

Litigation Management, Inc.'s

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Reporter

THE REVIEW OF MEDICAL INFORMATION MANAGEMENT FOR LITIGATION

Published as an Educational Service to the Corporate, Insurance, and Defense Legal Community by Litigation Management, Inc.

Volume VIII, Number 1

June 2005

DISTINGUISHING BETWEEN ASBESTOSIS AND SILICOSIS: *A Lawyer's Primer on the Proper Diagnosis of Each Disease*

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The aim of this paper is to identify a broad range of tests and findings in medical records that defense attorneys will need to master in order to discuss the case in depth with their chosen expert witnesses, as well as perform effective cross examination of plaintiffs' witnesses. Mentioning any aspect of the diagnosis of asbestos-related or silica-related maladies, particularly the more controversial findings, should not be interpreted as the authors' endorsement of its relevance or efficacy.

Asbestos-related disease claims have been clogging American courthouses — but not doctors' offices — since the 1970's. In the past, progressive judges attempted innumerable "fixes," none of which succeeded in clearing their dockets permanently. Even the litigants themselves tried to orchestrate a "global" settlement mechanism, but it was rejected by the United States Supreme Court — thus perpetuating the morass.

Now rumors are rife that the United States Congress, at long last,

will legislate an end to the litigation crisis before any (or many) more bankruptcies take their toll on the owners, investors, and insurers of some of this country's largest and most essential businesses. Equally widespread are rumors of the inevitable demise of the "Fairness in Asbestos Injury Resolution Act of 2005" (the "FAIR Act"), because the three chief constituencies affected — former producers of asbestos and asbestos-containing products, their insurers, and organized labor — cannot agree on (1) how many dollars the trust fund will need to compensate potentially hundreds of thousands of claimants; (2) which companies should contribute; (3) what amounts (or tiers) individual payers should contribute; and (4) over what period of time. This may be the classic example of the original proponents regretting what they wished for and launching a circular blame game to explain the impasse.

The Rand Corporation's Institute for Civil Justice estimated that, through 2002, 730,000 people filed asbestos

bodily injury claims, and defendant companies and insurers paid \$70 billion in settlements and court judgments — with 59 percent of this amount going to the attorneys on both sides. An additional 100,000 asbestos-related lawsuits were filed in 2003, and about 600,000 claims are pending. Actuaries project that, without government intervention, the cost of asbestos injury litigation could reach \$265 billion. It is little wonder that certain entrepreneurial lawyers and operators of mobile screening trailers continue to salivate over the potential profits. They are not above testing medical and legal limits to bring more claimants into the system, nor are they above using the same tactics to increase case filings in silica litigation.

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Prognosticators at both ends of the trial table suggest that even if “asbestos” goes away tomorrow, “silica” waits in the wings of the mass torts arena. It is precisely this dual disease issue that has most recently derailed swift passage of the FAIR Act.¹

Asbestos and silica have been used since Biblical times in countless industrial, commercial, and residential applications and products. Both are naturally occurring minerals in virtually inexhaustible supply. Both are relatively inexpensive and thus desirable ingredients in many thousands of products. Neither substance, nor the illness claims they spawn, is going to exit the stage of American courtroom drama any time soon. To equip juries with the facts to reach correct verdicts, defense lawyers first must understand which diagnostic tools are employed to differentiate between illnesses caused by asbestos and silica. If the attorneys do not perceive the differences, they cannot educate juries on the subtle differences between asbestosis and silicosis. Uninformed jurors might saddle one industry with paying for the illnesses of the other — or even worse, allow double windfalls for a single, distinct condition.

The relatively recent wave of silica-related lawsuit filings around the country, perhaps not surprisingly, has increased in direct proportion to rumors that the FAIR Act of 2005 would be enacted. Similarly, the up-tick in silica cases might be linked to asbestos-related tort reform legislation in several of the more active and problematic states. For example, in Texas, Mississippi, Georgia, Illinois, Ohio, and Florida, there are ongoing efforts to tighten venue rules, eliminate forum shopping, and require objective signs of impairment before an asbestosis or pleural plaque claim receives a trial date. Are the silicosis claims “real,” or are some folks hedging their bets, should an asbestos compensation fund or other tort reforms leave them out in the cold? Paradoxically, silica-related deaths are decreasing,² while silicosis case filings are skyrocketing. The silica plaintiffs’ bar seems intent on following the path of least resistance by forcing the round peg of silicosis into the square hole of asbestosis. Fitting them both into the same mold will not do justice to either disease and lead to unnecessary frustration and confusion by courts, attorneys, and jurors alike.

Mass screening for asbestos-exposed workers proved so lucrative, that it was little wonder that the same laboratories, the same clinicians, the same x-ray readers, and the same law firms went back to the same well to tap into the same reservoir of alleged dust-disease victims. Mobile screening vans, with portable x-ray and spirometry equipment sharing space with the sponsoring lawfirm’s intake desk, were parked at union halls, motels, and shopping centers around the country. Reportedly, the persons screened were not asked to pay for the tests. No one can argue that such a one-stop operation is very “customer-friendly.”

Positive test results could turn a patient into plaintiff, before the mobile “lab” drove to its next location.

The screenings focused heavily on chest radiographs that were funneled to a few select “B-readers.” A relatively recent phenomenon has the B-reader who looks for silica exposure also documenting asbestos exposure, and vice versa. If the x-ray is not clear for either mineral, the increasingly popular diagnosis of mixed-dust pneumoconiosis (“MDP”) may be recorded. Re-screening of individuals already diagnosed with a nonmalignant asbestos lung disease (“ALD”) is turning up diagnoses of silicosis or MDP, either in an heretofore missed conjunction of maladies or, even more remarkably, only evidence of silicosis and no trace of asbestosis in previously diagnosed cases of ALD. This finding has led to a joke making the rounds among counsel handling dust-disease cases: the cure for asbestosis finally has been found — it’s silicosis!

Although a topic meriting its own article, it bears mentioning here in passing that at least one court is turning an increasingly critical eye upon the legitimacy of mass screenings. Federal District Judge Janis Graham Jack presides over In Re: Silica Products Liability Litigation, MDL No. 1553 (S.D.

Texas), encompassing nearly 10,000 silica injury claims. In mid-February Judge Jack conducted three days of well-publicized hearings into the accuracy and propriety of silica screenings.³ She was clearly shocked by the potentially fraudulent practices conducted

by several prominent screening outfits and B-readers. Defense lawyers have filed a motion seeking \$1.1 million in sanctions from the plaintiffs’ lawyers for “knowingly submitting and advocating bogus diagnoses.” Judge Jack is expected to issue a 100-page opinion on these matters to guide other courts around the country, particularly those courts that may receive the cases on remand. More conscientious jurists like Judge Jack are needed to shut down this mass tort locomotive completely before it leaves the station. Can lessons learned from the wholesale run-up of unimpaired ALD suits be implemented in time to avoid repeating the same mistake for silica exposures?

Despite certain similarities in exposure settings, diagnostic tools, and symptomology, asbestosis and silicosis are quite distinctive forms of pneumoconiosis.⁴ As far as similarities, both asbestos and crystalline silica (quartz) exposure can occur in a wide variety of occupational settings. The development of the respiratory symptoms associated with both of these minerals is dependent upon both the intensity and duration of the exposures (many significantly exposed workers, however, will never develop any occupational lung disease). Thus there are similarities between asbestos and silica, and the nonmalignant diseases associated with them. It is the differences between them, however, that are the focus of the remainder of this article.

“Paradoxically, silica-related deaths are decreasing, while silicosis case filings are skyrocketing.”

I. Comparison Between Asbestos/Asbestosis and Silica/Silicosis

Significant occupational or environmental exposures to asbestos and silica can cause nonmalignant respiratory disease — asbestosis and silicosis, respectively. As mentioned earlier, the size and shape of the dust particle as well as the proximity of the exposed individual to the dust source are key to the development of the illness. Dose is not simply the concentration of asbestos or silica in the atmosphere; rather, it is the number of particles or fibers in the subject's breathing zone actually inhaled and deposited in the periphery of the bronchial tree. Lungs are not harmed without inhalation of the dust.

Silicosis and asbestosis are both interstitial lung diseases. Interstitial lung disease, or interstitial pulmonary fibrosis, is a generic term that includes a multitude of chronic lung disorders, many caused by inhaled particulates, others by genetic abnormalities, and still others of either idiopathic or unknown origin. Virtually all such maladies produce a common patient complaint or symptom — shortness of breath, sometimes reported in medical records as “dyspnea on exertion.” This is simply feeling more breathless than similarly situated persons with healthy lung function, or than the patient experienced prior to the onset of disease. Because these diseases share certain common features, the comparison between these two diseases can generate confusion among lawyers, judges, and juries. Most independent medical experts would agree, however, that, outside of the litigation setting, clinicians are rarely confused in diagnosing silicosis or asbestosis.⁵

Most of the other diseases — particularly the malignancies — associated with either asbestos or silica inhalation lack the common features of the pneumoconioses. Asbestos exposure had been causally associated with diffuse malignant mesothelioma, for example, but silica exposure has not. Silica exposure has been associated with several autoimmune disorders, and even tuberculosis (“TB”). Diseases such as scleroderma, systemic lupus erythematosus (SLE), or TB are not caused by asbestos. Lung cancer arguably, especially in the litigation context, may be an area of dual association. Although lawyers may argue that lung cancer can be attributed to either asbestos or silica exposure, unbiased scientists tend to disagree. There is wide agreement among experts in pulmonary diseases that lung cancer in an individual (even a smoker) with full-blown

asbestosis can be attributed in part to inhalation of asbestos fibers. However, there is no such consensus over whether exposure to silica has the ability to induce lung cancer.

A. Silica and Silicosis

Silica — also known as highly purified quartz or silicon dioxide (SiO₂) — is the most common and abundant mineral in the earth's crust. It is common sand, found in abundance in gravel and all types of soils. Our beaches are nothing more than natural silica deposits, and silica is a constituent of the vast majority the earth's rocks. Silica is also commonly found in mines. Commercial applications of silica are virtually limitless. It has been used in foundry, sandblasting, and countless other manufacturing operations. If crystals of silica or sand are split into tiny enough particles, they can be inhaled deep into the lungs and later pose health risks.

The most common disease associated with silica exposure is silicosis (nodular pulmonary fibrosis). Silicosis can be asymptomatic and does not inevitably progress in severity. In essence, if an individual breathes in enough microscopic crystals of silica, the natural defense mechanisms of his respiratory system can be overwhelmed.⁶ Silicosis depends upon dose — the intensity and duration of exposure. It is “dose-related,” meaning the higher the amount of inhaled silica, the higher the risk of developing the disease. Confounding the prediction equation, however, is the wild card of individual variation or biosusceptibility among humans. This is seen, for example, in smokers, where the ten-year one-pack-a-day smoker may develop lung cancer, while the three-pack-a-day smoker shows no signs of illness. Because of differences in dose and variability of hosts, making accurate projections of disease is not possible, even from known levels of silica exposure.

Silicosis can occur in three different forms, depending upon the airborne concentration of respirable crystalline silica: chronic silicosis (subdivided into simple or complicated forms), accelerated silicosis, and acute silicosis.

David Weill, M.D., Associate Professor of Medicine in the Pulmonary and Critical Care Division at the University of Colorado Health Sciences Center, in his statement before the Senate Committee on the Judiciary Concerning Asbestos: Mixed Dust and FELA issues, explained:

Chronic simple silicosis is the most common form of the disease in the United States today. Chronic

“Despite certain similarities in exposure settings, diagnostic tools, and symptomology, asbestosis and silicosis are quite distinctive forms of pneumoconiosis.”

silicosis, in its simple form, typically requires more than twenty years of moderate exposure. It is characterized by rounded nodules, like tiny marbles, principally in the upper lobes of the lungs. In its lower grade forms, simple silicosis does not generally result in respiratory impairment. It may progress, or become more serious over time, especially in workers who continue to be exposed to silica. However, progression tends to be slow and depends on several factors.

In the complicated form of chronic silicosis, the small rounded nodules found in simple silicosis form conglomerate lesions, whose largest diameter exceeds [one] centimeter. Most patients with complicated silicosis have respiratory impairment with abnormal pulmonary function test findings typically indicating lung “restriction” or a reduction in the lungs’ capacity for inhaled air. Accompanying airway obstruction is not uncommon. Rare today, accelerated silicosis results from higher exposure to silica, usually over a period of five to ten years. This form of the disease may progress whether or not continued workplace exposure occurs. Chest x-rays can show either a pattern of small rounded nodules alone or in conjunction with larger conglomerate opacities.

These patterns may be described as small areas of inflammation and / or scarring. “Complicated” silicosis typically occurs in less than five percent of simple silicosis cases. Chronic simple silicosis is the most common diagnosis of those workers bringing claims after a mass screening; as such, it will be the main focus of the comparison to asbestosis.

“Accelerated” silicosis can develop five to ten years after initial exposure to silica. Individuals with accelerated silicosis can experience breathlessness, weakness, chest pain, cough, and sputum production. The radiographic appearance and symptoms of accelerated silicosis and acute silicosis are similar, but the clinical and radiographic progression of accelerated silicosis is rapid. Unlike chronic silicosis, accelerated silicosis forms an irregular fibrosis appearance. Accelerated silicosis can be serious. When death occurs from accelerated silicosis, it is usually caused by hypoxic respiratory failure.

In his statement before the Senate Committee, Dr. Weill explained acute silicosis (sometimes referred to as “silicoproteinosis”) as:

... [A] rapidly progressive, fatal disease. It occurs after massive exposures, over a relatively short period of time, from several months to a few years. In acute silicosis, the spaces in the lung where oxygen

exchange takes place (the “alveoli”) become filled with fluid and cells.

Accelerated and acute silicosis are rarely found in developed countries today, although there have been case reports many years ago of acute silicosis among sandblasters and workers drilling through silica-containing rock.

Some of the typical occupational exposures to silica include: sandblasting, foundry work, refractory work, silica flour work, glass manufacturing, ceramic manufacturing, mining, quarrying or tunneling, stone cutting, abrasive manufacturing, and abrasive blasting.

B. Asbestos and Asbestosis

“Asbestos” is not a precise scientific term. Rather, it is used to describe certain “families” of fibrous silicates, most with commercial applications. A fiber is characterized as having an aspect ratio in which the length is three or more times the width. Asbestos-bearing rock requires extensive mining and milling to remove the waste and to separate the fiber bundles into acceptable grades and sizes. It makes an excellent binder or matrix for many products and is an excellent heat insulator. It is flame resistant and impervious to attack by many common acids and corrosives. For years, it was called “The Magic Mineral” and was incorporated into countless products as varied as theatre curtains, oven pads, cigarette filters, and spray-on fireproofing.

There are two main branches in the asbestos family tree: serpentine, where chrysotile (white) is the sole fiber type, and amphibole, consisting of both amosite (brown) and crocidolite (blue) fiber types. These three fiber types have the most commercial uses. The noncommercial amphiboles include anthophyllite, actinolite, and tremolite — the latter being a common contaminant of chrysotile deposits. Serpentine fibers are relatively long and curly. The amphiboles by comparison are shorter and more “spiky.” Size is relative. “Long” might be 25 to 30 microns and short fibers are less than five microns in length. Respirable particles (not fibers) of silica by contrast are generally about ten microns across.⁷ Sophisticated light and electronic microscopy techniques are essential to visualizing individual fibers or particles in either environmental samples or in lung tissue.

Whereas silicosis is characterized by the presence of small nodules of scar tissue, usually in the apex of the lung, asbestosis involves diffuse interstitial fibrosis — typically bilateral at the bases of the lung parenchyma. The lesions form where gas exchange occurs, and the pattern is called intra-alveolar or peribronchiolar scarring. Chest x-rays of lungs of asbestotics show a hazy, ground-glass appearance of irregular, reticular (linear) densities. As its name implies, only “as-

bestos” causes asbestosis. All forms of asbestos fiber — serpentine and amphibole — are implicated, and all asbestos diseases are dose-related.

Asbestosis most often causes an objectively measurable confirmation of the patient’s complaint of shortness of breath; namely, a “restrictive ventilatory defect” on pulmonary function tests (“PFTs”).⁸ In its recent and somewhat controversial pronouncement, the American Thoracic Society (“ATS”) re-issued diagnostic criteria for nonmalignant asbestos lung disease. This 2004 document indicated that asbestosis also can manifest as an obstructive ventilatory defect — where the rate of airflow, as opposed to the volume of air, is reduced. These new ATS criteria even permit “mixed restrictive/obstructive” PFT results to support a diagnosis. Only time will tell whether the new ATS criteria will find as wide acceptance as its 1986 version.⁹ Asbestosis can also lead to a decrease in diffusion capacity (DLCO < 80% of predicted value), although numerous other pulmonary conditions also impair gas exchange, such as lung damage from cigarette smoking — chronic obstructive pulmonary disease (“COPD”) and emphysema.

C. Possibility of Having Both Diseases

Dr. Weill’s statement before the Senate Committee echoes the views of Drs. Paul Epstein and Laura Welch at footnote 5 herein:

Although asbestosis and silicosis are different diseases that look different on x-ray films, it is theoretically possible for one person to have both diseases. A person could be exposed to both silica and asbestos in sufficient quantities to cause either disease, but it would be extremely unusual for one person in a working lifetime to have sufficient exposure to both types of dust to cause both diseases.

II. Elements of the Differential Diagnoses

A. Overview

In general, because lung tissue often is not available in suspected cases of either silicosis or asbestosis, the diagnosis of either condition is based upon four interrelated clinical criteria: (1) a reliable **occupational history**, including sufficient latency (the appropriate time interval between exposure and detection of disease) to cause disease; (2) a **physical examination**; (3) the presence of characteristic **chest x-ray** evidence, or radiographic abnormalities consistent with the disease; and (4) the **exclusion** of other pulmonary diseases that can mimic either disease radiographically (e.g., sarcoidosis, fungal disease, berylliosis). Although a physician does not rely solely on a chest film to diagnose either illness, clear dis-

tinctions in the appearance and predominance of scarring caused by asbestosis, compared to silicosis, are well known. The chest x-ray alone is the tool that differentiates the shapes and patterns of opacities. Although several important differences exist that can distinguish radiographically between silicosis and asbestosis, there appears to be a predisposition among certain medical experts to ignore or minimize these differences.

Again, Dr. Weill explained it succinctly:

In evaluating pneumoconioses, chest x-rays are normally interpreted using the International Labor Organization (“ILO”) radiograph classification system.¹⁰ The purpose of the ILO system was to standardize the interpretation of chest x-rays using descriptions of the size, shape, and degree of involvement (i.e., the profusion) of radiographic abnormalities. The system is used to describe shape (regular or irregular) and size (regular: p, q, r, and irregular: s, t, u) characteristics of radiographic abnormalities. The extent of radiographic abnormalities (profusion) is numbered from normal (or 0) to increasingly abnormal (1, 2, and 3). The ILO classification scheme also addresses which of the six lung zones are involved (upper, middle, and lower in either the right or left lung). Also, particularly important when distinguishing between asbestosis and silicosis, the presence and type of pleural abnormalities are noted.¹¹

As discussed above, chronic (simple) silicosis produces silicotic inflammatory lesions, which appear as tiny round opacities, primarily in the upper zones of both lungs. Dr. Weill explained that:

“[o]n an x-ray, these nodules show up as small, rounded opacities, which would be rated on the ILO form as p, q, or r. In contrast, asbestosis is characterized by linear parenchymal fibrosis, which shows up on an x-ray as small irregular opacities [and rated on the ILO form as] (s, t, or u), primarily in the lower lobes of both lungs.

According to Dr. Weill:

[c]hronic (complicated) silicosis is even harder to confuse with asbestosis on chest x-rays than is the simple form of the disease. In complicated silicosis, the tiny round nodules found in simple silicosis join together and the opacities that show up on the x-ray film are large....

Asbestosis does not appear on x-ray as similar large opacities or amalgamations of tiny scars that have merged into large ones.

Differences	
Asbestos	Silica
Mesothelioma	No Mesothelioma
No Tuberculosis	Tuberculosis (and other fungal infections; e.g., histoplasmosis, coccidiomycosis)
Laryngeal and certain GI Cancers	Autoimmune Disease (rheumatoid arthritis, lupus, scleroderma, etc.)
Lower lung predominates	Upper and mid lung predominates
Irregular opacities (s, t, u on ILO form)	Rounded opacities (p, q, r on ILO form)
Pleural plaques which may calcify	No calcified plaques
No coalescence of opacities	Coalescence of round opacities
No large opacities	Large opacities
No hilar adenopathy	Hilar adenopathy
No mediastinal adenopathy	Mediastinal adenopathy
No eggshell calcification	Eggshell calcification
Honeycombing	Compensatory emphysema

Beyond the x-ray appearance of interstitial changes, asbestos exposure can lead to pleural plaques or pleural thickening. Silica does not lead to these pleural changes. Advanced asbestosis may appear as “honeycombing” of the parenchyma. Silicosis can exhibit a distinctive “eggshell calcification” pattern, and lymph node enlargement is common. Dr. Weill explained:

Not only are these appearances different in individual cases, these two broad radiographic patterns point one toward entirely different diagnostic categories. Diseases other than asbestosis fall into the reticular group. Examples include...idiopathic pulmonary fibrosis, radiation pneumonitis...chronic hypersensitivity pneumonitis, chemotherapy-induced lung disease, [and scleroderma]. Diseases other than silicosis fall into the nodular group, including sarcoidosis, berylliosis, coal workers' pneumoconiosis...metastatic cancer, [and histoplasmosis].

With the right experts, opposing counsel will generate opinions that wrestle inconsistent clinical findings into the diagnosis that fits the lawsuit. There is the safe haven of “inter-reader variability” to explain anomalous findings.¹² The biased expert also can attempt to explain inconsistent findings on the basis of individual susceptibility, much as the ATS now accepts mixed restriction and obstruction on PFTs as evidence of asbestosis. If the courts are effective “gatekeepers,” applying the *Daubert* test assiduously, such bogus opinions will be stricken. Too often, courts allow such opinions into evidence “for what it is worth” — small consolation for the defendant in a hostile jurisdiction. In truth, there are clear differences in the manifestation of asbestos- and silica-related diseases. No honest diagnostician should confuse them. From pathologic, radiographic, and clinical perspectives, asbestosis and silicosis are very distinct diseases.

B. Finding the Guideposts in Clinical Records

Because (1) silicosis and asbestosis can be found among workers with similar occupational histories, (2) these work-

ers may have been exposed to a multitude of other pneumoconiosis-producing agents on the job, and (3) these workers share other similarities (including widespread tobacco use), it is logical the clinician charged with making the correct diagnosis would have numerous findings to tease apart. Asbestosis and silicosis are “diagnoses of exclusion,” meaning that all other potential causes of the symptoms and clinical findings need to be eliminated. To evaluate the thoroughness of the clinical work-up of either an asbestosis or a silicosis case — regardless of whether it is performed by one of the “entrepreneurial” screening labs, the plaintiff’s own family doctor, or the defense IME expert, here are the basic elements that should be covered: (*See table A on following page.*)

C. Other Pneumoconioses/Mixed Dust Pneumoconioses

The advent of the plaintiffs’ bar filing “mixed dust” cases makes discussion of the unusual condition, mixed-dust pneumoconiosis (“MDP”), and other pneumoconioses relevant. Many times, after a screening, a worker will be diagnosed with silicosis, asbestosis, or the common catch all, MDP. Inorganic dusts cause several potentially related but different types of lung disease. There are over one hundred causes of diffuse interstitial lung disease. MDPs will occur when lung disease is produced after inhaling two or more dusts through a number of dust exposures. Of course, the first step in determining what, if any, disease may be present requires conducting a differential diagnosis, that is, a determination of which of two or more diseases with similar symptoms is the one from which the person is suffering. A diagnosis of MDP ideally is supported by the identification and quantification of inorganic particulates in lung tissue, and in any event, must include x-rays where mixed-dust fibrotic nodules predominate over all other linear and/or rounded opacities.

In December, 2004, researchers of pulmonary diseases produced a consensus paper in the journal, *Human Pathology*. Authors include numerous internationally prominent

TABLE A

<p>1. Chief Complaints</p> <ul style="list-style-type: none"> Shortness of Breath Dyspnea on Exertion Changes in Daily Routines Onset of Symptoms, and Associated Activity Cough — Dry vs. Productive Cough — Chronic (e.g. lasting > 6 weeks) vs. Acute Onset Wheezing Snoring <p>2. Past Medical History</p> <ul style="list-style-type: none"> Hospitalizations and Surgeries Chronic Illnesses Cancer Allergies Accidents (broken ribs) Social Habits (alcohol and tobacco) Illegal Drug Use <p>3. Review of Systems</p> <p>a. Respiratory</p> <ul style="list-style-type: none"> Asthma Tuberculosis (positive skin test, exposure to others) Pneumonia Fungal Infections (raised pigeons or chickens, mold) AIDS Sleep Apnea <p>b. Cardiovascular</p> <ul style="list-style-type: none"> Myocardial Infarction (heart attack) Bypass Graft / Stent Placement Stroke Emboli Deep Venous Thrombosis Angina Arrhythmias Pace maker / Defibrillator implanted Paroxysmal Nocturnal Dyspnea (sensation of waking up, gasping for air, or bolting to sitting position in bed to restore breathing) Intermittent Claudication (leg pain/difficulty walking) Congestive Heart Failure (ischemic heart disease, pedal edema / swollen ankles) Orthopnea (sleeping on two or more pillows) <p>c. Neurological and Musculoskeletal</p> <ul style="list-style-type: none"> Arthritis (Rheumatoid or Degenerative) Kyphosis / Scoliosis of the Spine Rib Fractures Joint Pain or Deformity Simple Deconditioning (too painful to walk or regularly exercise, “out of shape”) Obesity <p>d. Endocrine</p> <ul style="list-style-type: none"> Diabetes Mellitus Steroid Therapy Hypo- or Hyperthyroidis <p>4. Family History</p> <ul style="list-style-type: none"> Health status of parents/siblings, cause of and age at death Cancer clusters among blood relatives Residences (from childhood to present, toxins, molds, lead paint, asbestos) Secondary or take-home dust exposures Exposure to secondhand smoke Unusual hobbies, activities (such as model railroads with asbestos “mountains”) Dusty hobbies (woodworking) Pesticide exposures (agricultural family) <p>5. Occupational History¹³</p> <ul style="list-style-type: none"> All jobs Direct vs. Indirect Exposures Bystander vs. Mechanic All harmful agents in work place Ventilation Regularity, Frequency, Duration, Intensity (of all harmful exposures) Periodic or Employment Physicals, X-rays, PFTs 	<p>6. Physical Examination</p> <ul style="list-style-type: none"> General appearance (in the pink or “sickly”) Healthy Complexion and Skin Head, Eyes, Ears, Nose Mentally Oriented Normal Gait and Agility Distressed/ /Labored Breathing Number and Quality of Respirations Vital Signs (pulse, blood pressure, temperature) Palpation and Auscultation of the Chest (rales, crackles) Breath Sounds (wheezes, rasps, distant) Heart Sounds (skips, murmurs, gallops, bruits) Abdomen (obesity, enlarged liver/spleen) Extremities (clubbing, cyanosis) Surgical Scars Central nervous system evaluation <p>7. Laboratory Tests</p> <p>a. Chest Radiographs (X-rays, CT Scans, Hi-Resolution CTs)</p> <ul style="list-style-type: none"> Current and Historical Positioning (AP, PA, Lateral, Supine, Prone) Film Quality B-reading Parenchymal Abnormalities Pleural Abnormalities Skeletal or Cardiac Abnormalities Diaphragmatic Abnormalities Bilateral Symmetry or Defect Carcinoma/masses Accumulation of Fluid <p>b. Pulmonary Function Testing¹⁴</p> <ul style="list-style-type: none"> Current and Historical Historical Consistency or Progression Reliable Lab and Equipment Experienced Technician Raw Data Reproducibility (consistent flow curves from which predicted values selected) Accurate Age, Weight, Race and Patient Data Recorded <p>c. Spirometry¹⁵</p> <ul style="list-style-type: none"> FVC FEV¹ FEV¹ / FVC FEF 25% - 75% for small airway function FEF 75% <p>d. Lung Volumes</p> <ul style="list-style-type: none"> Plethysmography (Body Box) Helium or Nitrogen Dilution TLC FRC ERV RV RV/TLC AV <p>e. Diffusion Capacity</p> <ul style="list-style-type: none"> Is volume consistent with other PFTs? Is DLCO corrected for alveolar volume (AV)? <p>8. Invasive Procedures</p> <ul style="list-style-type: none"> Bronchoscopy Biopsy (needle or open) Bronchoalveolar Lavage Thoracentesis Thoracoscopy Closed Pleural Biopsy
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experts frequently appearing on both sides of asbestos lawsuits, including Victor L. Roggli, M.D., Jerrold L. Abraham, M.D., and Allen R. Gibbs, M.D. They summarize the main features of MDP in the Abstract:

We defined mixed-dust pneumoconiosis (MDP) pathologically as pneumoconiosis showing dust macules of mixed-dust fibrotic nodules (MDF), with or without silicotic nodules (SN), in an individual with a history of exposure to mixed dust....According to our definition of MDP, therefore MDF should outnumber SN in the lung to make the pathologic diagnosis of MDP...The clinical diagnosis of MDP requires the exclusion of other well-defined pneumoconioses....Irregular opacities are the major radiographic findings in MDP (ILO 1980), in contrast to silicosis, in which small rounded opacities predominate. Clinical symptoms of MDP are nonspecific. MDP must be distinguished from a variety of nonoccupational interstitial pulmonary disorders.

Honma, K. et al., "Proposed Criteria for Mixed-dust Pneumoconiosis: Definition, Descriptions and Guidelines for Pathologic Diagnosis and Clinical Correlation," *HUMAN PATHOLOGY*, 35 (2004); 1515-23.

Many of the occupational exposure sites and occupations normally associated with silica and asbestos exposures are also considered potential mixed-dust locations, including mines, quarries, steel mills, construction sites, shipyards, gypsum rock mines, zinc mills, foundries, titanium manufacturing facilities, sandblasting operations, and iron working operations. Frequently, a worker will have mixed exposures, and as a result, it is necessary to be thorough in determining whether (and what) other conditions exist in the worker, and whether injury from silica exposure can be eliminated as a cause.

Numerous substances have been alleged to contribute to mixed-dust pneumoconiosis. In addition to silica and asbestos, some of these substances include: talc (found in certain paints, rubber, ceramics, pharmaceuticals, insecticides, cosmetics, and lubricants), kaolin (China clay, found in certain ceramics, paper, paint, cement, soaps, and sometimes used as a binding agent added to silica sand), fuller's earth (binder/filler used in refining oils and filler in cosmetics), and mica (aluminum silicates that are sometimes ground up in paper and paint).

Other potential substances include: bentonite (exposures can occur during oil well drilling; bentonite may also be used as a binding agent added to silica sand), gypsum (plaster-board), vermiculite (insulators), beryllium (metals), cadmium (pigments, paints, ceramics, glass, plastics, and leather), manganese (mining and crushing), and nickel (electroplating, enamels).

Although beyond the scope of this article, it is also important to keep in mind that, depending upon the person's exposure history, certain fungal, animal, chemical, bacterial, and unknown causes that are associated with lung injury and mimic those caused by dust also must be ruled out, in addition to the more traditional diseases associated with exposure to silica, asbestos, and other minerals, elements, or compounds.

III. CONCLUSION

Of course, engaging the best medical experts in the field of diagnosing pneumoconioses is essential, but legal practitioners will not know whom to call without the basic understanding of the clues buried in a plaintiff's medical records. Utilization of clinically experi-

enced medical information specialists to uncover, analyze, and summarize these critical pieces of information should be strongly considered. Until defense counsel recognize the key differences between asbestosis and silicosis, and have the underlying factual data to clearly differentiate the two disease processes, they will be significantly limited in their ability to cross-examine their adversary's hired gun so as to expose a bogus diagnosis. Before defendants are subjected to the risk and expense of mass tort litigation over anything as amorphous and commonplace as "dust exposure," appreciating the differences among illness-producing agents is all-important. Was the culprit asbestos, or was it silica? Perhaps the better question would be: Was it tobacco, or beryllium, or talc, or kaolin, or mica, or portland cement, or nickel, or rust, or cotton, or coal, or ... any of dozens of other minerals, chemicals and organic compounds commonly found in the American workplace throughout the Twentieth Century? Applying the appropriate legal analysis to the critical information culled from the underlying medical documentation will ensure the greatest likelihood of success in the defense of these ever proliferating lawsuits.

"Frequently, a worker will have mixed exposures, and as a result, it is necessary to be thorough in determining whether (and what) other conditions exist in the worker."

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- 1 Greg Gordon, the Star Tribune's Washington Bureau Correspondent,
wrote on February 3, 2005, an article entitled: "Asbestos: Some
lawyers accused of double-dipping" in which he stated: "It's no acci-
dent that claims over silica dust diseases have soared as Congress has
edged toward adopting legislation settling the nation's asbestos injury
suits,' a legal scholar told Congress on Wednesday. Lester Brickman, a
law professor at New York's Yeshiva University, accused lawyers of
engaging in 'entrepreneurial, if not fraudulent,' double-dipping.
Appearing before the Senate Judiciary Committee, he cited data
showing that 60 percent of 8,629 plaintiffs in a mass silicosis case in
Corpus Christi, Texas, had also filed asbestos claims with Johns
Manville Corp.'s asbestos bankruptcy trust. Hundreds of companies
and their insurers want to permanently cap their mounting asbestos
liabilities. But they have voiced concerns that a small number of
 - unscrupulous lawyers could make an end-run around a trust fund by
finding doctors willing to label their clients' illnesses as silicosis or
other dust-borne diseases, so they can stay in the court system."
 - 2 Sean F. Mooney included the NIOSH chart attached as Exhibit 1
in *Silica: A Litigation Sandstorm*, Harris Martin's Columns: Silica
(February, 2005). The chart indicates a dramatic drop in deaths
caused (at least in part) by silica between 1968 (1157) and 1999
(187).
 - 3 A blizzard of news reports has come out of these hearings in Corpus
Christi, Texas. Judge Jack is quoted as commenting during the
February 17, 2005 examination of Ray Harron, M.D., a B-reader in
West Virginia who had his secretary rubber stamp his signature to
over 2500 silicosis diagnoses, that the doctor's testimony was "rais-
ing great red flags of fraud." See e.g., article by Neal Falgout in
February 18, 2005 edition of Caller-Times, "Case Judge: 'Red flags of
fraud' are raised." The hearing was recessed at Dr. Harron's request to
allow him to consult with his personal counsel.
 - 4 Pneumoconiosis encompasses any disease causing scarring of the
parenchyma of the lung from the inhalation of "dust." Virtually
any dust can cause a pneumoconiosis, if the dust particle is small
enough to reach the areas deep within the lung where air exchange
occurs and if the dose is sufficient to defeat the body's several
defense mechanisms.
 - 5 According to the transcript of the congressional hearing on the
FAIR Act of 2005, on February 2, 2005, Paul Epstein, M.D. charac-
terized the "dual occurrences of asbestosis and silicosis are clinical
rarity." Laura Welch, M.D., who frequently is called as a plaintiff's
expert in asbestos trials, testified at the same hearing, "Asbestosis
and silicosis really are different diseases, and they're separable from
each other based on the history of exposure, the chest x-ray and pul-
monary function test." Dr. Welch identified herself as the medical
director for the Center to Protect Workers' Rights, an institute affil-
iated with the building and construction trades of the AFL-CIO.
 - 6 All humans have defenses to combat the effects of the ubiquitous
"impurities" in the air, such as the mucociliary escalator, the moist
linings of the nasal passages and throat, and macrophages — scav-
enger cells that prowl the alveolar sacs in search of foreign objects
like pollen, bacteria, viruses, and dust to engulf and eliminate.
 - 7 A micron or micrometer is one millionth of a meter.
 - 8 Restriction, as the name implies, is the reduction in the total vol-
ume of the lungs of the patient compared to so-called predicted
values. The predicted values are based on "normal" populations of
persons of similar height, weight, age, gender, and race.
 - 9 Based on prominent pulmonologists publishing letters of criticism
or disagreement in the medical literature. See e.g., Martin L.,
Correspondence, "2004 Asbestos Disease Guidelines Ignore Mass
Screening Abuse," Am. J. Resp. & Critical Care Medicine, 171
(2005); 666-67.
 - 10 In the United States, the B-reader certification is overseen by the
National Institute of Occupational Safety & Health ("NIOSH"),
utilizing the standard ILO films.
 - 11 See International Labor Organization, Guidelines for the Use of
ILO International Classification of Radiographs of Pneumoconiosis
(2000).
 - 12 "Inter-reader variability" is where two equally credentialed and
experienced B-readers arrive at different interpretations of the
same films.
 - 13 The goal being to determine whether there was or can be an over-
all exposure assessment calculated in fiber/years or the equivalent.
Was or can there be an assessment of latency?
 - 14 The goal being to verify consistent measurements of restrictive,
obstructive, or mixed ventilatory defects.
 - 15 Note: It is beyond the space limitations to explain all of the varia-
tions on PFTs results to support or refute the written diagnosis in
the medical records.

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The views expressed herein are solely those of the authors and are not made on behalf of any of their clients.

Can Outsourcing The Medical Information Management Function Improve Your Client Service?

By Elizabeth B. Juliano and Dean F. Slejko

Your firm has been retained to handle litigation involving hundreds of cases in which medical issues are pivotal to the plaintiffs' allegations. As lead counsel in this multi-state litigation, you assemble a team from several of your firm's offices and begin work.

As the litigation progresses, you find that managing the medical information presents a number of challenges:

- Boxes of records, in no apparent order or organization, are arriving daily. How should the records be organized so that they can be analyzed efficiently, even when additional records arrive three weeks later?
- Many additional health care providers are identified in the records. Which providers have been contacted, which ones have responded, and how many records are still outstanding?
- You and your client assign case priorities, with many of the highest priority cases having milestone dates near each other. How can progress be tracked across all cases so that milestones are met?
- Your team begins to recognize recurring fact patterns among plaintiffs in the litigation. How can statistics across the entire plaintiff population be gathered to easily examine trends or anomalies?
- Your client has to present a status report on the litigation to management later today and asks for your help. How quickly can meaningful information be gathered that will fulfill your client's request?

Your team meets all of these challenges, but you wonder if retaining a medical information management (MIM) service provider might be a better solution. *If you outsource the MIM function, can the service provider help you better serve your client?*

MIM Service Provider Essentials

A MIM service provider can help you with client service in three significant ways:

- Handle the day-to-day challenges involved in the collection and analysis of medical records;

- Provide tools that help you understand the medical issues of the litigation, prepare each plaintiff's case for discovery and trial, and communicate the medical picture throughout your firm, to your client, and to local counsel; and
- Provide the ability to see the litigation as a whole in order to monitor trends, prioritize cases, and project costs.

Handling Day-to-Day Challenges

Your MIM service provider should have the organization, processes and procedures in place to:

- Efficiently follow up with providers who don't respond to requests for records
- Quickly track down new providers that were not previously identified and who may have critical information regarding the plaintiff
- Deal with provider pre-payment or other special requirements to locate, copy, and mail records
- Monitor which records have been received, those that are still outstanding, and receipt (or not) of "No Records Statements"
- Keep the review process flowing, even though the records arrive in bunches over the course of weeks, and sometimes months
- Update sections of the reports as new records arrive and identify those sections that have been updated so you don't waste time re-reading the report to learn what has changed
- Automatically and continually communicate the record collection and analysis status of each case so that you are informed of progress

These are details that require significant follow-up and maintenance time and effort. When they are handled in a way that is transparent to you and your client, it allows you to focus on client service activities such as *evaluating* the medical record findings, *monitoring* trends, *developing* strategy and *communicating* this information to your client.

Tools That Improve Your Client Service

Not only should a MIM service provider relieve you of these detail-oriented tasks, but they should also give you the means for providing the advocacy that your client expects from outside counsel. Consider some of the other tools and services that a MIM service provider can offer and how they can help improve your client service:

- *A secure web site that gives you continuous access to all case-related information* including scanned records, work products, status reports, and other litigation-related documents you wish to store. You and your partners access the same database of records and reports so that everyone sees the same information. Your client notices the excellent coordination among members of your team and attributes it to your leadership skills.
- *Customized types and styles of medical analysis work products that conform to the needs of the litigation as well as your personal preferences for information display.* Members of your team can quickly familiarize themselves with a case since the reports all have the same format. Your client sees how quickly your team has moved up the learning curve regarding this litigation.
- *Chronological charts of pertinent key words or phrases identified in each plaintiff's records.* Keywording facilitates use of the summary by promoting the ability to reference specific information quickly. You determine the type of information which is critical to the evaluation of the case, and by identifying these keywords, you can review the information at a glance to evaluate the relevance to the case.
- *Medical analysis work products that contain embedded hyperlinks* that allow you to directly access and view imaged records as they are referenced in the report. You spend less time searching for the source documents and more time preparing the defense of cases.
- *On-line split-screen viewing capability* that allows you to view medical analyses on the left side of your computer screen, and the records on the right side, a welcome change from balancing a report on one knee and a pile of records on the other. The combination of hyperlinked records and split-screen

format makes your team more productive, enabling you to keep on top of the growing caseload. Your client sees that your team is meeting all deadlines.

- *The ability to easily download work products and the linked records to your laptop computer.* Your team doesn't have to lug around file folders full of reports and records when conducting depositions in remote locations. Further, your team can work in various locations, i.e., on airplanes, without risking others viewing sensitive documents.
- *Case sorting functionality* to quickly examine the litigation by parameters you select, such as plaintiff name, diagnosis, counsel, assigned priority, deadline date, or venue. Your client comes to realize that you have everything under control, and now calls mainly to discuss litigation strategy.
- *Communication through a single point of contact* that keeps you apprised of all the medical-related activities in the litigation. Your client is confident of your grasp of the medical issues.
- *Established budgets for the services provided,* helping you to manage litigation costs. You know there will be no surprises when the invoice for MIM services arrives. Your client's Legal Finance Department acknowledges that litigation expenses are at or below budgeted levels.

Providing a Global View of the Litigation

Medically driven litigation can involve enormous amounts of information of various types including:

- Pleadings
- Medical records
- Employment, social security, educational, and worksite records
- Deposition transcripts (both historical and current)
- Expert witness reports and CV's
- Product or project files and related documents
- Briefs, motions and court orders
- Attorney work products

Since information developed early in the life of the litigation remains critical to the defense as the tort matures, you need to be able to easily access and analyze all of this historical data across all cases and throughout all phases of the litigation.

In addition to handling the medical information, your MIM service provider should offer the technology to conveniently store, view, and disseminate this critical information. The added capability to create your own custom compilation reports can help you examine statistics, monitor trends, prioritize cases, and project costs. Now, when your client calls requesting information, you can respond quickly with current information in the format your client requests.

Client Service from Behind The Scenes

There are obviously many attributes that go into providing top-notch client service, but the MIM service provider

can play a critical behind-the-scenes role in medically driven litigation. The three critical items to expect from your MIM service provider are the handling of day-to-day challenges in the collection and analysis of medical records, the tools and services that help you understand and communicate the medical issues of the litigation to your client, and the technology to provide a global view of the litigation.

When your client calls with a new assignment, either mass tort litigation or a single case, consider the support a MIM service provider can offer. It may raise your client service, and your client's ultimate satisfaction, to an even higher level.

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Did You Know?

- LMI medical analyses have contributed to Top 10 Defense Wins in 2003 and 2004 as named by *The National Law Journal*
- LMI cost-efficiently handles individual cases as well as mass tort litigation
- One LMI in-house client managing mass tort litigation states "the introduction of LMI into our medical information management program saved us no less than 20% in defense fees."

For more details, please contact ebj@medicineforthedefense.com.