Ventricular Diastolic Suction at Normal Arterial Pressures

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It has been shown previously that an empty ventricle can suck in a small amount of fluid during diastole, but the question of whether a normal ventricle does so was not answered. In the present experiments it was demonstrated that even a ventricle with a more nearly normal endsystolic residual volume draws blood into its cavity and that this ventricular *vis a fronte* is augmented by the action of epinephrine.

It has been established that the mammalian and reptilian ventricle can draw fluid into its cavity by a diastolic *vis a fronte*. However, from these observations, one cannot arrive at any conclusions as to the role of ventricular diastolic suction in normal ventricular filling since the existence of this phenomenon was demonstrated only in nearly empty ventricles. The common denominator of all previous experimental conditions was that the ventricles contained an abnormally small residual volume because their content was ejected against a negligible resistance (almost empty or severed arteries). It was furthermore shown that negative transmural pressures occurred in quiescent ventricles only when they contained a relatively small volume.

Therefore, the question still remained unresolved whether normal ventricular filling is aided by the ventricular diastolic *vis a fronte*. This problem was the subject of the present investigation. Since this problem could not be suitably studied in the unanesthetized closed chest mammal it was examined under conditions in which the nearest approximation to a normal residual volume could be achieved experimentally. Contrary to the conditions in previous experiments, a more nearly normal endsystolic residual volume was maintained by allowing the ventricle to eject against the resistance existing in the aorta under normal arterial pressure.

**Method**

In futile preliminary attempts to answer the question, numerous dog experiments were performed in which negative left intraventricular diastolic transmural pressures were observed and correlated with cardiac output and ventricular residual volumes. These experiments included modified heart-lung preparations and hearts with partially occluded or controlled ventricular inflow. It was generally found that some ventricular filling occurred in the absence of a positive ventricular filling pressure. However, the amount of blood drawn into the left ventricle and then expelled into the aorta was too small to sustain near normal aortic pressures and adequate coronary perfusion. This resulted in a reduced vigor of contraction which in turn diminished intraventricular diastolic suction. Thereupon, arterial pressure fell further, initiating a vicious cycle which led to rapid circulatory failure.

From these observations it became evident that, in order to maintain normal ventricular performance, the coronary and body tissue perfusion had to be supported by artificial means. This was done with the aid of a pump, by-passing the left ventricle as illustrated in figure 1.

These perfusion experiments were undertaken in 10 open chest dogs, 10 to 15 Kg. in weight, anesthetized with 30 mg. Kg. pentobarbital intravenously. The animals were fixed in the right lateral decubitus position and the chest was entered between the left fourth and fifth ribs. The heart was submerged by filling the chest (C) with donor blood (B), thereby establishing an accurate

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*Suction, as used here, is defined as a *vis a fronte* which draws fluid into a cavity by reducing the pressure in the cavity below that existing outside the cavity's orifice.
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zero atmospheric pressure reference level. The positive ventricular filling pressure existing in the intact atrium was abolished by extirpating the entire outer wall and appendage of the left atrium (A). The pulmonary veins (PV) then emptied directly into the blood lake (B), and the ventricle (V) could only receive fluid from the blood lake without any filling pressure at the mitral orifice. The edges of the left atrial wall and the pericardium (incised over the left atrial region) were prevented from partially occluding the mitral orifice by holding them open with a modified eyelid speculum or suitable clamps. Great care was taken to open the atrium under the fluid level in order to prevent air from being drawn into the ventricular cavity and resulting in coronary air embolism. The overflow of blood was drained from the chest through cannula D into a defoaming reservoir (R) and pumped from there with a Sigma Motor pump (S) to a perfusion reservoir (P). Perfusion into both femoral arteries (FA) could be adjusted with the aid of a bleeder valve (BV) in a compressed air line (CA) and an aneroid manometer (M,) so as to maintain various pressure levels in the arterial system. Manometer M, recorded fluctuations of the level of the blood lake, thus assuring a continuous tracing of the atmospheric zero pressure reference level. Manometer M, recorded aortic pressures (AO) via a cannula passed through the left carotid artery. Manometer M, recorded left intraventricular pressure via a polyethylene catheter inserted through a pulmonary vein or through the ventricular wall. M, Mi, and M, were modified Gregg optical manometers of adequate frequency response and sensitivity. The pressure in the perfusion reservoir was brought into equilibrium with the normal mean aortic pressure of the animal while the atrium was still intact. This permitted the extirpation of the atrial wall without a fall in the mean aortic pressure which was automatically maintained by perfusion from the reservoir, in some cases for over an hour. Such an arrangement had the following advantages: 1. Resistance to left ventricular ejection, which among other factors is dependent upon the diastolic aortic pressure, was only little changed from normal when the atrium was opened. 2. Coronary and body tissue perfusion remained practically unchanged. 3. By altering the arterial perfusion pressure the left ventricular residual volume could be increased or decreased above or below the amount existing at normal aortic pressures.

After taking control records with the left atrium open, 100 μg of epinephrine (Parke Davis) were injected into the femoral vein, or 40 μg added to the perfusing arterial blood, or 40 to 100 μg added to the blood in the chest cavity near the open left atrium. With the intravenous route the epinephrine had to circulate via pulmonary veins into the chest cavity where it became diluted before reaching the left ventricle. It was found that by this route relatively large doses of epinephrine were necessary to produce marked effects on ventricular contraction.

After each experiment the size of the extirpated left atrial wall area was measured and found to be considerably larger than the mitral orifice area.

RESULTS

Figure 2 shows a record from a representative experiment illustrating left intraventricular and aortic pressure pulses produced by a ventricle without positive ventricular filling pressure. This record was taken about 5 min. after the extirpation of the left atrial wall while the mean aortic pressure was maintained by pump perfusion at the level which existed under normal circulatory conditions before the opening of the atrium. As seen in the lower tracing, left intraventricular pressure was 115/0 mm. Hg. Aortic pressure rose from 98 to 112 mm. Hg with a near normal systolic contour (first and third beat). The midcilesural pressure (100 mm. Hg) was slightly above end-diastolic pressure, indicating some diastolic runoff. The heart had a pulsus alternans, as was often observed in these experiments when the heart began to weaken. The second beat in figure 2 illustrates how a less forceful ventricular beat resulted in a smaller systolic aortic pressure rise under these con-
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Fig. 2. Ventricular and aortic pressure curves of an intact left ventricle filling with zero ventricular filling pressure and ejecting against normal arterial pressure. From top to bottom: Aortic pressure in mm. Hg, atmospheric zero manometer (M0), intraventricular pressure in mm. Hg. Respiration stopped during recording so level of blood remains unchanged as indicated by horizontal tracing of zero manometer. Baselines of aortic and ventricular manometers and time tracing removed by retouching.

ditions. Continuous tracings of this type of aortic pressure pulses were obtainable for over 5 min. The aortic systolic pulses disappeared when the mitral orifice was manually or instrumentally occluded. This indicates that the ejected blood had entered the ventricular cavity through the mitral orifice and not through the thebesian vessels from the coronary arteries.

One may conclude from this experiment (1) that in the absence of a positive ventricular filling pressure the ventricle ejected with each contraction a certain amount of blood against the resistance existing in the aorta under normal arterial pressures and (2) that the ejected blood must have entered the ventricle by the action of a diastolic ventricular vis a fronte.

Figure 3 illustrates the effect of an abnormally high arterial perfusion pressure on ventriculard ejection. Record A represents the normal aortic pressure curve of the animal before the atrium was opened. Record B shows the aortic pressure curve after excision of the left atrial wall, when the perfusion pressure was raised from a normal level of about 130 mm. Hg to 170 mm. Hg. Under this condition the ventricular pressures amounted to 140/0.

The aortic pressure is essentially a horizontal line indicating that no detectable ejection of ventricular blood occurred. During isometric contraction one notices a slight impact vibration similar to that customarily observed in normal aortic pressure tracings (compare with record A).

Record C of figure 3 was taken 3 min. after B with the same pressure in the perfusion
reservoir but after 100 \gamma of epinephrine had been injected into the femoral vein. Intraventricular systolic pressures reached values of 270 mm. Hg. Aortic pressures were elevated to 244/213. The aortic pressure curve showed a marked pulsation in which the systolic rise and diastolic fall is similar in contour, though shorter than the normal (record A) indicating the existence of ventricular output.

From the experiment one may conclude that in the absence of a filling pressure an otherwise normal ventricle can produce systolic pressures which are as great as or even greater than normal. The systolic pressures, however, are not sufficient to overcome the resistance to ejection when the arterial pressure is artificially set at an abnormally high level. The ventricle contracts isometrically under such conditions. One may further conclude that epinephrine increases the force of ventricular contractions so much that the abnormally high resistance to ejection is overcome.

Figure 4 shows two segments of a continuous record taken from another experiment, illustrating the effect of epinephrine on the failing heart in the absence of a ventricular filling pressure. In A the arterial perfusion pressure was 80 mm. Hg. The force of ventricular contraction was too weak to overcome the resistance to ejection. The aortic pressure curve remained essentially a horizontal line. Segment B was taken with the same pressure in the perfusion reservoir about 4 min. later after 100 \gamma of epinephrine had been injected into the femoral vein. Ventricular systolic pressures increased from 58 to 98 mm. Hg and resulted in ejections into the aorta at slightly subnormal arterial pressure levels (97/83).

One may conclude from this that in the absence of a positive filling pressure the contractile force of the failing ventricle is so reduced that no portion of the ventricular content is ejected against the resistance existing at near normal arterial pressure levels. No ventricular diastolic \textit{eis a fronte} can be demonstrated under such condition. However, when the ventricle contractile force is increased by the action of epinephrine the failing ventricle can draw blood in and eject it into the aorta at near normal arterial levels.

Definitely measurable negative intraventricular transmural pressures were recorded in only 3 experiments. As pointed out previously, the resistance to inflow through a wide open mitral orifice was too small to detect subatmospheric ventricular pressures with conventional instruments.

**DISCUSSION**

The value of the present experiments lies in the demonstration that a more nearly normal ventricle actually draws in blood for its filling. Undoubtedly, the reservation must be made that a ventricle in an anesthetized open chest dog is not an entirely normal ventricle. Its endystolic residual volume is smaller than that in the closed chest, resting, unanesthetized animal. However, it can be assumed that the ventricle with the open atrium did not differ markedly from a normal ventricle of an open chest preparation as far as the endystolic residual volume is concerned. In either case this volume would be determined by the resistance to ejection which would be approximately the same at equal arterial pressures.

The results obtained from these experiments are only of qualitative nature. The amount of blood sucked into the ventricle cannot be determined from the contour of the aortic pressure pulses alone. Therefore, the percentage contribution of diastolic suction to normal ventricular filling remains unknown, and one cannot as yet draw any conclusions as to its biologic significance.

Even though the evidence derived from pressure pulse contours as to cardiac output is only qualitative, one may say that in the normal as well as in the failing heart the action of epinephrine consistently resulted in an increase in the amount of blood drawn into the ventricle. This may be caused by an increase of diastolic suction resulting from either an augmentation of the ventricular contractile

*The increased peripheral resistance to maintained perfusion plus ventricular output may account for the high aortic pressure level when epinephrine was acting.*
force or a decrease of the ventricular residual volume. That epinephrine leads to more negative intraventricular diastolic pressures in the nearly empty ventricle with a partially obstructed inflow has been previously shown.\(^2,7\) It is also known that pressures are more negative in ventricles of small volume.\(^8\) Probably both factors act in combination to increase diastolic suction with epinephrine.

With these data available the following concept may be suggested. It appears that in the normal heart a small amount of blood is drawn into the ventricle by the diastolic ventricular vis a fronte. On the basis of the relatively small aortic pulse pressures produced by the ventricle in the absence of a filling pressure this contribution to ventricular filling is probably less significant than that by the vis a tergo. With epinephrine, the ratio of vis a fronte to vis a tergo apparently shifts in favor of the former in effecting ventricular filling. The change of an organism from rest to exercise may also involve a similar shift of this ratio.

In the past the finding of a positive, intraventricular, transmural pressure has been interpreted to mean that ventricular diastolic suction cannot exist. It was thought that only the occurrence of negative intraventricular transmural pressures could be taken as evidence for the existence of a ventricular diastolic vis a fronte. A brief consideration of the physical forces will show that this conclusion is fallacious.

The concept that a vis a fronte could aid normal ventricular filling, even though intraventricular, diastolic, transmural pressure may remain positive is illustrated by two simple diagrams in figure 5. In a fluid system in which two forces are operating to fill a cylinder (ventricle), one force pushing (vis a tergo) and the other one pulling (vis a fronte), the pressure in the cylinder will be determined by: the pressure (vis a tergo) in the reservoir (atrioventricular system), the resistance to flow from the reservoir into the cylinder, and the impedance to inflow offered by the cylinder. Normally, the resistance to flow through the ativoventricular orifice is very small. In the presence of a vis a fronte (fig. 5A), symbolized by a plunger pulling with an elastic spring on the cylinder content, the filling would be facilitated because the pressure difference between the cylinder and the reservoir would be increased. In the example the frontal force would lower the pressure in the cylinder to 20 mm. H2O (fig. 5A) from the 40 mm. H2O...
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Existing in the absence of this force (fig. 5B). The pressure in the cylinder need not necessarily fall below atmospheric (negative, intraventricular, transmural pressure). In a system in which the resistance to flow (orifice) is negligible the pressure in the cylinder could only become negative if the *vis a fronte* exceeded the *vis a tergo*. The pressure relations in the absence of a frontal force are symbolized in figure 5B: in this case the plunger is pushed, corresponding to a diastolic expansion of ventricular walls by *vis a tergo* alone.

From this consideration it must be concluded that even the existence of a positive diastolic transmural pressure in a normally filling ventricle is theoretically compatible with the existence of a ventricular diastolic *vis a fronte*. It also illustrates that in such a case one cannot draw any conclusion from the pressure measurements alone as to the forces involved.

The implication drawn from the present experiments that a diastolic frontal force contributes to normal ventricular filling is, therefore, not in conflict with the finding that slightly positive, intraventricular, diastolic, transmural pressures are usually observed in the unanesthetized normal organism.

SUMMARY

In 10 experiments on anesthetized open chest dogs the problem of whether a normal ventricle draws in blood during diastole was studied. Under the experimental conditions the nearest approximation to a ventricle of normal endsystolic residual volume was established by allowing the ventricle to eject against the resistance existing in the aorta at normal arterial pressures. The heart was submerged by filling the chest cavity with donor blood. The left atrial wall was extirpated, thereby establishing zero ventricular filling pressure at the mitral orifice. The entire circulation and arterial pressures were maintained at normal levels by pumping pulmonary venous blood from the chest cavity into the femoral artery. Small systolic aortic pressure pulses were recorded at normal arterial pressure levels indicating the existence of ventricular ejection and thereby of diastolic suction. This was enhanced by epinephrine in the normal as well as in the failing heart. It was concluded that the intact mammalian ventricle working against normal aortic pressures draws a small amount of blood into its cavity with zero ventricular filling pressure. A concept was advanced that in an intact normal heart a positive intraventricular diastolic transmural pressure does not preclude the existence of a ventricular diastolic *vis a fronte*.

SUMMARIO IN INTERLINGUA

In 10 experimentos con anesthesiate canes a thorace aperte, le problema esseva studiate si le ventriculo normal effectua uu suction de sanguine in le diastole. Sub le conditiones del experimento le melior approximation de un ventriculo con normal volume residue termino-systolic esseva establite per lasser le ventriculo completar su ejection contra le resistentia que existe in le aorta sub le condizioni de normal pressiones arterial. Le corde esseva submergito per plenar le cavitate thoracica con sanguine exogene. Le pariete sinistro-atrial esseva extirpate, de manera que le pression de replenation ventricular al orificio mitral esseva zero. Le pression del circulation total e le pression arterial esseva mantenite a nivellos normal si per pumpar sanguine pulmono-venose ab le cavitate thoracica a in le arteria femoral. Basse systolic aortic pulsos de presision esseva registrate a normal nivellos de pression arterial, lo que indicava le existentia de ejection ventricular e assi de suction diastolic. Isto esseva augmentate per epinephrina in cordes normal si ben como in cordes insufficiente. Le conclusion esseva formulate que le ventriculo mammalian in stato intacte, travaliante contra normal pressiones aortic, tira un miere quantitate de sanguine a in su cavitate quando le pression de replenation ventricular es zero. Es presentate le conception que in le caso de un intacte corde normal, positive pressiones diastolic transmural intraventricular non exclude le existentia de un *vis a fronte* ventriculo-diastolic.
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