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SMANN, K. Ersatz kiesel-säurehaltiger Stoffe durch kiesel-säurefreie zur Bekämpfung der Silikose. [Substitution of Silica-Containing Materials by Silica-Free Materials in the Combat against Silicosis] *Zent. f. Arbeitsmed. u. Arbeitsschutz*. 1956, July, v. 6, No. 7, 167-71. [16 refs.]

Germany three-fourths of the compensation pay for occupational diseases is on account of pneumoconiosis; in the German coal mines the total loss from this disease is 4 times that for accidents. Practical measures for protection against dust have achieved only limited success and the substitution of materials free from silica gives the best prospect of eradicating the danger. Examples are given. During the period 1946-49 among 470 sandblasters in the iron and steel industry in Southern Germany 1.4 per cent. developed severe pneumoconiosis annually; silica-free materials were substituted, e.g. steel-shot and artificial abrasive wheels. Moulding sand can be removed from castings under exhaust draught and the sand is cleaned before re-use.

Reference is made to machines for cleaning castings which are economical and technically efficient as well as hygienic. Where steel-shot is unsuitable for cleaning and polishing, as with light metals, proprietary materials are in use in Germany. Hydroblast, at 100 atmospheres pressure, can be used to clean castings and cores with recovery of 80-90 per cent. of the sand. Bentonite is referred to as a substitute for quartz in moulding sand; it is said to give improved technical results and is easily removed from the metal. Parting powders without silica are mentioned. Reference is made to the use of artificial grinding wheels in place of grindstones in Southern Germany. Warning is given against the use of quartz as the binding material of artificial grinding wheels. It was officially stated in 1955 to reach as much as 30 per cent. of uncombined quartz in some cases; it is essential to keep the silica content to the lowest possible minimum; the same principle applies in the grinding of jewels and ornamental stones.

Reference is made to an Order of 1938 prohibiting the use of quartz sands as abrasive in grinding of steel; this has led to the use of emery and silicon carbide which can be graded to give good results. The higher cost of artificial abrasives is offset by higher working speed and the same applies to the glass industry. Sandblasting in the glass industry is prohibited for all females and for males under 18; workers over 18 should not be employed on this work for more than 2 years; silicon carbide can be used for the process. In the ceramic industry the risk of disease can be reduced by the use of powders free from, or low in, quartz for dusting unfired ware. In the refractories industry magnesite can replace silica. In the shoe industry a risk of silicosis arises from polishing with machines using sand-paper with high proportions of free silica in the abrasive; one worker sustained 100 per cent. disablement from disease after 12 years work on this process; another developed silico-tuberculosis after 14 years of work. Emery or carborundum should be used as the abrasive.

Similar practice should be observed in the wood-working industry. For stone-dusting in coal mines slate or chalk should be used. E. L. Middleton

ROBINSON, H. The Ventilation of the Battery Containers of Storage Battery Locomotives. *Safety in Mines Research Establishment*. [Portobello St., Sheffield.] *Research Rept.* No. 122. 1956, Feb., 22 mimeographed pp., 6 figs. & 1 pl. [2a. 3d.]

WILSON, M. E. & HARRON, D. C. Development of Mechanical Breather for Evaluation of Respiratory Equipment. *Arch. Indust. Health*. Chicago. 1956, June, v. 13, No. 6, Sect. 1, 561-6, 6 figs.

PATIGNY, J. Vision et visibilité dans les mines. [Vision and Visibility in Mines] *Arch. Belges Méd. Sociale, Hyg., Méd. du Travail et Méd. Légale*. 1956, Mar., v. 14, No. 3, 97-114, 7 figs.

The author discusses (1) the causes of poor visibility in mines—low intensity of illumination, poor reflectivity of surfaces and feeble contrast—and (2) the effects of adaptation to low levels of illumination and of glare.

He describes some tests in which the effects of illumination and glare on visibility were studied. As in other situations, the visibility in a mine with a given intensity of illumination depends on the nature of the visual task and on the quality of the lighting. Intensities of the order of only 0.5-1 lux do not make relatively detailed vision impossible if the contrast is sufficient, and especially if the background is light. With an intensity of only 0.1 lux one can see untarnished metallic objects that are not too small. With a black background, however, as, for example, at a coal-face, vision of detail is lost with illumination of about 0.5 lux.

Types of mine lighting are discussed briefly, and the author favours the cap-lamp, which illuminates the working surface. There is, however, the difficulty that a miner working within 3 or 4 yards of another may be exposed to glare from the other's cap-lamp. This effect can be reduced considerably by using lamps with matt reflectors which, at a distance, give a much more feeble light than is projected by polished reflectors. Thomas Bedford

ARLBERG, G. & HANSSON, C. J. Asbestos. En för Sverige ny yrkessjukdom. [Asbestosis. An Industrial Disease which is New for Sweden] *Svenska Läkartidningen*. 1956, v. 53, No. 21, 1376-83, 2 figs.

In 1953 a 63-year-old workman, whose employment had entailed contact with asbestos, complained of growing dyspnoea on exertion during the past year. He was found to suffer from asbestosis in the third stage, with numerous asbestos bodies in his sputum. Under treatment in hospital he showed some diminution of symptoms, and his vital capacity rose from 1,700 to 2,900 ml. Two months after his discharge from hospital he returned to it with pneumonia which proved refractory to treatment. This case led to a

scrutiny of the 60 men employed on asbestos work. Eight of them, between the ages of 43 and 63 years and with a history of exposure to asbestos dust for 5-17 years, were found to be suffering from first-stage or second-stage asbestosis. None of them, however, presented symptoms of asbestosis. The notification of the first case to the public health authorities was followed up by an application for the extension to asbestosis of the prophylactic measures already in force against silicosis. In Sweden all workers whose employment entails contact with asbestos are to be subjected to periodic radiological examinations. Protective masks are to be used during work entailing the slightest risk of inhalation of asbestos dust. Other measures, providing for better ventilation of workshops, are also to be adopted in view of the growing importance of the asbestos industry. During the 5-year period before the last war Sweden imported only 17.6 tons of asbestos, whereas during a similar period after the war the corresponding figure was 46.5 tons.

Claude Lillingston

BARTH, G., FRICK, W. & SCHNEIDERMANDEL, H. Die Aluminiumlunge. Verlaufsbeobachtungen und Neuerkrankungen in der Nachkriegszeit. [Pneumoconiosis due to Aluminium Dust] *Deut. med. Woch.* 1956, July 13, v. 81, No. 28, 1115-19, 6 figs. on pl. [25 refs.]

During the second world war pneumoconiosis due to aluminium dust was not uncommon and because of its bad prognosis was regarded as a serious industrial hazard. It was hoped that the better conditions of post-war work would eradicate the risk, but several new cases have been found in the last few years. Aluminium dust is manufactured in 2 forms, as stearin-coated polished particles used in paints and for other purposes and as uncoated particles known as pyro-dust which is required for making torpedo fuses and signal flares. Workers in the industry are exposed to clouds of dust since adequate protection by artificial ventilation methods has not been possible. Clinically, it was found that the pneumoconiosis in pre-war years when stearin-coated dust was mainly produced was slower in its onset and course than in the war years when pyro-dust was the chief aluminium dust to be manufactured. Pyro-dust is used also in peace time in fireworks. Some records are included of 13 workers who had worked for various periods of time before, during and after the war. There were 10 deaths in this group. Case histories of 3 of these workers are given in detail. Pathological changes were seen chiefly in the upper lobes of the lungs and these are described. In only 1 case was there evidence of tuberculous infection.

M. E. Deinfeld

SCHILLING, R. S. F. Byssinosis in Cotton and other Textile Workers. *Lancet*, 1956, Aug. 11 & 18, 261-5; 319-25, 11 figs. [Numerous refs.]

This article comprises the 2 Milroy lectures delivered before the Royal College of Physicians of London in February 1956. In the first lecture the author traced

the history of the disease from the time of its recognition in Lancashire in 1831; it was then called cotton spinners' phthisis. It is still a serious problem in the cotton industry in Lancashire. High mortality rates from respiratory diseases led to enquiries into their cause. In 1909 COLLIS examined a number of strippers and grinders of the carding engines to whom high mortality rates were reported, and the condition became more identified with the carding and was the subject of departmental inquiries and experimental investigations in the 1930s. The cotton carding process is described.

Since the period 1910-12 the Registrar-General given death rates for strippers and grinders separately from other cotton workers, and it has been possible to assess the effectiveness of dust control. A departmental committee of the Home Office concluded, in 1938, that conditions in the card-rooms had much improved, they recommended a compensation scheme for workers disabled by byssinosis, which was instituted in 1941. In the diagnosis of byssinosis from chronic bronchitis and emphysema a significant fact is that byssinosis is characterized by a history of tightness of the chest, breathlessness on Mondays or on the first day at work after an absence. These symptoms may get worse and extend to other working days and eventually give rise to severe and permanent disablement. This history is rarely found in chronic bronchitis and, in the absence of specific clinical and radiographic changes, the diagnosis by history is reasonably reliable for field studies. In a recent study of workers, aged 40-59 in 6 spinning coarser grades of cotton [this *Bulletin*, 1956, v. 31, 167], it was found that the nearer workers are to the carding engines the higher is the prevalence of the disease; in a later investigation in mills spinning finer cottons the prevalence was found to be much less.

Byssinosis among spinners cannot yet be dismissed as an insignificant problem; in 1921-23 and in 1933 cotton spinners had higher death rates than all workers from respiratory and cardio-vascular-renal diseases. Since the start of the compensation scheme the number of pensions awarded has increased from 3 in 1942 to 99 in 1955; but it must be noted that during this period the scheme has been extended to include workers who now account for about one-third of the cases in the cotton-ware industry has also been included. The limits regarding the amount of disablement have been relaxed. The compensation scheme now covers some 58,000 workers, of whom about 36,000 are men and girls.

In the second lecture reference is made to the literature on byssinosis in other cotton industries in Europe and the U.S.A. Byssinosis has been described among workers in hemp and flax, but not in jute. The pathological changes found in the lungs in byssinosis are akin to those of non-specific chronic bronchitis and emphysema, with deposits of dust; "bodies" comparable with asbestos bodies, but rounded, were described by GOWAN in the lungs of 3 Lancashire cotton workers. In the clinical history and pathogenesis the symptoms of the onset of dyspnoea is the basic feature. The author found a highly significant reduction in maximum voluntary ventilation in byssinosis patients.

ASBESTOSIS, AN OCCUPATIONAL DISEASE NEW TO SWEDEN*

by Gunnar Ahlborg and Carl-Johan Hansson,
of the Sahlgren Hospital, Göteborg

Our report is occasioned by nine cases of asbestosis, including one death, to our knowledge the first in Sweden from this occupational pneumoconiosis. The first patient applied in 1953 and occasioned an examination of the 60-odd workers employed in asbestos work in the Göteborg industry; the other eight cases were found in that group examination.

— Asbestosis has been a subject of research abroad for a long time. For further study of this the reader is referred to Hunter, 1955, and his bibliography, and to Noro, 1946, and Wegelius, 1947, who described in detail the occurrence of the disease in Finland. Here we shall only relate our experience and give a brief survey of the conditions under which the disease occurs in Sweden and of its clinical symptoms and prophylaxis.

Our own material, as said above, consists of 9 male workers aged 43 to 63, with times of exposure to asbestos dust of from 5 to 17 years. During this time, however, they have also been employed at other work or with different insulation material, so that the degree of exposure is very hard to judge. Seven of the cases got their asbestosis after insulation work and two after work at asbestos spraying. One of the latter, the one with lethal outcome, could be classed upon first examination as asbestosis Stage III (see below), the other as Stage II. Of the others, four may be regarded as being in Stage I-II and the rest in Stage I or incipient asbestosis.

Subjective complaints that can be attributed to the disease were reported only by the patient with Stage III. For that reason his case history will be recounted here in detail.

At the time of examination he was 63 years old and had been employed at asbestos work for 17 years, mostly at asbestos spraying, and used respiratory protection only sporadically. A year ago he was bothered by increasing shortness

*A lecture delivered to the Göteborg Medical Society on 29 February 1956.

of breath under exertion, by a dry night cough, and by evening fatigue, and at the examination he displayed conversational dyspnea, cyanosis of the lips and prominent veins, watchglass nails, and drumstick fingers. The lung examination showed damping and râle increasing toward the bottom of the lungs. The X-ray picture of the lungs corresponded to what is found in asbestosis Stage III, and in the sputum there were abundant asbestos bodies (Figure 1, p 6). He had tachycardia but otherwise no sign of heart disease in the physical examination of the heart; roentgenologically the heart showed general enlargement, and the electrocardiogram at the first examination showed nothing remarkable. Later a branch block developed.

He was treated with antibiotics and theophyllamine, whereupon the subjective symptoms grew less and the vital capacity rose from 1,700 to 2,900 ml. He could be discharged and cared for at home, but he returned after a few months with a pneumonia that resisted all treatment.

The autopsy (performed by Sourander) showed among other things a diffusely thickened, leathery visceral pleura on both sides. The upper parts of the lungs showed edema and signs of pneumonia. In the lower $\frac{2}{3}$ of the lungs the consistency was uneven, granular, and noticeably firm, in section markedly tough. The fibrous transformation of the parenchyma of the lungs increased in the caudal direction and exhibited a coarsely porous, trabeculated appearance with rounded cavities up to the size of hazel nuts between the reticular, slate-gray, fibrous stretches. Under the microscope the parenchyma of the lung was found to be strewn with broad strands of connective tissue and foci of a locally nodular appearance. The alveoli were found to be atelectatic. In the connective tissue there was an abundance of asbestos bodies and around them a rather heavy cell infiltration, consisting of alveolar cells, macrophages, and other multinuclear giant cells. In the bronchial glands, on the other hand, no asbestos bodies could be found.

PAD: Pneumoconiosis + pneumoniae lobaris et oedema lobi sup. pulm. amb. + bronchitis mucopurulenta + dilatatio sin. et hypertrophia dextr. cordis + sclerosis a. coron. cordis + stasis chron. hepatis.

The case that we classified as Stage II was discovered in 1953 and the patient desisted then from work with asbestos. In spite of that he has shown progression radiologically, but still without subjective complaints. For the other seven we still have too short a period of observation.

Asbestos

Asbestos is a mineral consisting chiefly of magnesium or iron silicate and varying somewhat in composition depending on the locality--Canada, South Africa, Finland, and several other countries. It consists of flexible fibers, so that it can be carded, spun, and woven into tapes or cloths. Considerable strength, high melting point, and poor heat-conducting capacity are the basis for its use as a friction material (brake bands) and insulation material.

In technology we are now dealing with extremes, in temperatures as elsewhere, and it is therefore not surprising that the use of asbestos increases every year. During a 5-year period before the war Sweden imported 17.6 tons; in a corresponding time after the war, since the shortage after the blockade was compensated for, the figure was 46.5 tons.

In Sweden asbestos is chiefly used as insulating material around pipes and the like on vessels, as fire-resistant material in ship's spaces or assembly rooms, in the manufacture of brake bands, and as a reinforcing material in cement and plastic.

In asbestos spraying for insulation against fire, the asbestos is first carded in a machine and then blown with air through a special spray pistol together with water and a binding medium against the surface of the wall, so that the mass forms a layer up to a decimeter in thickness. The dust formation is considerable.

The insulation work on ships is done with imported asbestos which as a rule has been carded. From this an insulating mass is made--often at the work site--consisting of asbestos, water, and some binding agent; this may either be allowed to dry into blocks that can be sawed or smeared directly on the part to be insulated, or else woven into a sort of ticking which is then filled with carded asbestos, sewn together, and installed on tanks or other large objects. The greatest dust production occurs in mixing the asbestos mass and in making the tickings, but considerable exposure to dust can also take place in wrapping pipes with asbestos cords and in tearing away old material.

In installations on land, safe insulating materials such as glasswool, rockwool, or cinder wool are often used, but shocks and vibrations on board, unfortunately, make their use impossible on most vessels.

Pathogenesis

Asbestosis results from inhalation of asbestos dust, which consists in large part of needles ca. 1 μ thick and from 1 to 70 μ in length. The exposure time for occurrence of asbestosis naturally varies with the degree of exposure, but is said to be about half of that for silicosis. Hunter, 1955, recounts a case in which the time between beginning of exposure and death from asbestosis was only 18 months. The irritation due to inhaled asbestos fibers gives rise to a progressive fibrosis of the lungs. In experiments with animals it has been found that short fibers give rise to an interstitial peribronchiolitic fibrosis of more diffuse type, while longer fibers produce nodules of approximately the same type as silicosis nodules (King, Klegg, and Rae, 1946). The progressing fibrosis is usually accompanied by atelectasis, emphysema, bronchiectases, and pulmonary heart.

In contrast to the conditions in silicosis, the modifications occur primarily in the lower $\frac{2}{3}$ of the lungs, with lowering of the hilus area and development of thickenings of the pleura.

From extensive experimentation with animals, King et al. in particular reached the conclusion that the fibrosis in asbestosis is due to purely mechanical irritation, in contrast to the fibrosis in silicosis, where there are strong reasons to suspect a purely chemical effect. For they could only induce asbestosis in the laboratory animals in mobile organs and by means of fibers that have retained their flexibility and sharpness and that have a certain length. Furthermore, they found no great signs of phagocytosis and no damage to the lymph glands. The particles thus are not removed from the lung to any great extent, and this contributes to the unfavorable prognosis in asbestosis, even if the patient promptly discontinues the work.

A characteristic feature is the presence in the lung and in sputum of what are called asbestosis bodies (Figure 1), which contain asbestos needles centrally and are covered at the pointed ends with a substance stainable with hematoxylin. The authors are inclined to interpret the formation of asbestos bodies as an attempt by the body to protect itself against the sharp fibers. It has also been possible to show in experiments with animals that inhalation of asbestosis bodies does not give rise to asbestosis. They are evidence not of asbestosis but of exposure to asbestos.

Clinical Picture

Clinically, asbestosis differs little from other lung fibroses. The subjective complaints usually do not appear until Stage III, in the form of shortness of breath, dry coughing, especially at night, and fatigue. Watch-glass nails and drumstick fingers may be found. The physical pulmonary findings are most often limited to rale; after the onset of pleura thickening and lung fibrosis, also increasing damping basally. Complications are commonly in the form of bronchitis, pneumonia, and pulmonary heart with their specific symptomatic flora, and are often the immediate cause of death. Tuberculosis as a complication is by no means as common in asbestosis as in silicosis.

Besides these complications, in very recent times a high frequency of lung cancer has been observed among patients with asbestosis. In a study of the causes of death in 130 asbestos workers, Doll, 1955, found that the supermortality was substantial, or 39 cases against 15 expected. The excess was due partly to vascular diseases, 22 cases against 7 expected, and partly to lung cancer, with no fewer than 11 cases against 0.8 expected. This relationship, too, makes the prognosis for asbestosis more serious than for silicosis.

Diagnosis is based on the work anamnesis and X-ray findings and can be completed by the finding of asbestosis bodies in the sputum.

The X-ray picture in asbestosis has been described in detail, among others by Wegelius, 1947, on the basis of 126 cases. The roentgenological lung modifications in our patients correspond to those described by Wegelius, with the exception that in one of our cases we found suspected calcifications of the pleura.

Like other pneumoconioses, asbestosis has been subdivided into three stages. In the literature two such stage classifications are defined, one from Germany by Saupe, 1938, and the other from America by Shull, 1936.

Shull in his stage classification puts greater weight on the pleura thickenings than Saupe does. Like the former, we observed such thickenings in several of our cases and for that reason considered that we got the most use out of his stage divisions in classifying our material. According to his classification the stages are characterized as follows:

Stage I. Incipient asbestosis. Slight interstitial fibrosis in both lungs down toward the bases.

Stage II. Moderately advanced asbestosis. More pronounced interstitial fibrosis, which extends to the surface of the lung in the basal $\frac{2}{3}$ of the lungs.

Slight thickening of the pleura and pericardium to the same height. Slight damage to the heart on the right side and moderate apical emphysema.

Stage III. Advanced asbestosis. Very pronounced interstitial fibrosis in the basal $\frac{2}{3}$ of the lungs, very marked apical emphysema, considerable dextral heart hypertrophy, but the contours of the heart do not appear clearly because of condensations in the lungs. Almost always a marked thickening of the pleurae and pericardium.

In accord with the animal experiments referred to earlier showing that asbestosis occurs only in mobile organs, X-ray modifications are found primarily in the basal, most mobile part of the lungs. The pleura modifications are also found there, parietally and in the pleura diaphragmatica. In one of our cases, classified as Stage II, these pleura thickenings were so impervious to X-rays that it must be assumed that calcifications had occurred in them. Figure 2 shows a schematic view of their spread in a patient who had worked with asbestos for 10 years and was subjectively free of complaints.

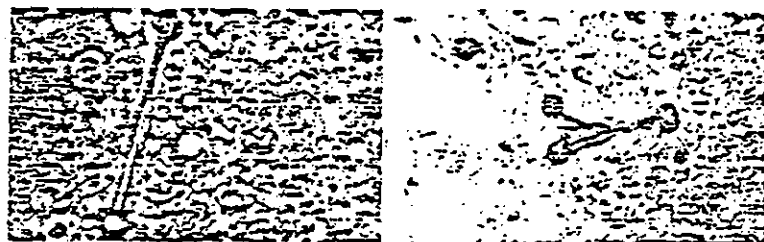


Fig. 1.

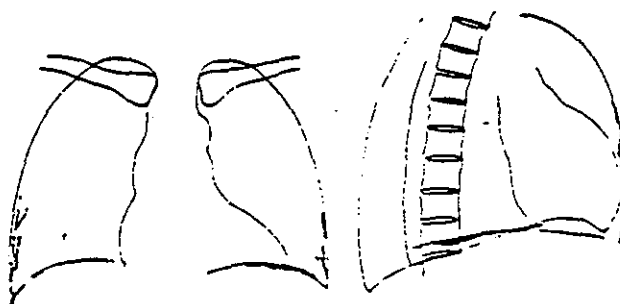


Fig. 2.

Because of the fibrous destruction of the basal parts of the lung, emphysema develops in the apices. In more advanced cases the condensations are so pronounced that normal lung tissue cannot be observed at all. The heart-lung boundaries are then largely blotted out.

As usual in pneumoconioses, the roentgenological differential diagnosis must be supported by the work anamnesis, and just as in the case of silicosis,

repeated examinations are needed before it can be said with certainty whether there is an incipient asbestosis or not. In asbestosis the conditions of exposure are somewhat easier to learn in anamnesis than is the case with silicosis, inasmuch as a workman is aware of whether he has worked with asbestos, but he has a hard time saying whether the mineral he is working on contains substances that cause silicosis or not.

The treatment, of course, is only symptomatic. In the progressing case, cortisone treatment may perhaps be considered, which might possibly arrest the advancing fibroses. Thus far, however, the risk of spread of latent infections in the already greatly reduced respiratory tissues has restrained us from such therapeutic experiments.

Prophylaxis

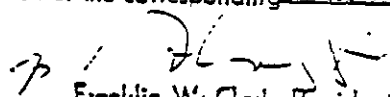
Obviously, an effective prophylaxis against this disease is needed. In England, where the processing and use of asbestos was begun very early, it has been possible through prophylactic occupational hygiene measures to reduce the morbidity from around 3 percent of asbestos workers to practically zero for the time being. In Sweden, too, similar measures have been taken since attention was called to the danger. Thus the first of our cases was reported in the fall of 1953 to the Royal Medical Board with a request for regulations concerning preventive measures similar to those in effect for silicosis. Proposals are now being worked out concerning periodic X-ray examinations of all who are employed in asbestos work.

Extensive dust surveys have been carried out by the State Institute of Public Health and are now being evaluated.

As soon as the first cases became known, the factory inspector in Göteborg served notice that protective masks were to be worn by all workers at places where the slightest risk of inhaling asbestos dust could be imagined. But that safety measure must be regarded more or less as provisional. The factory inspection service is now engaged in working out, wherever possible, other technical protection measures, which will come down in the main to ventilation and local removal of dust by suction at certain moments in the work. More detailed studies in countries where the asbestos risk was observed earlier would be necessary, however, before it would be possible to arrive at specific regulations for the asbestos industry in Sweden.

Through such occupational hygiene measures it can be hoped that this first asbestosis case in Sweden will also be the last. Asbestosis is too serious a disease for anything other than complete protection for workers employed in asbestos work to be acceptable.

I certify that the staff member who translated the foregoing is thoroughly familiar with the Swedish and English languages and that it is a true and complete translation of the corresponding Swedish document.


Franklin W. Clark, President

Language Service Bureau, Inc.
Dupont Circle Building
Washington, D.C. 20036

1956

SVENSKA LÄKARTIDNINGEN

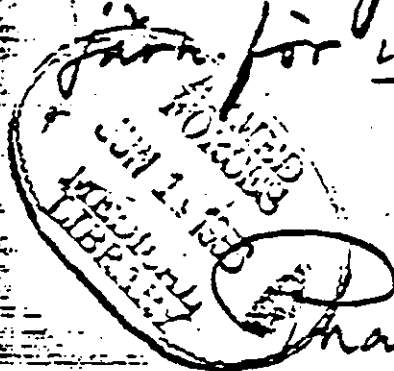
ORGAN FÖR SVERIGES LÄKARFÖRBUND

Arg. 53 Nr 21 25 maj 1956

GER LIST MED. LIT."

1951: Intrafer

Nu: Imferon -
färd. för intramuskulär
injektion



Pharmacia

Summary.

ACTH and Cortisone in Dermatology. Advantages and Risks.

The use of ACTH and cortisone can prolong life in patients suffering from certain fatal dermatoses, and in others relieve distressing symptoms, prolong remissions in some chronic skin conditions, or completely suppress symptoms of acute, self-limiting, allergic reactions. The indications are briefly discussed.

A series of 135 patients treated with these drugs for certain dermatoses over periods of time varying up to 2 years is presented. In view of the high incidence of complications (over 27 % in this series) the importance of maintaining very strict indications and constant supervision is stressed.

Literatur:

1. Sulzberger, M. B.: Proceedings Tenth International Congress of Dermatology, 1952.
2. Hellerström, S., Hollström, E., Hård, S. & Magnusson, B.: Acta Derm.-Ven. 33: 285, 1953.
3. Sulzberger, M. B.: Modern Trends in Dermatology. Ed. R. M. B. MacKenna. Butterworth's medical publications, 1954.
4. Pillsbury, M. & Urbach, F.: Medical Uses of Cortisone. Ed. D. W. Lukens. The Blakestone Co. Inc. New York, 1954.
5. Sulzberger, M. B. & Baer, R. L.: Year Book of Dermatology and Syphilology, 1952. The Year Book Publishers, Chicago.
6. Bollet, A. J., Block, R. & Bunim, J. J.: J. Am. M. Ass. 158: 459, 1955.
7. Hård, S.: Nord. Med. 53: 929, 1955.
8. ———: Sv. Läkartidsn. 53: 666, 1956.
9. Witten, Y. H.: Ann. N. York Acad. Sc. 61: 524, 1955.
10. Gemzell, C., Hård, S. & Siltén, A.: Acta Derm.-Ven. 35: 327, 1955.
11. Matkinson, F. D. & Ferguson, E. H.: J. Invest. Derm. 25: 281, 1956.
12. Livingston, C. S., Hildebrand, J. F., Key, J. S. & Smith, R. W.: A.M.A. Arch. Derm. 72: 313, 1955.

Från Sahlgrenska sjukhuset, Göteborg.

Asbestos,

En för Sverige ny yrkessjukdom!

Av

Gunnar Ahlberg (och) Carl-Johan Hansson, Göteborg

Vårt meddelande är föranlett av 9 fall av asbestos, varav ett dödsfall, och oss veterligt de första i Sverige av denna yrkespneumoconios. Den första patienten sökte 1953 och föranledde en undersökning av det 60-tal arbetare, som sysslade med asbestarbete inom Göteborgsindustrin; därvid upptäcktes de övriga 8.

¹ Föredrag i Göteborgs läkaresällskap 22.2.1956.

Asbestos har utomlands varit föremål för undersökningar sedan ganska länge. För närmare studium av dessa hänvisas till Hunter (1935) och till hans litteraturanvisningar samt till Noro (1946) och Wegelius (1947), vilka senare utförligt beskrivit sjukdomens förekomst i Finland. Här skall endast relateras våra erfarenheter och ges en summarisk översikt över sjukdomens uppkomstbetingelser i Sverige, dess klinik och profylax.

Vårt eget material utgöres som sagt av 9 manliga arbetare i åldern 43 till 63 år och med en expositionstid för asbestdamm av 5 till 17 år. Under denna tid ha de emellertid även sysslat med annat arbete eller med annat isoleringsmaterial, varför expositionsgraden är mycket svår att bedöma. 7 av fallen ha föll sin asbestos efter isoleringsarbete, 2 efter arbete med asbestsprutning. Ett av de senare, det med letal utgång, kunde vid första undersökningen rubriceras såsom asbestos stadium III (se nedan), det andra till stadium II. Av de övriga få 4 anses tillhöra stadium I—II, de återstående stadium I eller börjande asbestos.

Subjektiva besvär, som kunna hänföras till sjukdomen, ha endast angivits av patienten med stadium III. Hans sjukdomshistoria skall därför här närmare relateras.

Vid undersökningen var han 63 år och hade under 17 års tid sysslat med asbestarbete, mest med asbestsprutning, och använde därvid endast andningskydd sporadiskt. Sedan ett år tillbaka besvärades han av ökande andfåddhet vid ansträngning, nattlig torrhosta och kvällströtthet, och vid undersökningen föredde han samtalsdyspnöe, läppeyanos och ökad väntekning, urgelnaglar samt trumpinnesfingrar. Lungfyndet utgjordes av nedåt tilltagande dämpning och rassel. Röntgenbilden av lungan överensstämde med den, man finner vid asbestos stadium III och i spulum fanns rikligt med asbestkroppar (fig. 1). Han hade tachycardi men i övrigt inga tecken på hjärtsjukdom vid fysikalisk undersökning av cor; detta var röntgenologiskt allmänförstorat och ekg vid första undersökning utan säker anmärkning. Senare utvecklade sig ett grenblock.

Han behandlades med antibiotika och theophyllamin, varvid de subjektiva besvären minskade, och vitalitetskapaciteten steg från 1.700 till 2.900 ml. Han kunde utskrivas och vårdas i hemmet men återkom efter ett par månader med en pneumoni, som motstod all behandling.

Sektionen (Sourander) visade bl. a. en diffust förtjockad, svålig pleura visceralis bilateralt. Lungornas övre delar visade ödem och tecken på pneumoni. Konsistensen var i ned-

re 23 av lungorna ojämna, knottig och anmärkningsvärt fast, i snittet synnerligen seg. Den fibrösa omvandlingen av lungparenchymet ökade i kaudal riktning och uppvisade ett grovporöst, trabekulerat utseende med upp till hasselnstora, rundade kaviteter mellan de nätförmiga, skiffergrå, fibrösa straken. Mikroskopiskt fann man lungparenchymet genomsett av breda bindvävsstråk och härdar av ställvis nodulärt utseende. Alveolerna ledde sig atelektatiska. I bindväven fanns rikligt med asbestkroppar och omkring dessa en rätt kraftig cellinfiltration, bestående av alveolarceller, makrofager och en och annan flerkärnig jättecell. I bronchialkörtlarna däremot kunde inga asbestkroppar återfinnas.

PAD: Pneumoconiosis + pneumoniae lobaris et oedema lobi sup. pulm. amb. - Bronchitis mucopurulenta + Dilatatio sin. et hypertrophia dx cordis + Sclerosis a. coron. cordis + Stasis chron. hepatitis.

Det fall, som vi rubricerat såsom stadium II, upptäcktes 1933 och avkopplades då från arbete med asbest. Trots detta har han visat röntgenologisk progress, dock fortfarande utan subjektiva besvär. För de övriga 7 ha vi ännu för kort observationstid.

Asbest.

Asbest är en bergart, huvudsakligen bestående av magnesium- eller järnsilikat och med något olika sammansättning, beroende på syndorten. Canada, Sydafrika, Finland med flera länder. Den består av flexibla trådar, vilket gör att den kan kardas, spinnas och vävas till band eller tyg. Betydande hållfasthet, hög smältpunkt och dålig värmeledningsförmåga ligger till grund för dess användning som friktionsmaterial (bromsband) och isoleringsmaterial.

Inom tekniken rör vi oss ju för närvarande med ytterligheter även beträffande temperaturer, och det är därför inte överraskande, att användningen av asbest ökar för varje år. Under en femårsperiod före kriget importerade Sverige 17,6 ton, under motsvarande tid efter kriget, sedan bristen efter avspärningen ersatts, 46,5 ton.

I Sverige används asbest främst allt som isoleringsmaterial runt rörledningar och dylikt på fartyg, som brandisoleringsmaterial i fartygsutrymmen eller samlingslokaler, vid bromsbandstillverkning samt som armeringsmaterial i cement och plast.

Vid asbestsprutning för brandisolering kardas först asbesten i en maskin och blåses sedan med luft genom en spe-

ciell sprutpistol tillsammans med vatten och ett bindemedel mot väggytan, så att massan bildar ett upp till decimeter-tjockt lager. Dammbildningen är betydande.

Vid isoleringsarbeten på fartyg utgår man från importerad, i regel kardad asbest. Av denna tillverkas — ofta på arbetsplatsen — isoleringsmassa bestående av asbest, vatten och något bindemedel, vilken antingen får torka till såghara block eller smetas direkt på den detalj, som skall isoleras; eller också vävas madrass-var, som sedan fyllas med kardad asbest, sys och monteras på behållare eller andra större föremål. Största dammproduktionen uppstår vid blandning av asbestmassa och vid tillverkning av madrasser, men även vid lindning av rör med asbestsnören och rivning av gammal material kan betydande dammexposition uppkomma.

Inom anläggningar i land använder man ofta som isoleringsmaterial otarlig glas-, sten- eller slaggull, men skakningarna ombord omöjliggöra tyvärr dessas användning på de flesta fartyg.

Patogenes.

Asbestos uppkommer genom inhalation av asbestdamm, vilket till största delen består av ca 1 my tjocka nalar med en längd mellan 1 och 70 my. Expositionstiden för uppkomsten av asbestos varierar givetvis med expositionsgraden men anges ungefär till hälften av den för silikos. Hunter (1953) relaterar ett fall, där tiden mellan börjande exposition och exilus i asbestos endast utgjorde 18 månader. Retningen genom inhälerade asbestfibrer ger upphov till en fortskridande fibrosering av lungorna. Vid djurförsök har man funnit, att korta fibrer ge upphov till en interstitiell peribronchiolitisk fibrosering av mera diffus typ, medan längre fibrer ge noduli av ungefär samma typ som silikosnoduli (King, Klegg och Rae, 1946). Den progredierande fibroseringen ger på vanligt sätt atelektas, emfysem, bronchiectasier och cor pulmonale.

I motsats till förhållandena vid silikos uppträder förändringarna främst i nedre $\frac{2}{3}$ av lungan med neddragning av hilusområdet och uppträdande av pleuraförtjockningar.

Genom omfattande djurexperimentella undersökningar ha framför allt King och medarbetare kommit till den slutsatsen, att fibroseringen vid asbestos uppkommer genom rent mekanisk retning, i motsats till fibroseringen vid silikos, där man ju har starka skäl att misstänka en rent kemisk retning. De kunde nämligen endast erhålla asbestos hos försöksdjuren i rörliga organ och genom fibrer, som ha

kvar sin flexibilitet och spetsighet och som ha en viss längd. Vidare fann man ingen större ansats till phagocytos och ingen förstoring av regionära lymfkörtlar. Partiklarna föras sålunda icke i någon större utsträckning bort från lungan, vilket bidrager till den dåliga prognosen vid asbestos, även om patienten tidigt avbryter arbetet.

Karakteristiskt är förekomsten i lungan och i sputum av s. k. asbestkroppar (fig. 1), som centralt innehålla asbest-



Fig. 1.

nålar, i de spetsiga ändarna klädda med ett med hämatoxylin färgbart ämne. Man har velat tolka bildningen av asbestkroppar såsom ett försök från kroppens sida att skydda sig mot de spetsiga fibrerna. Det har också på djurförsök kunnat visas, att inhalation av asbestkroppar icke ger upphov till asbestos. De äro bevisande icke för asbestos men väl för asbestexposition.

Klinik.

Asbestos skiljer sig kliniskt icke nämnvärt från andra lungfibroser. De subjektiva besvären uppträda vanligen först i stadium III i form av andfäddhet, torrhosta, framför allt nattetid, och trötthet. Urginsnaglar och trumpinnesfingrar kunna förekomma. Det fysikaliska lungfyndet inskränker sig oflöst till rassel, efter inträdd pleuraförtjockning och lungfibrosering uppträder tillagande dämpning basalt. Komplikationer äro vanliga i form av bronchiter, bronchiektasier, pneumonier och cor pulmonale med deras speciella symptomflora och utgöra ofta den omedelbara orsaken till exitus. Tuberkulosen är som komplikation inte alls så vanlig vid asbestos som vid silikos.

Utom dessa komplikationer har på sista tiden hos patienter med asbestos uppmärksamats den höga frekvensen av cancer pulmonis. Vid undersökning av dödsorsaken hos 130 asbestarbetare fann Doll (1955), att överdödligheten var

betydande eller 39 fall mot väntade 15. Överskottet berodde dels på vasculära sjukdomar med 22 fall mot väntade 7, dels på cancer pulmonis med icke mindre än 11 fall mot väntade 0,8. Även detta förhållande gör asbestosens prognos allvarligare än silicosisens.

Diagnosen ställes på yrkesanamnes och röntgenfynd och kan fullständigas genom fynd av asbestkroppar i sputum.

Röntgenbilden vid asbestos har utförligt beskrivits bl. a. av Wegelius (1947) på grundval av 126 fall. De röntgenologiska lungförändringarna hos våra patienter överensstämmer med de av Wegelius beskrivna, med undantag av att vi funnit suspekta pleuraförkalkningar i ett av våra fall.

Liksom andra pneumoconioser har även asbestosen indelats i tre stadier. I litteraturen finnas två dylika stadiindelningar definierade, den ena från Tyskland av Saupe (1938), den andra från Amerika av Shull (1936).

Shull lägger i sin stadiindelning större vikt vid pleuraförtjockningarna än vad Saupe gör. Vi ha liksom den förre iakttagit sådana förtjockningar i flera av våra fall och ha därför ansett oss ha mest nytta av hans stadiindelning vid klassificeringen av vårt material. Enligt hans indelning karakteriserar de olika stadierna på följande sätt:

Stadium I. Incipient asbestos. Lätt interstitiell fibros i båda lungorna ner mot baserna.

Stadium II. Måttligt avancerad asbestos. Mer uttalad interstitiell fibros, som sträcker sig ut till lungytan på de basala $\frac{2}{3}$ av lungorna. Lätt förtjockning av pleurae och pericardium i samma höjd. Lätt högerförstoring av hjärtat och måttligt apicalt emfysem.

Stadium III. Avancerad asbestos. Mycket uttalad interstitiell fibros med förtätningar i de basala $\frac{2}{3}$ av lungorna, mycket markerat apicalt emfysem, tydlig högersidig hjärthypertrofi, men konturerna av hjärtat framträda otydligt på grund av förtätningarna i lungorna. Nästan alltid uttalad förtjockning av pleurae och pericardium.

I överensstämmelse med tidigare relaterade djurexperimentella undersökningar, visande att asbestos endast uppstår i rörliga organ, finner man röntgenförändringarna främst i lungans basala, mest rörliga delar. Även pleuraförändringarna finner man där, parietalt samt i pleura diaphragmatica. I ett av våra fall, klassificerat som Stadium II, var dessa pleuraförtjockningar så röntgentåla, att man måste förutsätta, att det uppkommit förkalkningar inom desamma. Fig. 2 visar en schematisk bild av dessas utbred-

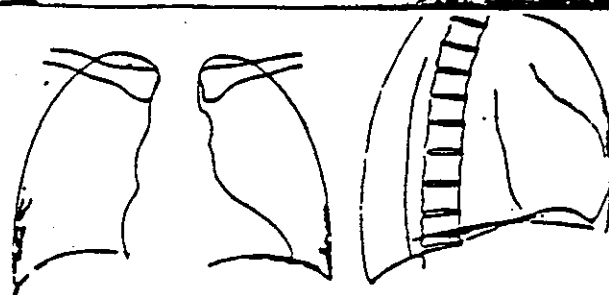


Fig. 2.

ning hos en patient, som i 10 års tid arbetat med asbest och som var subjektivt besvärsfri.

På grund av den fibrösa destruktjonen av de basala partierna inom lungan uppkommer på spetsarna emfysem. I mera avancerade fall äro de stråkformiga förtätningarna så uttalade, att normal lungvävnad överhuvud taget icke kan iakttagas. Hjärt-lunggränserna äro då till största delen utsuddade.

Den röntgenologiska differentialdiagnosen måste som vanligt vid pneumoconioser stödja sig på yrkesanamnesen, och liksom vid silicos fordras det upprepade undersökningar för att med säkerhet kunna säga, om det rör sig om incipient asbestos eller inte. Expositionsbetingelserna vid asbestos är emellertid lättare att anamnestiskt få fram än vid silicos, beroende på att en arbetare väl känner till om han arbetat med asbest men ju har svårt att ge uppgift om, huruvida den bergart, han bearbetar, innehåller silicosframkallande ämnen eller inte.

Behandlingen är givetvis endast symtomatisk. Man kan kanske i progredierande fall diskutera cortisonbehandling, som möjligen skulle kunna hejda den fortskridande fibroseringen. Risken för utbredning av tysta infektioner i den redan starkt reducerade respiratoriska vävnaden har emellertid hittills avhållit oss från sådana terapiförsök.

Profylax.

Effektiv profylax är givetvis nödvändig mot denna sjukdom. I England, där man mycket tidigt började bearbeta och använda asbest, kunde man också genom profylaktiska yrkeshygieniska åtgärder nedbringa morbiditeten från omkring 3 % hos asbestarbetare till för närvarande praktiskt taget 0. Även i Sverige ha liknande åtgärder vidtagits, sedan

man uppmärksammat faran. Sålunda anmälades det första av våra fall 1953 till Kungl. medicinalstyrelsen med begäran om bestämmelser angående förebyggande åtgärder i likhet med dem, som gäller för silicis. Förslag håller nu också på att utarbetas beträffande periodiska röntgenundersökningar av alla i asbestarbete sysselsatta.

Omfattande dammundersökningar ha även utförts av Statens institut för folkhälsan och håller på att bearbetas.

Så fort de första fallen blivit kända, meddelade yrkesinspektören i Göteborg anvisning, att skyddsmask skulle bäras av alla arbetare på platser, där minsta risk för inhalation av asbestdamm kunde tänkas förekomma. Denna skyddsåtgärd får dock i viss mån betraktas som ett provisorium. Man är f. n. inom yrkesinspektionen — i samarbete med arbetsgivarna — sysselsatt med att, där så är möjligt, utarbeta andra tekniska skydd, vilka i huvudsak kommer att gå ut på ventilation och lokala utsugningar vid vissa arbetsmoment. Närmare studier i länder, där asbestosrisken tidigare observerats, torde emellertid bli nödvändiga, innan man kan komma fram till särskilda bestämmelser för asbestindustrin i vårt land.

Genom sådana yrkeshygieniska åtgärder kan man hoppas, att dessa första asbestofall i vårt land också skall bli de sista. Asbestosen är en alltför allvarig sjukdom, för att man skall kunna acceptera annat än fullständigt skydd för inom asbestarbete sysselsatta arbetare.

Litteratur:

- Doll, R.: Brit. J. Ind. Hyg., 12: 81, 1955.
Hunter, D.: The Diseases of Occupations. London, 1955.
King, Klegg & Rae: 1946. Cit. Hunter.
Koro, L.: Acta path. et microbiol. Scand., 23: 53, 1946.
Lampe, E.: Röntgenatlas der Asbestose der Lungen. Thieme, 1938.
Shull, J. R.: Radiology, 27: 279, 1936.
Wegelin, C.: Acta Radiol., 28: 139, 1947.

Obstipationens behandling

Av

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Förstoppningen är en av civilisationens mer eller mindre besvärliga förbannelser. Vi låter i stor utsträckning maskinerna utföra kroppsarbetet och behöver därför mindre mat. Då blir det också mindre avfall allen helst som vi hittat på