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is irritating to the skin. The new insecticide reported here is a product known as alpha naphthyl isothiocyanate, usually mixed with pyrethrum to obtain the toxic effect.

# Current Comment

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IN "an X-Ray Study of the Lungs of Workmen in the Asbestos Industry, Covering a Period of Ten Years," ARIAL W. GEORGE, M.D., and RALPH D. LEONARD, M.D.,* describe an intensive study of workers exposed to the asbestos hazard in a large industrial plant in Massachusetts, where brake linings were manufactured. This study was begun in 1928. The asbestos contained somewhat less than 30% silica. Dry weaving was first used, later changed to wet weaving. Of all cases examined in 1928 and 1929, 12 were diagnosed as asbestosis by x-ray findings. These were put on compensation or given a lump-sum settlement. They represented a very small number of the total employees. Three cases were definitely advanced, but without many of the usual clinical signs, and would not change their occupation. These remained with the plant until it was closed in 1936, and one of these became a litigant.

Within a short time after the closing of the plant, 90 other workmen brought in claims for disability due to asbestosis. These were examined and positive diagnoses of asbestosis made on 12 with three additional cases questionable. About 13% of these 90 cases claimed symptoms. The insurance carrier immediately arranged compensation or settlement in excess of $50,000 for these 12. Of the remaining 78, 30 came to trial. The Legal Aid Society furnished able counsel and the late John B. Hawes, Jr., M.D., became the medical expert for them and made a positive diagnosis in the 30 cases. The referee committee of three so-called "Panel Doctors" (one a roentgenologist), whose diagnosis is final, examined 10 of these 30 cases without finding definitely stated asbestosis, but their findings were reported as, "consistent with," or "a slight degree of," or "the probability of" asbestosis. They were obviously depending upon the history of exposure and not on the x-ray findings alone. This feature of exposure appeared also to influence Dr. Hawes in some 80% of his positive diagnoses. He based his diagnosis and prognosis upon: (1) History of exposure, (2) difficulty in breathing on exertion, (3) cough, (4) rapidity of heart action on exertion, and (5) "that asbestosis was a progressive disease, once contracted, whether the individual remained in the industry or was removed."

The authors find also that a large number of asbestosis cases gave the history of coughing up blood. Irritation on the hands may develop into open ulcers whose bases show asbestos fibers imbedded in the deep layers of the skin. Probably a similar condition may develop in the air passages. Certain individuals seem sensitive and others immune, for which no satisfactory explanation is at hand unless possibly the method of breathing.

Re-examination of the 12 original cases, made at the time of one of the hearings, showed them to be free of all symptoms and, except for several women in their group, they were doing laborious work in new occupations. This was eight or nine years after their original cessation of exposure. It is evident that if a history of exposure is eliminated, a positive clinical diagnosis of asbestosis cannot be made and reliance must be placed upon the x-ray examination. Further, it appears that asbestos dust will not give symptoms until there are visible changes in the x-ray film.

The more characteristic findings are described in detail, briefly summarized as: Bilateral distribution limited to the lower lobes of both lungs, at first as a "ground glass" or "smoky" appearance; absence of gross nodulation — what nodulation there is has a fine, evenly distributed quality; no characteristic hilum or root shadow changes although there is often some accentuation of the bronchial shadows which present a laminated appearance in the lower lobes (probably localized emphysema with atelectasis); accentuation of interlobar pleural septa; lower chest pleuritic adhesions with obliteration of the costophrenic angles; emphysema; and more dilatation of the right heart than seen in other pneumoconioses but no demonstrable dilatation of the pulmonary artery. No cases of complicating tuberculosis were found nor was there evidence of asbestosis lighting up a preexixsting tuberculosis.

Differential diagnosis would include other pneumoconiosis, especially silicosis; tuberculosis; lower lobe congestion from various causes; accentuation of the bronchial shadows in the lower lobes; and chronic bronchitis or bronchiectasis, but neither of these latter conditions usually show an associated "ground glass" appearance.

The authors conclude that each asbestos industry and exposure must be an especial study in itself; the percentage of silica must be taken into consideration and whether the methods used were dry or wet; asbestosis is not a progressive disease after the individual is removed from the dust exposure; there is no evidence of any causal relation to tuberculosis; final diagnosis must depend mainly on x-ray evidence, there being no pathognomonic clinical findings; and that an amount of asbestosis dust in the lungs, sufficient to produce symptoms, will give visible evidence on the x-ray film.

* In *Radiology*, Vol. 33 196-202 (August) 1939.

# Toxic Fumes

## —*in Massachusetts Industries*—

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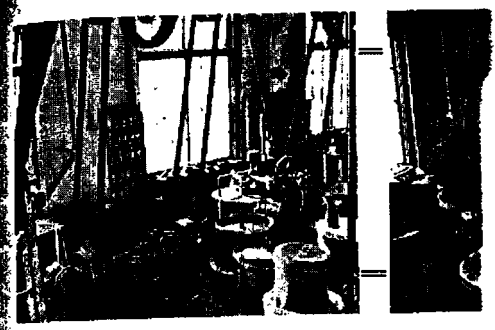
MOST occupational diseases fall roughly into one of three groups:

1. Dust diseases;
2. Occupational dermatoses;
3. Poisoning by toxic fumes and gases.



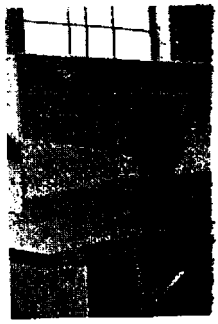
Of these three categories the first have received the most attention; the second are the most numerous; and the third are the most varied and form the subject of this discussion.

While the dust diseases affect primarily the lungs, and dermatoses are by definition skin dis-



**LEFT: Dope room of an artificial leather factory. sources of benzol vapor. RIGHT: Same, after insta ment. Even with tubs of dope and mixers covered of solvent vapor escape. Ventilation is th**

eases, toxic gases and fumes may act selectively any part of the body. For example, the lining the nose, the very doorway to the system, is tacked by chromic acid; while the bladder, almo the last organ which contacts waste matter befo it is eliminated, is the site of tumors caused b some amino compounds used chiefly in makin dyes. Many other toxic fumes attack the lower respiratory tract, liver, kidneys, blood-forming mechanism and nervous system. In variety of action on the body the gases and fumes are certainly the most interesting of the occupational hazards.



**LEFT: Table for fur volved exposure to table. Without pro RIGHT: Acid pickli**

It has been shown innumerable times that most industrial poisonings occur through the respiratory tract, i.e., they are caused by breathing in the toxic agent, which may be in the form of a gas, fume, or dust.

The industrial hygienist recognizes this as ax matic, but others do not always do so.

We recently learned of several cases of lead p soning among painters engaged in coating str tural steel with red lead paint.

Three theories of how these cases occurred w advanced. The paint was partly sprayed and pa ly brushed. Water was supplied the painters in open pail, and it was found that this was freque ly well flavored with red lead paint from the spr