

BLOOD GASOMETRY AFTER DOSED EFFORT IN SUBJECTS EXPOSED TO ASBESTOS

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ABSTRACT

The authors examined 125 workers occupationally exposed to asbestos. Fifteen workers developed radiological signs of asbestosis with a functional respiratory impairment.

In the remaining 110 subjects, all with more than 15 years of exposure but without radiological signs of fibrosis, the authors carried out the usual functional respiratory tests. In addition, blood gasometry test was performed before and after work (120 watts × 10 minutes).

A decrease in PaO₂ which persisted or appeared under effort (hypoxaemia, often connected with hypocapnia) was a precious sign of a complex functional disorder allowing in the case of a long exposure a preradiological diagnosis of asbestosis.

Within an epidemiological study conducted on behalf of the Sanitary Assessorship of the Campania Region among workers of a cement-asbestos manufacture exposed to asbestos by inhalation we performed blood gasometry before and after work.

The purpose of the study was to prove that hypoxaemia which occurs through the stress caused by muscular work, is a sign of functional respiratory impairment which precedes radiological alterations typical of fibrosis.

SUBJECTS AND METHOD

The subjects taken in the study were 125 workers exposed to the inhalation of cement-amiant dust. They were hospitalized and clinically examined.

Fifteen subjects showed radiological signs of fibrosis (14 of a slight degree and one of a more advanced type). The remaining 110 showed no radiological signs of fibrosis and/or pleural alterations.

The following respiratory function tests were carried out in all subjects:

- 1) Spirography (Expirograph; FRC computer Godart);
- 2) CO-transfer, in stable equilibrium, according to Filley's method modified by Bates, through Godart Mark II diffusion test, both at rest, and at work - 100 watts;

3) Gas-analysis (PaO_2 , PaCO_2 , pH) basic acid equilibrium by Radiometer;
 4) Haemoglobin dosage in the arterial blood (g Hb/100 ml: HbO_2 /100 ml, HbCO /100 ml) by Co-oximeter IL 182, at rest, and after a charge of 120 watts for 10 minutes at the Dargatz cycloergometer.

All patients were also submitted to the usual clinical and laboratory examinations, besides radiography, so that a possible pathological association and/or fibrosis of a different kind could be discovered.

RESULTS

The results of the investigations suggest that the functional syndrome of asbestosis was present in a characteristic sequence: a block of alveolar capillary perfusion, hypoxaemia, pulmonary hyperventilation and hypocapnia. The syndrome was observed in 15 cases of asbestosis which were radiologically confirmed (Fig. 1).

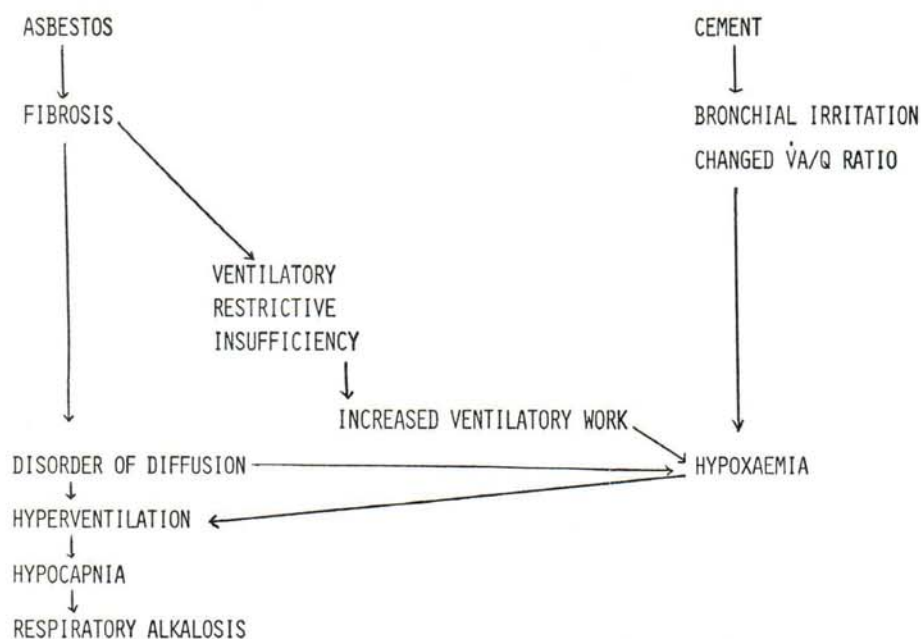


FIG. 1 - Scheme of functional alterations for exposure to cement-asbestos dust.

The period of employment and exposure to asbestos of 107 subjects was longer than 15 years (Table 1).

Spirography was normal in 65 out of 110 subjects, while a restrictive ventilatory failure was found in 29 (26.4%) and mixed (obstructive-restrictive)

ventilatory insufficiency in 15 (13.6%) subjects. Ventilatory obstructive insufficiency was found only in one subject. In five subjects with mixed failure the emphysema-index CVR/CPT was above 35.

Blood gasometry at rest showed a decrease of PaO₂ in 25 subjects. Of these 15 regained normal PaO₂ values during dosed work and only in 15 subjects the values were reduced even at effort.

TABLE 1
Percentage of subjects with impaired transfer of carbon monoxide in the lung.

| Group of workers | T _{CO} :T _{CO/V} | At rest | During work (100 watts) |
|--|------------------------------------|---------|----------------------------|
| Workers exposed to asbestos (N = 110) | Normal (\geq theoretic) | 66.8 | 68.0 |
| | Decreased ($<$ theoretic) | 33.2 | 32.0 |
| Workers exposed to asbestos with hypoxaemia (N = 23) | Normal | 30 | 0 |
| | Decreased ($<$ theoretic) | 70 | 100.0 |

Among 85 subjects with normal blood gasometry at rest, 13 presented a pathological decrease at effort.

On the whole 23 subjects (21%) showed hypoxaemia at effort, with a mean value of PaO₂ 80 = 7.2 mm Hg; in 87 subjects with normal PaO₂ the mean value of PaO₂ was 98 ± 6.4 mm Hg. Among 23 subjects with hypoxaemia six showed hypocapnia at rest (PaCO₂ 36 mm Hg) and 10 (43%) showed incipient hypocapnia or manifest hypocapnia at effort (mean value of PaCO₂ was 34.1 ± 1.3 mm Hg). Among these 10 subjects only three showed also respiratory alkalosis.

Of the 23 subjects with hypoxaemia after effort 70% had a reduced CO-transfer at rest; at effort a constant decrease was observed in all subjects (Table 1).

One third of all workers showed a decrease in CO-transfer confirming the results obtained in our previous research in workers exposed to amiant.

The frequency of hypoxaemia was connected with the length of work: in most workers hypoxaemia appeared after 21 years of work. No hypoxaemia was found among the subjects with less than 16 years of service (Table 2).

Spirography showed insufficient respiration in 45 workers. However, hypoxaemia was not always accompanied with ventilatory failure. The transfer of CO was reduced in 32% of all workers.

Not all the subjects with a decreased CO-transfer were affected by hypoxaemia. On the contrary, all the patients with hypoxaemia showed a pronounced decrease of CO-transfer both at rest and/or, at least, after effort.

TABLE 2
Hypoxaemia and length of exposure.

| Years of exposure | No of cases | Hypoxaemia by effort | |
|-------------------|-------------|----------------------|------|
| | | N | % |
| 15 | 7 | 0 | 0 |
| 16-20 | 37 | 7 | 8.9 |
| 21-25 | 50 | 12 | 24.0 |
| 25-30 | 16 | 4 | 25.0 |
| Total | 110 | 23 | 20.9 |

The smallest percentage of pathological results was related to the O_2 tension by effort (decrease in 21% of the workers).

However, hypoxaemia caused by diffusion disorders was not compensated, but it increased during work.

Hypoxaemia was therefore, a characteristic consequence of the alveolar capillary block caused by asbestosis. It could be explained only if connected with fibrosis which was not yet manifest and thus unrevealed by radiography. The functional changes were interpreted almost exclusively as caused by amiant fibres, as cement with the traces of silica caused only an irritation of the respiratory tract, contributing to some extent to hypoxaemia (see the scheme in Fig. 1). In our opinion the determination of PaO_2 after dosed effort (120 watts \times 10 minutes) might indicate hypoxaemia in workers exposed to asbestos dust, which is the more significant the more it is associated with hypocapnia.