Interrelationships of Cardiac Output, Blood Pressure, and Peripheral Resistance during Normal Respiration in Normotensive and Hypertensive Individuals

By Arnold H. Williams, M.D., and Arthur L. Gropper, M.D.

The effects of normal respiration upon the heart, blood pressure, and peripheral circulation were studied in normotensive and hypertensive subjects. Changes of right and left ventricular output were measured by the ballistocardiogram and the pressure-pulse method of Remington, peripheral blood flow by plethysmographs. The central and peripheral changes were interrelated and evidence is presented that the latter are chiefly due to the effects of intrathoracic pressure upon cardiac filling.

For many years it has been known that quiet respiration is accompanied by certain changes in the dynamics of the circulation. On inspiration the output of the right ventricle increases, that of the left decreases,1-6 arterial pressure falls7-11 and regional blood flow is diminished.12-14 During expiration the direction of the changes is reversed. Their origin has been attributed to the effects of variations of intrathoracic pressure upon the filling of the right heart which in turn affects the filling of the left heart.2,4-6 The depth, type and rate of respiration7,11 influence the magnitude of the changes to some degree. There is no information on the effects of quiet respiration upon peripheral resistance.

Most of the above studies have been made on laboratory animals. Rather drastic procedures were used and either central or peripheral circulatory changes were investigated. The present study represents an attempt to interrelate all the changes in man, using technics designed to cause as little disturbance to the subject as possible. In addition, the reactions of normotensive and hypertensive individuals were compared.

Methods

Simultaneous measurements were made of the variations of the output of each ventricle, of arterial pressure and of volume flow through various peripheral areas. Blood pressure was measured directly by a Hamilton optical manometer15 through a 20 gage hypodermic needle in the brachial artery. Respiration was recorded by a pneumograph strapped about the thorax. Directional changes in regional blood flow were estimated with photoelectric plethysmographs applied usually to the lobe of the ear, the toe tip and finger tip. In a few instances they were followed in the forearm and hand with an air plethysmograph. Similar changes were found in other regions of the body, i.e., the scrotum, cheek, tongue, and rectovaginal septum. These instruments did not provide quantitative measurements of blood flow, but directional changes were estimated from the diastolic volume of the part. All electrical signals were led through direct current amplifiers into Sanborn galvanometers; recordings were made on a photokymograph run at a speed of 5 or 25 mm. per second. Inasmuch as the outputs of left and right ventricles vary in opposite directions during respiration, it was necessary to devise methods for separating these two functions. Total cardiac output from beat to beat was calculated from tracings taken on a high frequency ballistocardiograph of Wilkins' design.16 The oscillations of the table were recorded by means of a photoelectric pressure recorder with a suitable rubber membrane. An attempt to estimate beat-to-beat variations of left ventricular stroke volume utilizing the K wave of the ballistocardiogram was unsuccessful. In this study the percentile changes of left ventricular output from inspiration to expiration were estimated using the pressure-pulse method of Remington.19 Estimates of the percentile changes of right ventricular output were

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TABLE 1.—Circulatory Changes during Quiet Respiration

<table>
<thead>
<tr>
<th>Name, Sex and Age</th>
<th>Diagnosis</th>
<th>Condition of Patient during Respiration</th>
<th>Pulse Rate (per min.)</th>
<th>Blood Pressure (mm. Hg)</th>
<th>Left Ventricular Stroke Index†</th>
<th>Ballistocardiographic Stroke Index (cc. per beat)</th>
<th>Peripheral Resistance†</th>
<th>Type of Ballistocardiogram</th>
</tr>
</thead>
<tbody>
<tr>
<td>R. J. M, 33</td>
<td>Alcoholism</td>
<td>calm</td>
<td>79.8</td>
<td>73.2</td>
<td>+8.3</td>
<td>57.1 ± 0.3 (+3.5) 29.9 ± 25.7 +16.3 ± 28.3 ±2.89 ± 2.81 ±2.8</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>R. S. M, 34</td>
<td>Pneumonia, Convalescent</td>
<td>agitated</td>
<td>47.4</td>
<td>46.0</td>
<td>+7.6</td>
<td>61.4 ± 0.3 (+3.2) 27.7 ± 42.1 +37.1 ± 31.1 ±1.55 ± 1.57 ±1.3</td>
<td>M</td>
<td></td>
</tr>
<tr>
<td>O. H. M, 40</td>
<td>Malnutrition</td>
<td>calm</td>
<td>85.1</td>
<td>81.9</td>
<td>+3.2</td>
<td>56.8 ± 0.3 (+3.2) 37.7 ± 42.1 +37.1 ± 31.1 ±1.55 ± 1.57 ±1.3</td>
<td>M</td>
<td></td>
</tr>
<tr>
<td>L. M, 22</td>
<td>Pneumonia, Convalescent</td>
<td>tense</td>
<td>56.6</td>
<td>54.3</td>
<td>+4.6</td>
<td>60.2 ± 0.3 (+3.4) 27.3 ± 42.1 +37.1 ± 31.1 ±1.55 ± 1.57 ±1.3</td>
<td>M</td>
<td></td>
</tr>
<tr>
<td>B. M, 24</td>
<td>Tonsilitis, Convalescent</td>
<td>calm</td>
<td>60.8</td>
<td>58.7</td>
<td>+2.7</td>
<td>55.1 ± 0.3 (+3.4) 27.7 ± 42.1 +37.1 ± 31.1 ±1.55 ± 1.57 ±1.3</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>T. B. M, 47</td>
<td>Indolent Ulcer</td>
<td>calm</td>
<td>75.0</td>
<td>71.0</td>
<td>+4.5</td>
<td>50.1 ± 0.3 (+3.5) 25.6 ± 41.8 +37.1 ± 31.1 ±1.55 ± 1.57 ±1.3</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>T. C. M, 33</td>
<td>Alcoholism</td>
<td>tense</td>
<td>76.0</td>
<td>71.0</td>
<td>+2.6</td>
<td>60.6 ± 0.3 (+4.9) 27.7 ± 42.1 +37.1 ± 31.1 ±1.55 ± 1.57 ±1.3</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>J. V. M, 20</td>
<td>Reiter’s Syndrome</td>
<td>tense</td>
<td>70.0</td>
<td>73.3</td>
<td>+4.0</td>
<td>66.9 ± 0.3 (+3.9) 39.2 ± 42.1 +37.1 ± 31.1 ±1.55 ± 1.57 ±1.3</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>R. F. M, 24</td>
<td>Duodenal Ulcer</td>
<td>tense</td>
<td>75.3</td>
<td>68.0</td>
<td>+1.9</td>
<td>69.2 ± 0.3 (+2.4) 27.7 ± 42.1 +37.1 ± 31.1 ±1.55 ± 1.57 ±1.3</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>M. B. M, 44</td>
<td>Pneumonia, Convalescent</td>
<td>calm</td>
<td>102.0</td>
<td>98.4</td>
<td>+3.7</td>
<td>45.5 ± 0.3 (+3.7) 27.7 ± 42.1 +37.1 ± 31.1 ±1.55 ± 1.57 ±1.3</td>
<td>N (Ms on Insp.)</td>
<td></td>
</tr>
<tr>
<td>S. S. M, 44</td>
<td>Alcoholism</td>
<td>calm</td>
<td>71.4</td>
<td>68.3</td>
<td>+4.5</td>
<td>75.0 ± 0.3 (+1.1) 27.7 ± 42.1 +37.1 ± 31.1 ±1.55 ± 1.57 ±1.3</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>Mean...</td>
<td>21.5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table concluded on following page
### TABLE 1—Continued

<table>
<thead>
<tr>
<th>Name, Sex and Age</th>
<th>Diagnosis</th>
<th>Condition of Patient during Expt.</th>
<th>Surface Area (Sq. M.)</th>
<th>Resp. rate (per min.)</th>
<th>Pulse Rate (per min.)</th>
<th>Blood Pressure (mm. Hg)</th>
<th>Left Ventricular Stroke Index (cc. per beat)</th>
<th>Ballistocardiographic Stroke Index (cc. per beat)</th>
<th>Peripherai Resistance</th>
<th>Type of Ballistocardiogram</th>
</tr>
</thead>
<tbody>
<tr>
<td>L. F., M., 24</td>
<td>Essential Hypertension</td>
<td>agitated</td>
<td>1.42 ± 0.18</td>
<td>99.4 ± 90.9 ± 10.4 ± 3.7</td>
<td>267 ± 29 ± 228.3 ± 917.9</td>
<td>117 ± 0.17 ± 133.8 ± 31.9</td>
<td>+5.4 ± 1.45 ± 5 ± 74.1 ± 4.54 ± 0.2 ± 24.4 ± 28.1 ± 4.8 ± 224.4 ± 30.7 ± 5.17 ± 3.39 +4.3</td>
<td>M.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. S., M., 50</td>
<td>Essential Hypertension</td>
<td>calm</td>
<td>1.08 ± 0.21</td>
<td>80.4 ± 71.5 ± 12.5 ± 91.4</td>
<td>211 ± 533 ± 397.0 ± 65.2</td>
<td>110.5 ± 113.0 ± 6.1</td>
<td>+2.3 ± 2.3 ± 69.2 ± 4.73 ± 22.4 ± 62.0 ± 29.5 ± 5.04 ± 3.36 +6.35</td>
<td>N.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A. S., M., 30</td>
<td>Essential Hypertension</td>
<td>calm</td>
<td>1.00 ± 0.20</td>
<td>97.2 ± 94.9 ± 1.9 ± 3.3</td>
<td>170.3 ± 174.8 ± 1.4 ± 2.6</td>
<td>109.6 ± 91.4 ± 0.2</td>
<td>+63.5 ± 64.5 ± 5 ± 4.4 ± 21.8 ± 42 ± 34.7 ± 5.54 ± 3.97 ± 0.55</td>
<td>N.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ibid, M., 30</td>
<td>Essential Hypertension</td>
<td>calm</td>
<td>1.00 ± 0.41</td>
<td>98.5 ± 81.7 ± 3.3 ± 1.5</td>
<td>149.0 ± 152.9 ± 2.7 ± 0.6</td>
<td>108.7 ± 108.7 ± 0.2</td>
<td>+60.6 ± 61.3 ± 1.3 ± 21.4 ± 22 ± 19.1 ± 2 ± 33.3 ± 3.72 ± 1.0</td>
<td>N.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N. J., M., 22</td>
<td>Glomerulonephritis, Hypertension</td>
<td>calm 1 °9 ± 24</td>
<td>96.5 ± 81.7 ± 3.3 ± 1.5</td>
<td>149.0 ± 152.9 ± 2.7 ± 0.6</td>
<td>108.7 ± 108.7 ± 0.2</td>
<td>+60.6 ± 61.3 ± 1.3 ± 21.4 ± 22 ± 19.1 ± 2 ± 33.3 ± 3.72 ± 1.0</td>
<td>N.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>W. B., M., 56</td>
<td>Malignant hypertension, Uremia</td>
<td>calm 1 °9 ± 24</td>
<td>95.5 ± 95.5 ± 0.0 ± 0.0</td>
<td>200.8 ± 214.5 ± 0.8 ± 0.8</td>
<td>100.0 ± 102.0 ± 2.0</td>
<td>+67.0 ± 70.3 ± 4.9 ± 55.3 ± 3.2 ± 41.4 ± 49.5 ± 2.91 ± 2.89 ± 0.7</td>
<td>M.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean ± 95.3</td>
<td>Arteriosclerotic hypertension</td>
<td>calm ± 1 °9 ± 24</td>
<td>83.6 ± 80.8 ± 3.5 ± 3.5</td>
<td>187.8 ± 196.6 ± 4.5 ± 4.5</td>
<td>97.9 ± 100.0 ± 2.3 ± 2.3</td>
<td>+63.9 ± 21.4 ± 39.0 ± 28.7 ± 37.7 ± 34.2 ± 4.14 ± 4.23 ± 1.3</td>
<td>M.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>F. T., M., 67</td>
<td>Arteriosclerotic hypertension</td>
<td>calm ± 1 °9 ± 24</td>
<td>73.3 ± 74.0 ± 1.7 ± 1.7</td>
<td>250.2 ± 241.9 ± 1.1 ± 1.1</td>
<td>98.9 ± 96.0 ± 0.2 ± 2.1</td>
<td>+85.8 ± 81.6 ± 1.4 ± 55.3 ± 29.8 ± 69.2 ± 45.8 ± 3.3 ± 3.3 ± 3.3</td>
<td>M. (calculated as N)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E. H., F., 20</td>
<td>Coarctation of Aorta, 5 months pregnancy</td>
<td>tense ± 1 °9 ± 24</td>
<td>88.2 ± 80.6 ± 0.4 ± 0.4</td>
<td>102.9 ± 107.9 ± 2.1 ± 2.1</td>
<td>98.1 ± 100.6 ± 2.6</td>
<td>+50.6 ± 51.2 ± 1.2 ± 31.6 ± 26.3 ± 20.1 ± 27.6 ± 4.52 ± 4.6 ± 1.8</td>
<td>N. (no K wave)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Percentile changes are expressed in terms of inspiratory values except for the BCG values, which are expressed in terms of expiratory values.

* Respiration irregular
† Calculated by the pressure pulse of Remington and co-workers.19
‡ Formulas for calculation of resistance

\[
\text{Diastolic pressure} = \left( \text{BCG stroke index}_{\text{max}} \times [0.4 \times (\text{Systolic pressure}_{\text{exp}} - \text{Diastolic pressure}_{\text{exp}})] \right)
\]

\[
\text{Resistance}_{\text{exp}} = \left( \frac{\text{BCG stroke index}_{\text{max}} - [0.5 \times \% \text{change left ventricular stroke index} \times \text{BCG stroke index}_{\text{mean}}]}{\text{Diastolic pressure}_{\text{exp}} - \text{Diastolic pressure}_{\text{exp}}]} \right)
\]

\[
\text{Resistance}_{\text{exp}} = \left( \frac{\text{BCG stroke index}_{\text{max}} - [0.5 \times \% \text{change left ventricular stroke index} \times \text{BCG stroke index}_{\text{mean}}]}{\text{Diastolic pressure}_{\text{exp}} - \text{Diastolic pressure}_{\text{exp}}} \right)
\]

§ Type of BCG complex: N = normal, L = late downstroke, M, = late M, M, = double i or mid M.20

Output (SV, R) were made by subtracting the changes of left ventricular output (SV, L), as obtained from the pressure-pulse method, from the total percentile changes recorded in the ballistocardiogram (SV, R + SV, L).°

* The respiratory variations of the ballistocardiogram reflect changes in the output of both ventricles. They vary in opposite directions during the respiratory cycle; their sum is recorded by the ballistocardiograph. The ballistocardiographic variations are Patients were obtained from the clinic and wards of St. Louis City Hospital. Eleven were normoten-

Inspiration \( \text{SV} \), R = BCGmean - \( \frac{\Delta \text{SVL} + \Delta \text{BCG}}{2} \)

Expiration \( \text{SV} \), R = BCGmean + \( \frac{\Delta \text{SVL} - \Delta \text{BCG}}{2} \)
sive, 11 suffered from "essential" hypertension of varying degrees of severity, and 2 had hypertension from other causes. The two groups were comparable in age and included 22 men and 2 women. The laboratory was air-conditioned at a constant temperature of 78°F, and all experiments were carried out in semidarkness. The only disturbing stimulus was the arterial puncture, done after periarterial novocaine infiltration. In most instances measurements were obtained during quiet relaxed breathing (table 1).

![Figure 1](http://circ.ahajournals.org/)  
**Fig. 1.** Respiratory variations of peripheral blood flow. These plethysmographic records, selected from various patients, show that peripheral blood flow varies with respiration in all parts of the body, but stops during apnea although α waves continue (lower right). The vertical lines point to the decrease of blood flow during inspiration (up in the respiratory tracing). In all records the volume changes due to the pulse are superimposed on the slower respiratory changes. The bracket at the bottom of each record represents a time interval of two seconds. The label R.V. (upper right) indicates rectovaginal septum.

A representative 20 second section of each record was selected for analysis and beat-to-beat measurements were made of each function. The peak changes of blood pressure and both measures of cardiac output, during inspiration and expiration, were separated and averaged in all of the respiratory cycles of the strip. The ballistocardiographic changes were calculated according to the altitude formula of Starr and Schroeder.20 Left ventricular output was estimated from the data of table 2 of Remington and co-workers,19 the pressure-pulse values being obtained from the arterial pressure tracings. Peripheral resistance was calculated as the ratio of the mean arterial pressure to the mean ballistocardiographic stroke index, mean blood pressure being considered as diastolic pressure + 40 per cent of the pulse pressure (see footnote to table 1). Directional changes of volume of the extremities were estimated from diastolic values of the plethysmographic tracings.

**Results**

Respiratory variations of blood flow were found in every peripheral area in which measurements were made. They stopped during periods of apnea (fig. 1). The relationship of these changes to those of cardiac output and blood pressure can be seen in figure 2. When the beat-to-beat calculations were made of cardiac out-
output by the pressure-pulse and ballistocardiographic methods, it was found that the two changes in opposite directions. Blood pressure, systolic more than diastolic, and peripheral blood flow mirrored the changes in left ventricular output. Changes in the volume of the forearm followed the level of blood pressure with a lag of approximately one beat (fig. 3). This interrelationship of central and peripheral changes was constant in all parts of the body studied.

![Fig. 2. The interrelationship of the central and peripheral circulatory changes during respiration. This record, taken at a slow camera speed, shows the interrelationships of circulatory changes during respiration in a 30 year old hypertensive male (A. S.) From top to bottom are the electrocardiogram (lead II), respiration (the peaks indicate inspiration), the ballistocardiogram (BCG), brachial blood pressure (with the scale in mm. of Hg at the left), volume of the forearm (down indicates an increase), volume of the pinna of the ear, and of the second toe (up indicates an increase). The variations have been underlined for accentuation in the central portion of the record. During inspiration the ballistocardiographic complexes increase in size, blood pressure falls, and the volume of the peripheral parts diminish. All these changes are in the opposite direction during expiration. The bracket at the bottom of the record indicates a two second interval.

The respiratory variations of blood pressure were often of considerable magnitude, systolic varying as much as 26 mm. Hg and diastolic, 6 mm. Hg. The ballistocardiographic stroke index changed by as much as 69 per cent, while the maximal change of the pressure-pulse index was only 7.2 per cent, the greatest individual variations being found in hypertensive patients (table 1). A comparison of the respiratory changes of right and left ventricular stroke

output, as calculated by these methods, showed those of the right to be very much greater than those of the left in every individual but one (fig. 4). There was an apparent difference in the variation of left ventricular stroke volume between the normotensive and hypertensive

![Fig. 3. Beat-to-beat relationships of central and peripheral circulatory changes during respiration. Three respiratory cycles of a hypertensive patient (A. S.) have been plotted. During the inspiratory phase, between the first two vertical lines, the ballistocardiographic (BCG) stroke index shows an increase of right ventricular stroke volume while the pressure-pulse stroke index shows a decrease of the output from the left side of the heart. The opposite changes occur during expiration (between the second and third vertical lines). The discrepancy between the magnitude of the two stroke indexes is explained in the text. The changes of systolic, and to a lesser extent diastolic, blood pressure parallel those of the output of the left heart as does the volume of the forearm (S and D indicate systolic and diastolic volume respectively). However, the latter shows a lag of one beat. The pulse rate varied between 73 and 75 per minute, the black dots showing individual beats.

* In 2 patients left ventricular output decreased during expiration, and in one the output, calculated from the ballistocardiograph, decreased during inspiration (table 1).
were of minor degree, the calculated values for peripheral resistance might possibly be interpreted as compensatory (peripheral resistance decreasing during a rise of blood pressure) in 6 of the 11 normotensive individuals but in only 4 of the 13 hypertensives; however, only 6 showed changes greater than 3 per cent (table 1).

Fig. 4. Percentile respiratory changes of right and left ventricular stroke indexes. The black portion of each bar represents percentile changes of left ventricular stroke index (pressure-pulse method) while the total height of each bar represents the total percentile changes of the ballistocardiogram. The open portion of each bar, therefore, represents the changes of right ventricular stroke index (that portion of the ballistocardiographic change due to variations in the output of the right heart). In every case except the first normotensive it is considerably greater than that of the left heart. This variation is more pronounced in the hypertensive group. In the 2 cases marked with an x, left ventricular stroke volume increased during inspiration; in the patient marked with a check the ballistocardiogram showed a decrease during inspiration.

DISCUSSION

Validity of the Methods Used

The methods used for the estimation of right and left ventricular stroke volume are open to criticism but there are no others which provide beat-to-beat measurements of these functions. It is probable that they are adequate to indicate general trends for reasons to be brought out below, but they cannot be considered to be quantitative. Both are probably semiquantitative. The plethysmographs afford only direct measurements of directional changes of blood flow as they merely measure alterations in the volume of the part and in the amplitude of the pulse.

The present paper is the first attempt to quantitate the respiratory variations of the output of the right heart by the ballistocardiogram. While the kinetic energy of the blood ejected by the left ventricle is thought to contribute a somewhat greater proportion to the forces which produce the ballistocardiographic complex, the respiratory variation of the complexes is believed to be largely a reflection of changes of right ventricular ejection.17, 18 Although other factors are known to affect the respiratory variations, i.e., positive pressure breathing21 which diminishes right ventricular output,22 myocardial disease,23 and pooling in the splanchnic bed,24 they do not appear to be of significance in the present study. The values of respiratory changes of right ventricular stroke volume estimated by cardiac catheterization in man2 are of the order of magnitude of those calculated above (table 1). The respiratory changes observed in the stroke volume of the left heart were small. The method used provides only an estimate of the alterations in that the calculations are based on arbitrary values of functions which are known to differ from individual to individual. Consequently, it might be inadequate for the quantitation of such small variations of stroke index. It is difficult to conceive, however, that the method could be so inaccurate as to be erroneous in regard to direction. It is striking that the calculated percentile changes of left ventricular stroke volume are also within the range estimated by cardiac catheterization in man.5

When the values of the mean ballistocardiographic stroke indexes were corrected so as to be comparable to those obtained with direct Fick methods, for example, BCGmean + 0.18 BCGmean25 they gave a good correlation with the pressure-pulse stroke index in 9 of the 11 normotensive patients and in 9 of the 13 hypertensives, provided that errors up to 20 per cent were allowed in both methods.* The corre-
Correlation was good without this correction in the normotensives (table 2). A correlation of this range is not insignificant in view of the indirect methods used.

On the other hand no correlation was found between the magnitude of the respiratory variations of blood pressure and those of left ventricular stroke index in each individual, as entirely understood. There are two major schools of thought.24, 27

1. The rate of filling of the right heart depends upon intrathoracic pressure, if systemic venous pressure is constant. During inspiration intrathoracic pressure falls and consequently venous return and filling are increased. Right ventricular output and pulmonary arterial

Table 2.—Correlation of Stroke Index by Ballistocardiographic and Pressure-Pulse Methods

<table>
<thead>
<tr>
<th>Patients</th>
<th>BCG*</th>
<th>BCG + 0.18</th>
<th>BCG (A) +20%</th>
<th>(A) -20%</th>
<th>P.P. + (B)</th>
<th>P.P. - (B)</th>
<th>Correlation of 3 cc. or Less</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normotensives</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>R. J.</td>
<td>28.3</td>
<td>33.4</td>
<td>40.1</td>
<td>—</td>
<td>58.7</td>
<td>—</td>
<td>47.0 No</td>
</tr>
<tr>
<td>R. S.</td>
<td>33.6</td>
<td>39.7</td>
<td>47.6</td>
<td>—</td>
<td>71.7</td>
<td>—</td>
<td>57.4 No</td>
</tr>
<tr>
<td>O. H.</td>
<td>53.1</td>
<td>62.7</td>
<td>—</td>
<td>50.1</td>
<td>54.7</td>
<td>—</td>
<td>— Yes</td>
</tr>
<tr>
<td>L. M.</td>
<td>45.3</td>
<td>53.5</td>
<td>64.1</td>
<td>—</td>
<td>61.2</td>
<td>—</td>
<td>— Yes</td>
</tr>
<tr>
<td>B. M.</td>
<td>55.5</td>
<td>65.5</td>
<td>—</td>
<td>52.4</td>
<td>54.0</td>
<td>—</td>
<td>— Yes</td>
</tr>
<tr>
<td>T. B.</td>
<td>54.6</td>
<td>64.4</td>
<td>—</td>
<td>—</td>
<td>63.6</td>
<td>—</td>
<td>— Yes</td>
</tr>
<tr>
<td>T. C.</td>
<td>39.4</td>
<td>46.5</td>
<td>—</td>
<td>37.2</td>
<td>38.1</td>
<td>—</td>
<td>— Yes</td>
</tr>
<tr>
<td>J. V.</td>
<td>35.8</td>
<td>42.3</td>
<td>—</td>
<td>33.8</td>
<td>38.2</td>
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* Ballistocardiographic stroke index.
† Ballistocardiogram corrected so as to be within the range of cardiac output determined by the Fick principle.25
‡ Pressure-pulse stroke index. This equals that obtained by the Fick principle within ± 20 per cent.19
§ Correlation within 5 cc. where errors up to 20 per cent are allowed in both methods (see text and references 19 and 25).

might be expected. The calculated change of peripheral resistance did not account for the difference. It is possible that the effects of intrathoracic pressure may have obscured the correlation.

Theoretic Considerations

The mechanism of the various circulatory changes accompanying quiet breathing is not pressure increase; the pulmonary vascular bed is enlarged more than enough to compensate for the increased stroke volume, and therefore blood pools in the lungs as a result of these events. Filling of the left heart is reduced, its output diminished, and blood pressure falls. All these changes are reversed during expiration. The pulmonary vascular bed therefore acts as a cushion to prevent large variations in systemic pressure.25
2. The stroke volume of the right heart is constant and is unaffected by intrathoracic pressure. All the above described changes are due to alterations of pulmonary vascular resistance which are caused by variations of intrathoracic pressure. Resistance is increased during inspiration, as a result of inflation of the lung, leading to decreased filling of the left ventricle.

This study does not attempt to decide which of these views is correct but strongly favors the first.

A. It is fortunate that the large respiratory variations in output of the right heart do not cause similar alterations in the output of left heart with consequent fluctuations of arterial pressure. Only in conditions severely limiting left heart output are these normal changes exaggerated to the point where the so-called "paradoxical pulse" becomes obvious. Their extent may afford a clinical index of certain functions of the pulmonary circulation and heart. Those individuals showing large variations in right heart output did not necessarily have large variations in the output of the left.

B. As has been suggested by Hamilton, part of the respiratory variation of blood pressure is due to direct transmission of changes of intrathoracic pressure through the large central blood vessels to the periphery. Consequently, intrathoracic pressure exerts a direct but small effect upon blood pressure. The primary cause of the respiratory variation of blood pressure, however, is probably cardiac in origin. If changes of blood pressure were due to intrathoracic pressure alone, one should obtain identical changes of systolic and diastolic pressures which closely followed the fluctuations of intrathoracic pressure. Diastolic changes were considerably smaller. The peaks of the changes of systolic pressure usually but not necessarily paralleled the position of the chest wall (if one can consider the pneumograph to follow intrathoracic pressure). In addition the variation of systolic pressure exceeded 5 mm. Hg, the normal respiratory change of intrathoracic pressure, in 10 patients of the series.

C. As deep inspiration is known to cause vasoconstriction, it appeared conceivable that some neurogenic mechanism dependent upon respiration was affecting arteriolar size and, in turn, peripheral flow. It is not necessary, however, to postulate such a reflex. Application of the existing knowledge of the time of response of blood vessels to stimulation of an autonomic nerve makes it unlikely that the variations of flow are neurogenic in origin. They are not abolished by the intravenous injection of tetraethylammonium chloride in doses sufficient to cause a pronounced fall of blood pressure in man, but do disappear immediately on voluntary cessation of respiration. Thus it seems likely that the variations of flow are due to the changes of central blood pressure. The peripheral changes of flow in part cushion the changes of pressure.

Differences between Normotensive and Hypertensive Subjects

1. The finding that respiratory variations of the ballistocardiogram are on the average greater than normal in hypertensive subjects is similar to the experience of Brown and de Lalla, and suggests that alterations in output of the right heart may be greater in hypertension. However, this disease can result in weakening of the left ventricle which is known to increase the respiratory variations of the ballistocardiogram.

2. The respiratory changes of left ventricular output were found to be smaller in the hypertensive group but this apparent difference is probably due to factors inherent in the calculations. A small error in the estimation of blood pressure at low levels gives greater increments of stroke volume by the pressure-pulse method than at hypertensive levels.

3. Respiratory variations of blood pressure were on the average greater in the hypertensive group. This finding was to be expected because of the characteristics of the pressure volume curve of the aorta, that is, a given increase

* Although no explanation is given for these findings, the most plausible one is that the kinetic energy of the blood ejected by the left ventricle is reduced. Consequently the blood expelled by the right ventricle exerts a greater effect upon the size of the complexes. However in many instances these results may result, to a great extent, from the secondary effect of pooling of blood in the peripheral circulation and inadequate venous return to the heart.
of volume causes a greater change of blood pressure at high than at low levels. As observed by others some of the changes of blood pressure were marked.  

4. The calculated changes of resistance indicated that the expiratory increase of peripheral blood flow cushioned the elevation of blood pressure in many normotensive individuals. In contrast, this occurred in only 3 of the hypertensive group (table 1). Apparently peripheral blood flow was not as readily increased. This finding is to be expected since peripheral resistance is elevated in hypertension. However, this interpretation is open to criticism.

**SUMMARY AND CONCLUSIONS**

1. During the inspiratory phase of quiet respiration in normotensive and hypertensive individuals (a) the size of ballistocardiographic complex grows larger, indicating an increase of right ventricular stroke index; (b) left ventricular stroke index, estimated by the pressure-pulse method, decreases; (c) blood pressure falls, systolic more than diastolic; (d) regional blood flow diminishes. The opposite changes occur during expiration.

2. The interrelationships of these changes are discussed.

3. It is suggested that the combined use of the ballistocardiographic and pressure-pulse methods provides a semiquantitative measure by which the respiratory variations of right and left ventricles may be separated.

4. The large respiratory variations in the output of the right heart are not correlated with those of the left. The discrepancy between the two may afford an index of clinical variations in the heart and pulmonary circulation.

5. Respiratory variations in regional blood flow are probably secondary to changes of arterial pressure, and are not under nervous control.

6. It is possible that the cushioning effect of the peripheral circulation is not normal in some cases of hypertension.

**Acknowledgment**

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Interrelationships of Cardiac Output, Blood Pressure, and Peripheral Resistance during Normal Respiration in Normotensive and Hypertensive Individuals
ARNOLD H. WILLIAMS and ARTHUR L. GROPPER

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