Effects of Acute Hypoxia on the Volume of Blood in the Thorax

By H. W. Fritts, Jr., M.D., J. E. Odell, M.D., P. Harris, M.D., E. W. Braunwald, M.D., and A. P. Fishman, M.D.

The results presented in the first paper of this series confirm the observations of others that acute hypoxia frequently raises the pulmonary arterial pressure in normal man. This pressor effect cannot be solely attributed to an augmented cardiac output, nor can it be ascribed to an elevated left atrial pressure. It seems, therefore, to arise from a change in the dimensions of some portion or portions of the pulmonary vascular bed.

Whether this change involves an alteration in the pulmonary blood volume has not been settled, since experiments in man and in animals have yielded conflicting results. The present study was designed to investigate the relation between volume and pressure by using two dissimilar methods. The first entailed measuring the central blood volume before and during hypoxia; the second involved recording the change in the relative weights of the two ends of the body when hypoxia was induced.

Methods
Measurement of Central Blood Volume

The central blood volume was measured by using the dye-dilution method of Hamilton. For this purpose, a no.-8 cardiac catheter was advanced until its tip lay in the main pulmonary artery and a Cournand needle was inserted into the right brachial artery. The patient was then allowed to rest for 15 minutes before observations were begun.

The experimental protocol comprised 2 20-
HYPOXIA AND THORACIC BLOOD VOLUME

Figure 1
Schematic drawing of a teeter-board.

Figure 2
Effect of (A) hypoxia and (B) norepinephrine on the relative weights of the 2 ends of the body. Response marked CAL indicates effect of placing a 50-Gm, weight 10 cm. from fulcrum on head-end of the board.

Figure 3
Effect of hypoxia on central blood volume.

erable variation between the 2 measurements in individual patients. When the difference between the volumes measured in each patient was divided by the first volume, the range extended from -11 to +12.6 per cent, with an average difference of -1.9 per cent. With the assumption that these differences had a Gaussian distribution, their standard deviation was 7.33 per cent. Hence, 2.58 standard deviations, the number necessary to include 99 per cent of the observations, was ±18.91 per cent.

Patients in Whom the Central Blood Volume Was Measured before and during Hypoxia

The volumes measured in the 17 patients who were made hypoxic are recorded in table 1. The values obtained while they breathed 21 per cent oxygen gave an average volume of 0.79 L./M.², and a standard deviation of 0.119 L./M.². The volumes measured during hypoxia gave an average value of 0.81, and a standard deviation of 0.566 L./M.². When analyzed by the “t” test of Fisher, the difference between the averages was not significant (p>.10).

The changes in volume are depicted graphically in figure 3. Each bar represents a single patient. The length of the bar represents the percentage change in volume calculated as the difference between the ambient air and hypoxic values divided by the ambient air value. In only 2 patients (J.O'C. and J.H.) did the percentage change exceed the limits
of ±18.91 per cent calculated for all 8 subjects.

Despite the lack of change in the central blood volume, hypoxia exhibited its usual pressor effect on the pulmonary arterial pressure. Thus, the average value during the control period was 13.7 mm. Hg and during the period of hypoxia it rose to 18.7 mm. Hg. This difference would have arisen by chance less frequently than 1 in 100 times (p. <.01).

Patients Studied on the Teeter-Board

None of the 9 patients studied on the teeter-board demonstrated any change in the position of the center of gravity during hypoxia, despite the fact that arterial saturations as low as 62 per cent were induced. A typical record is shown in figure 2A, and is contrasted with a tracing recorded during an infusion of norepinephrine (fig. 2B). In the latter instance, the center of gravity shifted toward the head-end of the body.

Discussion

In the studies reported in the preceding paper, the cardiac output was measured by the Fick principle and the importance of the steady-state was emphasized. In the present studies, the cardiac output was measured by the dilution principle and safeguards of the same sort were applied. The most frequent difficulty was imposed by an unstable densitometer baseline during the hypoxic period. This unsteadiness was caused by variations in the oxygen saturation of the blood in the systemic arteries, and in 12 patients the fluctuations were so large that the data were discarded. In the patients included in this report, baselines recorded over a period of 20 seconds showed variations that were less than 5 per cent of the maximum height of the curve.

The central blood volume as measured in these studies included not only the blood in

### Table 1

Changes in Arterial Blood Oxyhemoglobin Saturation, Pulmonary Arterial Pressure, Cardiac Index, Mean Circulation Time, and Central Blood Volume during Acute Hypoxia in Seventeen Normal Subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Arterial blood O₂ sat, %</th>
<th>Pulmonary pressure s/dm mm. Hg</th>
<th>Cardiac output L/min/M²</th>
<th>Mean circulation time sec</th>
<th>Central blood volume L/M²</th>
<th>Pulmonary arterial pressure s/dm mm. Hg</th>
<th>Cardiac output L/min/M²</th>
<th>Mean circulation time sec</th>
<th>Central blood volume L/M²</th>
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</thead>
<tbody>
<tr>
<td>J.O.</td>
<td>98</td>
<td>19/6,12</td>
<td>4.1</td>
<td>10.9</td>
<td>.74</td>
<td>90</td>
<td>20/8,14</td>
<td>3.6</td>
<td>11.7</td>
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<tr>
<td>C.D.</td>
<td>93</td>
<td>18/9,13</td>
<td>3.0</td>
<td>12.8</td>
<td>.64</td>
<td>88</td>
<td>18/9,13</td>
<td>3.2</td>
<td>12.3</td>
</tr>
<tr>
<td>J.O'C.</td>
<td>91</td>
<td>26/10,16</td>
<td>4.5</td>
<td>14.1</td>
<td>1.06</td>
<td>86</td>
<td>33/14,22</td>
<td>5.3</td>
<td>14.8</td>
</tr>
<tr>
<td>P.M.</td>
<td>95</td>
<td>12/5,9</td>
<td>3.4</td>
<td>12.2</td>
<td>.69</td>
<td>85</td>
<td>16/7,11</td>
<td>4.7</td>
<td>8.9</td>
</tr>
<tr>
<td>M.B.</td>
<td>94</td>
<td>16/6,9</td>
<td>3.3</td>
<td>15.2</td>
<td>.84</td>
<td>83</td>
<td>18/7,12</td>
<td>3.9</td>
<td>14.0</td>
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<tr>
<td>E.J.</td>
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<td>3.0</td>
<td>14.8</td>
<td>.74</td>
<td>83</td>
<td>29/10,18</td>
<td>3.9</td>
<td>11.0</td>
</tr>
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<td>A.O.</td>
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<td>4.8</td>
<td>12.1</td>
<td>.97</td>
<td>80</td>
<td>23/10,15</td>
<td>6.9</td>
<td>8.9</td>
</tr>
<tr>
<td>C.G.</td>
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<td>18/8,13</td>
<td>4.3</td>
<td>11.1</td>
<td>.80</td>
<td>79</td>
<td>24/11,17</td>
<td>4.1</td>
<td>12.4</td>
</tr>
<tr>
<td>J.L.</td>
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<td>3.5</td>
<td>11.7</td>
<td>.68</td>
<td>76</td>
<td>26/12,19</td>
<td>4.5</td>
<td>9.7</td>
</tr>
<tr>
<td>W.M.</td>
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<td>28/11,18</td>
<td>3.5</td>
<td>13.0</td>
<td>.76</td>
<td>74</td>
<td>30/12,20</td>
<td>4.6</td>
<td>11.5</td>
</tr>
<tr>
<td>V.P.</td>
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<td>4.7</td>
<td>9.9</td>
<td>.78</td>
<td>74</td>
<td>22/14,18</td>
<td>5.1</td>
<td>10.5</td>
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<tr>
<td>H.C.</td>
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<td>.62</td>
<td>72</td>
<td>40/14,27</td>
<td>3.6</td>
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<tr>
<td>J.H.</td>
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<td>3.2</td>
<td>14.8</td>
<td>.79</td>
<td>71</td>
<td>33/19,26</td>
<td>3.2</td>
<td>11.9</td>
</tr>
<tr>
<td>A.P.</td>
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<td>13/6,10</td>
<td>4.1</td>
<td>14.8</td>
<td>1.01</td>
<td>70</td>
<td>19/11,15</td>
<td>5.2</td>
<td>10.5</td>
</tr>
<tr>
<td>A.C.</td>
<td>96</td>
<td>21/9,15</td>
<td>4.1</td>
<td>11.1</td>
<td>.76</td>
<td>68</td>
<td>34/17,24</td>
<td>5.8</td>
<td>8.7</td>
</tr>
<tr>
<td>O.S.</td>
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<td>22/10,16</td>
<td>4.3</td>
<td>10.9</td>
<td>.78</td>
<td>66</td>
<td>33/18,25</td>
<td>5.9</td>
<td>9.4</td>
</tr>
<tr>
<td>G.Y.</td>
<td>96</td>
<td>24/11,16</td>
<td>4.6</td>
<td>9.2</td>
<td>.71</td>
<td>65</td>
<td>31/14,22</td>
<td>5.1</td>
<td>6.9</td>
</tr>
<tr>
<td>Average</td>
<td>95</td>
<td>20/9,14</td>
<td>3.9</td>
<td>12.3</td>
<td>.79</td>
<td>77</td>
<td>26/12,19</td>
<td>4.6</td>
<td>10.7</td>
</tr>
</tbody>
</table>
the pulmonary vessels, but that contained in the chambers of the left heart and that segment of the aorta and its branches that lay at points temporally equidistant from the aortic valve. In keeping with the observations of Doyle, Wilson, and Warren, our results indicated that hypoxia did not alter this volume of blood.

Furthermore, no shift in the center of gravity could be demonstrated when hypoxia was induced in patients lying on the teeter-board. These results confirm and extend earlier observations from this laboratory and agree with those published by Honig and Tenney. According to the calibration, the teeter-board would have disclosed a change in the pulmonary blood volume of 50 ml. or less, if the blood moved through a distance of 10 cm. along the length of the body. These observations support the measurements of the central blood volume in indicating that the volume of blood in the thorax does not change appreciably during hypoxia.

Summary
Two different methods were used to study the effect of acute hypoxia on the volume of blood in the thorax. The first method entailed estimating the central blood volume by the Stewart-Hamilton dye-dilution technic; the second involved the use of a teeter-board. The 2 methods gave comparable results. Both indicated that the volume of blood in the thorax is not appreciably altered by hypoxia.

Summario in Interlingua
Duo differente methodos esseva usate pro studiar le effecto de hypoxia acute super le volumine del sanguine in le thoraece. Le priame del duo methodos consisteva in estimare le volumine de sanguine central per medio del technica a dilution de colorante secondo Stewart-Hamilton; le secunde utilisava un planca basculante. Le resultatos obtenite per le duo methodos esseva comparabile. Ambas indicava que le volumine de sanguine in le thoraece non es alterate appreciabilemente per le effectos de hypoxia.

References
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