Ventilation-perfusion inequality in patients with non-alcoholic liver cirrhosis

G. Hedenstierna, C. Söderman, L.S. Eriksson, J. Wahren

ABSTRACT: Ventilation-perfusion relationships were studied in patients with non-alcoholic liver cirrhosis. Spirometry was essentially normal but the transfer factor of the lung (DLco) was reduced by on average 34% of predicted. Arterial oxygen tension (Pao2) ranged from normal down to 6.9 kPa. Varying degrees of ventilation-perfusion (VA/Q) abnormalities (multiple inert gas elimination technique) were observed with increased dispersion of the perfusion distribution (log suQ, 0.90; range 0.32–1.71; upper normal limit, 0.60) and the presence of both regions of low VA/Q ratios (between 0.1 and 0.005) (mean 4.1%; range 0–18.8%) and shunt (VA/Q ratios below 0.005) (mean 3.9%; range 0–19.8%). There was a close similarity between measured and calculated Pao2 in normoxaemic patients, but calculated values exceeded measured Pao2 in hypoxaemic patients. The difference between calculated and measured Pao2 correlated inversely to DLco (r=0.65, p<0.05). An inverse correlation was also noted between DLco and the sum of shunt and low VA/Q regions (r=0.87, p<0.001). It is concluded that hypoxaemia in non-alcoholic liver cirrhosis patients can be accounted for by intrapulmonary shunting and VA/Q mismatch, and possibly a "diffusion-perfusion" defect in patients with more severe gas exchange impairment.

Chronic liver disease with cirrhosis is frequently accompanied by impaired arterial oxygenation and sometimes hypoxaemia [1]. Using multiple inert gas elimination technique [2] several recent studies have demonstrated a ventilation-perfusion (VA/Q) mismatch in severely liver-diseased patients, mainly with alcoholic cirrhosis, and with varying smoking habits [3–7]. In three of these studies, the VA/Q mismatch could fail to fully explain the impaired arterial oxygenation [3–5]. However, additional factors such as diffusion limitation and extrapulmonary shunting were discussed in the two other papers on patients with more severe gas exchange impairment and considerable hypoxaemia [6, 7].

In patients with non-alcoholic cirrhosis impairment of arterial oxygenation has become an important issue, since severe hypoxaemia is a contraindication to liver transplantation [8]. To what extent intrapulmonary VA/Q mismatch, with or without other associated gas exchange disturbances, may explain the hypoxaemia in this group of patients has not been fully established. Better understanding of the causes of hypoxaemia may improve the perioperative care of the liver transplanted patient, and possibly widen the indications for such surgery.

In the present study we have evaluated the VA/Q relationships and diffusion capacity of the lung in essentially nonsmoking patients with advanced non-alcoholic liver cirrhosis and varying arterial oxygen tension (Pao2).

Material and methods

Patients

Fourteen patients with liver cirrhosis admitted to the hospital for assessment of a possible liver transplantation were investigated (table 1). Their mean age was 37 yrs (range, 18–50 yrs), mean weight 65 kg (range, 51–83 kg) and mean height 171 cm (range, 155–197 cm). The diagnosis was primary sclerosing cholangitis in four patients, primary biliary cirrhosis in four, chronic active hepatitis in three, cryptogenic cirrhosis in two, and tyrosinaemia in one patient. One patient had previously had bleedings from oesophageal varices and nine had slight ascites. None of the patients had systemic hypertension or cardiac or renal dysfunction. All had normal chest X-ray. Two patients were moderate smokers (less than ten cigarettes per day) (patients no. 5 and 10). All patients were hospitalized at the time of the study. The study was approved by the local ethics committee, and informed consent was obtained from each patient.
Clinical, pressure were measured. The difference between the and into the pulmonary artery. Pulmonary vascular occlusion pressure, obtained by inflating the balloon at oscopy to a right-sided hepatic vein (ten patients). The difference. The catheter was then advanced to the heart two pressures thus reflects the hepatic venous pressure sup ine posi ti on. A triple lum en thermistor-tipped catheter (Swan was introduced percutaneously after Ganz 7F Edwards Labo r ato ri es, Santa Ana, CA)

Catheterization and haemodynamic measurements

Lung function tests

Slow (static) and forced vital capacity manoeuvres (VC and FVC) were recorded by a low resistive bellows spirometer (Ohio 810), and the forced expired volume in one second as a percentage of FVC (FEV/FVC) was calculated. The transfer factor of the lung was analysed by the single breath CO method (DLco) (equipment: Mijnhardt diffusimat), and calculated according to standard techniques [9]. It was also corrected for haemoglobin concentration in blood and divided by alveolar volume [9]. Ref. values ALAT<0.7 µkat·l⁻¹, alkaline phosphatase<4.2 µkat·l⁻¹, prothrombin complex 70-130%, albumin 35-46 g·l⁻¹, haemoglobin 115-145 g·l⁻¹.

Ventilation-perfusion relationships

Six gases (sulphur hexafluoride (SF₆), ethane, cyclopropane, enflurane, diethylether, and acetone) were dissolved in isotonic saline and infused into a vein at a rate of 3 ml·min⁻¹. After 40 min of infusion, under steady-state conditions, arterial and mixed venous blood samples were taken and mixed expired gas collected for analysis by gas chromatography (Sigma 3, Perkin Elmer). Blood gas partition coefficients were determined by a two-step procedure. For technical details, see [11]. Arterial/mixed venous and mixed expired/mixed venous gas concentration ratios (retention and excretion, respectively) were plotted against blood gas partition coefficients. By formal mathematical analysis with enforced smoothing these relationships were transformed into a multicompartmental plot of blood flow and ventilation against VA/Q [2, 12]. Of the available information related to the VA/Q distribution data are presented on the mean VA/Q ratio of the ventilation and perfusion distributions (Vₐ/ₚ, Qₐ/ₚ).
the dispersion around the means, expressed as the logarithmic standard deviation of ventilation and perfusion distributions (log snV, log snQ), shunt (percentage perfusion of lung regions with VA/Q ratios <0.005), "low VA/Q regions" (Qlow; percentage perfusion of lung regions with 0.005 <VA/Q ratios <1), "high VA/Q regions" (Vhigh; percentage ventilation of lung regions with VA/Q ratios >100), and dead space (VD; percentage ventilation of lung regions with VA/Q ratios >100).

Blood gas analysis

Arterial and mixed venous blood were drawn for blood gas analysis, using standard techniques (analyser: ABL-2, Radiometer).

Statistics

Standard statistical methods were employed, using Student’s t-test and linear regression analysis when necessary. Data in the text and tables are presented as mean±SEM.

Results

Lung function

FEV₁/FVC was at the lower normal limit in two patients (patients no. 2 and 10). Otherwise there were no signs of airway obstruction. Mild reductions in vital capacity were shown in two patients (patients no. 2 and 9). The transfer factor, whether in absolute value (OLeo), or after correcting for haemoglobin concentration and normalizing for alveolar volume (Kco), was moderately reduced. Individual data are shown in table 1.

Table 2. - Ventilatory and haemodynamic variables

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>V̇E l·min⁻¹</th>
<th>Q̇T l·min⁻¹</th>
<th>HR b·min⁻¹</th>
<th>C(a-v)O₂ mmHg</th>
<th>RA PA mmHg</th>
<th>PCW mmHg</th>
<th>PS mmHg</th>
<th>PVR mmHg²·min⁻¹</th>
<th>SVR mmHg F¹·min⁻¹</th>
<th>HVPG mmHg</th>
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Mean V̇E: minute ventilation; Q̇T: cardiac output; HR: heart rate; C(a-v)O₂: arterial-mixed venous oxygen content difference; RA: mean right atrial; PA: mean pulmonary arterial; PCW: mean pulmonary capillary wedge; PS: mean systemic arterial; PVR: pulmonary vascular resistance; SVR: systemic vascular resistance; HVPG: hepatic venous pressure gradient; * RA assumed to be 7.5 mmHg.

Circulation

Cardiac output ranged from normal to high values (4.4–15.7 l·min⁻¹), maintained by a heart rate of mean 75 beats/min and a large stroke volume in most patients [13] (table 2). Pulmonary artery pressures were all normal to low and the pulmonary capillary wedge pressure was also within normal limits. The calculated pulmonary vascular resistance was in the lower range of normal [13]. Systemic vascular pressures and the calculated systemic vascular resistance were normal or low. The arterial-mixed venous oxygen content difference was low in most patients, and ranged from 18–47 ml·l⁻¹ blood (table 2).

The hepatic venous pressure difference was increased in 5 of 10 patients studied, the highest value being 27 mm Hg (upper normal limit used in our laboratory, 9 mm Hg) (table 2).

Gas exchange and ventilation-perfusion relationships

Minute ventilation was normal to high (table 2) and arterial carbon dioxide tension normal to low (table 3). The measured Pao₂ was 11.4 kPa (85 mm Hg) or less in six patients. This value is used as the lower normal limit at our laboratory for the present age group (see also [14]). Severe hypoaxaemia was seen in patients no. 10, 12 and 13 (table 3). Mixed venous oxygen tension was normal to high in all patients, even in those with more severe arterial hypoaxaemia (table 3).

The technical quality of the derived VA/Q distributions was assessed by summing up the squared differences between measured and predicted retentions of the six infused inert gases (remaining sum of squares, RSS). An indication of acceptable quality of the VA/Q distributions is a RSS of 6 or less in half of the experimental runs [15]. In this study it was 6 or less in 11 of 14 experiments. The calculated distributions...
showed a wide range of $\dot{V}/Q$ abnormalities (table 3, fig 1). Only one patient (patient no 6) had a clearly normal $\dot{V}/Q$ distribution with normal log $\delta Q$, no regions of low $\dot{V}/Q$ and almost no shunt (fig 1). Two patients had normal log $\delta Q$, no perfusion of low $\dot{V}/Q$ regions, but moderately increased shunts (patients no. 3 and 4). Another 2 patients had borderline values on log $\delta Q$ (upper normal limit 0.60) [16], no or minor perfusion of low $\dot{V}/Q$ regions, and no or very low shunt (patients no. 1 and 2). In the remaining nine patients log $\delta Q$ was clearly increased, well beyond the upper normal limit. In all these patients there was considerable perfusion of low $\dot{V}/Q$ regions with extreme values of 9, 10 and 19% of cardiac output in patients no. 10, 12 and 8, respectively. In addition, a significant shunt was seen in 4 of these patients. Thus, a total of 6 patients had shunts which accounted for more than 2% of cardiac output with extreme values of 17 and 20% in patients no. 13 and 12, respectively (table 2).

Knowing the $\dot{V}/Q$ distribution, cardiac output, mixed venous oxygen tension ($P_{O_2}$), the haemoglobin concentration and the slope of the dissociation curve, the arterial $P_{O_2}$ can be calculated by means of an iterative procedure [17, 18]. All parameters were measured or calculated, except for $P_{O_2}$ (a determinant of the slope of the haemoglobin dissociation curve) which was assumed to be 3.8 kPa (28.5 mm Hg), corresponding to the mean of data presented in previous studies [3, 6, 7]. However, the calculated $P_{O_2}$ does not take any diffusion limitation for $O_2$ into account, nor will it be affected by extrapulmonary right to left shunting. A higher calculated than measured $P_{O_2}$ may thus indicate an additional cause of hypoxaemia besides intrapulmonary shunt and $\dot{V}/Q$ mismatch. A comparison of calculated and measured $P_{O_2}$ showed a good correspondence in
Comparison between Pao$_2$ m and Pao$_2$ c. Note the similarity between measured and calculated values in the normoxaemic range, and the increasing difference when Pao$_2$ is reduced. Broken line: identity line; solid line: regression line, for the six hypoxaemic patients (Pao$_2$m <11.5 kPa): Pao$_2$c = 1.68 + 0.85 Pao$_2$m; r = 0.99, p<0.001. (See also text).

Fig. 2. - Comparison between measured arterial oxygen tension (Pao$_2$m) and that calculated from the VA/Q distributions (Pao$_2$c). Note the similarity between measured and calculated values in the normoxaemic range, and the increasing difference when Pao$_2$ is reduced. Broken line: identity line; solid line: regression line, for the six hypoxaemic patients (Pao$_2$m <11.5 kPa): Pao$_2$c = 1.68 + 0.85 Pao$_2$m; r = 0.99, p<0.001. (See also text).

Fig. 3. - Correlation between DLco and the degree of gas exchange abnormality, expressed as the sum of shunt and perfusion of lung regions with low VA/Q ratios (low VA/Q), in percentage of cardiac output. Regression equation: DLco = 77.3 + 1.19 (shunt + low VA/Q); r = 0.87, p<0.001, n=9. A similar but weaker correlation was noted between Kco and the sum of shunt and low VA/Q (not shown in figure; r = 0.75, p<0.01).

Fig. 4. - Correlation between the difference of calculated and measured Pao$_2$ and DLco. Regression equation: calculated Pao$_2$ - measured Pao$_2$ = 1.15 - 0.014 DLco; r = 0.65, p<0.05, n=9.

material can be objected to, since the normal and the hypoxaemic values should not be expected to fit the same regression line).

A significant inverse correlation between the transfer factor of the lung (DLco, Kco), and gas exchange impairment, expressed as the sum of shunt and perfusion of low VA/Q regions was noted (fig. 3). Separate analyses of DLco on shunt and on low VA/Q showed weaker correlations (r = -0.72 and -0.65, respectively). The possible impedance of arterial oxygenation by diffusion impairment was tested by comparing the difference between calculated and measured Pao$_2$ (Pao$_2$c - Pao$_2$m) with DLco. An inverse correlation of borderline significance (r = 0.65, p<0.05) was noted, with increasing Pao$_2$c - Pao$_2$m when DLco was decreased (fig. 4).

No correlations were seen between Pao$_2$ or any VA/Q variable, on the one hand, and spirometry, on the other. Also, no correlation was shown between the degree of portal hypertension, as determined by the hepatic venous pressure gradient, and VA/Q mismatching.

Discussion

The present study demonstrated that patients with non-alcoholic liver cirrhosis have a pulmonary dysfunction which produces both moderate areas of low VA/Q ratios and shunt. These observations are similar to those made in patients with alcoholic cirrhosis [3-7]. However, in these studies, many patients were smokers, making it difficult to exclude smoking as a contributory factor to the VA/Q disturbance. In the present investigation the patients were essentially nonsmokers, and no clinical and almost no functional signs of airway obstruction were seen. Two patients had slightly reduced vital capacities, and one of these (no. 2) had primary biliary cirrhosis which can be associated with fibrosing alveolitis [1]. However, a comprehensive clinical examination and chest X-ray
revealed no signs of fibrosis. Interstitial oedema in
dependent lung regions and cranial displacement of the
diaphragm because of ascites have also been suggested
to cause hypoxaemia in cirrhotic patients, by compressing
small airways and reducing ventilation in these
regions [19, 20]. None of the present patients had
signs of oedema, or a shift of the diaphragm, on chest X-ray.
Moreover, ascites was minor in those who had it, and the two patients with the largest \( V_{A}/Q \) disturbances had no ascites at all, as assessed by clinical
evaluation and ultrasound recording. It can thus be
concluded that there were no clear findings of a
ventilatory impairment, although subtle changes in the
small airways may not be detected by the tests used. In
two previous studies, no correlation was shown
between tests on small airway function and respiratory
and inert gas exchange data [3, 5].

Impaired hypoxic pulmonary vasoconstriction was
proposed by Daoou et al. [21] as a cause of hypoxaemia in patients with cirrhosis. More recently, it was
suggested that cirrhotic patients exhibit progressive
abnormalities of systemic and pulmonary haemodynamics
together with increasing impairment of hypoxic pulmonary vasoreaction, and that this would be
the main cause of the altered pulmonary gas exchange
in cirrhosis [3]. It has also been shown that at least
some cirrhotic patients have dilated microvascular
channels which permit intravenously injected
microspheres of 20–40 \( \mu \) in diameter to pass through the
lung circulation [22–24]. It is tempting therefore to
suggest that such vessels produce shunt, which, together
with loss of hypoxic pulmonary vasoconstriction, cause
the \( V_{A}/Q \) disturbance in liver cirrhosis. It has also been
proposed that a diffusion gradient for oxygen may exist
dilated pulmonary vessels [25], resulting in a "diffusion-perfusion" defect [26]. This theory is supported
by theoretical calculations of oxygen transport in larger
vessels [27]. If present, such a mechanism may be
contributing to reduced \( D_lco \) and hypoxaemia in liver
cirrhosis. The present finding of an inverse correlation
between gas exchange impairment (sum of shunt and
perfusion of regions with low \( V_{A}/Q \) ratios), and
reduction in the transfer factor of the lung (fig. 3) may
indicate that both functional disturbances worsen in
parallel, but it may also suggest that the reduced transfer
factor merely reflects the ventilation-perfusion mismatch.

In an attempt to throw further light on the possibility of a diffusion limitation for oxygen, measured and cal-
culated \( P_{ao} \), have been compared in a number of recent
studies, but varying results have been obtained. Thus,
in studies on patients with no or only moderate hypoxaemia, all gas exchange impairment could be explained
by intrapulmonary shunt and \( V_{A}/Q \) mismatch as evidenced by the corresponding measured \( P_{ao} \) and
calculated \( P_{ao} \) [3–5]. On the other hand, in patients with
more severe hypoxaemia a higher calculated than
measured \( P_{ao} \) indicated additional causes of hypoxaemia
[6, 7]. In the present study we had the opportunity
to examine both normoxaemic and severely hypoxaemic patients. We found a good correspondence between
measured \( P_{ao} \) and calculated \( P_{ao} \), when oxygenation
was normal, but an increasing difference between
calculated and measured values with decreasing \( P_{ao} \) as
shown in figure 2. Thus, with increasing gas exchange
impairment there was an increasing contribution to
hypoxaemia by another factor than intrapulmonary shunt
and \( V_{A}/Q \) mismatch, suggesting the coexistence of
diffusion limitation for \( O_2 \). This was further supported
when we compared the difference between calculated
\( P_{ao} \), and measured \( P_{ao} \) ( \( P_{ao,c} - P_{ao,m} \) ) with the reduction in \( D_lco \). We found a weak correlation with
increasing \( P_{ao,c} - P_{ao,m} \) when \( D_lco \) was decreased (fig.
4). On basis of these observations we propose that
there is a diffusion limitation for both \( CO \) and \( O_2 \) which
in some cases can cause reduced arterial oxygenation
and add to the impaired oxygenation caused by shunt
and \( V_{A}/Q \) mismatch. Since, to our knowledge, there is
no morphological evidence of thickened alveolar-
capillary membranes, we suggest that the higher \( P_{ao,c} \)
than \( P_{ao,m} \) may be an indication of a "diffusion-
perfusion" defect.

A weakness in the analysis of \( P_{ao} \), is that a fixed slope
factor (\( P_{ao} \)) was used instead of constructed individual
haemoglobin dissociation curves. However, the use of
another \( P_{ao} \) in all patients would not alter the correlation,
only cause a parallel shift of the regression line. If, on
the other hand, \( P_{ao} \) varied considerably between the
patients, the effect on the correlation will be less pre-
dictable.

Finally, the discussion on the difference between
calculated and measured \( P_{ao} \), postulates that any vessel
dilation that has not produced intravascular inert gas
gradients. Previous calculations suggest that the inert gases,
as presently used, should not be diffusion limited [28].
Our own data showed a good fit of the derived \( V_{A}/Q \)
distributions to the retention data (small remaining sum
of squares, RSS), and no effect of the weight of the
inert gases on their elimination could be disclosed. This
would have been the case if there was a diffusion limi-
tation for the inert gases. Thus, a heavy inert gas, such
as enflurane, showed no greater error in fitting with the
calculated retention curve (mean error, data from all
patients: 0.24) than a light gas like ethane (mean error:
0.30; no significant difference between the two gases
(paired t-test)).

In conclusion, patients with non-alcoholic liver
cirrhosis suffer from \( V_{A}/Q \) mismatch and shunt similar
to that shown in alcoholic cirrhosis. Moreover, with
more prominent gas exchange impairment, the effect of
an additional factor, probably diffusion limitation, for
oxygen, emerges. The findings can fit with pulmonary
vascular relaxation and dilated microvascular
channels.

References

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RÉSUMÉ: Les relations ventilation-perfusion ont été étudiées chez des patients atteints de cirrhose hépatique non éthylique. La spirométrie est fondamentalement normale, mais le facteur de transfert pulmonaire (Dlco) est réduit en moyenne de 34% des valeurs prédites. La tension artérielle en oxygène (Pao₂) a des valeurs qui s'étalent depuis la normale jusqu'à 6,9 kPa. Différents degrés d'anomalies du rapport ventilation-perfusion (V/Q) ont été observés au moyen de la technique d'élimination de gaz inertes multiples: il s'agit d'une accentuation de la dispersion de la distribution de la perfusion log snr o.00; extrême 0.32-1.71: limite supérieure de la normale, 0.60) et de la présence de deux types de régions: celles avec une ratio V/Q basse (entre 0.1 et 0.005) (moyenne 4.1%, extrême 0-18.8%) et celles avec shunt (ratio V/Q inférieure à 0.005) (moyenne 3.9%; extrême 0-19.8%). Chez les patients nonoxémiques, il y a une similitude étroite entre la Pao₂ mesurée et calculée. Par contre, chez les sujets hypoxémiques, les valeurs calculées sont supérieures aux valeurs mesurées. La différence entre les valeurs calculées et mesurées de Pao₂ est en corrélation inverse avec la Dlco (r=0.65, p<0.05). L'on a noté également une corrélation inverse entre Dlco et la somme des régions à shunt et à rapport V/Q bas (r=0.87, p<0.001). L'on conclut que l'hypoxémie, chez les patients atteints d'une cirrhose hépatique non éthylique, peut être expliquée par le shunt intra-pulmonaire et par la non congruence de la ventilation et de la perfusion, et peut-être par un défaut diffusion-perfusion chez les patients dont l'atteinte des échanges gazeux est plus sèvere.