Mottled chest radiograph and gas transfer defect in chronic liver disease

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In a prospective study of 170 patients with various types of chronic liver disease, a pulmonary gas transfer defect was found in 20% and mottled shadowing in the chest radiograph in 6%. The presence of these abnormalities was not related to the cause of the liver disease.

Reduction of transfer factor in liver disease was not accompanied by a restrictive ventilatory defect and was found in most cases with mottled radiographs.

The incidence of finger clubbing and the levels of both venoarterial admixture and cardiac output were higher in patients with mottling than in those with normal radiographs. Mottling was also seen in the chest radiographs of five other patients with hepatic cirrhosis who had previously been investigated for cyanosis due to intrapulmonary shunting.

Despite earlier reports of active chronic hepatitis and primary biliary cirrhosis occurring in patients with fibrosing alveolitis, we suggest that these radiographic changes in liver disease are usually caused by a pulmonary vascular disorder, rather than by coincidental lung disease, and are a local manifestation of a generalized vasodilated state. The low incidence of diffuse lung disease complicating chronic liver disease was confirmed by reviewing the hospital necropsy records.

In one patient the radiographic mottling disappeared and the physiological evidence of a circulatory disorder became less marked during a period of improved hepatocellular function.

Some of the cardiorespiratory disorders in chronic liver disease (CLD) have received considerable attention. They include hypoxaemia due to intrapulmonary arteriovenous shunts (Rydell and Hoffbauer, 1956), increased cardiac output (Kowalski and Abellmann, 1953), respiratory alkalosis (Vanamee et al., 1956), and pulmonary hypertension (Senior et al., 1968). It is less well known that mottled shadows may sometimes be seen at the lung bases on chest radiographs (Chrispin and Lessof, 1965); post-mortem studies have suggested that these are due to dilated precapillary pulmonary blood vessels (Berthelot, Walker, Sherlock, and Reid, 1966). Isolated cases of CLD have also been reported with reduced pulmonary transfer factor (Karlish, Marshall, Reid, and Sherlock, 1967; Cotes, Field, Brown, and Read, 1968; El Gamal, Stoker, Spiers, and Whitaker, 1970). Radiographic mottling and gas transfer defect are both characteristic features of diffuse lung disease and it has recently been reported that fibrosing alveolitis is occasionally associated with both active chronic hepatitis (Turner-Warwick, 1968) and primary biliary cirrhosis (Mason, McIlmurray, Golding, and Hughes, 1970).

METHODS

We present in this paper the results of pulmonary investigations in 170 patients with CLD. Special attention is given to the physiological findings in CLD with mottled chest radiographs and these are compared with the physiological pattern seen in diffuse lung disease. The incidence of diffuse lung disease in CLD was also determined by use of hospital necropsy records.

MATERIALS AND METHODS

We investigated 170 patients with CLD who had not been assessed from a respiratory viewpoint beforehand (the prospective study). The diagnosis of the liver disorder had been based on the clinical picture, liver biopsy, and immunological status. Seventy-three had active chronic hepatitis, 37 primary biliary cirrhosis, and 27 alcoholic cirrhosis; 33 had cirrhosis which was cryptocogenic or of other aetiology. Cases with severely decompensated liver disease, ascites or chronic bronchitis were excluded. Clinical evidence of a pulmonary disturbance was sought by a full history and physical examination of the chest. Dyspnoea was graded according to the revised Medical Research

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Council's questionnaire on respiratory symptoms. Recent chest radiographs were studied with the patients' identities concealed from the examiner. Vital capacity (VC) was measured by a dry spirometer (Vitalograph). Pulmonary transfer factor for carbon monoxide (TF) was measured by the single breath method of Ogilvie, Forster, Blakemore, and Morton (1957) modified by using the calculation of breath-holding time proposed by Jones and Meade (1961) and of alveolar volume from the dilution of helium by the test breath. Measured values of TF were corrected for the effect of anaemia by the regression formula of Dinakara et al. (1970).

Twenty-six patients with CLD consented to further physiological investigations after a full explanation of their nature and purpose. Details of these procedures are given elsewhere (Stanley and Woodgate, 1971). In brief, simultaneous collections were made of arterial blood, mixed venous blood, and expired air while breathing air and oxygen successively in the supine position. Gasometric analysis of these samples allowed determination of the alveolar-arterial O₂ differences (A-aDo₂) by the alveolar air equation, cardiac output (Qt), and venoarterial admixture (Qva) by the standard shunt formula.

For comparison VC, TF, A-aDo₂, and Qva were determined in eight successive patients attending this hospital with diffuse lung disease; four had fibrosing alveolitis and four had pulmonary sarcoidosis. Mixed venous blood was not sampled in these subjects, but an arteriovenous O₂ difference of 4·5 vol % was assumed for the calculation of Qva.

VC and TF were expressed as percentages of the predicted values for the patients' heights and ages given by Cotes (1968). Approximate 95% confidence limits for these measurements were set by the use of Cotes' estimates of their standard deviations in healthy subjects. Thus, normal ranges for VC and TF were assumed to be 75-125% and 70-130% of the predicted values respectively. The mean VC and TF of 20 normal control subjects studied in this laboratory were 99% and 101% of these predicted values. Gasometric data obtained in 14 healthy volunteers, whose mean age was 42 years, were also used to establish the following normal ranges: arterial Po₂ 78-106 mmHg, A-aDo₂ breathing ambient air less than 25 mmHg, and Qva less than 3% of the cardiac output.

We also reviewed the radiographs and physiological data of five patients with hepatic cirrhosis previously seen at our hospital who had been investigated for severe arterial unsaturation (the retrospective study). The necropsy records provided information on the pulmonary histology of 120 patients with CLD who died during the period 1965-70. They included 52 with active chronic hepatitis and 18 with primary biliary cirrhosis: their lung sections were re-examined by light microscopy and evidence of fibrosing alveolitis was sought. These patients also included 11 whose lung function had been assessed in life, 10 in the prospective study and one in the retrospective study.

Obtainable from W. J. Holman, Ltd., Dawlish, Devon

### TABLE I

<table>
<thead>
<tr>
<th>Cause of CLD</th>
<th>No. of Cases</th>
<th>Mean Age (yr)</th>
<th>Dyspnoea</th>
<th>Rales</th>
<th>Low Transfer Factor</th>
<th>Mottled Chest Radiograph</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(uncorrected for anaemia)</td>
<td>(corrected for anaemia)</td>
</tr>
<tr>
<td>Active chronic hepatitis</td>
<td>73</td>
<td>38</td>
<td>13 (18%)</td>
<td>0</td>
<td>20 (27%)</td>
<td>15 (21%)</td>
</tr>
<tr>
<td>Primary biliary cirrhosis</td>
<td>37</td>
<td>54</td>
<td>12 (12%)</td>
<td>0</td>
<td>10 (22%)</td>
<td>7 (19%)</td>
</tr>
<tr>
<td>Alcoholic cirrhosis</td>
<td>27</td>
<td>50</td>
<td>10 (30%)</td>
<td>2</td>
<td>6 (12%)</td>
<td>5 (18%)</td>
</tr>
<tr>
<td>Other causes of cirrhosis</td>
<td>33</td>
<td>49</td>
<td>10 (30%)</td>
<td>2</td>
<td>13 (39%)</td>
<td>8 (24%)</td>
</tr>
<tr>
<td>Total</td>
<td>170</td>
<td>46</td>
<td>40 (23%)</td>
<td>2</td>
<td>49 (29%)</td>
<td>35 (20%)</td>
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</table>

### TABLE II

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yr)</th>
<th>Liver Disease Cause Duration (yr)</th>
<th>Dyspnoea Grade</th>
<th>Clubbing</th>
<th>Vital Capacity (% predicted)</th>
<th>Transfer Factor % predicted</th>
<th>Arterial O₂ Tension (mmHg)</th>
<th>Alv-art. O₂ Diff. Breathing Air (mmHg)</th>
<th>Cardiac Index (1/min/m²)</th>
<th>Venoarterial Admixture (% cardiac output)</th>
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<tr>
<td>1</td>
<td>17</td>
<td>Fasciol. 10</td>
<td>1</td>
<td>+</td>
<td>+</td>
<td>54</td>
<td>66</td>
<td>41</td>
<td>6·2</td>
<td>11·0</td>
</tr>
<tr>
<td>2</td>
<td>26</td>
<td>ACH 12</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>37</td>
<td>85</td>
<td>27</td>
<td>8·8</td>
<td>9·2</td>
</tr>
<tr>
<td>3</td>
<td>30</td>
<td>Crypt. 13</td>
<td>1</td>
<td>-</td>
<td>+</td>
<td>38</td>
<td>69</td>
<td>43</td>
<td>5·6</td>
<td>7·3</td>
</tr>
<tr>
<td>4</td>
<td>56</td>
<td>Crypt. 6</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>71</td>
<td>66</td>
<td>40</td>
<td>5·3</td>
<td>7·3</td>
</tr>
<tr>
<td>5</td>
<td>47</td>
<td>PBC 6</td>
<td>0</td>
<td>+</td>
<td>+</td>
<td>86</td>
<td>88</td>
<td>30</td>
<td>6·1</td>
<td>6·2</td>
</tr>
<tr>
<td>6</td>
<td>59</td>
<td>Alc. 13</td>
<td>1</td>
<td>-</td>
<td>+</td>
<td>57</td>
<td>67</td>
<td>43</td>
<td>4·9</td>
<td>10·8</td>
</tr>
<tr>
<td>7</td>
<td>14</td>
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<td>0</td>
<td>+</td>
<td>+</td>
<td>61</td>
<td>78</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>8</td>
<td>48</td>
<td>ACH 8</td>
<td>0</td>
<td>+</td>
<td>+</td>
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<td>9</td>
<td>43</td>
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<td>-</td>
<td>+</td>
<td>68</td>
<td>84</td>
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</table>

Fasciol. = hepatic fascioliasis; ACH = active chronic hepatitis; Crypt. = cryptogenic cirrhosis; PBC = primary biliary cirrhosis; Alc. = alcoholic cirrhosis

¹Corrected for effect of anaemia
RESULTS

The prospective study The findings are summarized in Table I. Dyspnoea was common, although usually mild (grade I). More severe dyspnoea (grades II to IV) was present in 5%. Crepitations were heard in only two patients and on clinical grounds were attributed to heart failure rather than to lung disease. The measured levels of TF were low in 29% and in 20% after correction for anaemia. Mottled radiographs were seen in 6%. The incidence of reduced TF and abnormal radiographs was not related to the type of liver disease: in particular, these disturbances were not more frequent in cases of active chronic hepatitis or primary biliary cirrhosis.

Findings in cases with mottled radiographs (Table II) A striking aspect of the liver disease was its long duration in most cases. Eight of these nine patients had clubbing, compared with an incidence of only 8% in the 161 patients with normal radiographs. Four were mildly dyspnoeic, but none had clinical evidence of lung disease. Erythrocyte sedimentation rate was variable but often normal. Abnormal immunological tests were found only when appropriate to the underlying liver disease.

Physiological comparisons between CLD and diffuse lung disease Reduction of TF in CLD was often marked in those with radiographic mottling (Fig. 1). Indeed, cases of CLD with abnormal radiographs could not be distinguished from cases of diffuse lung disease by measurement of TF. By contrast, VC was virtually normal in CLD irrespective of the radiographic appearance but tended to be low in cases of lung disease (Fig. 2).

The levels of arterial Po2 and A-aDo2 in cases of CLD with mottled radiographs and patients with diffuse lung disease were similar (Fig. 3). Cardiac indices (Fig. 4) were usually higher in cases of CLD with mottled shadowing than in patients with
a normal chest radiograph. An increase of Qva was common in CLD but was present in only one patient with diffuse lung disease (Fig. 5). However, it was especially striking that Qva was higher in six of the seven cases of CLD with mottled shadowing than in any of the other patients.

![Graph showing cardiac indices in cases of chronic liver disease with either normal or mottled chest radiographs.](image)

**TABLE III**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yr)</th>
<th>Liver Disease</th>
<th>Dyspnœa Grade</th>
<th>Vital Capacity (% predicted)</th>
<th>Transfer Factor (% predicted)</th>
<th>Arterial O(_2) Tension (mmHg)</th>
<th>Venoarterial Admixture (% cardiac output)</th>
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<tr>
<td>10</td>
<td>60</td>
<td>ALC</td>
<td>2</td>
<td>99</td>
<td>49</td>
<td>52</td>
<td>35</td>
</tr>
<tr>
<td>11</td>
<td>33</td>
<td>Crypt.</td>
<td>2</td>
<td>102</td>
<td>38</td>
<td>(84%)</td>
<td>38</td>
</tr>
<tr>
<td>12</td>
<td>30</td>
<td>Crypt.</td>
<td>8</td>
<td>95</td>
<td>49</td>
<td>46</td>
<td>35</td>
</tr>
<tr>
<td>13</td>
<td>35</td>
<td>Crypt.</td>
<td>12</td>
<td>95</td>
<td>34</td>
<td>58</td>
<td>15</td>
</tr>
<tr>
<td>14</td>
<td>10</td>
<td>ACH</td>
<td>1</td>
<td>105</td>
<td>51</td>
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<td>28</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>2</td>
<td>100</td>
<td>51</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

 注1: Corrected for effect of anaemia
注2: % Arterial O\(_2\) saturation (measured by manometric Van Slyke apparatus)

ACH = active chronic hepatitis; ALC = alcoholic cirrhosis; Crypt. = cryptogenic cirrhosis

THE RETROSPECTIVE STUDY The findings in the five patients previously investigated because of arterial desaturation are shown in Table III. The liver disease was of no particular aetiology but had usually been of long duration. Case 5 had chronic bronchitis and bronchiectasis, but the remaining four patients had no clinical evidence of lung disease although they all complained of dyspnoea. All had cyanosis, finger clubbing, and high levels of venoarterial admixture. Retrospective examination of their chest radiographs revealed mottling in every case. Although none had significant reduction of vital capacity, all had considerable impairment of gas transfer.

**Histological study** The necropsy findings of 120 cases of CLD revealed a wide variety of common lung diseases, such as chronic bronchitis, emphysema, and pneumonia, but no evidence of fibrosing alveolitis was seen. Two cases of primary biliary cirrhosis had widespread intrapulmonary granulomas; marked radiographic mottling had been observed in one of these patients (case 9 in Table II) and full details will be reported elsewhere. Lung tissue was also available in a second case with mottling and a reduced TF (case 10 in Table III). This showed panacinar emphysema which had not been suspected clinically. However, this emphysema could not have produced the radiographic changes, although it was possibly responsible for the gas transfer defect. The pulmonary vessels had not been injected and no useful comments could be made about their structure. No pulmonary disease was found in two other cases in whom values of TF below 60% of predicted normal had been measured during life.
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FIG. 6. Case 1. Chest radiographs (a) September 1968. There are coarse nodular shadows which are most marked in the right lower zone. The hilar vascular shadows are enlarged and the peripheral vascular markings are accentuated. (b) Detail of the right lower zone showing the nodular shadows.
Chest radiographs The mottling was always bilateral with nodular shadows which were most pronounced in the lower lung zones, but in detail there were two distinct appearances. Usually the nodules were soft, coarse (1–4 mm), irregular, and superimposed on a clear background. An associated feature was accentuation of the pulmonary vessels both in the hilar regions and peripherally. The small vessels were especially prominent at the costophrenic angles where they often resembled septal lines, but linear shadows in this region could always be traced centrally to join other vessel markings and never extended laterally to touch the lung margin. These appearances were noted in cases 1 to 8 in the prospective study and in all the cases in the retrospective study. The most obvious changes of this type were seen in case 1 (Fig. 6a, b). A second type of shadowing was seen in case 9 of the prospective study. The nodules were much finer and in the right lower zone coalesced to form a ground-glass haze. The vascular markings were normal. She was the only case of CLD with nodular shadowing who had a normal level of cardiac index and venoarterial admixture. At necropsy she was found to have diffuse intrapulmonary granulomas.

CASE REPORT

Case 1 is reported in detail primarily to demonstrate that the radiographic shadowing and some of the associated physiological changes may resolve during clinical remission of the underlying liver disease. The case also shows that increased venoarterial admixture is not the sole cause of arterial hypoxaemia in CLD.

This young man had been admitted to hospital in 1959 when aged 7 years. He had hepatosplenomegaly and a blood eosinophilia of 15,000/mm³. Liver biopsy showed cirrhosis and infiltration of the portal tracts with eosinophils and lymphocytes. Investigations for a parasitic infestation including Fasciola hepatica were negative. A portacaval shunt was performed in 1961 for recurrent gastrointestinal bleeding. Finger clubbing and increased vascular markings on chest radiography were noted in 1964. The next year he developed precoma; he responded initially to a low protein diet, but in July 1968 he required additional neomycin therapy to prevent recurrent encephalopathy. Over this period (1964-68) his effort tolerance slowly deteriorated until he was unable to hurry on level ground. In September 1968 he was transferred to the Royal Free Hospital for further assessment.

Physical examination showed a small, icteric, and ill-looking youth. There was gross clubbing of the fingers and toes. His pulse (76/minute) was bounding. Blood pressure (110/65 mmHg) and venous pressure were normal. The second heart sound in the pulmonary area was accentuated but normally split. An ejection systolic murmur was audible over the whole praecordium. There was no peripheral oedema. Examination of the lungs was normal. The liver was enlarged, but the spleen was impalpable and there was no ascites.

Laboratory studies included the following: Haemoglobin 10·8 g/100 ml; WBC 17,500/mm³ with polymorphs 11,550, lymphocytes 4,900, monocytes 875, and eosinophils 175; and ESR 116 mm/hour; total serum bilirubin 14 mg/100 ml, alkaline phosphatase 90 iu/l, aspartate transaminase 90 iu/l, albumin 1·8 g/100 ml and globulin 5·6 g/100 ml. In the serum the Fasciola hepatica complement fixation test had become strongly positive (dilution 1:320). Chest radiography showed prominent hilar and peripheral vascular markings with widespread nodularity in both lower and middle lung zones (Figs. 6a, b). The electrocardiogram was normal.

Cardiorespiratory data are given in Table IV. Vital capacity was within normal limits, but transfer factor was very low even after correction for anaemia (54% predicted normal). A-aO₂ breathing room air was 41 mmHg. It was calculated that the venoarterial admixture was 11% of the cardiac output, which must have contributed a large part of the arterial hypoxaemia. Measurements of pressure and saturation at different sites during right heart catheterization revealed no evidence of congenital heart disease and the systolic murmur was attributed to the high cardiac index (6·2 l/min/m²).

Although a diagnosis of hepatic fascioliasis had not been fully substantiated, his deteriorating condition seemed to warrant a trial of specific therapy for this condition and he was given a course of emetine hydrochloride. Over the next 18 months the hepatic failure lessened and his effort tolerance improved. Dietary restrictions and neomycin therapy were withdrawn and he had no recurrence of encephalopathy. The total serum bilirubin fell to 7·5 mg/100 ml and the serum albumin level rose, although varying widely from month to month (2·0-3·5).

<table>
<thead>
<tr>
<th>TABLE IV</th>
</tr>
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<tbody>
<tr>
<td>CASE I: CARDIOPULMONARY DATA</td>
</tr>
<tr>
<td>Chest radiograph</td>
</tr>
<tr>
<td>Transfer factor¹</td>
</tr>
<tr>
<td>Vital capacity (ml)</td>
</tr>
<tr>
<td>Arterial oxygen tension (mmHg)</td>
</tr>
<tr>
<td>Alv-art. O₂ difference breathing air (mmHg)</td>
</tr>
<tr>
<td>Venoarterial admixture (%) cardiac output</td>
</tr>
<tr>
<td>Cardiac index (1/min/m²)</td>
</tr>
<tr>
<td>Pulmonary arterial pressure (mmHg)</td>
</tr>
</tbody>
</table>

¹Values in parentheses are corrected for effect of anaemia ²Lower values for 1968, higher values for 1970

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He was readmitted in May 1970 for further cardio-pulmonary investigation and the findings are shown in Table IV. The arterial $O_2$ tension had risen somewhat, together with a reduction of the elevated $A-aDO_2$ breathing air. Furthermore, the level of venoarterial admixture had fallen to within normal limits. Both cardiac index and pulmonary artery pressure were lower than previously. The chest radiograph (Fig. 7) no longer showed basal nodular shadowing or increased peripheral vascular markings and the hilar vascular shadows were less prominent. The TF had not improved and it seemed likely that the remaining hypoxaemia was largely due to ventilation-perfusion inequality, which may also occur in cirrhosis, as described by Cotes et al. (1968). The pulmonary hypertension, although extensively documented in liver cirrhosis (Senior et al., 1968), remains unexplained.

DISCUSSION

Since pulmonary gas transfer has often been regarded as normal in liver disease, we have reviewed the measurement of transfer factor in hepatic cirrhosis given by other workers. Reduced TF has been found in five previous cases studied by the single breath method (Karlish et al., 1967; Cotes et al., 1968; El Gamal et al., 1970). The steady state TF was also very low in one out of five cases reported by Blackburn et al. (1960). Using the hypoxic breathing procedure the transfer factor for oxygen has been calculated in 27 cases (Williams, 1960; Heinemann, Emirgil and Mijnssen, 1960; Abellmann, Kramer, Verstraeten, Gravallese, and McNeely, 1961; Chiesa, Ciappi, Balbi, and Chiandussi, 1969): among these were eight with values below 15 ml/min/mmHg, which has been given as the lower limit of normal (Comroe et al., 1962). Thus our values of transfer factor agree with earlier data, and the occurrence of impaired gas transfer in CLD seems well established. In cases of CLD without ascites a decreased TF is not usually associated with any restrictive ventilatory defect. This contrasts with most lung diseases in which a decreased TF tends to be accompanied by a small vital capacity. The
level of TF provided a useful index of the overall
efficiency of pulmonary gas exchange in CLD as
cases with diminished carbon monoxide uptake
usually had an increased A-aDO2 breathing room
air. Several possible mechanisms may limit gas
transfer in CLD. In some patients anaemia reduces
the mass of haemoglobin in the pulmonary capillaries
available for combination with carbon
monoxide. Coincidental lung disease may be
present in others. However, the level of TF
corrected for the effect of anaemia was often low
when lung disease was not apparent clinically or,
in two cases, at subsequent necropsy. Cotes
and his colleagues (1968) suggested that transfer
limitation in their patients might have reflected a
decreased surface area for gas exchange produced
by uneven pulmonary blood flow. Although more
recent work with radioactive xenon has shown that
the predominant regional inequality of lung func-
tion in cirrhosis resides in the distribution of
ventilation rather than blood flow (Ruff et al.,
1971), it remains possible that some pattern of
pulmonary inhomogeneity may be responsible for
the reduction of TF in liver disease.

The basal nodularity sometimes apparent on
chest radiography has been attributed to dilatation
of small pulmonary blood vessels (Berthelot et al.,
1966); this would also be in keeping with the
observed accentuation of vascular markings. The
high cardiac outputs giving increased pulmonary
flow rates in the cases with nodular shadows
provided an additional reason for their increased
vascular plethora. A physiological consequence
of this pulmonary vasodilatation was increased
venoarterial admixture, presumably due to opening
of multiple intrapulmonary arteriovenous anas-
tomoses. The increased vеноarterial admixture,
hyperkinetic circulation, and finger clubbing often
seen in the cases with radiographic shadows may
be concurrent manifestations of a generalized
vasodilatated state in CLD (Stanley and Woodgate,
1971). It is thus of special interest that all these
signs regressed in one patient during a period of
clinical improvement. Remission of cyanosis due
to intrapulmonary shunting in liver disease has
previously been reported in a child (Silverman,
Cooper, Moller, and Good, 1968) and it appears
that at least some of the circulatory changes caused
by CLD are not immutable. Alternatively, the
radiographic nodularity could be related to inter-
stitial oedema; this has been suggested as the cause
for hypventilation of the dependent lung zones
found with radioactive xenon in hepatic cirrhosis
(Ruff et al., 1971). However, true Kerley B lines
were not seen in any of our cases with nodularity.

Finally, one clinical aspect must be stressed.
Fibrosing alveolitis is occasionally accompanied by
active chronic hepatitis or primary biliary cirrhosis,
and in cases of CLD with both radiographic
mottling and reduced TF a diagnostic problem may
sometimes arise. For example, the first case report
of CLD with a low single breath TF (Karlish et al.,
1967) concerned a young man with active chronic
hepatitis: his chest radiograph showed nodular
shadowing which was initially attributed to fibros-
ing alveolitis, but precapillary vasodilatation was
the only pulmonary disorder found at necropsy.
In our cases with this combination of physio-
logical and radiographic disturbances we have
considered the overall picture to be unlike fibrosing
alveolitis, but without histological proof this
opinion must be guarded. Although differences
between CLD with radiographic mottling and
diffuse lung disease in the measurements of vital
capacity and vеноarterial admixture were observed,
their clinical importance is probably limited. The
vital capacity in CLD may be reduced by ascites
(Abelmann, Frank, Gaensler, and Cugell, 1954)
and increased venidoarterial admixture may occur in
advanced diffuse lung disease (Arndt, King, and
Briscoe, 1970). Indeed, the exclusion of pulmonary
disease in some cases of hepatic disease may not
be possible without lung biopsy, which may be
hazardous due to coagulation deficiencies. It is
thus fortunate that our necropsy material confirms
that the incidence of fibrosing alveolitis in CLD is
truly rare.

We wish to thank Professor Sheila Sherlock for per-
mission to study patients under her care and Dr.
Frances Gardner for allowing us to use the equipment
of the Department of Cardiology. Also we gratefully
acknowledge the skilled technical assistance given by
Mr. Donald Wagstaff, Mrs. Carol Shaw, and Miss
Harriet Copperman.

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Diffusing capacities and ventilation: perfusion ratios in
patients with the clinical syndrome of alveolar capillary
Arterial changes in the lungs in cirrhosis of the liver—
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Thorax 1972 27: 315-323
doi: 10.1136/thx.27.3.315

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