The Pulmonary Diffusing Capacity in Congenital 
and Rheumatic Heart Disease

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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 mem-
brane. 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 hyper-
tension; 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 steno-
sis, this figure undoubtedly is close to the pulmonary artery systolic pressure, and therefore per-
mits 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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case (no. 11, IVSD), in whom arterial oxygen saturation was 79 per cent. However, values less than 93 per cent (the lower limit of normal in this laboratory) were found in 4 additional patients. In patient no. 16 arterial oxygen saturation fell from a resting value of 91 to 69 per cent following the standard exercise test, suggesting that reversal of shunt with exercise had occurred.

The 13 patients with rheumatic heart disease have been grouped in tables 1 and 2 according to the manometric or clinical estimation of pulmonary artery pressures. Five of these patients were considered by clinical examination to have normal pulmonary artery pressures; in the 2 instances in which doubt existed (nos. 18 and 20) catheter measurements were obtained. The remaining 8 cases were, with 1 exception, in an advanced or unquestionably progressive stage of the disease; case no. 24 presented very mild symptoms but definite resting pulmonary hypertension.

A control series of 19 normal subjects was selected on the basis of good general health and absence of known cardiac or pulmonary disease. Four of the group were hospitalized patients and 15 were doctors, technicians, or hospital personnel.

The standard technics used in physiologic diagnosis and evaluation of the cardiac patients have been described in a previous report from this laboratory. In the present study oxygen saturation determinations in some instances were made with a Wood-type oximeter as an adjunctive device at cardiac catheterization. Shunt localization in case no. 10 was accomplished by injection of radioactive iodinated serum albumin through the catheter at different sites in the right heart. Cardiac output in some of the subjects with rheumatic heart disease was determined by the dye-dilution technic, which in this laboratory correlated satisfactorily with the Fick method.

The resting diffusing capacity for carbon monoxide (DLCO) by the single-breath technic was carried out as described by Ogilvie et al. Subjects and patients came to the laboratory in the postprandial state. Estimation of the total lung capacity and its subdivisions was performed by spirometry and the nitrogen washout technic with the subject seated. The disappearance rate of carbon monoxide from the alveolar air was then measured during a breath-holding period of 10 to 13 seconds. The subject performed as complete an expiration as possible and then was switched into an atmospheric circuit previously washed out with the test gas. Inhalation was as rapid as possible and as complete as could be achieved without a maximum effort. The command for a complete expiration was given 10 seconds after inspiration was completed, but the duration of breath holding was timed by the graphic tracing from the beginning of inspiration to the collection of the alveolar

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Diagnosis</th>
<th>Am. Heart failure</th>
<th>Pulmonary Venous Drainage</th>
<th>Pulmonary artery pressure (mm. Hg)</th>
<th>Pulmonary blood flow (L/min.)</th>
<th>Systemic blood flow (L/min.)</th>
<th>Arterial O2 saturation (%)</th>
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<td>2</td>
<td>Anomalous</td>
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<td>3.5</td>
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<td>IASD</td>
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<td>14.0</td>
<td>5.2</td>
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Rheumatic Heart Disease

Normal pulmonary artery pressure at rest, or mild symptoms

<table>
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<tr>
<th>Case No.</th>
<th>Diagnosis</th>
<th>Am. Heart failure</th>
<th>Pulmonary Venous Drainage</th>
<th>Pulmonary artery pressure (mm. Hg)</th>
<th>Pulmonary blood flow (L/min.)</th>
<th>Systemic blood flow (L/min.)</th>
<th>Arterial O2 saturation (%)</th>
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<tr>
<td>17</td>
<td>MS</td>
<td>14</td>
<td>2.5</td>
<td>2.5</td>
<td>97</td>
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<td></td>
</tr>
<tr>
<td>18</td>
<td>MS</td>
<td>16</td>
<td>4.0</td>
<td>4.0</td>
<td>91</td>
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Pulmonary arterial or left atrial hypertension at rest, or moderate to severe symptoms

<table>
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<th>Case No.</th>
<th>Diagnosis</th>
<th>Am. Heart failure</th>
<th>Pulmonary Venous Drainage</th>
<th>Pulmonary artery pressure (mm. Hg)</th>
<th>Pulmonary blood flow (L/min.)</th>
<th>Systemic blood flow (L/min.)</th>
<th>Arterial O2 saturation (%)</th>
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<tr>
<td>22</td>
<td>MS, MI</td>
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<td>3.0</td>
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<td>MS, AI</td>
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<td>2.8</td>
<td>2.8</td>
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<tr>
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<td>MI</td>
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<tr>
<td>25</td>
<td>MS, AS</td>
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<td>MS, AS</td>
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<tr>
<td>28</td>
<td>MI</td>
<td>36</td>
<td>1.9</td>
<td>1.9</td>
<td>91</td>
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*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 (M.²)</th>
<th>Vital capacity (L.)</th>
<th>Residual volume/total lung capacity %</th>
<th>Mixing capacity (L./min.)</th>
<th>Max. breathing capacity (L./min.)</th>
<th>DLco pred. (ml./min./mm. Hg)</th>
<th>Residual volume/Max. obs. pred. (%)</th>
<th>Residual volume/Max. obs. pred. (%)</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>obs. % pred.</td>
<td>%</td>
<td>obs. % pred.</td>
<td>Ht. BSA (L.) capacity index (L./min.)</td>
<td>% pred.</td>
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<tr>
<td>1</td>
<td>19</td>
<td>M</td>
<td>171</td>
<td>1.84</td>
<td>4800</td>
<td>99 21</td>
<td>0.67</td>
<td>165 117</td>
<td>40 28 143</td>
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<tr>
<td>2</td>
<td>20</td>
<td>M</td>
<td>173</td>
<td>1.71</td>
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<td>77 26</td>
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<td>51 28 182</td>
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<td>3</td>
<td>31</td>
<td>M</td>
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<td>3250</td>
<td>72 25</td>
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<td>33 26 127</td>
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<td>45</td>
<td>M</td>
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<td>134 123</td>
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<td>17</td>
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<td>2830</td>
<td>85 28</td>
<td>1.23</td>
<td>75 75</td>
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<td>8</td>
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<td>F</td>
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<td>84 33</td>
<td>1.03</td>
<td>79 91</td>
<td>32 24 133</td>
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</table>

#### Congenital Heart Disease

- Pulmonary hypertension, absent or mild
- Pulmonary hypertension, moderate or severe

#### Rheumatic Heart Disease

- Normal pulmonary artery pressure at rest or mild symptoms
- Pulmonary artery or left atrial hypertension at rest or moderate or severe symptoms


Average: 35 - 165 - 1.64 - 3301 - 91 - 32 - 1.07 - 110 - 110 - 22 - 25 - 88 - 23- - 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 $DL_{CO}$ 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 diffusing capacity by the steady-state method of Filley, MacIntosh and Wright\footnote{1} was accomplished in 5 of the cases of IASD, 2 cases of IVSD, and 1 case of rheumatic heart disease. In our hands this technic gave erratic results in resting subjects. This was thought to be caused by difficulty in obtaining the extreme accuracy of $P_{aco}$ determination necessary for the estimation of the mean diffusion gradient. Those data that were obtained suggested that the $P_{aco}$ 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 $DL_{CO}$ 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.\footnote{2} Predicted values for lung volumes in seated, nonfasting subjects were derived from the data of Needham et al.,\footnote{3} 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 $DL_{CO}$ and body height ($DL_{CO} = 37.4 \times$ 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, $DL_{CO}$ observed/$DL_{CO}$ 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 $DL_{CO}$ 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 seculundum 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
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 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. 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.
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 Weiss 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

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Summary in Interlingua

Le capacitate pulmonar de diffusion esseva determinate per medio del technica a inspiratio unie de monoxido de carbon in 16 patientes con defectos de septo intracardiac, 13 patientes con rheumatic morbo de valvula cardiae, e 19 subjectos normal. Valores significativamente elevate esseva trovate in 10 patientes con defectos de septo intracardiac, inclusive 9 in qui le fluxo de sanguine pulmonar esseva elevate. Valores normal o insignificativamente elevate occurreva usualmente in casos in que defectos de septo intracardiac esseva accompaniata de grados moderate o marcate de hypertension pulmonar. Un significativamente reducida valor esseva obtenite in 1 patiente con defecto de septo interatrial complicate per marcate grados de hypertension pulmonar e in 4 ex 13 subjectos con morbo rheumatic de valvula cardiae. In 2 subjectos con defectos de septo interatrial, le capacitate diffusori esseva initialmente elevate sed deveniva normal post que le defecto habeva esseite claudite. Le datos es compatibile con le conception que le fluxo de sanguine pulmonar e le capacitate diffusori es magnitudes interrelationate e que le complicationes pulmono-vascular in patientes con defectos de septo intracardiac es etiam capace, in certe casos, a causar un compromiso del diffusion per un mechanismo plus directe. Iste considerationes suggere que determinationes serial del capacitate diffusori es possibilmente de valor clinica in establir le prognose e in determinar le urgentia de chirurgia corrective in le caso individual.

References


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.