Pulmonary Function in Left Ventricular Failure, Including Cardiac Asthma

By Richard S. Cosby, M.D., Ellery C. Stowell, Jr., Ph.D., W. Ray Hartwig, B.S., and M. Mayo, A.B.

Usually comprehensive studies of pulmonary function in hypertensive patients during left ventricular failure and cardiac asthma are presented. These findings are compared to those in mitral stenosis with congestive failure and in pulmonary emphysema with right heart failure. All patients were severely dyspneic and bedridden.

Although Harrison¹ and others have presented detailed studies of blood gases in left heart failure and cardiac asthma, and a number of papers on the experimental production of cardiac asthma and pulmonary edema are available,² there are no complete studies of the markedly abnormal respiratory patterns in this state. McCann³ in a recent review has referred to the elevation of arterial pCO₂ in the later stage of cardiac decompensation as “replacing the initial hypocapnia.” Rodbard⁴ has emphasized the importance of bronchospasm in cardiac asthma. Our study will attempt to describe the respiratory pattern of left-sided heart failure in an effort to define the physiologic characteristics of such a state. Particular attention will be paid to the respiratory defects found in those patients with cardiac asthma. Studies of the respiratory patterns in mitral stenosis and in emphysema will be used for comparison and contrast.

Material and Methods*

Eight patients with hypertensive heart disease in congestive failure (group I), 14 patients with rheumatic heart disease, mitral stenosis, and congestive failure (group II), and 16 patients with emphysema and right heart failure (group III) were subjected to an evaluation of their pulmonary function. The patients were selected specifically for their severe degree of dyspnea, irrespective of the presence or absence of wheezy respiration. All were bedridden at the time of examination. The usual lung volume studies were obtained, and analyses of distribution and diffusion were performed according to the techniques of Riley and co-workers.⁵ In addition, arterial oxygen saturations following positive pressure breathing, exercise, and 100 per cent oxygen were obtained in some of the patients. The expiratory flow pattern was analyzed by recording a pneumotachogram simultaneously with expiratory pO₂ and CO₂ curves, with both an infrared analyzer and a Beckman mass spectrometer. The mass spectrometer furnished information on alveolar oxygen, alveolar carbon dioxide, and pulmonary clearance. The linearity of response with gas concentration, the rapid inherent response time of the instrument, and its ability to monitor all respiratory gases consecutively rendered it invaluable.

Results

The results of the ventilatory pulmonary function studies in all 3 groups are summarized in table 1. Because of the non-normal distribution of the data the measurements are summarized as medians in each category and non-parametric tests of statistical significance, in the analysis of variance and the 95 per cent confidence limits, were utilized.⁶ In table 1, patients in group I (hypertensive heart disease with congestive heart failure) show a marked reduction in vital capacity, maximum breathing capacity, air velocity index, and breathing reserve expressed as a per cent of maximum breathing capacity. In these 4 categories the hypertensive patients are significantly different from the patients with rheumatic heart disease. The ventilatory abnormalities in the hypertensive group are obstructive in nature.

In comparison with the cases in group I,
patients in group II (rheumatic heart disease and mitral stenosis with congestive heart failure) have only a moderate ventilatory impairment; moreover, they show none of the bronchospastic features present in the hypertensive patient. This is more clearly brought out by the significantly higher ventilation factor in the patients with rheumatic heart disease. The ventilation factor of Motley7 averages 3 measurements: maximum breathing capacity, timed vital capacity, and residual air, expressed as a per cent of the predicted value in each case.

As expected, the patients in group III (emphysema with right heart failure) show marked differences from the group with mitral stenosis and congestive failure. The characteristic ventilatory defects in emphysema are clearly depicted in table 1—diminished vital capacity and maximum breathing capacity, low air velocity index, diminished ventilation factor, and a markedly low ratio of breathing reserve to maximum breathing capacity. The similarity of these data to comparable measurements in hypertensive heart disease with congestive failure is striking and again emphasizes the obstructive nature of respiration in both states. To be sure, these measurements are considerably more abnormal in emphysema, showing the greater obstructive element, as exemplified by the comparison of the breathing reserve to maximum breathing capacity in both groups.

Blood gas studies in the 3 groups are summarized in table 2. Patients in group I show a significantly low arterial pO2 and a significantly high arterial pCO2. Note that the high arterial pCO2 leads to a low calculated alveolar pO2, a factor that appears to be responsible for the normal aeration gradient found in this group. The arterial oxygen saturation is somewhat below the normal, the transfer gradient is double the normal value, the per cent venous admixture is increased, and the oxygen diffusing capacity is somewhat reduced. There is a slight increase in arterial oxygen saturation on exercise and a slight decrease in the change in "true oxygen" after exercise.

In patients in group II the median value of arterial pO2 is practically normal and the value of arterial pCO2 is below normal. The aeration gradient is somewhat smaller than in group I, probably because of the lower arterial pCO2. The transfer gradient in group II is again double the normal value; the per cent venous admixture is within the normal range, although an occasional patient did show marked evidence of shunting. The oxygen diffusing capacity is lower than in group I, primarily due to impaired diffusion in patients with high pul-

### Table 1.—Ventilatory Measurements

<table>
<thead>
<tr>
<th></th>
<th>Hypertensive (8)</th>
<th>Rheumatic (14)</th>
<th>Pulmonary (16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital capacity (% normal)§</td>
<td>44.7†</td>
<td>73.5†</td>
<td>44.5</td>
</tr>
<tr>
<td>Maximum breathing capacity (% normal)</td>
<td>40.7*</td>
<td>58.8*</td>
<td>33.5*</td>
</tr>
<tr>
<td>Air velocity index</td>
<td>0.666†</td>
<td>0.965†</td>
<td>0.658</td>
</tr>
<tr>
<td>Tidal volume ml</td>
<td>434</td>
<td>536</td>
<td>448</td>
</tr>
<tr>
<td>Ventilation (L./min./M.²)</td>
<td>4.46</td>
<td>5.00</td>
<td>5.01</td>
</tr>
<tr>
<td>Physiologic dead space as % tidal volume</td>
<td>51.0</td>
<td>40.2</td>
<td>58.0</td>
</tr>
<tr>
<td>Alveolar ventilation (L./min./M.²)</td>
<td>2.40</td>
<td>3.64</td>
<td>2.24</td>
</tr>
<tr>
<td>Timed vital capacity in 3 seconds as % vital capacity</td>
<td>93.7</td>
<td>93.0</td>
<td>88.5</td>
</tr>
<tr>
<td>Breathing reserve as % Maximum breathing capacity</td>
<td>70.0†</td>
<td>85.0†</td>
<td>60.2†</td>
</tr>
<tr>
<td>Ventilation factor (%)</td>
<td>45.5†</td>
<td>59.3†</td>
<td>40.9</td>
</tr>
</tbody>
</table>

* Differences between groups significant at the 5 per cent level.
† Differences between groups significant at the 1 per cent level.
‡ Significant at the 5 per cent level—hypertensive vs. rheumatic.
§ All ventilatory measurements expressed at body temperature, saturated ambient pressure.
PULMONARY FUNCTION IN VENTRICULAR FAILURE

Table 2.—Blood Gases

<table>
<thead>
<tr>
<th></th>
<th>Hypertensive (8)</th>
<th>Rheumatic (14)</th>
<th>Pulmonary (16)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Median</td>
<td>95% Confidence Limits</td>
<td>Median</td>
</tr>
<tr>
<td>Arterial pO₂ mm. Hg</td>
<td>84.6*</td>
<td>47.9–100.1</td>
<td>94.1*</td>
</tr>
<tr>
<td>Arterial pCO₂ mm. Hg</td>
<td>44.6*</td>
<td>35.5–47.7</td>
<td>36.0*</td>
</tr>
<tr>
<td>% arterial O₂ saturation</td>
<td>93.8‡</td>
<td>82.5–98.1</td>
<td>96.8</td>
</tr>
<tr>
<td>% change in above after exercise</td>
<td>+0.2‖</td>
<td>−0.3–+3.5</td>
<td>+2.3‡</td>
</tr>
<tr>
<td>No. reaching 100% after 100% O₂</td>
<td>All</td>
<td>—</td>
<td>5 of 7</td>
</tr>
<tr>
<td>Aeration gradient (mm. Hg)</td>
<td>48.0†</td>
<td>39.3–56.8</td>
<td>39.0†</td>
</tr>
<tr>
<td>Transfer gradient (mm. Hg)</td>
<td>19.0‡</td>
<td>6.6–15.1</td>
<td>20.0</td>
</tr>
<tr>
<td>% venous admixture</td>
<td>11.5</td>
<td>3.0–20.0</td>
<td>5.0</td>
</tr>
<tr>
<td>O₂ diffusing capacity</td>
<td></td>
<td>12.3</td>
<td>8.1–16.5</td>
</tr>
<tr>
<td></td>
<td>3.0–5.0</td>
<td>3.0–20.0</td>
<td>5.0</td>
</tr>
<tr>
<td></td>
<td>—</td>
<td>—</td>
<td>44.0</td>
</tr>
</tbody>
</table>

* Differences between groups significant at the 5% level.
† Differences between groups significant at the 1% level.
‡ Significant at 5% level—pulmonary vs. hypertensive.
§ Significant at 5% level—pulmonary vs. rheumatic.
‖ No. in groups too small for statistical analysis.

Table 3.—Ventilation and Blood Gas Studies in Eight Cases of Hypertensive Heart Failure

<table>
<thead>
<tr>
<th></th>
<th>Hypertensive (8)</th>
<th>Rheumatic (14)</th>
<th>Pulmonary (16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital capacity* % normal</td>
<td>20.0</td>
<td>24.9</td>
<td>38.0</td>
</tr>
<tr>
<td>Maximum breathing capacity % normal</td>
<td>10.9</td>
<td>3.1</td>
<td>15.0</td>
</tr>
<tr>
<td>Timed vital capacity as a % of vital capacity</td>
<td>95.5</td>
<td>86.0</td>
<td>90.0</td>
</tr>
<tr>
<td>Air velocity index</td>
<td>0.545</td>
<td>0.123</td>
<td>0.395</td>
</tr>
<tr>
<td>Ventilation factor (%)</td>
<td>13.0</td>
<td>22.0</td>
<td>22.0</td>
</tr>
<tr>
<td>Breathing reserve as a % of Maximum breathing capacity</td>
<td>44.0</td>
<td>71.0</td>
<td>21.0</td>
</tr>
<tr>
<td>Physiologic dead space as a % tidal volume</td>
<td>52.3</td>
<td>40.0</td>
<td>51.0</td>
</tr>
<tr>
<td>Alveolar ventilation L./min./M.²</td>
<td>1.40</td>
<td>3.45</td>
<td>2.32</td>
</tr>
<tr>
<td>Arterial pO₂ (mm. Hg)</td>
<td>86.0</td>
<td>80.0</td>
<td>84.6</td>
</tr>
<tr>
<td>Arterial pCO₂ (mm. Hg)</td>
<td>47.0</td>
<td>42.0</td>
<td>44.5</td>
</tr>
<tr>
<td>% arterial oxygen saturation</td>
<td>97.3</td>
<td>94.0</td>
<td>90.0</td>
</tr>
<tr>
<td>Aeration gradient (mm. Hg)</td>
<td>59.0</td>
<td>48.0</td>
<td>49.0</td>
</tr>
<tr>
<td>Transfer gradient (mm. Hg)</td>
<td>15.0</td>
<td>22.0</td>
<td>17.0</td>
</tr>
<tr>
<td>Wheezing</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

* All ventilatory measurements expressed at body temperature, saturated, ambient pressure.
† Patient in pulmonary edema.

The other 2 groups, a factor undoubtedly related to the marked increase in the per cent venous admixture. The oxygen diffusing capacity is low, about equal to that found in group II. Striking differences are found in measurements taken after exercise. In group III, there is a marked fall in arterial oxygen saturation, and the true oxygen rise.

Table 3 shows the respiratory data in indi-
Individual patients in group I. The first 5 patients show severe bronchospastic respiration; these are considered to have cardiac asthma. They show a decrease in vital capacity, and a still greater decrease in maximum breathing capacity and breathing reserve. The other 3 patients had been admitted to the hospital in congestive heart failure, but did not present wheezy respiration at the time of examination. The ventilatory measurements are, on the whole, less abnormal in these 3 cases.

In their blood gas studies, the first 5 patients show a definite increase in arterial pCO₂; minimal arterial oxygen desaturation is present in 3 of the 5 cases, and 1 of these, in pulmonary edema, has an arterial saturation well below 90 per cent. It appears that the first 5 patients have true respiratory insufficiency in the same sense, but not in the same degree as do our patients with severe emphysema. Although the actual resting ventilation is increased above the normal range, a marked increase in the ratio of physiologic dead space to tidal volume is present, and thus the alveolar ventilation is inadequate. It is remarkable that such patients are able to maintain nearly normal arterial oxygen saturation with such diminished lung volumes, bronchospastic respiration, and arterial CO₂ retention.

Figures 1, 2, and 3 are diagrams of simultaneous pneumotachograms and pCO₂ and pO₂ curves of typical patients in each group, taken during expiration. These alveolar CO₂ and O₂ curves were obtained by the Beckman mass spectrometer. They portray the breathing pattern characteristic in hypertensive cardiac asthma, mitral stenosis with congestive failure, and also emphysema with right heart failure. In the typical patient in group I (fig. 1) the pneumotachogram shows a prolonged obstructive-type expiration with a low tidal volume. The normal curves are shown in each case for comparison. The alveolar pCO₂ level is somewhat above the normal level and the alveolar pO₂ level is reduced.

In the typical patient in group II (fig. 2), the pneumotachogram shows an increased tidal volume and no obstruction to expiration. The alveolar pCO₂ level is below the normal level, but the alveolar pO₂ is normal.

In the typical patient in group III (fig. 3),

**Fig. 1.** Single breath analysis of alveolar pO₂, alveolar pCO₂, and tidal volume in hypertensive heart disease with congestive failure. Curves show decreased alveolar pO₂, increased alveolar pCO₂, diminished tidal volume (stippled), prolongation of expiration and tachypnea. Normal single breath alveolar pO₂, alveolar pCO₂ curves and tidal volume (black area) are presented for comparison.

**Fig. 2.** Single breath analysis of alveolar pO₂, alveolar pCO₂ and tidal volume in rheumatic heart disease with mitral stenosis, and congestive heart failure. Curves show elevated alveolar pO₂, diminished alveolar pCO₂, large tidal volume (stippled) and no evidence of obstruction to expiration. Mild tachypnea was present. Normal single breath alveolar pO₂ and alveolar pCO₂ curves and tidal volume (black area) are presented for comparison.
FIG. 3. Single breath analysis of alveolar \( pO_2 \), alveolar \( pCO_2 \) and tidal volume in emphysema with right heart failure. Curves show diminished alveolar \( pO_2 \) and a marked increase in alveolar \( pCO_2 \). A marked prolongation of expiration was present, characteristic of ventilatory insufficiency. Normal single breath alveolar \( pO_2 \) and \( pCO_2 \) curves and tidal volume (black area) are presented for comparison.

the resemblance to the pattern found in hypertensive heart disease with congestive heart failure is again evident. Here, even in the presence of a normal tidal volume, a pronounced obstruction to expiration is present. There is a markedly high alveolar \( pCO_2 \) level, and a low alveolar \( pO_2 \).

**DISCUSSION**

The ventilatory measurements outlined in table 1 clearly demonstrate a marked difference between the hypertensive and the rheumatic patient with mitral stenosis in congestive heart failure. An obstructive-type ventilation is characteristic in the hypertensive in failure, and is most uncommon in the patient with mitral stenosis and heart failure.

Because of such differences, analyses of ventilatory and respiratory measurements in heart failure must take into consideration the underlying etiologic states. In the past, many authors studied lung volumes in heart disease as a whole. Richards and associates,\(^8\) for example, measured the lung volumes of patients with congestive heart failure, regardless of etiology. These authors stated that as heart failure progressed, the complementary air diminished markedly, the reserve air became smaller than normal, with a marked decrease in vital capacity, and in extreme failure, the functional residual air was also smaller than the normal. Evidence was presented that increased heart size was an important factor in the production of these changes. No differentiation was made between hypertensive, coronary, or rheumatic heart failure.

The studies presented in table 1 emphasize the dissimilarities present in the 2 cardiac groups, particularly the markedly bronchospastic respiration characteristic of the hypertensive patient, and the lower vital capacity, breathing reserve and maximum breathing capacity found in group I.

The specific entity “cardiac asthma” was studied by Harrison,\(^1\) who emphasized the marked decrease in vital capacity and concomitant increase in ventilation present in this state. Heyer\(^9\) studied 11 patients with cardiac asthma; 3 had cardiac asthma due to syphilitic aortic insufficiency, 4 had hypertensive heart disease, 2 had arteriosclerotic heart disease, and 2 had rheumatic heart disease with mitral stenosis. The spiromgrams in these patients were identical to those taken on patients with bronchial asthma. Prolongation of expiration was present in both conditions. The expiratory-inspiratory ratio (in seconds) was 1.61 in the normal; in cardiac asthma it was 2.67, and in bronchial asthma it was 2.14. Patients who did not show prolonged wheezing on physical examination might still have a prolongation of expiration well above the normal range. Patients with cardiac asthma improved after receiving aminophylline, with a definite increase in vital capacity.

Quite similar findings were reported by Plotz,\(^10\) who studied 9 patients; 5 had cardiac asthma due to hypertensive heart disease, 3 had cardiac asthma due to coronary heart disease, and only 1 had cardiac asthma due to rheumatic heart disease. These 9 patients were given epinephrine. The average vital capacity before epinephrine was 2.15 l., with an average increase of 425 ml. following epinephrine. Patients with basal rales but without prolonged expiration were not relieved by epinephrine.
He concluded that epinephrine relieved the bronchial spasm of cardiac asthma with an increase in vital capacity.

The data in table 3 showing the ventilatory data of individual patients with hypertensive heart disease with congestive heart failure present 5 patients who compare with those described by Harrison, Heyer, and Plotz. The bronchospastic pattern described by Heyer and Plotz was essentially that of hypertensive heart disease and cardiac asthma, since only 3 of the combined 20 patients in the 2 series had rheumatic disease with mitral stenosis and cardiac asthma. The rarity of cardiac asthma in the presence of mitral stenosis is well known; none of the patients in our group II showed bronchospastic respiration or clinical wheezing.

Much attention has been given recently by Frank's group, Richards, and Cosby and associates to the ventilatory problems in mitral stenosis, with and without congestive heart failure. It was shown by Cosby and co-workers that the degree of dyspnea was more closely correlated with the height of the pulmonary artery pressure and with vital capacity than with maximum breathing capacity. The vital capacity was reduced but not to levels as low as those seen in hypertensive heart disease with failure. Little evidence of bronchospasm was present.

Frank and co-workers stressed the increasing minute ventilation in this condition and pointed out that effective alveolar ventilation was maintained throughout all stages of disability. These authors re-emphasized the absence of obstructive defect. Richards emphasized the discrepancy between the very moderate degree of ventilatory impairment and the marked degree of exertional dyspnea.

The relation between gas exchange and ventilation has been considered an important measurement of the degree of disability and has been employed to evaluate the cardiac element in dyspnea. McMichael, for example, noted a poor correlation between vital capacity and cardiac output but an excellent inverse correlation between the ventilatory equivalent for carbon dioxide and cardiac output. This was particularly useful in measuring sequential changes in the same patient. He considered the ventilatory equivalent for carbon dioxide more critical than that for oxygen, since it was less dependent upon the respiratory quotient. Frank and co-workers and Lindgren found an increased ventilatory equivalent for oxygen in mitral stenosis, but there was no relationship between the ventilatory equivalent and the degree of disability.

In the 3 groups studied in this laboratory, the only significant measurement related to gas exchange on exercise was the change in "true" oxygen on exercise. Patients with emphysema were able to increase the amount of oxygen extracted from inspired air slightly, although the increase was not a normal one. On the other hand, patients in the rheumatic group showed a significant decrease in "true" oxygen on exercise. Measurements of ventilatory equivalents were not conclusive, although all were somewhat elevated at rest. Because of the unstable cardiorespiratory state of most of these patients it would seem likely that a derived measurement so dependent upon ventilation would have greater variability than the per cent change in "true" oxygen on exercise.

Several authors have studied blood gas exchange in heart disease. Varnauskas has recently reported on 49 hypertensive patients in varying degrees of congestive failure, who showed a mean arterial desaturation of 3 per cent below the normal. Harrison reported on 5 patients with similar disability; 2 fell below 90 per cent. The most extensive study of the alveolar-arterial gradient in cardiac subjects has been presented by Storstein. He studied 4 patients with hypertensive heart disease, 15 patients with rheumatic heart disease, and 8 patients with pulmonary disease and emphysema. Median arterial oxygen saturations were as follows: 93.8 per cent (91.9 to 94 per cent) in the hypertensive group; 93.2 per cent (80.0 to 98.7 per cent) in the rheumatic group; and 83.0 per cent (76.9 to 90.7 per cent) in the pulmonary group. Median alveolar-arterial gradients were as follows: 23.4 mm. (18.5 to 37.7 mm.) in the hypertensive group; 21.5 mm. (6 to 33 mm.) in the rheumatic group; and 47.5 mm. (31.3 to 55.0 mm.) in the pulmonary group. There is close agreement between Storstein's data and
the data shown in table 2 relative to arterial oxygen saturation and the transfer gradient. The patients in our group III did not show quite such striking increases in the transfer gradient as did Storstein's\textsuperscript{17} comparable group. However, the agreement in the 2 series as a whole is noteworthy, especially on consideration of the fact that Storstein\textsuperscript{17} made no mention of the degree of disability in any of his patients; moreover, the arterial pO\textsubscript{2} in Storstein's\textsuperscript{17} series was calculated indirectly from the oxygen dissociation curve, while in our groups the arterial pO\textsubscript{2} was measured directly by Riley's technic.\textsuperscript{5}

With respect to abnormalities of distribution and diffusion, there are no specific studies available on patients in congestive heart failure. The data of Fowler and associates\textsuperscript{18} and Carroll and associates\textsuperscript{19} refer to patients with mitral stenosis, with or without heart failure. Of the 13 patients studied by Fowler's group\textsuperscript{18} only 1 was described as being in congestive failure at the time of examination. These authors specifically chose patients with increasing levels of pulmonary artery pressure and noted that in the 5 showing a decrease in oxygen diffusing capacity, 4 had markedly increased pulmonary artery pressure. Of the other 8 patients with lower pulmonary artery pressure, 5 showed an increase in per cent venous admixture and 6 showed an increase in the ratio of dead space to tidal volume.

The data in table 2 confirms those of Fowler and associates\textsuperscript{18} and Carroll and associates,\textsuperscript{19} revealing a marked reduction in oxygen diffusing capacity in the presence of elevated pulmonary artery pressure. A significant increase in per cent venous admixture was present in only 1 patient with rheumatic heart disease and 1 patient with hypertensive heart disease, both in pulmonary edema. The ratio of physiologic dead space to tidal volume was somewhat higher in the hypertensive than in the rheumatic group in our series.

Finally, the small group of patients with hypertensive heart disease and the severest degree of cardiac asthma deserves special mention. The presence of CO\textsubscript{2} retention in cardiac asthma has not been previously emphasized, although Peters and Barr\textsuperscript{20} had found increases of arterial pCO\textsubscript{2} up to 52 mm. in cardiac asthma. McCann\textsuperscript{3} believed that the initial hypocapnia in heart disease was replaced by elevation of the arterial pCO\textsubscript{2} as congestive failure became more severe. It seems most significant that those with the most pronounced degree of cardiac asthma had the lowest vital capacities and the highest arterial pCO\textsubscript{2} in the cardiac groups. This small group strongly resembled the patients with emphysema and right heart failure. Nevertheless their arterial oxygen saturation was minimally reduced, an effect that may have been due to the positive pressure effect of bronchospastic respiration.

**Summary**

In hypertensive heart disease with congestive heart failure, a marked reduction is present in vital capacity, maximum breathing capacity, air velocity index, and breathing reserve expressed as a per cent of maximum breathing capacity. Although not all of the patients complained of the wheezing characteristic of cardiac asthma, the majority of the patients with hypertensive heart disease in failure have an obstructive type of ventilatory impairment. Minimal abnormalities of diffusion, moderate increases in the physiologic dead space and minimal increases in the per cent venous admixture are present. Only in an occasional patient with frank pulmonary edema is marked arterial desaturation present. When cardiac asthma is severe, the vital capacity and maximum breathing capacity are markedly reduced, and true respiratory insufficiency with CO\textsubscript{2} retention ensues.

In rheumatic heart disease, mitral stenosis and failure, at a comparable level of disability, only moderate ventilatory impairment exists. The vital capacity is the measurement that most clearly reflects the ventilatory disability and obstructive ventilatory features are extremely rare. Ventilation tends to be greater than in hypertensive heart disease and thus the aeration gradient is significantly lower. The arterial pCO\textsubscript{2} is significantly reduced below the normal range. Greater abnormalities of diffusion are present than in the hypertensive group, and there is also an increase in the ratio
of physiologic dead space to tidal volume. Significant arterial oxygen desaturation is rare.

Patients with pulmonary emphysema at a comparable level of disability follow the known established patterns of respiratory impairment. There is a marked increase in residual air, and the obstructive ventilatory insufficiency strongly resembles the pattern found in hypertensive heart disease. However, the transfer gradient and per cent venous admixture are both far greater than in hypertensive heart disease, the arterial pCO₂ is higher and arterial oxygen saturation is lower than in cardiac patients with congestive failure. Thus blood gas measurements rather than ventilatory measurements more adequately separate the cardiac and pulmonary groups.

**Conclusions**

The characteristic respiratory pattern in patients with hypertensive heart disease with congestive failure and with cardiac asthma is one of obstructive ventilatory insufficiency, low vital capacity and maximum breathing capacity, minimal arterial oxygen desaturation, and arterial pCO₂ retention. In contrast, patients with mitral stenosis and congestive failure show hyperventilation, only moderate ventilatory impairment with no obstructive features, and a low arterial pCO₂. Patients with pulmonary emphysema, at a comparable level of disability, show a higher residual air and a lower breathing reserve. There is a greater degree of arterial oxygen desaturation, a far higher transfer gradient, a larger increase in per cent venous admixture, and a higher arterial pCO₂ than in patients with heart disease and congestive failure.

**Acknowledgment**

We gratefully acknowledge the valuable statistical advice of Dr. John C. Talbot, and the assistance of Dr. Robert W. Oblath, Dr. Lawrence M. Herman, Dr. J. Louis Freibrun, and Dr. Irwin Hoffman in the performance of cardiac catheterizations.

**Summario in Interlingua**

In morbo cardiac hypertensive con congestive disfallimento cardiae, un marcate reduction se manifesta in le capacitate vital, le capacitate respiratori maximal, le indice del velocitate aeree, e le reserva respiratori exprimite in pro cento del capacitate respiratori maximal. Ben que non omne le patientes se plangeva del rhoncho caracterisic de asthma cardiae, le majoritate del patientes con morbo cardiac hypertensive in disfallimento suffe de un typo obstructive de disturbation ventilatori. Es a notar minimal anormalitates de diffusion, moderate augmentos del spatio morte physiologic, e minimal augmentos in le procentage del admixture venose. Marcate dissaturation arterial se trova solmente in rar patientes con franc edema pulmonar. Quando le asthma cardiae es sever, le capacitate vital e le capacitate respiratori maximal es marcate-mente reduce, e ver insufficientia respiratori se disveloppa con retention de CO₂.

In morbo cardiac rheumatic, stenosis mitral, e disfallimento, il existe a comparabile nivellos de invaliditate solmente moderate grados de disturbation ventilatori. Le capacitate vital es le mesura que reflecte le plus clarmente le incapacity ventilatori, e obstructive tractos ventilatori es extrememente rar. Le ventilation tende a esser plus grande que in morbo cardiac hypertensive, e assì le gradient de aeration es significativamente plus basse. Le pCO₂ es reduce a grados significativamente infra le limites normal. Plus grande anormalitates de diffusion es presente que in le gruppo hypertensive, e il se nota etiam un augmento in le proportion inter le spatio morte physiologic e le volumine del aire currente. Grados significative de dissaturation arterial de oxygeno es rar.

Patientes con emphysema pulmonar a nivellos comparabile de incapacity obedí le establite e cognoscite modello de disturbation respiratori. Il occurre un marcate augmento del aire residue, e le obstructive insufficientia ventilatori es multo simile a lo que es trovate in morbo cardiac hypertensive. Tamen, le gradient de transferimento e le pro cento del admixture venose es ambes multo plus grande que in morbo cardiac hypertensive, le pCO₂ es plus alte e le saturation arterial de oxygeno es plus basse que in patientes con disfallimento congestive. Assì, mesurationes del gases del sanguine serve plus adequademente
que mesuraciones ventilatorias a separar le grupo cardiac e le grupo pulmonar.

Le sequente conclusiones es presentate: Le configuration characteristic del respiracion in patientes con morbo cardiac hypertensive associate con disfallamento congestive e con asthma cardiac es distinguite per obstructive insufficiencia ventilatoria, basse capacitatie vital e maximal capacitatie respiratorii, minimal dissaturation arterial de oxygeno, e retencion arterial de pCO2. In contrasto con isto, patientes con emphysema pulmonar a comparable niveles de incapacitate monstra un plus grande volumine de aire residue e plus basse reservas respiratorii. Il se nota in iste casos un plus alte grado de dissaturation arterial de oxygeno, un multo plus alte gradiente de transferimento, un plus grande augmento del procentage de admiotion venose, e un plus alte pCO2 arterial que in patientes con morbo cardiac e disfallamento congestive.

APPENDIX—DEFINITIONS

1. Vital capacity is the maximal volume of gas that can be expelled from the lungs by forceful effort following a maximal inspiration (ml).

2. Inspiratory reserve volume (formerly complemental air) is the maximal amount of gas that can be inspired from the end-inspiratory position (ml).

3. Expiratory reserve volume (formerly reserve or supplemental air) is the maximal volume of gas that can be expired from the end-expiratory level (ml).

4. Residual volume is the volume of gas remaining in the lungs at the end of a maximal expiration, customarily expressed as a per cent of the total capacity (ml).

5. Maximal breathing capacity is the maximal volume of gas that a subject can breath out per minute, usually measured over a 15-second interval (l/min).

6. Air velocity index is the per cent predicted maximum breathing capacity divided by the per cent predicted vital capacity.

7. Tidal volume is the volume of gas inspired or expired during each respiratory cycle (ml).

8. Ventilation is the volume of air expired per minute, expressed in liters per minute per square meter body surface.

9. Physiologic dead space is the volume of air in the trachea and bronchi (anatomic dead space) and in addition air from alveoli where circulation is reduced or absent. It is calculated by the Bohr equation, expressing the relationship of arterial and expired pCO2. It is customarily expressed as a per cent of tidal volume.

10. Alveolar ventilation is the total ventilation minus the dead space ventilation, expressed in liters per minute per square meter body surface.

11. Timed vital capacity is the volume of the total vital capacity expired in 3 seconds, expressed as a per cent of the vital capacity.

12. Breathing reserve as a per cent of maximum breathing capacity is the maximum breathing capacity minus resting ventilation divided by the maximum breathing capacity.

13. Ventilation factor is an average of (1) the 3-second timed vital capacity as a per cent of predicted vital capacity; (2) the maximum breathing capacity as a per cent of its predicted value; and (3) the normal residual air as a per cent of total lung volume divided by the observed residual air as a per cent total lung volume.

14. Aeration gradient is the pO2 of inspired air minus the pO2 of the alveolar air.

15. Transfer gradient is the pO2 of alveolar air minus the pO2 of arterial blood.

16. Venous admixture is the quantity of mixed venous blood reaching the peripheral arterial blood from right to left shunts such as bronchial and thebesian veins and abnormally from capillary blood in alveoli in which ventilation is reduced in relation to perfusion. It is expressed as a per cent of cardiac output.

17. Oxygen diffusing capacity is a measure of the permeability to oxygen of the alveolo-capillary membrane of the lung as a whole. The oxygen intake per minute divided by the calculated mean oxygen pressure gradient along the length of the capillary is the oxygen diffusing capacity, expressed as ml oxygen per minute per mm Hg.

18. True oxygen is the amount of oxygen extracted from inspired air, expressed in per cent per liter of expired air.

19. Ventilatory equivalent for carbon dioxide (or oxygen) is the minute volume of air in liters divided by each 100 ml of oxygen consumed or carbon dioxide produced.

REFERENCES


I affirm likewise of the blood in the veins, that the blood does always, and every where, run out of the lesse into the greater, and hastens towards the heart from every part: whence I gather that whatsoever quantity which is continually sent in, the arteries do receive by the veins, that the same does return and does at last flow back thither from whence it is first driven, and that by this means the blood moves circularly, being driven in its flux and reflux by the heart, by whose force it is driven into all the fibres of the arteries, and that it does afterwards successively by a continual flux return through the veins, from all those parts which draw, and streyn it through; sense it self teaches us that this is true, and collections from things obvious to sense takes away all occasion of doubt.—William Harvey, _De Circulatione Sanguinis_, 1649.
Pulmonary Function in Left Ventricular Failure, Including Cardiac Asthma
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