important factor in the causation of the collapsing pulse. It becomes necessary, then, to test experimentally the effect of changes in the peripheral resistance.

There are three methods by which it is possible to obtain varying degrees of increased resistance: (1) By stimulation of the splanchnic nerves; (2) by the injection of adrenalin; (3) by compressing the abdominal aorta.

While the first method in general is the most satisfactory, it was found that under the circumstances in which the records were obtained in this research it was impracticable. The dissection necessary to stimulate these nerves, following an already extensive cutting operation on the thorax and the production of a valvular lesion, was more than the animal was able to stand. It was necessary, then, to employ only the methods of abdominal compression and adrenalin injection.

If one accepts the dictum of Corrigan, that the collapsing pulse is due to the regurgitation of blood from the aorta, then any condition which retards the forward flow of blood must, of necessity, increase regurgitation, and thus render more pronounced the collapse of the pulse.

That the collapse is not increased by such a means, but entirely abolished, a glance at tracings Nos. 27 and 28 will show. They repre-

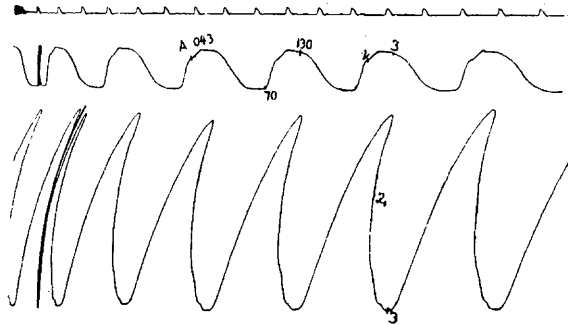


Fig. 28.—Carotid and ventricular volume curve of aortic insufficiency during compression of abdominal aorta. Note the change in the character of the pulse. A, summit of primary wave, is reached in .043 of a second after the commencement of systole. Systolic pressure, 130 mm. of Hg.; pressure at end of systole, 130 mm. of Hg.; pressure at end of diastole, 70 mm. of Hg.; systolic output, 13.5 c.c.

sent the changes in pulse form and volume curve which follow compression of the aorta at its bifurcation.

Change in the Pulse Form.—These curves show in a striking manner the truth of the statement of Marey regarding the conditions which modify the systolic portion of the pulse. There being a great disparity between the rate of inflow and the rate of outflow, the summit of the primary wave A is reached in a relatively shorter period during compression of the aorta. When the flow was unrestricted the time taken by the primary wave to attain its maximum was .065 second, while now it is reached in .043 second. The “systolic plateau,” for the same reason, becomes ascending, and the dicrotic notch occupies the summit of the

curve. It will be seen to coincide exactly with the end of systole, as measured by the plethysmograph, .01 second in each.

The fall of pressure is now entirely diastolic, since the systolic plateau does not descend. The pressure which exists in the aorta at this moment is 130 mm. Hg., as contrasted with 70 mm. Hg. in the corresponding part of the ordinary curve of aortic insufficiency. There is thus established the ideal condition for regurgitation. Notwithstanding that the diastolic fall of pressure is more gradual than it is even in the normal tracing with intact valves, and only reaches the abscissa .06 second before the succeeding systole; *or*, in other words, the *conditions most favoring regurgitation caused the cessation of the collapsing quality in the pulse.*

Change in the Volume Curve.—The effect on the systolic contraction is merely an exaggeration of that which occurs in the ordinary state of insufficiency. The tension transmitted to the ventricular walls before contracting is greater, and the “loading” effect is more pronounced. The systolic contraction is still more protracted and the rate of systolic discharge slowed. It will be seen from the record that the diminished rate of output is more marked toward the end of systole, the tracing becoming more oblique as the apex is approached.

There is, however, this distinction. The tension is now so great that the tone of the ventricular wall is no longer able to counteract it, and a considerable regurgitation results. The distance from the base line at the end of diastole is shortened.

Further, so great is the resistance to be overcome in the aorta during the latter part of systole that the ventricle fails to effect so complete an emptying of its cavity. The difference in the quantity of residual blood is small, however, and does not amount to more than 2 c.c.

The record shows conclusively that regurgitation can not account for the collapsing pulse. Even with the increased systolic output and the greater pulse pressure, due to regurgitation, when the aorta is compressed, an increase of the resistance to onward flow of blood abolishes its collapsing character, and, conversely, a diminution of the resistance will exaggerate it, although this can not be tested experimentally, when the aortic valves are damaged, as the diastolic pressure is already reduced to a minimum. It is easy, however, to follow the successive changes in this type of pulse which result from the gradual relaxation of an increased resistance.

The position assumed by the dicrotic notch becomes gradually lower on the descending limbs of the pulse curve and the systolic plateau becomes first horizontal and then descending. This indicates a gradual

lowering of the pressure, which is transmitted to the ventricles at the commencement of their relaxation, since it is the pressure which exists in the aorta at the moment of closure of the aortic valves.

An increase of the rapidity of capillary escape, then, may be said to have a preservative influence on both the heart and the arterial system. It diminishes the magnitude of the pressure at the end of systole, and, therefore, prevents the transmission of high pressure to the ventricular walls during their relaxation. It also slows the rate of rise of the primary wave, and thus counteracts the tendency to dilatation of the arteries, as would be caused by the sudden stretching attending the passage of a steep wave of pressure.

The injection of adrenalin effects a change in the pulse curve which is identical with that of compression of the abdominal aorta. It raises the aortic pressure at the commencement of diastole, and, therefore, dis-

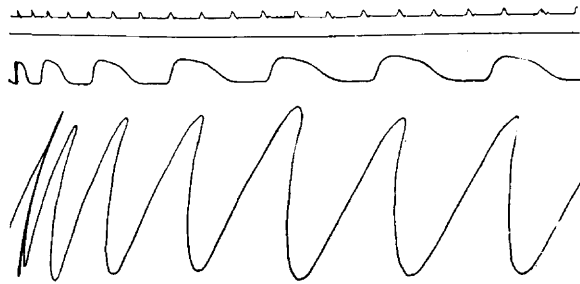


Fig. 29.—Carotid and ventricular volume curve of aortic insufficiency to show the effect of the injection of adrenalin. It is similar to that produced by compression of the abdominal aorta. Compare with the ordinary tracing of aortic insufficiency No. 27.

tends the ventricle by the regurgitation of blood. But the contraction of the peripheral arterioles retards the escape of blood through the capillaries, and the pulse ceases to be collapsing. On account of the stimulating action which it exercises on the ventricular contraction, the systole is more complete, and the effect it produces on the volume curve differs from that of abdominal compression in that the quantity of residual blood is diminished. Tracing No. 29 was taken on a fast drum when the drug had produced its maximum effect.

CLINICAL APPLICATION OF EXPERIMENTAL RESULTS.

The most important points which have been brought out from the experimental investigation of the pulse and blood pressure changes in aortic insufficiency are briefly:

1. That the so-called water-hammer pulse owes its abruptness and rapid fall not to regurgitation into the ventricle, but to an increased blood flow through the capillaries.

2. That this increased capillary flow is due, in a recent insufficiency, to reflex inhibition of the vasomotor center, probably induced by stimulation of the depressor nerve effected by the mechanical action of the pressure transmitted from the aorta.

3. This pressure simultaneously increases the ventricular tone.

4. That so long as the tonus is maintained a slowing of the pulse can not have any appreciably harmful influence on the amount of blood which regurgitates.

It becomes necessary, then, to apply these results to the aortic insufficiency which is due to disease, and to inquire as to how far they will help to explain the clinical discrepancies previously mentioned.

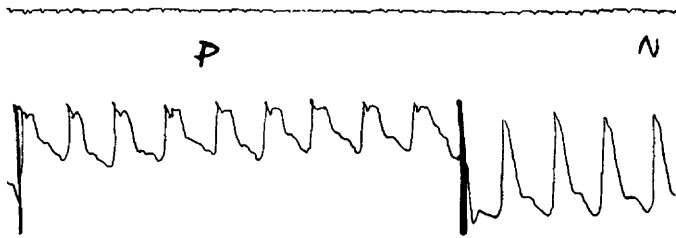


Fig. 30.—Tracing from the radial artery to show the effect of terminal constriction. P, during constriction. N, Tourniquet removed.

It is obvious that if the main fall of pressure is due to an increased rate of capillary flow it will follow that any condition which retards it should cause the disappearance of the collapse. This can readily be accomplished in the case of the radial artery by constricting the terminal vessels of the hand by the application of an elastic tourniquet. If a sphygmographic tracing be taken from a typical case of aortic insufficiency in the manner recommended by Lewis,³⁰ with weight instead of band fixation of the sphygmograph, the effect of decreasing the out-flow from the artery will at once become apparent.

Such a tracing before and during terminal constriction is shown in Figure 30. The patient suffered from pure uncomplicated aortic insufficiency. He had a systolic blood pressure of 130 mm. Hg., and a diastolic blood pressure of 65 mm. Hg. and subjectively a typically collapsing pulse. The instrument used was a Jacquet chronosphygmograph. This instrument being a heavy one, it sufficed only to lay it over the artery

30. Jour. Physiol., Lond., 1906, xxxiv, 393.

without encircling the wrist with the usual sphygmograph band. The tourniquet was applied below the level of the thumb, well away from the sphygmograph, so that on being tightened the tracing could not be affected by the wrinkling of the skin. The contrast is striking. Before the terminal vessels are constricted the amplitude of the pulse is great, the apex of the wave pointed, and the systolic plateau is a steep decline. During constriction, on the other hand, the amplitude becomes small and the trough of the waves does not reach the abscissa, thus indicating an artery which does not empty itself. The systolic plateau is more horizontal, and the dicrotic notch assumes relatively and absolutely a higher position on the tracing. Indeed, the tracing so transformed is the counterpart in every detail of the tracings of aortic insufficiency which Sansom describes as "having a resemblance with the tracings indicating high tension."

A similar result, although in a totally different manner, has been obtained by diminishing the rapidity of capillary flow by the aid of venous constriction. As has been pointed out by Bayliss and Starling,³¹



Fig. 31.—Series of tracings of the brachial artery taken with varying degrees of venous obstruction. When the pressure in the cuff is lower than the diastolic pressure the arterial inflow to the limb is not affected, while the venous return is restricted. The numbers indicate the different pressures in the cuff.

the capillary flow and pressure are greatly influenced by changes in the venous pressure, the flow being greatest when the pressure is least. If, by constricting the limb with a cuff bearing a pressure less than the diastolic blood pressure, the free inflow to the limb is not retarded, any desired amount of venous restriction may be obtained. A tracing, No. 31, from the brachial artery under such circumstances, is shown above. It will be seen that the collapse disappears and the catacrotic limb of the pulse curve becomes gradual in its fall.

So strong has been the stimulus of a dominant and preconceived idea that clinicians have sought to extend this idea into the explanation of many of the other attendant features of aortic insufficiency. Thus it is universally held that the reason why the collapse is more marked when the limb is elevated is due to the greater ease with which the blood can flow back into the ventricle. Apart from the physical impos-

31. *Jour. Physiol., Lond.*, 1894, xvi, 157.

sibility of there being a retrograde blood current where there is a driving strength of even 50 mm. Hg., as indicated by the diastolic pressure, this view can readily be controverted by a simple experiment.

If, while the limb is elevated, the upper arm is encircled in a sphygmomanometer cuff, and the pressure raised to a point below diastolic pressure, so as to obstruct the venous return only, the intensified sensation of the collapse is now no longer felt. This experiment, while disproving the theory that the collapse is due to regurgitation, at the same time renders it evident that the elevation of the limb increases the sensation of the collapsing pulse, not by increasing backflow, but because it increases the capillary flow by hastening the venous return.

But the capillary pulsation so common in this condition is also ascribed directly to regurgitation. It is said that it is due to the empty condition of the peripheral vessels in diastole which results from regurgitation into the ventricle. In view of what has been said above, it is only necessary to quote an observation of Gibson's³² while commenting

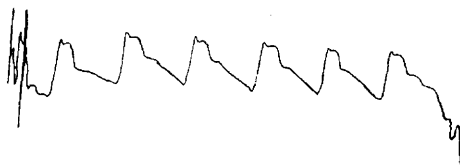


Fig. 32.—Apparently normal tracing from a case of aortic insufficiency which gave subjectively a collapsing pulse. The effect of an increase of systolic pressure is seen in tracing No. 34.

on this phenomenon: "It must be allowed that it may occur in conditions permitting a free passage of blood through the arterioles and capillaries into the veins." He further states that a direct venous pulse may develop in the peripheral veins, the pulsation following systole and passing from the periphery to the center.

Granted, then, that the collapsing pulse has its causation in the periphery and not in the center, the explanation of the clinical discrepancies previously mentioned becomes at once apparent. The collapsing pulse can exist only when the diastolic blood pressure is low, since when the systolic pressure remains constant the diastolic pressure is regulated by the rapidity of flow from the peripheral vessels. In other words, it is possible to receive the impression of a collapsing pulse only when the arterial walls are subjected to a slight distending force during the diastolic phase of the cardiac cycle.

32. *Loc cit.*, p. 493.

It has, however, been frequently observed in those cases in which the diastolic pressure is low that when the systolic pressure is also low the pulse may give to the finger the impression of a sudden decline, while the sphygmograph tracing may show a more or less sustained pulse wave.

Tracing No. 32 is taken from such a case in which, with a systolic pressure of 75 mm. Hg. and a diastolic of 55 mm. Hg., the pulse felt typically collapsing. The patient, aged 20, suffered from aortic insufficiency, the result of acute rheumatism.

This discrepancy has its explanation in the fact that the pulse is not felt during the whole period of time in which the systolic wave is passing, and it is only that part of it which corresponds to the summit of the pulse wave that impresses itself on the finger. In order that a low pressure pulse, in which the pulse amplitude is small, may be palpated satisfactorily, the finger must be allowed to rest but lightly over the

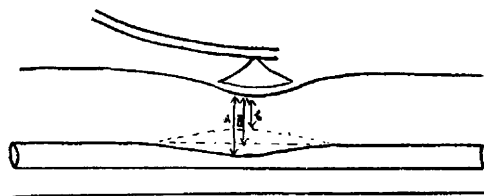


Fig. 33.—Diagram to illustrate the effect which different degrees of pressure exerted by the sphygmograph button will have on the excursion of the arterial wall during the passage of the pulse wave.

artery. There is, therefore, communicated to it only that part of the pulse which arises from the excursion of the arterial wall beyond its diastolic limit, i. e., the apex of the pulse wave. This is further evidenced by the fact long recognized by clinicians that if the wrist or forearm of a patient suffering from this disease be grasped lightly by the hand the water-hammer quality is much intensified. Under such circumstances the vessel, distended by the wave produced by the ventricular systole, is felt only at the summit of its distension. When, however, the sphygmograph records the largest amplitude of wave for a given pulse the pressure which is exerted by its pad is approximately equal to the pressure which obtains within the vessel.

During the interval between the pulse waves the arterial walls are somewhat approximated, as in Figure 33, and in this condition the systole of the ventricle will cause them to execute a greater movement. But, owing to the low diastolic pressure and to the fact that the pulse amplitude is small, the arterial wall is stretched but slightly. The arterial

recoil, therefore, will not be forcible, and it will return gradually to the position assumed during diastole.

The low systolic pressure in the patient mentioned above being an indication of a feebly contracting left ventricle, the effect of treatment was to raise it from 75 mm. Hg. to 105 mm. Hg., while the diastolic pressure remained as before, viz., 50 mm. Hg. The change resulting in the sphygmographic tracing is interesting, for it presented a fairly typical collapsing pulse, while at the same time it gave the collapsing sensation to the finger. This change in the character of the tracing can readily be explained by the greater stretching force to which the artery was subjected during the passage of the primary wave. Haycraft³³ has recently shown that within wide limits the arterial walls obey Hooke's law, which means that the amount of extension is proportional to the force which produces it. It follows, then, that the more the vessel is distended the more rapid will be the rate of recoil. It thus becomes evident how it is that the finger can always receive the sensation of a

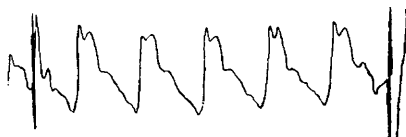


Fig. 34.—To show the effect of an increase in systolic pressure. The pulse is more collapsing.

collapsing pulse when the diastolic blood pressure is low, while it is only in those cases in which the pulse pressure is excessive that it can with certainty be recorded by the sphygmograph.

It is also easy to explain the absence of the collapsing pulse in the percentage of cases previously mentioned. These cases must have presented high diastolic pressures, although they are not recorded, since it is only within the last two years that we have been in a position to measure the diastolic pressure clinically. The high diastolic pressure in the majority of these cases was probably the result of the accompanying arteriosclerosis which most of the cases presented. It is to be noted, also, that the absence of the collapsing pulse need not be due to a small pulse pressure, i. e., the difference between systolic and diastolic pressure, or, conversely, a large pulse pressure need not necessarily be accompanied by a collapsing pulse.

A collapsing pulse can only be seen in those cases in which the diastolic blood pressure is low. The fact that a high diastolic blood pressure should cause the sphygmograph tracing to assume a gradually sloping

33. Jour. of Physiol., Lond., xxxi, 392.

line, even when there is a great range between the systolic and diastolic pressures, is due to a condition depending on the physical property of the arterial wall. Roy³⁴ showed that while Hooke's law is obeyed when the pressures are normal, when they are exceeded the extensibility of the artery rapidly diminishes.

From the figures given by Roy of the extensibility of the arterial walls at different pressures two pulse curves are drawn (Fig. 35).

In both instances the excess of systolic over diastolic pressure is the same, viz., 60 mm. Hg., but they differ in that the one, the diastolic pressure, is below normal, viz., 50 mm. Hg., while in the other it is somewhat above normal, viz., 80 mm. Hg. In the lower curve the amplitude is double that in the upper, the angle between the upstroke and downstroke is more acute and the dicrotic notch relatively lower. In the upper curve

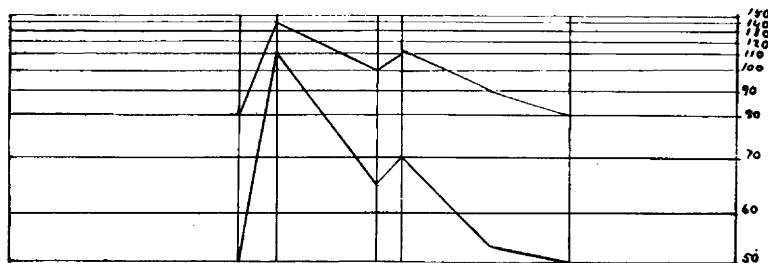


Fig. 35.—Diagrammatic representation of two pulse curves indicating the same pulse pressure, but at different diastolic pressures. The horizontal lines indicate the extensibility of the artery at given pressures (after Lewis).

upstroke and downstroke are oblique and the angle between them is widened.

In the curve taken at the low pressure there is more movement of the arterial wall for the same change of pressure. The one would be classified as a "collapsing" pulse, the other as "sustained."

THE LOW PERIPHERAL RESISTANCE IN AORTIC INSUFFICIENCY.

Starling²¹ has pointed out that in disturbed conditions of the circulation the reflex mechanism called into play aims more at sparing the heart from overexertion than in counteracting the original vascular disorder. So in aortic insufficiency the function of a reflex dilatation of the peripheral vessels is a conservative one, being the method adopted by Nature to relieve the heart of the increased work thrown on it as a consequence of the transmission of pressure from aorta. This reflex action, however, is wholly distinct from that which occurs in old standing

34. Jour. of Physiol., Lond., 1881, iii, 135.

cases, which are due to disease, for now there are produced in the arterial system conditions which obviate the necessity of a continuous action on the part of the vaso-dilators.

The great changes in pressure between systole and diastole have the effect of stretching and widening the coats of the peripheral arterioles so that they remain permanently dilated. This dilation subserves the function of the reflex inhibition of the vasomotor center, which takes place after an experimental insufficiency. It was shown by Romberg and Hasenfeld¹⁰ that several months after an insufficiency had been produced in rabbits the vasomotor nerves were less active than normal and the same degree of peripheral constriction could not be effected by their stimulation.

The question, then, immediately arises why the ventricle becomes hypertrophied and dilated to such an extraordinary extent, if an appreciable quantity of blood does not regurgitate, and the increased work thrown on the heart can be greatly obviated by a peripheral dilatation.

In order to make the point clear, certain properties of the contractility of muscle must be mentioned briefly. Haidenhain³⁵ has shown that when the tension on a skeletal muscle is increased before it contracts, the energy of the contraction becomes greatly augmented. The apex of a frog's heart ceases to beat under ordinary circumstances, but it can be made to contract by distending the cavity of the ventricle with fluid. Gaskell³⁶ also showed that after the apex of the frog's heart is rendered quiescent by crushing a ring of tissue above it, it can be made to contract again by clamping the aorta.

The excitory effect of increased tension in both cardiac and skeletal muscle is thus firmly established. Moreover, this mechanical effect involves a great expenditure of cardiac energy, for it is accompanied by an augmentation of all the chemical processes which accompany contraction, as is evidenced by the increase in heat production. In aortic insufficiency, instead of a pressure equal only to the atmospheric pressure as in the normal ventricle, the intraventricular pressure tracings show that there is a pressure on the ventricular walls during diastole, equal to the diastolic pressure in the aorta. The ventricle is thus locally stimulated to increased exertion and its contraction becomes similar to that in which a muscle is extended before it contracts. So long as the heart is able to supply the increased demands thus made on its energy there may neither be hypertrophy nor dilatation, and we have the authority of Krehl³⁷ that every case of aortic insufficiency is not accompanied

35. Quot. by Gaskell, Shaefer's Text-book of Physiol.

36. Schaefer's Physiol., vol. ii, 170.

37. Clin. Path., trans. Hewlett, p. 38.

by an enlargement of the left ventricle. When, however, the cardiac muscle becomes inefficient it will give way to the stretching influences which bear on it during diastole, and the cavity of the ventricle will enlarge. This is immediately followed by hypertrophy. But in so doing the increased work which the heart now accomplishes, as a consequence of the greater systolic output, has the effect of raising the mean pressure in the arterial system. There is thus established a condition in which there is a greater transmission of pressure and a still greater expenditure of energy. The condition is a progressive one, for the greater the hypertrophy and dilatation the greater the transmission of pressure and consequently a greater waste of cardiac energy.

It is thus evident how potent a factor is a low peripheral resistance in the preservation of the heart in aortic insufficiency. It not only lessens the tendency toward the regurgitation of blood, but it also reduces

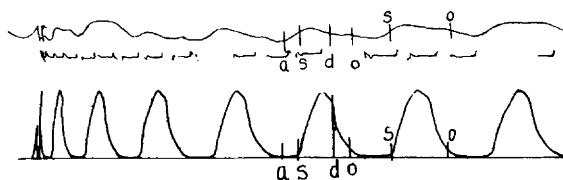


Fig. 36.—Simultaneous tracing from the left auricle and the left ventricle after the production of aortic insufficiency. a, auricular systole; s, ventricular systole; d, ventricular relaxation; o, opening of the mitral valves. There is a fall in the auricular pressure during the contraction of the ventricle. The valves must, therefore, be competent.

the influence of the increased tension on the ventricle as a stimulating agent.

It may not be out of place to mention here the frequency with which a transient mitral insufficiency may develop in the course of an experiment in which the aortic valves have been damaged.

A simultaneous tracing of the intraauricular and intraventricular pressures gives a ready means of detecting such incompetency of the mitral valves, for instead of the normal fall in pressure, which occurs in the auricle during ventricular systole, there is a systolic rise of pressure when an insufficiency develops. Such is seen to take place in tracings Nos. 36, 37 and 38. These three tracings were taken at different times during the course of one experiment.

In No. 36, there being a fall of pressure in the auricle during the contraction of the ventricle, s-d, the mitral valves are competent. In No. 37 there is a systolic rise of pressure, which is to be interpreted as indicating an inadequate closure of the auriculo-ventricular orifice.

Tracing No. 38 was taken an hour later, and it shows that the ventricle has recovered, for there is now a systolic fall in pressure in the auricles.

This effect is probably due to periodic changes in the ventricular tonus, which, when lowered, permits of ventricular dilatation, and this is followed by a relative insufficiency at the mitral opening. This view is supported by the result obtained by stimulating the peripheral end of the vagus, which produces a marked diminution in the ventricular tone.

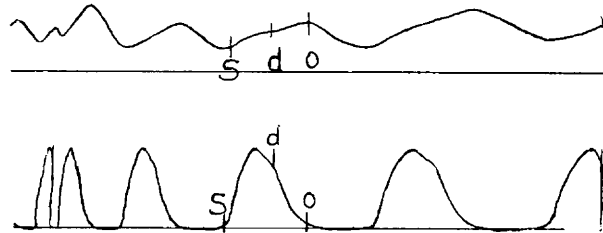


Fig. 37.—Same as No. 36, but there is a rise in auricular pressure during the contraction of the ventricle, s-d. The valves are incompetent.

Tracing No. 39 shows the effect of such stimulation. It will be seen that immediately before stimulation the ventricular systole, s-d, is accompanied by a slight fall in auricular pressure (the valves are competent), while during stimulation there is a marked rise in pressure (an insufficiency has developed). O marks the commencement of the fall of auricular pressure and, therefore, the time of opening of the mitral valves.



Fig. 38.—Same as No. 37, one hour later. The mitral insufficiency is no longer present, for now the pressure in the auricle falls during ventricular systole, s-d. Lettering as above.

It is clear that the production of a relative insufficiency at the mitral orifice, in addition to an aortic lesion, may, to a great extent, be avoided by lowering the diastolic blood pressure. For, when the diastolic pressure is sufficiently low, the ventricular tone is able to overcome it and no dilatation results.

The conclusion, then, which is to be drawn from the experimental and clinical study of this disease is that the condition which is most favorable in a patient suffering from aortic insufficiency is a low diastolic blood pressure. This is supported by the fact, long recognized by clinicians, that the outlook in a case of aortic insufficiency resulting from rheumatic endocarditis is more favorable than one in which the lesion has been the result of arteriosclerosis (Osler³⁸). The reason is sufficiently obvious, for in the former the diastolic pressure is low, while in the latter, as a consequence of the rigid arteries, the diastolic blood pressure is high.

Finally, this is not without a bearing on the question of the use of digitalis in this disease. It has been contended that this drug is contra-indicated because by prolonging ventricular diastole it favors increased



Fig. 39.—Simultaneous tracing from the left auricle and left ventricle after production of aortic insufficiency to show the effect of vagal stimulation. The diminution in tone causes a dilatation of the ventricle and a relative insufficiency at the mitral orifice. Before stimulation the pressure in the auricle falls during the ventricular systole, while during stimulation there is a rise of pressure from s-d.

regurgitation. But it has been proved already that a mere lengthening of the period of diastasis is not harmful, if there be no diminution of the ventricular tone, since the pressures in the ventricle and aorta are equalized as soon as the ventricular filling is completed. The harmful influence of digitalis can not result from its action in slowing the heart rate. It is due to the action which the drug possesses on the peripheral arterioles. As is well known, it has a powerful constricting action on these vessels, and thus, by raising the diastolic pressure, is its effect deleterious. Theoretically, strophanthus should be a more useful drug, since, as shown by Fraser,³⁹ it does not possess this constricting quality to the same degree.

38. *Princ. and Pract. of Med.*, 1898, p. 517.

39. *Action and Uses of Digitalis and Its Substitutes, with Special Reference to Strophanthus*. Read before Brit. Med. Assoc., Cardiff, 1885, p. 15.

SUMMARY OF CHIEF CONCLUSIONS.

1. The work of Henderson is confirmed in that the cardiac cycle is not diphasic but triphasic, and consists of systole, the period of the ventricular discharge; diastole, the period of ventricular relaxation and filling; diastasis, the period of rest.

2. The effect of the production of aortic insufficiency in the dog is to increase the amount of systolic output by only a fraction of a cubic centimeter of blood.

3. The volume of blood which regurgitates is negligible.

4. The transmission of pressure to the ventricle increases the ventricular tonus.

5. It also produces a reflex inhibition of the vasomotor center.

6. The fall of pressure in aortic insufficiency is due to the diminished peripheral resistance thus induced, and is not caused by loss of blood from regurgitation.

7. The increase of pulse pressure—the difference between maximum and minimum pressure—is due to a lowering of the diastolic pressure. There is no increase in the systolic pressure.

8. The main fall in pressure is systolic in time and is due to an increased blood flow through the capillaries.

9. So long as the tonus of the ventricle is maintained, a slowing of the heart rate does not favor increased regurgitation.

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