Asymptomatic Pulmonary Atelectasis in Drug Addicts*

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DURING ROUTINE CHEST X-RAY EXAMINATIONS OF MANY DRUG ADDICTS ADMITTED TO THE HOSPITAL FOR THE PURPOSE OF DETOXIFICATION, WE RECENTLY OBSERVED A NUMBER OF PATIENTS WHO DEMONSTRATED PLATE-LIKE ATELECTASIS WITHOUT ANY CLINICAL SYMPTOMS. THE PURPOSE OF THIS PAPER IS TO REPORT THESE FINDINGS AND TO DISCUSS THE POSSIBLE PATHOPHYSIOLOGIC MECHANISM INVOLVED.

Our group comprised ten men from 34 to 62 years of age and four women between 39 and 62 (Table 1). None presented symptoms referable to the bronchopulmonary tree, inasmuch as there was neither cough, fever, hemoptysis nor dyspnea. Characteristically, there was no chest pain or previous history of lung disease. The only common factor was that they were all mainliners who practiced a daily intravenous injection of varying amounts of heroin. The amount consumed ranged anywhere from 3 to 12 bags per day, each bag containing approximately one grain of heroin. In addition, several patients also took other drugs—namely secobarbital (Seconal), pentobarbital (Nembutal), sodium secobarbital and sodium amobarbital (Tuinal) and alcohol (Table 1). They did not employ an aseptic technique, and the material used was in powder form, which probably contained the main substance (heroin) mixed with some inert material to make up bulk. Externally they appeared apathetic and "foggy" presenting the evidence of repeated venous punctures in the upper extremities and at times in the lower, with some areas

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Table 1

<table>
<thead>
<tr>
<th>No.</th>
<th>Name</th>
<th>Sex</th>
<th>Age</th>
<th>Quantity of Heroin Daily</th>
<th>Addict to Heroin</th>
<th>Other Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>W.G.</td>
<td>M</td>
<td>62</td>
<td>5-6 bags</td>
<td>9 years</td>
<td>7-8 sodium secobarbital and sodium amobarbital</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(Tuinal) capsules.</td>
</tr>
<tr>
<td>2</td>
<td>J.G.</td>
<td>M</td>
<td>39</td>
<td>10-12 bags</td>
<td>12 years</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>M.G.</td>
<td>F</td>
<td>62</td>
<td>6-8 bags</td>
<td>30 years</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>R.R.</td>
<td>M</td>
<td>37</td>
<td>4 bags</td>
<td>12 years</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>J.B.</td>
<td>M</td>
<td>40</td>
<td>3-5 bags</td>
<td>20 years</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>B.G.</td>
<td>F</td>
<td>39</td>
<td>3-4 bags</td>
<td>10 years</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>J.N.</td>
<td>M</td>
<td>39</td>
<td>3 bags</td>
<td>20 years</td>
<td>Alcohol (excessive amounts), sodium secobarbital</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>and sodium amobarbital (Tuinal) or secobarbital</td>
</tr>
<tr>
<td>8</td>
<td>H.W.</td>
<td>M</td>
<td>40</td>
<td>4-5 bags</td>
<td>10 years</td>
<td>Alcohol (excessive amounts).</td>
</tr>
<tr>
<td>9</td>
<td>E.H.</td>
<td>F</td>
<td>45</td>
<td>5 bags</td>
<td>28 years</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>W.S.</td>
<td>M</td>
<td>29</td>
<td>8 bags</td>
<td>2 years</td>
<td>Alcohol (excessive amounts).</td>
</tr>
<tr>
<td>11</td>
<td>H.M.</td>
<td>M</td>
<td>52</td>
<td>3 bags</td>
<td>19 years</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>R.W.</td>
<td>F</td>
<td>58</td>
<td>4-5 bags</td>
<td>34 years</td>
<td>Pentobarbital (Nembutal) 10-12 capsules daily.</td>
</tr>
<tr>
<td>13</td>
<td>A.C.</td>
<td>M</td>
<td>34</td>
<td>4-6 bags</td>
<td>1 year</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>H.L.</td>
<td>M</td>
<td>38</td>
<td>4 bags</td>
<td>18 years</td>
<td></td>
</tr>
</tbody>
</table>
PULMONARY ATELECTASIS IN DRUG ADDICTS

of old phlebitis. Two demonstrated the puffy-hand syndrome, and one also had puffy feet, in all probability the result of localized thrombophlebitis. An x-ray film of the chest taken on admission, before the beginning of the detoxification program, demonstrated horizontal linear densities in the lower lung fields (Fig. 1A to 4B). These lines were situated about 1-3 cm above the diaphragm and were either unilateral or bilateral and both single and multiple. They appeared typical of the plate-like atelectasis described by Fleischner.

DISCUSSION

According to Fleischner and his co-workers, linear shadows seen in the lung on x-ray examination may be due to three conditions—namely: 1) interlobar pleuritis, 2) foci of atelectasis or 3) healed pulmonary infarction. The first cause may easily be ruled out here, since the location of the shadows in our patients was not anatomically in the area of the interlobar fissures. The second and third etiologic possibilities, however, require consideration and speculation.

There is some confusion as to the exact meaning of atelectasis. By definition, atelectasis denotes incomplete expansion of the lung parenchyma and a shrinkage of alveolar spaces. It implies a functionless lung,

![Figure 1A (upper): Bilateral atelectasis in a 62-year-old man addict (PA view). Figure 1B (lower): Close-up view of thickened linear atelectasis in left base. Note the location at the base parallel to the diaphragm.](image1)

![Figure 2A (upper): Lateral view of chest demonstrating linear atelectasis at the base. Figure 2B (lower): Close-up view of same patient demonstrating multiple linear atelectasis.](image2)
which may be either completely or only partially airless. Our patients demonstrated partial atelectasis, thus indicating airlessness in only a small segment of the lung.

Atelectasis is always secondary to other pathologic processes which interfere with respiration. Often it constitutes an inflammatory process in the abdomen below the diaphragm—especially in the subphrenic region—which interferes with the diaphragmatic motion and produces decreased respiration on the side involved. At times, the appearance of a plate-like focus of atelectasis is the first evidence of disease below the diaphragm and may give a clue as to side involved. Pain on respiration following abdominal surgery, chest-wall trauma, pleurisy or pulmonary infarction may diminish breathing and thus decrease ventilation of the affected side, causing retention of bronchial secretions with obstruction and eventual atelectasis.

Atelectasis is also frequently associated with infections of the respiratory tree, bronchiectasis and occasionally tuberculosis. In such instances it usually follows obstruction of secondary or tertiary bronchi by mucous plugs. Repeated episodes of bronchial asthma with markedly increased secretions of tenacious material can also be responsible
for plugging of some bronchi, resulting in atelectasis. Certain lesions of the central nervous system, with direct involvement of the respiratory center situated in the reticular formation of the medulla, may depress breathing, permitting the retention of secretions, with secondary obstruction and atelectasis. None of the above factors, with the possible exception of undetected pre-existing infections, seemed to apply to the patients under our supervision, however.

It is well known that morphine and its derivatives, when used in large doses beyond the therapeutic range, have a depressant effect on respiration both centrally and locally. Heroin, the drug employed by the addicts in this group, is a morphine derivative which is hydrolized to morphine and therefore acts in the same manner.

Egbert and Bendixen demonstrated that when morphine was given in the therapeutic doses to postoperative patients, it did not grossly depress ventilation or the exchange of gases, but did cause a diminution in deep breathing by depressing the reflex urge to maintain normal pulmonary mechanics. They felt that this may explain the increased incidence of postoperative atelectasis following the consumption of large doses of morphine.

In the lungs of dogs, ventilation without periodic breathing leads to a decrease in pulmonary distensibility presumably caused in part by atelectasis, but this trend is reversed by a deep breath. In the normal human lung, practically all alveoli are patent, even during rest, and are ventilated by a quiet tidal breath. Man, when awake, automatically takes a deep breath about eight to ten times an hour (more than three times the average tidal time). It is assumed that alveolar collapse or atelectasis is prevented by the occasional deep breath. Ferris and Pollard found a reduction in compliance—i.e., hypoventilation in individuals who consciously refrained from sighing. Egbert and Bendixen discovered that patients who received morphine postoperatively for the relief of pain failed to take spontaneous deep breaths for a time, and they assumed that such respiration at constant volume is more likely to result in atelectasis. Hence, since morphine can depress pulmonary mechanics by reducing the sighing normally present and can decrease the excursions of the chest-wall, causing hypoventilation, we believe that heroin, the drug employed by our group of addicts was capable of having a similar effect.

The symptoms of acute atelectasis vary from none whatever, if the region involved is small, to severe dyspnea, tachypnea, cyanosis and cough, in cases where a large part of the lung is affected. The signs, when present in massive atelectasis are chiefly dullness to flatness on percussion, suppression of breath sounds and displacement of the diaphragm, chest wall and mediastinal structures toward the lesion. With re-aeration of the lung, these findings are reversible. In segmental and linear atelectasis, the physical signs are minimal and hardly detectable as was noted in our patients.

It has recently been shown that atelectasis develops because of an alteration in alveolar surface tension. Fractionation of washed lung extracts has yielded a material (surfactant) that seems to be liberated by the cells in the alveolar walls. This material is a phospholipid of the lecithin type and acts as an antialveolar factor. Adult human atelectasis is basically a mechanical phenomenon, with secondary deficiency of an antialveolar surfactant, often resulting in a vicious circle of self-perpetuating alveolar collapse difficult to reverse. This very likely explains the persistence of the linear shadows referred to above.

Fleischner and his co-workers have shown that the end stage of a pulmonary infarct may present itself as a linear shadow at the base of the lung, closely resembling the density of plate-like atelectasis. It is interesting to note that many addicts choose to indulge the habit in groups and congregate in congested quarters which necessitate sitting on the floor with feet crossed for several hours. It is conceivable that since the lower extremities are compressed, thrombophlebitis may have developed here in
some cases with subsequent pulmonary embolization. Kent and Reid have reported the occurrence of pulmonary emboli in a group of healthy young men in the Navy, who had been in a cramped position for many hours, either in a submarine or a long bus ride. Although the patients they described complained of chest pain and had some fever, this does not negate the possibility of a similar condition in our group. While on continuous drug injection, addicts are insensitive to pain and feel quite euphoric. Since the bags of narcotics contain impurities, it is also conceivable that some of this material may be lodged in the pulmonary vascular tree, causing pulmonary embolization. The linear shadows seen in our patients are not too dissimilar to those observed in the end-healed stage of pulmonary infarction.

Septal lines, commonly referred to as Kerley lines, may at times resemble linear atelectasis. However, these lines, although situated at the bases of the lungs, are much finer in appearance. They are most commonly bilateral and are located above the lateral costophrenic angles. They denote increased pulmonary venous pressure in the interlobular septa and are associated with a number of pathologic states predominantly: 1) chronic left heart failure, 2) fibrotic pulmonary disease and 3) lymphangitic involvement secondary to neoplasm. However, in our cases, none of the above existed, hence Kerley lines, as the etiology of the linear shadows described above, need not be considered.

SUMMARY
Observations on 14 drug addicts revealed that under the influence of heroin, they presented signs of atelectasis on X-ray examination without any detectable symptom or clinical manifestation. This finding may very likely be explained on the basis of either diminished depth of respiration and decreased sighing or silent pulmonary embolization. We tend to favor the former because the physiologic effect of the drug employed results in diminished breathing and hypoventilation, both of which are conducive to the production of atelectasis. The latter concept may also prevail, but proof is lacking. Further study with such tools as angiography and lung scanning are indicated for more definitive answers.

RESUMEN
La observación de 14 narcómanos ha revelado que bajo la influencia de la heroína presentaban signos radiológicos de atelectasia sin expresión clínica ni sintomática. Esto puede muy bien ser explicado por la disminución en la profundidad de los movimientos respiratorios o por embolia pulmonar silente. Nos inclinamos a lo primero, ya que el efecto fisiológico de la droga empleada resulta en disminución de la amplitud de los movimientos respiratorios e hipoventilación, ambos efectos conducivos a la atelectasia.

La segunda posibilidad es factible, pero no ha sido probada.

Nuevos estudios mediante la angiografía y la exploración con radioisótopos están indicados para llegar a comprobaciones definitivas.

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REFERENCES
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