Medical Treatment Guidelines

Occupational Interstitial Lung Disease

Effective May 2, 2022

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The NYS Workers’ Compensation Board would like to thank the members of the New York Workers’ Compensation Board Medical Advisory Committee (MAC). The MAC served as the Board’s advisory body to adapt the American College of Occupational and Environmental Medicine (ACOEM) Practice Guidelines to a New York version of the Medical Treatment Guidelines (MTG). In this capacity, the MAC provided valuable input and made recommendations to help guide the final version of these Guidelines. With full consensus reached on many topics, and a careful review of any dissenting opinions on others, the Board established the final product.

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A. **GENERAL GUIDELINE PRINCIPLES**

The principles summarized in this section are key to the intended application of the New York State Medical Treatment Guidelines (MTG) and are applicable to all Workers' Compensation Medical Treatment Guidelines.

A.1 **Medical Care**

Medical care and treatment required as a result of a work-related injury should be focused on restoring functional ability required to meet the patient’s daily and work activities with a focus on a return to work, while striving to restore the patient’s health to its pre-injury status in so far as is feasible.

A.2 **Rendering Of Medical Services**

Any medical provider rendering services to a workers’ compensation patient must utilize the Treatment Guidelines as provided for with respect to all work-related injuries and/or illnesses.

A.3 **Positive Patient Response**

Positive results are defined primarily as functional gains which can be objectively measured. Objective functional gains include, but are not limited to, positional tolerances, range of motion, strength, endurance, activities of daily living (ADL), cognition, psychological behavior, and efficiency/velocity measures which can be quantified. Subjective reports of pain and function may be considered and given relative weight when the pain has anatomic and physiologic correlation in proportion to the injury.

A.4 **Re-Evaluate Treatment**

If a given treatment or modality is not producing positive results within a well-defined timeframe, the provider should either modify or discontinue the treatment regime. The provider should evaluate the efficacy of the treatment or modality 2 to 3 weeks after the initial visit and 3 to 4 weeks thereafter. These timeframes may be slightly longer in the context of conditions that are inherently mental health issues, and shorter for other non-musculoskeletal medical conditions (e.g. pulmonary, dermatologic etc.). Recognition that treatment failure is at times attributable to an incorrect diagnosis a failure to respond should prompt the clinician to reconsider the diagnosis in the event of an unexpected poor response to an otherwise rational intervention.

A.5 **Education**

Education of the patient and family, as well as the employer, insurer, policy makers and the community should be a primary emphasis in the treatment of work-related injury or illness. Practitioners should develop
and implement effective educational strategies and skills. An education-based paradigm should always start with communication providing reassuring information to the patient. No treatment plan is complete without addressing issues of individual and/or group patient education as a means of facilitating self-management of symptoms and prevention of future injury.

Time Frames

A.6 Acuity
Acute, Subacute and Chronic are generally defined as timeframes for disease stages:
- Acute – Less than one month
- Subacute - One to three month, and
- Chronic - greater than three months.

A.7 Initial Evaluation
Initial evaluation refers to the acute timeframe following an injury and is not used to define when a given physician first evaluates an injured worker (initial encounter) in an office or clinical setting.

A.8 Diagnostic Time Frames
Diagnostic time frames for conducting diagnostic testing commence on the date of injury. Clinical judgment may substantiate the need to accelerate or decelerate the time frames discussed in this document.

A.9 Treatment Time Frames
Treatment time frames for specific interventions commence once treatments have been initiated, not on the date of injury. It is recognized that treatment duration may be impacted by disease process and severity, patient compliance, as well as availability of services. Clinical judgment may substantiate the need to accelerate or decelerate the time frames discussed in this document.

A.10 Delayed Recovery
For those patients who fail to make expected progress 6-12 weeks after an injury and whose subjective symptoms do not correlate with objective signs and tests, reexamination in order to confirm the accuracy of the diagnosis and re-evaluation of the treatment program should be performed. When addressing a clinical issue that is not inherently a mental health issue, assessment for potential barriers to recovery (yellow flags/psychological issues) should be ongoing throughout the care of the patient. At 6-12 weeks, alternate treatment programs, including formal psychological or psychosocial evaluation should be considered. Clinicians
must be vigilant for any pre-existing mental health issues or subsequent, consequential mental health issues that may be impacting recovery. For issues that are clearly and inherently mental health issues from the outset (i.e. when it is evident that there is an underlying, work-related, mental health disorder as part of the claim at issue), referral to a mental health provider can and should occur much sooner. Referrals to mental health providers for the evaluation and management of delayed recovery do not indicate or require the establishment of a psychiatric or psychological condition. The evaluation and management of delayed recovery does not require the establishment of a psychiatric or psychological claim.

**Treatment Approaches**

**A.11 Active Interventions**
Active interventions emphasizing patient responsibility, such as therapeutic exercise and/or functional treatment, are generally emphasized over passive modalities, especially as treatment progresses. Generally, passive and palliative interventions are viewed as a means to facilitate progress in an active rehabilitation program with concomitant attainment of objective functional gains.

**A.12 Active Therapeutic Exercise Program**
Active therapeutic exercise program goals should incorporate patient strength, endurance, flexibility, range of motion, sensory integration, coordination, cognition and behavior (when at issue) and education as clinically indicated. This includes functional application in vocational or community settings.

**A.13 Diagnostic Imaging And Testing Procedures**
Clinical information obtained by history taking and physical examination should be the basis for selection of imaging procedures and interpretation of results. All diagnostic procedures have characteristic specificities and sensitivities for various diagnoses. Usually, selection of one procedure over others depends upon various factors, which may include: relative diagnostic value; risk/benefit profile of the procedure; availability of technology; a patient’s tolerance; and/or the treating practitioner’s familiarity with the procedure.

When a diagnostic procedure, in conjunction with clinical information, provides sufficient information to establish an accurate diagnosis, a second diagnostic procedure is not required. However, a subsequent diagnostic procedure including a repeat of the original (same) procedure can be performed, when the specialty physician (e.g. physiatrist, sports medicine physician or other appropriate specialist) radiologist or surgeon documents that the initial study was of inadequate quality to make a diagnosis. Therefore, in such circumstances, a repeat or complementary diagnostic procedure is permissible under the MTG.
It is recognized that repeat imaging studies and other tests may be warranted by the clinical course and/or to follow the progress of treatment in some cases. It may be of value to repeat diagnostic procedures (e.g., imaging studies) during the course of care to reassess or stage the pathology when there is progression of symptoms or findings, prior to surgical interventions and/or therapeutic injections when clinically indicated, and post-operatively to follow the healing process. Regarding serial imaging, (including x-rays, but particularly CT scans), it must be recognized that repeat procedures result in an increase in cumulative radiation dose and associated risks.

A given diagnostic imaging procedure may provide the same or distinctive information as obtained by other procedures. Therefore, prudent choice of procedures(s) for a single diagnostic procedure, a complementary procedure in combination with other procedures(s), or a proper sequential order in multiple procedures will ensure maximum diagnostic accuracy, minimize the likelihood of adverse effect on patients, and promote efficiency by avoiding duplication or redundancy.

A.14 Surgical Interventions
Consideration of surgery should be within the context of expected functional outcome. The concept of "cure" with respect to surgical treatment by itself is generally a misnomer. All operative interventions must be based upon positive correlation of clinical findings, clinical course and imaging and other diagnostic tests. A comprehensive assimilation of these factors must lead to a specific diagnosis with positive identification of pathologic condition(s). For surgery to be performed to treat pain, there must be clear correlation between the pain symptoms and objective evidence of its cause. In all cases, shared decision making with the patient is advised. The patient should be given the opportunity to understand the pros and cons of surgery, potential for rehabilitation as an alternative where applicable, evidence-based outcomes, and specific surgical experience.

A.15 Pre-Authorization
All diagnostic imaging, testing procedures, non-surgical and surgical therapeutic procedures, and other therapeutics within the criteria of the Medical Treatment Guidelines and based on a correct application of the Medical Treatment Guidelines are considered authorized, with the exception of the procedures listed in section 324.3(1)(a) of Title 12 NYCRR. These are not included on the list of pre-authorized procedures. Providers who want to perform one of these procedures must request pre-authorization from the carrier before performing the procedure.
Second or subsequent procedures (the repeat performance of a surgical procedure due to failure of, or incomplete success from the same surgical procedure performed earlier, if the Medical Treatment Guidelines do not specifically address multiple procedures) also require pre-authorization.

A.16 Psychological/Psychiatric Evaluations

In select patients, mental health evaluations are essential to make, secure or confirm a diagnosis. Of course, the extent and duration of evaluations and/or interventions by mental health professionals may vary, particularly based on whether: the underlying clinical issue in the claim is inherently a mental health issue; or there is a mental health issue that is secondary or consequential to the medical injury or illness that is at issue in the claim in question; or there is a pre-existing, unrelated mental health issue that has been made worse by, or is impeding the recovery from (or both) the medical injury or illness that is at issue in the claim in question.

Tests of psychological function or psychometric testing, when indicated, can be a valuable component of the psychological evaluation in identifying associated psychological, personality and psychosocial issues. Although these instruments may suggest a diagnosis, neither screening nor psychometric tests are capable of making a diagnosis. The diagnosis should only be made after careful analysis of all available data, including from a thorough history and clinical interview.

A professional fluent in the primary language of the patient is strongly preferred. When such a provider is not available, services of a professional language interpreter must be provided.

Frequency: When assessing for a pre-existing, unrelated mental health issue that has been made worse by, or is impeding the recovery from (or both) a work-related, medical injury or illness, then a one-time visit for initial psychiatric/psychological encounter should be sufficient, as care would normally be continued by the prior treating provider. If psychometric testing is indicated by findings in the initial encounter, time for such testing should not exceed an additional three hours of professional time. For conditions in which a mental health issue is a central part of the initial claim, or in which there is a mental health issue that is secondary or consequential to the work-related, medical injury or illness, that is part of the claim in question, then more extensive diagnostic and therapeutic interventions may be clinically indicated, and are discussed in detail in the Medical Treatment Guidelines for such mental health conditions.
A.17 Personality/Psychological/Psychosocial Intervention

Following psychosocial evaluation, when intervention is recommended, such intervention should be implemented as soon as possible. This can be used alone or in conjunction with other treatment modalities. For all psychological/psychiatric interventions, there must be an assessment and treatment plan with measurable behavioral goals, time frames and specific interventions planned.

- Time to produce effect: two to eight weeks.
- Optimum duration: six weeks to three months.
- Maximum duration: three to six months.
- Counseling is not intended to delay but rather to enhance functional recovery.

For PTSD Psychological Intervention:

- Optimum duration three to six months.
- Maximum duration: nine to twelve months.

For select patients, longer supervision and treatment may be required, and if further treatment is indicated, documentation of the nature of the psychological factors, as well as projecting a realistic functional prognosis, should be provided by the authorized treating practitioner every four weeks during the first six months of treatment. For treatment expected to last six to twelve months, such documentation should be provided every four to eight weeks. For long-term treatment beyond twelve months, such documentation should be provided every eight to twelve weeks. All parties should strive for ongoing and continuous communications, in order to facilitate seamless, continuous and uninterrupted treatment.

A.18 Functional Capacity Evaluation (FCE)

Functional capacity evaluation is a comprehensive or more restricted evaluation of the various aspects of function as they relate to the patient’s ability to return to work. Areas such as endurance, lifting (dynamic and static), postural tolerance, specific range-of-motion, coordination and strength, worker habits, employability, as well as psychosocial, cognitive, and sensory perceptual aspects of competitive employment may be evaluated. Components of this evaluation may include: (a) musculoskeletal screen; (b) cardiovascular profile/aerobic capacity; (c) coordination; (d) lift/carrying analysis; (e) job-specific activity tolerance; (f) maximum voluntary effort; (g) pain assessment/psychological screening; (h) non-material and material handling activities; (i) cognitive and behavioral; (j) visual; and (k) sensory perceptual factors.

In most cases, the question of whether a patient can return to work can be answered without an FCE.
An FCE may be considered at time of MMI, following reasonable prior attempts to return to full duty throughout course of treatment, when the treating physician is unable to make a clear determination on work status on case closure. An FCE is not indicated early during a treatment regime for any reason including one to support a therapeutic plan.

When an FCE is being used to determine return to a specific job site, the treating physician is responsible for understanding and considering the job duties. FCEs cannot be used in isolation to determine work restrictions. The authorized treating physician must interpret the FCE in light of the individual patient's presentation and medical and personal perceptions. FCEs should not be used as the sole criteria to diagnose malingering.

A.19 Return To Work
For purposes of these guidelines, return to work is defined as any work or duty that the patient is able to perform safely. It may not be the patient’s regular work. Ascertaining a return to work status is part of medical care, and should be included in the treatment and rehabilitation plan. It is normally addressed at every outpatient visit. A description of the patient’s status and task limitations is part of any treatment plan and should provide the basis for restriction of work activities when warranted. Early return to work should be a prime goal in treating occupational injuries. The emphasis within these guidelines is to move patients along a continuum of care and return to work, since the prognosis of returning an injured worker to work drops progressively the longer the worker has been out of work.

A.20 Job Site Evaluation
The treating physician may communicate with the employer or employer’s designee, either in person, by video conference, or by telephone, to obtain information regarding the individual or specific demands of the patient’s pre-injury job. This may include a description of the exertional demands of the job, the need for repetitive activities, load lifting, static or awkward postures, environmental exposures, psychological stressors and other factors that would pose a barrier to re-entry, risk of re-injury or disrupt convalescence. When returning to work at the patient’s previous job tasks or setting is not feasible, given the clinically determined restrictions on the patient’s activities, inquiry should be made about modified duty work settings that align with, the patient’s condition in view of proposed work activities/demands in modified duty jobs. It should be noted, that under certain circumstances, more than one job site evaluation may be indicated.

Ideally, the physician would gain the most information from an on-site inspection of the job settings and activities; but it is recognized that this may not be feasible in most cases. If job videos/CDs/DVDs are available
from the employer, these can contribute valuable information, as can video conferences, conducted from the worksite and ideally workstation or work area.

Frequency: one or two contacts
- 1st contact: Patient is in a functional state where the patient can perform some work.
- 2nd contact: Patient has advanced to state where the patient is capable of enhanced functional demands in a work environment.

The physician shall document the conversation.

Other

A.21 Guideline Recommendations And Medical Evidence
The Workers’ Compensation Board and its Medical Advisory Committee have not independently evaluated or vetted the scientific medical literature used in support of the guidelines, but have relied on the methodology used by the developers of various guidelines utilized and referenced in these Guidelines.

A.22 Experimental/Investigational Treatment
Medical treatment that is experimental/investigational and not approved for any purpose, application or indication by the FDA is not permitted under these Guidelines.

A.23 Injured Workers As Patients
In these Guidelines, injured workers are referred to as patients recognizing that in certain circumstances there is no doctor-patient relationship.

A.24 Scope Of Practice
These Guidelines do not address scope of practice or change the scope of practice.
Occupational Interstitial Lung Disease
Effective date will coincide with the launch of OnBoard: Limited Release

B. Occupational Interstitial Lung Disease Guideline

B.1 Overview
Occupational lung disease is often classified into several different categories, of which Interstitial Lung Diseases (ILD) is one of the main categories and work-related asthma is another (Work related asthma is addressed in the NY Occupational Asthma/Work Related Asthma Medical Treatment Guideline).

This guideline is intended as an evidence-based approach to the diagnosis and treatment of Occupational Interstitial Lung Diseases (ILD). The guideline covers inorganic dust-related diseases (e.g., silicosis, asbestosis, and coal workers’ pneumoconiosis (CWP)), and the immunologically mediated diseases such as chronic beryllium disease (CBD) or hypersensitivity pneumonitis (HP). Occupational exposure history, presentation, and diagnostic and screening test results form the foundation for diagnosis and treatment plans.

ILDs are a heterogeneous group of more than 100 diseases that inflame and/or scar the lung parenchyma and which are classified together because of similar clinical, roentgenographic, physiologic, and/or pathologic features. ILD describes disorders affecting the lung interstitium. Acute injury to the interstitium is manifested mostly by edema and inflammation, while chronic injury is characterized by fibrosis, the end stage of chronic inflammation. ILD sometimes referred to as “pulmonary fibrosis” or “interstitial fibrosis” is a group of chronic, generally irreversible conditions manifested by a vigorous immune and/or inflammatory response and exuberant fibroblast activity that results in excessive collagen deposition.

B.2 Introduction
Occupationally related ILD fall into four often clinically overlapping categories:
- Pneumoconiosis
- Hypersensitivity Pneumonitis (HP)
- Other Granulomatous Diseases
- Toxic Inhalation Injury

ILD associated with pneumoconioses and autoimmune processes tends to progress through stages, ultimately reaching a similar “end stage” condition. This condition is characterized by:
- restrictive disease
- pulmonary hypertension
- cor pulmonale
- congestive heart failure
- lung infections due to loss of host defense mechanisms

ILD, as it advances, is often associated with a chronic dry cough, which may require suppression particularly when it interferes with sleep.
Table 1: Most Common ILD Conditions, Etiologic Agents, Latency

ILD is a large category of disease; the list of conditions below is not all inclusive of all work-related ILD.

All listed exposures may have increased risk of occupational ILDs where there is sufficient frequency, intensity and duration of exposures.

<table>
<thead>
<tr>
<th>Category</th>
<th>Condition/Examples</th>
<th>Occupational Exposure (Etiologic Agent)</th>
<th>Latency (Time of exposure to onset of symptoms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumoconiosis (Three most common)</td>
<td>Silicosis</td>
<td>Crystalline silica</td>
<td>Years to decades</td>
</tr>
<tr>
<td></td>
<td>Asbestosis</td>
<td>Asbestos Minerals</td>
<td>Decades</td>
</tr>
<tr>
<td></td>
<td>Coal Workers</td>
<td>Coal Mine Dust, Graphite</td>
<td>Decades</td>
</tr>
<tr>
<td>Hypersensitivity Pneumonitis (Organic Respirable Dusts/Low Molecular Weight) Sensitizing Chemicals</td>
<td>Farmers Lung Bird Fanciers Lung</td>
<td>Animal proteins, plant proteins, bacteria, fungi, and paints, foam, PVC fumes, diisocyanates.</td>
<td>Defined as acute, subacute and chronic. As early as hours in acute.</td>
</tr>
<tr>
<td>Other Granulomatous Diseases</td>
<td>Berylliosis Hard Metal Disease (Cobalt, Tin)</td>
<td>Beryllium, Cobalt Tin. Hard Metal</td>
<td>Years to decades</td>
</tr>
<tr>
<td>Toxic Inhalation Injury</td>
<td>Irritant inhalation injury(diffuse alveolar related to nitrogen oxide) ex. Nitrogen Dioxide, Ozone, Phosgene or ionizing radiation (Gases)</td>
<td>ex. Nitrogen Dioxide, Ozone, Phosgene or ionizing radiation (Gases)</td>
<td>Hours</td>
</tr>
</tbody>
</table>
Table 2: Industrial Exposure

<table>
<thead>
<tr>
<th>Category</th>
<th>Industry</th>
<th>Occupational Activities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumoconiosis (Three most common)</td>
<td>Mining, oil and gas, construction, foundry, pottery, manufacturing</td>
<td>Drilling, mining, excavating, abrasive blasting, grinding, cutting</td>
</tr>
<tr>
<td>Silicosis</td>
<td>Sandblasting, foundry workers, tunnel diggers, ceramic workers</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pneumoconiosis: Asbestosis</td>
<td>Power plant, foundry, demolition, ship building, brake and clutch linings, asbestos cement, asbestos textile, fireproofing, insulation</td>
<td>Removal of, disturbing old asbestos-containing construction materials (e.g., insulation), insulation application, brake and clutch work,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pneumoconiosis: Coal Workers</td>
<td>Mining, electricity generation and storage, metals</td>
<td>Coal mining/handling, battery manufacture, pencil making</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypersensitivity Pneumonitis</td>
<td>Wood and food products, animal rearing, farming, painting, chemicals manufacturing</td>
<td>Cleaning, water sprays, shredding,</td>
</tr>
<tr>
<td></td>
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</tr>
<tr>
<td>Other Granulomatous Diseases</td>
<td>Nuclear, aircraft, tools, electronics</td>
<td>Machining, grinding, smelting, metal product manufacturing</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toxic Inhalation Injury</td>
<td>Chemical production, manufacturing, Transportation</td>
<td>Generally acute exposure in the course of accidents or other disasters</td>
</tr>
</tbody>
</table>

**B.3 Key Concepts**

**B.3.a Latency**

Latency can be defined as the time interval between initial exposure and onset of symptoms/clinical diagnosis. The concept of latency is important in occupational ILD as most of the occupational ILDs have a long latency time.

**B.3.b Relationship between Latency and Exposure (See Tables 1 and 2)**

**B.3.c Comorbidities**

As per the International Agency for Research on Cancer (IARC), asbestos exposure is associated with an increased risk for lung cancer (with far greater risk, or interaction, with cigarette smoking), mesothelioma (involving pleural or peritoneal serosal membranes), laryngeal and ovarian cancer. Other studies show that asbestos may be associated with increases in cancers in other sites such as pharyngeal, stomach, colon, and kidney cancers. Asbestos exposure has also been associated with risk for airway disease.
• CWP is associated with an elevated risk of autoimmune disorders, principally rheumatoid arthritis (aka, “Caplan’s syndrome”). Thus, workers with CWP may have associated autoimmune disorders and develop systemic clinical manifestations. CWP has also been associated with risk for airway disease.

• Silicosis also increases risk for lung cancer, pulmonary tuberculosis, autoimmune disease, renal disease, and airways diseases. There is also an interaction of increased lung cancer and cigarette smoking, although not as strong as that one for asbestos exposure.

B.4 Conditions

B.4.a Silicosis

Exposure to sufficient respirable silica leads to silicosis, an irreversible disease which is associated with a variety of systemic and pulmonary conditions. Patients with silicosis or silica exposure have an increased risk for lung cancer. The IARC reclassified silica as a Group I substance (“carcinogenic to humans”) in October 1996.

Silicosis is still the most common occupational disease worldwide and at least 1.7 million U.S. workers are exposed to respirable crystalline silica.

B.4.a.i Etiologic Agent
Silicosis results from exposure to crystalline silicon dioxide. Exposure to silica in other forms such as glass and other amorphous forms of silica has not been associated with silicosis. However, crystalline silica is present in sand. Silica exposure occurs in a variety of industries and occupations, including construction, sandblasting, and mining.

B.4.a.ii Condition Considerations
Exposure to silica can result in one of three different disease patterns: chronic silicosis, subacute/accelerated silicosis and acute silicosis. The most common form is chronic silicosis, which is usually seen after more than ten years of exposure. Subacute silicosis results from shorter, heavier exposures, usually after two to five years of latency. Acute silicosis is often seen following intense exposure to fine silica-containing dust over a several month period.

Chronic silicosis may progress to massive, accreted fibrotic zones in the lung (“conglomerative silicosis”) that result in:

• respiratory failure,
• pulmonary hypertension,
• cor pulmonale with right heart failure.

Patients with silicosis also have increased risk for:
• Chronic bronchitis, defined by chronic sputum production, with or without obstructive impairment in pulmonary function tests,
• Exposure to silica at levels below those associated with simple silicosis has been associated with chronic airflow limitation and/or mucus hypersecretion and/or pathologic emphysema,
• lung cancer,
• pulmonary tuberculosis,
• autoimmune disease,
• renal disease.

B.4.a.iii Latency
Silicosis typically becomes clinically apparent over a period of years, exceptions are rare but include accelerated silicosis.

B.4.a.iv Diagnosis
The diagnosis of silicosis is typically made clinically, based on occupational history of sufficient exposure with appropriate latency, objective radiographic evidence (chest radiograph and/or high resolution CT), assessment of pulmonary function and consideration of alternative differential diagnoses.

B.4.b Asbestosis

B.4.b.i Condition Considerations
Exposure to asbestos can result in one of several different disease patterns. Asbestosis refers to the diffuse type of pulmonary fibrosis that results from inhaling asbestos fibers. Pleural thickening, in the form of discreet pleural plaques (calcified or uncalcified) is the most common manifestation of asbestos exposure. Diffuse pleural thickening, rounded atelectasis and non-malignant asbestos-related pleural effusion are other manifestations of pleural disease caused by asbestos exposure.

B.4.b.ii Comorbidities
Common with asbestos-related disease. Asbestos exposure is associated with an increased risk for:
• Lung cancer (with far greater risk, or interaction, with cigarette smoking).
• Mesothelioma (involving pleural or peritoneal serosal membranes).
• Laryngeal and ovarian cancer.

Individuals with asbestosis experience variable rates of disease progression, ranging from mild to severe respiratory impairment. Asbestosis symptoms and radiographic findings are worsened by cigarette smoking history and other exposures such as diesel fuel fumes.
B.4.b.iii Latency
The symptoms of asbestosis can take decades after exposure to show up.

B.4.b.iv Diagnosis
The diagnosis of asbestosis and other asbestos-related diseases is typically made clinically, based on occupational history of sufficient exposure with appropriate latency, objective radiographic evidence (chest radiograph and/or HRCT), assessment of pulmonary function, and consideration of alternative differential diagnoses.

B.4.c Coal Workers’ Pneumoconiosis (CWP)
Coal dust is a mixture of carbon and complex organic materials and minerals, including variable amounts of silica and silicates.

B.4.c.i Condition Considerations
- CWP is a distinct disease, distinguishable pathologically from silicosis, although the two may occur together particularly in miners who drilled or cut through rock.
- CWP differs histologically from silicosis in the morphology of the lesion.
- Coal workers’ pneumoconiosis (CWP) is often associated with bronchitis and some degree of airways obstruction.
- CWP may progress to large intrathoracic fibrotic masses, usually visible on chest x-rays in the upper and mid lung fields (“progressive massive fibrosis”), which are associated with severe respiratory impairment.

B.4.c.ii Comorbidities CWP is associated with an elevated risk of:
- Autoimmune disorders, principally rheumatoid arthritis (aka, “Caplan’s syndrome”).
- Workers with CWP may have associated autoimmune disorders and develop systemic clinical manifestations.

B.4.c.iii Latency
- CWP Pneumoconiosis typically becomes clinically apparent over a period of decades.
- Exceptions are rare but include CWP associated with high exposure levels.

B.4.c.iv Diagnosis
The diagnosis of CWP is typically made clinically, based on occupational history of sufficient exposure with appropriate latency, objective radiographic evidence (chest radiograph and/or HRCT), assessment of pulmonary function, and consideration of alternative differential diagnoses.
B.4.d Hypersensitivity Pneumonitis (HP)

Hypersensitivity Pneumonitis (HP), also known as extrinsic allergic alveolitis, can be caused by inhalation of organic dust with antigenic properties or exposure to low-molecular weight sensitizing chemicals.

B.4.d.i Condition Considerations

HP is a large family of disorders of immune response often associated with granulomatous pathological changes. HPs tend to be highly specific to occupation or environmental settings.

Inhaled causative agents include:

- Animal proteins
- Plant proteins
- Bacteria
- Fungi
- Diisocyanates.
- Paints
- Trimellitic anhydride
- Epoxy resins
- “Bordeaux mixture” (a pesticide made from copper sulfate used in vineyards)

These dusts arise from:

- Renovation of buildings (especially demolition or exposing damp interior walls), exposure to contaminated water or persistently wet spaces (humidifiers, hot tubs, saunas, and unventilated showers)
- Handling birds
- Occasionally from sensitization to other animals (such as farmer’s lung)
- Insects (such as miller’s lung, the antigen to which is a wheat weevil protein)
- Amoebae (humidifier lung)
- Pesticide powder (pyrethrum HP)
- Spores of a thermophilic actinomycete bacteria resulting in furrier’s lung
- Animal-derived dusts
- Grain dusts
- Mold spores

HP often begins with wheezing and airways obstruction. Untreated and unmanaged, it may progress to respiratory insufficiency and profound impairment. Pigeon breeders’ lung famously is associated with clubbing, unlike most hypersensitivity pneumonitides.
B.4.d.ii Latency

- In HP, sensitization may occur in the first few weeks after beginning exposure, in others it may be delayed for months or years.
- The acute, predominant airways symptoms of HP develop in a sensitized individual over days to weeks and may progress over weeks to interstitial inflammation and ultimately to fibrosis.
- Rarely also hyperacute or sudden in onset, similar to some eosinophilic pneumonias or some drug-induced pneumonitis.

B.4.d.iii Differential Diagnosis

HP should be included in a differential diagnosis of an acute influenza-like or febrile disorder in a patient with a history of exposure to inhaled antigens. However, it may also suggest rheumatological or autoimmune lung disease and infection (mycoplasma, Legionella spp., or diffuse mycosis) as a cause of interstitial disease, the latter especially in a host with a compromised immune system. A history of exposure to birds should also raise the possibility of other diseases including psittacosis.

While there are no well-established risk factors for development of HP, personal and familial susceptibility may play a role.

B.4.e Other Granulomatous Diseases

ILD caused by chronic immune and foreign-body responses to antigens in the lung, which may be metal dusts and, therefore, also considered pneumoconioses.

B.4.e.i Prominent examples include:

- Beryllium (Beryllium Disease)
- Cobalt in cemented tungsten carbide (Hard Metal Disease)
- Work-related Sarcoidosis

B.4.e.ii Condition Considerations

The clinical manifestations of hard metal disease are overall similar to other pneumoconioses. The pathology findings of hard metal disease are that of a giant cell interstitial pneumonia. The interstitial fibrosis is accompanied by activated macrophages that fill alveoli and is part of a dysfunctional foreign body reaction.

Chronic beryllium disease is a systemic granulomatous inflammatory disorder that is very similar to sarcoidosis. The tissue response is mediated by immune mechanisms and may not localize to an area of dust accumulation. This may manifest in systemic, body-wise disease manifestations, although less frequently than in sarcoidosis.
B.4.e.iii Latency
These disorders are uncommon, problems develop at different exposure levels in different people. It can be decades before these disorders become clinically apparent and the clinical presentations are variable.

B.4.e.iv Condition Considerations
- This condition usually results from irritant inhalation injury (e.g., diffuse alveolar injury related to nitrogen oxides).
- Diffuse interstitial fibrosis should be distinguished from more common idiopathic interstitial fibrosis either of the “usual interstitial pneumonia” or the “nonspecific interstitial pneumonia” types.
- Extensive fibrosis, which may occur following recovery from diffuse alveolar damage by toxic inhalation, is refractory to direct management.
- Advanced forms of all of the occupational ILDs may have a similar clinical presentation to diffuse interstitial fibrosis.

B.4.f Toxic Inhalation Injury
ILD due to toxic inhalation injury is generally the result of severe lung injury after an acute exposure to high concentration of noxious gases, fumes or mists.

B.5 History Taking and Physical Examination
Occupational exposure history, presentation, and diagnostic screening test results form the foundation for diagnosis and treatment plans.

B.5.a History of Present Illness
The History of Present Illness (HPI) should document:
- Occupational and non-occupational pulmonary exposures.
- Occupation: current/past and types of work activities (such as: construction, demolition, mining, manufacturing, drilling). See table below for examples.
- Time spent at each job, including jobs held years to decades in the past.

<table>
<thead>
<tr>
<th>Industry</th>
<th>Mining, oil and gas, construction, foundry, pottery, manufacturing</th>
<th>Power plant, foundry, demolition</th>
<th>Mining, electricity generation and storage, metals</th>
<th>Wood and food products, animal rearing, farming</th>
<th>Nuclear, aircraft, tools, electronics</th>
</tr>
</thead>
</table>

Exposures to:

- Dusts: organic dusts (fungi, bacteria, plant and animal proteins) and inorganic mineral dusts (silica, asbestosis, coal).
- Metals (Beryllium (old light bulbs, aerospace), tin, cobalt).
- Toxic and inflammatory fumes, gas, vapors, aerosols.
- History of exposure should include non-occupational exposures to these agents with a description of exposure, duration of exposure, and intensity of exposure.
- Intensity of exposure: ideally with environmental measurements (industrial hygiene data) or at least a qualitative description of intensity of exposure, e.g., daily, weekly, monthly, yearly, etc.
- Include questions detailing the individual’s responsibilities and exposure (e.g. did you work in the office, etc.).

Symptoms:

- Symptoms and when symptoms began
- Including complaints of:
  - Throat tightness
  - Shortness of breath
  - Difficulty with inspiration or expiration
  - Harsh sounds
  - Cough
  - Sputum production

Duration, onset and frequency of symptoms.

- Symptom development including:
  - Aggravation and alleviation of symptoms in relationship to work environment
  - Changes in work environment
  - Changes in symptoms in relation to days worked and not worked
  - Progression of symptoms
- Pulmonary imaging and testing.
- Previous treatments.
- Relationship to work: This includes a statement of the probability that the illness, or injury is work-related.
- Ability to perform job duties and activities of daily living.

B.5.b Past History
• Past medical history including but not limited to prior pulmonary exposures and treatments (include prone to bronchitis, pneumonia).
• Review of systems includes, but is not limited to, symptoms of rheumatologic, neurologic, endocrine, neoplastic, and other systemic diseases.
• Detailed smoking history (including marijuana, vaping, etc.).
• Detailed medication history including use of Amiodarone, chemotherapeutic agents, and nitrofurantoin.
• Vocational and recreational pursuits.
• Prior imaging studies.
• Past surgical history.
• Allergy history.

B.5.c Physical Examination

An occupational pulmonary physical examination should include the following elements:
• Vital signs, including measured respiratory rate, O2 saturation.
• Overall functional abilities, including ease of movement, walking and changing positions, dressing and undressing while assessing signs and symptoms of dyspnea.
• Assessment of respiratory status (e.g., rate, depth, use of accessory muscles, nasal flaring).
• Inspection for stigmata of pulmonary disease as well as potential etiologies including:
  o Mucous membrane abnormalities
  o Nasal polyps/swelling/discharge
  o Clubbing (asbestosis, idiopathic pulmonary fibrosis, some hypersensitivity pneumonitides)
  o Anterior-posterior diameter
  o Scoliosis
  o Kyphosis
• Palpation for:
  o Chest wall abnormalities
  o Adenopathy and neck masses
• Percussion for resonance to identify:
  o Aeration
  o Diaphragm level
  o Suggestion for fluid interface or consolidation
• Auscultation for:
  o Inspiration to expiration ratio
  o Adventitious breath sounds (crackles, wheeze (often a secondary manifestation of HP and a primary manifestation of eosinophilic pneumonia) rales, rhonchi)
Pleural rubs, as well as timing, location and persistence of lung findings

- Cardiac examination with attention to findings of cor pulmonale and heart failure.
- Dermal examination for signs of disease, i.e., erythema nodosum (sarcoidosis).

**B.5.d Diagnostic Approach**

The diagnoses of occupational ILD typically is made clinically, based on occupational history of sufficient exposure with appropriate latency, objective radiographic evidence (chest radiograph and/or HRCT), assessment of pulmonary function (including consistent changes in ventilatory capacity, static lung volumes or gas-exchange), and consideration of alternative differential diagnoses.

In a worker with a typical clinical picture (including exposure history, latency, and radiographic presentation), lung biopsy is rarely needed to provide a diagnosis of occupational ILD. Pathologic examination of lung tissue may at times be required in settings where clinical or radiographic features are inconclusive or atypical.

**Recommended: Follow-Up Diagnostic Tests**

Periodic medical follow up, including pulmonary function tests and imaging studies in the medical evaluation of pulmonary occupational disease.

**B.6 Diagnostic Testing**

**B.6.a Spirometry**

Spirometry is a useful initial test of lung function. Spirometry provides physiologic evidence for occupational ILD and differentiates between obstructive and restrictive lung patterns of lung function. Spirometry should be performed on all patients as a key component in the diagnosis and monitoring of occupational interstitial lung disease. However, ILD is not defined by spirometry. Abnormal spirometry results should lead to further testing including confirmation by lung volume testing according to ATS accepted recommendations or referral to a specialist.

Ideally, the modern diagnostic evaluation of pulmonary occupational disease should include measurements of lung volumes and diffusing capacity. As per clinical necessity, further analysis of gas exchange physiology, cardiopulmonary exercise testing and/or six-minute walk test should be used to supplement the diagnostic and therapeutic evaluations of occupational lung disease.
Technique – Diagnostic spirometry testing:

- Should be performed using recommended equipment and procedures by an appropriately trained technician.
- Should be performed in accordance with the most recent recommendations or requirements of American Thoracic Society, as well as Occupational Safety and Health Administration (OSHA), NIOSH, and Mine Safety and Health Administration (MSHA).
- When diagnostic spirometry is abnormal, testing should first be repeated on another occasion, if possible, to ensure that a worker was maximally inhaling, blasting out hard, and exhaling fully during the test.
- If results remain abnormal, short term reversibility of the spirometry results should be assessed, most often by repeating the spirometry testing after the individual has undergone a standardized short-acting bronchodilator inhalation protocol.
- Once a satisfactory test has been recorded for the worker, diagnostic interpretation may compare his/her largest results with normal ranges derived from appropriate similar populations.

Interpretation – There are several steps in the interpretation of spirometry testing performed as part of the evaluation of workers at risk of occupational ILD. First, the interpreter must review and comment on test quality and determine whether acceptability criteria are met. If the test is considered adequate for interpretation, then adjust for age, height, gender and race/ethnicity using appropriate reference tables for normal or predicted values.

For patients who have previously completed spirometry, changes in test results are evaluated over time.

_Spirometry for Occupational Interstitial Lung Disease Diagnosis and Monitoring_

**B.6.a.i Spirometry**

**Recommended** in the diagnostic work-up and monitoring of individuals with occupationally related interstitial lung diseases.

*Indications – Diagnostic*: Patients with history and/or chest radiography consistent with ILD and workplace exposure consistent with plausible etiologies (e.g., worker complaining of chronic or intermittent cough, shortness of breath, or decreased physical abilities). Spirometry should generally be postponed if there has been recent surgery, respiratory infections, or recent cardiac problems.

*Indications – Monitoring/Surveillance*: Periodic spirometry (yearly) with longitudinal evaluation of loss of pulmonary function is recommended for workers in occupations with exposures that are either known or thought to be associated
with development of occupational lung disease. Longitudinal evaluation is accomplished by tracking FEV1 loss over a period of time, since the FEV1 is the most repeatable lung function parameter. Such evaluation should be calculated when spirometry tests are of adequate technical quality. In general, a loss of FEV1 in excess of 50 ml/year is considered a loss of pulmonary function in excess of the aging effect. The American College of Occupational Medicine (ACOEM), the American Thoracic Society (ATS) and the National Institute of Occupational Safety and Health (NIOSH) all have different proposed methodologies to calculate the loss of pulmonary function and determine if such loss is above the expected age-related loss of pulmonary function. Computerized software is available to calculate trends over time, such as NIOSH's Spirola.

Evidence for Use of Spirometry

B.6.b Static (Full) Lung Volumes

Measurement of static lung volumes, including Total Lung Capacity (TLC), Functional Residual Capacity (FRC) and Residual Volume (RV), is indicated to complement the information obtained on a spirometry test when further clarification of diagnosis is indicated.

The finding of a reduced FVC on spirometry could be due to several disease processes. In order to fully clarify a reduced FVC on spirometry, measurement of static lung volumes is required to confirm the diagnosis of a true restrictive disorder, i.e., a reduced TLC below lower limits of normal.

Static lung volumes can be used in obstructive diseases as well to assess the existence of air trapping, for example in emphysema or asthma. In these conditions, the TLC is increased as is the RV/TLC ratio.

Measurement of static lung volumes can be accomplished by inert gas dilution or body plethysmography.

**Recommended** – in the assessment of Occupational ILD to clarify a reduced FVC on spirometry, especially when the FEV1/FVC ratio is normal.

**Indications** – Static lung volumes are recommended in the assessment of Occupational ILD to clarify a reduced FVC on spirometry, especially when the FEV1/FVC ratio is normal.

B.6.c Measurement of Oxygenation

Measurement of oxygenation can be accomplished by non-invasive oximetry or by arterial blood gas sampling.
Non-invasive oximetry measures oxyhemoglobin or oxygen saturation of the hemoglobin. It is a simple method commonly used in the outpatient setting. Arterial blood gas is helpful in accurately measuring the partial pressure and saturation of oxygen and allows the calculation of alveolar-arterial oxygen gradient.

**Recommended** – Non-invasive oximetry measurements of oxygenation (pulse oximetry) in the evaluation and management of Occupational ILD.

**Recommended**. Arterial blood gas measurements in select patients where accurately measuring the partial pressure and saturation of oxygen and calculation of alveolar-arterial oxygen gradient is indicated.

**Indications** – Measurements of oxygenation are recommended in the assessment of Occupational ILD.

### B.6.d  Chest Radiographs

Evaluation of pulmonary occupational disease should include imaging studies. At minimum, a chest radiograph PA and Lateral should be part of the diagnostic work-up. It is preferable that chest radiographs should be interpreted according to the International Labor Organization Classification for Pneumoconiosis.

Radiographs provide structural anatomic information about the lung parenchyma and pleura that informs the differential diagnosis of occupational ILD and also provides information about the extent of involvement and progression of disease. However, although radiographs may assist in the diagnosis of occupational lung diseases, they are less sensitive and specific than CT/HRCT.

Radiographs should be interpreted by a physician with **appropriate training, experience, and skills** in interpretation of radiographs for diagnosis of ILD and occupational lung disease. To document the patterns and severity of radiographic appearances of pneumoconiosis, radiographs are preferably interpreted according to the International Labour Organization (ILO) classification\(^8\) by readers who have "B" reader certification for this classification system or individuals with appropriate training and skills. The Board recognizes that other standard-setting organizations require “B” reader qualifications for interpretation of radiographs in certain situations.

**Evidence for Use of Chest Radiographs**

#### B.6.d.i  Posterior-Anterior (PA) and Lateral Chest Radiographs

**Recommended** for the diagnosis of occupational interstitial lung disease.
Performed – Physicians who interpret chest radiographs for
diagnosis of occupational lung disease should have appropriate
training, experience, and skills and have "B" reader certification
for the ILO classification system or individuals with appropriate
training and skills.

B.6.e High Resolution Computed Tomography (HRCT) Scans
/Computed Tomography (CT)

HRCT/CT should be considered in the evaluation of occupational ILD,
when additional diagnostics are required based on clinical findings
(including spirometry and chest X-Ray). Readers of HRCT/CT scans for
occupational lung disease should have appropriate training and
experience. It is recommended that a specialized thoracic radiologist
review the chest CT scan.

B.6.e.i HRCT/CT

Recommended - in the evaluation of occupational ILD to
confirm or exclude the diagnosis of ILD

Indications/Technique: HRCT/CT may be helpful in confirming
or excluding a diagnosis of occupational ILD.
- When indicated HRCT/CT of the chest should include lung,
mediastinal and high-resolution windows.
- HRCT/CT is generally performed in the supine position, but
prone imaging may be of use in certain circumstances, for
example, confirmation that subtle peripheral and/or basilar
findings represent interstitial abnormality.
- Inspiratory/expiratory imaging is particularly useful when
considering air trapping with associated with HP.

Recommended - in the diagnostic work up of pneumoconiosis
and other pulmonary occupational diseases especially in those
lung diseases that result in increased risk for lung cancer, as
this imaging study not only has diagnostic value but can be
used as a screening test for early detection of lung cancer

Evidence for the Use of HRCT

B.6.f Magnetic Resonance Imaging (MRI) of the Chest

Not Recommended - as a primary diagnostic tool for occupational ILD.

B.6.g PET/CT Scans of the Chest

Recommended - in select cases in the evaluation of cancer associated
with ILD (lung cancer and mesothelioma) and certain other comorbid
conditions.
B.6.h Carbon Monoxide Diffusing Capacity (DL\textsubscript{co})

DL\textsubscript{co} (Diffusing capacity of the lungs) is a test that measures the movement of gas from the lungs (alveoli/ air spaces) to blood flowing in the pulmonary capillaries. DL\textsubscript{co} is typically used to describe the single breath diffusing capacity test which measures this diffusion. In this test the patient inhales a known amount of CO and the difference between what is inhaled and the CO measured in the exhaled gas is measured as the diffusing capacity (for a gas) of the lungs into blood. The test indirectly assesses the ability of the lungs to transfer oxygen to blood through the use of a calibrated test gas, CO.

Using appropriate methods for the test and adjustments for the results the test can be used to assess lung function and the presence of several lung diseases including ILD. The test should be performed according to the ATS/ERS statement published in 2017. This statement includes the methods and adjustments that must be made to obtain a valid test.

https://protect2.fireeye.com/url?k=a4914a5d-f8b5bc3c-a493b368-0cc47a6d17e02ee7efdddf3f04a65&u=https://www.thoracic.org/statements/resources/pft/DLCO.pdf

Further:
- At least two DL\textsubscript{co} tests should be performed and the average reported.
- The two measurements for the DL\textsubscript{co} should agree within 10%.
- It is important to obtain smoking status as cigarette smoking may cause measurable baseline levels of CO causing an increased back-pressure and carboxyhemoglobin.
- It is important to have available the patient's hemoglobin, as anemia will lower the measured diffusion. Equations for correction of anemia are available.

B.6.h.i Carbon Monoxide Diffusing Capacity (DL\textsubscript{co})

**Recommended** - for use in diagnosing occupational lung disease.

**Indications** – DL\textsubscript{CO} may be used to help in diagnosing gas exchange abnormalities in patients with lung disease.

**Advantages and Limitations** – DL\textsubscript{CO} may be affected by different diseases and exposures (Table 3). These **must** be considered when interpreting the test results.

**Evidence for the Use of DL\textsubscript{co}**
Table 3. Diseases /Conditions Associated with Alterations in DLCO

<table>
<thead>
<tr>
<th>Diseases/Conditions that Decrease DL&lt;sub&gt;CO&lt;/sub&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Reduced effort or respiratory muscle weakness</td>
</tr>
<tr>
<td>• Thoracic deformity preventing full inflation</td>
</tr>
<tr>
<td>• Anemia</td>
</tr>
<tr>
<td>• Pulmonary emboli</td>
</tr>
<tr>
<td>• Hb binding changes (e.g., HbCO, increased Fl, O&lt;sub&gt;2&lt;/sub&gt;)</td>
</tr>
<tr>
<td>• Valsalva maneuver</td>
</tr>
<tr>
<td>• Lung resection</td>
</tr>
<tr>
<td>• Emphysema</td>
</tr>
<tr>
<td>• Interstitial lung disease (e.g., IPF, sarcoidosis)</td>
</tr>
<tr>
<td>• Chronic beryllium disease (CBD)</td>
</tr>
<tr>
<td>• Pulmonary edema</td>
</tr>
<tr>
<td>• Pulmonary vasculitis</td>
</tr>
<tr>
<td>• Pulmonary hypertension</td>
</tr>
</tbody>
</table>


B.6.i Biological Sampling

B.6.i.i Invasive Procedures

Including, but not limited to, bronchoscopy, bronchoalveolar lavage analysis and lung biopsy are not routinely required to diagnose occupational lung disease, but should be included as part of the diagnostic armamentarium when clinically indicated and/or necessary to confirm or exclude a specific diagnosis. Often specific CT findings are considered diagnostic in certain conditions.

B.6.i.ii Sputum Samples and Bronchoalveolar Lavage (BAL)

If there is insufficient objective clinical evidence obtained from physical examination, chest radiographs and spirometry, additional testing including biological sampling may be indicated to confirm the diagnosis of occupational ILD.

B.6.i.ii.a Sputum Sample (both induced and spontaneous)

**Recommended** - in select patients as an aid for the diagnosis of occupational lung disease.

**Indication/Technique** - If insufficient clinical objective evidence is obtained from physical examination, chest radiographs and spirometry, additional testing including sputum sampling may be indicated to confirm the diagnosis of
occupational ILD. Sputum sampling may support the diagnosis of occupation lung disease but is not required given the availability of modern testing (i.e. HRCT). Sampling is done by having the patient cough to attempt to produce the sputum from deep within the lungs. It is recommended that each sample be at least 15mL to help increase the sensitivity of the sample.

B.6.i.ii.b Bronchoalveolar Lavage

**Recommended** - in select patients as an aid for the diagnosis of occupational lung disease.

*Indications/Technique* – To assist in the diagnosis of occupationally-related interstitial lung disease. BAL may support the diagnosis but is not required given the availability of modern testing (i.e. HRCT).

BAL should be performed according to the ATS guidelines on performance of BAL for ILD.

B.6.i.iii Bronchoscopy and/or Lung Biopsy

**Recommended** - in very select patients to confirm or exclude diagnosis in specific cases

_Evidence for the Use of Bronchial Alveolar Lavage (BAL) and Sputum_

B.7 Management of Occupational Interstitial Lung Disease

Management of workers diagnosed with occupational ILD is aimed at preventing further loss of lung function by decreasing inflammation and preventing the progression of lung scarring.

- Avoid additional provocative exposure to protect from disease progression.
  - Exposure assessment for workers diagnosed with occupational ILD to determine whether a worker might return to a specific job/exposure including use of PPE.
  - Avoid source of the problem.
  - Stop smoking and avoid passive smoke exposure.
  - Avoid airway irritants such fragrances, solvents and dust.

- Pharmacological treatment.
  - Follow established guidelines for treatment of ILD.
  - Bronchodilators, inhaled corticosteroids, cytotoxic drugs or immunotherapy.
Monitor Progress
  o Periodic medical follow-up, including PFTs and imaging studies for pulmonary ILD
  o Six-minute walk test as a means to monitor response to treatment or progression of disease

Minimize and manage potential complications of ILD
  o Immunization against pneumococcal pneumonia and influenza
  o Monitor for acute flare-up
  o Aggressive management of respiratory infections with a low threshold for hospitalization
  o Specific management of co-morbidities (including potential opportunistic infections, cancer)
  o Supportive (supplemental) oxygenation if desaturation is documented during exertion or sleep
  o Management of cardiac complications (e.g. pulmonary hypertension, right-sided heart failure, CHF).

Screen for lung cancer

Pulmonary Rehabilitation to improve functional capacity
  o Alternate efficient breathing methods
  o Evaluate and maximize home environment to save exertional energy
  o Maintain caloric intake

B.7.a Lung Transplantation

Recommended - In advanced or rapidly progressive cases, evaluation for lung transplantation should be performed.

B.7.b Pharmacological Treatment

Recommended – the goal of pharmacologic treatment for occupational ILD primarily addresses symptoms and limitations, it cannot reduce fibrosis. Recommendations for the pharmacological treatment of ILD should follow those of ATS or similarly recognized guideline issuing organizations.

Workers with clinical findings consistent with a given type of occupational ILD should be referred to a physician with training and experience in medical management of that condition.

B.7.c Exposure Assessment

Recommended - that an exposure assessment be completed for workers diagnosed with occupational interstitial lung disease.

Exposure Assessment for Workers with Occupational ILD - Exposure data from industrial hygiene surveys and Safety Data Sheets (formerly
known as Material Safety Data Sheets) and other sources such as area or personal monitoring data should be reviewed and considered for each worker diagnosed with occupational ILD.

**Rationale for Recommendations** - Exposure assessment data are necessary to determine past and present exposures to specific agents, to ascertain the degree of respiratory hazards that exist, and to identify appropriate personal protective equipment to reduce exposure.

The ability of a worker to use appropriate personal protective equipment to protect from further exposure is dependent upon pulmonary function and the physical demands of the job. Generally speaking, workers with severe to very severe respiratory impairment may not have sufficient inspiratory capacity to work while wearing respirators that increase the work of breathing (such as half-or full-face filtering respirators), and likewise may not be able to perform the functions of an occupation requiring moderate physical activity.

**B.7.d 6-Minute Walk Test (6MWT)**

The 6-minute walk test is a prognostic tool used for monitoring individuals to assess performance/functional ability over time. The test measures the distance a patient can walk on a flat, hard surface in a period of 6 minutes.

**Recommended** - in individuals with interstitial lung disease as a means to monitor response to treatment or progression of the disease.

**Indication/Technique** – To measure the response to medical interventions in patients with moderate to severe lung disease. It may also be used as a measure of functional status of patients as well as a predictor of morbidity and mortality.

Absolute contraindications for the 6MWT include:
1. History of unstable angina.
2. Heart attack within the previous month.

Relative contraindications for the 6MWT include:
1. Resting tachycardia (>120 beats/minute).
2. Uncontrolled hypertension.

Reasons for immediately stopping the test are chest pain, intolerable dyspnea, leg cramps, staggering, excessive diaphoresis, and pale or ashen appearance.

**Evidence for the Use of the 6-Minute Walk Test**
Appendix 1: The ILO Classification

The ILO Classification depends on 22 standard reference radiographs that are used to formally identify and characterize pneumoconiosis and related pulmonary abnormalities arising from occupational exposure. These reference radiographs demonstrate a variety of types and severities of lung abnormalities that frequently arise from occupational dust exposure. Proper use of the classification involves a visual comparison of the test subject’s x-ray film side-by-side with the standards. The test subject is assigned the classification pertaining to the standard radiograph or radiographs to which it is most similar in appearance. Ie. Category 0/0, 1/1, 2/2 or 3/3; and the types p/p, q/q, r/r, s/s, t/t or u/u where applicable. The person undertaking the classification, typically a physician formally trained in the use of the ILO Classification, completes a data entry sheet where they record their classifications of each of the various abnormalities. In addition, ancillary information on the quality of the radiograph and the presence of other medical findings is noted. The ILO classification system has been shown to be related to the amount and composition of dust retained in the lung.
## Appendix 2: Evidence Tables

### Evidence for the Use of Spirometry


<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Score</th>
<th>N</th>
<th>Test Used</th>
<th>Comparison Test</th>
<th>Population</th>
<th>Length of Follow-up</th>
<th>Outcome measures</th>
<th>Results</th>
<th>Conclusion</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Miller 1994</td>
<td>7.0</td>
<td>2611</td>
<td>Spirometry</td>
<td>Chest radiography, History</td>
<td>Insulators working pre 1970s with asbestos exposure</td>
<td>None</td>
<td>Radiography, Smoking status, FEV₁, FVC, FEV₁/FVC</td>
<td>Non-smokers with asbestos exposure: 172/515 (33%) had abnormal FVC, 31/515 (6%) had reduced FEV₁/FVC. Smokers: 971/2096 (46%) had abnormal FVC, 518/2096 (25%) with reduced FEV₁/FVC.</td>
<td>&quot;That reduced FVC and reduced FEV₁/FVC are both more frequent in insulators who have smoked (compared with NS insulators or smokers in the general population) suggests an interaction between asbestos and smoking in producing both these physiologic abnormalities.&quot;</td>
<td>Eighty-seven percent of participants had 30+ years exposure to insulation. Diagnosis of asbestosis made with chest radiography only. No baseline data on other exposures or disease. Data suggest spirometry is sensitive to radiographic findings in workers exposed to asbestos. Sensitivity increased in workers with smoking history.</td>
</tr>
<tr>
<td>Wang 1999</td>
<td>7.0</td>
<td>130</td>
<td>Spirometry</td>
<td>Chest radiography, DLco</td>
<td>Male Chinese refractory plant workers</td>
<td>None</td>
<td>Radiography, FEV₁, FEV₁/FVC ratio</td>
<td>Radiographic hyperinflation was related to silicosis diagnosis. Relationship between radiographic hyperinflation was stronger than silicosis when looking at decreased spirometry values (p &lt;0.05).</td>
<td>&quot;The findings indicate that emphysema associated with silicosis is likely to be responsible for the pulmonary obstruction and decreased diffusing capacity.&quot;</td>
<td>Authors had access to environmental readings on dust exposure. &quot;Controls&quot; younger and still working while majority of &quot;cases&quot; were retired. Evaluated smoking in regression analysis. Data suggest silicosis causes decrease in FVC, FEV₁, FEV₁/FVC that correlates with chest radiograph findings. Emphysema common in silicosis patients.</td>
</tr>
<tr>
<td>Kilburn 1994</td>
<td>6.5</td>
<td>2,662</td>
<td>Spirometry TLC</td>
<td>Chest radiography</td>
<td>1,146 men with asbestosis and 1,146 age-matched exposed to asbestos without a diagnosis of asbestosis, 320 unexposed controls</td>
<td>None</td>
<td>Chest radiography Spirometry values Smoking status Symptoms</td>
<td>Never smoked: Controls compared to exposed group had no significant change in FVC, FEV1, FEF25-75. Controls compared to asbestosis group had significant difference in all parameters (p &lt;0.004).</td>
<td>“Asbestos exposure reduced flows and produced air trapping after 20 years in workers who never smoked. Smoking increases these abnormalities.”</td>
<td>Case-control study design. Occupational exposure measured by interview. Used smoking as stratification. Data suggest spirometry values may be used in diagnosing and screening for asbestosis in conjunction with chest radiography.</td>
</tr>
<tr>
<td>Barnhart 1988</td>
<td>6.5</td>
<td>40</td>
<td>TLC Spirometry</td>
<td>Chest radiography DLco P(A-a)O2</td>
<td>Cases referred to occupational medicine because of concern with asbestosis</td>
<td>None</td>
<td>Chest radiography TLC FEV1 FVC DLco P(A-a)O2</td>
<td>Group 1 (interstitial fibrosis and COPD) had no case of restriction on TLC. There was decreased FEV1 (p&lt;0.001) compared to Group 2 which had only interstitial fibrosis on chest radiography.</td>
<td>“[I]n patients with asbestos exposure, radiographic fibrosis, and COPD, the TLC is an insensitive test for indicating functional effect of asbestos-induced fibrosis. In the setting of airflow obstruction, caution should be used in excluding adverse respiratory effect due to asbestos exposure through the use of TLC.”</td>
<td>Much of data collected by retrospective chart review. Two readers read chest radiographs. Asbestos exposure done by patient interview. Data suggest TLC is an insensitive measure of lung restriction due to asbestos exposure in patients who also have COPD. Multiple measures should be taken into consideration in diagnosis of asbestosis.</td>
</tr>
<tr>
<td>Leung 2005</td>
<td>6.5</td>
<td>1,576</td>
<td>Spirometry</td>
<td>Chest radiography</td>
<td>Cases referred to the statutory Pneumoconiosis Medical Board for assessment</td>
<td>None</td>
<td>FVC FEV1/FVC (FER)</td>
<td>55.6% had normal spirometry; 7.6% had reduced FVC with normal FER; 8.4% had reduced FVC and FER. On regression analysis: age, smoking, history of TB, size of lung nodules and PMF were independent predictors of airflow obstruction.</td>
<td>“In an occupational compensation setting, disease indices and history of tuberculosis are independent predictors of both airflow obstruction and reduced capacity for silicotic patients.”</td>
<td>Patients diagnosed with silicosis if they had nodules scored as &gt;1/0 in ILO classification system. A record review study. Data suggest patients with radiographic evidence of silicosis may have decreased lung function, but that more than half have normal values on spirometry.</td>
</tr>
<tr>
<td>Rosenman 2010</td>
<td>6.0</td>
<td>526</td>
<td>Spirometry</td>
<td>Chest radiography</td>
<td>“Confirmed” silicosis patients either by chest</td>
<td>None</td>
<td>Radiography FVC, FEV1, FEV1/FVC ratio</td>
<td>Obstruction on spirometry: “Both obstructive and restrictive patterns were observed regardless of</td>
<td>Obtained chest radiography and spirometry values by medical record review.</td>
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<tr>
<td>Study</td>
<td>Year</td>
<td>N</td>
<td>Spirometry</td>
<td>Radiology or biopsy</td>
<td>Smoking status</td>
<td>Radiographic Findings</td>
<td>Spirometric Findings</td>
<td>Notes</td>
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<tr>
<td>Brodkin 1993</td>
<td>6.0</td>
<td>812</td>
<td>Spirometry</td>
<td>Chest radiography</td>
<td>None</td>
<td>Radiography FVC, FEV₁, FEV₁/FVC</td>
<td>OR for restrictive ventilator impairment: Cough 0.91 (p = NS), phlegm 0.83 (p = NS), wheezing 2.18 (p &lt;0.01), [smoking ever/never] 0.85 (p = NS), parenchymal small opacities 1.41 (p &lt;0.001), pleural thickening 1.06 (p = NS).</td>
<td>These results support the validity of the ATS questionnaire as an epidemiological tool and emphasize the importance of clinical history in assessing respiratory status.</td>
<td>Data suggest report of wheezing, dyspnea have strongest association with ventilatory defects. Reported a significant correlation of radiographic findings with ventilatory defects.</td>
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<tr>
<td>Kilburn 1985</td>
<td>6.0</td>
<td>257</td>
<td>Spirometry</td>
<td>Chest radiography</td>
<td>None</td>
<td>Radiography FVC, FEV₁, FEF₂₅₋₇₅, FEF₇₅₋₁₆₀, DLCO</td>
<td>14/43 (33%) nonsmokers had 1/1 radiographs with normal spirometry values. Current and ex-smokers had a downward trend in same values.</td>
<td>These shipyard workers had minimal to moderate asbestosis with much pleural disease and little functional impairment when compared to a smoking-specific reference population.</td>
<td>Used PA and lateral chest radiographs with 3 different B readers to diagnosis asbestosis in shipyard workers. Included smoking as a variable. Data suggest earlier asbestosis does not cause a significant drop in FEV₁, FVC in older shipyard workers.</td>
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<tr>
<td>Aaron 1999</td>
<td>5.5</td>
<td>1,831</td>
<td>Spirometry</td>
<td>Helium dilution Plethysmography</td>
<td>Uncertain</td>
<td>TLC VC, FEV₁, FVC, FEV₁/FVC</td>
<td>Sensitivity: 193/225 (86%); Specificity: 1,329/1606 (83%); PPV: 193/470 (41%); NPV: 1,329/1,361 (97.6%)</td>
<td>[T]he accuracy with which spirometric measurement of FVC and expiratory flow rates can diagnose the presence of a restrictive impairment. Patients whose FVC fall above the 95% CI of Uncertain what type of patients included in study. Does not appear to have any occupationally-related cases. Data suggest spirometry is useful in ruling out a restrictive lung disease diagnosis.</td>
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Non-Occupational Interstitial Lung Disease

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<tr>
<th>Study</th>
<th>Year</th>
<th>N</th>
<th>Spirometry</th>
<th>Radiology or biopsy</th>
<th>Smoking status</th>
<th>Radiographic Findings</th>
<th>Spirometric Findings</th>
<th>Notes</th>
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</table>

**Radiography or biopsy or both**

17.3% of non-smokers (NS) 26.5% of smokers (S) Restriction: 30.1% NS, 28.1% S Mixed: 22.4% NS, 25.7% S

17.3% of non-smokers (NS) 26.5% of smokers (S) Restriction: 30.1% NS, 28.1% S Mixed: 22.4% NS, 25.7% S

**Smoking status obtained by interview of worker or next of kin or medical record review. Data suggest both restrictive and obstructive results may occur in workers with silicosis on spirometry. Less than half of workers diagnosed with silicosis had abnormal spirometry.**
### Boros 2004

<table>
<thead>
<tr>
<th>4.0</th>
<th>1,173</th>
<th>Spirometry</th>
<th>Whole body plethysmography</th>
<th>Mean age 44.3 – with HP (74), sarcoidosis (568), pulmonary fibrosis (194), connective tissue disease (51), and pneumoconiosis (23)</th>
<th>None</th>
<th>TLC</th>
<th>VC</th>
</tr>
</thead>
<tbody>
<tr>
<td>882/1,173 (75.2%) both indices were above (LLN), 267/1,173 (22.8%) TLC was markedly reduced, 209/1,173 (17.8%) VC reduced (p &lt;0.01), 185/1,173 (15.8%) had both indices reduced.</td>
<td>&quot;Our results indicate that the spirometric measurement of VC is not enough for the detection of restriction, and may result in missing the diagnosis of diminished lung volume in almost 10% of patients. Thus in order to assess lung function reliably in ILD patients, the measurement of TLC seems to be essential.&quot;</td>
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</tbody>
</table>

### Sircar 2007

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<thead>
<tr>
<th>4.5</th>
<th>1,730</th>
<th>Spirometry</th>
<th>None</th>
<th>Coal miners</th>
<th>FEV₁, Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>Odds ratios: Compared to below 30ml/year loss, 1.39 (0.99-1.97) 60ml/year to 90ml/year 1.90 (1.32-2.76) more than 90ml/year loss of FEV₁.</td>
<td>&quot;Risk of death increases in individuals with rates of decline above about 60ml/year and is statistically significant with declines of 90ml or more. These results should be useful to healthcare providers in assessing lung function declines observed in individuals.&quot;</td>
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How each patient was originally diagnosed not described. Small number of pneumoconiosis patients. No separation of results based on diagnosis. Making this study difficult to assess in terms of occupational lung disease. Data suggest that in generalized ILD patients both VC and TLC is useful.

### Other

- Making this study difficult to assess in terms of occupational lung disease. Data suggest that in generalized ILD patients both VC and TLC is useful.
## Evidence for Use of Chest Radiographs

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Score (0-11)</th>
<th>N</th>
<th>Test Used</th>
<th>Comparison Test</th>
<th>Population</th>
<th>Length of Follow-up</th>
<th>Outcome Measures</th>
<th>Results</th>
<th>Conclusion</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sun 2008</td>
<td>9.5</td>
<td>90</td>
<td>Chest x-ray</td>
<td>HRCT</td>
<td>Mine-machine manufacturing workers in China involved in sand casting</td>
<td>None</td>
<td>Radiography classifications</td>
<td>Of 30 employees without silicosis on x-ray, 8 (28%) had evidence of silicosis on HRCT.</td>
<td>&quot;HRCT is not currently accepted as a diagnostic tool for the detection of pneumoconiosis…HRCT scans should be considered for the better and earlier diagnosis of pneumoconiosis.&quot;</td>
<td>Both x-ray and HRCT scan readers blinded to diagnosis status. All patients male. No background information given such as smoking status or other exposures. Data suggest HRCT will detect evidence of silicosis earlier than x-ray.</td>
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<tr>
<td>Paris 2004</td>
<td>9.5</td>
<td>706</td>
<td>PA chest x-ray</td>
<td>High resolution CT, basilar crackles, age, cumulative exposure index to asbestos fibers, Total Lung Capacity</td>
<td>Retired asymptomatic workers with documented asbestos exposures. Average age 65.2, 89% male.</td>
<td>None</td>
<td>ILO classification, Plethysmography, CEI, Clinical examination</td>
<td>Compared to HRCT scan as gold standard: Small irregular opacities in x-ray: Sn: 46% Sp: 80%. Pleural abnormalities: Sn: 66% Sp: 47%. Basilar crackles: Sn: 46% Sp: 76%. Low TLC: Sn: 27% Sp: 85% CEI: Sn: 95% Sp: 18%</td>
<td>&quot;Our findings confirm that HRCT can detect early-stage asbestosis in people who have been highly exposed to asbestos whose X-ray can be considered normal…Moreover, HRCT screening does not seem warranted for people with low occupational exposure (CEI &lt;25 fibers/ml x years)...&quot;</td>
<td>All participants had no known asbestos related disease. X-rays and HRCT scans read by 3 independent readers blinded to patient status. Data suggest a combination of clinical exam, exposure history and testing increases both sensitivity and specificity in diagnosing asbestosis.</td>
</tr>
<tr>
<td>Vallyathan 1996</td>
<td>8.0</td>
<td>430</td>
<td>PA X-ray</td>
<td>Autopsy results</td>
<td>Coal miners in West Virginia exposed to medium to high rank bituminous coal</td>
<td>None</td>
<td>Pathology X-ray readings</td>
<td>298/430 (69%) of films were classified as &gt;0/1 (41%) classified as 2/1 or greater.</td>
<td>Overall the study showed good agreement between the predicted probabilities and observed responses of a profusion category &gt;= 0/1 for pathologic CWP lesions. However, the study also showed that CXR were insensitive for detecting minimal CWP lesions, and were unreliable indicators in X-rays were PA and read by 3 different readers. Average age of death 68, but no data on cause of death. Data suggest that PA x-rays may assist in diagnosis of CWP but will often miss smaller lesions less than 3-5mm in diameter.</td>
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<tr>
<td>Year</td>
<td>Authors</td>
<td>Study Design</td>
<td>Study Population</td>
<td>Diagnosis Method</td>
<td>Results</td>
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<tr>
<td>Wain 1984</td>
<td>8.0 50</td>
<td>PA X-rays only</td>
<td>Autopsy results</td>
<td>X-ray findings Autopsy results</td>
<td>Patients with plaques on autopsy from a Veterans hospital. Controls. Prevalence of pleural plaques on autopsy 5.8%. 7/25 (28%) of autopsy-confirmed cases had evidence of plaques on x-ray. None of controls had evidence of plaques on x-ray. “It is clear that an accurate occupational history is essential for the recognition of relationships between asbestos and pleural plaques, carcinoma, and asbestos body counts.” Occupational/exposure history obtained through chart review. X-rays PA only. All male veterans. Data suggest PA x-rays have high specificity but low sensitivity for detection of pleural plaques in patients exposed to asbestos.</td>
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<tr>
<td>Kipen 1987</td>
<td>7.0 138</td>
<td>PA X-ray</td>
<td>Autopsy results</td>
<td>X-ray findings Autopsy pathology</td>
<td>Asbestos insulation workers who died from lung cancer. All 138 cases had histologic evidence of parenchymal fibrosis. 10/138 (10%) negative for any fibrosis on x-ray. “Discrepancies in the results of radiological and pathological examination for interstitial fibrosis were present in 18% of those heavily exposed insulators… These findings indicate the primacy of the history of asbestos exposure, irrespective of the presence of absence of non-malignant x-ray changes (asbestosis)…” Consensus of 3 x-ray readers taken. No mention of blinding done. Data suggest that a negative x-ray does not rule out moderate to severe interstitial fibrosis in workers exposed to asbestos.</td>
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<tr>
<td>Ruckley 1984</td>
<td>7.0 261</td>
<td>X-ray</td>
<td>Lung tissue</td>
<td>ILO classification Emphysema Years Death</td>
<td>Male coal miners. 45% of men with no opacity on x-ray had simple pneumoconiosis. In x-rays with p type opacities 89% had simple pneumoconiosis. In x-rays with q or r irregularities 61% had simple pneumoconiosis. Intra-observer variation was small, inter-observer variation evident. Lungs with no x-ray opacities had fewer foci that were small and rarely palpable. “This study has shown that the composition of dust retained in the lung, as well as its amount, makes an important contribution to the radiographic appearances of pneumoconiosis.” Study used lung tissue to confirm dust burden and emphysema diagnosis. No good baseline data on participants such as smoking status/years exposed. Data suggest certain level of dust burden must be met before x-ray opacities are seen. Certain types of opacities signify different types of dust exposure in coal miners; 45% of lungs with simple pneumoconiosis had normal chest x-ray.</td>
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<tr>
<td>Year</td>
<td>Duration</td>
<td>Sample Size</td>
<td>Imaging</td>
<td>Histology</td>
<td>Control Group</td>
<td>Findings</td>
<td>Discussion</td>
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<tr>
<td>1972</td>
<td>7.0</td>
<td>98</td>
<td>X-ray</td>
<td>Lung tissue</td>
<td>Coal miners in England</td>
<td>Correlation between pneumoconiosis score and lung dust content was $r = 0.90$. Iron and other mineral contents of coal is important in disease status.</td>
<td>Used one of the lungs to determine dust burden. Data suggest the higher the amount of dust in lungs the more opacities seen on chest x-ray.</td>
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<tr>
<td>1987</td>
<td>7.0</td>
<td>71</td>
<td>X-ray</td>
<td>Lung biopsy</td>
<td>Coal miners</td>
<td>Lungs classified as category O may contain several pinhead fibrotic lesions up to &gt;3mm in diameter. Subjects with predominately p opacities contained more macules and pinhead fibrotic nodules than those of subjects presenting q or r opacities.</td>
<td>“For the main, homogenous group of 98 miners, the correlation between the simple pneumoconiosis score and the coal and other mineral contents was 0.9.”</td>
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<tr>
<td>2008</td>
<td>6.5</td>
<td>53</td>
<td>X-ray</td>
<td>HRCT, Spirometry, Helium Dilution, DLco</td>
<td>Workers exposed to silica - mainly sandblasters and stone cutters</td>
<td>Small opacities: concordance between radiographs and CT scans was 56.8%. For large opacities, concordance was 70.5%.</td>
<td>The results of this study and others make increasingly clear the complexity of the relation between what is seen on a chest radiograph and what is present in the lungs of coalworkers, and emphasize the fundamental importance of the character of the dust lesions and the composition of the dust itself.</td>
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<tr>
<td>1998</td>
<td>6.5</td>
<td>240</td>
<td>X-ray</td>
<td>Symptom questionnaire, Chest CT scans, Dust exposures, Spirometry</td>
<td>Coal workers 1. Exposed with x-ray findings at baseline 2. Exposed without x-ray findings 3. Less exposed without x-ray findings</td>
<td>Exposed group with x-ray findings: 24/78 (31%) had worsened x-rays at 4 years, 10/78 (13%) had developed CWP. Exposed group with normal x-rays: 6/78 (8%) had worsened x-rays. Less exposed group with normal x-rays: 1/78 had worsened x-rays.</td>
<td>Exposed group with x-ray findings and pneumoconiosis were more often observed in coal miners with micronodules on lung CT scans, wheezing, low values of MMEF and FEF25%, and high dust exposure at the first examination.</td>
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<tr>
<td>Year</td>
<td>Study</td>
<td>Subjects</td>
<td>Methods</td>
<td>Diagnosis</td>
<td>Findings</td>
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<tr>
<td>Musk 1981</td>
<td>Coal miners</td>
<td>Spirometry, Pulmonary function test with closed circuit helium dilution, Plethysmography, Exercise test, Symptom Questionnaire</td>
<td>9 years</td>
<td>ILO classification</td>
<td>Men with r opacities had a reduction in lung compliance over men with q opacities.</td>
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<tr>
<td>Brodkin 1993</td>
<td>Various workers exposed to asbestos</td>
<td>Spirometry, Symptoms-Questionnaire</td>
<td>None</td>
<td>X-ray ILO classification, Symptoms</td>
<td>Pre and post bronchodilator response X-ray ILO classification Symptoms. 324/816 (40%) had unremarkable chest x-ray. 219/816 (27%) had pleural abnormalities 100/816 (12%) had parenchymal abnormalities 169/816 had both Parenchymal small opacities on x-ray increased odds of restrictive ventilator pattern by OR 1.41 (1.32-1.52) (p &lt;0.001). No significant findings on x-ray and obstructive ventilator pattern.</td>
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<tr>
<td>Larson 2012</td>
<td>Citizens of Libby, MT who were participating in a community screening program</td>
<td>Spirometry, HRCT scans</td>
<td>None</td>
<td>X-ray ILO category</td>
<td>Some had HRCT scans (363/6476) Participants with HRCT scan 3% had parenchymal abnormalities not seen on x-rays. 77% (5003/6476) had normal spirometry. No trends between prevalence of abnormal spirometry with surrogate of amphibole exposure.</td>
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<tr>
<td>Study</td>
<td>Year</td>
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<td>Test</td>
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<tr>
<td>Collins 1988</td>
<td>5.0</td>
<td>895</td>
<td>X-ray</td>
<td>Symptom questionnaire, Work history and smoking questionnaires</td>
<td>None</td>
<td>X-ray ILO classification Symptoms Spirometry</td>
<td>Men with small opacities who were smokers 2 or 3 times more likely to report breathlessness, cough and sputum. Dust exposure increased changes of reporting same symptoms. Both age and dust exposure related inversely to lung function. “[T]he presence and profusion of small irregular opacities should be taken into consideration when assessing the severity of coal workers’ simple pneumoconiosis.”</td>
<td>Included detailed occupational exposure history, including dust samples. They also included smoking. Data suggest the small irregular opacities seen on x-ray also correlate with decreased lung function in coal workers.</td>
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<tr>
<td>Cockcroft 1983</td>
<td>5.0</td>
<td>124</td>
<td>X-ray</td>
<td>Physical exam</td>
<td>Years</td>
<td>Smoking Age Underground exposure</td>
<td>Increasing age associated with increasing irregularity of small opacities (p &lt;0.001). Smoking associated with increasing irregularity of small opacities (p &lt;0.01). “Our findings suggest that irregular opacities are related to underground exposure and should probably be considered to be part of simple coal workers’ pneumoconiosis.”</td>
<td>Included detailed occupational exposure history, and smoking status. Data suggest the irregular opacities may signify CWP with or without small regular opacities irrespective of age and smoking status.</td>
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<tr>
<td>Hurley 1982</td>
<td>4.5</td>
<td>2,600</td>
<td>X-ray</td>
<td>Symptoms, dust exposure</td>
<td>10 years</td>
<td>Classification Dust exposure</td>
<td>Men who worked longer hours in coalmining had higher prevalence of coal worker pneumoconiosis. Little evidence that exposures to quartz dust influenced the chances of developing CWP. “The radiological signs…can therefore be regarded as an indirect measure of increased risks of reduced breathing capacity, disability, and excess mortality.”</td>
<td>Included detailed occupational exposure history, including dust samples. Data suggest that overall coal dust exposure burden results in greater findings on x-ray, but higher exposure to quartz in this cohort did not seem to have an effect on development of CWP classified by x-ray.</td>
<td></td>
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</tr>
<tr>
<td>Amandus 1976</td>
<td>4.0</td>
<td>6,166</td>
<td>X-ray</td>
<td>Spirometry Symptoms</td>
<td>None</td>
<td>X-ray findings Symptoms Spirometry</td>
<td>Smoking, age, and years underground contributed significantly to prevalence of irregular lesions. “This study shows that there is a statistical association between cigarette smoking and the presence of irregular opacities. The results also suggest that other factors such as bronchitis, age, and exposure to coal dust are involved in the development of these lesions.”</td>
<td>Included smoking status. No other confirmatory test other than symptoms and some spirometry values. Data suggest that smoking, age, and years underground are associated with irregular opacities in underground coal miners.</td>
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**Evidence for the Use of HRCT**
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<tr>
<th>Author/Year</th>
<th>Score (0-11)</th>
<th>N</th>
<th>Test Used</th>
<th>Comparison Test</th>
<th>Population</th>
<th>Length of Follow-up</th>
<th>Outcome Measures</th>
<th>Results</th>
<th>Conclusion</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Collins 1993</td>
<td>10.0</td>
<td>21</td>
<td>High resolution CT scan</td>
<td>Chest radiography, Spirometry, Arterial blood gases, Physical history</td>
<td>Coal miners</td>
<td>None</td>
<td>Radiography, Spirometry</td>
<td>Of 9 patients who had negative chest radiography, 4 had evidence of nodules on HRCT scan consistent with CWP. Only miners with a history of smoking had airflow limitations.</td>
<td>&quot;For detecting evidence of coal dust accumulation in lung parenchyma and identifying focal emphysema, HRCT was more sensitive than standard chest radiography. However, despite earlier detection of parenchymal abnormalities, abnormal pulmonary function attributable to coal dust could not be identified.&quot;</td>
<td>Small sample size. Each radiograph PA and read by 2 blinded readers. Each HRCT scan read by 2 blinded radiologists. Excluded miners with evidence of airflow obstruction on spirometry. Data suggest HRCT scans are more sensitive than chest radiography in detecting nodules in miners. This earlier detection does not correlate well to functional limitations.</td>
</tr>
<tr>
<td>Newman 1994</td>
<td>9.5</td>
<td>40</td>
<td>High resolution CT scan</td>
<td>Chest radiography, Lung biopsy</td>
<td>Various workers exposed to beryllium and either positive of BeLT surveillance testing or had symptoms and chest radiography consistent with beryllium disease</td>
<td>None</td>
<td>Radiography, Biopsy</td>
<td>15/28 (54%) of biopsy confirmed cases had abnormal chest radiographs. 25/28 (89%) of biopsy confirmed cases had abnormal HRCT scans 10/13 (77%) of the normal chest radiographs and abnormal HRCT scans.</td>
<td>&quot;Thin-section CT was more sensitive than chest radiography in detection of beryllium disease, but the diagnosis was missed in up to 25% of cases with histologic proof.&quot;</td>
<td>All cases had biopsy confirmed beryllium disease and positive BeLT immunological testing. Two groups: 1) workers without symptoms but had positive BeLT immunological testing on surveillance; and 2) workers with symptoms and positive chest radiographs. Data suggest HRCT is more sensitive in detecting lung pathology in beryllium disease, but it still missed up to 25% of cases.</td>
</tr>
<tr>
<td>Gamsu 1995</td>
<td>9.5</td>
<td>30</td>
<td>High resolution CT scan</td>
<td>Biopsy</td>
<td>Workers exposed to asbestos in shipyards or construction; 6 lungs came from autopsy</td>
<td>None</td>
<td>Radiography, Biopsy</td>
<td>Pathology normal in 5/30 (15%) of cases. HRCT negative in 14/30 (48%) and positive in 16/30 (52%). When two findings were needed to diagnose the Specificity went from 60% to 100%, Sensitivity went from 88% to 78%.</td>
<td>&quot;[H]igh-resolution CT detection of asbestosis, a combination of the cumulative number of different findings and an assessment of the extent and severity of the abnormalities could be complimentary. We also conclude that asbestosis can be present histopathologically with normal or near normal high-resolution CT scan.</td>
<td>The 6 lung samples did not have pleura present and little to no clinical data available. Baseline data sparse. CT Scans and pathologists blinded. Data suggest HRCT scans are both sensitive and specific in the diagnosed of asbestosis.</td>
</tr>
<tr>
<td>Gevenois 1994</td>
<td>8.0</td>
<td>83</td>
<td>High resolution CT scan</td>
<td>Chest radiography</td>
<td>Patients involved in medicolegal evaluations</td>
<td>None</td>
<td>Radiography</td>
<td>2/9 (22%) of the patients with negative chest radiography had a positive CT scan.</td>
<td>&quot;[T]hese data point out the limited value of CR, graded according to the ILO classification to evaluate low grade CWP in exposed workers, especially when the opacities described on CR are</td>
<td>Two different readers on both chest radiography and HRCT. No baseline data noted. Data suggest HRCT is more sensitive in detecting micronodules in silicosis than chest radiography.</td>
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<tr>
<td>Study</td>
<td>N.</td>
<td>N.</td>
<td>Diagnostic Method</td>
<td>Patient Characteristics</td>
<td>Medical Imaging</td>
<td>Findings</td>
<td>Summary</td>
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<tr>
<td>Lynch 1995</td>
<td>8.0</td>
<td>63</td>
<td>High resolution CT scan</td>
<td>Open lung biopsy</td>
<td>Various</td>
<td>None</td>
<td>Radiography</td>
<td>HRCT was able to distinguish between HP and IPF in 90% of cases if they were definite, 60% if diagnosis was probable.</td>
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<td>Ziora 2005</td>
<td>7.5</td>
<td>20</td>
<td>High resolution CT</td>
<td>FEV₁, FVC, DCO</td>
<td>Patients diagnosed with HP</td>
<td>None</td>
<td>FEV₁, FVC, DCO</td>
<td>All patients had a diminished DCO. 19/20 (95%) had FVC and FEV₁ &lt;70% predicted and FEV₁/FVC &gt;/= 75%.</td>
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<tr>
<td>Huuskonen 2001</td>
<td>7.5</td>
<td>651</td>
<td>High resolution CT</td>
<td>Chest radiography</td>
<td>Workers exposed to asbestos fibers</td>
<td>None</td>
<td>Radiography</td>
<td>85/602 (14%) had a diagnosis of asbestosis. Chest radiography with ILO Sn: 51%, Sp: 89%. HRCT Sn: 70%.</td>
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<tr>
<td>Eterovic 1993</td>
<td>7.0</td>
<td>35</td>
<td>High resolution CT scan</td>
<td>Chest radiography</td>
<td>Workers in asbestos cement plant and controls</td>
<td>None</td>
<td>Radiography</td>
<td>HRCT had a higher probability score in advanced asbestosis patients than in early asbestosis. (p=0.013) Chest radiography had more advance ILO scores as asbestosis disease advanced.</td>
<td></td>
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<tr>
<td>Mosiewicz 2004</td>
<td>6.5</td>
<td>64</td>
<td>High resolution CT scan</td>
<td>Chest radiography</td>
<td>Iron foundry workers with silicosis</td>
<td>None</td>
<td>Radiography</td>
<td>HRCT and radiography were 88%-94% consistent when the findings were nodules. HRCT scan detected irregular. In this study, we confirmed that CCT and HRCT are more sensitive than CR to detect silicosis.</td>
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High-resolution CT features can be used to distinguish IPF from HP in most but not all cases. Desquamative interstitial pneumonitis cannot reliably be distinguished from acute or subacute HP.

We have found a relatively strong correlation between nodules and examined spirometric and diffusion parameters, which suggests that the presence of intraluminal granulation tissue in bronchioles and adjacent aveoli may impair the ventilatory and diffusion capacity in HP patients.

The examined HRCT scoring method proved to be a simple, reliable, and reproducible method for classifying lung fibrosis and diagnosing asbestosis also in large populations with occupational disease, and it would be possible to use it as a part of an international classification.

Although HRCT is evidently a more sensitive technique than conventional computed tomography or chest radiography for an early radiological diagnosis or asbestosis, its qualitative analysis may seem less sensitive than some simple lung function tests.

Results of HRCT correlate well with results of conventional radiography in the assessment of nodular changes in silicosis of iron foundry workers. HRCT scans are more sensitive in detecting smaller nodules.
<table>
<thead>
<tr>
<th>Year</th>
<th>Study</th>
<th>Subjects</th>
<th>Imaging Methods</th>
<th>Diagnostics</th>
<th>Findings</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aberle 1988</td>
<td>6.0 63</td>
<td>High resolution CT scan</td>
<td>CT scan, Chest radiography, PA and Lateral Spirometry</td>
<td>Workers diagnosed with clinical asbestosis and controls</td>
<td>Nodules in 45%-75% of patients with negative chest radiography for intralobular nodules and peripheral subpleural nodules. Enables significantly more frequent detection of nodular changes of small sizes, especially those localized under the pleura.</td>
<td>HRCT can complement the clinical and radiologic assessment of subjects who have had asbestos exposure. Lack of baseline characteristics such as smoking status. No mention of other exposures or health conditions. Data suggest HRCT more sensitive than CT or chest radiography in detecting subpleural lines and position may affect outcomes.</td>
</tr>
<tr>
<td>Hanak 2008</td>
<td>4.5 69</td>
<td>High resolution CT scan</td>
<td>Some patients had spirometry and some physical exams</td>
<td>Patients with a diagnosis of HP up to 9 years</td>
<td>CT scan, fibrosis. All-cause mortality</td>
<td>CT findings of parenchymal fibrosis are associated with reduced survival in patients with HP and may serve as a useful prognostic indicator. Retrospective medical record review from Jan 1997 to Dec 2002. Death data collected December 2006. Good background data. Data suggest fibrosis seen on HRCT is similar to biopsy in that it is indicative of a higher mortality rate.</td>
</tr>
<tr>
<td>Lynch 1988</td>
<td>4.0 260</td>
<td>High resolution CT scan</td>
<td>None</td>
<td>Asbestos exposed workers with inconclusive chest x-rays for asbestos related lung disease</td>
<td>None</td>
<td>27 of 260 workers had focal lung masses for a total 43 lesions. Careful interpretation of CT and high-resolution CT features and close surveillance can obviate the need for biopsy in the majority of instances. Poorly-defined centrilobular micronodules and branching linear structures were the thin-section CT findings most frequently seen in patients with arc-welder’s pneumoconiosis. All workers exposed to asbestos in construction or shipyards. All at least 10 years since exposure. Some had IV contrast. CT scans not directly compared to any other diagnostic tool so a comparison is not able to be drawn. No biopsies done.</td>
</tr>
<tr>
<td>Han 2000</td>
<td>4.0 85</td>
<td>High resolution CT</td>
<td>Clinical history in 53/85, Spirometry in 53/85</td>
<td>Welders in shipyards or assembly plants who had alleged lung abnormalities</td>
<td>None</td>
<td>79% of welders were smokers. 54/85 (64%) welders had positive findings on HRCT. 6/43 (14%) of smokers had similar findings. Poorly-defined centrilobular micronodules and branching linear structures were the thin-section CT findings most frequently seen in patients with arc-welder’s pneumoconiosis. Lack of baseline data. Two different radiologists read images. Data suggest there are findings on HRCT in workers with clinical signs or symptoms relate to welding.</td>
</tr>
<tr>
<td>Topcu 2000</td>
<td>5.0 26</td>
<td>High resolution CT</td>
<td>Chest radiography</td>
<td>Workers already diagnosed with asbestosis</td>
<td>None</td>
<td>24/26 (92%) had evidence of asbestosis on HRCT. 9/26 had apical pleural thickening, 7/26 had apical honeycombing. We suggest that the HRCT protocol for examining asbestos-exposed individuals with pleural plaques on chest X-rays should include the whole thorax, since the asbestosis-related pathologies Small numbers. Did not really compare findings in light of diagnosing asbestosis. Other exposures not well explained. Discussed tobacco use. Data suggest HRCT scans should include the spices if pleural plaques</td>
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</table>
may involve all parts of the lung."

Evidence for the Use of $\text{DL}_{\text{CO}}$

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Score</th>
<th>N</th>
<th>Test Used</th>
<th>Comparison Test</th>
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<th>Conclusion</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Eterovic 1993</td>
<td>7.0</td>
<td>35</td>
<td>Single breath $\text{DL}_{\text{CO}}$</td>
<td>Biopsy Chest radiographs HRCT- prone and supine Spirometry Stress testing on bicycle ergometer</td>
<td>Workers of chrysotile asbestos cement factory</td>
<td>None</td>
<td>$\text{DL}_{\text{CO}}$ Biopsy results</td>
<td>14/15 (93%) with advanced asbestosis had reduced $\text{DL}_{\text{CO}}$.</td>
<td>[A] biphasic mid-expiratory flow rate and change in $\text{DL}_{\text{CO}}$ (initial increase followed by a decrease) in non-smoking subjects may be the earliest functional abnormality indicative of future interstitial asbestosis.”</td>
<td>Uncertain where 13 control subjects came from. No mention if controls had biopsy or not. Data suggest changes in DLCO may be useful in diagnosis of asbestos related disease.</td>
</tr>
<tr>
<td>Dujic 1992</td>
<td>7.0</td>
<td>14</td>
<td>Single breath $\text{DL}_{\text{CO}}$</td>
<td>HRCT PA and LAT chest radiography Spirometry</td>
<td>Asbestos cement workers, average age of 42</td>
<td>9 years</td>
<td>$\text{DL}_{\text{CO}}$ (Dm and Vc) FVC FEV$_1$ ILO scores</td>
<td>DLCO increased (p&lt;0.0005) but remained in normal range. HRCT showed pleural thickening in 6 employees.</td>
<td>“Lung function test were suggested to be more sensitive than chest radiographs in detection of early asbestosis.”</td>
<td>Participants asymptomatic at start of study and had normal spirometry and chest radiographs. Exposed to predominately chrysotile asbestos; 11 non-smokers, 3 smokers. No controls. Small sample size. Data suggest decreases in DLCO may be monitored in employees exposed to asbestos before symptoms occur to help identify earlier onset disease.</td>
</tr>
<tr>
<td>Abejie 2010</td>
<td>6.5</td>
<td>454</td>
<td>Single breath $\text{DL}_{\text{CO}}$</td>
<td>PA chest radiography Spirometry</td>
<td>Chrysotile exposed workers compared to electronic workers as controls</td>
<td>None</td>
<td>$\text{DL}_{\text{CO}}$ FVC FEV$_1$/FVC ILO classification</td>
<td>Chest radiograph: 36% emphysema, 31% asbestosis, 15% both. When employees with asbestosis on chest radiograph excluded, employees exposed to asbestos had lower $\text{DL}<em>{\text{CO}}$ and FVC vs. controls. Employees with chest radiographs consistent with asbestosis had lower $\text{DL}</em>{\text{CO}}$ and FVC values vs.</td>
<td>“…our study showed that asbestos exposure with or without radiographic asbestosis is significantly associated with reduced DLCO and restrictive lung impairment. However, asbestos exposure was not significantly associated with reduced FEV$_1$/FVC.</td>
<td>Controls were younger, smoked less. Data suggest DLCO and FVC are lower in employees both exposed to asbestos and with findings on chest radiography consistent with asbestosis.</td>
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Evidence for the Use of Bronchial Alveolar Lavage (BAL) and Sputum

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Score (0-11)</th>
<th>N</th>
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<th>Comparison Test</th>
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<th>Length of Follow-up</th>
<th>Outcome Measures</th>
<th>Results</th>
<th>Conclusion</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Teschler 1996</td>
<td>7.5</td>
<td>135</td>
<td>BAL</td>
<td>Sputum tissue samples</td>
<td>Workers exposed to asbestos dust: Group 1 classified as high exposure, Group 2 as medium, Group 3 as None</td>
<td>Asbestos bodies (light microscopy, at 400x) in BAL and sputum in all subjects. Lung tissue in 21 subjects.</td>
<td>33% of subjection in group 1, 68% in group 2; 45% in group 3 had ABs in BAL but not Sputum. Open lung biopsy had ABs in all samples. Samples with less than 1,000 ABs/cm³ had no Abs in sputum samples.</td>
<td>“…many subjects with positive BAL fluid analysis had negative sputum results. These findings suggest that BAL is the superior of the two methods for assessing lung AB content.”</td>
<td>Tissue samples done only on 21 subjects. Data suggest BAL is more sensitive than Sputum in detecting FBs in subjects. No correlation is made between FBs and disease burden.</td>
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<tr>
<td>Study</td>
<td>Year</td>
<td>N</td>
<td>Method</td>
<td>Occasional exposure</td>
<td>Common finding</td>
<td>Conclusion</td>
<td>Notes</td>
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<tr>
<td>Vathesatogkit</td>
<td>2004</td>
<td>7.0</td>
<td>BAL Chest radiography HRCT scan Spirometry Dlco</td>
<td>Utility workers and controls None Asbestos bodies (light microscopy, at 40x) Respiratory symptoms Chest radiographs HRCT scans Spirometry Dlco</td>
<td>AB found in 10/30 subjects (33%) and 0/30 controls. AB positive subjects had reduced FEV1 and diffusion capacity (p &lt;0.05). HRCT scans showed higher prevalence of parenchymal disease (p &lt;0.05).</td>
<td>“In asbestos-exposed subjects, the presence of AB in BAL cytospin slides should be viewed as a clinically important finding, and their HRCT scans should be reviewed carefully for evidence of interstitial lung disease.” Two blinded pathologist read slides for AB. Data suggest detection of Asbestos bodies in utility workers represents an indicator of exposure, but not necessarily related to asbestos diseases.</td>
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<tr>
<td>Corhay</td>
<td>1990</td>
<td>4.5</td>
<td>BAL Chest radiography Spirometry Dlco</td>
<td>Steel workers and controls (white collar workers) 5 year repeat BAL in 7 subjects. Others, none. Asbestos bodies (light microscopy, at 200x)</td>
<td>Chest radiographs normal in 65 steel workers. ABs found in 38/65 (58.5%) of steel workers and 6% of controls. Smoking habits and presence of COPD did not influence AB counts.</td>
<td>“This study shows that steel workers may be subject to a nontrivial exposure to asbestos in an industrial plan environment.” Not compared to tissue samples. No sputum samples taken. Data suggest steel workers may be exposed to asbestos as part of their job.</td>
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<tr>
<td>Karjalainen</td>
<td>1994</td>
<td>4.0</td>
<td>BAL Exposure data</td>
<td>Exposed workers None</td>
<td>Asbestos bodies (light microscopy, at 200x)</td>
<td>Concentration of AB/ml found in 85% exposed to asbestos, and 7% of those not likely exposed. Patients with asbestosis (n = 9) showed higher average concentrations of AB (median 13) than patients with pleural disease only (median 2.4).</td>
<td>“…the correlation between AB concentration and exposure history was greater than in earlier studies on workers exposed to chrysotile.” No other biological testing done other than BAL. Broke analyses down by type of job. Data suggest higher concentrations of ABs seem to correlate with higher exposure and more significant disease but the correlation is not linear.</td>
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<tr>
<td>Alexopoulos</td>
<td>2011</td>
<td>7.0</td>
<td>Induced Sputum Broncho-alveolar lavage Chest radiography Spirometry ECG</td>
<td>Romanian brake factory workers without pneumo-coniosis None Total number and vitality of cells Number of dust cells Iron laden macrophages Asbestos bodies (AB)</td>
<td>In the six workers who reported using PPE none had asbestos bodies in IS or BALF. 14/39 (36%) had AB in BALF. Of those 7/14 (50%) has AB in IS.</td>
<td>IS “usefulness for screening of workers should be further evaluated because the inflammatory response in our study lacks specificity since it might have been induced [by] asbestos, dust and smoking.” At least 15 years of exposure to asbestos at &gt;5 fibers per mL. Chest radiographs &lt;= 1/0 ILO classification by two physicians. BAL performed in right middle lobe. Sputum induction done by inhaling saline then asked to cough. Study suggests IS may be helpful in proving insight for both</td>
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Evidence for the Use of the 6-Minute Walk Test

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<th>Author/Year</th>
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<th>N</th>
<th>Test Used</th>
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<th>Length of Follow-up</th>
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<th>Conclusion</th>
<th>Comments</th>
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</thead>
<tbody>
<tr>
<td>Du Bois 2011</td>
<td>5.0</td>
<td>822</td>
<td>6-minute walk test</td>
<td>Spirometry</td>
<td>Patients with confirmed IPF</td>
<td>None</td>
<td>FVC, DLCO, resting alveolar-arterial oxygen gradient (AaPo2), UC San Diego Shortness of Breath Questionnaire (UCSD SOBQ), St.</td>
<td>Distance walked during the 6MWT was correlated with FVC, DLCO, Resting AaPo2, UCSD SOBQ.</td>
<td>&quot;[O]ur results demonstrate that the 6MWT is a reliable, valid, and responsive measure of exercise tolerance in patients with IPF, and that a decline in 6MWD of 24-45 meters indicates clinical decline.</td>
<td>Data obtained during a drug study. Large sample size. All with IPF. Minimal clinical difference of 24-45 meters. Data suggest 6 minute walk test useful in determining...</td>
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<tr>
<td>Author</td>
<td>Year</td>
<td>Age</td>
<td>Gender</td>
<td>Study Design</td>
<td>Control Group</td>
<td>Test Performed</td>
<td>Details / Findings</td>
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<tr>
<td>Pimenta</td>
<td>2010</td>
<td>5.0</td>
<td>60</td>
<td>Retrospective</td>
<td>None</td>
<td>Whole-body plethysmography DLco</td>
<td>Mean distance: ILD patients 430 meters; Controls: 602 meters. SpO2 Median desaturation distance ratio: ILD patients: 10 Controls: 2.5. “Desaturation distance ratio is a promising concept and a more reliable physiologic tool to assess pulmonary diseases characterized by involvement of the alveolar-capillary membrane, such as interstitial lung diseases.” Mean age 60 for ILD patients from a tertiary referral clinic. Data suggest combination of distance and desaturations during 6MWT helps to diagnose ILD patients. Data seems weak for diagnosis of ILD as it doesn’t compare to other lung disorders, nor control for other conditions.</td>
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<tr>
<td>Alhamad</td>
<td>2010</td>
<td>4.5</td>
<td>59</td>
<td>Retrospective</td>
<td>None</td>
<td>Spirometry</td>
<td>6-minute walking distance and lowest oxygen saturation (DSP). Forced expiratory volume (FEV), (FEV1), total lung capacity (TLC), and defined product of 6Used predicted values based on age and gender.</td>
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<tr>
<td>Modrykien</td>
<td>2010</td>
<td>4.0</td>
<td>58</td>
<td>Retrospective</td>
<td>None</td>
<td>Echocardiography, distance saturation product (DSP), and pulse oximetry (SPO2)</td>
<td>Patient with pulmonary arterial hypertension (PAH) and pre-transplant diagnosis of IPF. Mean age 60 for ILD patients. Data suggest combination of distance and desaturations during 6MWT helps to diagnose ILD patients. Data seems weak for diagnosis of ILD as it doesn’t compare to other lung disorders, nor control for other conditions.</td>
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<tr>
<td>Flaherty</td>
<td>2006</td>
<td>4.0</td>
<td>197</td>
<td>Categorical</td>
<td>None</td>
<td>FVC DLco SaO2</td>
<td>Categorical baseline distance was a weak predictor of mortality for the entire cohort (p = 0.038). Baseline desaturation SaO2 &lt;= 88% had a mean survival time of 3.21 years vs. 6 month. 6 minute walk test stopped when SaO2 was &lt;=86%. No oxygen allowed during testing. Retroactive study design. Mortality appears to be all cause mortality. Data suggest in patients with IPF, desaturations at baseline, not 6MWD is a better predictor of subsequent mortality.</td>
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</table>

George’s Respiratory Questionnaire (SGRQ). and SGRQ (p <0.001). |
|                      | 6.83 years (p = 0.006) |   |   |   |   |   |
Appendix 3: Low-Quality / Supplementary Studies

The following low-quality-supplementary studies were reviewed by the Evidence-based Practice Interstitial Lung Disease Panel to be all inclusive, but were not relied upon for purpose of developing this document’s guidance because they were not of high quality due to one or more errors (e.g., lack of defined methodology, incomplete database searches, selective use of the studies and inadequate or incorrect interpretation of the studies’ results, etc.), which may render the conclusions invalid. ACOEM’s Methodology requires that only moderate- to high-quality literature be used in making recommendations. (196)

### SPIROMETRY

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Score</th>
<th>N</th>
<th>Test Used</th>
<th>Comparison Test</th>
<th>Population</th>
<th>Length of Follow up</th>
<th>Outcome Measures</th>
<th>Results</th>
<th>Conclusion</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ng 1987</td>
<td>NA</td>
<td>81</td>
<td>Spirometry</td>
<td>Chest radiography</td>
<td>Granite workers</td>
<td>10 years</td>
<td>FEV₁, FVC, radiography, exposure data</td>
<td>Workers classified as having simple silicosis had a FVC of 4% below predicted. Complicated silicosis had FVC 13% below predicted.</td>
<td>&quot;The progression of simple silicosis is thus accompanied by appreciable declines in lung function and is strongly affected by previous levels of exposure to dust.&quot;</td>
<td>No additional exposures considered. Smoking evaluated. Data suggest spirometry values decline with progression of silicosis as seen on chest radiographs and may be used in monitoring programs.</td>
</tr>
<tr>
<td>Cowie 1998</td>
<td>NA</td>
<td>242</td>
<td>Spirometry</td>
<td>Chest radiography DCO</td>
<td>Gold miners in South Africa</td>
<td>4.5 years</td>
<td>FEV₁, FVC, FEV₁/FVC DCO</td>
<td>FEV₁ loss over 4.5 years was average of 37ml/year in workers without evidence of silicosis and 125ml/year for worst cases (p = 0.000001). FVC: 15ml/year vs. 116 ml/year, DCO 0.54 vs. 1.37</td>
<td>&quot;[T]his study of a sample of a cohort of older gold miners reexamined 4.5 years…has shown a substantial loss of lung function attributable to the presence and degree of silicosis.&quot;</td>
<td>No additional exposures considered. Evaluated at smoking and age in data analysis. Data suggest spirometry and carbon monoxide diffusion decrease with time in workers with silicosis more than workers without silicosis.</td>
</tr>
<tr>
<td>Wang 2006</td>
<td>NA</td>
<td>1,884</td>
<td>Spirometry</td>
<td>None</td>
<td>Coal mine workers</td>
<td>&gt;10 years</td>
<td>FEV₁</td>
<td>Individuals with short-term declines found to be 3-18 times more likely to have long-term declines</td>
<td>&quot;Our findings provide guidance for interpreting periodic spirometry results from individuals exposed to respiratory hazards.&quot;</td>
<td>Not a true diagnostic study, no comparison test, no real diagnosis given.</td>
</tr>
<tr>
<td>Hankinson 1986</td>
<td>NA</td>
<td>NA</td>
<td>Spirometry</td>
<td>None</td>
<td>Healthy volunteers for normal values</td>
<td>None</td>
<td>FVC, FEV₁, FEV₁/FVC</td>
<td>None</td>
<td>&quot;This paper is a brief guide for those in the medical profession attempting to establish or improve their medical surveillance programs for occupational respiratory diseases.&quot;</td>
<td>Study is for background information, not comparing spirometry to any other diagnostic test. Had set of healthy volunteers to get &quot;normalized&quot; values.</td>
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</table>

See Hankinson 1986 for details
<table>
<thead>
<tr>
<th>Wang 2005</th>
<th>NA</th>
<th>449</th>
<th>Spirometry</th>
<th>Symptoms</th>
<th>Newly hired Chinese underground coal miners</th>
<th>3 years</th>
<th>FVC, FEV₁, FEV₁/FVC</th>
<th>FEV₁ slope averaged - 39ml/year in miners; 160ml/year in referents</th>
<th>&quot;Dust and smoking affect lung function in young, newly hired Chinese coal miners. FEV₁ change over the first three years of employment in non-linear.&quot;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beeckman 2001</td>
<td>NA</td>
<td>634</td>
<td>Spirometry</td>
<td>Symptoms, mortality, illnesses</td>
<td>Coal miners</td>
<td>18 years</td>
<td>FVC, FEV₁, Mortality Diagnosis</td>
<td>Higher proportion of coal miners with symptoms than coal miners without (p &lt;0.05). CS group had more symptoms of respiratory illness than RF group. CS had more deaths from cardiovascular disease or nonmalignant respirator disease.</td>
<td>&quot;The results of this study document the potential consequence of rapid declines in lung function, and emphasize the importance of recognition and effective interventions for individuals who experience accelerated losses of FEV₁.&quot;</td>
</tr>
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</table>

**CHEST RADIOGRAPHS**

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Score (0-11)</th>
<th>N</th>
<th>Test used</th>
<th>Comparison Test</th>
<th>Population</th>
<th>Length of Follow up</th>
<th>Outcome measures</th>
<th>Results</th>
<th>Conclusion</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attfield 1995</td>
<td>3.5</td>
<td>3,194</td>
<td>X-ray</td>
<td>Symptoms Employment status</td>
<td>Coal Miners</td>
<td>None</td>
<td>X-ray findings Employment status</td>
<td>53% were current miners, 47% ex-miners. (14% left for health reasons) CWP was 7-9%.</td>
<td>&quot;[T]he results described here indicate that the present coal mining work force is still at risk of developing CWP over a life time's work.&quot;</td>
<td>Included detailed occupational exposure history, and smoking status and dust exposure levels.</td>
</tr>
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</table>

**BRONCHIAL ALVEOLAR LAVAGE AND SPUTUM**

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Score (0-11)</th>
<th>N</th>
<th>Test Used</th>
<th>Comparison Test</th>
<th>Population</th>
<th>Length of Follow-up</th>
<th>Outcome Measures</th>
<th>Results</th>
<th>Conclusion</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Christman 1991</td>
<td>3.5</td>
<td>86</td>
<td>BAL</td>
<td>Symptoms</td>
<td>Granite workers Controls</td>
<td>Up to 5 years</td>
<td>Dust (silica) particles in BALF and in collected macrophages using polarized light microscopy. Control subjects averaged 4.35% of macrophages with particles. Granite workers had up to 50% with particles. The difference was significant (p&lt;0.0001)</td>
<td>&quot;With further understanding, BAL may become a more useful tool for the evaluation of workers with occupational exposure to dusty trades.&quot;</td>
<td>Participants not necessarily diagnosed with any specific disease. Other possible exposures not defined. Data suggest BALF may aid in detecting dust exposure in granite workers.</td>
<td></td>
</tr>
<tr>
<td>Dodson 1993</td>
<td>NA</td>
<td>5</td>
<td>BAL</td>
<td>None</td>
<td>Foundry workers</td>
<td>None</td>
<td>Ferruginous bodies (200x and 400x) Ferruginous bodies were seen by electron microscopy and light microscopy.</td>
<td>&quot;Our present study of lavage samples from foundry workers confirmed the presence of classical ferruginous bodies as reported in previous studies of tissue samples...&quot;</td>
<td>Small numbers, no comparison test.</td>
<td></td>
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</tbody>
</table>
### 6-MINUTE WALK TEST

| Author/Year | Score | N   | Test Used            | Comparison Test | Population                                      | Length of Follow-up | Outcome Measures                                                                 | Results                                                                 | Conclusion                                                                 | Comments                                                                 |
|-------------|-------|-----|----------------------|-----------------|-------------------------------------------------|---------------------|----------------------------------------------------------------------------------|--------------------------------------------------------------------------|--------------------------------------------------------------------------|
| Buch 2007   | 3.5   | 163 | 6-minute walk test   | Spirometry      | Patients with interstitial lung disease secondary to systemic sclerosis (SSc). | No follow-up.       | FVC, single breath diffusing capacity ($DL_{CO}$), Borg Dyspnoea Index            | No correlation found between 6MWT, pulmonary function, and Borg Dyspnea Index. | "The lack of criterion validity and the poor correlation with gas-exchange measurements raises important questions on the overall suitability of this test in SSc-ILD." | Data from a drug study. Patients with Systemic Sclerosis Interstitial Lung Disease. No normative values for age, gender, ethnicity used. Data suggest 6 minute walk test not effective predictor of dyspnea in these patients. |
| Chetta 2001 | 2.5   | 40  | 6-minute walk test   | Spirometry Body plethysmography, Carbon monoxide transfer capacity, Oximetry | Interstitial lung disease patients with history of breathlessness | None                | Walk Distance, Age, Breathlessness, FVC, SpO2                                     | Mean walk distance 487 m. 24/40 (60%) had >2% fall in oxygen saturation. | "Our study confirms that the 6MWT is a simple and inexpensive test that can provide a global evaluation of sub-maximal exercise capacity in ILD patients. Furthermore, we demonstrate that in these patients walk distance and oxygen desaturation during walk, but not breathlessness perception after walk, can be predicted by resting lung function." | Used second walk test to allow for learning effect. Different causes of ILD were included including sarcoidosis, idiopathic, etc. Patients had the disease from 1-19 years. No comparison diagnostic study included. Data suggest 6MWT may be used in ILD patients. |

### OTHER

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Score</th>
<th>N</th>
<th>Test Used</th>
<th>Comparison Test</th>
<th>Population</th>
<th>Length of Follow-up</th>
<th>Outcome Measures</th>
<th>Results</th>
<th>Conclusion</th>
<th>Comments</th>
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<tbody>
<tr>
<td>Gibbons 2001</td>
<td>NA</td>
<td>79</td>
<td>6-minute walk distance</td>
<td>Age Height Gender</td>
<td>Healthy participants to develop reference values for 6 minute walk distance.</td>
<td>None</td>
<td>6MWD</td>
<td>Best 6MWD average 698 meters. Distance inversely related to age (p &lt; .001). Distance directly related to height (p &lt; .001).</td>
<td>&quot;Selection of appropriate predicted 6MWD values for interpretation of performance should be guided by subject age and degree of test familiarization provided.&quot;</td>
<td>Distance used for test was 20 meters. This is different than ATS recommended 30 meters. Normative values needed based on age, height, and gender.</td>
</tr>
<tr>
<td>Reference</td>
<td>Use of 6MWD</td>
<td>6-Minute Walk Distance</td>
<td>Age, gender, height, weight, spirometry, Oxygen saturation, degree of dyspnea (Borg scale) Pulse rate.</td>
<td>Participants</td>
<td>Distance walked</td>
<td>Results</td>
<td>Notes</td>
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<td>Enright 1998</td>
<td>NA</td>
<td>6-minute walk distance</td>
<td>Age range 20-80 years. related to gender (p &lt;0.0002)</td>
<td>Healthy participants to develop reference values for 6 minute walk distance. Aged 40-80.</td>
<td>Men 576 m; women 494 m. Age, weight and height also influenced distance.</td>
<td>These reference equations may be used to compute the percentage predicted 6MWD for individual adult patients performing the test for the first time, using the standardized protocol.</td>
<td>Distance used for test was 100 feet. This is different than ATS recommended 30 meters. They excluded BMI &gt;35 kg/m2 and FEV1 &lt;70%. Reference values are valid only for first time performing the 6MWT.</td>
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<tr>
<td>Troosters 1999</td>
<td>NA</td>
<td>6- minute walk distance</td>
<td>Age, gender, height, weight</td>
<td>Healthy elderly volunteers.</td>
<td>Distance averaged 631 m. Males had 84m more than females on average (p &lt;0.001). There was a correlation with age and height (p &lt;0.01)</td>
<td>The six minute walking distance can be predicted adequately using a clinically useful model in healthy elderly subjects. Its variability is explained largely by age, sex, height and weight. Results of the six minute walking distance may be interpreted more adequately if expressed as a percentage of the predicted value.</td>
<td>Performed in 50m long hallway. Patients encouraged every 30 seconds. Study proposes a formula for normative values in 6MWD and states that a % of predicted is a more accurate result than absolute distance.</td>
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<td>Jenkins 2010</td>
<td>NA</td>
<td>Repeated 6-min walk distance maximum of 4 weeks after first.</td>
<td>Patients with COPD, interstitial lung disease (ILD), bronchiectasis and asthma before starting a pulmonary rehabilitation</td>
<td>None</td>
<td>6MWD increased in patients on second test. (p &lt;0.001) with at least 80% of patients in each cohort.</td>
<td>Respiratory diagnosis influences the magnitude of the learning effect for the 6MWT. The findings support the recommendation of a practice 6MWT at baseline assessment in order to provide an accurate measure of the effects of rehabilitation on 6MWD.</td>
<td>Retrospective study. Appears to be a learning effect for 6MWD after first test, but not after second.</td>
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<td>Garin 2009</td>
<td>NA</td>
<td>Mortality</td>
<td>Patients with scleroderma and or idiopathic pulmonary fibrosis</td>
<td>Uncertain</td>
<td>No significant difference between scleroderma patients and IPF. Lower extremity pain was primary limitation to walk distance for 15-20% of subjects.</td>
<td>Pain limitations confound the utility of the 6MWT, particularly in SSc patients without both ILD and PH… 6MWT distance is not always reflective of the same physiological process.</td>
<td>Retrospective record review. In patients with systemic scleroderma pain and other factors may limit walk distance more than dyspnea.</td>
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<tr>
<td>Baughman 2007</td>
<td>NA</td>
<td>6-minute walk distance</td>
<td>Spirometry, St. George Respiratory Questionnaire Fatigue assessment scale, Dyspnea score</td>
<td>Sarcoidosis patients, 130/142 (92%) had extrapulmonary manifestation</td>
<td>73/142 had distance &lt;400 m; 32/142 had distance &lt;300 m.</td>
<td>6MWD was reduced in the majority of sarcoidosis patients. Several factors were associated with a reduced 6MWD, including FVC, oxygen saturation with exercise, and self-reported respiratory health.</td>
<td>Participants were patients referred to tertiary sarcoidosis clinic. Most had extrapulmonary illnesses. FVC % predicted was 82% (17-</td>
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<td>151%) FEV₁, 76% (16-155%)</td>
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Appendix 4: References


