THE ROLE PLAYED BY THE VENTRICULAR RELAXATION
PROCESS IN FILLING THE VENTRICLE

LOUIS N. KATZ

From the Department of Physiology, Western Reserve University, School of Medicine, Cleveland

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At one time it was believed that the relaxing ventricle sucked blood into its chamber by some sort of activity during relaxation, such as an elastic recoil, a sudden stretching resulting from the filling of the coronary arteries, a marked asynchronous cessation of contraction, or even an actual contraction of some fibers at this time (cf. Ebstein, 1904; Tigerstedt, 1921, and Prince, 1915, for literature). The evidence presented in favor of this view has been refuted or, at least, shown to be compatible with the current idea of passive filling. For example, the recording of an intraventricular pressure below atmospheric with the minimal manometer and with the early graphic methods, by Goltz and Gaule (1878), de Jager (1883), Rolleston (1887), von Frey and Krehl (1890) and Porter (1892) among others, has been shown to be an instrumental error by Von den Velden (1906), Straub (1912) and Wiggers (1928). The latter two have failed to obtain pressures within the ventricle below that surrounding the heart when employing instruments of high precision constructed along the lines laid down by O. Frank (1903). Similarly, the claim of Mosso and Pagliani (cf. Ebstein, 1904) and of Stefani (1893) that the heart can fill when the pressure surrounding it is higher than in its chamber has been refuted by Roy (1879), Tunnicliffe (1896) and Martin and Donaldson (cf. Ebstein, 1904). The observation that the dead heart will fill after it has been artificially compressed (Chassaignac and Fick) (cf. Ebstein, 1904), does not necessarily apply to the conditions occurring in the living active heart (Von den Velden, 1906). The notch on the volume curve during early diastole to which diverse significance has been attached by Straub (1910) and de Heer (1912) is an instrumental artefact (Wiggers and Katz, 1920). The presence of a pressure gradient between the auricle and ventricle during the entire diastole with maxima early in the period and during auricular contraction (Porter, 1892) is equally compatible with the view of passive filling as with that of suction (Henderson, 1906).

Evidently the proof of a suction action by the relaxing ventricle is lacking. The current view of ventricular filling conceives the ventricles as being entirely passive, i.e., that the ventricular walls are stretched by
the force of the pressure head in the veins and auricles and that the energy of filling comes entirely from this pressure head, which is created by the accumulation of blood returning to the veins and auricles.

The idea that the ventricle plays no rôle in filling ignores the fact that the ventricle, like ordinary skeletal muscle, is an elastic body which has one elastic state when contracted and another when relaxed (Frank, 1895; Gasser and Hill, 1924). The process of contraction can be viewed as a change from the relaxed to the contracted elastic state and the process of relaxation as the reverse. Wiggers (1927) has shown that if the ventricle is not fully relaxed when filling begins, the completion of relaxation will facilitate filling. In fact, if the ventricle were to stay fully contracted as occurs with certain poisons, the amount of filling which the accumulation of fluid in the veins might produce is obviously much smaller than when relaxation occurs. A priori, these considerations indicate that the relaxing ventricle is not entirely passive but plays some rôle in filling.

The intact mammal is not very suitable for evaluating the rôle of ventricular relaxation in filling. This evaluation can be more readily accomplished in an isolated system where the other variable factors can be kept constant and relaxation compared with contraction. In this system a filling reservoir should be employed of such large cross section area that the removal of fluid from it to fill the heart will not appreciably alter the fluid level in it. The reservoir will keep the pressure in the system and against the walls of the ventricle constant unless some change is induced by activity of the heart. The use of a heart such as the turtle's with the auricles removed will further simplify matters by reducing the active chambers to one. Similarly, the use of rigid glass tubes to connect the ventricle with the filling chamber will eliminate the variable effect of elastic tubes. A narrow constriction in the tubes through which the ventricle fills will exaggerate any pressure variations which activity of the ventricle might produce and so make it more appreciable. The onset of filling may be made to coincide closely with the beginning of relaxation, in order to get the full effect of relaxation, by making the ventricle fill from the same reservoir chamber that it empties into. This arrangement will also make the extracardiac conditions during filling and emptying practically identical and thus allow a better comparison of the two phases. With the above arrangement contraction and relaxation would occur under conditions which approached an isotonic state. An investigation of the rôle of relaxation in filling was undertaken along these lines.

**Method.** A set-up modified from that previously described (Katz, 1928) was employed to satisfy the above mentioned conditions. A diagrammatic representation of the arrangement is given in figure 1. The turtle ventricle was connected with a reservoir, \( R \), containing isotonic sodium chloride, by means of a glass tube, \( N \), and cannula, \( I \), constricted
at the end. This cannula was inserted into the ventricular chamber via the aorta. In these experiments the stopcock, A, was turned 180° from the position shown in the diagram to exclude the tubes G and H. The saline reservoir was of such large cross section area that the fluid level dropped only 1 mm. for each 6.5 cc. saline removed. Intraventricular pressure was recorded by a modified Wiggers manometer, P, inserted into the ventricular cavity via the A-V orifice. Ventricular volume was recorded from an air-filled chamber, J, connected with a Frank segment capsule, Q, by a rubber tube, F. During the course of an experiment stopcock C was turned 90° and the side tube B was completely closed off. The pressure and volume changes were simultaneously recorded on bromide paper without parallax by using a double beam of light from the author’s (1924) duo-slit lamp. Such records were obtained at various levels of the reservoir. The volume curve was calibrated by means of the graduated pipette L, and the pressure curve, by varying the height of the reservoir, E, after the heart had ceased to beat.

RESULTS. The records obtained demonstrate without fail that the pressure early during diastole falls below the zero level of the system. A typical record is shown in figure 2. During ejection of fluid, which is indicated by the decrease in the volume curve, V, the pressure, P, rises for a time and then falls, almost reaching the zero level of the system by the end of ejection (indicated by the horizontal line in fig. 2). During filling which is indicated by the rise of the volume curve the pressure falls below the zero level at first and then returns to it. The reduction in pressure during filling is almost equal to the elevation of pressure during ejection in this particular experiment.

A reduction of pressure below the zero level of the system occurred in all the preparations during filling as long as the ventricle was rhythmically active. The reduction in pressure below the zero level was found to be greatest when the zero level of the system was highest1 and when the

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1 In these experiments, the optimum filling pressure was not reached.
ventricular activity was most vigorous. The amount of pressure reduction during relaxation varied more than the amount of pressure elevation during contraction as the reservoir level was varied. At no time was a pressure below atmospheric recorded within the ventricle.

Fig. 2. Simultaneous pressure, $P$, and volume, $V$, curves obtained under nearly isotonic conditions. Horizontal black line is base line of pressure curve. At bottom, white line is time; each change in level represents one second.

Fig. 3. Simultaneous pressure, $P$, and volume, $V$, curves obtained under nearly isotonic conditions and under isometric conditions. Points $X$ indicate time when conditions changed from isotonic to isometric. Base line and time as in figure 2.

The objection may be raised that the reduction in pressure during relaxation is an artefact caused by an overswing of the system and is not produced by the relaxing ventricle. The gradient of the change is too steep and the pressure drop is of too long duration to be explained as a
free oscillation of the system; it is rather like a forced phenomenon. Furthermore, the variation in pressure and volume at first go in opposite directions unlike the true free oscillations of the system sometimes recorded after filling is over (cf. fig. 2) where the pressure and volume changes are parallel and in the same direction. However, further proof was devised to show that the pressure reduction was not an artefact but actually evidence of activity on the part of the relaxing ventricle. It was based on the idea that the filling of the ventricle with fluid tended to decrease the

Fig. 4. Diagram of $P/V$ ratios in relaxed and contracted states, showing diagrammatically changes during contraction and relaxation. Discussed fully in text.

amount and abbreviate the period of pressure reduction. On this basis, a greater reduction in pressure would be anticipated if ventricular filling were prevented. Filling could be readily prevented with the apparatus employed by turning stopcock A (fig. 1) 90° at or near the end of the ejection period, thereby cutting off the ventricle from the reservoir. This procedure was carried out a number of times and, without fail, it was found that the pressure drop was magnified. A typical record is shown in figure 3. The first beat of the record shows the volume, $V$, and pressure, $P$, changes when the ventricle was allowed to fill. The changes are essentially
identical with those already described and illustrated by figure 2. At X in figure 3, just before the end of ejection, stopcock A was turned and the ventricle separated from the reservoir. The ventricle, therefore, did not fill as shown by the horizontal level maintained by the volume curve, V, and the pressure, P, rapidly dropped more than double the distance below the zero level found when the ventricle did fill.

This experiment is similar to that of making the ventricle contract isometrically where, as is well known, the pressure rises higher than under nearly isotonic conditions. The relaxing ventricle therefore behaves qualitatively in a manner similar to that of the contracting ventricle when the conditions are changed from nearly isotonic to isometric conditions. The reason for the increase in the pressure change when the relaxation (or contraction) of the ventricle occurs isometrically over that under nearly isotonic conditions, lies in the change in elastic state of the heart muscle during the process of relaxation (or contraction). During relaxation the heart, like skeletal muscle, changes from a body with a high elasticity coefficient to one with a low elasticity coefficient; i.e., the ratio $P/V$ decreases (where $P$ is the pressure and $V$ is the volume). During contraction the changes are in the reverse direction (cf. Frank, 1895; Gasser and Hill, 1924), i.e., the ratio $P/V$ is increased. A similar change can be produced in the ratio $P/V$ by changing the numerator, $P$, by altering the denominator, $V$, or by doing both. If the ventricle can fill during relaxation, the ratio $P/V$ is decreased by an increase in the volume, $V$, and a decrease in the pressure, $P$; but when isometric conditions prevail the change in $P/V$ is accomplished by decreasing $P$ alone. Hence, the change in $P$ is larger under isometric conditions than when filling can occur.

These facts can be illustrated in the diagram of figure 4. The upper curve AED represents the $P/V$ ratios of the fully contracted ventricle (cf. Frank, 1895) and the lower curve AFC, the ratios of the fully relaxed ventricle. Now, if we start with the fully relaxed ventricle at point F, then the changes occurring during contraction when the ventricle is connected with the reservoir (as in fig. 1) would fall along a curve such as the dotted line FHE (fig. 4), the temporary rise of pressure being due to the friction in the tubes and the inertia of the fluid retarding the ejection of fluid. During relaxation, if filling occurs, the changes will fall along a curve such as the dotted line EGF; the temporary fall in pressure being due to the friction in the tubes and the inertia of the system (possibly plus the initial momentum in the opposite direction) causing a lag in the movement of fluid toward the heart. The degree to which the pressure will fall during filling
will depend on the relation of the length and calibre of the tubes to the rate of relaxation. The shorter and wider the tubes or the slower the change in elastic state, the less marked will be the pressure drop. The faster the change in elastic state and the longer and narrower the tubes, the more marked will be the pressure drop. When filling is prevented, making the relaxation isometric, the pressure drop will be at a maximum, i.e., the vertical height $EB$. In other words, the drop in pressure during relaxation is due to the inability of the volume changes to keep pace with the change in the ratio $\frac{P}{V}$, and the extent of the pressure drop is a direct function of this lag in flow of fluid toward the ventricle.

Although the pressure changes during relaxation can be varied by changing the degree of filling, by altering the rate of filling, or by preventing filling entirely, nevertheless the curve representing the $\frac{P}{V}$ ratios of the fully relaxed ventricle is not affected by any of these factors. It was found in these experiments (cf. fig. 5) that in any single heart the

![Diagram](http://ajplegacy.physiology.org/)

**Fig. 5.** A comparison of the $P/V$ ratios of the fully relaxed ventricle, relaxing nearly isotonically (closed circles) and relaxing isometrically (open circles), with the smooth curved line.
$P/V$ ratios of the fully relaxed ventricle coincided reasonably with the same smooth curve both when the volume was set and relaxation was isometric (cf. open circles, fig. 5) and when the pressure was set and relaxation nearly isotonic (cf. closed circles, fig. 5).

Discussion. The experiments cited in this report indicate that, under the conditions of these experiments, filling occurs as a result of the relaxation process which reduces the elastic coefficient $P/V$ to a minimum value. By so doing, the pressure within the ventricle drops and so establishes a pressure gradient which tends to cause a movement of fluid toward the heart.

Several questions remain to be discussed: 1. What are the sources of energy for the change in elastic state? 2. Do these experiments indicate that the relaxing ventricle aspirates blood? 3. What is the relation of the results of these experiments to the common observations on the isolated muscle suspended on mercury? 4. Does the relaxing ventricle play a similar role in filling in the intact animal?

On an energetic basis, the fact that the pressure in the ventricle is reduced below the zero level of the system indicates that some work has been done and some source of potential energy has been converted to free energy. The ultimate source of such energy is some potential store created by the contraction process. It may come in part from the exothermic chemical reaction, presumably a neutralization of acids similar to that which Hill, Meyerhoff and their collaborators have shown occurs during the relaxation of skeletal muscle. The store of mechanical energy, built up during contraction, which would be converted to free energy might be in several forms. During contraction the potential energy of the heart muscle would increase because the muscle changes from a body with a low to one with a high elasticity coefficient. It would also increase because the center of gravity of the heart would be raised in the system used in these experiments. There might be some energy stored in the strain of some of the non-contractile elements in the heart. During relaxation these sources of energy and possibly others would be set free and could do work, at least sufficient to tend to drop the pressure within the ventricle below the zero level of the system. The reason that the pressure does not go below atmospheric is of course due to the limit set to the conversion of potential to free energy. The heart has a minimum elastic state which allows the pressure to approach atmospheric zero but never to fall below it. In other words, the lower limit of the pressure is set at atmospheric pressure by the minimum elastic state of the heart.

While the energy which enlarges the heart and lowers the pressure within its chamber is created by the heart, the energy for fluid movement comes from the pressure head in the reservoir. The filling heart in this regard differs in no way from any suction or aspirating pump in which
some source of energy lowers the pressure in the pump, but the energy for
flow comes from the pressure head in the reservoir regardless of what this
level is in relation to atmospheric pressure. The only apparent difference
between the ventricle and other aspirating pumps is the fact that the heart
cannot lower the pressure within its chamber below that surrounding it.
Nevertheless it can draw fluid into its chamber. Under these experimental
conditions, therefore, ventricular filling is a process similar in nature to
emptying. The mechanism for both filling and emptying is dependent

![Pressure curve and volume curve of the mammalian heart (after Wiggers), discussed in text.](image-url)

on some reversible physico-chemical process which alters the elastic
coefficient of the ventricle between two definite limits, the maximum for
the contracted and the minimum for the relaxed ventricle.

The observations here reported are readily correlated with the experi-
ments on skeletal muscle placed on mercury. It has been reported that
when a muscle is placed on mercury and stimulated, it will shorten and not
lengthen again. As a matter of fact, at first there is a slight lengthening
and eventually as the resting length decreases, a state is reached where
there is no shortening either. There is no doubt that the absence of
lengthening and later of shortening is due to the inability of the reduced change in elastic state, which decreases as the resting length decreases, to overcome the friction and other resistances preventing change in length. A few observations bearing on this point have been made by the author on the isolated frog gastrocnemius connected with a balanced lever and so arranged that lengthening will occur against gravity. It was found that during relaxation the gastrocnemius even with a small initial length can push the lever and so must do a little work. The amount of this work, although much less than that done in contraction, is nevertheless quite noticeable.

Granting then that the isolated ventricle connected to a reservoir, as in these experiments, can exert a sucking action and draw fluid into its chamber, the question is whether or not the mammalian ventricle actually does draw blood into its chamber when the circulatory system is intact. Evidence on this question can be obtained from an analysis of the combined ventricular volume and pressure curves of the dog's heart such as Wiggers prepared (cf. fig. 6).

Before discussing the curve, it should be borne in mind, that beside the possible sucking action of the ventricle two other factors are concerned in filling the ventricle, namely, 1, auricular activity, and 2, elevation of the pressure head in the auricles and veins by blood returning to them. During filling, the changes are different in the various phases. During rapid inflow (VI to VII) the pressure drops and the volume increases; during diastasis (VII to VIII) and early auricular activity, both pressure and volume increase, only to decrease somewhat later in auricular activity. The fact that the pressure decreases during rapid inflow while the ventricle is filling, indicates that the ventricle is relaxing since the ratio \( \frac{P}{V} \) is decreasing. Were filling entirely due to a passive distention, during this phase, one would expect to find an increase in both the volume and pressure as is the case during distention of any elastic body and is actually the case during diastasis and early auricular activity. The fact that the pressure drops during filling indicates that the ventricle is relaxing at a faster rate than it can fill. Such a state of affairs is inconceivable if the filling were entirely passive during this phase. In other words, since the large and rapid increase in volume in the rapid inflow phase is accompanied by a decided drop in pressure, the inference is obvious that a decrease in the elasticity coefficient is primarily responsible for the pressure drop and for filling. In no other way can one rationally explain, it seems to me, the opposite changes in volume and pressure. The relaxing ventricle, therefore, not only can but does exert a sucking action to draw blood into its chamber. The relative importance played by this sucking action, by auricular activity, and by passive filling due to the accumulation of
blood returning to the heart, remains to be worked out under various conditions for the two ventricles.²

**SUMMARY**

1. Experiments were made on the isolated turtle ventricle connected to a reservoir, which tended to keep the pressure of the system constant, in order to see what rôle ventricular relaxation played in filling the heart chamber.

2. It was found that during relaxation the pressure within the ventricle dropped below the zero level of the system. The amount of this drop was a direct function of the initial length of the muscle in the range studied.

3. The drop in pressure was not due to an overswing, for when filling was prevented at the onset of relaxation by making relaxation isometric, the pressure drop below the zero level of the system was magnified. Other considerations are discussed which also disprove that it is an artefact.

4. The minimum elastic state of the ventricle can be expressed by a smooth curve, along which the ratios of the relaxed heart fall, regardless of whether the diastolic volume or the diastolic pressure is set.

5. The drop of pressure during relaxation below that set by the reservoir is taken as evidence that the ventricle is drawing in fluid.

6. These experiments are not contradicted by those on skeletal muscle suspended on mercury.

7. The sources of energy for the sucking action are mainly potential stores, created by the contraction process, such as energy of position and of elastic state. The distinction is made between the source of energy for the sucking action and for the flow of fluid. The distinction is shown to be the same as in an aspirating pump.

8. The conclusion is drawn that relaxation is of similar nature to contraction. Both are dependent on some reversible physico-chemical process which alters the elastic state of the muscle between two limits, the minimum for the relaxed and the maximum for the contracted heart.

9. Evidence is given to show that the ventricle not only can but does exert a sucking action in the intact mammal.

² An idea of the possible relative importance of the passive filling compared with that due to sucking action may be indicated by comparing the gradient of the volume curve during diastasis with that occurring during rapid inflow. If we assume that the gradient of filling due to the accumulation of the blood returning to the veins is of a similar order during rapid inflow as it is during diastasis, which is probably correct, then the sucking action of the ventricle is of considerable importance in filling since it changes the gradient of filling tremendously. This argument is suggestive but, frankly, not conclusive.
RELAXATION PROCESS IN VENTRICULAR FILLING

BIBLIOGRAPHY

GOLTZ, F. AND J. GAULE. 1878. Pflüger's Arch., xvii, 100.
DEHEER, J. L. 1912. Pflüger's Arch., exlviii, 1.
HENDERSON, Y. 1906. This Journal, xvi, 325.
KATZ, L. N. 1928. This Journal, lxxxvii, 348.
PRINCE, A. L. 1915. This Journal, xxxvii, 43.
1912. Pflüger's Arch., exliii, 69.
WIGGERS, C. J. AND L. N. KATZ. 1920. This Journal, liii, 49.