Pulmonary Function in Rheumatic Heart Disease and Its Relation to Exertional Dyspnea in Ambulatory Patients

By John R. West, M.D., Harry A. Bliss, M.D., John A. Wood, M.D., and Dickinson W. Richards, Jr., M.D.

Pulmonary and cardiocirculatory function were studied in 21 ambulatory patients with rheumatic heart disease. A pattern of pulmonary function was observed which deviated from normal to a degree insufficient to account for the exertional dyspnea of these patients. The pathogenesis of this symptom is, therefore, discussed in relation to the observed cardiocirculatory abnormalities which were often pronounced.

WITH few exceptions, notably the reports by Peabody and co-workers on maximal pulmonary ventilation induced by rebreathing expired air, studies directed at the problem of dyspnea of cardiac origin have been limited to determinations of pulmonary ventilation at rest and during exercise as well as measurement of various lung volumes in patients with heart disease.

While the presence of functional impairment of the lungs has been deduced from various abnormalities noted in these tests, particular emphasis has been placed upon the value of the vital capacity determination. According to one viewpoint the importance of this measurement lies in the beliefs, first, that dyspnea in heart disease is simply a manifestation of insufficiency of the pulmonary ventilatory apparatus due to both an increased ventilatory requirement and a decreased ventilatory capacity and, second, that the vital capacity affords a reliable index of this ventilatory capacity.

According to another opinion, however, one which stresses the point that cardiac dyspnea is largely "reflex" in nature, the vital capacity is important chiefly because it is a good index of pulmonary elasticity, a decrease in which is considered to be the main factor in the origin of "reflex" dyspnea. All of these concepts are, however, open to some doubt and are deserving of re-examination. Vital capacity has not proved to be a reliable index of ventilatory capacity in many forms of chronic pulmonary disease and may not be so in the case of heart disease. A causal relationship, furthermore, between the symptom of dyspnea in cardiac patients and the postulated insufficiency of the ventilatory apparatus, while plausible, has not been demonstrated.

Studies emphasizing dynamic concepts of pulmonary function as well as lung volume measurements have proved to be more useful than the latter alone in defining patterns of pulmonary dysfunction in chronic pulmonary disease and might be expected, therefore, to provide a means of evaluating the performance of the lungs in heart disease. The present report deals with a series of such pulmonary function studies as well as various cardiocirculatory measurements in a group of patients with rheumatic heart disease who presented varying degrees of exertional dyspnea.

METHODS OF STUDY

The pulmonary function of 21 individuals suffering from chronic, inactive rheumatic heart disease was studied according to methods previously described (table 1). Eighteen of these patients, group I, with uncomplicated heart disease, were free from edema, ascites, obvious hepatomegaly and pulmo-
Table 1.—Clinical Findings in 21 Patients with Rheumatic Heart Disease, Inactive, at the Time of the Physiologic Studies

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>B.S.A.</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. D.</td>
<td>F</td>
<td>23</td>
<td>1.56</td>
<td>N.S.R., M.S. No exertional dyspnea. No digitalis. Never in cardiac failure.* Class I. A</td>
</tr>
<tr>
<td>J. S.</td>
<td>M</td>
<td>42</td>
<td>1.72</td>
<td>N.S.R., M.S. Dyspnea only on strenuous exertion. No digitalis. Never in cardiac failure. Class I. A</td>
</tr>
<tr>
<td>M. R.</td>
<td>F</td>
<td>32</td>
<td>1.45</td>
<td>N.S.R., M.S. Dyspnea on mild exertion. Digitalized. Previous cardiac failure. Class II. C</td>
</tr>
<tr>
<td>E. S.</td>
<td>M</td>
<td>36</td>
<td>1.80</td>
<td>N.S.R., M.S., M.I., E.H. Dyspnea on moderate to severe exertion. Digitalized. Previous cardiac failure. Class II. C</td>
</tr>
</tbody>
</table>

**Group II**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age</th>
<th>B.S.A.</th>
<th>Remarks</th>
</tr>
</thead>
</table>

* The term "cardiac failure" as used here means right ventricular failure.

Abbreviations: B.S.A. = body surface area in square meters; N.S.R. = normal sinus rhythm; Aur. Fib. = auricular fibrillation; M.S. = mitral stenosis; M.I. = mitral insufficiency; A.S. = aortic stenosis; A.I. = aortic insufficiency; E.H. = enlarged heart.
nary roles at the time of study, although several gave
a history of having had right ventricular failure or
pulmonary edema or both at some time in the past.
While a few of the patients were asymptomatic the
majority complained of persistent exertional dyspnea
which in some was quite severe. According to the
criteria of the New York Heart Association\textsuperscript{18} there
were five in class I, five in class II, seven in class
III and one in class IV. Three other patients,
group II, presented definite clinical evidence of
intrinsic pulmonary disease in addition to heart
disease. They, too, were free from signs of conges-
tive failure or pulmonary edema at the time of
study, but each complained of severe exertional
dyspnea. In all but four of the cases it was possible
to measure cardiac output and right heart pressures
by the cardiac catheterization technic shortly before
or soon after the pulmonary function studies. Data
on 10 additional patients, similar in every way to
those in group I but studied only during cardiac
catheterization, are also included in graphic form
only for the purpose of illustrating the relationship
of pulmonary ventilation to the cardiac index and
mean pulmonary arterial pressure during a steady
state of exercise.

Cardiac catheterization was always performed in
the morning hours with the patient in a postab-
sorptive state and, in general, without premedication.
Venous cutdown, insertion of the catheter and
simultaneous collection of samples of blood and
expired air were performed in the usual manner for
the determination of cardiac output by the direct
Fick method. Expired air was measured in a Tissot
gasometer and analyzed for oxygen and carbon
dioxide in a Scholander micro gas analyzer.\textsuperscript{19} Samples
of mixed venous blood were withdrawn anaerobically
from the right or left pulmonary artery just beyond
the bifurcation, except in one instance (table 2) when
right auricular blood was obtained. Arterial blood
was obtained from an indwelling needle seated
in a peripheral artery. The oxygen contents of both
bloods were determined immediately after with-
drawal by the manometric method of Van Slyke
and Neill.\textsuperscript{20} Intracardiac and intravascular pressures
were recorded photographically via resistance wire
pressure transducers and ballistic galvanometers.\textsuperscript{21}
Systolic and diastolic pressures reported here represent
arithmetic mean values over at least two
respiratory cycles and mean pressures were calcu-
lated over the same time period after planimetric
integration of the areas beneath the curves.

Observations at rest were made while the patients
were lying in a comfortable position on a fluoro-
scopic table. Exercise was performed in the same
position, the patient moving weighted pedals with
his feet. In an attempt to achieve as steady a meta-
bolic state during exercise as was present at rest the
patients were required to perform the exercise for
8 or 10 minutes as a rule, and collection of samples
of blood and expired air for measurement of cardiac
output was delayed until exercise had been under-
way for five minutes or more.\textsuperscript{*} The pressure values
for exercise which are reported were recorded just
prior to or just after the cardiac output determina-
tion during exercise.

RESULTS AND COMMENTS

Since it was possible in these studies to ob-
tain more information about the heart and
circulation than had been available in the
majority of previous studies, an attempt has
been made to correlate any deviations from
normal in pulmonary function with abnor-
malities in cardiocirculatory dynamics. In many
instances deviations from normal in the latter
sphere were quite severe in that marked reduc-
tions in cardiac output and elevations in pul-
monary arterial pressure were observed fre-
cently (table 2).

1. Lung Volumes. There were no striking
deviations from normal in the conventional
lung volumes of the patients in group I.
While the vital capacity in three patients
(M. D., N. W. and L. A.) was found to be less
than 85 per cent of that predicted, the average
for the entire group was 99 per cent and the
lowest value noted was 80 per cent. Residual
volume and total lung capacity were also rela-
tively unimpaired, the latter differing signifi-
cantly from the predicted values in only 1 of
the 18 patients (N. W., a poorly developed
youth who had led a markedly restricted life
since early childhood). The mean ratio of
residual volume to total lung capacity in this
group was 23 per cent and the maximum value
observed was 34 per cent. In contrast, each of
the three patients in group II had a significant
reduction in vital capacity, an increase in
residual volume, and an abnormally high ratio
of residual volume to total lung capacity.

In view of some previous work on the lung
volumes in heart disease\textsuperscript{3, 5, 11} the normal findings
in this group of patients is noteworthy since it
has become rather widely accepted that
alterations in the vital capacity, residual
volume and total lung capacity may result
from engorgement of pulmonary blood vessels

\textsuperscript{*} Three exceptions should be noted: in the case of
patients H. C., A. A., and A. B. cardiac output was
measured during the fifth minute of the exercise
period.
whether or not there is associated edema of the lungs or of the abdominal viscera.\textsuperscript{22} The findings in these patients, who had no clinical evidence of fluid retention, would suggest that reported previously.\textsuperscript{22} The finding, on spirometry, in several instances of a slight degree of expiratory prolongation at the extreme expiratory position in the patients of group I

\textbf{Table 2.—Physiologic Findings Relative to Pulmonary and Cardiocirculatory Function in 21 Patients with Rheumatic Heart Disease, Inactive}

<table>
<thead>
<tr>
<th>Patient</th>
<th>VC % of Pred.</th>
<th>TLC % of Pred.</th>
<th>RV/TC X 100</th>
<th>MBC % of Pred.</th>
<th>Oxygen Cons. cc/min./M.2 BSA</th>
<th>Arterial Oxygen Saturation</th>
<th>Arterial pCO2</th>
<th>Index of Intrapulmonary Mixing % Nt</th>
<th>Oxygen Cons. cc/min./M.2 BSA</th>
<th>Cardiac Index L./min./M.2 BSA</th>
<th>Pulmonary Arterial Pressure mm. Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. D.</td>
<td>120</td>
<td>109</td>
<td>120</td>
<td>107</td>
<td>430</td>
<td>97</td>
<td>98</td>
<td>41</td>
<td>43</td>
<td>2.5</td>
<td>—</td>
</tr>
<tr>
<td>J. S.</td>
<td>110</td>
<td>104</td>
<td>19</td>
<td>77</td>
<td>123</td>
<td>383</td>
<td>94</td>
<td>97</td>
<td>45</td>
<td>45</td>
<td>1.1</td>
</tr>
<tr>
<td>J. R.</td>
<td>109</td>
<td>97</td>
<td>14</td>
<td>98</td>
<td>118</td>
<td>423</td>
<td>96</td>
<td>91</td>
<td>37</td>
<td>38</td>
<td>1.2</td>
</tr>
<tr>
<td>M. R.</td>
<td>90</td>
<td>98</td>
<td>27</td>
<td>95</td>
<td>110</td>
<td>476</td>
<td>95</td>
<td>98</td>
<td>38</td>
<td>37</td>
<td>1.0</td>
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<tr>
<td>M. D.</td>
<td>84</td>
<td>102</td>
<td>34</td>
<td>82</td>
<td>132</td>
<td>303</td>
<td>92</td>
<td>89</td>
<td>34</td>
<td>33</td>
<td>1.5</td>
</tr>
<tr>
<td>H. R.</td>
<td>90</td>
<td>—</td>
<td>—</td>
<td>93</td>
<td>145</td>
<td>317</td>
<td>93</td>
<td>98</td>
<td>31</td>
<td>22</td>
<td>—</td>
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<tr>
<td>N. W.</td>
<td>80</td>
<td>75</td>
<td>15</td>
<td>82</td>
<td>140</td>
<td>427</td>
<td>98</td>
<td>97</td>
<td>45</td>
<td>47</td>
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</tr>
<tr>
<td>E. S.</td>
<td>88</td>
<td>92</td>
<td>27</td>
<td>75</td>
<td>150</td>
<td>438</td>
<td>95</td>
<td>93</td>
<td>40</td>
<td>39</td>
<td>1.3</td>
</tr>
<tr>
<td>E. C.</td>
<td>113</td>
<td>120</td>
<td>25</td>
<td>77</td>
<td>117</td>
<td>310</td>
<td>98</td>
<td>100</td>
<td>54</td>
<td>33</td>
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</tr>
<tr>
<td>H. C.</td>
<td>91</td>
<td>94</td>
<td>26</td>
<td>82</td>
<td>107</td>
<td>347</td>
<td>100</td>
<td>95</td>
<td>32</td>
<td>2.1</td>
<td>2.1</td>
</tr>
<tr>
<td>V. H.</td>
<td>94</td>
<td>104</td>
<td>27</td>
<td>90</td>
<td>121</td>
<td>289</td>
<td>98</td>
<td>95</td>
<td>37</td>
<td>35</td>
<td>1.2</td>
</tr>
<tr>
<td>A. A.</td>
<td>88</td>
<td>88</td>
<td>23</td>
<td>105</td>
<td>113</td>
<td>290*</td>
<td>97</td>
<td>—</td>
<td>33</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>L. A.</td>
<td>82</td>
<td>97</td>
<td>34</td>
<td>87</td>
<td>139</td>
<td>372</td>
<td>95</td>
<td>99</td>
<td>40</td>
<td>40</td>
<td>2.4</td>
</tr>
<tr>
<td>R. D.</td>
<td>103</td>
<td>100</td>
<td>27</td>
<td>60</td>
<td>143</td>
<td>288*</td>
<td>96</td>
<td>92</td>
<td>36</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>A. B.</td>
<td>108</td>
<td>108</td>
<td>14</td>
<td>81</td>
<td>120</td>
<td>517</td>
<td>98</td>
<td>—</td>
<td>36</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>J. M.</td>
<td>87</td>
<td>93</td>
<td>25</td>
<td>100</td>
<td>145</td>
<td>574</td>
<td>98</td>
<td>92</td>
<td>37</td>
<td>42</td>
<td>1.2</td>
</tr>
<tr>
<td>G. M.</td>
<td>101</td>
<td>110</td>
<td>26</td>
<td>71</td>
<td>129</td>
<td>—</td>
<td>96</td>
<td>—</td>
<td>37</td>
<td>1.6</td>
<td>—</td>
</tr>
</tbody>
</table>

* Patient performed slightly substandard exercise.
† Mixed venous blood sample withdrawn from right auricle.
‡ Thirty step test, duration: one minute.
§ Steady state exercise.
¶ Cardiac output determined during fifth minute of exercise.

Abbreviations: VC = vital capacity; TLC = total lung capacity; % of Pred. = per cent of predicted; RV/TC = residual volume/total lung capacity; Oxygen cons. = oxygen consumed; cc./min./M.2 BSA = cubic centimeters per minute per square meter of body surface area; Ex. = exercise; Rec. = during the first minute of recovery after exercise; % Nt = per cent of nitrogen in alveolar air after seven minutes breathing 100 per cent oxygen; L./min./M.2 BSA = liters per minute per square meter of body surface area; s/d, m = systolic/diastolic, mean.

blood vessel engorgement alone, with or without cardiac enlargement, is not likely to result in a significant change in vital capacity or residual volume. These observations confirm similar findings in one other group of patients is perhaps pertinent to this matter. Observed in the absence of other stigmata of obstructive breathing, this may represent the early effect of a change in resilience of the lung (a decrease in compliance) secondary to engorgement of
the vascular bed, not sufficient to affect lung volume measurements in these patients but indicative of the effect more advanced circulatory abnormalities (pulmonary edema) might have.

2. Maximum Breathing Capacity. The maximum breathing capacities of 15 of the 18 patients in group I were less than the values predicted, but the average deviation from normal was small, the mean value for the group being 86 per cent of that predicted (table 2). The maximum breathing capacities of the three patients in group II were, in contrast, much lower than normal (table 2). There was no correlation in either group between maximum breathing capacity and resting cardiac index or mean pulmonary arterial pressure at rest. There was, also, no correlation between maximum breathing capacity and vital capacity in the patients in group I. The only instances of seriously reduced maximum breathing capacity among the patients in group I were noted in two cases (R. D. and G. M.) who were greatly debilitated as a result of prolonged hospitalization and bed rest. It would seem, therefore, that factors relating to general physical fitness are more important determinants of maximum breathing capacity in these patients with rheumatic heart disease than are specific circulatory changes. Recent work of another sort in this laboratory bears this out. Preliminary studies on a number of patients with mitral stenosis as well as normal individuals have shown that maximum breathing capacity determined during steady treadmill exercise is not different from that determined at rest. Were pulmonary vascular pressures, per se, an important determinant of maximum breathing capacity, physical exertion itself would tend to reduce this in most patients with mitral stenosis since an increase in pulmonary arterial pressure, with exercise, and undoubtedly pulmonary venous pressure as well, is the rule with this disorder.24

3. Spirograms. The findings with respect to lung volumes and maximum breathing capacity were, in general, reflected in the spiographic records. Normal spiograms were seen in the 18 cases of group I, except for the previously mentioned slight prolongation of expiration noted at the extreme expiratory position in a few instances. The three patients, on the other hand, whose clinical findings indicated intrinsic pulmonary disease were found to have spirometry that were quite abnormal: all demonstrated considerable expiratory prolongation and two (H. N. and F. L.) performed maximal voluntary ventilation in the high

Table 3.—Physiologic Findings Relative to Pulmonary Ventilation during Routine Pulmonary Function Study in 21 Patients with Rheumatic Heart Disease, Inactive

<table>
<thead>
<tr>
<th>Patient</th>
<th>Pulmonary ventilation, L./min./M² BSA, BTPS</th>
<th>Oxygen consumption, cc. per liter of ventilation, STPD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Exercise</td>
</tr>
<tr>
<td>Group I</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A. D.</td>
<td>3.7</td>
<td>13.1</td>
</tr>
<tr>
<td>J. S.</td>
<td>3.8</td>
<td>8.7</td>
</tr>
<tr>
<td>J. R.</td>
<td>5.2</td>
<td>11.6</td>
</tr>
<tr>
<td>M. R.</td>
<td>3.7</td>
<td>15.0</td>
</tr>
<tr>
<td>M. D.</td>
<td>6.8</td>
<td>14.6</td>
</tr>
<tr>
<td>H. R.</td>
<td>4.3</td>
<td>17.1</td>
</tr>
<tr>
<td>G. K.</td>
<td>3.6</td>
<td>12.6</td>
</tr>
<tr>
<td>N. W.</td>
<td>4.1</td>
<td>11.8</td>
</tr>
<tr>
<td>E. S.</td>
<td>5.0</td>
<td>14.6</td>
</tr>
<tr>
<td>E. C.</td>
<td>4.7</td>
<td>14.0</td>
</tr>
<tr>
<td>H. C.</td>
<td>—</td>
<td>10.6</td>
</tr>
<tr>
<td>V. H.</td>
<td>3.6</td>
<td>11.7</td>
</tr>
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<td>A. A.</td>
<td>3.7</td>
<td>13.1</td>
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<td>L. A.</td>
<td>4.4</td>
<td>11.7</td>
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<tr>
<td>R. D.</td>
<td>4.6</td>
<td>9.2</td>
</tr>
<tr>
<td>A. B.</td>
<td>4.1</td>
<td>11.0</td>
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<tr>
<td>J. M.</td>
<td>4.3</td>
<td>12.3</td>
</tr>
<tr>
<td>G. M.</td>
<td>4.6</td>
<td>—</td>
</tr>
</tbody>
</table>

Group II

<table>
<thead>
<tr>
<th>Patient</th>
<th>Pulmonary ventilation, L./min./M² BSA, BTPS</th>
<th>Oxygen consumption, cc. per liter of ventilation, STPD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Rest</td>
<td>Exercise</td>
</tr>
<tr>
<td>H. N.</td>
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<td>9.9</td>
</tr>
<tr>
<td>H. H.</td>
<td>3.9</td>
<td>15.0</td>
</tr>
<tr>
<td>F. L.</td>
<td>5.9</td>
<td>13.8</td>
</tr>
</tbody>
</table>

Abbreviations: L./min./M² BSA, BTPS = liters per minute per square meter of body surface area at body temperature and pressure, saturated with water; STPD = dry, 760 mm. Hg pressure, 0 centigrade.

inspiratory position with a small tidal volume in a manner characteristic of pulmonary emphysema.

4. Pulmonary Ventilation. The mean minute volume was slightly greater than normal at rest, during exercise, and in the recovery period in the patients comprising group I, (table 3, fig. 1). This hyperpnea was probably
true physiologic hyperventilation and not merely a manifestation of shallow breathing since arterial pCO₂* was not infrequently low after exercise as well as at rest. A reciprocal change in the oxygen consumption per liter of ventilation,† which was in keeping with this hyperpnea was also seen (table 3). Similarly, hyperventilation in relation to oxygen consumption was a feature of the performance of the group I patients and 10 other rheumatic subjects in a similar state of compensation during the mild steady exercise performed during cardiac catheterization, in which studies it was noted that the greatest degrees of rela-

tension within the lung tissue itself. It is also possible, in view of the relation between cardiac output and ventilation, that factors related to the transport of oxygen, carbon dioxide, or other metabolites may exert an influence upon ventilation via pathways not

![Graph](image)

**Fig. 1.** Mean ventilatory response to the 30-step test of 17 patients with rheumatic heart disease. Dashed line and open circles indicate the mean response predicted for this group of patients.

**Fig. 2A.** Relation of the oxygen removal rate (oxygen consumption per liter of ventilation) to the cardiac index during a steady exercise in a group of 19 patients with rheumatic heart disease. Twenty-two observations are included because three patients were studied before and after mitral commissurotomy.

**Fig. 2B.** Relation of the oxygen removal rate (oxygen consumption per liter of ventilation) to the mean pulmonary arterial pressure during a steady exercise in a group of 20 patients with rheumatic heart disease. Twenty-three observations are included because three patients were studied before and after mitral commissurotomy.

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* pCO₂ equals the partial pressure of carbon dioxide in arterial blood.

† Comparison of ventilation among patients is facilitated by using this means which makes allowance for the small variations in the severity of the exercise, and hence oxygen consumption, which occurred.

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* RHD - 17 Cases

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5. **Index of Intrapulmonary Mixing.** The index of intrapulmonary mixing was normal in all but 2 of the 21 patients (table 2). One of the two (A. B.) was a young individual who was completely asymptomatic, while the other (H. H.) was a patient with restrictive fibrosis involving the right lung in addition to advanced rheumatic heart disease. Since, however, the majority of these patients were found to hyperventilate to some extent at rest it is recognized that certain degrees of uneven alveolar ventilation might well have escaped detection by this test.

6. **Oxygen Consumption.** Oxygen consumption was normal at rest in every case in group I, but was distinctly less than normal during the minute of standard exercise of the pulmonary function test in 6 of the 17 patients who were exercised (table 2). The low oxygen intake in the case of two patients (A. A. and R. D.) may be simply a reflection of subnormal physical exertion since only 22 and 24 steps were taken respectively by these patients instead of the usual 30. There was, however, no feature which clearly distinguished the four others, (M. D., H. R., V. H. and E. C.) from those with normal oxygen intakes except for the fact that each was found at another time to have a very low resting cardiac output, lower in each case than that found in any of the patients with normal oxygen consumptions during the standard exercise. The significance of this finding lies in the illustration it affords of the restriction which may be imposed by chronic rheumatic heart disease upon oxygen consumption during activity, in connection with which it should be noted that the level of resting cardiac output (or, rather, the resting arteriovenous difference) determines the extent to which oxygen consumption can increase with exercise just as does the ability to increase the cardiac output over the resting level.

Oxygen consumption was normal at rest in each of the three patients in group II, but it was definitely lower than normal during the period of standard exercise (table 2). In each case, however, there was inadequate physical exertion during the test.

7. **Arterial Blood Gases.** Arterial pCO₂ was normal or slightly low after exercise as well as at rest in every case in group I. Arterial oxygen saturation also was normal after exercise as well as at rest in the majority of these patients (table 2). A value of 92 per cent was noted at rest in one patient (M. D.), however, in whom the oxygen saturation after exercise was 89 per cent. Three other patients (J. R., R. D. and J. M.) were noted to have small drops in arterial oxygen saturation after exercise from values which were normal at rest.

The cause of this reduction in oxygen saturation with exercise is not known. It may be due to impaired diffusion of oxygen across the alveolar capillary membrane,56 or simply a manifestation of normal physiologic shunts26 in normal or moderately increased amount but with a low mixed venous oxygen content. In contrast to the findings of others,56 however, there was no correlation between the level of arterial oxygenation and the degree of clinical disability found (tables 1 and 2). No effect, furthermore, of anoxia upon the degree of hyperventilation was noted.

One patient in group II (H. N.) was found to have appreciable arterial anoxia at rest and after exercise without evidence of carbon dioxide retention. The two others were normal in this respect (table 2).

**Discussion**

Among the many questions raised by these studies those pertaining to the origin of exertional dyspnea in the ambulatory rheumatic cardiac patient are of major interest since this is the foremost clinical manifestation of the disease.

It is apparent that the pulmonary functional pattern (as defined by these tests) of the 18 patients with uncomplicated heart disease differed greatly from that observed in the case of the patients with associated intrinsic pulmonary disease. Altered lung volumes, abnormal spiographic records, and reduced maximum breathing capacity in the latter group pointed out the presence of overdistention of the lungs and definite impairment of chest bellows action, functional patterns indistinguishable from those found in similar cases of chronic pulmonary disease without heart dis-
ease. On the other hand, there was nothing in the performance of the patients without intrinsic pulmonary disease to suggest the existence of abnormalities in pulmonary function which would be likely to result in exertional dyspnea. Although hyperventilation was noted at all stages of activity in these patients it was entirely too slight* in degree, even during exercise, to result in manifest ventilatory insufficiency. Factors, therefore, other than those relating to impaired ventilatory function must be concerned with the development of exertional dyspnea in these cardiac patients; and the major circulatory changes in chronic rheumatic heart disease, that is, reduction in cardiac output and elevation of pulmonary vascular pressures, should be examined in this light.

A restricted ability to transport oxygen to and to remove metabolites from the skeletal muscles secondary to a low and relatively fixed cardiac output can be considered an important factor in limiting the work tolerance of patients with rheumatic heart disease for obvious reasons. Easy fatigability, weakness, perhaps weight loss, seem likely to be related to this chronic reduction in blood flow; and in most patients the first two of these disturbances contribute greatly to the pattern of physical disability exhibited. Similarly, a limited cardiac output might conceivably result in labored breathing during exertion as a result of undue exhaustion of the respiratory muscles themselves despite the fact that in most circumstances the general manifestations of the reduction in blood flow could be expected to bring physical activity to a halt before such local influences become manifest. In the face, however, of possibly increased effort in breathing in the cardiac patient the “reserve” of the respiratory muscles might be impaired sufficiently to result in dyspnea at relatively low levels of minute volume. Studies on the work of breathing under various conditions will doubtless aid in solving this problem.

The importance of increased pulmonary vascular pressures in the production of dyspnea is equally ill defined. Although it has been shown that pulmonary hypertension per se does not bring about a reduction in vital capacity or maximum breathing capacity to any great extent, changes in pulmonary physical properties secondary to high pressures in the pulmonary veins, capillaries or arteries might still be important in the development of dyspnea. A reduction in pulmonary compliance secondary to increased intravascular pressure, for example, would necessitate increased energy expenditure in moving air into and out of the lungs which could certainly result in increased effort in breathing, a possible effect of which has been mentioned previously. Another possibility is that alterations in pulmonary elasticity might result in changes in the velocity of air flow at various phases of the respiratory cycle which may give rise to a sense of urgency to breathing not experienced by the normal individual at comparable minute volumes. Finally, pulmonary venous and arterial hypertension might conceivably lead to some form of distress, unrelated to the actual movement of air into and out of the lungs, as a result of

---Fig. 3. Mean ventilatory response to the 30-step test of 25 patients with the syndrome of alveolar-capillary block. Dashed line and open circles indicate the mean response predicted for this group of patients.

* The relative mildness of the hyperpnea seen in these patients is well brought out by comparison with that seen in a group of patients with the syndrome of alveolar-capillary block, a disorder in which hyperventilation is often of sufficient magnitude to tax ventilatory capacity19. 20 (fig. 3).
stimuli arising in distended veins or arteries. The phenomenon of orthopnea suggests that some such mechanism may exist because discomfort noted in the supine but not in the upright position cannot be ascribed to ventilatory insufficiency as discussed herein despite the changes in lung volumes which occur with changes in posture.28

Thus the phenomenon of exertional dyspnea in these patients which seems not to be due to ventilatory insufficiency cannot be definitely attributed to any specific circulatory abnormality either, from such evidence as is now available. The relative importance of inadequate cardiac output and increased pulmonary vascular pressure in its development is not known, although there are various hypothetic mechanisms whereby each of these disturbances could result in dyspnea. Current studies of problems related to the mechanics of breathing will probably throw some light on the importance of effort or energy expenditure in breathing in this matter, a question which has been impossible to evaluate by means employed in the present studies, while examination of patients before and after successful mitral commissurotomy will perhaps aid in differentiating the influences of vascular congestion and inadequate cardiac output upon this wholly subjective yet clinically all important manifestation of heart disease.

CONCLUSIONS

1. Twenty-one patients with rheumatic heart disease and varying degrees of diminished cardiac reserve have been studied with respect to pulmonary and cardiocirculatory function. Eighteen of these were ambulatory individuals who presented no clinical signs of right ventricular failure or pulmonary edema despite the presence, in some instances, of advanced hemodynamic abnormalities secondary to heart disease. Three others had chronic pulmonary disease in addition to heart disease, but also, were not in congestive failure.

2. Although deviating slightly from normal in several respects the pattern of pulmonary function of the 18 patients without pulmonary disease or congestive failure does not account for the exertional dyspnea which these patients have. It suggests, rather, that dyspnea on exertion at the stage of cardiac insufficiency exemplified by this group of patients is a complex matter with multiple facets. Of these, weakness and easily induced fatigue of skeletal musculature including the muscles of respiration are possibly directly related to inadequacy of cardiac output while increased pulmonary vascular pressures may result in an increased effort in breathing as well as some form of subjective distress secondary to vascular distention per se.

3. In the absence of fluid retention, that is, of pulmonary edema, hydrothorax, or other evidences of this abnormality, or specific disease of the lungs, the presence of even severe degrees of pulmonary vascular engorgement may not result in appreciable alterations in the lung volumes, maximum breathing capacity and gross ventilatory response to mild exercise.

4. Intrinsic pulmonary disease, when it occurs concomitantly with compensated rheumatic heart disease, induces alterations in lung functions which may as a rule be readily differentiated from those due to heart disease alone.

SUMARIO ESPAÑOL

La función pulmonar y cardiocirculatoria se estudió en 21 pacientes ambulatorios con enfermedad reumática del corazón. Un patrón de función pulmonar se observó con desviación de un grado que es insuficiente para explicar la disnea esforzosa de estos pacientes. La patogénesis de este síntoma se discute en relación a las anormalidades cardiocirculatorias que a menudo fueron marcadas.

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