SECTION ON CARDIOVASCULAR DISEASES

Heart Disease of Pulmonary Origin

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It behooves clinical investigators in a University Hospital group to survey from time to time, recorded clinical material on pressing diagnostic and therapeutic problems. Heart disease of pulmonary origin is a pertinent and pressing topic for, when fully developed, it has been considered hopeless by too many physicians. We have, therefore, reviewed our cases in an attempt to reemphasize precipitating and perpetuating conditions, determine predisposing factors and evaluate early diagnostic signs. We hope to touch upon possible prophylactic measures and argue for the establishment of an effective a program of management as possible. We first studied the records of 208 cases, who had been cross filed under the diagnosis of chronic cor pulmonale. It soon became apparent that minimal criteria for the diagnosis of chronic cor pulmonale had to be established, and after considerable study we arrived at those set down in Table I. We were acutely aware that chronic cor pulmonale is an end result of an insidious process. Furthermore we were fully conscious of the difficulty of arbitrarily drawing a line separating definite cor pulmonale from pulmonary disease with positional electrocardiographic changes.

The application of our criteria, as outlined here, has limited our study to patients with definite respiratory disease and pulmonary hypertension, and definite right atrial and right ventricular enlargement; as shown by the electrocardiogram or the teleradiogram. In addition, we have accepted the presence of right heart failure, without left failure, as presumptive evidence of right ventricular enlargement, even when other signs were equivocal. Patients with tricuspid or pulmonary valvular

<p>| TABLE I |</p>
<table>
<thead>
<tr>
<th>CRITERIA FOR DIAGNOSIS OF PURE COR PULMONALE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Evidence of Hypertension of the Pulmonary Circuit</td>
</tr>
<tr>
<td>A. Midsternal or Epigastric Heave</td>
</tr>
<tr>
<td>B. Pulmonary Diastolic Shock</td>
</tr>
<tr>
<td>C. Sharply Accentuated &amp; Split P2</td>
</tr>
<tr>
<td>2. Definite Evidence of Right Ventricular Enlargement</td>
</tr>
<tr>
<td>A. ECG-Right Axis Deviation, P1 &amp; P2 Tall and Sharp; High R in V1</td>
</tr>
<tr>
<td>B. Radiogram</td>
</tr>
<tr>
<td>C. Right Heart Failure without Left Heart Failure</td>
</tr>
<tr>
<td>3. Absence of Significant Valvular Lesions, Systemic Hypertensive or Ischemic Heart Disease with Myocardial Infarction</td>
</tr>
<tr>
<td>4. Symptoms and Signs of Pulmonary Disease</td>
</tr>
</tbody>
</table>

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disease, constrictive pericarditis, systemic hypertension, ischemic myocardial changes and congestive failure of the left ventricle and a few rare or equivocal conditions were specifically excluded. Patients with mitral stenosis and congenital cardiac lesions were intentionally omitted, even though they were known to have increased resistance to pulmonary flow, and pulmonary hypertension. The vascular changes were secondary to heart disease and no primary lung disease was present.

Pulmonary function studies have added much to our concepts of the physiology of the cardio-respiratory system. However, these are mainly of value in the early detection quantitating, localizing and differentiating the pathology in the lung. Respiratory tests have been utilized only to a limited extent in studying secondary cardiac changes and were neglected in most of our cases, even though they promise much in early diagnosis.

In Table II, we have grouped the findings of our 208 cases, with which we began our study. By our criteria, there were among these 208 records only 51 indisputable pure chronic cor pulmonale cases. An additional 32 had pulmonary disease, many with various electrocardiographic findings, but in these we could not demonstrate right heart enlargement or failure. Another 60 cases were excluded at the beginning because of complicating cardiovascular diseases. In 44 the cardiac changes were insignificant and in 21 there was no evidence to substantiate pulmonary origin of the clinical picture.

In Table III, we have listed some of the unusual conditions which have

### TABLE II
### ANALYSIS OF 208 CASES DIAGNOSED AS COR PULMONALE

| 1. Definite Pure Cor Pulmonale | 51 |
| 2. Lung Disease with Some ECG Changes, But No Demonstrable Hypertrophy or Failure of the Right Ventricle | 32 |
| 3. Cases with Complications: Systemic Hypertension, Infarctions, Left Failure, etc. | 60 |
| 4. Cases of Pulmonary Disease in Which Cardiac Changes were Minimal or Absent | 44 |
| 5. Cases in Which the Primary Pathology was Extra Pulmonary | 21 |
| **TOTAL** | **208** |

### TABLE III
### UNUSUAL CONDITIONS SIMULATING CHRONIC COR PULMONALE

| 1. Carcinoma of Lung impinging on Pulmonary Artery |
| 2. Intracardiac Aneurysm |
| 3. Thrombosis of Pulmonary Artery |
| 4. Essential Pulmonary Hypertension |
| 5. Constrictive Pericarditis |
| 6. Tricuspid Valvular Disease |
TABLE IV
AGE, SEX AND RACE OF 51 PATIENTS WITH COR PULMONALE

<table>
<thead>
<tr>
<th>Age</th>
<th>No. of Cases</th>
<th>Per Cent of Total</th>
<th>Sex and Race</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 - 10 years</td>
<td>2</td>
<td>4</td>
<td>White Male</td>
</tr>
<tr>
<td>10 - 20 years</td>
<td>2</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>20 - 30 years</td>
<td>1</td>
<td>2</td>
<td>Colored Male</td>
</tr>
<tr>
<td>30 - 40 years</td>
<td>4</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>40 - 50 years</td>
<td>10</td>
<td>20</td>
<td>White Female</td>
</tr>
<tr>
<td>50 - 60 years</td>
<td>15</td>
<td>30</td>
<td></td>
</tr>
<tr>
<td>60 - 70 years</td>
<td>14</td>
<td>28</td>
<td>Colored Female</td>
</tr>
<tr>
<td>70 - 80 years</td>
<td>3</td>
<td>6</td>
<td></td>
</tr>
</tbody>
</table>

been confused with cor pulmonale in this series. The exclusion of some of these cases may seem arbitrary, as for example, bronchogenic carcinoma impinging on the main pulmonary artery is clearly lung disease with secondary right heart failure. However, we felt it desirable to exclude those conditions without significant impairment of pulmonary function.

We have analyzed our cases in regard to age, and these data are shown in Table IV. Though there was a rather widespread distribution, the high incidence in the middle aged groups has been demonstrated. This is probably due to several factors. Lung disease must be present for a considerable time to produce changes, and the development of atherosomatous degeneration will decrease cardiac reserve and serve to precipitate right heart decompensation. Two status asthmaticus deaths in two of our young patients were of great interest, these children seen in the first decade of life had mucoviscidosis, with recurrent bouts of respiratory infection and with chronic bronchial infections. Both died before the age of two and incidentally, both had relative anemias instead of the polycythemias, usually present in pulmonary hypoxia.

The sex and race incidences of patients with cor pulmonale shown in Table IV indicate an overwhelming preponderance of white males in our group which is also a finding worthy of some attention. There must

TABLE V
ETIOLOGY OF 51 CASES OF PURE CHRONIC COR PULMONALE

<table>
<thead>
<tr>
<th>Etiology</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma-Bronchitis-Emphysema</td>
<td>33</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td>6</td>
</tr>
<tr>
<td>Congenital Cyst of Lung</td>
<td>3</td>
</tr>
<tr>
<td>Pulmonary Fibrosis</td>
<td>2</td>
</tr>
<tr>
<td>Pneumoconiosis</td>
<td>2</td>
</tr>
<tr>
<td>Coeliac Disease</td>
<td>2</td>
</tr>
<tr>
<td>Other Conditions</td>
<td>3</td>
</tr>
</tbody>
</table>
be more than one factor responsible for this, and we have considered
a greater exposure of men to industrial dusts and colds. The higher inci-
dence of untreated asthmatic bronchitis and of atheromatous changes in
males were judged significant. The probably more frequent use of toba-
ccco by men might appeal to some as contributing to this disproportion. We
have no explanation for the racial difference except that the non-cau-
sions in this area, are of lower socio-economic status and might have
succumbed to other causes before the full-blown cardio-pulmonary syn-
drome had time to develop.

In Table V, the asthma-emphysema syndrome is shown to be the
principal etiology. These individuals invariably had had chronic plastic
bronchitis and episodes of status asthmaticus were common. Two of
them had complicating kyphoscoliosis and emphysema. The cases of
congenital cysts of the lung are of interest for while they are relatively
rare, they may be amenable to radical therapy, and two of the three
patients have been subjected to surgical resection, with improvement.
The cases of pneumoconiosis have been rare in our area but are important
to note, in that they may probably be avoidable, and have been prevented
by proper industrial hygiene.3

The Electrocardiogram in Cor Pulmonale

In analyzing in detail the electrocardiograms of these patients, we found,
as was to be expected, that sinus tachycardia was extremely common, and
in fact, was the predominant rhythm. It was interesting to find that
the incidence of atrial fibrillation was only 6 per cent, which is rather
rare in contrast to its frequency in conditions characterized by left atrial
dilatation. This may be related to the lack of myocarditis in our patients.
We found, in those with dilated conus arteriosus, the typical electro-
cardiographic picture of right axis deviation, electrically vertical position
and clockwise rotation of the heart. Peaked P waves in II, III and AVF,
were more frequently seen in younger patients who were not likely to
have complicating myocardial disease. Evidence of right ventricular

<table>
<thead>
<tr>
<th>TABLE VI</th>
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</thead>
<tbody>
<tr>
<td><strong>Cor Pulmonale</strong></td>
</tr>
<tr>
<td>17 Cases</td>
</tr>
<tr>
<td>Maximum</td>
</tr>
<tr>
<td>Minimum</td>
</tr>
</tbody>
</table>

| **Uncomplicated** Emphysema | **Plasma** | **RBC Mass** | **Total Blood** |
| 6 Cases                      |**Average**|   + 3.3    |    + 4.3    |     + 5.8    |
| Maximum                       |   45.0    |   +12.0    |    +19.0    |     +19.0    |
| Minimum                       |   40.5    |   - 9.0    |    - 8.0    |     - 9.0    |

(where possible, the values found before digitalization were used)
hypertrophy, in precordial leads was not commonly seen. The tracing of V3R, and V4R would probably have added to our evidence but these leads were not recorded routinely on most of our patients.\textsuperscript{4} We analyzed various R:S ratios in the different leads, as well as many other possibly significant points but were unable to discover any further additions to the previous diagnostic features. Vector cardiography promises some help in the early recognition of slight right ventricular hypertrophy.

*Heart Failure and Blood Mass Alterations*

In 80 per cent of our 51 cases of cor pulmonale congestive heart failure was present and all of these were subsequently digitalized. It is notable that 13 (25.5 per cent) of these patients developed abnormal rhythm or other signs of digitalis intoxication sometime during our observation. This complication was probably due to efforts to push too far the cardiac

### TABLE VII

**Blood Volume Studies, Plasma, RBC Mass and Total in Patients with Cor Pulmonale in Failure and in Compensated States in Percentages Above and Below Normal Values**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Weight Kg</th>
<th>V.P. Cm. Sahline</th>
<th>Hb. Cnt. Per Cent</th>
<th>Hct.</th>
<th>P.V. Icm</th>
<th>RBC Mass Cm.</th>
<th>Total B.V. P.V. and RBC M.</th>
</tr>
</thead>
<tbody>
<tr>
<td>E. L. P.</td>
<td>42</td>
<td>53</td>
<td>18</td>
<td>20.1</td>
<td>66</td>
<td>+11</td>
<td>+83</td>
<td>+44</td>
</tr>
<tr>
<td>J. D. C.</td>
<td>54</td>
<td>87</td>
<td>21</td>
<td>19.2</td>
<td>61.8</td>
<td>+2</td>
<td>+33</td>
<td>+17</td>
</tr>
<tr>
<td>O. R.</td>
<td>57</td>
<td>53</td>
<td>22.5</td>
<td>15.4</td>
<td>58</td>
<td>+32</td>
<td>+95</td>
<td>+61</td>
</tr>
<tr>
<td>R. B.</td>
<td>25</td>
<td>60</td>
<td>27</td>
<td>16.8</td>
<td>58.5</td>
<td>-18</td>
<td>+46</td>
<td>+11</td>
</tr>
<tr>
<td>C. K.</td>
<td>48</td>
<td>66</td>
<td>20</td>
<td>16.3</td>
<td>58.5</td>
<td>+37</td>
<td>+57</td>
<td>+46</td>
</tr>
<tr>
<td>C. O. D.</td>
<td>68</td>
<td>76</td>
<td>24</td>
<td>17.0</td>
<td>56</td>
<td>+16</td>
<td>+23</td>
<td>+21</td>
</tr>
<tr>
<td>W. H.</td>
<td>61</td>
<td>90</td>
<td>25</td>
<td>17.0</td>
<td>55</td>
<td>-20</td>
<td>+64</td>
<td>+14</td>
</tr>
<tr>
<td>J. A.</td>
<td>49</td>
<td>62</td>
<td>30+ 31</td>
<td>16.5</td>
<td>54.8</td>
<td>+34</td>
<td>+63</td>
<td>+41</td>
</tr>
<tr>
<td>B. E.</td>
<td>59</td>
<td>50</td>
<td>12</td>
<td>15.6</td>
<td>55</td>
<td>-5</td>
<td>+23</td>
<td>+20</td>
</tr>
<tr>
<td>W. E. T.</td>
<td>67</td>
<td>64</td>
<td>7</td>
<td>17.0</td>
<td>49</td>
<td>-23</td>
<td>+26</td>
<td>+0</td>
</tr>
<tr>
<td>L. L.</td>
<td>42</td>
<td>61</td>
<td>28</td>
<td>14.2</td>
<td>48</td>
<td>+27</td>
<td>+21</td>
<td>+24</td>
</tr>
<tr>
<td>J. C.</td>
<td>43</td>
<td>56</td>
<td>11</td>
<td>15.0</td>
<td>45</td>
<td>+3</td>
<td>+10</td>
<td>+6</td>
</tr>
<tr>
<td>A. A.</td>
<td>68</td>
<td>45</td>
<td>12</td>
<td>14.6</td>
<td>42</td>
<td>+12</td>
<td>+5</td>
<td>+9</td>
</tr>
<tr>
<td>C. W.</td>
<td>63</td>
<td>77</td>
<td>10</td>
<td>12.9</td>
<td>40.5</td>
<td>-9</td>
<td>-8</td>
<td>-9</td>
</tr>
<tr>
<td>J. W. S.</td>
<td>35</td>
<td>79</td>
<td>11</td>
<td>14.5</td>
<td>43.5</td>
<td>+7</td>
<td>-7</td>
<td>+1</td>
</tr>
</tbody>
</table>
glycoside to control this rather refractory form of failure.

Digitalis and diuresis of 5 of the cor pulmonale patients with failure resulted in decrease of plasma volume to below normal values. Oddly enough, the erythrocyte mass also decreased markedly in three of these cases by an average of 44 per cent while two showed no significant change. There has been subclinical jaundice demonstrated in some of these instances as evidence of a hemolytic component, but it did not seem to be of the magnitude to explain these differences. The possibility of shift of the red blood cell mass and erythrocyte sequestration were considered.

The severe cases almost invariably had polycythemia, but when we tabulated our data, the hemoglobin values ranged from 22 gms. per cent to 10.0 gms. per cent with an average of 16.1 gms. per cent which is just above the upper limits of normal for our laboratory. Nearly 40 per cent of the 51 patients had a normal value but 5, or 9.8 per cent had a complicating anemia. The results are summarized in Table VI and show the hematocrit varied from 66 per cent to 48 per cent with an average of 56.4 per cent. The venous pressure was, of course, increased in those patients with right ventricular failure. It has been noted by Reilly and by Lewis et al that the total blood volume is expanded in most cases of heart failure6,7 four of which had cor pulmonale.

Detailed blood volume studies on 17 of our patients were done using Cr131 to study the red blood cell mass by the methods of Sterling and Grey8 and plasma volume studies were done with I131 by the method of Crispell et al.9 We studied six cases with uncomplicated emphysema for comparison. The results are shown in Table VI as change in per cent above and below our normal for an individual of a similar age, sex, and body surface area. These are shown in detail in Table VII. Although there was some variation of plasma volume and only three showed a significant increase of 30 per cent or more, the red blood cell mass was consistently elevated with value ranging from +21 per cent to +83 per cent with an average increase of +48.1 per cent.

The total blood volumes were derived by addition of plasma volume and red blood cell mass and ranged from no increase to an increase of 46 per cent in the cor pulmonale patients with an average increase of 27 per cent. In contrast only four of the six emphysema patients showed increases with values ranging from —8.1 per cent to +19.0 per cent and an average of +9.0 per cent. These findings were not considered outside of the normal range.

**Bronchopulmonary Measures**

In the management of these patients, we would first emphasize prophylaxis, that is, control of pulmonary disease to delay or prevent cardiac changes. We have learned to practice vigorous treatments of asthmatics and desensitization to allergin if such are demonstrable.

First and foremost in management are measures aimed at alleviation of bronchopulmonary condition and any contributing focus as sinusitis or tonsillitis. We would again emphasize the value of antibacterial agents for the prompt control of bronchial infection in a primary or recurrent
flare-up. Bronchodilators, as anti spasmodics, aminophyllin, wetting agents and mucous digestants have been used frequently in recent years in our hospital. The most common plan has been a combination of aerosols as Isuprel, aerolone and Alevaire, in equal parts, either by a No. 40 De Vilbiss nebulizer, or with intermittent positive pressure oxygen apparatus of the Monaghan type. Aminophyllin (0.25-0.5 gm.) intravenously is often life saving. Abdominal pads and binders or pneumoperitoneum to elevate the diaphragm, with the hope of increasing the bellows action and augmenting the vital capacity may sometimes help. Most of our patients receive instruction in diaphragmatic breathing exercises, to increase the efficiency of the abdominal bellows.¹⁰ All of these measures seem to add to longevity and postpone complications.

Industrial safeguards which are being more and more applied should be advocated as they will, in time, tend to reduce the incidence of pneumoconiosis and subsequent fibrosis. We have found potassium iodide in a saturated solution, carefully administered in gradually increased dosage to produce rather marked symptomatic relief in a large number of patients, particularly those with an allergic bronchitis component. Adrenal cortical steroids are apparently of some value in those allergic patients with eosinophilia, and prednisone or prednisilone would be preferable to cortisol or hydracortisone because of the decreased tendency to salt and fluid retention and potassium excretion.

Cardiovascular Procedure

Management of cardiac complications has largely been directed toward an attempt to correct myocardial failure. Venesection with blood letting until the hemoglobin is 13 gm. and the hematocrit is below 50, is usually beneficial. Digitalis, when indicated by clinical signs of failure with increased venous pressure is of value, and its efficacy may tend to vary directly with the extent to which ischemic myocardial changes complicate the picture. Mercurial diuretics are of value in reducing the hypervolemia of heart failure. Diamox has been most commonly used in these patients, because of the well known complication of respiratory acidosis and elevated CO₂.¹¹ We have used 250 to 500 mgs. of diamox every day or every other day, adjusting the dosage to the individual requirements. Reduction of the blood volume in acute episodes with phlebotomy has rarely been necessary, and should be done carefully. In hypervolemic polycythemic patients 500 cc. is usually sufficient to bring the hemoglobin to 12-13 gm. and the hematocrit to 45 to 50 gm.

In addition to these rather specific measures, we have advocated reduction of activity to reduce oxygen requirements of the tissues and a sedative as phenobarbital in moderate doses by mouth which seemingly has been of aid. Recently, the use of radio Iodine I₁₃¹ in euthyroid patients has been advocated, in an effort to reduce metabolic needs by intentionally inducing a hypothyroid state. It would seem preferable to use drugs of a temporary antithyroid type, as propyl-thiouracil or tapezol. These agents have been used to a limited extent, and the therapy seems to be of some definite value. We have felt that the relatively heroic iodine destruction of
thyroid tissue should be reserved for the most severe cases, at least until further studies make optimal dosage a more predictable quantity.

Finally, we must briefly mention the psychotherapeutic aspect of this disease, which, in common with other chronic, progressive conditions, continues to be a significant feature from the patient's point of view at any rate. An aggressive, confident, or optimistic attitude by the doctor must be an integral part of the successful management of these unfortunate victims.

The adoption of a nihilistic attitude by the physician, can contribute as much to a patient's decline as any other factor.

Discussion

In these studies we have been impressed with the frequency of the insidiously developing pulmonary disease in patients in the wake of recurrent bronchitis. Patients are asymptomatic between attacks for 20 to 30 years because of the great 20 fold reserve of the lungs during the first phase of the disease. The subsequent development of chronic hypertensive coronary artery heart disease or chronic valvular disease, may obscure the signs of predominant right heart enlargement. Under these circumstances, the pulmonary condition is often subclinical and overlooked until an acute respiratory infection precipitates symptoms.

It is certainly advantageous to the patient to have had a potential or a subclinical diagnosis prophetically made in the first phase by a wise and cautious physician, before symptoms of pulmonary origin develop, for then long postponement may be accomplished. Every recurrent or chronic cough, every asthmatic attack especially with increasing residual air in the lung, gradual decrease of exercise tolerance and fatigability, should suggest developing emphysema in the middle aged man.

The respiratory dead space increasing more rapidly than normal with aging, causes the second phase of symptoms to progress more rapidly over a period of five to eight years. The chest expansion and movement of the lung borders decrease, the timed vital capacity drops off as the residual air volume increases and the anteroposterior diameter increases slowly, almost imperceptibly. The electrocardiographic signs of right axis deviation, S1 increasing, PII and especially VI sharpening and R developing in V1, V2 and V3, and S over the left precordium appear late. Vectorcardiography, and radiological studies help in the detection of early or slight right cardiac enlargement.

In the third phase, a heart disease of pulmonary origin, symptoms and signs of myocardial failure, appear and threaten dissolution within a few years. The development of pulmonary hypertension and polycythemia adds to the loudness and splitting of the P2 sound and gradually leads to the appearance of signs of right heart failure, engorged neck veins, cyanosis, enlarged liver, ascites, and edema, all of which are rather late findings. Supportive treatment, coronary vasodilators, and cardiac tonics are usually effective, for a time at least, even in this stage of chronic pulmonary disease.

However, the diagnosis and the developing cardiopulmonary process
should be made in its incipiency long before symptoms develop and steps
taken to postpone the progression by prompt early vigorous treatment
of the pulmonary condition. Unfortunately, the majority of patients do
not present themselves until the disease is far advanced. We must be
alert to the facts that every worker in a dusty trade, every asthmatic
patient, every person with recurrent bronchitis, bronchiectasis, fibrosing
tuberculosis, pneumonitis, sarcoidosis, bronchopulmonary disease of
spirochetal, parasitic a protozoal origin, repeated sprays of pulmonary
embolism\textsuperscript{13} or cystic disease of the lung may develop primary cor
pulmonale.

Secondary cor pulmonale results from capillary engorgement, alveolar
edema in valvular disease as develops in mitral stenosis and in Eisen-
mengers complex or pulmonary valvular disease. It is necessary to rule
out the presence of signs of constrictive pericarditis, or valvular disease
and coronary artery lesions which may be obscured by progressive
emphysema,\textsuperscript{14} and masquerade as heart disease of pulmonary origin.

**SUMMARY**

1. Criteria for definite, pure, chronic cor pulmonale have been set
down and 51 patients who meet these qualifications were studied. By
far, the greatest incidence is in middle aged white males, though it is
seen in all ages, both sexes, and in various racial groups.

2. The electrocardiogram shows the changes rather late, and in general
was of relatively little value in assessing the degree of cardiac change;
yet, the ecg. finding may be the first to focus attention on the presence
of a right ventricular overload.

3. Detailed blood volume studies were done in 17 patients with cor
pulmonale and six individuals with emphysema, using isotopes I\textsubscript{131} human
serum albumin to determine plasma volume, and Cr\textsubscript{51} for estimation of the
total red blood cell mass. The emphysema patients were not consistently
abnormal, but those with definite cor pulmonale showed a variable increase
of total blood volume, and a consistent increase of red blood cell mass.

4. Follow-up studies after digitalization of patients with failure showed
decrease in plasma volume invariably, and frequently, a marked inex-
plicable decrease of red blood cell mass.

5. In management of these patients, we have strongly emphasized the
importance of prophylaxis, early recognition, and energetic treatment in
the early phases, even before cardiac changes are manifest. We must
admit that the patients who have met our criteria represent the most
advanced and refractory ones. While the long term prognosis is poor,
current drugs and practices offer the patient comfort, ability to work,
and probably a longer life span than was previously possible.

**RESUMEN**

1. Un criterio definido y puro sobre el cor pulmonale crónico se ha
establecido ye se han estudiado 51 enfermos que reunieron las característi-
cas. La mayor incidencia, con mucho, se encuentra en los hombres de
mediana edad, aunque se ve todas las edades en ambos sexos y en varios grupos raciales.

2. El electrocardiograma muestra los cambios más bien tarde y en general fué de escaso valor para estimar el grado de la alteración cardiaca; sin embargo puede ser que ECG por sus cambios sea el primero que llame la atención sobre la presencia de abolsamiento del ventrículo derecho.

3. Se hicieron estudios detallados de volumen sanguíneo en 17 enfermos con cor pulmonale y en seis individuos con enfisema, usando seroalbúmina marcada con I_131 para determinar volumen plasmático y Cr-51 para la estimación de la masa celular total de eritrocitos. Los enfermos de enfisema no fueron siempre anormales pero aquéllos con cor pulmonale definido mostraron un aumento variable en el volumen total sanguíneo y consistente aumento de la masa celular de eritrocitos.

4. El seguimiento de los enfermos después de digitalización con desfallecimiento mostró decrecimiento de volumen del plasma invariablemente y frecuentemente, un inexplicable decrecimiento del volumen celular de eritrocitos.

5. En el tratamiento de estos enfermos hemos recalado fuertemente la importancia de la profilaxis, descubrimiento rápido, y tratamiento enérgico de modo temprano aún antes de que haya alteraciones cardíacas.

Debemos admitir que los enfermos que reunieron las características según nuestro criterio representan los más avanzados y refractarios.

Si bien el pronóstico a la larga es malo, las drogas actuales y los métodos ofrecen al enfermo confort, capacidad de trabajo y probablemente una prolongación de la vida mayor de lo que era posible antes.

**RESUME**

1. L'auteur a déterminé les critères qui permettent de définir le coeur pulmonaire chronique pur, et a étudié 51 malades qui entrent dans cette catégorie. La fréquence la plus grande est de loin celle des individus de sexe masculin, de race blanche, moyennement âgés, encore que cette affection soit constatée à tous les âges, dans les deux sexes, et chez divers groupes raciaux.

2. L'électrocardiogramme montre des altérations plutôt tardives et n'a en général que peu de valeur pour déterminer l'importance de l'altération cardiaque; toutefois les constatations électrocardiographiques peuvent être les premières à signaler l'existence d'une anomalies ventriculaire droite.

3. Des études détaillées sur le volume sanguin furent faites chez 17 malades atteints de coeur pulmonaire, et six atteints d'emphyème, avec détermination du volume plasmatic y évaluation de la masse totale des globules. Les malades atteints d'emphysème ne montrèrent pas constamment des anomalies, mais ceux qui étaient atteints de coeur pulmonaire montrèrent une augmentation variable du volume sanguin total, y une augmentation importante de la masse des globules rouges.

4. Des contrôles après traitement des malades par la digitaline suivi d'échec, montrèrent invariablement une diminution du volume plasmatic et fréquemment une nette et inexplicable diminution de la masse des globules rouges.
5. Dans la conduite du traitement de ces malades, l'auteur insiste fortement sur l'importance de la prophylaxie, de la découverte précoce de l'affection et d'un traitement énergique dans la phase de début, avant même que des altérations cardiaques ne se soient manifestées. On doit admettre que les malades qui répondent aux critères que l'auteur a proposés représentent les cas les plus avancés et les plus sérieux. Bien que le pronostic à long terme soit mauvais, les drogues et médications courantes permettent d'offrir au malade un certain bien-être, la possibilité de travailler, et probablement une survie plus longue que celle qui était auparavant possible.

ZUSAMMENFASSUNG

1. Es wurden Merkmale niedergelegt für ein fest begrenztes reines chronisches cor pulmonale und 51 Kranke untersucht, auf die diese Kennzeichnung zutraf. Das bei weitem häufigste Vorkommen liegt bei weisen Männern im mittleren Alter, wenngleich man es in jedem Alter, bei beiden Geschlechtern und bei verschiedenen Rassen sieht.

2. Das Elektrocardiogramm zeigt die Veränderungen ziemlich spät und erwies sich im allgemeinen als von relativ geringem Wert bei der Bestimmung des Grades der cardialen Veränderung; der EKG-Befund kann den ersten auf den Brennpunkt gerichteten Augenmerk für das Bestehen einer Überbeanspruchung des rechten Ventrikel's sein.


REFERENCES


