Experimental Evidence of Ventricular Diastolic Suction

By GERHARD A. BRECHER, M.D., PH.D.

Intraventricular pressures were measured during brief periods of mitral orifice occlusion to determine whether or not the ventricle can exert a sucking force during diastole. This procedure was based on the premise that with a wide open mitral orifice negative diastolic transmural pressures cannot be detected in the ventricle because the adjoining atrium has collapsible walls. During mitral orifice closure, negative intraventricular pressures were recorded, and Ringer's solution was sucked against the force of gravity into the ventricle. It is concluded that the mammalian ventricle is capable of sucking blood from the atrium into its cavity.

ACCORDING to present concepts the ventricle is filled only by the vis a tergo, i.e., the pressure available in the atrium during ventricular diastole. This view is based on the observations that negative intraventricular pressures have never been reliably recorded and that, in an open chest dog or cat, fluid is not sucked by the ventricle through a cannula from a reservoir situated below heart level.

Despite this commonly accepted evidence, interest has been renewed in the problem of the "active" diastole (for historical review see Brecher). Evidence in support of the belief that work can be performed by the ventricle in diastole has been furnished by Chassaignac and, recently, by Bloom. They showed that the beating, excised, mammalian ventricle, submerged in water or saline and thus without effective filling pressure, can take in fluid and pump it out.

The present study was made to determine whether or not negative transmural pressure and suction can develop during diastole in the mammalian ventricle.

The experimental approach was based on the consideration that the lack of a detectable negative intraventricular transmural pressure in the normal heart does not prove the absence of a diastolic sucking force. Such a force may exist, but may not be revealed by the present techniques of intraventricular pressure measurement.

Failure to reveal diastolic suction by pressure measurements could be attributed to the fact that part of the common veno-atrioventricular cavity has pliable walls. Under normal conditions fluid may be transferred with such small impedance from one part of the cavity (atrium) into the other (ventricle) that a detectable negative transmural pressure might not develop in the ventricle even if it did suck in blood during diastole. The pliable veno-atrial walls would offer no structural resistance to accommodate a smaller volume when blood empties into the ventricle. Hence, negative pressures may be detectable only in that part of the common cavity which has semi-rigid walls (ventricle), if the connection with the atrium were interrupted.

METHOD

Acute experiments were undertaken in 11 dogs, 10 to 22 Kg., in weight, anesthetized with 30 mg./Kg. pentobarbital intravenously and fixed in the right lateral decubitus position. The chest was entered between the left fourth and fifth ribs (fig. 1). A cannula of 6 mm. lumen was inserted via left atrial appendage into the left ventricle and connected with thick walled Tygon tubing to a reservoir R of 250 ml. of Ringer's solution. A rubber flap valve V prevented reflux of blood from the ventricle to the reservoir but permitted Ringer's solution to flow from a reservoir toward the ventricle. Flow from the reservoir toward the ventricle was visualized by adding methylene blue to the reservoir fluid. In the first group of experiments outflow from the reservoir (decreasing weight) was recorded with a strain gage flowmeter S (Alexander). In the second group of
results

Reservoir Outflow. Figure 2 shows a segment of a strain gage flow meter record from an experiment in which the fluid level of the reservoir (R in fig. 1) was 28 mm. below the fluid level in the chest. In this record a constant weight of the reservoir (= 0 outflow) produces a horizontal tracing. From the beginning of the record to the point marked X the mitral orifice was open, permitting an unimpeded inflow of blood from the atrium into the ventricle. Then the mitral orifice was closed for 7 sec. (from X to Y) by tightening a ligature (L in fig. 1). During this period 7 heart beats occurred. The decline of the tracing shows that 5.8 ml. of Ringer's solution flowed from the reservoir into the ventricle. At the point marked Y the ligature was released and unimpeded atrial flow into the ventricle was resumed.

The following may be concluded from these findings: When ventricular filling from the atrium is prevented by a temporary closure of the mitral orifice the ventricle can suck fluid from a reservoir situated below the hydrostatic level of the ventricle as indicated by the decline of the tracing (from X to Y). On the other hand, when the ventricle is filled from the atrium through the wide open mitral orifice, fluid is not sucked from the lower reservoir into the ventricle. This is indicated by the horizontal course of the tracing at the beginning and end of the record.

A detailed examination of the record in figure 2 reveals that the outflow from the reservoir into the ventricle (X to Y) occurred rhythmically. The tracing declines maximally during ventricular diastoles D but minimally during systoles S. The systolic contractions are recognizable in the tracing by a transmitted vibration artifact. This time relation indicates that the emptying of fluid from the reservoir occurred primarily during ventricular diastoles.

Phasic Variations of Ventricular Inflow. The low resonance frequency of the strain gage flowmeter (6 cycles/sec.) limited further analysis of ventricular inflow within the cardiac cycle. Therefore, the rate of flow through the intraventricular cannula was phasically recorded with a high fidelity flow recorder (bristle flowmeter) simultaneously with intraventricular pressures. Figure 3 depicts two segments of a continuous record from an experiment in which the reservoir (R in fig. 1) was 30 mm. below the hydrostatic level of the ventricle. The two heart beats shown in segment A of figure 3 were recorded while ventricular inflow from the atrium was unimpeded. Early diastolic pressure was 0 mm. of water and end-diastolic pressure was +54 mm. of water. No flow from the reservoir occurred during diastole. The brief backflow at the beginning of systole and the small fluid column oscillations during systole were caused by some bulging of the rubber flap valve. Segment B, taken 2 sec. later, shows the effect of closing the mitral orifice. Intraventricular pressures descended as low as -108 mm. of water at early
Fig. 2. Strain gage flowmeter record demonstrating ventricular diastolic suction. Calibration in ml. (For better reproduction the vertical time lines were removed by retouching.)

Fig. 3. Effect of ventricular diastolic suction upon flow from a reservoir 30 mm. below heart level. A. Mitral orifice open. B. Mitral orifice occluded. Flow in ml./min. Intraventricular pressures in mm. water.
diastole, rising to —65 mm. water at the end of diastole (between fifth and sixth heart beat). A marked forward flow occurred during each diastole. The brief back flow component at the beginning of each systole and fluid column oscillations during systole can be attributed to a slight incompetence and bulging of the rubber flap valve.

A detailed analysis of these tracings reveals that inflow into the ventricle from the —30 mm. reservoir began at the moment when the intraventricular pressure reached —33 mm. water. Flow was rapidly accelerated during the first 100 msec. during each diastole. Maximal flow rates of 113 ml./min. were reached during the first and fifth diastole. Flow was maintained at a high, though moderately declining rate throughout the entire diastole. Flow decelerated rapidly within 30–50 msec. to 0 with the advent of systole. Total inflow during the first diastole amounted to 0.3 ml. as calculated by measuring the area under the flow curve. Average inflow per minute amounted to approximately 43 ml. (heart rate 150). It is noted that diastolic suction even occurred during the brief period between the third heart beat and the subsequent extrasystole. It occurred also during the compensatory pause though the negative pressure reached immediately after the extrasystole was less (—70 mm. water) than after regular systoles.

The following may be concluded from these findings. A substantial negative transmural pressure can develop in the ventricle during diastole when the fluid connection between the ventricle and atrium is interrupted. This negative pressure is maximal at early diastole but persists throughout the entire diastole. Owing to the negative intraventricular pressure, fluid can be sucked during diastole from a constant pressure reservoir situated below the hydrostatic level of the ventricle. Suction is more pronounced during diastoles following strong heart beats.

Intraventricular Pressure Measurements. During the experiments it was observed that the rubber flap valve always bulged slightly during ventricular systole. Thus the fluid column between reservoir and ventricle was set into rhythmical motion as seen in the flow-meter tracings of figure 3. As is well known, the net movement of such a pendulating fluid column can be unidirectional under certain hydrodynamic conditions (greater inertia in one direction than in the other). This could simulate a suction effect by the ventricle. In order to rule out all possible artifacts arising from a pendulating fluid column intraventricular pressures were measured after clamping the tubing from the reservoir to the ventricular cannula in a third group of experiments.

The average values of negative intraventricular diastolic pressures recorded for 6 consecutive beats during temporary closure of the mitral orifice are presented in table 1. In some experiments epinephrine was given (0.1 ml./Kg., 1:50,000). Negative transmural pressure developed consistently in all experiments, but varied in degree in different animals. In all cases the negative pressure persisted throughout the entire diastole. Generally, the pressures became somewhat more negative under the action of epinephrine.

Physical Model. A physical analogue was constructed to test the soundness of the hypothesis that ordinarily negative pressure cannot be demonstrated in the ventricle because of the collapsibility of the veno-atrial walls (fig. 4). The semirigid walls of a cylinder (= ventricle) were connected to a pliable rubber bag (= atrium) leaving a wide opening (= mitral orifice). The rubber bag was filled with water from a constant pressure reservoir (= pulmonary capillary bed) situated 1 to 5 mm. above the hydrostatic level of cylinder and bag (= heart). Suction (= diastole) and pressure (= systole) was created in the

<table>
<thead>
<tr>
<th>No. of Experiment</th>
<th>Diastole Without Epinephrine</th>
<th>Diastole With Epinephrine</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Early</td>
<td>Late</td>
</tr>
<tr>
<td>1</td>
<td>—96</td>
<td>—55</td>
</tr>
<tr>
<td>2</td>
<td>—32</td>
<td>—23</td>
</tr>
<tr>
<td>3</td>
<td>—28</td>
<td>—20</td>
</tr>
<tr>
<td>4</td>
<td>—80</td>
<td>—43</td>
</tr>
<tr>
<td>5</td>
<td>—18</td>
<td>—5</td>
</tr>
<tr>
<td>6</td>
<td>—85</td>
<td>—60</td>
</tr>
</tbody>
</table>
FIG. 4. Physical model illustrating the effect of a structure with collapsible walls (= atrium) upon negative pressures in a structure of semirigid walls (= ventricle).

cylinder by moving a piston $P$. "Aortic" and "mitral" valves assured unidirectional flow. A ligature $L$ placed around the "atrium" permitted temporary closure of the "mitral orifice."

The negative pressures recorded from the cylinder during suction were too small to be measurable when fluid was transferred without an appreciable resistance from the collapsible rubber bag into the cylinder. However, negative intracylindrical pressure was measurable when the "mitral orifice" was closed by tightening the ligature $L$. The pressure and flow records obtained from the physical analogue resembled closely those of the living animal shown in figure 3.

It can be concluded that without creating a detectable negative transmural pressure fluid can be drawn by a suction force into a rigid walled cavity (= ventricle) from another cavity which has pliable walls (= atrium) when resistance to flow is negligible.

**DISCUSSION**

These experiments demonstrate that negative pressure and suction can develop in the mammalian ventricle. In the light of this information it will not be possible to maintain the present concept that the ventricle is filled exclusively by the *vis a tergo*.

However, we must express caution as to the quantitative significance of intraventricular diastolic suction for the normal ventricular filling. The experimental conditions under which the existence of negative intraventricular pressures was revealed preclude such quantitation. The residual volume of the ventricle became very small after mitral orifice occlusion. Although in several experiments suction was seen to occur during the first diastole following sudden mitral occlusion it may well be that in the presence of large residual volumes intraventricular suction is less pronounced or even abrogated.

The experiments offer suggestive evidence that intraventricular suction is mainly caused by an elastic recoil of the ventricular walls. This view is supported by the observation that the lowest negative pressures were recorded after the strongest ventricular contractions and that negative pressures and suction could also be produced by massage of the fibrillating and dead ventricle.

**SUMMARY**

The problem of negative ventricular diastolic pressures and diastolic suction was studied in 11 anesthetized open chest dogs. The hydrostatic level of the heart was established by filling the chest with saline. An intravenous cannula was connected via tubing and a flap valve to a reservoir of Ringer's solution situated below the hydrostatic level of the heart (from 0 to $-100$ mm.).

No diastolic ventricular suction occurred when the mitral orifice was open. Upon temporary occlusion of the mitral orifice, Ringer's solution was sucked against the force of gravity into the ventricle during diastole. Simultaneously recorded intraventricular diastolic transmural pressures were negative, ranging during early diastole from $-13$ to $-108$ mm. H$_2$O in various animals. It was demonstrated in a physical analogue that an active sucking force can exist in a cavity of semi-rigid walls (= ventricle) without creating a negative transmural pressure detectable by present technics provided that flow from an adjoining cavity with pliable walls (= atrium) is practically unimpeded. Correspondingly, negative intraventricular transmural pressure cannot be recorded reliably when the mitral orifice is open because the atrial walls of the common atrioventricular cavity are collapsible. The existence of a ventricular sucking force and
the possibility of developing recordable negative intraventricular transmural pressures could, therefore, be revealed only when the mitral orifice was closed.

As a result of these findings, one cannot maintain the commonly accepted view that the ventricle fills only passively by arrectro. The mammalian ventricle is capable of sucking blood into its cavity during diastole.

However, no conclusions can be drawn from these experiments as to the quantitative significance of diastolic suction for filling a ventricle which contains a normal or large residual volume.

Acknowledgment

The author wishes to express his appreciation to Dr. Eric Ogden and Leo Sapirstein for their generous help and valuable criticism and to Mrs. Bella Kleinman and Mr. Eugene Scott for their skillful assistance.

Addendum

While this paper was in press, an exchange of manuscripts with Dr. Walter Bloom of the Department of Medicine, Emory University School of Medicine, revealed that he had also recorded negative intraventricular pressures in the dog.

Summario in Interlingua

Le problema de negative pressiones diastolic ventricular e de suction diastolic esseva studiata in 11 anesthesiate canes a thorace aperte. Le nivello hydrostic del corde esseva establite per plenar le thorace con solution salin. Un cannula intravenose esseva connectite via tubos e un valvula con un reservoir de solution de Ringer que esseva placiate infra le nivello hydrostatic del corde (ab 0 a 100 mm).

Nulle diastolic suction ventricular occurreva quando le orificio mitral esseva aperte. Post occlusion temporari del orificio mitral, solution de Ringer esseva sugite, in diastole, contra le fortia de gravitate a in le ventriculo. Le registration simultanea de transmural presiones diastolic intraventricular monstrava valores negative, con un variation in varie animales de inter minus 13 e minus 108 mm H2O durante le prime phase diastolic. Il esseva demonstrate per medio de un analogo physic que un active fortia de suction pote existir in un cavitate de parietes semirigide (= ventriculo) sin crear un negative pression transmural de grados detegibile per le nunc disponibile technicas, providite que il ha un practica mente non-impedite fluxo ab un cavitate adjacente con parietes elastic (= atrio). Correspondentemente, un negative pression transmural intraventricular non es precisemente registrable quando le orificio mitral es aperte, proque le parietes atrial del commun cavitate atrio-ventricular es collabibile. Le existentia de un fortia de suction ventricular e le possibilitat de disveloppamento de registrabile negative pressiones transmural intraventricular poteva, per consequente, revelar se solmente quando le orificio mitral esseva claudite.

Resulta ab iste constatationes que il es impossible mantener le opinion currente que le ventriculo es plenate solmente passivemente per le action de un via a tergo. Le ventriculo mammalian es capace de suger sanguine a in su cavitate durante le diastole.

Nonobstante, iste experimentos non permette deducer conclusiones relative al significatation quantitative del suction diastolic in le plenation de un ventriculo que contine un normal o un grande volume residue.

References

1 Guasp, F. T.: El ciclo cardiaco, consideraciones criticas sobre la interpretation clasica y nuevas ideas sobre el mismo. Privately published monograph by Guasp from the Medical Faculty of the University of Salamanca, Madrid, 1954.


Experimental Evidence of Ventricular Diastolic Suction

GERHARD A. BRECHER

Circ Res. 1956;4:513-518
doi: 10.1161/01.RES.4.5.513

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1956 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7330. Online ISSN: 1524-4571

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circres.ahajournals.org/content/4/5/513

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation Research can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation Research is online at:
http://circres.ahajournals.org/subscriptions/