The Coronary Blood Flow in Pulmonary Emphysema and Cor Pulmonale

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The coronary blood flow was determined in 9 individuals with pulmonary emphysema alone and emphysema with cor pulmonale. Intracardiac pressures, cardiac output and pulmonary functions were also studied. In 5 of the 9 cases coronary blood flow was within the normal range. In one case with a high cardiac output the coronary blood flow was elevated in proportion. The presence of cor pulmonale did not appear to influence the results.

The coronary blood flow in a group of 9 patients with pulmonary emphysema and cor pulmonale was estimated by means of the nitrous oxide desaturation method described by Bing and co-workers. The cardiopulmonary functions of these patients were under study and whenever it was possible to intubate the coronary sinus the coronary blood flow was determined.

METHODS

The pulmonary function studies were performed according to accepted procedures. Coronary blood flow per 100 Gm. of heart muscle was estimated by bringing the patient into equilibrium with a low concentration of inhaled nitrous oxide and determining the integrated arteriovenous difference during desaturation. Since Gregg has shown that the coronary sinus blood originates mainly from the left ventricular muscle, this method measures chiefly the coronary blood flow per 100 Gm. of left ventricular tissue. The experimental work of Eckenhoff and Gregg show a fairly good correlation between the nitrous oxide method and other methods of measuring coronary blood flow in the experimental animal. Cardiac output was determined by the Fick procedure of collecting mixed venous blood from the pulmonary artery, arterial blood and expired air simultaneously. Analysis of the blood gases was carried out on the Van Slyke apparatus and the expired air was analyzed by the Scholander method.

RESULTS

Preliminary work in our laboratory showed that satisfactory nitrous oxide desaturation curves could be constructed in patients with pulmonary emphysema. The curves had a flattened contour with slower desaturation which leveled off a minute or two later than in patients without pulmonary disease (fig. 1). The preliminary observations were borne out by the additional cases studied. The delay in desaturation was found to correlate principally with the pulmonary outflow obstruction and trapping as reflected in the index of intrapulmonary mixing. Patients with normal ventilation did not show delay in nitrous oxide desaturation. It was found, however, that an additional 5 to 10 minutes was needed to bring the arterial and coronary venous blood into equilibrium with the gas mixture as compared with patients without pulmonary disease.

Table 1 shows the results in these nine patients. Note that six had elevation of the pulmonary artery pressure and one in whom this was not recorded had elevation of the right ventricular pressure; 5 had elevation of the right ventricular end diastolic pressures. The hematocrit reading and presumably the blood volume was not unusually high in 3 of these 5 cases. The coronary blood flows are near the average normal of 60-100 ml/100 Gm./min. as given by Bing. This correlated fairly well with the cardiac index (fig. 2). In one patient (A. G.), with a marked elevation of the cardiac index, a correspondingly high coronary blood flow was found. Table 1 also relates the cardiac index to the coronary blood flow, the coronary arteriovenous oxygen difference, the myocardial oxygen consumption or MMRO₂ (myocardial metabolic rate for oxygen). These results did

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not show a significant variation from the normal range. The presence or absence of cor pulmonale did not appear to influence the results. Patient R. A. also suffered from extensive sarcoidosis; his emphysema was mild and his reduction in pulmonary function was due to impaired inspiratory reserve. There was no appreciable delay in the nitrous oxide desaturation.

**Table 1.** Results of Physiologic Studies

<table>
<thead>
<tr>
<th>Patient</th>
<th>Pulmonary Artery</th>
<th>Right Ventricle</th>
<th>Cardiac Index</th>
<th>Coronary Flow</th>
<th>AVOD Diff.</th>
<th>MMRO2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mm. Hg</td>
<td>mm. Hg</td>
<td>L./min</td>
<td>ml/100 Gm./min.</td>
<td>ml/100 ml.</td>
<td>ml/100 Gm./min.</td>
</tr>
<tr>
<td>G. S.† ‡</td>
<td>44/23</td>
<td>44/7</td>
<td>1.7</td>
<td>75</td>
<td>13.8</td>
<td>10.4</td>
</tr>
<tr>
<td>W. F.†</td>
<td>1.6</td>
<td>73</td>
<td>12.0</td>
<td>9.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>R. A.†</td>
<td>27/10</td>
<td>27/3</td>
<td>2.3</td>
<td>71</td>
<td>13.5</td>
<td>9.6</td>
</tr>
<tr>
<td>S. N.*</td>
<td>56/23</td>
<td>56/11</td>
<td>3.1</td>
<td>80</td>
<td>16.0</td>
<td>14.2</td>
</tr>
<tr>
<td>A. G.*</td>
<td>41/5</td>
<td>6.6</td>
<td>152</td>
<td>9.0</td>
<td>13.7</td>
<td></td>
</tr>
<tr>
<td>P. Y.*</td>
<td>43/27</td>
<td>43/10</td>
<td>2.9</td>
<td>107</td>
<td>12.3</td>
<td>13.2</td>
</tr>
<tr>
<td>J. O.*</td>
<td>55/24</td>
<td>55/6</td>
<td>2.7</td>
<td>90</td>
<td>12.1</td>
<td>10.9</td>
</tr>
<tr>
<td>L. E.†</td>
<td>25/10</td>
<td>25/3</td>
<td>3.6</td>
<td>97</td>
<td>13.3</td>
<td>12.9</td>
</tr>
<tr>
<td>M. T.*</td>
<td>52/24</td>
<td>52/8</td>
<td>2.8</td>
<td>77.3</td>
<td>12.1</td>
<td>9.4</td>
</tr>
</tbody>
</table>

* Indicates emphysematous patients with cor pulmonale.
† Indicates emphysematous patients without cor pulmonale.
‡ Elevated pulmonary artery pressure due to left ventricular failure.

The normal values for the coronary blood flow were thought to be some evidence against extensive invasion of the myocardium by sarcoid.

**Fig. 1.** A, curves obtained from patient without pulmonary disease. B, with pulmonary emphysema.

**Fig. 2.** Graph showing correlation between coronary blood flow in ml/100 Gm/min. (abscissa) and cardiac index, in L./min. (ordinate).
DISCUSSION

Animal studies indicate that hypoxia is the greatest stimulus to increased coronary blood flow. It appears that these patients have either compensated for this or that critical lowering of the O₂ tension was not present. One patient did have definite elevation of his coronary blood flow with a high cardiac index, moderate unsaturation, and a high normal hematocrit of 52.

It has been recently suggested by Corday following experimental work in animals and anatomical dissection in cadavers that the left coronary artery could be compressed between the aorta and pulmonary artery in pulmonary hypertension. This might account for the pathologic picture of patchy myocardial necrosis and pain seen in cor pulmonale. In two patients who had the highest pulmonary artery pressure in this series and who also had very large pulmonary arteries, the coronary blood flow was found to be in the upper range of normal.

In many cases of cor pulmonale, because of the marked positional changes in the electrocardiogram, very little information is obtainable concerning the left ventricle. It has been postulated that right heart failure supervenes in cor pulmonale, especially in the older patients, because of associated degenerative heart disease. Our studies indicate that the coronary blood flow at rest was not impaired in any of these patients and whether or not co-existing coronary disease was impeding a higher basal coronary flow is a matter of conjecture.

SUMMARY

Coronary blood flow was measured in nine individuals with varying degrees of pulmonary emphysema alone and emphysema with cor pulmonale. Determinations of other cardiac and pulmonary functions were also studied. In 8 of the 9 cases, coronary blood flow was within the normal range. In one individual with high cardiac output coronary blood flow was also elevated but the ratio of coronary blood flow to cardiac output was maintained. The presence of cor pulmonale did not appear to influence the results significantly.

SUMMARIO IN INTERLINGUA

Le coronari fluxo sanguinee esseva mesurate in 9 individuos con varie grados de emphysema pulmonar, sol o in combination con corde pulmonal. Determinationes de altere functiones cardiac e pulmonar esseva etiam facite. In 8 del 9 casos, le coronari fluxo sanguinee esseva intra limites normal. In un individuo con alte rendimento cardiac, le coronari fluxo sanguinee esseva etiam elevate, sed le proportion de coronari fluxo sanguinee e rendimento cardiac esseva mantenite. Le presentia de corde pulmonal non pareva influentar le resultatos significativemente.

REFERENCES

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