

THE DIASTOLIC FILLING OF THE MAMMALIAN HEART. BY HERMANN STRAUB, DR. MED., *Stuttgart.*

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EXPERIMENTS on the volume of the heart have been carried out by several investigators, e.g. Johansson and Tigerstedt¹, Stefani², and François-Franck³. In 1906 Henderson⁴ published a paper on the subject in which he gives a critical account of previous experiments as well as a description of his own investigations. His statements may be briefly summarised as follows.

During the period of discharge the volume curve of the ventricles shows a sudden and steep descent, beginning with the moment of the opening of the semilunar valves and accounting for 90 per cent. of the total output from the ventricle. At the bottom the volume curve is rounded and comes to a blunt point, at the apex of which the semilunar valves close. For a brief period immediately following the completion of systole all the heart valves are probably closed. Yet during this period, according to Henderson, the volume curve rises sharply, a slight notch in the curve sometimes marking the end of this brief period. He attributes this initial increase in the bulk of the ventricles to the rush of blood from the aorta into the coronary vessels. During the period of filling of the ventricles the moment of opening of the auriculo-ventricular valves is marked by a rapid movement upwards of the volume curve. The line traced by the lever rises steeply and often nearly straight almost to its summit, then curves quickly and runs for some time *parallel to the abscissa*. Thus the filling of the ventricles occupies only the early part of diastole and is as rapid a process as the emptying in systole. According to this author, the volume curve during the latter part of diastole runs parallel to the abscissa, indicating that during this period no movement of blood occurs into the ventricles. Henderson suggests the name "diastasis" for this period, and states that differences in the rate of the heart beat depend almost entirely on lengthening or shortening of this period. In a very rapidly beating heart the period of diastasis entirely disappears, the volume curve becoming simply a series of sharp up and down strokes. He suggests that the contraction of the auricles increases the ventricular volume at the most to the extent of a few drops.

¹ Johansson and Tigerstedt. *Skand. Arch. f. Physiol.* i. 1889, p. 331, ii. 1891, p. 409.

² Stefani. *Arch. Ital. de Biol.* 18, 1893, p. 119.

³ François-Franck. *Arch. de Physiol.* 1890, p. 395.

⁴ Yandell Henderson. *Amer. Journ. of Physiol.* 16, 1906, p. 325.

The correct registration of the heart volume or ventricular volume no doubt affords a difficult problem. The changes are rapid and excessive. The acceleration of the moving parts of any registering instrument is therefore considerable, and the demands on its mechanical perfection unusually large. Moreover, the circulation through the heart is very easily influenced by the recorder unless very special care be taken.

It is therefore indispensable that the instrument used for registration of volume changes of the heart should be subjected to a rigorous criticism. At Professor Starling's suggestion I have undertaken this criticism, and have also made observations on the changes in the volume of the heart by a method which is, I think, more delicate than those previously used.

The degree of accuracy of previous methods.

The theory of registering instruments has been treated so exhaustively by Otto Frank¹ that there should be no difficulty in determining whether any given instrument is able to record correctly certain movements. Frank has shown that, for this purpose, it is necessary to determine three constants of the apparatus, namely, (1) a constant of mass (M), (2) a constant of damping (K), (3) a constant of elasticity (E). Both mass and friction contribute to the deformation of the registered curve, but since the latter can always be kept within such limits that its influence may be neglected, the constant of mass must be regarded as the more important. The relation of the mass-constant to the elasticity-constant is determined by a process called by Frank "dynamic standardisation," i.e., by registration of the oscillations of the registering system when it is set into movement apart from any movement at the point at which it is connected with the receiving apparatus. The time (T) of one oscillation of the system is given by the equation

$$T = 2\pi \sqrt{\frac{M}{E}}$$

In order to get a true record T must be smaller than the time of any of the waves to be registered. The ratio $\frac{1 \text{ second}}{T} = N$ is the number of oscillations per second.

To test the reliability of previous methods I submitted them to the 'dynamic standardisation.' I used a Marey tambour of the same

¹ O. Frank. *Ztschr. f. Biol.* 44, p. 445, 1903, *ibid.* 48, p. 489, 1906, *ibid.* 50, p. 309, 1908, *ibid.* 53, p. 429, 1910.

diameter as Henderson's, 12 cm. The ground plate of the stylus had a diameter of 9.5 cm. The tambour was connected with a light lever of straw. For the air transmission I applied a piece of rubber tubing similar to that used by Henderson. For a rough determination of the oscillations of this instrument it seemed to me to be sufficient to clamp the end of the rubber tubing and to cause the movements by sudden compression and relaxation of the tubing near its end. In this way I recorded the curve of Fig 1.

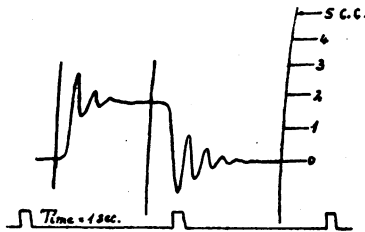


Fig. 1.

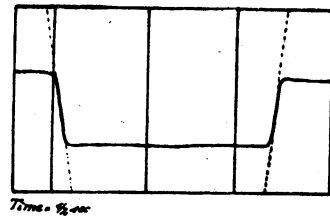


Fig. 2.

The instrument has a very slow period, its N is 9 oscillations per second. No doubt slight improvements are possible, but this N is far from the minimum wanted for our purpose. It might at first appear that Henderson's tambour had better qualities. He tested by means of a large syringe, and states that the stylus of the tambour followed without appreciable lag movements of the plunger of the syringe of considerably greater volume and rapidity than those occurring in the experiments. But every part of the registering system contributes to the oscillations according to its *mass*, more exactly

$$T = \sqrt{T_1^2 + T_2^2 + \dots T_n^2}.$$

By far the greatest mass of the whole system is represented by the lever, as Frank demonstrates. So a test of the instrument without the lever is of no use. As a matter of fact the mass of Henderson's apparatus is too great for his purposes.

Still the deformation due to instrumental inertia can be diminished or abolished by increasing the *damping*. Many details of Henderson's curves lead to the conjecture that he used such a method. But this affords a deformation of the curve as well as increasing the mass. The damping must be the smaller the weaker the moving forces are. For the given case this condition allows only a very slight damping. To

make the instrument aperiodic, even with a slight damping, there is only one way, that is to diminish the mass.

In the case of instruments for the registration of changes in volume the *elasticity* coefficient is of considerable importance. This coefficient determines the relation between the changes of pressure and the corresponding changes of volume: $E = \frac{\Delta p}{\Delta v}$. For pressure recording instruments it is necessary to make this value great; for volume registering instruments it must as nearly as possible equal 0, i.e., the instrument must follow the volume without any change in pressure, so that it works so far as possible isotonicly. The resistance to movement of an instrument for the registration of volume must be inconsiderable as compared with the forces acting at the connecting part of the system. In the case of the heart, for instance, this viscus must be able to expand with diastole and cause a corresponding movement of the recording instrument without the exertion of any pressure on the latter. If a rubber membrane be employed for recording the changes in volume this will be more stretched at the end of diastole, when the volume of the heart is at its maximum. At this moment, however, the pressure differences which cause the inflow of blood into the ventricle are very small, so that the amount of inflow is likely to be influenced by the slightest compression in consequence of the tension of the rubber membrane. It is true that by starting with a negative pressure inside the tambour one might avoid this disadvantage, but in this case the negative pressure would cause a suction action at some other point of the curve which would in itself modify the process of filling. Henderson pays some attention to this point and states that his tambour showed no rise of pressure for the volume changes in question. Yet to fulfil this condition strictly an extremely thin rubber membrane would be necessary, and such a membrane it would be hardly safe to use against the heavy weight of a lever *plus* the friction of the point against the blackened surface. On the other hand, a sufficiently elastic membrane presents the danger already discussed of an actual resistance by back pressure to the diastolic filling of the heart.

These considerations raise some doubt as to whether the method employed by Henderson is really adequate to give a true record of the changes in the heart volume. We have good reason to believe that the mass of the Marey's tambour used by him was too great and that therefore too much damping was required to make it aperiodic. Moreover, the elasticity of the rubber membrane must have altered to a certain

extent, at any rate, the mechanical conditions of the ventricular expansion, and tended to have hindered the inflow of blood towards the end of diastole. In addition to these possible errors it must be recalled that in Henderson's experiments the intracardiac pressure was recorded at the same time as the heart volume by means of a tube introduced into the left ventricle from the carotid artery. There was thus insufficiency of the aortic valves which in itself would represent an abnormal condition.

Observations.

An improvement of the method could only be made by diminishing the mass moved, which is almost entirely due to the mass of the lever. This can be easily effected by dispensing altogether with a lever and using the method of photographic registration. As will be shown later, the period of oscillation of the instrument can be diminished to such an extent that we may dispense with any other improvement such, e.g. as shortening the length of the tubing used for air transmission and increasing its diameter. By such methods one might get a Marey tambour sufficient for most problems of volume registration, but for the reasons mentioned above, I was anxious to diminish the elasticity of the membrane more than it is advisable to do with a rubber sheet.

(Elasticity always equals $\frac{\Delta p}{\Delta v}$.) A membrane which will keep its form in spite of its comparatively weak elastic force is afforded by the excessively thin wall of a soap bubble. The pressure inside the bubble will be extremely small and will not be appreciably altered by an increase in its volume. The internal pressure of a soap bubble is proportional to the surface tension and is inversely proportional to the radius, so that with a large radius it will be almost nothing and will not be practically altered for small changes of the radius caused by volume changes. A soap bubble has already been used by several previous investigators¹ for volume registration.

For the dynamic standardisation of the soap bubble I used the method described above for the Marey tambour. In recording the highest point of the soap bubble was photographed in the same way as later on for the experiments. A curve obtained is given in Fig. 2. The dotted tangent shows that the speed of the volume change is about the same as the greatest found later on during the experiments. The acceleration is even greater than any during the experiments. Though

¹ W. Straub. *Ztschr. f. exp. Pathol. u. Ther.* 1, p. 1.

there is damping only by the friction of the air transmission, which of course is very small, it is nevertheless sufficient to make the instrument just aperiodic for the acceleration required. By using a still greater acceleration I found a frequency of oscillation $N = 56$. This instrument seemed to me not to want further improvement.

The practical arrangement of the experiments was as follows. The experiments were performed on cats, anæsthetised with A. C. E. and kept under artificial respiration. The sternum was divided in the middle line and the mammary artery tied. The pericardium was opened near the apex of the heart and three cuts were made in it down to its attachment to the vessels. The cardiometer used was in principle the same as Henderson's, only the bulb was made of glass. A set of cardiometers of different sizes was at my disposal, so that a convenient shape could always be used. The window of the cardiometer was closed by a thin rubber membrane with a hole. The size of the hole is of the greatest importance to avoid leaking of the instrument as well as a compression of the atrio-ventricular groove, which would cause serious alteration of the blood-flow.* By a rubber tube the cardiometer was connected with a glass funnel. I used a set of funnels of different sizes according to the volume changes of the heart. For the cat I generally used one or other of two funnels with 2.7 cm. and 4 cm. diameter respectively. The opening of the funnel was directed upwards and was covered with a layer of soap solution only very slightly curved. The soap bubble was blown by help of a T-piece inserted in the rubber tube and closed by a clamp. The blood pressure was recorded from one carotid by means of an elastic rubber manometer. In some experiments I recorded also the auricular contraction of the right auricle by the suspension method. The funnel and the points of the recording levers were placed in front of a lantern and the image of the levers and the upper surface of the soap bubble projected with a photographic lens on a slit behind which, in a dark room, an ordinary kymograph with photographic paper was running at a speed of about 3.5 cm. per second. The time was marked by the stylus of a metronome beating before the slit and thus interrupting the light every half second. The records obtained in this way show an abscissa, the changes of the soap bubble and the curves traced by the points of the levers. Every half second a vertical line of the time marker connects corresponding points of the curves.

After the experiment the volume changes indicated by the soap bubble were gauged by means of a syringe. The gauges are given on the margin of the curves. As long as the curvature of the soap film

is small the volume differences are nearly in linear proportion to the height of the apex of the bubble. With increasing height (h) the volume changes are more and more proportional to the cube of the height, as follows from the formula of the volume of a segment of a sphere. $V = \pi h \left(\frac{\rho^2}{2} + \frac{h^2}{6} \right)$. ρ is the radius of the funnel.

From the normal heart beating at the rate of 120 per minute the tracings recorded were as in Fig. 3. During the systolic discharge, marked by a downstroke of the soap bubble, the volume curve shows the shape described by Henderson. Then the volume rises again rapidly and for a long time with about the same speed as during the downstroke caused by the systolic discharge. The end of this period is marked by a notch, which is more distinct the slower the heart's rate is, and disappears completely in a quickly beating heart.

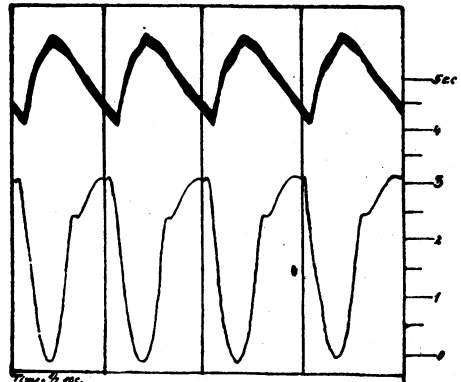


Fig. 3.

After the notch the volume resumes its upward movement with diminished speed and reaches a more or less prominent point near the top of the curve. The following part is not quite regular in different tracings even when obtained from the same heart. The conditions influencing this part will be discussed later. Generally one sees a slight notch and a second tip. Then at once the next discharge follows.

For identification of the points of the volume curve corresponding points of the pressure curve were used. It was first necessary to determine the loss of time between the opening of the aortic valves and the upstroke of the lever. For this purpose, immediately after death, the heart in connection with the vessels and the transmission used during the experiment was brought in front of the lantern and filled with liquid under a pressure corresponding to the blood pressure. Then the heart was suddenly compressed, and the compression photographed together with the following upstroke of the lever. From the tracing obtained in this way the loss of time between the compression and the resulting movement of the lever was calculated and found to be $1/12$ of a second.

Then in the curves previously recorded this time interval was traced back from the point, where the pressure pulse rises, and so the point of opening of the aortic valves determined. This point I connected by a dotted line with the corresponding point of the soap bubble curve. It

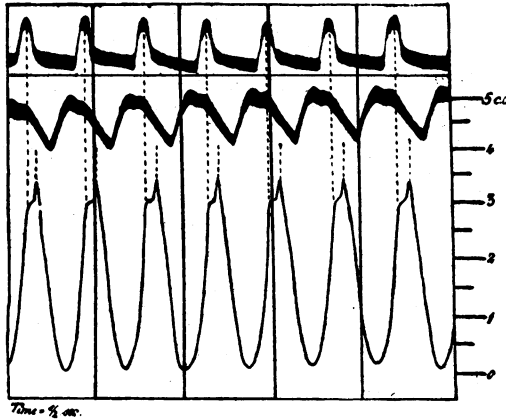


Fig. 4.

was always found, as theoretically would be expected, coincident with the beginning of the downstroke of the volume curve. This shows that the volume begins to diminish at the moment of the opening of the aortic valves and that during the "Anspannungszeit" no diminution in volume takes place.

Another point of the heart's cycle was marked in some curves by recording the auricular contraction by means of the suspension method. Figs. 4 and 5 were obtained in this way. The maximum of auricular contraction is connected by another dotted line with the soap bubble curve and there it meets the tip mentioned above as occurring near the top of the curve. This tip therefore is due to the increased inflow of blood caused by the auricular contraction. In Fig. 5 a weak stimulation of the vagus was used, and in

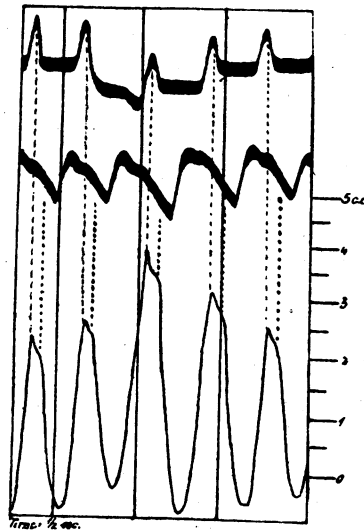


Fig. 5.

this way a relaxation of the auricle was caused. Under this condition the auricular contraction must move a greater volume of blood contained in the distended auricle, and the volume curve of the ventricle indeed shows the increased inflow.

The time interval between the auricular contraction and the opening of the semilunar valves is occupied by the closure of the auriculo-ventricular valves and by the "Anspannungszeit." To the mechanical disturbances produced by these events is due probably the somewhat irregular part of the curve at this point. The intraventricular pressure may vault the valves a little against the auricles and thus cause an apparent *diminution* in volume of the ventricles, as in Fig. 5. The action of the papillary muscle, contracting before the fibres of the heart's wall, as we know, may pull the valves against the ventricle and thus cause an apparent *increase* in volume as in Fig. 4. Or both mechanisms may be balanced, and this perhaps is the regular mechanism, as in Fig. 3.

For the notch during the upstroke of the volume curve the other curves give no explanation. This notch may be found in the curves of several previous investigators, such as Stefani, though much deformed by the inertia of the instruments used. In the rapidly beating heart it entirely disappears. The course of the curve seems to me to suggest the following explanation. After the contraction the heart by its own elasticity, like a compressed rubber ball, springs back to a certain volume with about the same speed as it diminished in volume during the contraction. This period of "elastic diastole" is ended by the notch of the curve. The rest of the curve indicates a "passive diastole," when the heart is filled from the veins as a result simply of pressure differences. The notch itself at first looks like an oscillation of the recording instruments. The dynamic standardisation demonstrates that this explanation does not apply. The notch is not due to the recording instruments, but signifies an oscillation of the heart muscle itself near the point of equilibrium of its elasticity and an oscillation of the blood following the movement of the heart wall with a certain inertia.

Fig. 6 demonstrates a somewhat stronger stimulation of the vagus. With the slowing of the heart rate the notch becomes more and more distinct. The total volume of the heart is increased, but though the output is augmented the systole is less complete. At the end of the curve the heart dilates so much that the soap bubble touches the lever of the falling pressure record and bursts.

In Fig. 7 the heart beat is completely stopped by strong vagus stimulation. Two auricular contractions cause a remarkable inflow of

blood into the fully relaxed ventricle. The waves following the auricular contractions are not oscillations of the recording instrument, whose period is incomparably quicker, but are the expression of waves of the blood in the relaxed ventricle. Henderson's somewhat similar curve on page 349 of his paper shows the great deformation due to increased damping.

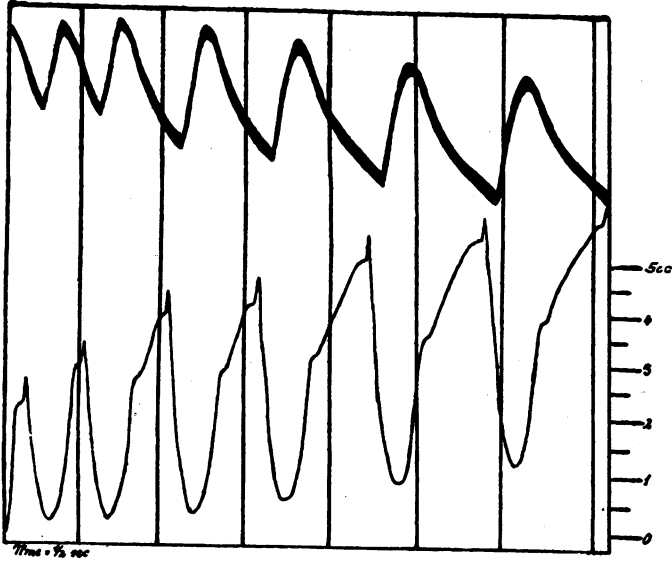


Fig. 6.

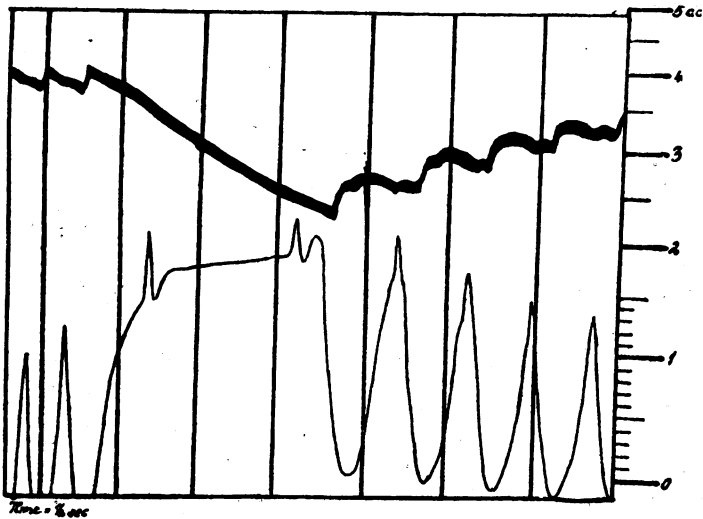


Fig. 7.

It is remarkable that none of my curves shows any portion parallel to the abscissa. Even during the long diastole due to the vagus stimulation of Fig. 7 the volume is still slowly but distinctly increasing. The different conclusion of Henderson must be attributed to his apparatus. It may be that the increased pressure caused by stretching the rubber membrane at the end of diastole has exercised such a resistance to distension as to prevent the ordinary inflow of blood into the ventricle. It may also be that the curve is deformed by an oscillation of the apparatus. The rapid movement of the systole or of the first part of diastole may cause the tambour to oscillate, and such an oscillation may hide the true upward movement of the curve. The rubber curtain of the cardiometer, if the hole in it be too small, may compress the atrio-ventricular groove and thus hinder the inflow of blood into the ventricles. In any case my curves demonstrate that there is no period of "*diastasis*."

SUMMARY.

For a true registration of the heart's volume an instrument is wanted with very small mass, damping and elasticity.

With the opening of the aortic valves the volume curve shows an instantaneous and steep descent. Near the bottom it rounds away to a blunt point.

After completion of the systole the volume curve assumes at once the upward movement and completes most of the diastole with a speed about equal to the corresponding reverse movement during the systole. This period ends by a notch due to the oscillations of the heart muscle and of the blood rushing into the ventricles. This part of diastole seems to be due to the elastic reaction of the ventricles: "*elastic diastole*."

The notch at the end of this period is more distinct the slower the heart rate.

After the notch the ventricles dilate with diminished speed being filled from the veins simply by the pressure difference: "*passive diastole*."

The auricular contraction causes a distinct increase in the volume of the ventricles.

The mechanism of the closure of the atrio-ventricular valves causes the last part of the curve to assume different shapes according to the prevalence of the outward push of the pressure or the inward pull of the papillary muscles on the valves.

There is no period of "*diastasis*" in the relaxing ventricle.