The Pulmonary Diffusing Capacity in Congenital and Rheumatic Heart Disease

By J. Howland Auchincloss, Jr., M.D., Robert Gilbert, M.D., and Robert H. Eich, M.D.

The pulmonary diffusing capacity was determined both in patients with intracardiac septal defects and in patients with valvular heart disease in an effort to find out whether pulmonary congestion and hyperemia as they occur in cardiac disease have different effects on the process of gas diffusion and whether these effects vary at different stages of the disease process.

THE pulmonary diffusing capacity (DL) is a measurement relating the flow rate of gas across the alveolo-capillary membrane to the pressure gradient. Estimation of DL is possible by a variety of technics, and in recent years it has resulted in a materially improved understanding of diffuse pulmonary parenchymal disease and of the effect of chronic airway obstruction on the pulmonary parenchyma. The effect of heart disease on DL has not received comparable attention; however, Carroll, Cohn, and Riley have demonstrated that DL at rest may or may not be reduced in disease of the mitral valve.

Classically, the magnitude of DL has been considered to depend upon the permeability of the diffusing surface and the amount of area available for diffusion. In addition, Roughton and Forster have recently shown that DL is also affected by the volume of blood in ventilated pulmonary capillaries. It would seem reasonable that both the surface area and capillary blood volume might be increased under conditions of passive congestion, as in mitral valve disease, and under conditions of increased pulmonary blood flow (pulmonary hyperemia) such as occurs in septal defects with left-to-right shunts. Also, it might be expected that either of these conditions could eventually affect the permeability of the membrane. Thus, alterations of the diffusing capacity in cardiac disease could be a result of the effects of the disease on any or all of the determinants of diffusing capacity and might give valuable information concerning both the circulatory and parenchymal state of the lungs.

MATERIALS AND METHODS

The clinical and hemodynamic data for the 29 patients included in the study are summarized in table 1. In 16 of these patients the diagnosis of interatrial or interventricular septal defect had been established by right heart catheterization. Atrial defects (IASD) with or without anomalous pulmonary veins existed in 13 patients; in 7 of these the mean resting pulmonary artery pressure was 22 mm. Hg or less. The remaining 6 patients with IASD exhibited evidence of pulmonary hypertension; in 4 cases this was established by direct measurement (mean pulmonary artery pressure 33 to 72 mm. Hg), but in 2 patients (nos. 15 and 16) the catheter failed to enter the pulmonary artery. Pulmonary hypertension was considered likely in 1 of these 2 patients (no. 15) because she had undergone multiple episodes of severe right heart failure. Pulmonary hypertension was considered to be present in patient no. 16 because the catheter entered the right ventricle, where a systolic pressure of 84 mm. Hg was recorded. In the absence of clinical evidence of pulmonic stenosis, this figure undoubtedly is close to the pulmonary artery systolic pressure, and therefore permits an estimation of pulmonary artery mean pressure (50 mm. Hg) for correlative purposes.

Three patients had interventricular septal defects (IVSD). Pulmonary artery pressure was normal in 1 and elevated in 2.

In the entire group (IASD and IVSD) shunt reversal at rest was clinically manifest in only 1

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PULMONARY DIFFUSING CAPACITY IN HEART DISEASE

Pulmonary hypertension absent or mild

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Diagnosis</th>
<th>Arterial O2 saturation (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>IVSD</td>
<td>94</td>
</tr>
<tr>
<td>2</td>
<td>Anomalous pulmonary venous drainage† IASD</td>
<td>93</td>
</tr>
<tr>
<td>3</td>
<td>IASD</td>
<td>96</td>
</tr>
<tr>
<td>4</td>
<td>IASD</td>
<td>97</td>
</tr>
<tr>
<td>5</td>
<td>IASD</td>
<td>91</td>
</tr>
<tr>
<td>6</td>
<td>IASD</td>
<td>95</td>
</tr>
<tr>
<td>7</td>
<td>IASD</td>
<td>97</td>
</tr>
<tr>
<td>8</td>
<td>IASD</td>
<td>99</td>
</tr>
</tbody>
</table>

Pulmonary hypertension moderate or severe

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Diagnosis</th>
<th>Arterial O2 saturation (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>IVSD</td>
<td>95</td>
</tr>
<tr>
<td>10</td>
<td>IASD, right-to-left shunt</td>
<td>91</td>
</tr>
<tr>
<td>11</td>
<td>IVSD, right-to-left shunt</td>
<td>79</td>
</tr>
<tr>
<td>12</td>
<td>IASD, ost. prim. and second.</td>
<td>97</td>
</tr>
<tr>
<td>13</td>
<td>IASD, ost. prim. and second.</td>
<td>92</td>
</tr>
<tr>
<td>14</td>
<td>IASD, tricuspid insufficiency</td>
<td>97</td>
</tr>
<tr>
<td>15</td>
<td>IASD, ost. prim.</td>
<td>94</td>
</tr>
<tr>
<td>16</td>
<td>IASD, ost. prim.</td>
<td>91</td>
</tr>
</tbody>
</table>

Rheumatic Heart Disease

Normal pulmonary artery pressure at rest, or mild symptoms

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Diagnosis</th>
<th>Arterial O2 saturation (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>17</td>
<td>MS</td>
<td>97</td>
</tr>
<tr>
<td>18</td>
<td>MS</td>
<td>97</td>
</tr>
<tr>
<td>19</td>
<td>MS</td>
<td>91</td>
</tr>
<tr>
<td>20</td>
<td>MS</td>
<td>91</td>
</tr>
<tr>
<td>21</td>
<td>MS</td>
<td>91</td>
</tr>
</tbody>
</table>

Pulmonary arterial or left atrial hypertension at rest, or moderate to severe symptoms

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Diagnosis</th>
<th>Arterial O2 saturation (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>22</td>
<td>MS, MI</td>
<td>90</td>
</tr>
<tr>
<td>23</td>
<td>MS, AI</td>
<td>95</td>
</tr>
<tr>
<td>24</td>
<td>MS</td>
<td>95</td>
</tr>
<tr>
<td>25</td>
<td>MI</td>
<td>93</td>
</tr>
<tr>
<td>26</td>
<td>MS, AS</td>
<td>93</td>
</tr>
<tr>
<td>27</td>
<td>MS, progressive dyspnea, hemoptysis</td>
<td>93</td>
</tr>
<tr>
<td>28</td>
<td>MS</td>
<td>93</td>
</tr>
<tr>
<td>29</td>
<td>MI</td>
<td>91</td>
</tr>
</tbody>
</table>

*Pulmonary artery mean pressure estimated from right ventricular systolic pressure.
†Left atrial pressure.

IVSD, interventricular septal defect; IASD, interatrial septal defect; MS, mitral stenosis; MI, mitral insufficiency; AI, aortic insufficiency; AS, aortic stenosis.
### Table 2.—Lung Volumes and Diffusing Capacity

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Sex</th>
<th>Ht. (cm.)</th>
<th>BSA (M²)</th>
<th>Residual volume/total lung capacity (L)</th>
<th>Mixing index ( % N₂)</th>
<th>Max. breathing capacity (L/min.)</th>
<th>DLCO pred. (ml./mm. Hg)</th>
<th>% pred.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Vital capacity (L), obs. % pred.</td>
<td></td>
<td>obs. % pred.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### Congenital Heart Disease

**Pulmonary hypertension, absent or mild**

| 1    | 19  | M   | 171   | 1.84 | 4800 | 99  | 21  | 0.67 | 165 | 117  |
| 2    | 20  | M   | 173   | 1.71 | 4820 | 77  | 26  | 0.95 | 137 | 105  |
| 3    | 31  | M   | 167   | 1.64 | 3250 | 72  | 25  | 0.84 | 144 | 125  |
| 4    | 35  | M   | 184   | 2.11 | 4120 | 81  | 30  | 0.70 | 158 | 112  |
| 5    | 45  | M   | 176   | 1.73 | 3390 | 77  | 37  | 0.90 | 134 | 123  |
| 6    | 49  | M   | 173   | 1.91 | 4180 | 101 | 30  | 0.97 | 128 | 110  |
| 7    | 17  | F   | 163   | 1.58 | 2830 | 85  | 28  | 1.23 | 75  | 75   |
| 8    | 32  | F   | 162   | 1.58 | 2690 | 84  | 33  | 1.03 | 79  | 91   |

#### Pulmonary hypertension, moderate or severe

| 9    | 16  | M   | 173   | 1.90 | 4830 | 96  | 17  | 0.54 | 199 | 134  |
| 10   | 20  | M   | 175   | 1.83 | 4730 | 90  | 31  | 0.46 | 193 | 139  |
| 11   | 30  | M   | 175   | 1.98 | 4400 | 90  | 24  | 0.44 | 161 | 115  |
| 12   | 39  | M   | 171   | 1.72 | 3650 | 83  | 37  | 0.67 | 138 | 121  |
| 13   | 19  | F   | 152   | 1.35 | 1350 | 49  | 54  | 1.03 | 41  | 48   |
| 14   | 29  | F   | 166   | 1.52 | 2210 | 64  | 42  | 0.73 | 73  | 83   |
| 15   | 38  | F   | 158   | 1.80 | 1930 | 66  | 46  | 0.90 | 41  | 43   |
| 16   | 47  | F   | 155   | 1.32 | 2060 | 79  | 40  | 2.16 | 54  | 83   |

**Average** 30 — 168 1.72 3453 81 33 0.90 120 102 34 27 126

**Range** 16-49 — 152-1.32-1350-49-17-0.44-41-43-11-20-50-

#### Rheumatic Heart Disease

**Normal pulmonary artery pressure at rest or mild symptoms**

| 17   | 23  | M   | 155   | 1.58 | 3710 | 88  | 25  | 0.69 | 54  | 46   |
| 18   | 29  | F   | 162   | 1.49 | 3420 | 105 | 27  | 0.64 | 82  | 96   |
| 19   | 31  | F   | 155   | 1.52 | 3260 | 111 | 27  | 1.47 | 117 | 136  |
| 20   | 35  | F   | 163   | 1.63 | 3290 | 104 | 23  | 1.24 | 106 | 118  |
| 21   | 38  | F   | 155   | 1.48 | 3410 | 122 | 30  | 0.57 | 90  | 114  |

**Pulmonary artery or left atrial hypertension at rest or moderate or severe symptoms**

| 22   | 31  | M   | 174   | 1.73 | 3510 | 73  | 32  | 0.83 | 130 | 107  |
| 23   | 33  | M   | 183   | 1.87 | 3980 | 77  | 30  | 1.59 | 221 | 170  |
| 24   | 46  | M   | 182   | 1.96 | 5560 | 120 | 26  | 2.28 | 154 | 125  |
| 25   | 47  | M   | 161   | 1.76 | 3970 | 109 | 29  | 1.10 | 149 | 137  |
| 26   | 25  | F   | 162   | 1.59 | 1730 | 49  | 40  | 0.54 | 74  | 78   |
| 27   | 30  | F   | 159   | 1.47 | 2690 | 86  | 41  | 0.86 | 87  | 104  |
| 28   | 38  | F   | 167   | 1.58 | 2460 | 75  | 41  | 1.08 | 106 | 126  |
| 29   | 50  | F   | 165   | 1.61 | 1920 | 65  | 41  | 0.97 | 59  | 78   |

**Average** 35 — 165 1.64 3301 91 32 1.07 110 110 22 25 88

**Range** 23-50 — 155-1.47-1730-49-23-0.54-54-46-14-22-54-

183-1.96-5560-122-41-2.28-221-170-28-32-127
sample and was therefore always in excess of 10 seconds. The valve that switched the patient into the circuit for collection of the alveolar sample was turned when the operator sensed that expiration had begun, and the short time delay necessary for turning the valve allowed about 700 to 2,000 ml. of dead space and alveolar air to escape. The alveolar sample, collected in a 6 liter anesthesia bag, was transferred to the gas analyzers under positive pressure. Thermal conductivity (helium) and infra-red (carbon monoxide) meters were connected in series, and all analyses were performed after the removal of water vapor. The helium analyses were performed both before and after the removal of carbon dioxide by absorption, and thus the percentage of carbon dioxide could be estimated with an accuracy of 1 per cent. Appropriate corrections were then applied to both the helium and carbon monoxide analyses, since both meters were to some extent sensitive to carbon dioxide in the amount present in alveolar air. Great care was taken with both meters to check the deflection to known gas mixtures with each analysis. The stability of calibration was checked every few months and varied only within narrow limits during the 1 year period in which both initial and follow-up studies were performed.

The test gas consisted of 12.05-12.70 per cent helium, 0.308-0.492 per cent carbon monoxide, 20 per cent oxygen and the remainder nitrogen. The variation in inspired carbon monoxide concentration was greater than desired; however, this appeared to have no effect on DLCO as judged by determinations in a normal subject, where both the higher and lower concentrations were used at the same sitting.

In preliminary experiments, determination of the resting diffusion capacity by the steady-state method of Filley, MacIntosh and Wright was accomplished in 5 of the cases of IASD, 2 cases of IVSD, and 1 case of rheumatic heart disease. In our hands this techinc gave erratic results in resting subjects. This was thought to be caused by difficulty in obtaining the extreme accuracy of PaCO2 determination necessary for the estimation of the mean diffusion gradient. Those data that were obtained suggested that the PaCO2 determination is even more critical where the diffusion capacity at rest is increased, inasmuch as part of the effect of an augmented DL in a resting subject may be to reduce the mean value of the diffusion gradient. As a result of these practical and theoretical considerations the method was abandoned in the present study, and the data are not reported.

In 15 patients the interval between DLCO determinations and catheterization studies was less than 6 months. In 3 patients an interval of more than 2 years separated the 2 studies. However, clinical findings failed in any case to suggest a degree of change that would have invalidated correlation of the data from the different procedures.

Maximum ventilatory capacity was determined with a modified 9 liter spirometer. Predicted values for the maximum ventilatory capacity were derived from the formula of Baldwin et al. Predicted values for lung volumes in seated, nonfasting subjects were derived from the data of Needham et al., a factor of 1.06 being used to raise the figures of ambient temperature and pressure saturated with water vapor to those of body temperature.

**RESULTS**

In the 19 normal subjects (table 3) a significant correlation was found between DLCO and body height (DLCO = 37.4 × body height (M.) − 36.5; S.D. = ± 3.25 ml./min./mm. Hg; r = 0.71). For all normal subjects and for patients a predicted value was computed from this formula, from which the percentage ratio, DLCO observed/DLCO predicted × 100, was derived. The mean of these values for the normal subjects was 100 per cent with a standard deviation of ± 12.5 per cent and range of 85 to 127 per cent. Values differing by more than 6.5 ml./min./mm. Hg from predicted are considered to be elevated or reduced, inasmuch as they fall outside of the 95 per cent confidence limits established by the normal series.

By these criteria, 10 of the 16 patients with congenital heart disease had elevated values of DLCO and 1 patient had a reduced value (table 2). In 5 patients the test was normal.
In the group with rheumatic heart disease, the values were normal in 9 patients and reduced in 4 (fig. 1).

Because both patient groups contained individuals with disease processes of greatly differing degrees of severity, 3 graphs are presented in which DLco as percentage of predicted value is plotted against various parameters (figs. 2-4). Thus, if allowance is made for differences in clinical severity (fig. 2), in the vital capacity measurement (fig. 3), and in the level of pulmonary artery pressure (fig. 4), patients with intracardiac septal defects tend to have higher values for diffusing capacity than patients with rheumatic heart disease.

Figure 5 relates the resting pulmonary blood flow to DLco. Resting pulmonary blood flow was normal or reduced in all the rheumatic subjects and was elevated in all but 4 of the 16 congenital patients. No instances of an elevated DLco in the presence of a reduced value for pulmonary blood flow were encountered, and, conversely, no reduced values for DLco were found in patients with elevated pulmonary blood flows.

The results of serial determinations of DLco are presented in figures 6 and 7. In the normal subjects followed over a 1 year period, the largest change between any 2 values expressed as a percentage of the greater value was 21 per cent. This could well be a summation of technical errors, inasmuch as variations of a similar magnitude would occasionally occur at a single sitting. The average variation at a single sitting, however, was 7 per cent.

Serial determinations were also performed in 4 patients with congenital heart disease and in 4 patients with rheumatic valvular heart disease. The normal degree of variation was exceeded in only 1 case (R.A., case 5), in whom a fall of 27 per cent from the original value of DLco occurred prior to corrective surgery, with a subsequent further fall postoperatively. In figure 7 this is expressed as
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FIG. 2 Left. Single-breath diffusing capacity in congenital and rheumatic heart disease with patients grouped according to clinical severity. Single-breath diffusing capacity is expressed as a percentage of the predicted value.

FIG. 3 Right. Relationship between single-breath diffusing capacity and vital capacity. Both vital capacity and DLco are expressed as percentage of predicted. Values on all 19 normal subjects fell within the trapezoid. Diagonal line is line of identity.

A fall from 179 per cent of the predicted figure to a final value of 107 per cent of predicted. This patient had an ostium secundum type of IASD with pulmonary artery pressure of 35/12 and a pulmonary blood flow of 19 L./min./M.² BSA at the time of the original examination. During the ensuing 9 months he noted increased fatigability and was found at a second catheterization to have an unchanged value of pulmonary artery pressure; the pulmonary blood flow was found to be 11 L./min./M.² BSA, but this change is of questionable significance. Satisfactory closure of the defect was accomplished, and the final measurement of DLco was made when he was in a symptomatically improved state.

In another patient with IASD (A.A. case 8) a significantly elevated DLco fell to normal postoperatively, and in 1 case of mitral stenosis (R.L., case 27) a rise of 17 per cent occurred after a successful valvuloplasty. In 3 other cases of mitral stenosis the values fell insignificantly or were unchanged. These 3 valvuloplasties were at least partially successful both from the technical standpoint and from the evaluation of symptoms in the early months of follow-up.

DISCUSSION

This study indicates that elevation of the resting diffusing capacity is probably a common occurrence in patients with intracardiac septal defects. Although this observation cannot be clearly correlated with any single associated finding in the other tests performed, the data suggest that the level of pulmonary blood flow is of major importance. This conclusion is based principally on the grouping of points in figure 5. The degree of scatter of these points is considerable, but such scatter is to be expected if the number of causes of biologic variation and technical error are considered. In particular, the estimation of pulmonary blood flow at rest by the Fick technic is inaccurate when the flow is markedly increased.

Recent work by Roughton and Forster strongly indicates that the value of DLco is

A GROUP

B GROUP

Symptoms:

none or mild

moderate or severe

Figure 2: Single-breath diffusing capacity in congenital and rheumatic heart disease with patients grouped according to clinical severity.

Figure 3: Relationship between single-breath diffusing capacity and vital capacity. Both vital capacity and DLco are expressed as percentage of predicted. Values on all 19 normal subjects fell within the trapezoid. Diagonal line is line of identity.
Whatever the change in the pulmonary capillary circulation may be in patients with uncomplicated shunts, the conditions are likely to be similar to those present in vigorous exercise in normal subjects. Under both circumstances large pulmonary blood flows are tolerated with no, or minimal, rises in pulmonary artery pressure. If it be assumed that in all of our patients with septal defect this uncomplicated condition existed at one time, it can be concluded that the finding of a normal diffusing capacity in the presence of pulmonary hypertension (fig. 4) represents a fall from a previously elevated value. Our data do not determine whether this fall in Dlco is primarily related to the decrease in pulmonary blood flow or to damage to the alveolo-capillary membrane related to the process of vascular obstruction. Edwards\textsuperscript{13} has described cellular fibrous intimal thickening in small pulmonary arteries and arterioles in pathologic material from patients with IASD and moderate pulmonary hypertension. These lesions may be completely occlusive but are focal; because of this fact it seems likely that areas of lung are functionally removed from the process of gas exchange, while in other areas the capillary bed is fully expanded. With severe pulmonary hypertension the occlusive lesions were present in vessels as wide as 300 microns. Presumably, once initiated, this process of focal vascular occlusion progresses, and its severity must therefore be related to its duration. Such considerations render more plausible some of the individual values of Dlco obtained. For example, both cases 10 and 16 had marked pulmonary hypertension with normal values for pulmonary blood flow. In case 10, a 20 year old male, Dlco was elevated, while in case 16, a 47 year old woman, it was markedly reduced.

In this study, Dlco in mitral valve disease was normal or reduced; similar results for Dlco were obtained by Carroll, Cohn, and Riley.\textsuperscript{5} It is thus obvious that active hyperemia, as exemplified by IASD, and passive congestion caused by valvular heart disease do not have identical effects on the pulmonary

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**FIG. 4 Top.** Relationship between single-breath diffusing capacity and mean pulmonary artery pressure.

**FIG. 5 Bottom.** Relationship between single-breath diffusing capacity and pulmonary blood flow. Diffusing capacity was elevated only in the presence of increased pulmonary blood flow, and was never below normal when pulmonary blood flow was high.

affected not only by the resistance offered by the pulmonary membrane to gas diffusion but also by resistance encountered by the CO molecule in its passage across the red-cell membrane and into chemical union with hemoglobin. This latter resistance is reduced as the capillary blood volume increases. It is impossible from the present data to demonstrate that the increased value for Dlco represents only a functional expansion of the alveolo-capillary membrane as opposed to an increase in capillary blood volume. In fact, under conditions of increased pulmonary blood flow it seems unlikely that either type of change could occur without the other.
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Figure 6: Top. Variations in single-breath diffusing capacity in normal subjects studied over 1 year.

Figure 7: Bottom. Variations in single-breath diffusing capacity in patients with congenital and rheumatic heart disease studied over 1 year. Time of corrective cardiac surgery is indicated by the vertical arrows.

capillary bed. It may be that while both conditions produce capillary dilatation, associated effects of pulmonary congestion cause a reduction in diffusing capacity. These associated effects in advanced cases have been well described by Parker and Weiss14 and are not limited to the pulmonary capillary. Fibrosis of the interstitium, thickening of the alveolar membrane, and edema (both interstitial and intra-alveolar) collectively provide ample explanation of the reduction in diffusing capacity that occurs in advanced cases. This knowledge, plus the failure of DLCO to rise in 3 of 4 cases studied after valvuloplasty, suggest that a reduced DLCO in mitral valve disease may be an indication of permanent damage to the lung parenchyma, rather than simply the result of congestion or diminished pulmonary blood flow.

It is not proposed that a single determination of DLCO materially improves the evaluation of the individual patient with intracardiac septal defect. It would, however, appear to have possible use when serially determined. If such determinations show a significant fall from an initially elevated value, it becomes difficult to escape the conclusion that progressive deterioration is occurring in the pulmonary circulation. The symptomatic and clinical evidences of these changes are by no means clear-cut, particularly in the early stages. There would appear, therefore, to be potential usefulness in a safe, reproducible procedure that correlates with events taking place in the pulmonary capillary bed.

SUMMARY

The pulmonary diffusing capacity was determined by the single-breath carbon monoxide technic in 16 patients with intracardiac septal defects, 13 patients with rheumatic valvular heart disease, and 19 normal subjects. Significantly elevated values were found in 10 patients with intracardiac septal defects, in 9 of whom the pulmonary blood flow was elevated. Normal or insignificantly elevated values usually occurred where intracardiac septal defects were accompanied by moderate or marked degrees of pulmonary hypertension. A significantly reduced value was obtained in 1 patient with interatrial septal defect complicated by marked pulmonary hypertension and in 4 of the 13 subjects with rheumatic valvular heart disease. In 2 subjects with interatrial septal defect the diffusing capacity was initially elevated but became normal following closure of the defect. The data
are consistent with the concepts that pulmonary blood flow and diffusing capacity are related quantities and that the pulmonary vascular complications in patients with intracardiac septal defects may also cause impaired diffusion by a more direct mechanism in some cases. In view of these considerations, serial determinations of diffusing capacity may have clinical value in prognosis and in determining the urgency of the need for corrective surgery.

ACKNOWLEDGMENT

We wish to express our thanks to Miss Elizabeth Coulter, Mrs. Mary Burdick, and Miss Eileen McAuliffe for their technical assistance.

SUMMARIO IN INTERLINGUA

Le capacitate pulmonar de diffusion esseva determinate per medio del technica a inspira-
tion unie de monoxido de carbon in 16 pa-
tientes con defectos de septo intracardiac, 13 pacientes con rheumatic morbo de valvula car-
diae, e 19 subjectos normal. Valores significativa-
tivamente elevate esseva trovate in 10 pa-
tientes con defectos de septo intracardiac, 
incluse 9 in qui le fluxo de sanguine pulmonar 
esseva elevate. Valores normal o insignifica-
tivamente elevate occurreva usualmente in 
casos in que defectos de septo intracardiac 
esseva accompaniata de grados moderate o 
marcate de hypertension pulmonar. Un 
significativalemente reducite valor esseva obtenite 
in 1 paciente con defecto de septo interatrial 
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The 1,346 persons working at the Brookhaven National Laboratory since 1953 were classified by history and physical examination with the following results. The average age was 34.6 years (±10.0) and five sixths were males. The 135 individuals who denied ever adding sodium chloride to their food had an incidence of hypertension (140 mm. Hg systolic, 90 mm. Hg diastolic or higher) of 0.7 per cent. In contrast, 10.5 per cent hypertensive individuals were found in the group of 581 individuals who routinely added salt to food before tasting it. The incidence of hypertension in the high salt group as compared with the low salt group was greater in non-overweight persons and much greater in overweight persons. The distinction by history between the low- and high-salt-intake groups was validated by the results of urine sodium analyses done in a number of cases. It was proposed that the level of sodium intake is the primary etiologic factor in the development of essential hypertension.

Rogers
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