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IN THIS ISSUE :

EDITORIAL OBSERVATIONS :

Safety Support	5
Responsibility and Safety	5
Recovery Effort	6
Robert J. Sullivan	6

ACCIDENT :

N.R.A. Industrial Safety and Health Standards	7
The Development of Safety Glass	10
New Code for Window Cleaners	11
Fifty Years' Service	12
The Eyes and Their Relation to Safety —by JAS. H. DELANEY, M.D.	13
How to Organize Safety Work on Field Jobs —by GEORGE E. SANFORD	16
Accident Causes in Different Industries —by G. STEVENSON TAYLOR, O.B.E.	17
Menace of Youthful Drivers	21
The Fool-Proof Highway of the Future —by Dr. MILLER McCLINTOCK	22
Accident Proneness—by ERIC FARMER, M.A. (Continued from June)	23
Industrial Fatalities in New York State	24
Safety Glass Committee	24

SPECIAL FEATURES

N.R.A. codes contain individual safety requirements, on page 7.

Youth as automobile drivers, on page 21.

Injuries involving the nervous system, paper of eminent neurologist, on page 35.

FIRE :

A Costly Lesson in Fire Prevention ..	25
Fighting Fire with First-Aid	27
The Ignition Temperature of Wood	29
Liquefied Petroleum Gases	30
Self-Inspection Blanks for Plants ...	31

HEALTH :

The Traumatic Neuroses—by JOSEPH SMITH, M.D.	35
Industrial Dusts and Their Control—by R. C. STRATTON	38
Compensation for Silicosis Held Urgent	39
Variation in Lung Lesions Produced by Silica —by TIMOTHY LEARY, M.D.	41
Industrial Nursing:	
The Future in Occupational Disease Coverage	43
Some Common Defects Among Young Male Workers	43
Classification of Causes of Blindness	43
A Nurse Was on Her Toes	43
Death Rates by Occupation	43
Occupational Disease Conference Held in New York	44
Occupational Diseases in Industry	45
Summer Accidents	47

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VARIATION IN LUNG LESIONS PRODUCED BY SILICA AND THE SILICATES*

by TIMOTHY LEARY, M. D.

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ONE of the interesting phenomena in connection with silicosis is the appearance of this condition apparently suddenly, coming in full bloom into recognition following the establishment of industrial accident compensation legislation, and all well within the last generation. It almost seemed as if this were a wholly new disease, and yet reason taught that it must have existed since man began to quarry and chip stone. That modern methods of machine drilling, polishing, and sand blasting added tremendously to the hazard there can be no question, but that dusty trades have tended to kill off human beings relatively early in their lives for many generations there can be no question. In a recent conversation with Commissioner Noonan of the Connecticut Industrial Accident Board, he said that he was commenting upon this apparently new disease with an old judge in a Connecticut town, when the judge drew him to the window and pointing to a graveyard on the hill opposite said, in substance: "That graveyard has been filled for some generations with the bodies of men who worked in (naming a local industry), and who died of tuberculosis as young men. The hazard was understood and looked upon as part of the game of life. It was accepted as fate that a man who went to work in that plant would in all human probability die young."

Another factor influencing the late discovery of silicosis as a disease has been the location of hazardous industries in general outside of the large cities. The peculiar sentimental horror of postmortem examinations still persists outside of the cities, and these examinations, when rarely performed, were carried out by men with limited training. If tuberculosis was present, although we now know it to be a secondary process, its lesions were so spectacular and so mentally satisfying as to the cause of death, that no further investigation was thought necessary. It was only when microscopic and chemical examination was practiced, and the character of the lesions disclosed evidence of some initiating factor, that suspicion of the influence of the occupation was aroused.

My experience with pneumokoniosis was largely limited to anthracosis until the industrial accident act focussed attention upon silicosis. The commonest form of dust exposure is to smoke dust. A very definite condition of more than ordinary anthracosis is commonly found in what I have termed "cigaretteosis." The confirmed cigarette smoker is apparently able for years to excrete the inhaled smoke dust, and may present normal appearing lungs at postmortem examination. Ultimately, however, the excretory mechanisms which mechanically

move the dust toward the trachea by ciliary action, or take it out of the lung tissue by phagocytic cells and lymph drainage, become inefficient. In the lungs of such individuals cross section discloses a dark brown tint of the cut surface, particularly in the upper lobes, and scrapings from the surface have a distinctly dirty color. On microscopical examination the alveoli are found containing free pigment and large numbers of phagocytic cells (histiocytes), which have engulfed the fine particles of pigment. Some alveoli are completely blocked with compacted masses of these cells. That the shortness of breath associated with intensive cigarette smoking may be, in at least small part, due to a less efficient respiratory function of the lungs suggests itself.

The impression exists that tuberculosis is a necessary sequence of silicosis in order to produce death. This is not true, particularly in asbestosis. I have material from four cases of asbestosis, in one of which (material kindly supplied by Dr. Sydney C. Darymple) death was due to a brain tumor and the asbestosis was only an associated condition. In a second case death was due literally to strangulation of the lungs which were completely encased in a layer of rigid fibrous tissue. In this case the lungs notably the left were contracted away from contact with the parietal pleura. There was thus established what might be called a pneumothorax, though no openings were found in the lungs through which air might have escaped. The lungs on section revealed a well preserved vesicular structure with practically no fibrosis except the enveloping subpleural layer and no gross evidence of emphysema, though microscopically there was a moderate degree of fine emphysema. In a third case the striking lesion was an emphysema. In a third case the striking lesion was an emphysema, marked by the universal distribution of series of groups of rounded cavities of generally small dimensions through both lungs. The lungs completely filled the pleural cavities and did not collapse on opening the chest. There was no suggestion of tuberculosis in these cases. The outstanding symptom in both cases was dyspnoea, in spite of the contrasting lesions. The breathing in each case was almost purely abdominal in type. The deaths were apparently due to exhaustion.

In a fourth case recently seen there is generalised distribution of foci of emphysema about pigmented scarred foci, as in the third case cited above, but in the lower lobe of the left lung there is diffuse fibrosis and an early tuberculosis.

The contrast between these cases of asbestosis and pure cases of silicosis is striking. Lungs from two

*Address delivered at New England Safety Conference April 30, 1934.

was dyspnoea, exhibit a fibrosis which is diffuse in the upper lobes with small islands of vesicular lung tissue between the masses of scar tissue. In the lower lobes occur typical silicotic nodules, discrete, lying in a rather abundant vesicular lung tissue. The least degree of silicotic change appears in the posterior portions of the lung, just above and below the hilum and again in the region above the diaphragmatic surface in the base. Fibrosis throughout the lung structure, with moderate subpleural fibrosis, is the characteristic picture.

A most interesting case of pneumokoniosis occurred in a mason whose functions included the relining of furnaces with fire brick. These lungs disclosed at the apices dense ebony like masses of blackened scar tissue, and a layer of varying thickness of the same tissue enveloping the lungs in great part beneath the pleura. In the dense apical mass at the left apex is a sharply isolated old cavity, measuring 5 centimeters in greatest diameter. The wall was smooth and contained a watery fluid with a gray deposit on the lining. Smears revealed no tubercle bacilli, though there is little doubt that the lesion was tubercular. There were nodal slaty masses scattered through the vesicular lung substance quite generally, varying from 0.3 to 0.5 centimeters. The distribution of the lesions in this case, the older process apical with an isolated cavity, and the character of the distribution beneath the pleura, together with the microscopical evidence of nodal masses having the character of healed tubercles throughout the enveloping subpleural

process left little doubt that this man had had a bilateral apical tuberculosis which had spread along the subpleural tissue. It was evident that the scarring due to anthracosis had led to the healing of the tubercular process and the isolation of the apical cavity. However there was a greater degree of fibrosis than anthracosis alone would account for. Ashing of the lung showed a silica content of the ash of about 25 per cent. It is a question whether his dust exposure shouldn't be credited with curing his tuberculosis. The death was sudden, suggested a coronary death, though the coronary arteries were relatively normal, and was not at all characteristic of a death from silicosis.

The cases of pure silicosis, *i. e.* without tuberculosis, and of asbestosis without tuberculosis illustrate the varied picture which the lungs may show. From three of my asbestosis cases the influence of a widespread focal emphysema, occurring in regions where the asbestosis bodies are massed, makes plain that this lesion together with the fixation of the lung by thickening of the subpleural tissue, is responsible for the dyspnoea and the relative insufficiency of the respiratory mechanism. Fibrosis within the lung is slight in degree and of minimal importance.

On the other hand fibrosis within the lung replacing a large part of the vesicular substance, together with subpleural thickening and adhesions, bring about the same interference with respiratory efficiency in the silicotic.



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