PLAINTIFF'S
EXHIBIT
DUP-83

Section 4

SERVICES ALLIED TO OCCUPATIONAL MEDICINE

Chapter 15

Medicolegal Aspects of Occupational Medicine

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A significant development of the last twenty years has been the forging of a link between the industrial physician and the attorney engaged in industrial work. The prodigious increase of workmen's compensation litigation has resulted in an interdependence that could never have been predicted when one remembers the hostility with which the two professions viewed each other during the turn and infancy of the twentieth century.

The legion of medicolegal questions that arise daily turns our minds back to the centaurs of mythology, concerning whom we ask, "Were they man or horse"? The intricate questions arising in industrial medicine and industrial litigation cause us to stop briefly and ask, "Is it medicine or law"? The answer is "Both."

To practice successfully in industrial litigation involving injuries and occupational diseases of men in industry, the lawyer must be reasonably well acquainted with medical problems and must be able to discuss his cases with physicians who can and will give the time and effort to assist him.

RECORDS

To practice successfully in industrial medicine, a well-rounded industrial physician should have an understanding of the basic law involved, the responsibility or nonresponsibility that may result from a particular set of facts, and an appreciation of the role he is to play in a future drama of litigation – where he and his records will be the key witnesses.

In the trial of industrial litigation involving injury or disease to a workman, the plant physician and his record can be and usually are the

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adopt this criterion, there is also some logic to *the old rule*. In giving testimony that may adversely affect the rights of a litigant, of which our courts are very jealous, it is elementary that the person so testifying should be subject to cross-examination. If the witness is relating something from a record that he or she did not make, obviously the cross-examination can elicit nothing as to the making, the accuracy, the sufficiency, or the meaning of the particular record.

This basic principle that is embodied in general terms under the so-called "hearsay rule" has prevented many important and perhaps dispositive bits of evidence from being introduced into a case and requires intelligent consideration in the making of records, so that a similar fate is not suffered in important litigation.

It is, therefore, paramount that every record be authenticated by the signature of the person making it; only by this method will you perpetuate the identity of your necessary witnesses. This identity being known, you will have the ability to present such vital testimony through the lips of the person whose record it is, since such a record was made at a time when the witness knew its accuracy, even though there might be a perfectly logical lack of any personal recollection at the time of the trial.

Histories should be taken in detail, since the story told by the claimant *ante litem*, at a time when there is little reason for exaggeration or, perhaps, falsification, will be very persuasive to the court, jury, or commissioner before whom the case is tried.

Records, in so far as medicolegal matters are concerned, usually refer to the plant hospital or plant doctor's records. They embrace the treatment card, nurses' charts, annual physical examination cards, special physical examination records, x-rays, consultation reports, and any forms that may be of use and that have been adopted by a particular industry. As an illustration of the latter, some use is made today of a form requesting plant hospital treatment for a personal condition which, at the time of the request, is alleged by the employee to be unrelated to his work. In order to perpetuate the record and to prevent a claim of work-relationship at a later date when some economic benefit might incite the desire to change the story, these forms can be used where applicable and should be retained in the medical file after execution by the employee.

As an aid to the immediate end of preparing efficient records and the ultimate end of their successful use in litigation, certain general comments are perhaps in order and are recorded here.

1. All histories, regardless of whether the onset of discomfort is claimed to be occupational or nonoccupational, should be taken in full and reported on the plant hospital card or other record.
2. The examining doctor or attending nurse should sign each entry, noting the time and the date.
3. The history should be taken in the attendant's own handwriting and retained in its original form unless immediately transcribed by type-

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writer. If the latter is done, the signature of the attendant should be in longhand.

4. In the cases where litigation is involved or where, at the time of the injury, it is apparent that a plant responsibility exists, the physician in charge should supervise the examination or examine personally if practical, as the value of limiting the number and selecting the most capable of the necessary witnesses is obvious.
5. Records of all visits and the clinical data obtained should be kept in each case. These records should be made at the time of the visit and, as heretofore suggested, should be signed by the attendant.
6. Where x-rays are taken by a technician, a record should be kept, showing the identity of the technician and a positive identification of the x-ray as that of the patient whose picture it purports to be.
7. Records should never be destroyed during the period of employment, nor after death or termination of employment until the possibility of litigation is removed by the applicable statute of limitations.
8. Terms that may be embarrassing or difficult to explain should not be used in the records.

With reference to the last point, it must be remembered that the medical records, during litigation, will be used for direct testimony, for cross-examination, and for scrutiny by the court or commissioner. Any ambiguity or unfortunate expression will be accentuated, either naturally or purposely, by opposing counsel and may prove embarrassing. As an example, the writer personally dislikes the use of the two familiar terms of "industrial lung" and "postconcussional syndrome." The term "industrial lung" appears to denote a degree of abnormality that may be entirely inconsistent with the findings and, when placed in the hands of a jury, court, or commissioner, may result in the defendant's losing the case on the classification by his own record of a lung that may be normal for a man industrially employed.

The term "postconcussional syndrome" is widely used but it is submitted that it lacks specificity. How much better to particularize and indicate the nature of the brain damage causing the symptoms! It may be difficult to do, but it is obviously easier to make a more accurate diagnosis at the time of the injury or shortly thereafter than it is to do the same thing under the fire of cross-examination several years later, when memory is dim and opposing counsel refuses to accept the term which means nothing more than a conglomeration of symptoms as a substitute for a more specific diagnosis.

In a general summary of the comments relating to records, it is urged that records:

1. Be complete.
2. Be authenticated by the signature of the examiner.
3. Be prepared to withstand cross-examination.
4. Be retained until all possible need for them has passed.

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COMPENSATION

Workmen's compensation today presents many, if not most, of the medicolegal questions that must be resolved by the coordinate efforts of the company attorney and the plant physician. It is in this field that the link of interdependence is necessarily the strongest.

Despite the fact that workmen's compensation is in its fifth decade of existence, dating back to the first surviving act in Wisconsin in 1911 (although abortive attempts, all declared unconstitution, began with an elective act in Maryland in 1902), there is still much misunderstanding as to its principles and its purposes. In legal circles one of the common proverbs of the early century was, "There is no liability without fault." This cliché has given way in many instances, and one of the most notable is in workmen's compensation.

The existence or absence of negligence, a synonym for fault, is immaterial in workmen's compensation. No one except the uninformed cares whether the employer was careless or whether the accident was the result of the stupidity of the injured workman, or even his failure to wear safety equipment or abide by safety regulations. The spirit of workmen's compensation is that of social legislation; the burden was meant to be carried by an enlightened industry as a cost of production. Even as obsolete and depreciated machinery and equipment are protected for replacement by an obsolescence and depreciation reserve, so depreciated, damaged, and obsolete employees, rendered so by injury or disease, must be repaired, replaced and sustained out of the price of the article manufactured and represent a very real cost of production.

Since this is the philosophy of workmen's compensation, it is irrelevant to discuss or consider the ordinary liability factors, such as negligence and contributory negligence. The fundamental question is to determine whether an injury is related to the employment, and the various states have, by legislative flat, created rules for such determination.

The many statutes are reasonably similar on this point, making the compensability of the condition dependent upon whether it is the result of an accident or exposure "arising out of and in the course of" the employment. It should be specifically noted, however, that a few of the statutes differ, some requiring only that the injury arise by accident "in the course of" the employment; others provide, in the alternative, that the test be that the accident should arise "out of or in the course of" the employment; still others delete entirely the requirement that the incident be accidental, but provide for compensation for personal injuries "arising out of and in the course of" the employment. However, the majority follow the uniform pattern first expressed above.

Since the gateway to compensation liability is generally the conjunctive phrase "arising out of and in the course of", it is incumbent to have a better understanding of its meaning. All but one of the words are

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monosyllabic; their analysis and interpretation should be easy. This natural assumption is incorrect, and each case must be considered on its own facts and fitted to the definition. The late Mr. Justice Murphy of the United States Supreme Court termed the phrase "deceptively simple and litigiously prolific" and noted that many thousands of cases have attempted to define its limits. Perhaps the most common interpretation of the words "in the course of" is the following taken from McNicol's case, a Massachusetts decision (215 Mass. 497):

> "It is sufficient to say that an injury is received 'in the course of the employment when it comes while the workman is doing the duty which he is employed to perform."

From this definition, and thousands of cases interpreting it, it can be said that "in the course of" relates primarily to time and place, contemplating normal hours of employment and the customary place of employment. It has no reference, however, to causal relationship.

The corresponding definition of "out of" selected from the same case is:

> "It arises out of the employment when there is apparent to the rational mind upon consideration of all the circumstances a causal connection between the conditions under which the work is required to be performed and the resulting injury."

Here the question of causal relationship is considered. Assume that the accident answers the test of being "in the course of" employment in that it occurred at the normal place and during the hours of work, can it also meet the test of arising "out of" it? Was it a risk that was created or accentuated by the particular work, or could this accident or injury have happened with equal frequency had the victim been at home or on the street, with the work performed by the employee playing no part in the occurrence?

Each accident, exposure, or incident must be tested by the application of this phrase to determine whether it should be accepted as a compensable matter, and that decision in turn, at least theoretically, determines whether or not treatment should be rendered by the plant physician. In many cases, however, and as a practical matter, it is difficult to determine which decision is made first. If treatment, in fact, is rendered an employee before the legal question as to compensability is considered, such treatment alone may be very persuasive in affirmatively deciding that the case is compensable. It is a delicate treadmill and emphasizes the value of an understanding by the industrial physician of the issues involved.

Here, again, is the field of medicolegal practice. The well-rounded industrial physician is able, except in emergency cases which require immediate attention, to evaluate the history given by the patient on his initial visit and to determine preliminarily whether or not treatment should be rendered on the basis of plant responsibility.

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Some attorneys and, in fact, some courts, inevitably at any suggestion of corporate benevolence, take the position that the rendering of treatment for any condition is an implied realization and acceptance of company responsibility. This is a patently ridiculous attitude but it must be dealt with in self-defense. Two methods come to mind:

1. Refuse to treat any personal condition which is unrelated to the employment excepting, of course, emergency cases.
2. If treatment is rendered for such personal condition, utilize some type of form which can be signed by the person applying for such treatment and thus permanently record the fact that such treatment is rendered for an admittedly nonoccupational condition.

Since all compensation litigation involves the presentation of medical testimony, an awareness of the theories by which such testimony will be presented is essential. The tremendous increase in the amount and profundity of workmen's compensation litigation has not evolved any new basis for the medical case. While the primary or basic structure may be further subdivided, essentially each case will still be presented on one of three basic theories.

1. Causation.
2. Activation.
3. Aggravation.

The theory of causation is utilized when the claimant alleges, by his medical testimony, that the accident or exposure caused his condition. He alleges that he was not only symptom-free prior to the incident, but that he was also nonpathological.

Activation concedes the prior existence of pathology, but denies any prior symptoms. It is based on inciting a passive, latent condition into an active, disabling one.

Aggravation admits the prior existence of both pathology and symptomatology, but claims an exacerbation or increase in the disability due to the incident.

Since the ultimate medical theory of a claimant's case may not be revealed until the trial when testimony is taken, an active imagination, coupled with an appreciation of the possibilities, can do much in the direction of adequate defense preparation.

A single illustration should suffice. Assume that an employee has received certain burns of the upper extremities, resulting in scar tissue, and that several years later an epithelioma appears at or near the site of the burns. What theory will be presented, and what must the defense be prepared to refute? Will it be that the burn caused the cancer, or will it turn along the lines of activation? As a third alternative, is it likely that the claimant's testimony will claim an aggravation of a preexisting cancer, due to the trauma?

Further possibilities present themselves for the industrial physician's consideration. Will an effort be made to prove that the original trauma

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was the inciting factor, or will this be subordinated to the theory that scar tissue, by constant friction on adjacent healthy tissue, is to blame?

Perhaps all of these theories may be discarded in favor of a general approach. There may be an attempt to show that cancers are divided into latent and active cancers and that there is no necessary or inherent rate of growth of a latent cancer; that certain local immunities may develop that will restrain the growth of cancer and that a so-called X factor has been recognized as a restraint on growth; that trauma of any sort has been known to release that X factor and permit the development of endogenous cancerous growth.

While there is a certain amount of overlapping, it will be noticed that there were six possible theories in the foregoing illustration upon which the claimant could predicate his case by medical testimony. These and kindred medicolegal problems are not the responsibility of either the attorney or industrial physician singly, but must be considered their joint responsibility for defense in proper cases. Just as a baseball battery with only a pitcher is an anomaly, so the defense of a compensation case without close cooperation of the attorney and the industrial physician is a defense with half a team.

Much emphasis is placed today upon traumatic neurosis and its part in medicolegal work. In certain areas where workmen's compensation litigation abounds, it is the rule rather than the exception that every traumatic injury is alleged to result in neurological as well as orthopedic damage. Not only head injuries, but every finger injury, every foot injury, every back injury, every hernia, in short – all injuries – are alleged by the respective claimants to have resulted in neurological disability, and compensation is sought for the neurological involvement which allegedly accompanies the bodily insult.

Whether we approach the problem as a lawyer or as a physician, we must recognize that there is a fairly logical basis for the allegation in certain cases. If an unhappy family life, a wayward child, or job frustration can be related to a disordered nervous system – either on an organic or a functional basis – it can certainly be argued that a serious injury, with attendant pain, mortification (as in the enucleation of an eye), or even litigation anxiety may also be a factor. However, to say that each and every trauma has a measurable effect in inducing neurological disorder is stretching credulity too far, and the industrial physician must be alert to discover and record, at the time of the initial visit and throughout subsequent treatment, anything that may tend to establish or negate the existence of a traumatic neurosis in order that a just decision can be achieved in each case.

The courts, juries, and commissioners hearing industrial litigation have come to realize that each individual has a different threshold of emotional stability and that the degree of trauma involved is no absolute criterion as to the possibility of causal relationship between it and an alleged

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traumatic neurosis or psychoneurosis. One man may be feeble and his same neurological or psychiatric damage, needing only the slightest impetus to propel him into it, whereas another may withstand successfully the severest kind of ordeal.

As a corollary to the thinking expressed above, everyone who expects to be involved in compensation litigation should be aware of one of its fundamentals. It is elementary that industry, in the hiring of its employees, takes the man as it finds him. Any pre-existing weakness such as hypertension, arteriosclerosis, relaxed inguinal rings or low emotional reserve, rendering an employee more susceptible to injury or disease, is no bar to his recovery of compensation, and the mere fact that a more healthy man would not have suffered a similar fate or, indeed been involved in any accident, is unimportant and irrelevant. This becomes particularly significant at the time of the pre-employment examination, when new employees are being hired. Their predisposition to occupational injury or disease is a matter to be considered in the light of this principle of workmen's compensation law, and the hiring of such an individual must be based on the acceptance of a calculated risk.

The susceptibility to accident of any employee is thus increased by his own physical condition, including any pre-existing weakness. If we consider the word "accident" in its ordinary sense, the above statement may be hard to justify. However, "accident" has undergone a metamorphosis: whereas, it formerly included only unforeseen, fortuitous, and catastrophic events, today it is much more liberally interpreted under our workmen's laws. The majority rule today, and the trend everywhere, is to consider any injury "accidental" if the result is unexpected or unforeseen. In other words, it is not the extraneous circumstances that determine a given set of facts to be an accident, but rather the effect upon the individual and his physical well-being.

Under this category the following have been held to be "accidental": frost-bite, sun-poisoning, back-strain as the result of normal work, coronary occlusion suffered by a hypertensive while walking up a flight of twenty-six steps, an occlusion suffered by an employee of a fish market while sweeping the floor, a strangulated hernia suffered by a fireman while stoking a furnace. In short — even though the extraneous and surrounding circumstances are normal — if the effect of such effort on the individual resulted in physical damage, the overwhelming weight of authority holds it to be an accidental injury.

REMARKS

The social concept that lies behind workmen's compensation is exemplary, and the theory that the risk of inevitable injury in industry should be transferred from the shoulders of the workman to the buying public as a cost of production cannot be seriously challenged. As with all social

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gains, the momentum can carry the pendulum too far, and the basic problem for industry is to keep the cost of production down by litigating and defeating unwarranted, exaggerated, and fictitious claims, and by safety and health programs that will lessen the frequency of such litigation.

The difficulty of successfully accomplishing that end has not been lessened by the change in public thinking that has taken place in the past generation. We find an increasing liberality in the administration of our statutes, with an added factor being noted in many cases in the nature of a desire to award a sum of money to the claimant at the expense of the defendant for no other reason than the better ability of the latter to pay. The representatives of industry who find themselves involved in industrial litigation should be aware of the existence and importance of this factor, as it must be dealt with in an accurate appraisal of any case. We may not like the change in world thinking, but to deny its existence is to invite errors in judgment.

In litigation matters, medical theories that may seem untenable will be urged by claimant's counsel and his witnesses in an effort to establish the compensability of a case. No matter how bizarre such theories may seem, they must be respected for purposes of litigation. The court, jury, or commissioner routinely fail or refuse to evaluate the respective abilities of opposing experts, and the mere fact that claimant's expert presents an unusual theory will not lessen its credibility. The best, and perhaps only, method of defeating a claim based upon what the industrial physician believes to be an untenable theory is by a thorough knowledge of the facts and medicine involved. Laughing the theory out of court never works. Regardless of how erudite and important your witnesses may be, the lesson will be learned early that there is no halo around their heads in the court room. Plaintiff's witnesses will undoubtedly appear just as credible to a court or jury. For that reason, it is imperative that the witness do nothing to upset this delicate balance unfavorably by his action on the witness stand. A sincere, courteous demeanor, with a serious rather than jocular treatment of the case, a continual equanimity with no unjustified show of ill temper, markedly but firmness of conviction, are all assets to the expert witness. If our witness is blessed with an agreeable personality, he should carry it with him to the stand and not shed it at the counsel table as so often happens. If his normal personality is less than agreeable, he must remember that, in addition to giving learned information as an expert and fact testimony from his records, he must also do a selling job from the witness stand, and for that purpose and for the limited period of time involved, he should be on his good behavior and be as personable as possible.

From his intimate contact with the industrial physician in and out of litigation over a period of many years, the author has reached a few conclusions concerning the role of a plant doctor.

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problem. Some dusty trades are not, however, readily amenable to engineering control. Mines are notorious in this respect and currently continue to contribute many new cases. Even in such industries the prevalence rates, speed of progression, incidence of complications, and life expectancy have been favorably modified by partial dust suppression, pre-employment and periodical medical examinations, isolation of tuberculous subjects and earlier exclusion of silicotic workers from high hazard areas.

Aluminum inhalation has been added as a valuable adjunct in those industries where dust suppression cannot be achieved. Used prophylactically, pulmonary oxidized metallic aluminum inhaled as an aerosol for fifteen minutes twice daily, before and after the working shift, has prevented or retarded silicosis in some 40,000 miners, foundry workers and ceramic industry employees whose health would have remained in serious jeopardy because of prevailing dust exposures. Aluminum inhalosis has also proved valuable in mitigating, retarding or preventing the progression of established silicosis. The validity of this method has been established by extensive animal experimentation, and there is no risk attendant on its proper use.

No other known medicinal aids, other than palliative agents, exist. Generally, it is better to keep the silicotic physically active as long as possible in order to retard fibrotic contraction of the lung. After emphysema, tuberculosis, or cor pulmonale have supervened, some reduction in physical exertion becomes necessary. Complete bed rest, however, often hastens the patient's demise. The decision as to which course to follow is often very much a Hobson's choice.

II. SILICATOSES

While the foregoing discussion has been concerned primarily with the disease produced by inhaled silicon dioxide, the silicates present an extensive array of naturally occurring minerals widely used in industry. With the exception of the fibrous silicates, the majority of the silicates are relatively inert, exciting little more pulmonary reaction than phagocytosis. This is the reason for stressing the importance of free versus combined silica. Some of the silicatoses can, however, be as serious as the most severe case of silicosis.

Asbestosis. This is by far the most important among the silicatoses. Six varieties of asbestos fibers are commonly implicated, viz. chrysotile, amosite, crocidolite, anthophyllite, tremolite and actinolite. Since more than 90 per cent of the asbestos used in the U.S.A. is chrysotile, cases occurring in this country are most probably chrysotile-derived. Crocidolite is more commonly mined and used in Europe and Africa. This information leaves scope for precise identification of the industry which has caused the asbestosis since the different types of asbestos fibers can be distinguished in biopsy or autopsy material.

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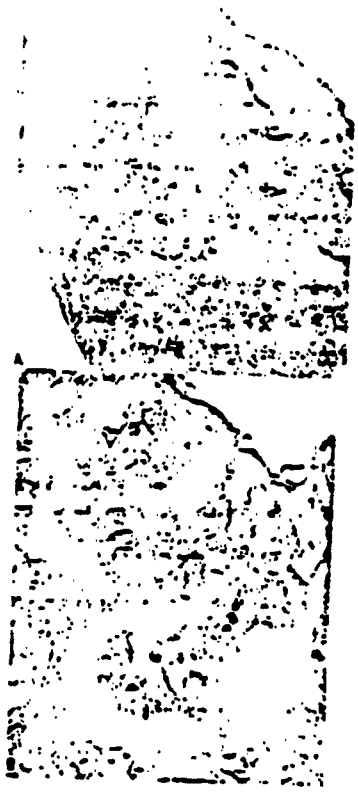

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FIG. 38

ASBESTOSIS

HISTOPATHOLOGY

A. DOMINANTLY SUBPLEURAL ASBESTOSIS. The main zone of collagen deposition is in the subpleural zone on either side of the lamina elastica. Fibrous subacinar dip toward along venous channels. Dilated bronchi within areas of condensation. Disposed fine focal perivascular fibrosis. *Miner exposed to chrysotile dust 32 years.* Initially symptomless. Died of cor pulmonale.

B. ADVANCED ASBESTOSIS. Dense linear perivascular fibrosis with minimal vesicular emphysema. *Textile worker exposed to amosite dust 11 years.* Progressive exertional dyspnea. No cough. Died of cor pulmonale.

C. TALCOSIS. Irregular interstitial fibrosis, mainly perivascular and perihaemic. Marked dimension of lower bronchi and bronchioles. *Mill worker exposed to monolite talc dust 17 years.* Terminal progressive dyspnea and productive cough. Died of cor pulmonale.

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Asbestosis is provoked wherever exposure to asbestos dust occurs. However, in textile industries there appears to be a greater prevalence of severe disease. In contrast with silicosis, the asbestotic reaction in the lung is excited not by mineral particles but by relatively long fibers. Thus, if asbestos is pulverized to particles no longer than 5 microns, very little disease develops and the process advances much more slowly than when the aerosol contains a predominance of fibers of 10 to 50 microns. Again, if the fibers are introduced into the lung in a high caliber form (e.g. compound multiple fibrils), less disease results than when the individual fibers are split into their component fibrils of submicron caliber.

Asbestosis is essentially an interstitial pulmonary lesion in which all component tissues are involved with focal emphasis of the process. The lesions range in severity from mere alveolar mural cellular infiltration of a microscopic nature, not detectable by radiography or even on gross anatomical examination, to massive consolidation with associated vascular occlusion, bronchiectasis, and carnification.

The interstitial lesions result after fine short fibers have been ingested by phagocytes and transferred into the lymphatic channels of the alveolar septa. Here fibrocytes proliferate, new capillaries form and reticulin, collagen and new elastic fibers are laid down. This type of lesion cannot readily be distinguished from other interstitial pneumonitides unless the asbestos fibrils are detected by oil immersion or electron microscopy. Rarely, some of these fibrils will be rendered obvious by conversion into an asbestos body through proteinaceous encapsulation and ferrous pigmentation. If the disease remains limited to this stage, disability may remain limited being merely due to increased work of breathing.

With extension of the process three major complications arise. There may be progressive invasion of venous adventitia with perivascular fibrosis, intimal hyperplasia, and ultimately occlusion of the vascular lumen. Relatively large venous channels may become involved but arteries tend to escape early damage. This obstruction to the venous channels leads to progressive elevation of pulmonary venous and marked cor pulmonale. Heart failure is the usual cause of death in these cases. The asbestotic origin of the venous obstruction is readily proven by demonstrating the asbestos fibers in the peri- and endovascular granulation tissue. Perl's stain often is sufficient for this purpose as it will demonstrate the presence of iron deposits in the proteus sheaths around the asbestos fibers. Another tell-tale feature consists of the deposition of abundant perivascular elastic laminae and fibers.

While the stage of the alveolar mural invasion is not radiographically detectable, except for blurring of lower pulmonary markings, the stage of perivenous fibrosis is characterized on the x-ray by the development of a coarse web and the effacement of the normal vascular pattern. The

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extent of the latter damage is often underestimated until tomography or angiography are resorted to.

Massive fibrosis tends to develop mainly in the parts of the lung which are in constant agitation, such as the lung apex, the supradiaphragmatic zone of the basal lobes, and the para-cardiac components. The consolidation is effected by progressive interstitial invasion, atelectasis, trapping of fibers within alveolar spaces where phagocytes surround them and organization takes place, and by abundant pleural thickening on both sides of the lamina elastica. In these zones carnification is often so complete that all semblance of pulmonary architectonics is completely lost. Major blood vessels can only be identified by elastic stains which reveal their elastic skeletons.

Radiographically, these consolidated zones present as diffuse and sprawling opacities along the upper mediastinum, above the diaphragm and on either side of the heart, whose silhouette becomes blurred: the so-called "shaggy heart" feature. Calcification may occur in these dense zones.

Symptomatically, these patients usually are merely dyspneaic on exertion unless there is marked accompanying vascular obstruction. In such cases cor pulmonale will be the predominating lesion. The thorax is usually small and immobile while breath sounds, vocal fremitus and resonance are markedly impaired over the affected zones. Bronchitis and bronchiolitis are not common in asbestosis, though some of these air passages may become involved in the fibro-granulomatous reaction. Advanced cystic bronchiectasis may develop as a result of the fibrotic distortion of the lung. At first there is mere bronchial distension and some fluid or mucus may become trapped in the dilated ducts. Later infection supervenes with all the attendant inflammatory processes leading to chronic suppurative bronchiectasis. The symptoms in these cases do not differ much from conventional varieties of bronchiectasis except that hemoptysis is less common and cor pulmonale develops early.

Tuberculosis may occur in asbestotic subjects, but is not more prevalent among these than it is in the general population. The lesions are not any more severe than those usually seen and often are limited by the fibrous barriers caused by the asbestosis.

Pulmonary carcinoma has been observed with such high frequency in employees of the asbestos industry that a causal relationship has been accepted by most authorities. Indeed, pulmonary carcinoma is compensable as an occupational disease in England, Germany and South Africa. Most of the carcinomata are squamous cell epitheliomata. Pleural mesotheliomata is also quite prevalent, especially in crocidolite industries.

Since the lymphatic system is not completely destroyed in asbestosis, a neoplasm may spread diffusely throughout the lung in spite of extensive interstitial fibrosis. This does not readily occur in silicosis.

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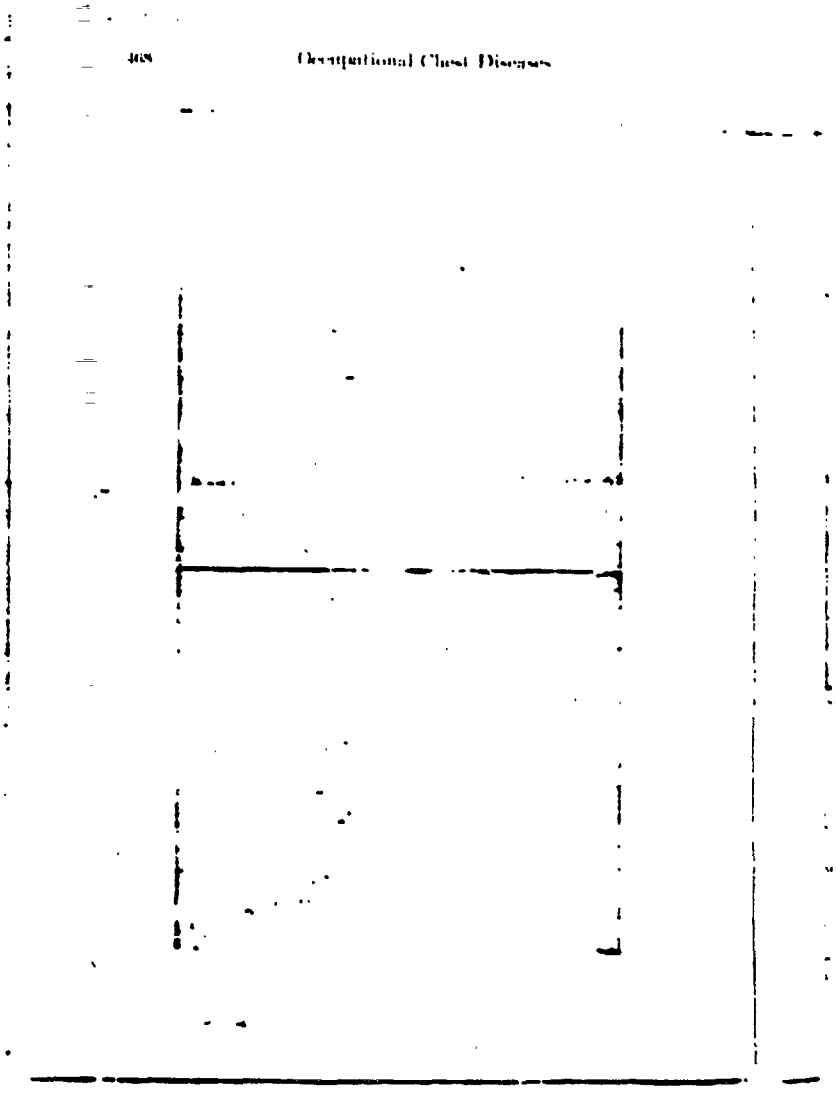

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Asbestos bodies not infrequently are demonstrable in the sputum, where they may serve as a diagnostic clue. However, severe asbestosis is compatible with their absence. Again, asbestos bodies may be quite prevalent without coexisting asbestosis. This is possible because the majority of asbestos bodies are formed in the alveolar spaces when fibers have become engulfed by phagocytes. These asbestos bodies may become incarcerated in granulomatous tissue by atelectasis and secondary inflammation.

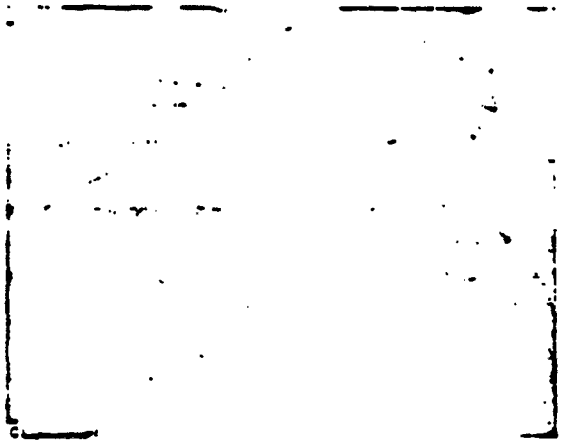

FIG. 37

ASBESTOSIS

RADIOGRAPHIC FEATURES

A. EARLY CONVEXITIVE ASBESTOSIS. Linear pulmonary fibrosis and ground glass opacification. Commencement shrinkage of upper lobes. Incipient cardiomegaly. Diaphragmatic adhesions. Textile worker heavily exposed to chrysotile dust 7 years. Slight exertional dyspnea and tachycardia. Discontinued work.

B. ADVANCED CONVEXITIVE ASBESTOSIS. Marked interstitial infiltration. Regional opacification. Paracardiac infiltration and "shaggy heart." Reduced lung volume. Marked cardiomegaly. Same case as A, 6 years after discontinuation of exposure to chrysotile dust. Marked cardiopulmonary disability. Died less than a year later from advanced chronic cor pulmonale.

C. ADVANCED CONVEXITIVE ASBESTOSIS. Extensive bilateral diffuse infiltration of lung fields. Thickened pleura and subpleural cyst. Part of peripheral opacification due to congestive cardiac failure. Heart markedly enlarged. Negro miner exposed 6 years to crocidolite dust. Marked dyspnea and anasarca. Died two years later from pulmonary carcinoma at the age of 35.

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In industries in which asbestos is handled extensively, warts are not uncommonly experienced at points of cutaneous trauma, e.g., hands, collar, belt, cuff zones. Many of these acanthotic lesions are indistinguishable from benign warts, but occasionally the telltale carcinoma asbestos fibers are identifiable. Later these warts, if left untreated, have a strong tendency to progress to malignant anaplasia. They should, therefore, be radically extirpated as soon as they are identified.

Important features in all cases of asbestosis are the existence of a lag period before the onset of the symptoms and the inexorable progression of the disease despite cessation of dust exposure. There is evidence to support the view that the first few years of exposure to asbestos fibers are as important determinants of the final outcome as are most of the remaining years of exposure. Thus, in one verified case, a single month of exposure to asbestos dust in a textile plant in one country was followed thirty years later in another country by fulminating fatal asbestosis. The intervening period was one of good health and no further asbestos dust exposures had occurred. Continued exposure will, of course, aggravate the process. The severity of the asbestosis is thus a function of the duration of the exposure multiplied by the period which has intervened between the inception of exposure and the onset of the disease. This principle at the same time accounts for the progression of asbestosis after exposure has been discontinued. Cases are currently cropping up in men and women who had worked as pipe insulators on liberty ships during the second world war without since having had any further asbestos exposure.

Talcosis: Two varieties of this condition occur. The first is caused by tremolite talc and the disease is essentially asbestosis with the same range of severe lesions found after chrysotile, amosite or crocidolite exposures. Talc bodies similar to asbestos bodies also occur. This condition should be carefully distinguished from the pulmonary reaction to other forms of commercial talc which may consist of pure magnesium silicate (alpine talc) or may be a mixture of the latter with anauxite, chromite, dolomite, magnesite, and spinell. Most of these substances are relatively inert. When breathed in massive quantities, and especially where there is some pre-existing pulmonary lesion (e.g. virus pneumonitis), they may cause respiratory incapacitation, usually transient, through extensive alveolar mural cellular infiltration. Because of the multiplicity of uses of talc, such cases may occur in diverse industries where talc is used as a slipping agent (e.g. paper mills, rubber industry). These pathogenic talcs have no place as surgical or toiletry accessories.

Rock Wool is a synthetic fibrous silicate extruded from slag. It is extensively used as an insulating material. When its long fibers are inhaled repeatedly and in great quantity, pulmonary lesions closely similar to those seen in asbestosis may result. Once more, the clue to diagnosis is the discovery of "rock wool bodies" in the granulation tissue

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problem. Some dusty trades are not, however, readily amenable to engineering control. Mines are notorious in this respect and currently continue to contribute many new cases. Even in such industries the prevalence rates, speed of progression, incidence of complications, and life expectancy have been favorably modified by partial dust suppression, pre-employment and periodical medical examinations, isolation of tuberculous subjects and earlier exclusion of silicotic workers from high hazard areas.

Aluminum inhalation has been added as a valuable adjunct in those industries where dust suppression cannot be achieved. Used prophylactically, pulmonary oxidized metallic aluminum inhaled as an aerosol for fifteen minutes twice daily, before and after the working shift, has prevented or retarded silicosis in some 40,000 miners, foundry workers and ceramic industry employees whose health would have remained in serious jeopardy because of prevailing dust exposures. Aluminum inhalation has also proved valuable in mongrang, retarding or preventing the progression of established silicosis. The validity of this method has been established by extensive animal experimentation, and there is no risk attendant on its proper use.

No other known medicinal aids, other than palliative agents, exist. Generally, it is better to keep the silicotic physically active as long as possible in order to retard fibrotic contraction of the lung. After emphysema, tuberculosis, or cor pulmonale have supervened, some reduction in physical exertion becomes necessary. Complete bed rest, however, often hastens the patient's demise. The decision as to which course to follow is often very much a Hobson's choice.

II. SILICATOSES

While the foregoing discussion has been concerned primarily with the disease produced by inhaled silicon dioxide, the silicates present an extensive array of naturally occurring minerals widely used in industry. With the exception of the fibrous silicates, the majority of the silicates are relatively inert, causing little more pulmonary reaction than phagocytosis. This is the reason for stressing the importance of free versus combined silica. Some of the silicatoses can, however, be as serious as the most severe case of silicosis.

Asbestosis. This is by far the most important among the silicatoses. Six varieties of asbestos fibers are commonly implicated, viz. chrysotile, amosite, crocidolite, anthophyllite, actinolith and tremolite. Since more than 90 per cent of the asbestos used in the U.S.A. is chrysotile, cases occurring in this country are most probably chrysotile-derived. Crocidolite is more commonly mined and used in Europe and Africa. This information leaves scope for precise identification of the industry which has caused the asbestosis since the different types of asbestos fibers can be distinguished in biopsy or autopsy material.

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