The Lung in Trauma

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SUMMARY

The lung, as a target organ in shock, is discussed. So-called 'shock lung' is characterized by pulmonary oedema and resultant hypoxaemia. This pulmonary oedema has a multifactoral origin and was present in all my 22 cases. Arterial pO₂ values averaged 56 mmHg, with a low of 35 mmHg and a high of 68 mmHg. Hyperventilation and hypocarbia accompany this hypoxaemia, initially; all three pulmonary causes of hypoxaemia may play a role in the production of the hypoxaemia. Inhalation of 100% oxygen, however, does not offer complete relief, indicating primarily a shunt effect. The significant points in our efforts to solve this problem are documented.


Circulatory resuscitation in traumatic shock reached a zenith during the last decade. With the realization of the importance of the interstitial volume, our morbidity figures showed an improvement beyond all expectations. Previously, many patients succumbed to so-called irreversible shock, a diagnosis made less frequently nowadays. Pulmonary insufficiency has become more prevalent, however, and we have come to see the picture shown in Fig. 1 more often. This shows the typical 'bat's wing' appearance of pulmonary oedema. These patients develop a progressive hypoxaemia, with an initial hypocarbia. Although this condition is reversible, it sometimes resists all attempts at treatment.

In 1926 Haldane said 'The lack of oxygen not only stops the machine but also wrecks the machinery'. The earlier the distress of the patient is recognized, the better the prognosis.

I wish to analyse my 22 cases in the light of early recognition and physiological approach to treatment.

METHODS AND CLINICAL MATERIAL

Twenty-two cases, showing typical changes, have been selected. The nature of the initial trauma is shown in Table I. Resuscitation of the low flow state was done according to Shires et al. and under guidance of central venous pressure, urinary output, packed cell volume determinations, and cardiac output, roughly estimated by pulse pressure.

After resuscitation, all patients were submitted to intensive care and biochemical status was controlled periodically. This included acid-base, arterial and venous pO₂, serum electrolytes, serum albumin and lactate determinations. Cardiac output was computed in 2 patients.

With slight degrees of hypoxaemia, test inhalation of 100% oxygen was done. If lowering of the alveolar-arterial pO₂ became evident, and pulse rate decreased, the patients were given a nose catheter or BLB mask with the percentage of inhaled oxygen controlled by arterial pO₂ determinations. Arterial pO₂ above 75 mmHg was aimed at (normal for Pretoria is approximately 84 mmHg).

With progressive alveolar-arterial pO₂ differences, mechanical ventilation with oxygen-enriched air via an intratracheal tube was considered, to obtain arterial pO₂ of 70-90 mmHg. When long-term (more than 48 hours) ventilation was envisaged, tracheostomy was performed. CO₂ was added to obtain an arterial pCO₂ of 30-35 mmHg.

Increased tidal volumes, with normal rates, were used in preference to expiratory positive pressure, or resistance to expiration. This method is not as detrimental to venous return because the rate, being kept within normal limits, means intrathoracic pressure is not influenced to such a great extent.

Reduction of pulmonary oedema was attempted by increasing plasma oncotic pressure (albumin) and by the use of diuretics.
TABLE I. LIST OF INJURIES IN 22 PATIENTS

<table>
<thead>
<tr>
<th>Injury Description</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flail chest, intra-abdominal injury and fractures</td>
<td>10</td>
</tr>
<tr>
<td>Multiple fractures and intra-abdominal injury</td>
<td>6</td>
</tr>
<tr>
<td>Septic shock after colon or gynaecological injury</td>
<td>4</td>
</tr>
<tr>
<td>Major surgery, e.g. ruptured abdominal aorta aneurysm</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>22</td>
</tr>
</tbody>
</table>

RESULTS

Average $pO_2$ in the 22 selected patients was 56 mmHg. Fig. 2 shows the average arterial, as compared with alveolar, $pO_2$ values in 7 of these patients who did not survive. The most prevalent symptoms and signs present in all 22 patients were tachycardia, hypoxaemia, hyperventilation, alkalosis, excess resuscitative demands and pretrauma respiratory disease. Cyanosis due to hypoxia was not always obvious on clinical examination but tachycardia, unexplained by other factors, was the most persistent finding. A positive response to 30 minutes of 100% oxygen inhalation, indicated by slowing of heart rate, verified the suspicion of hypoxaemia, while blood gas analysis confirmed it.

The pulse rate response to oxygen inhalation is shown in Fig. 3. This hypoxia is possibly the cause of the persistent hyperventilation seen in patients with pulmonary insufficiency, although it can also be caused by pain, excitement, micro-emboli from the site of injury, as well as by acid-base changes in the cerebrospinal fluid.

Contrary to what one would expect in hypoxia, these patients all presented with a mixed respiratory and metabolic alkalosis. This can be explained by, firstly, spontaneous hyperventilation; secondly, by the lack of bicarbonate excretion, either through a volume deficit or a post-traumatic aldosteronism. The excess bicarbonate may be due to sodium citrate metabolism from transfused bank blood. Thirdly, from continuous suction via a stomach tube.

Fourteen of our patients presented with pretraumatic limitation of respiratory reserve. These patients pose the same problem after trauma as they do after routine surgery.

Postmortem Appearance

Macroscopically the lungs are larger and heavier than normal. Typically, a non-compliant, stiff lung is seen. This is due to pulmonary oedema which can be proved on microscopic examination. The gas-blood barrier is widened sometimes to over 300%. Hyaline and fibrinous deposits are seen in the alveoli. Atelectasis is not prominent. On electron microscopy both pneumocytes I and II are prominent and increased in numbers.

These findings are not pathognomonic of the lung in trauma; it has been seen in rats by Kistler et al. after concentrated oxygen inhalation. Most of the patients on whom postmortem examinations have been done, had been exposed to high oxygen concentrations. The fact that all these patients had been submitted to vigorous mechanical ventilation, designed to combat atelectasis, may be the explanation of the absence of this condition on microscopic section.

DISCUSSION

Pulmonary oedema seems to play a major role in the causation of post-traumatic pulmonary insufficiency. This is borne out in Roentgen studies and postmortem findings in our cases as well as those of other workers. All three pulmonary causes of hypoxaemia can be explained by oedema, viz. limitation of diffusion, uneven ventilation in relation to blood flow, and physiological shunt. The first two become insignificant by inhalation of 100% oxygen and the entire alveolar-arterial oxygen difference is then due to shunting. The extreme widening of the gas-blood barrier added to increased flow of acidotic,
desaturated blood may indicate that the hypoxaemia is due in part to limitation of diffusion.

It seems likely that, in shock, the pliancy of the lung varies from one area to another due to congestion in dependent parts. With less ventilation going to these parts, desaturated blood will be mixed with arterial blood. By pressure on and filling of alveoli, pulmonary oedema can also produce non-ventilated but perfused areas in the lung. Pulmonary oedema is due to simple diffusion of fluid out of the capillaries. This is caused by either raised capillary pressure or loss of oncotic pressure as well as loss of capillary function through stretching of pores.

By infusing fractionated plasma factors from septic dogs into healthy animals, Clowes et al. produced severe lung lesions with fractions of molecular weight of 1000-10,000. Pulmonary vascular pressure was raised by 50%. This group will contain peptides, such as kinins and fibrinopeptides, both of which are thought to raise resistance in the postcapillary area.

Massive transfusions, besides raising intravascular pressure, also dilute plasma proteins, with loss of oncotic pressure.

Some workers also found raised interstitial albumin of lung tissue, indicating the possibility of altered capillary function.

Although very little evidence of atelectasis is found at postmortem examination, it plays a significant role in the production of hypoxaemia in injured patients. The most common cause is retention of secretions. Two additional causes of atelectasis are loss of normal breathing patterns and lack of surfactant activity of the phospholipids in the alveoli.

Surfactant activity in shock has been extensively examined. Pneumocyte II, which is presumably responsible for surfactant turnover, is changed in shock through altered pulmonary blood flow. A 'dry lung' is essential if accelerated degradation of surfactant is to be prevented. Why the pneumocyte II changes in shock take place is not clear. If it receives its oxygen supply from the pulmonary or bronchial system, the change will be through the impact of the low flow state. If, however, it receives oxygen directly from the alveolus, the role of these cells in producing atelectasis becomes of secondary importance.

Finally, aspiration of stomach contents is common in critically ill patients and can aggravate or initiate this lung condition by introduction of very acid stomach contents or septic material into the bronchial tree.

Our experience supports the findings of other workers in this field. Pertinent points in our series are the following: obese patients and heavy tobacco smokers are predisposed to pulmonary insufficiency after trauma; pulmonary oedema, of multifactorial origin, with possibly atelectasis, is the main cause of the hypoxaemia; contamination of the tracheobronchial tree with external noxious factors must be guarded against; and serial blood gas determinations are essential for early diagnosis.

REFERENCES