Ventricular Suction in the Turtle

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In two different experimental environments the isolated turtle ventricle was found to fill and eject blood in the absence of a positive filling pressure. It is concluded that the ventricle can "suck" fluid using energy liberated from its wall during diastole.

The question of ventricular "suction," that is "Does energy derived from the ventricle contribute to its filling in diastole?" has been debated from the time Erasistratus to the present and has recently been critically reviewed by Brecher. A recent film and abstract by Bloom offer support of the "suction" concept.

The ultimate proof of suction as a physiologic aid to filling should rest on a demonstration that, in the absence of a filling head, the ventricle develops a drop in intraventricular pressure which is sufficient to and actually does produce inflow. Because of the very low resistance of the A-V orifice the pressure difference necessary for inflow is very small and has not been measurable directly with existing apparatus except when inflow is impeded artificially. The pressure difference, then, must be inferred from the fact of flow.

A positive filling head can be eliminated by the total immersion of the ventricle in fluid at any depth. In this way the filling head at any point within the heart chambers is exactly counter-balanced by the external pressure at that depth. In this way any inflow indicates that the heart wall has been instrumental in producing a lowering of intracardiac pressure and the consequent inflow of fluid.

Two series of experiments on the excised ventricle of the turtle are here presented to throw light on this question.

Methods

Series I. The heart was removed from a turtle and attached to a glass tube which passed through the cut atria into the ventricular cavity. A ligature tied into the atrioventricular groove and around the truncus aortac prevented fluid from entering or leaving the ventricle except through the tube (fig. 1A). This tube passed through a rubber stopper and was connected, by a short rubber connector bent at right angles, to a horizontal serologic pipette. A duplicate pipette connected to a tube passing through the stopper was arranged parallel to the first but was not connected to the heart. These tubes were placed exactly level with each other to provide equal pressure inside and outside the ventricular wall. A bottle filled with Ringer's solution to the complete exclusion of air was then fitted to the stopper to enclose the heart as a plethysmograph. With this arrangement ventricular filling and ejection must be quantitatively reflected by the to and fro movements of the saline meniscus in the "cardiac" pipette. Reciprocal movements in the "plethysmograph" pipette reflect changes in heart volume.

The heart was electrically driven at 12 beats/min. and was immersed in fluid between 10 and 15 C.

Series II. The aorta of a heparinized (200 units) turtle was cannulated with a blunted 13 gage needle, care being taken to avoid damage to the aortic valves. The heart was excised without damage to the A-V valves and immersed in Ringer's solution. In another series of experiments it was found that the aorta of the turtle can be cannulated with the maintenance of valvular competence. A tube connected to the cannula was arranged to deliver ejected fluid at a level which could be adjusted precisely with respect to the level in the beaker in which the heart was immersed (fig. 1C). The ejected fluid was measured by clock and graduate cylinder. In this experiment the level in the receiving beaker determined only the diastolic aortic pressure against which the ventricle must work. The filling was from the beaker of saline in which the whole heart was submerged.
Table 1.—Ejection by Hearts in Series II

<table>
<thead>
<tr>
<th>Ejected fluid ml.</th>
<th>Time min.</th>
<th>Height of outflow level mm.</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>20</td>
<td>7</td>
</tr>
<tr>
<td>15</td>
<td>33</td>
<td>8</td>
</tr>
<tr>
<td>15</td>
<td>27</td>
<td>8</td>
</tr>
<tr>
<td>25</td>
<td>18</td>
<td>8</td>
</tr>
<tr>
<td>24</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>48</td>
<td>17</td>
<td>9</td>
</tr>
</tbody>
</table>

Results

Series I. Under these conditions fluid consistently flowed into the ventricular cavity during diastole. Figure 1B shows that during the first few beats the amount ejected, the amount flowing back into the heart, the residual and diastolic volumes are decreasing toward equilibrium values. In 15 such experiments the average intake for the greatest beat was 0.085 ml. (range 0.05-0.14) and the average for the tenth beat was 0.045 ml.

When the level of the plethysmograph pipette was raised above that of the cardiac pipette (both being kept strictly horizontal) some filling continued until the pressure difference exceeded 4 to 8 mm. of water pressure.

By rotation of the whole apparatus through 180° around the axis of the pipettes it was demonstrated that the heart's behavior was independent of its position and that fluid re-enters the heart whether the heart is above or below the meniscus. This experiment showed further that any density difference between the heart and the saline did not affect the filling.

Series II. Six such tests on 3 hearts are presented in table 1 which shows a small but definite amount of fluid ejected. This was accumulated in amounts large enough to eliminate any question of their being due to change in residual volume.

Discussion

The dynamics of submerged, collapsible containers is somewhat confusing. At first sight the hearts in these experiments appear to be subject to a filling pressure related to their depth. But the same head which might deliver fluid into the cavity is operating on the outside to oppose filling. Therefore only a self-expansion and consequent suction can fill the hearts in these experiments.

In the first series of experiments the filling indicated in figure 1B cannot have been caused by vis a tergo, for in none of these experiments was there a positive external "filling pressure"; in some this value was deliberately made negative. Clearly, during the whole of the phase of diastole in which inflow occurred the pressure in the ventricular
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cavity must have been less than that outside it, or there could have been no flow; but the arrangement of the equipment with an equal fluid head inside and outside the ventricle makes it inevitable that the transmural pressure causing the filling movement must be derived from the ventricular wall. Similarly, in the second series the 3/4 to 3 ml./min. of fluid which were ejected had entered a submerged heart at no external filling pressure. Again the motive force must have been derived from the heart itself since, at whatever depth, any filling pressure is exactly counterbalanced by the hydrostatic pressure on the outside.

These experiments appear to establish that, in certain circumstances, the turtle ventricle contributes to its own filling. Experiments in this laboratory (Brecher 1956) show the same to be true of the dog.

Satisfactory quantitation of this effect has not been achieved but it seems that at 0 filling pressure the heart can fill to the extent of about a tenth of its normal resting stroke; and further, that it can continue to achieve some filling against a pressure applied exterior to the ventricle of about a tenth of the probable normal venous filling pressure. If these quantities are representative, one must say that this effect is small but not necessarily negligible.

The geometry of the heart, no less than these observations, those of Brecher, and other observations in this laboratory, lead us to expect that this effect is more evident in a heart nearly empty of residual blood than it would be in a heart doing significant work and therefore, according to Starling's Law, somewhat distended.

The energy liberated in systole appears only in part as aortic tension potential and in part as the kinetic energy of aortic blood velocity. Most of the energy is lost to observation and is usually thought of as waste heat or inefficiency.

It seems reasonable to suppose that some of this lost energy may be stored as potential energy available for the self-filling here reported. We have no evidence whether this storage may be in the connective tissue "skeleton" of the heart or in the muscle fibers themselves; nor can we guess whether it may be stored as elastic potential of deformation or as chemical energy to be released during diastole. The former suggestion would seem to involve less departure from current concepts.

REFERENCES

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Circ Res. 1956;4:724-726
doi: 10.1161/01.RES.4.6.724

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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World Wide Web at:
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