In 1906 Henderson\(^1\) stated that the atria function as elastic reservoirs and not as force pumps. Since that time many estimates have been made of the quantitative role of atrial systole in cardiac performance. Recently the transport function of the atrium has been emphasized.\(^2\) In the present study the influence of atrial systole on forward, or effective, ventricular stroke volume was investigated by comparing ventricular function with and without prior atrial systole. A preliminary report\(^3\) of this work has been presented.

**Methods**

Experiments were performed on mongrel dogs of both sexes. The animals were anesthetized lightly with thiopental sodium, injected intravenously, followed by chloralose (50 to 100 mg/kg) in propylene glycol. Intermittent positive pressure breathing was maintained by a Harvard respiratory pump. An initial dose of 75 mg heparin was injected intravenously and subsequent injections of 10 mg were administered as needed to prevent blood clotting.

The experimental preparation employed was a right heart bypass as shown in figure 1. The neck was opened through a midline incision, the trachea cannulated (T.C.), and both vagi sectioned in the midcervical region. The chest was opened widely through a sternal split. A large cannula was introduced into the right atrium through the azygos vein. Loose ligatures were placed around the superior and inferior venae cavae. One large cannula was introduced through the right external jugular vein and passed to a point just below the ligature around the inferior vena cava. The main pulmonary artery was dissected free from the aorta and a ligature was placed loosely around it. A cannula was placed through the outflow tract of the right ventricle into the main pulmonary artery. An accessory blood oxygenator (Bl.Ox.), containing a heat exchanger (H.E.), and the pump system were filled with blood obtained from donor dogs that had been anesthetized lightly with thiopental sodium. The right heart bypass was established by opening the three venous
The electrodes were connected to separate Grass sutured to the right atrial appendage and a ligature around the superior vena cava, inferior vena cava, and pulmonary artery were tied. Next, a right atriotomy was performed and complete heart block was produced by tying a ligature around the bundle of His. Also, the sino-atrial node was excised and the atrium sutured.

One bipolar pacing electrode (P.EI.) was sutured to the right atrial appendage and a second bipolar pacing electrode (P.EL.) was sutured to the right ventricular outflow tract. The electrodes were connected to separate Grass stimulators (model S4) which were interconnected through a delay network. The pacing stimuli were 1 to 5 msec in duration and had liminal intensity. One bipolar electrode was sutured to the left atrium (A.EI.) and one to the left ventricle (V.EI.) in order to record electrograms from each of these chambers.

The rates of atrial and ventricular contraction were controlled by the Grass stimulator connected to the right atrium. Cardiac output was determined by the setting of the Sigmamotor pump (S.P.). Aortic pressure was regulated by a Starling resistance (S.R.) placed in the aortic arch of the aorta via the right common carotid shunt.

In each study the cardiac output and aortic pressure were selected. Next, the rate of the impulse generator connected to the right atrium was set at either 60 or 90/min depending on the spontaneous atrial rate. The delay network connecting the two impulse generators was adjusted so that the time between left atrial and left ventricular systole (A-V8 interval) was between 60 and 80 msec. Atrial asystole was induced without affecting ventricular stimulation by withdrawing the atrial pace. By this means a ventricular ejection preceded by an atrial systole was followed by a ventricular ejection not pre-

P23Db transducer (Tr.) connected to a large bore metal cannula which was inserted into the arch of the aorta via the right common carotid artery. Left atrial pressure was measured by a Statham SF1 transducer (Tr.) introduced through the appendage and left ventricular pressure was measured by a Statham SF1 transducer (Tr.) introduced through the apex dimple. Aortic flow was metered by a Medicor sine wave electromagnetic flow probe (E.M.F.) placed around the root of the aorta. All values were simultaneously recorded on an Electronics for Medicine DR8 photographic recorder. Forward or effective ventricular stroke volume (EVSV) was determined by planimetric integration of the flow tracing.

Aortic pressure was regulated by a Statham SF1 transducer (Tr.) introduced through the apex dimple.

TABLE 1

<table>
<thead>
<tr>
<th>Dog</th>
<th>Expt.</th>
<th>A</th>
<th>B</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>no.</td>
<td>HR = 60 to 90</td>
<td>HR = 120 to 150</td>
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<tr>
<td></td>
<td>no.</td>
<td>EVSVA</td>
<td>ECSVb</td>
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<td>I</td>
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</tr>
<tr>
<td>II</td>
<td>1</td>
<td>13.8</td>
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</tr>
<tr>
<td>III</td>
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<td>9.1</td>
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<tr>
<td>IV</td>
<td>1</td>
<td>16.2</td>
<td>12.9</td>
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<tr>
<td>VIII</td>
<td>1</td>
<td>8.5</td>
<td>6.7</td>
</tr>
</tbody>
</table>

Mean ± SD

HR: heart rate per minute; EVSVA: effective ventricular stroke volume in cc with an atrial systole; ECSVb: effective ventricular stroke volume in cc without an atrial systole.
ceded by an atrial systole. At least four atrial
asystoles were induced at each rate. Averaged
values of EVSV of the ventricular ejection pre-
ceded by an atrial systole were compared with
those of the ventricular ejection not preceded by
an atrial systole. The rate of stimulation was
then increased to the desired levels with cardiac
output and aortic pressure held constant and
the procedure repeated.

Results

The influence of atrial systole on effective
ventricular stroke volume (EVSV) at three
different heart rates when cardiac output and
mean aortic pressure were held constant in
18 experiments performed on eight dogs is
shown in table 1. In group A are the results
obtained with the heart rate between 60 and
90/min. The average stroke volume was
15.0 ± 4.6 cc in the ventricular ejections
which were preceded by atrial systole and
was 12.0 ± 3.9 cc in the ventricular ejections
which were not preceded by atrial systole.
The per cent change in EVSV at this heart
rate was -20 ± 5. In group B are the results
obtained with the heart rate between 120 and
150/min. The average EVSV was 10.0 ± 2.9 cc
in the ventricular ejections which were pre-
ceded by atrial systole and was 7.7 ± 2.4 cc
in the ventricular ejections which were not
preceded by atrial systoles. The per cent
change in EVSV at this heart rate was -23
± 9. In group C are the results obtained with
the heart rate between 180 and 210/min. The
average EVSV was 7.5 ± 2.1 cc in the ven-
tricular ejections which were preceded by
atrial systoles and was 4.6 ± 1.5 cc in the
ventricular ejections which were not pre-
ceded by atrial systoles. The per cent de-
crease in EVSV at this heart rate was -37
± 12. There was no significant difference in
the per cent change in EVSV that occurred
between group A and group B. However,
there was a significant difference in the per
cent change in EVSV that occurred between
group A and group C (P < 0.001) and be-
tween group B and group C (P < 0.001).

Typical recordings in one experiment of
the influence of atrial systole on effective
ventricular stroke volume (EVSV) at three
different heart rates with cardiac output and
mean aortic pressure held constant are shown
in figures 2, 3, and 4. At a heart rate of
90/min (fig. 2) the atrial bipolar electrogram

![Atrial asystole at heart rate of 90/min. AF: aortic flow; AP: aortic pressure; A.EL: left
atrial bipolar electrogram; V.EL: left ventricular bipolar electrogram. Time lines at one-second
intervals.](image)
(A.El.) and ventricular bipolar electrogram (V.El.) demonstrate that the first and second ventricular systoles were each preceded by an atrial systole, and the third ventricular systole was not preceded by an atrial systole. The effective ventricular stroke volume was 20.8 cc in the last ventricular ejection preceded by an atrial systole and 17.1 cc in that

![Diagram](attachment:image.png)

**FIGURE 3**

Atrial asystole at heart rate of 150/min. Symbols as in figure 2.

![Diagram](attachment:image.png)

**FIGURE 4**

Atrial asystole at heart rate of 210/min. Symbols as in figure 2.

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without an atrial systole. An 18% decrease in EVSV occurred at this heart rate.

At a heart rate of 150/min (fig. 3) the first three ventricular systoles were each preceded by an atrial systole and the fourth or last ventricular systole was not preceded by an atrial systole. The stroke volume was 13.9 cc in the last ventricular ejection preceded by an atrial systole and 10.8 cc in that without an atrial systole. A 22% decrease in effective ventricular stroke volume occurred at this heart rate.

At a heart rate of 210/min (fig. 4) the first three ventricular systoles were each preceded by an atrial systole, and the last three ventricular systoles were not preceded by atrial systoles. The EVSV was 10.0 cc in the last ventricular ejection preceded by an atrial systole and 6.3 cc in the first ventricular ejection without an atrial systole. A 37% decrease in EVSV occurred at this heart rate. Since a constant cardiac output was being pumped into the pulmonary artery, the EVSV of the last two ventricular ejections, which were also without preceding atrial systoles, increased to 7.8 and 8.7 cc. Other studies have shown that the increase in EVSV without the occurrence of atrial systole is accomplished by a higher mean left atrial pressure.

The dynamic effects of atrial systole on left atrial and left ventricular pressures were also investigated and typical recordings are shown in figures 5 and 6. The results obtained at a slow heart rate are shown in figure 5. Left ventricular pressure was recorded at two sensitivities, the lower one calibrated in mm Hg and the higher one in cm H2O, in order to emphasize left ventricular diastolic pressure (LVDP). Left atrial pressure (LAP) was recorded at the same sensitivity as the higher sensitivity left ventricular pressure (LVDP). The EVSV decreased from 19.3 cc when atrial systole was present to 14.5 cc when atrial systole was absent (a decrease of 25%). The a-wave, c-wave, x-descent, and v-wave were present in the ejection in which ventricular systole was preceded by an atrial systole. The a-wave

![Dynamic effects of atrial asystole at slow heart rate. LVP: left ventricular pressure; LAP: left atrial pressure; LVDP: left ventricular diastolic pressure; AF: aortic flow; A.EL: left atrial bipolar electrogram; V.EL: left ventricular bipolar electrogram. Time lines at one-second intervals. For description see text.](http://circres.ahajournals.org/figs/51.jpg)

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was absent and there was no x-descent when atrial systole was absent. The dashed line indicates the initiation of activation of the left ventricular electrogram. The points at which left ventricular end diastolic pressure was measured are indicated by arrows and correspond in time to the initiation of activation of the left ventricular electrogram. Left ventricular end diastolic pressure was 5 cm H2O higher in the ventricular ejection preceded by an atrial systole. Also in the beat with an atrial systole, the atrioventricular pressure reversal occurred 21 msec before the initiation of activation of the left ventricular electrogram, and in the beat without an atrial systole it occurred at the same time as the initiation of activation of the left ventricular electrogram.

Similar results were obtained at high heart rates as shown in figure 6. The EVSV decreased from 7.3 cc when atrial systole was present to 3.6 cc when atrial systole was absent (a decrease of 50%). The a-waves were prominent in the abbreviated diastoles of the ejections having atrial systoles and absent in the diastole of the ejection not having an atrial systole. Small x-descents were present in the beats having atrial systoles and the x-descent was reduced in the beat not having an atrial systole. Left ventricular end diastolic pressure was 4 cm H2O higher in the ventricular ejection preceded by an atrial systole. Also in the beats with atrial systoles, the atrioventricular pressure reversal occurred about 16 msec before initiation of activation of the left ventricular electrogram, and in the beat without an atrial systole it occurred at the same time as the initiation of activation of the left ventricular electrogram.

Discussion

There have been numerous studies of atrial function in the past. Some have assessed it in terms of ventricular filling, preclosure of the atrioventricular valve, and the relation between mean left atrial and left ven-

*FIGURE 6*

*Dynamic effects of atrial asystole at fast heart rate. Symbols as in figure 5. For description see text.*
atrial end diastolic pressure. In other studies cardiac output has been compared during effective and ineffective atrial activity.

Snyder and Wood determined the change in cardiac output that occurred in closed chest innervated dog preparations when atrial systole was made ineffective. They found that an ineffective atrial systole produced a 5 to 10% decrease in cardiac output over a wide range of heart rates (60 to 200 beats/min). Later, Sellers et al. studying dogs with chronic cardiac denervation but without heart block, found that a 15% decrease in cardiac output occurred when atrial systole was made ineffective over a comparable heart rate range. Skinner et al. found that maximal reduction in aortic flow approximated 30% with atrial systole absent or abnormally placed.

In the present study the experimental preparation allowed control of many variables which are difficult to control in the intact but admittedly more normal animal. The quantitative role of atrial systole was evaluated by determining its effect on forward or effective ventricular stroke volume at different heart rates with a constant cardiac output and aortic pressure. When ventricular ejections were not preceded by atrial systole, EVSV decreased between HR 60 to 90/min, 23 ± 9% between HR 120 to 150/min and 37 ± 12% between HR 180 to 210/min. In these experiments atrial systole contributed importantly in determining EVSV.

This drop in EVSV when there is no preceding atrial systole could be due to at least two mechanisms. These are: 1) The ventricle may fill less, have a lower left ventricular end diastolic pressure and volume and therefore eject less stroke volume; or 2) the ventricle may fill normally, but eject some volume backward into the atrium when the mitral valve closes. Evidence that the first mechanism is present was given in figures 5 and 6. Left ventricular end diastolic pressure was lower in the ejections without an atrial systole, indicating less ventricular filling. Evidence that the second mechanism is present was also shown in these figures. When atrial systole was absent the x-descent in the atrial pressure pulse was reduced or not present. Further, the atioventricular pressure reversal occurred before the initiation of activation of the left ventricular electrogram in the ejections preceded by atrial systoles and at the time of initiation of activation of the left ventricular electrogram in the ejections not preceded by atrial systoles. This suggests that mitral valve closure occurred earlier in the ejections with an atrial systole and thereby may have prevented some degree of early mitral regurgitation. Thus both mechanisms appear to play a role in the decreased EVSV that occurs when atrial systole is absent.

Summary
The influence of atrial systole on forward or effective ventricular stroke volume at different heart rates and at constant cardiac output was investigated in an open chest dog preparation. The procedure employed allowed a ventricular ejection preceded by an atrial systole to be followed by one which was not. A comparison was made between the effective ventricular stroke volumes of these two ejections. The observations support the view that atrial systole plays a significant role in determining effective ventricular stroke volume and that its influence is greater at higher heart rates when cardiac output is kept constant and stroke volume decreases. The data indicate further that the decrease in effective ventricular stroke volume is due to both a decreased ventricular filling and initial atioventricular regurgitation.

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