Atrial Activity and Systemic Blood Pressure during Anesthesia in Man

By Myron B. Laver, M.D., and Herman Turndorf, M.D.

Recent studies in the laboratory animal and in man have revived earlier interest in the contribution of atrial contraction to ventricular performance,1-4 and the influence of autonomic tone on displacement of the cardiac pacemaker.5-8 However, the hemodynamic consequences of acute alterations in the synchrony of atrial and ventricular contraction have never been clearly established in normal man. Several reports have demonstrated an improvement of cardiac output following conversion of atrial fibrillation to normal sinus rhythm.9,10 These studies, however, were conducted on patients with heart disease and a long-standing irregular rhythm, resulting from atrial fibrillation.

The present report represents an attempt to evaluate the significance of atrial contraction to ventricular performance during anesthesia in man. A recent chance finding of marked changes in arterial blood pressure following transition from sinus to nodal rhythm, has afforded a method for studying the effect of synchronous atrioventricular contraction on performance of the heart. The original observation indicated that the recorded alterations in arterial pressure were associated with a wandering pacemaker, low pressure occurring when the pacemaker site was located in the atrioventricular node and control pressures returning as the impulse was again initiated in the sinus node.

Methods

The present report includes data from 10 patients, aged 40 to 63, with no known symptoms attributable to cardiovascular disease, who were operated upon under halothane anesthesia and in whom a state of hypothermia was achieved by surface cooling. The preoperative electrocardiograms were considered to be normal. Premedication included atropine, pentobarbital, and chlorpromazine, given at least 1 hour prior to induction.

After a dose of 100 to 200 mg. of thiopental given intravenously, 2 per cent halothane was delivered from a calibrated or copper-kettle vaporizer* and given in 5 liters of oxygen via a face mask and a semi-closed breathing circuit. Five to 10 minutes later, repeated doses of 6 mg. of d-tubocurare were administered until complete apnea had been produced. An endotracheal tube was passed and ventilation was continued, without halothane, with use of a mixture of 4 liters of nitrous oxide and 2 liters of oxygen, and a pressure-controlled respirator was adjusted to deliver a tidal volume approximately 30 per cent higher than calculated from the Radford nomogram. A no.-18 Courmand needle was inserted into the radial or brachial artery and a no.-5 plastic ureteral catheter was threaded percutaneously from an antecubital vein into the superior vena cava or the right atrium. Position of the tip was identified by the appearance of the pulsatile venous pressure recording. Esophageal temperatures were measured with a thermistor probe taped to an esophageal electrocardiographic electrode and passed into the esophagus until a biphasic P wave indicated that the tip was located immediately behind the left atrium. This method has permitted quantitative comparison of temperatures among patients by providing a readily reproducible position in proximity to the heart. Such positioning may vary within wide limits if made by length measurements alone.

The arterial and central venous pressure tracings were recorded with Sanborn no. 267B transducers placed at a level midline between the anterior and posterior aspects of the chest. Surface cooling was achieved by circulating a precooled alcohol-water mixture from an electronically controlled unit through vinyl blankets.†

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Segment of record taken during a period of transition from sinus to nodal rhythm (A, upper), and from nodal to sinus rhythm (B, lower). Note the alteration in the venous pressure pattern and change in arterial systolic pressure as the P wave moves into or out of the QRS complex (change in systolic pressure in A = 20 mm. Hg, in B = 50 mm. Hg). The P-R interval increased to 0.12 second in B within seven additional beats not shown on the tracing. The airway pressure tracing indicates that the recording was taken during a period of apnea. The positive pressure reading is due to slow inflow of anesthetic gases during period of apnea. Paper speed: 25 mm./sec.
Arterial oxygen and carbon dioxide tensions were measured with modified Clark and Severinghaus electrodes housed in a constant temperature bath. The recording equipment for blood gas analysis consisted of a Sanborn pH amplifier model 350-3600 and an oxygen plug-in cell model 350-11 used with a low level amplifier.

Electrocardiographic monitoring was provided by an esophageal electrode and the standard limb lead II.

When all preparations were completed, anesthesia was changed to a halothane (0.3 to 1.0 per cent)-oxygen mixture, and maintained on this combination throughout the remainder of the procedure.

Atrioventricular dissociation was produced during the early stages of surgery by gradually decreasing the inspired halothane concentration immediately following the skin incision to a minimum of 0.3 per cent, or until the wandering pacemaker became evident on the electrocardiogram. The characteristic change from sinus to nodal rhythm, as seen in figure 1, consists of a progressive diminution of the P-R interval with the P waves gradually moving into the QRS complex. The change was not associated with a significant alteration in heart rate. If halothane is discontinued at this point, the P wave will be seen to move frequently in and out of the QRS complex while the characteristic arterial and venous pressure changes will be present each time the pacemaker site is displaced up or down within the atrium. Ultimately, the sinus node rhythm and the P-R interval become fixed and the arterial blood pressure continues to rise, indicative of the light anesthesia. Occasional ventricular extrasystoles were noted while the patients were being ventilated with 100 per cent oxygen but these disappeared promptly when halothane was reintroduced in the inspired mixture. Again, if these concentrations were increased gradually from 0.3 to 1.0 per cent, the pattern of atrioventricular dissociation reappeared. Frequently this sequence of events could be repeated several times, each occasion being accompanied by the described pressure changes.

In order to evaluate the possibility that the venous pressure tracings during nodal rhythm were due to partial tricuspid regurgitation, the interval between the R peak of the electrocardiogram and the C wave on the venous pressure tracing was measured in 10 complexes prior to the onset of nodal rhythm. This value was measured back from the first major peak of the altered venous pressure tracing, i.e., during nodal rhythm, and found to correspond exactly to the time of the R wave on the electrocardiogram. A similar analysis was applied to the interval between beginning of the P wave and the A peak of the venous pressure tracing. This time value was applied to the second major peak noted on the venous tracing during nodal rhythm and found to correspond exactly to the distance between

![Figure 2](http://circ.ahajournals.org/)

Figure 2

*Hemodynamic changes seen during the transition from sinus to nodal and back to sinus rhythm as they appear when recorded at slow paper speed. From top to bottom: arterial pressure, mean central venous pressure (variations due to changes in respiratory pressures), standard limb lead II of the electrocardiogram. Paper speed: 25 mm./sec. (fast segment), 0.5 mm./sec. (slow segment). Note the absence of P waves in the central segment and their re-appearance in the right-hand portion of the record. Arrow at top left indicates onset of nodal rhythm; arrow at right marks the return of normal sinus rhythm. Details of this type of change can be seen in figures 1A and 1B.*
Table 1
Data from Anesthetized Patients during Alterations in Atrioventricular Synchrony

<table>
<thead>
<tr>
<th>No.</th>
<th>Age &amp; sex</th>
<th>Operation</th>
<th>No. spontaneous changes</th>
<th>Temperature range of changes °C</th>
<th>Av. change in systolic BP NSR to NR mm. Hg %</th>
<th>Av. change in venous pressure peak NSR to NR mm. Hg %</th>
<th>Av. change in R-R interval NSR to NR sec. %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>57 F</td>
<td>Craniotomy aneurysm</td>
<td>14</td>
<td>31°-35°</td>
<td>−33.5</td>
<td>−25.8</td>
<td>Not recorded</td>
</tr>
<tr>
<td>2</td>
<td>40 M</td>
<td>Craniotomy aneurysm</td>
<td>16</td>
<td>29°-30°</td>
<td>−10.1</td>
<td>−10.4</td>
<td>+ 3                                      + 39.3</td>
</tr>
<tr>
<td>3</td>
<td>58 F</td>
<td>Meso-caval shunt</td>
<td>13</td>
<td>28°-30°</td>
<td>−9.3</td>
<td>−12</td>
<td>+3                                      + 37.1</td>
</tr>
<tr>
<td>4</td>
<td>63 F</td>
<td>Craniotomy aneurysm</td>
<td>8</td>
<td>29°-31°</td>
<td>−15.6</td>
<td>−17.3</td>
<td>Not recorded</td>
</tr>
<tr>
<td>5</td>
<td>58 F</td>
<td>Craniotomy tumor</td>
<td>1</td>
<td>26°</td>
<td>−20</td>
<td>−23.6</td>
<td>+ 2.5                                    + 20</td>
</tr>
<tr>
<td>6</td>
<td>57 F</td>
<td>Craniotomy tumor</td>
<td>9</td>
<td>29°-34°</td>
<td>−21.7</td>
<td>−16.6</td>
<td>Not recorded</td>
</tr>
<tr>
<td>7</td>
<td>52 F</td>
<td>Craniotomy tumor</td>
<td>4</td>
<td>29°-30°</td>
<td>−18.7</td>
<td>−13</td>
<td>Not recorded</td>
</tr>
<tr>
<td>8</td>
<td>56 F</td>
<td>Craniotomy tumor</td>
<td>5</td>
<td>34°</td>
<td>−34.5</td>
<td>−14.2</td>
<td>+ 6.4                                    + 31.2</td>
</tr>
<tr>
<td>9</td>
<td>51 F</td>
<td>Craniotomy tumor</td>
<td>9</td>
<td>31°-34°</td>
<td>−36.7</td>
<td>−20.4</td>
<td>+10.6                                    + 158</td>
</tr>
<tr>
<td>10</td>
<td>53 F</td>
<td>Craniotomy tumor</td>
<td>5</td>
<td>34°-34°</td>
<td>−26</td>
<td>−17.3</td>
<td>+ 4.2                                    + 93</td>
</tr>
</tbody>
</table>

|       | Mean      | S.D. ± 9.25 ± 4.75 | ± 2.95 ± 49.2 | ± 0.013 ± 4.32 | ± 0.004 ± 1.37 | ± 2.95 ± 49.2 | ± 0.013 ± 4.32 | ± 0.004 ± 1.37 | ± 2.95 ± 49.2 | ± 0.013 ± 4.32 | ± 0.004 ± 1.37 | ± 2.95 ± 49.2 | ± 0.013 ± 4.32 | ± 0.004 ± 1.37 |

*Normal sinus to nodal rhythm.
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the R wave during nodal rhythm and the aforementioned second peak.

Results

The arterial and venous pressure changes at the time of the shift from sinus to nodal rhythm are seen in figures 1 and 2. As figure 2 indicates, these changes resulted in a new and stable pressure level within 4 to 8 beats, subsequently maintained throughout the period of nodal rhythm. Figure 2 also demonstrates the hemodynamic pattern seen during slow-speed recording with the detailed counterpart illustrated in figures 1A and 1B.

Table 1 indicates the magnitude of these changes during variations between sinus and nodal rhythm in 10 anesthetized patients. These values were obtained by averaging the data from five pressure and electrocardiographic complexes immediately before and after the change in pacemaker site, all being recorded during periods of apnea. Absence of atrial contraction prior to ventricular systole was associated with a mean fall of arterial systolic pressure of 17.1 per cent and a mean rise in the peak venous pressure of 63.1 per cent, both changes being statistically significant (p < 0.02). The changes seen during the pacemaker shift were all in the same direction in each of the 84 instances recorded, but the magnitude of the pressure alteration, particularly the arterial, varied considerably and tended to be larger when the arterial pressure during sinus rhythm was high. The venous pressure values recorded in table 1 represent peak venous pressures, the characteristic tracing being shown to the right in figure 1A and to the left in 1B.

Both venous and arterial pressure changes were statistically significant (p < 0.02). The alterations in the R-R interval when a lower pacemaker became established were not statistically significant.

The relationship between the duration of the P-R interval and other measured parameters was studied by analyzing 250 consecutive complexes of the electrocardiogram, and of the arterial and venous pressures in two patients (nos. 9 and 10) during a period when the pacemaker shift occurred spontaneously on several occasions. Complexes distorted by passive movement were excluded from analysis. The tracings covered periods of 20 and 13 minutes, respectively, and were obtained prior to commencement of the intracranial part of the surgery, so that direct mechanical stimulation of the central nervous system would not provide a possible source of the arrhythmia. The data (table 2) are graphically illustrated in figure 3 where values for venous and arterial pressures and heart rate are plotted against the P-R interval. These show the inverse relationship between systolic and venous pressures as the onset of atrial systole tended to coincide with that of the ventricles.

Measurements of arterial carbon dioxide tensions indicated uniform hyperventilation throughout the period of the study (range: 12 to 37 mm. Hg). The arterial oxygen tensions were lower than expected in patients breathing high concentrations of oxygen but were not unusual in view of the venous admixture known to occur in the lungs during prolonged intermittent positive pressure ventilation in paralyzed patients (range: 192 to 460 mm. Hg).

The pacemaker shift was noted over a wide temperature range (26.7 to 34.7 C.) It has been noticed subsequently in normothermic individuals with similar pressure alterations.

Discussion

The present data suggest that rapidly reversible atrioventricular dissociation in anesthetized man can be produced readily and utilized to study the contribution of the atria during ventricular diastole and to closure of the atrioventricular valves. The conditions found effective for producing this change include halothane-oxygen anesthesia with inspired halothane concentrations well below 1 per cent; controlled ventilation with the use of d-tubocurare to produce muscle paralysis; and an exogenous stimulus, most conveniently supplied by the beginning of surgery. Notwithstanding the many unknown factors in the sequence of events, inhalation anesthesia
perms rapid variation in the blood levels of the responsible agent and allows pharmacologic control not obtained with drugs administered by other routes. Possibly, the sustained, controlled ventilation represents the key factor in the readiness with which a wandering pacemaker can be demonstrated.

Failure of atrial systole to occur within a certain time limit prior to ventricular contraction has a two-fold effect on ventricular performance. First, it is probable that atrial contribution to ventricular filling is abolished and this is immediately followed by a drop in arterial blood pressure. The data indicated that, over a wide range of systolic arterial pressures, this fall averaged 17.1 per cent of the pressure recorded during sinus rhythm. Jochim\(^6\) measured the time lapse between atrial and ventricular systole and found that a period of maximal atrial contribution to ventricular filling could be defined when both chambers contracted in rapid succession. Gesell\(^12\) and Wiggers and Katz\(^13\) have defined the volume contribution of the atria to ventricular filling in isolated hearts, but equivalent data from man have not been available, except in patients with atrial fibrillation.\(^10\) Figure 3 shows that the fall in systolic pressure was greater than the fall in diastolic pressure; no alterations occurred in the heart rate. Randall and McNally\(^14\) described a similar response to stellate ganglion stimulation in anesthetized man.

It is of some interest that the venous pressure patterns taken during nodal rhythm suggest a brief functional tricuspid regurgitation. In considering the magnitude of the venous pressure peak during nodal rhythm, we note that the last two patients listed in table 2 exhibited a venous pressure rise considerably higher than the remainder. Probably, the less extensive elevations represent atria and ventricles contracting simultaneously, with atrial systole occurring in the presence of closed atrioventricular valves. Inspection of the venous pressure tracings in these patients revealed the presence of a single peak during nodal rhythm, which probably repre-

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### Table 2

**Analysis of P-R Interval**

<table>
<thead>
<tr>
<th>Patient no. 1</th>
<th>0</th>
<th>0.02</th>
<th>0.04</th>
<th>0.06</th>
<th>0.08</th>
<th>0.10</th>
<th>0.12</th>
<th>0.14</th>
<th>0.16</th>
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<tr>
<td><strong>Systolic BP (mm. Hg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>N</td>
<td>19</td>
<td>3</td>
<td>5</td>
<td>12</td>
<td>17</td>
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<td>43</td>
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<td>23</td>
</tr>
<tr>
<td>(\bar{X})</td>
<td>123</td>
<td>128</td>
<td>125</td>
<td>135</td>
<td>154</td>
<td>153</td>
<td>153</td>
<td>163</td>
<td>167</td>
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<tr>
<td>S.D.</td>
<td>±0.47</td>
<td>±4.72</td>
<td>0</td>
<td>±10</td>
<td>±9.9</td>
<td>±8.9</td>
<td>±9.5</td>
<td>±11.2</td>
<td>±9.4</td>
</tr>
<tr>
<td>S.E.</td>
<td>±0.6</td>
<td>±2.7</td>
<td>0</td>
<td>±2.9</td>
<td>±2.4</td>
<td>±1.8</td>
<td>±1.5</td>
<td>±1.5</td>
<td>±1.9</td>
</tr>
<tr>
<td><strong>Diastolic BP (mm. Hg)</strong></td>
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<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>N</td>
<td>21</td>
<td>3</td>
<td>6</td>
<td>15</td>
<td>17</td>
<td>28</td>
<td>47</td>
<td>55</td>
<td>25</td>
</tr>
<tr>
<td>(\bar{X})</td>
<td>79</td>
<td>80</td>
<td>80</td>
<td>82</td>
<td>88</td>
<td>87</td>
<td>88</td>
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<tr>
<td>S.D.</td>
<td>±2.64</td>
<td>0</td>
<td>0</td>
<td>±2.52</td>
<td>±2.32</td>
<td>±2.72</td>
<td>±4.02</td>
<td>±3.66</td>
<td>±4.87</td>
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<tr>
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<td>0</td>
<td>±0.65</td>
<td>±0.57</td>
<td>±0.51</td>
<td>±0.59</td>
<td>±0.49</td>
<td>±0.93</td>
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<tr>
<td><strong>Venous pressure max. (mm. Hg)</strong></td>
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<td></td>
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<tr>
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<td>15</td>
<td>35</td>
<td>34</td>
<td>48</td>
<td>51</td>
<td>26</td>
</tr>
<tr>
<td>(\bar{X})</td>
<td>9</td>
<td>10.3</td>
<td>8.8</td>
<td>7.5</td>
<td>7</td>
<td>5.9</td>
<td>5</td>
<td>4.2</td>
<td>4.6</td>
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<tr>
<td>S.D.</td>
<td>±0.68</td>
<td>±1.87</td>
<td>±0.32</td>
<td>±0.83</td>
<td>±0.92</td>
<td>±0.83</td>
<td>±0.56</td>
<td>±0.42</td>
<td>±0.75</td>
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<td>±1.07</td>
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<td>±0.21</td>
<td>±0.16</td>
<td>±0.14</td>
<td>±0.08</td>
<td>±0.06</td>
<td>±0.15</td>
</tr>
<tr>
<td><strong>R-R interval (sec.)</strong></td>
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<td></td>
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<tr>
<td>N</td>
<td>22</td>
<td>2</td>
<td>7</td>
<td>17</td>
<td>38</td>
<td>27</td>
<td>46</td>
<td>53</td>
<td>23</td>
</tr>
<tr>
<td>(\bar{X})</td>
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<td>0.68</td>
<td>0.67</td>
<td>0.69</td>
<td>0.67</td>
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<tr>
<td>S.D.</td>
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<td>±0.03</td>
<td>±0.035</td>
<td>±0.026</td>
<td>±0.023</td>
<td>±0.02</td>
<td>±0.022</td>
<td>±0.024</td>
<td>±0.029</td>
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<tr>
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<td>±0.02</td>
<td>±0.013</td>
<td>±0.006</td>
<td>±0.005</td>
<td>±0.004</td>
<td>±0.003</td>
<td>±0.004</td>
<td>±0.007</td>
</tr>
</tbody>
</table>

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sents a fusion of the A and C waves seen during sinus rhythm. The pattern seen to the right in figure 1A and to the left in 1B is of considerably greater magnitude and is invariably associated with two peaks. The second peak according to our analysis probably represents atrial systole following contraction of the ventricles. A possible explanation is offered by the fact that atrial systole produces an increase in blood flow past the atrioventricular valves, thus leading to apposition of the valve leaflets secondary to a Venturi effect. Absence of this increased flow, as in complete heart block, results in ventricular systole being initiated with the leaflets widely separated, thus producing an initial regurgitant flow and the above venous pressure tracing during the early part of ventricular contraction.

The reflection of atrial contraction into the venous system during simultaneous contraction of atria and ventricles, was recognized in 1904 by Rihl. The effect of increased atrial pulsation independently of the ventricles has been shown to result in a fall in cardiac output. Daley, McMillan, and Gorlin have shown that acutely induced atrial fibrillation leads to valvular regurgitation, and similar changes have been demonstrated in patients with congestive heart failure.

Recent work in the isolated dog heart has emphasized the importance of atrial contraction to atrioventricular valve closure, and our data in man would appear to support these studies. Particularly pertinent is the work of Gelb, Donoso, and Moscovitz in which atrioventricular dissociation was produced in dogs by acute digitalization. The right atrial pressure patterns shown by these authors are strikingly similar to our own and, particularly in view of their magnitude, may actually represent regurgitation rather than atria contracting against a closed valve, as these authors suggest.

The alteration in atrioventricular synchrony was elicited during very light anesthesia and was initiated by external stimuli. We have no data on the nature of this stimu-
lus. Possibly, these arrhythmias are secondary to acutely altered autonomic tone, since they are similar to the findings of other workers who stimulated the stellate ganglion. Sarnoff et al. demonstrated the close relationship between the central nervous system and atrioventricular dynamics in the isolated dog heart. Rothenberger and Winterberg, by stimulating thoracic sympathetic fibers in the dog, were able to produce changes in rhythm similar to the ones presented in our study. The absence of cardioacceleration was attributed by these authors to the simultaneous stimulation of vagal fibers that course with those of the sympathetic to the heart and are difficult to separate anatomically.

It is likely that sympathetic stimulation superimposed on minimal anesthesia results in activation of pacemakers below the sinoatrial node. Expression of this altered autonomicity in man probably presupposes the absence of compensatory reflexes from the respiratory tract and explains why these arrhythmias are readily demonstrated during light anesthesia with complete muscle paralysis.

The presence of functional regurgitation in patients with an allegedly asymptomatic cardiovascular system suggests that great caution must be utilized in attributing falls in cardiac output to myocardial depression during periods of supraventricular arrhythmia, since part of the stroke volume is apparently returned into the atria. It also emphasizes the need for careful assessment of pressure recordings when ventricular function curves are drawn to evaluate the myocardial effect of anesthetic agents. As shown in the present study, both mean atrial pressure and stroke volume can be altered in the presence of an adequately functioning ventricle whenever inadequate valve closure results in loss of effective cardiac output by regurgitation.

Quantitation of atrial contribution to ventricular filling has been obtained in a variety of preparations. The methods set forth by the present study may allow further evaluation of this problem in man.
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Summary

Atrial contribution to ventricular performance was evaluated during halothane anesthesia in man by a method whereby atrioventricular dissociation is readily induced. Three conditions are outlined that facilitate production of this arrhythmia in man: general anesthesia with a halothane-oxygen mixture by use of an average inspired halothane concentration of 0.5 per cent; controlled ventilation following muscle paralysis with d-tubocurare; and an exogenous stimulus, most easily provided by the onset of surgery. The last factor is probably mediated via direct sympathetic stimuli to the heart.

The change from sinoatrial to atrioventricular nodal rhythm was associated with a fall in arterial pressure and a rise in central venous pressure; the heart rate remained unaltered. The changes in the systemic and central venous pressures were attributed to failure of atrial systole to contribute to ventricular filling during diastole.

It is suggested that tricuspid regurgitation may occur during nodal rhythm, since synchronous atrioventricular contraction is required for adequate closure of the atrioventricular valve. Further studies of this problem in anesthetized man with this particular pharmacologic tool are indicated.

References


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