Effects of Bladder Distention on Pulmonary Vascular Bed

By Alberto Agrest, M.D., and Aquiles J. Roccoroni, M.D.

Bladder distention is known to produce vascular and other autonomic reactions, which are much more evident in paraplegics than in normals and is more important the higher the level of spinal cord transection. These autonomic reactions include increased arterial pressure caused by systemic vasoconstriction, headaches, flushing of the face, bradycardia and sweating. We planned to investigate if the pulmonary vascular bed was included in this reaction and so demonstrate an example of nervous control of pulmonary vessels, a long debated subject. Our results seem to offer a good argument in favor of this nervous control and demonstrate that the pulmonary vascular bed participates in the general effects of bladder distention.

Methods

Hemodynamic studies were performed in 2 paraplegic patients. One of them (case 1), a 22-year-old male, had a spinal cord transection at C6 level, caused by a gunshot wound 2 years prior to this study. Case 2, a 32-year-old female, had a chronic transverse myelitis of unknown origin at C5 level. Both patients could breathe spontaneously, using diaphragmatic muscles and both of them had no control of their bladder functions.

Venous catheterization was performed with a single lumen cardiac catheter, guided under fluoroscopy and left in the pulmonary artery trunk just beyond the pulmonic valve. An indwelling Courand needle was placed in the femoral artery. Cardiac output was determined simultaneously, as described below according to direct Fick and Stewart-Hamilton principles.

Cardiac output determination by the dye dilution technic followed the injection of a 0.5 percent solution of Evans blue dye into the pulmonary artery main trunk just beyond the pulmonic valve. An indwelling Courand needle was placed in the femoral artery. Cardiac output was determined simultaneously, as described below according to direct Fick and Stewart-Hamilton principles.

Cardiac output determination by the dye dilution technic followed the injection of a 0.5 percent solution of Evans blue dye into the pulmonary artery main trunk and collection of samples from the femoral artery. Blood was collected into heparinized test tubes through the Courand needle and a plastic tube (22 cm. long and 2 mm. bore). Sampling of blood, starting 5 seconds after the injection was performed rhythmically, every 2 seconds, timed with a metronome set at 60 beats/min.

The amount of dye introduced was measured by weighing the syringe before and after the injection. The dead space of the catheter and manifold, through which the injection was performed, was measured by the weight difference of the system, when dry and when filled with distilled water. The dye remaining in the catheter was washed out by aspirating 10 ml. of blood immediately after the injection. Blood for hematocrit determination and for the plasma blank of the dilution curves was sampled from the artery prior to the injection.

Tubes were spun in a centrifuge at 3,000 r.p.m. for 30 minutes. Dye concentrations in plasma were read in a Beckman Model DU spectrophotometer at 6.250 A0 wavelength. The hematocrit was determined by centrifuging the heparinized blood in Wintrobe tubes for 30 minutes at 3,000 r.p.m. Dye concentrations were plotted in semilogarithmic paper, with extrapolation of the straight descending line previous to recirculation.

The cardiac output was computed from the dye dilution curve according to the method of Hamilton, Moore, Kinman and Sparling. Mean circulation time was computed according to the formula of Etsten and Li. Central blood volume was computed by the Stewart principle.

Total blood volume was measured with Evans blue dye, taking blood samples at the tenth and twentieth minute after the injection, but assuming the tenth minute samples as the value of complete homogenization in these patients, according to Gregersen's method.

Cardiac output by direct Fick method was determined by collecting expired gas in a Douglas bag during 5 minutes and sampling blood from the pulmonary and femoral arteries during the third minute. Blood gases were determined in duplicates according to the technic of Van Slyke and Neill, blood pH in a glass electrode with a Beckman G potentiometer. Expired air was measured in a calibrated Tissot spirometer and duplicate analysis for O2 and CO2 were carried out in a Scholander gas analyzer.

Femoral and pulmonary artery pressure were measured with a Statham strain-gage transducer, the zero point of reference being 10 cm. above the level of the table. A Sanborn Twin-Viso was used as recording system.
Table 1

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>B.S.</th>
<th>Sex</th>
<th>Respiratory Data</th>
<th>( V_{L} ) ( \text{L/min} \cdot \text{m}^{-2} )</th>
<th>( V_{A} )</th>
<th>( V_{E} ) ( \text{ml} \cdot \text{m}^{-2} )</th>
<th>( \text{PaCO}_{2} ) ( \text{mm Hg} )</th>
<th>( \text{Vo}_{2} ) ( \text{ml} \cdot \text{m}^{-2} \text{min}^{-1} \text{m}^{-2} )</th>
<th>pH</th>
<th>( \text{PaCO}_{2} ) ( \text{mm Hg} )</th>
<th>( \text{Vo}_2 % )</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>21</td>
<td>1.63</td>
<td>M</td>
<td></td>
<td>3.56</td>
<td>2.41</td>
<td>13</td>
<td>446</td>
<td>115</td>
<td>.856</td>
<td>96.5</td>
<td>48.9</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>3.12</td>
<td>M</td>
<td></td>
<td>1.63</td>
<td>1.91</td>
<td>15</td>
<td>340</td>
<td>98</td>
<td>.890</td>
<td>95.3</td>
<td>50.9</td>
</tr>
<tr>
<td>2</td>
<td>38</td>
<td>1.58</td>
<td>F</td>
<td></td>
<td>3.89</td>
<td>1.72</td>
<td>22</td>
<td>279</td>
<td>84</td>
<td>102</td>
<td>.826</td>
<td>94.2</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>4.05</td>
<td>F</td>
<td></td>
<td>3.46</td>
<td>2.09</td>
<td>20</td>
<td>231</td>
<td>92</td>
<td>118</td>
<td>.755</td>
<td>96.0</td>
</tr>
</tbody>
</table>

All pulmonary volumes are B.T.P.S.

\( V \) = expired volume; \( V_{A} \) = alveolar ventilation; \( f \) = respiratory rate; \( V_{E} \) = tidal volume;
\( V_{D} \) = dead space volume (includes instrumental dead space = 72 ml.); \( \text{PaCO}_{2} \) = \( \text{CO}_2 \) production;
\( \text{Vo}_2 \) = \( \text{O}_2 \) consumption; \( R \) = respiratory quotient; \( \text{SaO}_2 \) = oxygen arterial saturation;
\( \text{CcCO}_2 \) = arterial \( \text{CO}_2 \) content; \( \text{PaCO}_2 \) = arterial \( \text{PCO}_2 \).

Results

Table 1 shows the respiratory data of both patients with bladder empty and filled to a pressure of more than 25 cm. of water. Ventilation was within normal limits and changes with increased vesical pressure were small. Table 2 shows the hemodynamic data.

Cardiac output \( \text{ml} \cdot \text{sec}^{-1} \) =

\[ \text{Cardiac output} = \frac{\text{mean pulm. art. pressure} \times 1332}{1.332} \]

Total peripheral resistance =

\[ \text{Blood volume} = \frac{\text{mean femoral artery pressure} \times 1332}{1.332} \]

Central blood
Table 2

<table>
<thead>
<tr>
<th>Hemodynamic Data</th>
<th>Vesical pressure cm. H2O</th>
<th>C.I.f</th>
<th>C.I.H</th>
<th>D5-V02</th>
<th>M.C.T.</th>
<th>Qe mL./Kg.</th>
<th>Ret. %</th>
<th>H.R. mm. Hg</th>
<th>P.A.P. mm. Hg</th>
<th>F.P. mm. Hg</th>
<th>F.R. P.T.R. dynes sec. cm.²</th>
<th>Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>3.31</td>
<td>2.29</td>
<td>4.34</td>
<td>14.01</td>
<td>.688</td>
<td>75.8</td>
<td>43.5</td>
<td>75</td>
<td>12.1</td>
<td>2.6</td>
<td>8.5</td>
<td>+21</td>
</tr>
<tr>
<td>&gt;25</td>
<td>2.59</td>
<td>2.16</td>
<td>4.23</td>
<td>15.30</td>
<td>.547</td>
<td>83.0</td>
<td>43.0</td>
<td>85</td>
<td>20.4</td>
<td>6</td>
<td>12.6</td>
<td>-6</td>
</tr>
<tr>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change</td>
<td>%</td>
<td>-21</td>
<td>-6</td>
<td>+2</td>
<td>-12</td>
<td>-20</td>
<td>+9</td>
<td>0</td>
<td>-12</td>
<td>+48</td>
<td>+48 +57 +58</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>2.26</td>
<td>3.08</td>
<td>4.49</td>
<td>14.0</td>
<td>.720</td>
<td>66.9</td>
<td>44.0</td>
<td>93</td>
<td>13.1</td>
<td>4.4</td>
<td>8</td>
<td>97</td>
</tr>
<tr>
<td>30</td>
<td>2.44</td>
<td>3.33</td>
<td>4.67</td>
<td>14.35</td>
<td>.797</td>
<td>70.9</td>
<td>44.0</td>
<td>81</td>
<td>20.4</td>
<td>6.3</td>
<td>14</td>
<td>112</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Change</td>
<td>%</td>
<td>+7</td>
<td>+8</td>
<td>+3</td>
<td>+2</td>
<td>+10</td>
<td>+6</td>
<td>0</td>
<td>-13</td>
<td>-75</td>
<td>+17 +65 +9</td>
<td></td>
</tr>
</tbody>
</table>

C.I.f = cardiac index (Fick); C.I.H = cardiac index (Hamilton); D5-V02 = arterio venous O2 difference; M.C.T. = mean circulation time; Qe = "central" blood volume; Qs = blood volume; H.R. = heart rate; P.A.P. = pulmonary artery pressure; P.F. = femoral artery pressure; P.R. = total pulmonary resistance; P.T.R. = peripheral total resistance.

Discussion

Effects of bladder distention on autonomic mechanisms in paraplegic patients were reviewed and studied by Guttmann and Whitteridge in 1947, and by Hutch in 1955. These effects have been classified as: (a) mass involuntary, peripheral muscle spasm; (b) hyperactive reflex of sympathetic nervous system manifested by rise in arterial pressure, pilomotor and sudomotor responses; and (c) mass parasympathetic reaction observed as bladder spasm.

The effects that were secondary to the increased arterial pressure were as follows: (1) bradycardia due to carotid sinus and aortic arch stimulation; (2) headaches, due to passive cerebral vasodilation with increased cerebral blood flow; and (3) patchy vasodilation of the skin of the face and neck and of the nasal passages due to reflex or passive vasodilation. Guttmann also observed engorgement of the veins of the neck, marked dilation of the heart, tightness of the chest and shallow breathing, a situation resembling incipient heart failure.

Our subjects show increase in peripheral and pulmonary resistance. Since increased pulmonary artery pressure in this condition has not been described before, we shall discuss this point. Pulmonary artery pressure is increased when intrathoracic pressure rises during a sustained expiratory effort. Bladder distention may cause mass voluntary muscle spasm and increase intrathoracic pressure, but this is only a very transient effect that did not appear during our pressure recording. Normal respiratory cycles can be seen in the records (figs. 1 and 2) and excludes a Valsalva type of effect. Therefore one can accept increased pulmonary artery pressure as a circulatory effect.

Pulmonary arterial pressure may increase because of increase in cardiac output or in...
resistance. Cardiac output rise was shown not to occur and may be excluded.

A rise of resistance in this condition may occur as a consequence of left heart failure, pulmonary vein constriction or constriction of pulmonary arterioles. This point has not been elucidated in our experiments because "capillary" pulmonary pressure was not recorded, but the rather small increase in systemic arterial pressure and lack of rise in central blood volume speak against left heart failure.

Constriction of the venous side of the pulmonary vascular bed cannot be excluded, although lack of significant ventilatory changes would not favor this interpretation.

Pulmonary arteriolar vasoconstriction is left as the more plausible explanation. Is this increase in resistance mediated through a nervous or a humoral stimulus? Although a reflex phenomenon seems more probable, the fact that pulmonary artery pressure remains high, even when the vesical pressure has fallen to zero and returns to previous levels after about 10 minutes, calls for the possibility of a humoral mediator. Epinephrine, norepinephrine and histamine may be excluded, the first because cardiac output did not increase, the second because pulmonary resistance increased more than the peripheral resistance, and the third because the systemic pressure rose. Serotonin is still left as a possible humoral factor in this reaction that would explain the rise in pulmonary artery pressure. The other hemodynamic effects of this substance have numerous circulatory actions, so that any combination may be expected. Pathogenesis of the rise in pulmonary artery pressure with bladder distention, in spinal cord section, remains an open question.

Summary

Hemodynamic data during bladder distention in 2 patients with cervical cord transac-
tion are presented. Rise in pulmonary artery pressure and pulmonary resistance is shown to occur when vesical pressure is increased.

**Summario in Interlingua**

Es presentate datos hemodynamic obtenite durante distension del vesica in 2 patientes con transsection del cordon cervical. Es mostrate que augmentos del tension pulmono-arterial e del resistensia pulmonar occurre quando le pression del vesica es augmentate.

**References**


6. STEWART, G. N.: Pulmonary circulation time, the quantity of blood in the lungs and the output of the heart. Am. J. Physiol. 58: 20, 1921.


11. PAGE, I. H.: Serotonin (5 Hydroxtryptamine); the last four years. Physiol. Rev. 38: 277, 1958.
Effects of Bladder Distention on Pulmonary Vascular Bed
ALBERTO AGREST and AQUILES J. RONCORONI

Circ Res. 1960;8:501-505
doi: 10.1161/01.RES.8.3.501

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/8/3/501