



# Thomas Pavillion March 15, 2000

10:30–11:15 Session 1: Collateral Ventilation: Lung Collapse and Air Trapping  
*Stanley B. Reich, MD*

Session 2: Lines, Stripes, Edges, Angles, and Triangles  
*Andrew D. Sherrick, MD*

Session 3: Neonatal Intensive Care Radiology  
*George W. Gross, MD*

Session 4: Thoracic Cavitory Disease  
*Humberto O. Martinez, MD*

11:15–12:00 Session 1: Intervention in the Chest: Drainage and Biopsy  
*Roderick J. H. Robertson, MD*

Session 2: Developmental Abnormalities of the Lung:  
Radiographic Findings in the Adult  
*Carl J. Zylak, MD*

Session 3: Pitfalls in Plain Film and CT Analysis of the Mediastinum  
*Paul L. Molina, MD*

Session 4: Concepts of the Accuracy of HRCT in Interstitial Lung Disease  
*James F. Gruden, MD*

12:00–1:30 Lunch

1:30–2:15 Session 1: Imaging Algorithms in Pulmonary Embolism\*  
*Warren B. Gefter, MD*

Session 2: Pulmonary Aspiration Syndromes  
*Robert H. Choplin, MD*

Session 3: Asbestos-Related Lung and Pleural Disease  
*Philip C. Goodman, MD*

Session 4: Interventional Angiography in Trauma to Chest\*  
*Jaime Tisnado, MD*

\*Abstract not available at time of publication.

Wednesday, cont.



# Collateral Ventilation: Lung Collapse and Air Trapping

Stanley B. Reich, MD, FACR  
University of California Davis

**Objective of Workshop:** To correlate the physiology of collateral ventilation with cough, obstructive and non-obstructive collapse, air trapping, and emphysema – and their X-ray manifestations.

J. Kohn first described the interalveolar pores in 1893. In 1931 Van Allen and Lindskog evaluated the physiology of collateral respiration and related it to localized air trapping. In 1949 Churchill described the phenomenon of air trapping and indicated that it was a way of maintaining the spacial integrity of the lungs.

My odyssey with Drs. Culiner and Abouav started with a case of congenital obstruction of an upper lobe bronchus with air trapped behind the occluded bronchus.

Numerous experiments in various animals showed that collateral ventilation was always present in air breathing animals. Results also showed that air pressure of 30-40 mm of Hg (found in cough, sneeze and strain) would push air through the pores of Kohn into the adjacent alveoli where it would remain trapped at 30-40 mm Hg pressure – due to some kind of flap mechanism.

After a while, I realized that this was the mechanism of cough – for without it air could not go beyond an offending particle and expel it. However, this is hypoxic ventilation causing anoxic vasoconstriction in the areas of collateral ventilation.

In obstructive collapse the continuous secretion of mucous fills the alveoli and causes consolidation collapse – exceptions are allergic bronchopulmonary aspergillosis, congenital obstruction, and some cases associated with chronic bronchitis and COPD.

Non-obstructive collapse occurs as a result of: (1) Failure to cough due to pain and obtundation eg, post-operative segmental and subsegmental collapse. (2) Failure of inspiration and cough due to loss of chest wall and diaphragmatic motion. (3) Failure due to trapping from pleural disease. (4) Failures due to complete interlobar septa e.g., right middle lobe syndrome.

Air trapping – COPD is caused by (1) partial occlusion of bronchus allowing influx but not efflux of air. (2) Emphysema with air sac wall destruction. The lack of elastic tissue makes this area more compliant. (3) Collateral ventilation – positive pressure trapping because of bronchiolar obstruction at the 2-3 mm. bronchial diameter level (beyond the bronchial glands). This area is less compliant. (4) Combination of 2 and 3 – destruction and obstruction forming blebs.

These forms of air trapping coexist in smokers – destruction of air sac walls and obliterative bronchiolitis and affect pulmonary function tests in different ways.

Correlation with ventilation perfusion scans shows that areas of emphysema (alveolar wall destruction) lack perfusion, and have rapid filling of the destroyed area on inhalation with slow clearing.

With obliterative bronchiolitis there is little perfusion, slow wash in ventilation and very slow wash out.

Correlation with HRCT in emphysema shows enlarged air spaces without obvious walls on inspiratory films and slight diminution of these air spaces on expiration compared to marked emptying of normal lung.

HRCT in obliterative bronchiolitis shows relatively normal or slightly increased size of the area with inspiration but diminished perfusion as a result of hypoxic vasoconstriction. On expiration the involved areas maintain their size with marked emptying of normal lung.

The challenge will be to quantitative these different phenomena.

Illustrative cases of congenital obstruction with emphysema; usual obstructive collapse; examples of non-obstructive collapse (including right middle lobe syndrome); air trapping including Swyer-James; progressive massive fibrosis; allergic bronchopulmonary aspergillosis – as well as animal experiments demonstrating these phenomena will be presented.

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# Lines, Stripes, Edges, Angles, and Triangles

Andrew D. Sherrick, MD

The objective of this workshop is to help the participants gain a firm understanding of basic anatomic landmarks seen on frontal and lateral chest radiographs. A firm knowledge of basic Chest X-Ray (CXR) anatomy is important for accurate understanding and interpretation of chest images. This presentation will include discussion of mediastinal contours on CXR with CT correlation. In addition, the anatomy of some anatomic angles and triangles within the chest will be reviewed. At the end of this workshop the participants should have a basic understanding of the lines, stripes, and edges seen on a CXR and should be able to use this knowledge to aid them in detecting and diagnosing diseases of the chest.

**Paratracheal Stripe** – Normally the right side of the trachea is separated from the lung by a thin layer of fat forming the right paratracheal stripe; this is seen in the majority of patients and is usually 3-4mm in thickness. In up to half of patients the posterior tracheal wall is outlined by lung as it interposes between the spine and trachea forming the posterior tracheal stripe [1]. This stripe is typically less than 5mm in thickness on a lateral CXR. The esophagus may be situated in this region, in which case, the wall of the esophagus may contribute to this stripe (tracheoesophageal stripe). The left brachiocephalic vein can occasionally be seen as a convexity superiorly.

**Superior Pleuroesophageal Line or Stripe** – If there is air in the upper esophagus, the interface between the right lung and the right wall of the esophagus can be seen on a PA CXR as a shallow S extending from the lung apex to the azygos arch. This is usually a stripe measuring 3-5mm in thickness [10]. The left wall of the upper esophagus can occasionally be seen.

**Inferior Pleuroesophageal Line or Stripe** – If gas is present in the lower esophagus the right wall of the esophagus and the adjacent mediastinal fat and pleura may form a stripe where they come in contact with the right lung, this is termed the right inferior Pleuroesophageal stripe. This situation occurs less often on the left.

**Anterior Junction Line** – As the two lungs approximate anteriorly they are separated by four layers of pleura and an inconsistent amount of mediastinal fat. Since the septum dividing the two lungs is vari-

able in thickness this can form either a line or a stripe. On a PA CXR this can be seen oriented obliquely from upper right to lower left, the superior extent diverges to fade out as it reaches the clavicles [2]. The terms superior and inferior recesses have been used to describe the lung interfaces above and below the anterior junction region [9].

**Posterior Junction Line** – In some patients the lungs come in close contact posterior to the esophagus forming the posterior junction line. This line begins at the root of the neck well above the level of the clavicles and diverges inferiorly to encompass the aortic arch [6].

**Vascular Pedicle** – On a PA CXR the vascular pedicle extends from the top of the heart to the thoracic inlet. Its right border is formed by the right innominate vein and SVC, its left border by the left subclavian artery. In one study the width of the vascular pedicle in normal patients was 48mm (+/- 5mm)[4].

**Azygos Arch** – The azygos vein originates below the diaphragm and ascends through the aortic hiatus and then usually in front of or to the right of the thoracic vertebral bodies. It is joined at the T-8 or T-9 level by the hemiazygos vein. It passes anteriorly over the right mainstem bronchus and truncus anterior to empty into the posterior aspect of the SVC.

**Great Vessels** – The left subclavian artery arises as the third of the great vessels from the aortic arch. On a PA CXR it passes upward and lateral to the trachea in contact with the left mediastinal pleura forming an interface with the left upper lobe extending from the aortic arch to about the level of the medial end of the clavicle. On a lateral CXR, the posterior margin of the vessel may be seen through the posterior portion of the trachea coursing obliquely upward toward the neck.

**Left Superior Intercostal Vein** – This vein forms an arch on the left that is analogous to the azygos arch on the right. This vein drains the second, third, and fourth posterior intercostal veins, then passes forward and upward along the aortic arch to drain into the left brachiocephalic vein. About 75% of the time the vein connects to the accessory hemiazygos vein [7]. The aortic nipple is seen on a PA CXR in about 1% of normal subjects, however in one study



of patients with fibrosing mediastinitis it was seen in almost 50% of patients with SVC syndrome, due to dilatation of this vein from collateral flow [8].

**Aortopulmonic Window** – The AP window is located under the aortic arch and above the left pulmonary artery. It is bounded medially by the trachea and esophagus and laterally by the lung. The ligamentum arteriosum and the recurrent laryngeal nerve traverse this space.

**Azygoesophageal Recess** – Below the aortic arch the right lung makes contact with the mediastinal surface containing the esophagus and the azygos vein, this portion of lung is known as the azygoesophageal recess, and the interface is known as the azygoesophageal line.

**Preaortic Recess** – A portion of the left lower lobe known as the Preaortic recess is situated anterior to the descending aorta and posterior to the wall of the left main stem bronchus.

**Left Paraspinal Stripe or Line** – The left paraspinal line or stripe is seen in about 40% of PA CXRs, it extends from the region of the aortic arch inferiorly to about the T9-T12 level. It parallels the vertebral bodies and usually lies about half way between the left side of the descending aorta and the spine. As the aorta comes closer to the diaphragmatic hiatus, the aorta and the paraspinal line can overlap. Some believe that the paraspinal lines are Mach bands or edge-enhancing phenomena created by the retina in response to strong differences in transmitted illumination [6].

**Right Paraspinal Line** – The right paraspinal line is seen less often than the left. When seen it is usually thin and extends from T8-T12 regions.

**Cardiophrenic Angles** – On a PA CXR the cardiophrenic angles are formed by the borders where the heart and diaphragm meet. Many abnormalities can occur in this space, including pericardial cysts, prominent pleuropericardial fat, foramen of Morgagni hernias, lung masses, pleural masses and more.

**Costophrenic Angles** – The costophrenic angles are formed by the borders where the diaphragm meets the chest wall. Abnormalities in this region include pleural fluid, pleural masses, diaphragm masses, lung masses, foramen of Bochdalek hernias and more.

**Retrosternal Stripe** – On the lateral CXR the two lungs are in close relation to the back of the sternum. Some mediastinal fat may be interposed forming a vertical stripe [5].

**Retrosternal Clear Space** – The radiolucent lung behind the sternal body and in front of the ascending

aorta accounts for the well-recognized retrosternal clear space. This space is frequently not “clear” and when it is clear the depth of this space in normal patients is variable (18mm-45mm) [5].

**Cardiac Incisura** – On the lateral CXR the left lung is excluded from the anterior medial aspect of the inferior chest wall by the cardiac apex or the epicardial fat pad. This deficiency is termed the cardiac incisura. A similar appearance can occur on the right due to the presence of a fat pad [2].

**Retrotracheal Triangle** – On a lateral CXR the retrotracheal triangle is bordered anteriorly by the dorsal wall of the trachea, posteriorly by the upper thoracic vertebrae, and inferiorly by the aortic arch. A variety of abnormalities can be found in this space, including vascular abnormalities, esophageal abnormalities, thyroid masses, bronchogenic cysts and more [3].

**Posterior Wall of the Bronchus Intermedius** – The posterior wall of the bronchus intermedius is visible on the majority of well-performed lateral radiographs of the chest. On a well-positioned film it is usually seen projected over the bronchus to the left upper lobe. In the work by Proto et al. the posterior wall of the bronchus intermedius had a mean measurement of 1.3mm and a range of .5–3.0mm [5].

**IVC Shadow** – In most patients the posterior wall of the IVC can be seen as it enters the right atrium, on a lateral view.

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# Neonatal Intensive Care Radiology

*George W. Gross, MD*

This workshop will include discussion of the following topics with examples of the more common and/or important problems and issues:

1. Technical aspects of neonatal chest imaging, including equipment and projections
2. Radiation exposure and protection
3. Catheters, tubes, and monitors employed in the NICU
4. Normal appearances and normal variants
5. Approach to identifying and evaluating what is really important on the chest radiograph
6. Differentiating “medical” from “surgical” pulmonary disorders

7. Findings suggestive of congenital heart disease
8. Newer support techniques used in the NICU
9. Use of ultrasonography of the thorax in the NICU

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# Thoracic Cavitory Disease

Humberto O. Martinez, MD

The AIDS epidemic has already introduced us to opportunistic infectious organisms, such as *Pneumocystis carinii*, which has become a prevalent cause for pulmonary cavities. As we enter the new millennium, we will be challenged with the diagnoses of new causes of cavitory thoracic disease. Therefore, it is not only important to be familiar with the differential diagnoses of thoracic lucent defects, but also the role of plain film imaging of the chest and cross-sectional imaging particularly as it pertains to the distinction of parenchymal lesions from those caused by pleural or extra-pleural disease.

A true cavity is a gas filled space resulting from necrosis of lung parenchyma within a zone of consolidation, a nodule, or a mass, with liquefaction and evacuation of contents via the tracheo-bronchial tree. A true cavity can be differentiated from a pulmonary air cyst in that the later usually presents with well-defined walls no greater than 2 mm thick and larger than 1 cm in diameter. Radiographically, pulmonary cavities can present as single or multiple lesions varying in size and wall thickness and may or may not be associated with surrounding parenchymal infiltration. The most useful criterion in differentiating neoplastic from non-neoplastic cavitory lung disease is cavity wall thickness (measured at the thickest point). Other criteria useful in narrowing the differential diagnosis of cavitory lung disease include the presence of single or multiple lesions, as well as the presence of a surrounding infiltrate.

Many entities can give rise to cavitory lesions in the lung. These may be solitary or multiple and include infectious, inflammatory, and neoplastic etiologies. Often the most important question the Radiologist can answer is whether the lesion is neoplastic or non-neoplastic. Several studies have previously reported radiographic criteria to help differentiate benign from malignant lesions. Cavities with wall thickness greater than 15 mm were malignant in 95% of the cases, whereas those 4 mm. or less were benign in greater than 92% of the cases. All cavities with wall thickness of 1 mm. or less were benign. Those having wall thickness between 5 and 15 mm. had a 50% chance of being benign or malignant.

Other entities can present as lucent defects in the thorax which mimic cavitory lung disease. Etiologies include pleural disease such as empyema and localized pneumothorax. Post surgical entities such as

post-pneumonectomy space, plombage thoracoplasty, gastric pull-up, post traumatic causes such as traumatic rupture of diaphragm with herniating bowel in to the chest; and congenital causes such as diaphragmatic hernias.

The other radiographic criteria useful in narrowing the differential diagnosis of cavitory lung disease analyze the presence of single or multiple cavitory lesions. The presence of a single thick-walled cavitory mass usually implies malignancy, whereas the presence of multiple thin-walled cavities usually implies benign disease such as an inflammatory or infectious process.

The following table lists several causes of lucent defects in the thorax:

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## Congenital and Neonatal Diseases

- Bronchopulmonary dysplasia
- Wilson-Mikity syndrome
- Cystic adenomatoid malformation
- Pulmonary sequestration

## Airways Diseases

- Bullous emphysema
- Cystic bronchiectasis

## Infectious Diseases

- Tuberculosis and atypical mycobacterial infections
- Fungal infections: coccidioidomycosis, cryptococcosis, aspergillosis, blastomycosis,
- Staphylococcal pneumonia causing pneumatoceles
- Gram-negative pneumonias causing pneumatoceles
- Parasitic diseases: echinococcosis
- Opportunistic Infections: *Pneumocystis carinii*
- Others: nocardiosis

## Embolic Diseases

- Pulmonary thromboembolism
- Septic embolism

## Neoplastic Diseases

- Hematogenous metastases
- Pulmonary spread of laryngeal papillomatosis
- Hodgkin's disease and non-Hodgkin's Lymphoma
- Primary lung tumor (especially squamous cell)

## Autoimmune Diseases

- Rheumatoid necrobiotic nodules
- Wegener's granulomatosis
- Polyarteritis nodosa (very rare)

## Diseases of Unknown Origin

- Sarcoidosis
- Histiocytosis X

## Trauma

- Hydrocarbon ingestion with pneumatocele formation
- Traumatic lung cysts (pulmonary laceration)

## Conditions Mimicking Cavities

- Herniation of bowel into the thorax
  - Air-containing empyema
  - Lucite plombage
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# Intervention in the Chest: Drainage and Biopsy

*Roderick J. H. Robertson, MD*

The objectives of this workshop will be to discuss the indications and contra indications, choice of imaging, needle and drain type, analysis of results and the discussion of the complications and their treatment for mediastinal, pulmonary and pleural disease.

## Indications

The indications for biopsy are largely self evident. In our unit, lesions that appear likely to be lung cancer and have been staged as operable do not have a biopsy. Our main group for biopsy are those who are considered inoperable for whom tissue is acquired for further management whether this is mediastinal, pulmonary or pleural. The indication for radiological drainage in the mediastinum is usually infection, and this is often the case in the pleura too. Lung abscesses are drained if they are more than 4cm in size in an ill patient who is continuing to deteriorate despite good medical treatment.

## Contra Indications

Coagulation disorders are a contra indication to biopsy although fresh frozen plasma or platelets can be given if necessary. Such cover is particularly necessary in infections that have caused disseminated intra vascular coagulopathy which will not improve until the infection is treated. A relative contra indication is poor respiratory reserve. This group is one that needs particular assessment as the largest group for lung biopsy are those who have been turned down for surgery because of their lung function. Intractable coughing and confusion are also relative contra indications as they increase the complication rate.

## Choice of Imaging

For many, the imaging technique of choice used to be fluoroscopy, and this still has its place in some intra pulmonary lesions. A fluoroscopic biopsy has its disadvantages in that small lesions can be difficult to localise in both projections, and in larger lesions the needle may be placed into adjacent consolidation rather than the actual mass. CT, particularly fluoroscopic CT biopsy, is the preferred option. Slice thickness will depend on the size of the lesion, 10mm being satisfactory in larger lesions, but 3's or 5's may be necessary for small lesions. Larger masses that

abut the chest wall whether pleural, pulmonary or mediastinal can be done most easily using ultrasound guidance, but most biopsies are performed under CT guidance.

For drainage, ultrasound is the best technique for pleural collections. This shows septations more clearly than CT and allows the most effective choice for location of drains. CT is reserved for cases where drainage has initially failed, partly to ensure that a loculated collection has not been missed, and also to check that the end of the drain is not in a fissure or other mal-position. For both mediastinal and lung abscess drainage, CT guidance is preferred.

In lung abscess drainage, it is recommended that the patient is positioned so that the abscess is in the dependent lung. These abscesses are often under pressure with a ball valve effect, as on initial drainage, the patient may start to produce pus into the airways so if there is not a good cough reflex, may aspirate some of this into the other lung. This is less likely if the abscess is dependent.

## Needle/Drain Type

For biopsy, close correlation with the pathology department is always necessary. In our own department, trucut-core biopsies are obtained on all pleural and mediastinal masses. For both pleura and mediastinum, a needle that can be imaged with its inner stilette fully extended is preferred. This allows the operator to know that the core obtained will be from the particular part of the lesion required to be biopsied and also is reassuring in that the maximal penetration of the needle is already known. For lung biopsies, cytology is a preferred first option except in patients where there is a prior probability of a benign diagnosis such as in patients with rheumatoid disease and non smokers. In our department, cytology technicians are present at the time of biopsy. This allows the number of passes to be as small as possible, and also a core biopsy can be obtained if the cytology specimen appears unhelpful.

For drainage, size is not said to matter. Nevertheless, an 8 French drain is really only useful in patients with transudates or uncomplicated malignant effusions. With infection, whether mediastinal, intra pulmonary or pleural, 12 - 14 French drains are preferred



mainly because of the size of the drainage holes within the catheter. Streptokinase or other fibrinolytic treatment of pleural infection has gained widespread acceptance although the series have mixed results and the numbers overall are still small. Such treatment is restricted to pleural disease, and has been used with effect in malignant disease as well as in infection. The small numbers in the series is also reflected in the different regimes for administering streptokinase, usually 250,000i.u., but sometimes with 20mls saline, sometimes 100mls saline with dwell times of between half an hour and four hours, with either one administration per day or in some series, two.

In positioning patients for biopsy, the prone position is often favoured, but there are advantages in having the patient on their side with the affected lung dependent. This reduces pulmonary movement. This can allow a better approach to the lesion too. With mediastinal or pleural lesions, the space can be widened with initial injection of local anaesthetic and saline to widen the extra pleural space.

### Analysis of Results

There are several published series with established rates of diagnostic accuracy of over 90% for large pulmonary nodules (greater than 1.5cm). For smaller nodules, there is a variation in diagnostic accuracy from 74% to over 90%. Regular analysis of results is required. The regular attendance of cytology technicians for lung biopsies has both improved the diagnostic rate, but as importantly, has reduced the number of passes. The accuracy rate for both pleural and mediastinal biopsy is less well established and much depends on case selection. Similarly, analysis of effective drainage is complicated by case selection and the small numbers and quite large differences in results from published series.

### Complications

For biopsy, the commonest complication is pneumothorax with rates of up to 40% being reported. 20-30% pneumothorax are commonly reported figures. Initial reports suggested only 1% of patients require aspiration or drain placement for pneumothorax, but more recent reports have suggested rates of 5%. This may be, in part, due to a change in patient selection as smaller nodules are biopsied, but also more particularly in patients who are turned down for surgery in whom a smaller pneumothorax would require intervention. Haemoptysis rates depend on whether a fine needle or core biopsy is obtained. Other complications such as air embolism and tumour spread need to be remembered. Tumour spread is particularly important in biopsy of mesothelioma and pleural sarcoma.

In drainage of lung abscesses, aspiration of the abscess fluid into other parts of the lung after initial drainage has already been discussed. Haemoptysis and haemothorax are rare complications. Similarly rare is conversion of a lung abscess into a broncho-pleural fistula or a co-existent empyema. This latter occurrence has only occurred in one of our cases which was due to actinomycosis.

### Conclusion

There are few lesions or collections within the chest that cannot be safely biopsied or drained using imaging techniques. As imaging evolves, with more sophisticated CT evaluation as well as positron emission tomography and as surgical techniques change, including VATS, there will be a change in the type of patient referred. Nevertheless, while radiology results remain so good, image guided biopsy and drainage will remain a fundamental part of patient management.

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# Developmental Abnormalities of the Lung: Radiographic Findings in the Adult

Carl J. Zylak, MD, FRCPC, FACR

## Learning Objectives

1. Embryology of lung development
2. Imaging characteristics of the common anomalies encountered in the adult
3. Ability to differentiate from more sinister abnormalities.

Development anomalies of the lung are usually detected in the neonatal period and in early childhood. However, some are not encountered until later childhood or adulthood. As some of these anomalies can be confused with more sinister abnormalities an understanding of their imaging features will facilitate this distinction.

Anomalies encountered in adulthood can be classified into three broad categories. The first category represent lung bud anomalies, the second involves a combination of lung and vascular abnormalities and the third category relates to purely vascular anomalies. This presentation will highlight the common anomalies encountered in each of these categories.

## Lung Development

The first indication of normal development of the lungs appears in embryos at ovulation age 24 days. At 28 days, the right and left lung buds make their appearance from the ventral wall of the foregut. As the lung buds elongate, the respiratory portion of the gut is separated from the esophageal portion to form a tracheoesophageal septum. By day 30-32, the five lobar bronchi make their appearance. They then elongate and begin to branch. At 34-36 days, all the segmental bronchi are present, subsegmental buds then begin to appear and by days 38-40, all subsegmental bronchi are represented and many are undergoing further subdivision.

Lung bud anomalies include agenesis, aplasia and hypoplasia, bronchial atresia, lobar emphysema, cystic adenomatoid malformation and congenital bronchiectasis.

The opportunity for vascular anomalies relate to arteries originating from the thoracic aorta below the hilus of each lung and pulmonary arteries which may arise atypically from derivatives of embryonic aortic

arches. Abnormalities pertaining to the veins relate to their embryologic proximity to the splanchnic plexus which surrounds the developing lung buds, trachea and esophagus. This can result in anomalous venous drainage to commonly the superior vena cava, the right atrium and the left brachiocephalic vein. Other sites of drainage include the coronary sinus, inferior vena cava, azygos vein, and the portal vein.

This separation into bronchial and vascular anomalies is somewhat artificial as several of these anomalies demonstrate abnormalities of both the airway and vessels.

Vascular anomalies encountered therefore can relate to anomalous venous return, congenital aneurysm of the pulmonary arteries and combined venous and arterial abnormalities as seen in arteriovenous malformations.

Combined bronchial and vascular anomalies that may be encountered in the adult include the hypogenetic syndrome and bronchopulmonary sequestration, intralobar and extralobar.

## Imaging Features

### Lung Bud Anomalies

#### *Agenesis*

Agenesis of an entire lung will simulate the features following total pneumonectomy. The remaining lung is over inflated with accompanying shift of the mediastinum. Agenesis of a lobe may have quite a normal appearance on the chest radiograph or sometimes be confused with collapse of a lobe. Perfusion studies, be they nuclear scans, angiograms or CT, can readily establish these diagnoses.

#### *Bronchial Atresia*

The common sites of involvement in congenital bronchial atresia are the apical posterior segmental bronchus of the left upper lobe, segmental bronchi of the right upper lobe, middle lobe and occasionally the lower lobe. The radiographic features include the presence of a hilar mass with evidence of peripheral air trapping. The dilated bronchi contain retained secretions and can take a varied appearance. The branches may simulate horns as they often taper



peripherally. Occasionally, the dilated bronchi are purely air-filled and present as lucencies outlined by thin walls again taking interesting shapes such as hairpins, etc. Computed tomographic features are characteristic. The supplying bronchus is atretic. The dilated bronchi are either completely opaque or will demonstrate air fluid levels or occasionally be purely air filled. The adjacent peripheral lung is over inflated secondary to entrapment of air via collateral air drift. These patients are usually asymptomatic and do not require surgery. The major indication for surgery is repeated infections.

#### ***Congenital Lobar Emphysema***

Congenital lobar emphysema or neonatal lobar hyperinflation, is usually diagnosed in the neonatal period or infancy. However, occasionally the condition may be first encountered in the adult. In this latter scenario, particularly if the patient presents with a history of a recent stab wound to the thorax, the condition may be confused with a pneumothorax. Air trapping however is usually evident and there is often contralateral shift of the mediastinum. The presence of vessels within the over inflated lobe may be difficult to detect and hence the differentiation from pneumothorax may be exceedingly difficult.

#### ***Congenital Cystic Adenomatoid Malformation***

Congenital cystic adenomatoid malformation of the lung is an uncommon developmental anomaly. Pathologically, the lesion consists of adenomatoid proliferation of bronchioles that form cysts instead of normal alveoli. Three types have been classified pathologically. Type 1 consists of cysts between 2-10cm, Type 2 has numerous smaller, more uniform cysts measuring .5-2cm in diameter and Type 3 are solid appearing lesions which microscopically demonstrate tiny cysts. This disorder is usually discovered in neonates because of respiratory distress and may occasionally be discovered in older children and adults because of repeated infections. In the adult, the lower lobes are usually involved, with expansion of the involved hemithorax and compensatory shift of the mediastinum. The cysts may be single or multiple, air containing, fluid containing or contain both air and liquid.

#### ***Pulmonary Bronchogenic Cysts***

Pulmonary bronchogenic cysts are thought to result from a part of the tracheobronchial tree that has separated from the normal airways during the developmental branching process. Radiologically, the cyst, if fluid filled, will present as a well circumscribed nodule. If it becomes infected it can empty and present as an air filled space or as a mixture of air and liquid. The common location is the lower lobes.

#### ***Congenital Bronchiectasis***

Congenital bronchiectasis is postulated to be sec-

ondary to incomplete branching of the developing bronchial tree, a lobe or the entire lung can be involved. The lung is usually otherwise normal.

### **Combined Bronchial and Vascular Anomalies**

#### ***Hypogenetic Lung Syndrome***

The hypogenetic lung syndrome consists of abnormal development of the lung almost always on the right. The lung is small associated with a small pulmonary artery, anomalous venous return usually to the inferior vena cava and systemic arterial supply. Radiographic findings include a small lung with ipsilateral mediastinal shift. The retrosternal band of increased density on the lateral view, originally thought to represent deposition of adipose tissue, actually results from shift of the mediastinum and resultant decrease in the AP diameter of the lung. The anomalous draining vein usually connects with the inferior vena cava forming a familiar curvilinear density having the appearance of a sword, hence the appellation of the scimitar syndrome. CT and angiography confirm the presence of decreased volume of the lung, the anomalous draining vein and perfusion from systemic arteries originating from the descending thoracic or upper abdominal aorta. The affected lung is small demonstrating diminished vascularity.

#### ***Bronchopulmonary Sequestration***

Bronchopulmonary sequestration, intralobar and extralobar, represent an anomaly of tracheobronchial branching and retention of its embryonic systemic vascular supply. Its blood supply is from a systemic artery. Intralobar sequestrations reside within the normal lung parenchyma where as extralobar sequestrations have their own pleural enclosure within or below the diaphragm.

Radiographically, intralobar sequestrations can present as either an area of increased density simulating a pneumonia or as an air fluid collection situated usually in the lower lobes more commonly on the left. Occasionally the supplying artery can be demonstrated on plain films and is often seen on CT examination as a vessel originating from the aorta. With repeated infections, communication with the bronchial may develop. On CT examination, the adjacent lung is also abnormal consisting of over inflation likely secondary to air trapping rather than emphysema.

Extralobar sequestration is much less common also usually on the left related to the hemidiaphragm. Drainage is usually via the systemic venous system whereas with intralobar sequestration the drainage is to the pulmonary circulation. It is completely enclosed in a pleural sac. Radiographically, extralobar sequestration may present as a reasonably well defined mass at the base of the left hemithorax.



## Vascular

### *Total Anomalous Venous Return*

Total anomalous venous return is rare. A defect at the atrial level or communication between the aorta and pulmonary artery are necessary requirements for survival. The characteristic plain film feature is the familiar figure of eight or snowman.

### *Partial Anomalous Venous Return*

Partial anomalous venous drainage is an incidental finding in asymptomatic patients. The common site of involvement is anomalous drainage of the left upper lobe to the left brachiocephalic vein. This abnormality can be demonstrated on CT. The anomalous vein courses adjacent to the aorta and ascends to join the brachiocephalic vein.

### *Pulmonary Artery Aneurysm*

Aneurysms of the pulmonary arteries are rare anomalies. They are often associated with congenital cardiac defects. However, occasionally they can be encountered as an isolated finding in the adult.

### *Pulmonary Arteriovenous Malformation*

Pulmonary arteriovenous malformation refers to communications between pulmonary arteries and pulmonary veins. Radiographically they present as nodules on the conventional chest radiograph. The supplying artery and draining vein can sometimes be identified. On CT this arrangement is readily demonstrated. They may be single or multiple. They are

more commonly encountered in the lower lobes. Definitive evaluation consists of angiography or CT.

## Summary

In summary, this overview has highlighted the characteristic imaging features of developmental anomalies encountered in the adult population. Their characteristics may aid in the differentiation from more sinister abnormalities.

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# Pitfalls in Plain Film and CT Analysis of the Mediastinum

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## Introduction

Pitfalls in plain film and CT analysis of the mediastinum may be broadly categorized into vascular and non-vascular pitfalls. This discussion concentrates on some of the more common mediastinal vascular pitfalls. Both normal and aberrant vascular structures can be misinterpreted as representing enlarged mediastinal lymph nodes or other masses. Avoidance of such errors requires thorough knowledge of normal mediastinal anatomy and its common variations, careful review of multiple, contiguous CT sections, and the proper use of intravenous contrast material.

## Tortuous Innominate Artery

Tortuosity or ectasia of the innominate artery, particularly in elderly patients with atherosclerotic vascular disease, can occasionally be mistaken for paratracheal adenopathy or mass. Careful evaluation of sequential CT images above the aortic arch usually allows a correct diagnosis to be made. Enhancement of the tortuous vessel following administration of intravenous contrast material provides more definitive evidence when needed.

## Aberrant Right Subclavian Artery

An aberrant right subclavian artery (ARSA) originating from an otherwise normal left-sided aortic arch is the most common congenital mediastinal arterial anomaly, occurring in approximately 1% - 2% of the population. This anomalous vessel arises as the last branch of the aortic arch rather than from the innominate artery, and occurs as a result of interruption between the right common carotid artery and the ARSA in the developing double aortic arch. Its origin is frequently widened as the Kommerell diverticulum, representing the remnant of the distal right aortic arch.

Manifestations of ARSA on posteroanterior chest radiographs include an oblique edge extending to the right from the aortic knob, a tubular opacity or vessel projecting through the tracheal air column, and right paratracheal mass effect. On lateral radiographs, one

may see a retrotracheal opacity, anterior deviation of the trachea, and obscuration of the anterior portion of the aortic arch. On CT, the ARSA can be seen arising from the posteromedial portion of the aortic arch and crossing the mediastinum obliquely from left to right behind the esophagus. Often, the origin of the aberrant vessel is dilated, and the ipsilateral "innominate artery" appears smaller than normal.

## Left Pulmonary Artery

Partial volume averaging of the top of the left pulmonary artery may be mistaken for aortopulmonary window lymphadenopathy. The orientation of the artery imaged within the aortopulmonary window should be the same as that of the readily identified left pulmonary artery on the next more caudal scan. Adenopathy generally is oriented differently and has a less homogeneous appearance than the left pulmonary artery.

In some patients, the top of the left and main pulmonary arteries is located more cephalad than usual, at a level alongside the aortic arch, resulting in confusion with an anterior mediastinal mass or lymphadenopathy. Review of sequential CT images will show that the apparent "mass" is contiguous with the main pulmonary artery anteriorly, and with the left pulmonary artery posteriorly as it approaches the hilar area.

## Aortic Aneurysm and Dissection

Aneurysms arising from the aortic arch can simulate anterior mediastinal or lateral aortic lymphadenopathy. The presence of calcification in the wall of the mass and enhancement after intravenous contrast administration help confirm the diagnosis of an aneurysm. Although the diagnosis is generally straightforward, the appearance may be confusing when the aneurysm presents in an unusual location, when the lumen of the aneurysm is small with respect to the mural thrombus, or when intravenous contrast administration is suboptimal. Occasionally, enhancing mediastinal masses such as intrathoracic goiters may superficially mimic the appearance of an aneurysm.



The CT diagnosis of aortic dissection rests on identifying an intimal flap or a thrombosed false lumen surrounding the true lumen. However, several pitfalls exist that may simulate an intimal flap or false channel. These include streak artifacts, fusiform atherosclerotic aneurysms with intraluminal thrombus, partial volume averaging of calcification in the wall of an ectatic aorta mimicking displacement into the lumen, and improper identification of adjacent structures such as the pericardial recesses, left brachiocephalic vein, left superior intercostal vein, and enhancing atelectatic lung. Another cause of false-positive diagnosis of aortic dissection is artifact of the proximal ascending aorta arising from motion of the aortic wall during rapid image acquisition ( $\leq 1$  sec). Such artifacts, or apparent "intimal flaps", typically occur in the aortic root and are confined to one or two contiguous slices. The findings do not extend into the aortic arch or descending aorta and no associated mediastinal or pericardial hemorrhage is identified.

### Left Brachiocephalic Vein

The left brachiocephalic vein, usually larger than the adjacent arteries, can occasionally simulate a mediastinal mass or adenopathy, particularly when it has a tortuous, vertical, or anomalous course through the mediastinum. Administration of intravenous contrast material will result in opacification of the vein and separation from enlarged lymph nodes.

### Superior Intercostal Veins

The left superior intercostal vein, which courses anteriorly alongside the aortic arch to empty into the left brachiocephalic vein, may simulate lateral aortic lymph node enlargement or aortic dissection.

The right superior intercostal vein, particularly when distended, may mimic an enlarged posterior mediastinal lymph node. Sequential CT scans from cephalad to caudad will show the right superior intercostal vein coursing inferiorly adjacent to the spine to join the posterior aspect of the azygous arch.

### Persistent Left Superior Vena Cava

A persistent left superior vena cava, the most common anatomic variant of systemic venous return, occurs in 0.3% of the normal population. This vessel courses along the left side of the superior mediastinum and extends caudally lateral to the aortic arch and anterior to the left hilum before entering into the coronary sinus. In approximately 85% of cases a right superior vena cava is also present.

### Azygous Vein

When portions of a tortuous azygous vein arch from posterior to anterior are imaged on sequential transverse slices, retrobronchial, paratracheal, and pretracheal lymphadenopathy may be simulated. Review of serial CT scans and, if necessary, administration of intravenous contrast medium, will demonstrate a tortuous azygous arch extending forward from a prevertebral location to empty into the superior vena cava.

The azygous arch, ascending azygous vein and superior vena cava are enlarged in patients with azygous continuation of the inferior vena cava because of increased blood flow. In such patients, the dilated azygous venous system may simulate adenopathy anywhere along its course. Similar findings can be seen in patients with an obstructed or thrombosed superior vena cava resulting in dilatation of the azygous system.

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# Concepts of the Accuracy of HRCT in Interstitial Lung Disease

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## Introduction

The technique of thin section, or high-resolution, CT (HRCT) was first described in 1982 by Todo and colleagues [1]. Since that time, numerous articles and a major textbook have described the HRCT appearances of common and uncommon pathologic conditions of the lung parenchyma and bronchial tree [2]. Although there are many uses for HRCT in clinical practice, this review focuses in its role in establishing specific diagnoses in symptomatic patients such that biopsy is not required [3].

There are several methods by which tissue can be obtained for diagnostic purposes in patients with pulmonary disease. These include bronchoscopy with transbronchial biopsy (TBBx), open lung biopsy (OLB), and video-assisted thoracoscopic surgery (VATS). The distribution and appearance of HRCT abnormalities determine both the optimal location and type of biopsy; for example, TBBx is most appropriate in peribronchovascular disease such as sarcoidosis and lymphangitic carcinomatosis. OLB or VATS is often required for diagnosis of peripheral or nonsegmental disease [4]. In all cases, HRCT identifies appropriate locations for biopsy; this is of particular importance prior to VATS because of the small area of direct operator vision during the procedure. Proper interpretation of histopathologic specimens also requires HRCT correlation in many cases, particularly given the small samples obtained with many of these procedures.

Specific HRCT diagnoses obviate biopsy altogether. Diseases which can have diagnostic HRCT appearances include certain types of ILD as well as a variety of other disorders; many of these are reviewed below. The role of HRCT in the selection of patients for empiric therapy in lieu of immediate tissue diagnosis will not be addressed given the short length of this discussion. However, concepts of accuracy will be emphasized and the need for future research direction will be discussed. The bulk of the available HRCT accuracy literature will be reviewed in some detail.

## HRCT-Specific ILD Diagnoses

### Usual Interstitial Pneumonitis (UIP)

UIP, a nonspecific lung reaction, can occur in association with collagen vascular disease, as a manifestation of drug toxicity, or secondary to asbestosis; most cases, however, are spontaneous and termed idiopathic pulmonary fibrosis (IPF).

HRCT findings vary with the amount and degree of fibrosis present. In most patients, findings of fibrosis are dominant. Secondary lobules are distorted and angulated. Architectural distortion, traction bronchiolectasis, and irregular interfaces between parenchyma and vessels and bronchi are also present in most patients [5, 6]. Intralobular lines, which correspond to fibrosis within the lobule, and honeycomb cysts are commonly present in patients with advanced disease; interlobular septal thickening may also be present, but it is not conspicuous. HRCT shows the characteristic subpleural, posterior, and lower lung zone distribution of disease. The costophrenic angles are the site of initial and most severe abnormalities, and the diagnosis of UIP should not be made unless this predominance is present [7]. In patients with typical findings, HRCT is specific. However, HRCT does not allow distinction between IPF and UIP associated with the various other conditions listed above.

Ground glass attenuation (GGA), increased lung density which does not obscure underlying pulmonary vascular structures, is the dominant or sole abnormality present in patients with early disease (alveolitis). The HRCT appearance may be indistinguishable from other types of ILD or from diffuse infection. However, features of minimal fibrosis are often present in these cases at close examination. The characteristic costophrenic angle involvement and/or the appropriate clinical setting (patient with collagen vascular disease) prompts specific diagnosis in many of these cases.

### Sarcoidosis

Sarcoidosis is probably the type of ILD for which the need for tissue diagnosis is the most controversial,



even in patients with typical imaging and clinical findings. Some authors now consider HRCT diagnostic in select cases when associated with the appropriate clinical history [2].

The HRCT appearance of sarcoidosis, like most forms of ILD, varies with the stage of disease. The presence of isolated hilar and mediastinal adenopathy generally does not allow confident diagnosis; other causes of lymph node enlargement, particularly lymphoma, can cause an identical appearance. Calcification, particularly in the periphery of symmetrically enlarged nodes, is strongly suggestive if not diagnostic of sarcoidosis; the need for tissue diagnosis in these cases is controversial.

Parenchymal sarcoidosis often has a diagnostic HRCT appearance. Well-formed interstitial granulomas, the hallmark of the disease, result in the presence of small, dense nodules in a perilymphatic (interstitial) distribution; nodularity occurs along both the central bronchovascular bundles (axial interstitium) and the fissures and pleural surfaces (peripheral interstitium) [8-10]. The interlobular septa, although anatomically part of the peripheral interstitium, are not prominently affected. The findings are characteristically patchy or multifocal, and occur primarily in the upper lobes. The HRCT appearance can be diagnostic with or without associated adenopathy at this stage. Perilymphatic nodules can also occur in patients with silicosis, berylliosis, or lymphangitic carcinomatosis (LC); clinical and occupational history usually distinguishes between these entities.

#### **Silicosis, Coal Worker's Pneumoconiosis, and Berylliosis**

Silicosis and coal worker's pneumoconiosis (CWP) are different diseases, but cause similar or identical imaging findings. Both diagnoses require significant occupational exposure (at least 10 years) to silica or coal dust, respectively. Silicosis results in peribronchiolar layers of laminated connective tissue surrounded by areas of focal emphysema. HRCT findings reflect the underlying pathology; small peribronchiolar nodules correspond to the connective tissue lesions; however, perilymphatic nodules are also present [11]. Occasionally, perinodular emphysema is evident. The nodules, which may calcify, are most numerous in the posterior upper lung zones, and are bilateral and usually symmetric. Adenopathy, which may contain peripheral calcification, is common. In patients with longstanding disease, nodules enlarge; eventually, coalescent masses of parahilar fibrosis (progressive massive fibrosis) and peripheral paracatricial emphysema develop. HRCT enables specific diagnosis in patients with the appropriate history; in many instances, HRCT suggests the possi-

bility of silicosis before occupational information is available.

The lesion of CWP is the coal macule; this small accumulation of coal dust surrounds respiratory bronchioles. HRCT depicts small centrilobular nodules most numerous in the posterior and upper lung zones; the imaging findings can not reliably be distinguished from those of silicosis [12]. Progressive massive fibrosis can also occur with CWP.

Berylliosis is pathologically identical to sarcoidosis; this chronic granulomatous condition results from a cell-mediated response to beryllium, and can occur in ceramics workers, those in the nuclear weapons industry, and in fluorescent lamp manufacturers. HRCT findings are identical to those of nodular sarcoidosis; perilymphatic (interstitial) nodules occur in the upper lungs along bronchovascular bundles and along pleural surfaces. A diagnostic serum test is available and is useful in patients at risk for berylliosis [13].

#### **Lymphangitic Carcinomatosis (LC)**

LC, which results from metastatic forms of adenocarcinoma, is a perilymphatic disease which can cause either smooth or nodular thickening of the interstitial framework of the lung. This thickening is due to either tumor growth or obstructive edema within the interstitium, lymphatics, or vessels [14, 15]. The characteristic HRCT appearance consists of focal or multifocal smooth or nodular interlobular septal thickening (peripheral interstitial disease), often with apparent thickening of central airways and adjacent pulmonary arterial branches (axial interstitial disease). Septal thickening often outlines many contiguous lobules in patients with LC; the appearance is termed "polygonal arcades" [14]. The arterial branch, normally visible in the center of each lobule, often appears enlarged and irregular due to centrilobular axial interstitial disease [16]. While septal and peribronchovascular thickening is usually smooth, the abnormalities can be nodular, particularly in the interlobular septa [15]. LC characteristically does not cause findings of fibrosis.

A specific HRCT diagnosis of LC is relatively straightforward in most cases, but is generally not made unless there is a known history of primary malignancy or a visible lung nodule or mass likely to represent primary lung carcinoma. Rarely, LC is diffuse and may be indistinguishable from interstitial edema; in these cases, a post-diuresis scan may be required [17].

There is some overlap in the HRCT appearances of sarcoidosis, silicosis-CWP-berylliosis, and LC; all can cause perilymphatic (interstitial) nodules. Clinical information generally affords a specific diagnosis of one of these conditions. Clinical information is vital





to accurate HRCT interpretation in most cases, and if diagnostic uncertainty persists after integration of all available data, biopsy should be performed. HRCT depiction of perilymphatic disease generally prompts TBBX, and the imaging findings determine the appropriate biopsy sites.

### **Hypersensitivity Pneumonitis (HP)**

HRCT findings in patients with HP vary with the stage of the disease. Acutely, inflammatory cells fill the alveoli, causing nonspecific patchy or diffuse consolidation. Subacute HP, which occurs after resolution of the acute phase or as a baseline state between acute exacerbations in patients with continuous low-grade exposure to offending antigens, is characterized by alveolitis and peribronchiolar granulomatous inflammation and bronchiolitis. Radiographs may depict small nodules at this stage, but are often normal or nonspecific. HRCT demonstrates patchy areas of ground glass attenuation (GGA) and a background pattern of diffuse fuzzy centrilobular nodules, which are often poorly defined and also of GGA. These nodules, which measure less than 5 mm in diameter, are bilateral and involve all lung zones, and represent the bronchiolar and peribronchiolar inflammatory infiltrate [18]. Centrilobular GGA nodules can only be identified on very thin sections (1 or 1.5 mm), and can be quite subtle. Although other entities can also cause such nodules, the HRCT appearance is diagnostic of HP in the proper clinical setting.

If diagnostic certainty is insufficient to obviate tissue diagnosis, the HRCT findings (centrilobular nodules) suggest TBBx as the procedure of choice; in these cases, the granulomatous inflammation of HP may be difficult to differentiate from that of sarcoidosis. Final accurate diagnosis is often possible only with integration of clinical history and histopathologic and HRCT findings.

### **Eosinophilic Granuloma (EG)**

EG (pulmonary histiocytosis X) is characterized by peribronchiolar granulomatous inflammation in which there is a characteristic proliferation of histiocytes; later, this evolves into fibrosis, and cystic spaces form in the parenchyma. HRCT findings reflect the underlying pathology and the stage of disease. Small, often poorly-defined nodules are present in a peribronchiolar (centrilobular) distribution in patients with early disease; the appearance is similar to that of subacute HP, but the nodules of EG are less profuse and occur predominantly in the upper lungs. At this stage, HRCT may be suggestive of the diagnosis in the proper clinical setting (young smoker), but a specific diagnosis can only be made if characteristic lytic bone lesions are also present.

The combination of small centrilobular nodules and cysts, which occurs in subacute EG, is diagnostic [19, 20]. The cystic spaces may be thin or thick-walled and are round or coalescent; the latter cysts can have bizarre shapes. The costophrenic angles are typically spared. In some patients, the cystic lesions may be present without nodules; this appearance is also diagnostic. Advanced EG is more difficult to diagnose with HRCT (and with OLB). The characteristic cystic lesions may not be present; advanced fibrosis can result in an emphysema-like pattern. An HRCT diagnosis of severe emphysema in a young patient may prompt consideration of endstage EG in the differential diagnosis, but the appearance is not diagnostic.

### **Lymphangioleiomyomatosis (LAM) and Tuberos Sclerosis**

A progressive proliferation of spindle cells along the lymphatic vessels of the lung occurs in LAM. When this cellular growth occurs in the peribronchiolar lymphatics, areas of peripheral air trapping develop, leading to the formation of characteristic thin-walled parenchymal cysts. Spindle cells in the intrathoracic lymph nodes may lead to lymphatic dilatation and chyloous pleural effusions. A small minority of patients with tuberos sclerosis develop ILD indistinguishable from LAM.

The cystic lesions, often not evident on chest radiographs, have a diagnostic HRCT appearance. They range from several mm to several cm in diameter, and are thin-walled, round, and monotonous. Normal parenchyma and bronchovascular structures are present in areas between cysts [21, 22]. Unlike EG, the cysts of LAM do not coalesce, and the costophrenic angles are not spared. There is also no nodular phase of LAM. HRCT obviates lung biopsy in the proper clinical setting (young woman of childbearing age). Mediastinal and retrocrural adenopathy and pleural effusions (chylothoraces) may be present, but are not essential to the diagnosis. Occasionally, late or advanced disease may be difficult to differentiate from severe EG or emphysema.

## **HRCT-Specific Miscellaneous Diagnoses**

### **Bronchiectasis**

In many instances, HRCT depicts bronchiectasis in symptomatic patients in whom radiographs are nonspecific or normal; in these cases, an HRCT diagnosis of bronchiectasis, while not specific as to the underlying cause, obviates further testing or tissue diagnosis [23].

### **Emphysema**

HRCT is more sensitive than either chest radiography or conventional CT in the identification and



diagnosis of emphysema. Areas of low attenuation, with an upper lobe predominance, are characteristic of centrilobular emphysema; these low attenuation spaces have very thin or imperceptible walls and may coalesce with advanced disease. The HRCT appearance is diagnostic. Paraseptal emphysema, which involves the distal aspect of the secondary lobule, causes peripheral, subpleural cystic spaces which have very thin walls. The HRCT appearance, although diagnostic, may be confused with honeycombing and pulmonary fibrosis by inexperienced observers [17].

### **Bronchiolitis Obliterans**

Bronchiolitis obliterans (BO) is a nonspecific lung reaction characterized by peribronchiolar inflammation and plugs of granulation tissue within respiratory bronchioles. BO is the common endstage pathway for a variety of insults; it can follow viral infection (Swyer-James syndrome) or toxic fume inhalation, and can occur in association with various forms of connective tissue disease, particularly rheumatoid arthritis. Histologic BO is also present as a manifestation of rejection in lung transplant patients and of chronic graft-versus-host disease after bone marrow transplantation.

Chest radiographs may be normal or may demonstrate only large lung volumes in affected patients; HRCT depicts mild central airway dilatation and wall thickening with a peripheral pattern of mosaic perfusion. This heterogeneous parenchymal density can be striking, and is occasionally present without evident abnormalities of the central airways; in some cases, air trapping and the resultant mosaic perfusion pattern are only evident on expiratory HRCT. Peripheral bronchiolectasis is not a feature [24].

The HRCT diagnosis is specific only in the proper clinical setting and should not be made unless the history and pulmonary function tests are corroborative. In addition, if BO is clinically suspected, expiratory HRCT should be performed in addition to the standard end-inspiratory images [17].

### **Infectious Bronchiolitis**

Bacterial, mycobacterial, and viral infections of the bronchial tree can result in inflammatory changes particularly at the bronchiolar level.

HRCT does not allow a specific bacteriologic diagnosis, but the appearance is characteristic of infectious bronchiolitis. HRCT depicts impacted small airways as centrilobular tubular structures with nodular edges; the configuration resembles a “jack” used in the childhood game played with a rubber ball [23]. Peribronchiolar consolidation causes ill-defined nodular opacities centered on the small airways which are characteristically centrilobular in location

and do not reach the pleural surface or fissures [25]. HRCT diagnosis of infectious bronchiolitis obviates immediate transbronchial biopsy or bronchoalveolar lavage. Sputum cultures for bacterial and mycobacterial pathogens and empiric antibiotic therapy are appropriate [26].

### **Miliary Infection or Metastases**

Random (hematogenous) nodules are readily identified and localized with HRCT even when their diameter is quite small (several mm) [25]. The distribution is typical of either miliary infection or metastases. In patients with known malignancy, no tissue diagnosis is required in most cases. TBBx is necessary in most patients with miliary infection to establish a specific diagnosis.

## **Review of HRCT Diagnostic Accuracy in ILD**

Specific diagnoses based on HRCT are based predominantly on case series which report imaging features of pathologically proven entities and on the extensive clinical experience with the technique in the past 15 years. Scientific studies of the accuracy of HRCT are few and the conclusions that can be drawn from these investigations are limited.

**Mathieson** and colleagues first addressed the diagnostic accuracy of conventional CT, HRCT, and chest radiography in patients with ILD in a retrospective study of 118 patients imaged over a four-year interval [27]. Three observers interpreted radiographs and CT/HRCT images separately and without clinical information, and listed three diagnostic choices in decreasing order of probability for each examination. A “high-confidence level” diagnosis, the degree of which was not specified, was reached in 23% of all patients based on the CXR and in 49% with CT; these diagnoses were proven correct in 77% and 93%, respectively. CT/HRCT was most accurate in usual interstitial pneumonitis (UIP) (95% of “high-confidence” diagnoses correct), silicosis (100%), sarcoidosis (88%), and lymphangitic carcinomatosis (LC) (93%). These authors were the first to suggest that CT/HRCT could obviate lung biopsy, specifically in patients with UIP and silicosis in whom characteristic HRCT findings were present. Patient referral bias (referral for the test under evaluation was itself a criterion for study entry) limits application of the quoted accuracy values to populations other than that studied by the authors.

**Padley** et al. retrospectively evaluated 86 patients with pathologically proven ILD [28]. HRCT was performed with 3 mm sections at 10 mm intervals (closer to current technique); two observers reached a “confident” (again not formally defined) first-choice



diagnosis in 41% and 49% of all patients with CXR and HRCT, respectively, correct in 69% (CXR) and 82% (HRCT). This study confirmed the high accuracy of HRCT for UIP (90%), and first reported the accuracy of HRCT for EG (100%). Accuracy rates for “confident” diagnoses of sarcoidosis and HP were much lower in this study than in others [27, 29]; this may reflect lack of observer familiarity with imaging characteristics of these diseases or a high percentage of patients with these diagnoses in whom imaging findings were nonspecific. The overall lower accuracy of a “confident” diagnosis of ILD in this study when compared with the results of Mathieson et al. probably reflects variable ILD case mix. For example, Padley et al. included only three cases of silicosis and LC, but seven cases of cryptogenic organizing pneumonia (which has a nonspecific HRCT appearance).

Current state-of-the-art HRCT technique was first used by Grenier and colleagues in 140 ILD patients [29]. Three observers interpreted CXR and HRCT images without clinical information and arrived at correct “high-probability” (defined for the first time as greater than a 75% chance) specific diagnoses in 57%, 55%, and 47% of all cases of ILD, respectively. These values are low in comparison to those quoted in previous investigations, but Grenier et al. reported the percentages of all patients with ILD in whom HRCT enabled a correct “high probability” diagnosis. Other authors reported the percentages of “confident” diagnoses that were correct.

A correct “high probability” diagnosis was made at HRCT by the three observers in 59-88% of the patients with EG, 64-72% with sarcoidosis, 30-67% with UIP, and 27-55% with silicosis; there were too few patients with HP or LC to allow evaluation. That is, a correct “high probability” diagnosis of sarcoidosis, for example, was made at HRCT in 64-72% of all patients with sarcoidosis. The authors did not state whether false positive “high probability” diagnoses occurred. The wide variation in the percentage of “high probability” diagnoses for each specific ILD suggest the presence of interobserver variability. Although Grenier et al. concluded that an HRCT diagnosis of EG could eliminate open lung biopsy, they made no such statement regarding other forms of ILD [29].

Recently, in an attempt to assess HRCT in a clinical context, Grenier and colleagues studied clinical information, CXR, and HRCT in patients with ILD and applied a form of Bayesian analysis to the results [30]. The authors studied 308 patients; 208 retrospectively (Group A) and 100 prospectively (Group B). CXR and HRCT images were interpreted according to consensus of 2 experienced chest radiologists blinded to clinical or laboratory data. No specific ILD diagnosis was attempted based on imaging;

rather, a checklist (18 CXR and 28 HRCT findings) was completed for each case. Chi-square analysis (assuming independence of variables) determined symptoms (also completed on a checklist) and radiologic findings most relevant to the diagnosis of each specific ILD in Group A; this information was then applied prospectively to the study of Group B assuming an identical prevalence of each ILD in the two populations.

Specific diagnoses were made with a high level of confidence (probability greater than or equal to 95%) in Group A in 29% of cases based solely on clinical data, 9% on radiographs, and in 36% with HRCT alone. Combined clinical and radiographic data afforded correct high confidence diagnoses in 54%; this rose to 80% with the addition of HRCT. In group B, high confidence specific diagnoses were made on clinical, radiographic, and HRCT examination in 27%, 4%, and 49% of cases, respectively; this rose to 53% when clinical findings were combined with radiographs and to 61% with the addition of HRCT. Of importance is the fact that in both groups, HRCT with CXR and clinical data performed much better than did HRCT alone (80% and 61% v. 36% and 49% in group A and B, respectively). Accuracy studies in which HRCT interpretations were made without clinical information therefore underestimated the actual potential diagnostic impact of the technique.

Incorrect “high confidence” diagnoses, reported for the first time, occurred in 4% of patients in Group A even with integration of all available clinical and radiographic information. Many of these were semantic (rheumatoid lung and drug toxicity “misclassified” as IPF, hematogenous metastases “mistaken” for lymphangitic carcinomatosis). Of note, the fewer incorrect diagnoses were made as the probability level required for a “confident” diagnosis increased. The accuracy rates for HRCT quoted in earlier investigations did not account for this fact; the exact level of probability required for a “high confidence” diagnosis was never precisely specified. If “high confidence” was defined as a probability sufficient to obviate biopsy, for example, fewer patients with each disease would have a “confident” diagnosis made with HRCT, but these would be correct in a very high percentage of cases.

Incorrect “confident” diagnoses of significance did occur in the prospective portion of the study (group B); these included patients with LC considered to have sarcoidosis and HP, respectively, and four patients with silicosis believed to have sarcoidosis. These errors occurred despite integration of clinical, radiographic, and HRCT data. These misdiagnoses may have been due to the assumption of equal disease prevalence (a factor in the Bayesian analysis) in Groups A and B; in fact, the two groups differed



slightly in this regard. Thus, common diseases (sarcoidosis) were diagnosed instead of uncommon ones (silicosis). The model also failed to diagnose any case of HP because the model assumed independence of variables; for example, nodules and ground glass attenuation (GGA) were each on the HRCT checklist, but a centrilobular location of nodules was not. There was no way to report the presence of centrilobular GGA nodules on the checklist. This is relevant because this specific pattern is most characteristic of subacute HP.

Based upon a review of the above studies, the following conclusions are possible: 1) HRCT technique varied; in some studies, many patients did not undergo true HRCT. Current state-of-the-art HRCT should offer significant improvement in accuracy when compared the results in published studies; 2) the overall accuracy of HRCT depends on the case mix of ILD in the population under study. Accuracy rates are lower in populations with large numbers of patients with forms of ILD with nonspecific HRCT appearances than in other groups. Patient selection bias can have a significant impact on the case mix in retrospective studies. It is not the overall accuracy that is important, but the accuracy of HRCT in patients with each specific type of ILD. The percentage of patients with each type of ILD in whom HRCT enables a specific diagnosis depends on the degree of probability required to make that diagnosis; 3) HRCT accuracy is heavily dependent on the experience and ability of the interpreter; 4) most forms of ILD pass through various stages, some of which have characteristic HRCT findings; other stages have a nonspecific appearance. Accuracy values for a given ILD vary with both the type and severity of disease; 5) HRCT should always be interpreted with clinical information; an integrated approach to the diagnosis of ILD is essential.

## Conclusions

There is significant variability both within and between institutions with regard to the use of HRCT to render specific diagnoses without histologic or pathologic confirmation. In part, this reflects variability in the confidence and skill of radiologists; however, the attitudes, preferences, and experiences of individual pulmonary medicine specialists and/or thoracic surgeons are also important in this regard. Current reimbursement mechanisms encourage the performance of invasive diagnostic procedures; this is, however, changing rapidly in many parts of the country. Therefore, it is important to review and to update the way in which symptomatic patients are evaluated and managed. The impact of HRCT in the symptomatic patient in whom radiographs are normal or nonspecific must be explored in detail; if HRCT obviates further diag-

nostic procedures, whether invasive or noninvasive, it may be cost-effective rather than an added expense [26].

It cannot be overemphasized that the diagnostic evaluation of the patient with known or suspected lung disease, ILD or otherwise, should be a team approach involving pulmonologists, thoracic radiologists, and thoracic surgeons. There is no logic to a diagnostic approach that fails to include HRCT or employs HRCT only if bronchoscopy with biopsy is nondiagnostic, particularly patients with ILD. Similarly, in most cases, HRCT can not make a precise specific diagnosis without clinical data.

It is clear that in a significant percentage of symptomatic patients, HRCT findings are characteristic enough such that biopsy is redundant. HRCT should be interpreted only by experienced observers, and final reports of these studies should include levels of diagnostic certainty. Even in cases that ultimately require tissue diagnosis, HRCT a) directs appropriate site and type of lung biopsy and b) offers important supplemental information to pathologists interpreting limited biopsy specimens.

When evaluating the potential of HRCT to obviate biopsy, it is important to recognize that even OLB is not always accurate in the diagnosis of ILD, particularly in patients with advanced fibrosis [31]. Therefore, acceptable accuracy rates for HRCT-specific diagnoses may also be below 100%. Further systematic study of the accuracy of HRCT in clinical practice is needed.

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# Imaging Algorithms in Pulmonary Embolism

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## Pulmonary Aspiration Syndromes

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### Learning Objectives

The attendee will learn the major risk factors for aspiration

The attendee will learn the multiple manifestations of aspiration

The attendee will learn the role of various imaging modalities in the diagnosis of aspiration and its results.

Aspiration is the taking in of foreign material into the lungs with the respiratory current. It encompasses multiple entities occurring in differing

settings, having different management approaches and having differing radiographic manifestations. The incidence of aspiration varies widely depending upon the clinical setting. Radionuclide studies in many normal individuals show aspiration of small amounts during sleep or anesthesia. Three general groups of conditions that predispose to aspiration include states of depressed sensorium, neurologic disorders that interfere with swallowing, and structural disorders that interfere with swallowing. Some of the entities in each of these categories are shown in Table 1.

**Table 1:**  
**Common Causes of Pulmonary Aspiration Syndromes**

Depressed Sensorium	Neuromuscular Discoordination	Structural Disorders of the Aerodigestive Tract
Anesthesia Alcoholism Drug overdose Head injury Cerebral Infarction Cerebral hemorrhage Metabolic coma Seizure disorders	Multiple sclerosis Parkinson's disease Cranial neuropathy Muscular dystrophies Cerebral palsy Cerebral infarction Cerebral hemorrhage Head injury Choking Guillain-Barre syndrome Amyotrophic lateral sclerosis	Achalasia Esophageal carcinoma Laryngeal carcinoma Pharyngeal carcinoma Gastroesophageal reflux Esophagobronchial fistula Esophageal stricture Zenker's diverticulum





The consequences of aspiration are divided into several common forms: aspiration of gastric contents with or without chemical pneumonia, pleuropulmonary infections resulting from aspiration, aspiration of foreign materials into the lungs, and acute obstruction of the airway.

### Aspiration of Gastric Contents

Mendelson described the massive aspiration of gastric contents in 66 obstetrical patients in 1946. Forty of those patients developed dyspnea, tachycardia, wheezing and rhonchi and radiographic appearances suggesting pulmonary edema. All of those patients recovered. Five additional patients developed upper airway obstruction. Two of them died and two developed massive atelectasis. The remaining patients were asymptomatic.

Many studies have been conducted regarding aspiration of gastric contents. In summary, these suggest that the severity of injury is highest when particulate food is present, when the pH of the aspirate is below 2.5, and varies directly with the volume of aspirate. The risk of chemical pneumonitis decreases with age due to decreased secretion of gastric acid in elderly patients. Clinical manifestations of aspiration vary widely. With minor aspiration, there may be no symptoms. With major aspiration, patients may develop apnea, tachypnea, cyanosis, hypotension, fever, and adventitious respiratory sounds. Rarely, patients develop acute respiratory failure within hours of the episode. Patients may exhibit hypoxemia, and leukocytosis with a left shift.

Perioperative aspiration is a significant problem. Warner et al noted that patients undergoing emergency surgery are more likely to aspirate than those undergoing elective surgery (1/895 v 1/3886). The most common predisposition for aspiration is bowel obstruction. Other conditions are depressed sensorium, previous esophageal surgery, swallowing difficulty, and recent meal. Aspiration episodes are fairly evenly distributed throughout a surgical procedure. Thirty two percent of episodes occur during laryngoscopy and 35.9% occur during extubation with the remainder occurring during the procedure or post anesthesia recovery.

In the past 15 years, multiple forms of enteral feeding have been developed to improve patient's nutrition. Despite attempts to prevent it, aspiration may occur with any type of enteral feeding. Mullan et al reviewed their experience with nutritional support in patients with nasogastric tubes, gastric tubes, and jejunal tubes. Twelve of 276 (4.4%) developed an episode of aspiration. In only 4 of these did chest radiographs show infiltrates. All of these patients had fever. Mortality was not different in these 12 patients from the remainder of the cohort.

### Radiographic Manifestations of Gastric Aspiration

Radiographic manifestations of aspiration are non-specific. Opacities may be minimal or extensive and irregular, confluent, or nodular. The distribution of abnormalities depends on the distribution of the aspirated material. Liquid material flows according to gravity to the most dependent portion of the tracheobronchial tree. When patients are supine, this is the dorsal aspect of the midlungs with predominance on the right. In the erect or semierect position, this is the lung bases. Landay et al reported on 60 hospitalized patients with aspiration of gastric contents. Fifty-one of 60 (85%) had infiltrates on the initial radiograph and 3 developed them on subsequent studies. Infiltrates were bilateral in 41 and asymmetric in 35. There was a 26.7% mortality rate with eventual clearing in the remainder. Radiographic clearing began between days 1 and 9.

### Pleuropulmonary Infections Resulting from Aspiration

Pleuropulmonary infections result from aspiration of smaller amounts of material than are associated with chemical pneumonitis. The aspirate is contaminated with pathogenic bacteria that arise from the oropharynx or bacterial colonization of gastric secretions. When the aspiration occurs outside the hospital, anaerobic bacteria predominate as pathogens. The organisms most often implicated are *Bacteroides melaninogenicus*, *Fusobacterium nucleatum*, *Bacteroides fragilis*, *Peptostreptococcus* spp, and microaerophilic *Streptococcus*. In patients with hospital acquired aspiration related infections, there is a higher frequency of mixed aerobic-anaerobic infections. In addition to the anaerobic organisms just listed, aerobic pathogens include *Haemophilis influenzae*, methicillin-resistant *Staphylococcus aureus*, gram-negative enteric bacilli, *Pseudomonas pneumoniae*, and *Serratia marcescens*.

Determination of the presence of pneumonia may be quite difficult in hospitalized patients, especially those receiving mechanical ventilation. When an aspiration is witnessed, new opacities are identified on a chest radiograph, and clinical pneumonia develops in temporal relation to the event, the diagnosis is straightforward. Unfortunately, the diagnosis is usually not so certain. In a postmortem study of 24 patients with ARDS and possible pneumonia, Andrews et al found that the combination of clinical and radiographic criteria were accurate in only 29% for the presence or absence of pneumonia. Pneumonia was present in 58% of patients at postmortem examination. Thirty six per cent of these patients were thought to



have ARDS only. Twenty per cent of the patients with uncomplicated ARDS were thought to have pneumonia. Quantitative tracheal cultures were of no value in distinguishing patients with pneumonia and simple colonization.

In an attempt to improve on the ability to diagnose pneumonia in patients with mechanical ventilation, Chastre et al compared protected specimen brush cultures obtained bronchoscopically with the cultural and histologic diagnosis of lung specimens taken immediately after death. When protected specimen brush quantitative cultures grew organisms in a concentration  $>10^4$  colony forming units per milliliter histologic pneumonia was present and there were no false positives. Using the technique of protected specimen brush quantitative cultures, Fagon et al found that pneumonia was present on only 31% of patients who were undergoing mechanical ventilation and who had fever, pulmonary infiltrates, and purulent sputum.

Nosocomial pneumonia developing in patients receiving mechanical ventilation is thought to relate to microaspiration of upper airway oropharyngeal secretions or gastric secretions that have been colonized with bacteria. Prod'homme et al evaluated 244 patients receiving mechanical ventilation. They found that pneumonia developing within 4 days after intubation was most commonly due to *Staphylococcus aureus*, *Streptococcus pneumoniae*, and *Haemophilis influenzae*. On the other hand, pneumonia developing later than 4 days after initial intubation was most commonly due to gram negative bacilli. In 84% of patients with late onset gram negative bacillary pneumonia, gastric colonization with bacteria of the same species preceded the development of pneumonia. Gastric colonization was also present in 75% of patients who developed late onset staphylococcal pneumonia.

#### **Radiographic Manifestations of Pleuropulmonary Infections from Aspiration**

Patients with infection from aspiration may exhibit irregular, segmental or lobar opacities. Cavitation with or without air fluid levels is frequent. Variable wall thickness and variable amounts of surrounding lung opacity occur. Abscesses usually present in dependent portions of the lungs. In patients who have been unconscious, the posterior portions of the lungs are the target areas. In patients with neurological dysfunction or structural abnormality, the lung bases are the target areas. Pleural effusion may result from parapneumonic inflammation. An interface indenting the lung suggests empyema. Hydro-pneumothorax indicates a bronchopleural fistula.

It is difficult to identify the etiology of infiltrates on a patient in an ICU. Winer-Muram et al evaluated radiographs on 40 patients with fever, leukocytosis and

purulent tracheal secretions receiving mechanical ventilation. Pneumonia was diagnosed in these patients by quantitative protected specimen brush cultures. The accuracy of identification of pneumonia was only 57%.

#### **Aspiration of Foreign Materials into the Tracheobronchial Tree and Lungs**

Airway obstruction may develop after aspiration of solid or semisolid material. The clinical manifestations depend upon the size of the solid material and level of obstruction. If the airway is completely blocked, no airflow occurs and death rapidly ensues. When a bronchus is obstructed, patients may have wheezing, stridor, cough, and hypoxemia. In comparison to adults, children are much more likely to aspirate foreign bodies.

#### **Radiographic Manifestations of Foreign Material Inhalation**

The manifestations of foreign material inhalation depend upon the material inhaled. Radioopaque foreign bodies may be readily detectable and lodged in an airway. If small enough to enter a mainstem bronchus, the bodies usually go to the right due to the more obtuse angle of the right bronchus with the trachea than the left bronchus. If the foreign body is not radioopaque but lodges in a bronchus, it may cause segmental or lobar atelectasis. Occasionally, foreign bodies do not result in either atelectasis or radiographic opacities and are identified in the trachea and bronchi only retrospectively.

If the material is more liquid, identifiable abnormalities are dependent upon the innate density of the material. Near drowning results in water density infiltrates with a multifocal or diffuse distribution. Aspiration of  $Ba^{++}$  during a diagnostic exam may result in extensive radiodense infiltrates. Aspiration of mineral oil may result in low density infiltrates as identified by CT. Chemical shift magnetic resonance imaging may also identify infiltrates as being due to oily materials.

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# Asbestos-Related Lung and Pleural Disease

*Philip C. Goodman, MD*

Historically asbestos has been with us for thousands of years. Herodotus, the Greek historian, wrote of the use of asbestos in weaving cremation cloths. Other anecdotes tell of the use of asbestos in lamp wicks, tablecloths and socks. When large deposits of asbestos were discovered in Canada and South Africa at the height of the Industrial Revolution, use of this material spread rapidly throughout the world. Recognition of the dangers asbestos held for mankind were soon to follow. Even in the 1500s scattered accounts of pulmonary illness related to asbestos exposure were noted. Zenker, in 1867, first used the word pneumokoniosis, meaning dusty lungs, to describe the illness seen in coal and iron miners. Soon the term pneumoconiosis was being used to describe the disease seen when workers were exposed to asbestos. By 1930 the British Parliament and the International Labor Office in Geneva recognized the significance of disease caused by asbestos exposure and the term asbestosis first used in the 1920s to describe the lung problems noted in patients working in the asbestos industries. In the 1930s the association of asbestos exposure with lung and pleural disease was becoming more accepted. A case of neoplasm in a patient with asbestos exposure was reported in 1935 and other case reports soon followed. In 1964, finally, a careful review of hundreds of patients with long-term exposure to asbestos established that asbestos was indeed a carcinogen responsible both for lung cancer and mesothelioma.

Asbestos is a term which refers to a group of generally indestructible fibrous silicates which are pit mined from the earth. Major fiber types are serpentine and amphibole. A variety of fibers consisting of different chemical make-ups may be responsible for the diseases associated with asbestos exposure. Work groups at risk for exposure include asbestos miners and millers, textiles manufacturers, construction workers in all trades, shipbuilders, and certain members of the automotive industry.

Asbestosis refers to the delayed pulmonary disease caused by inhalation of asbestos. This is in contrast to asbestos-related pleural disease. It is thought that asbestos fibers are transported through conduct-

ing airways to the most distal portion of lung where enzymatic and inflammatory reactions ultimately result in fibrosis. Clinically patients generally present with shortness of breath which becomes progressively worse until they are dyspneic at rest. Late inspiratory crackles are characteristically heard on physical examination.

Chest radiology has been one of the major tools involved in diagnosing asbestosis for many years. The International Labor Office in Geneva through a series preceding scoring models published in 1980 the latest chest radiographic grading system for asbestos-related chest diseases currently available. Using this grading technique B-readers were initially charged with an epidemiologic role in monitoring asbestos-related lung and pleural disease. For the last few decades B-reading has served primarily as a tool for litigation. A discussion of B-reading will ensue during the course of this workshop. Briefly B-reading entails the use of standardized films to grade the subject chest radiograph in regards to profusion or degree of involvement, distribution of disease, quality of opacities, type, distribution, and extent of pleural disease, as well as incidental findings.

Pleural disease seen in patients exposed to asbestos include benign asbestos pleurisy, pleural thickening of both localized or plaque-like nature as well as diffuse involvement, and malignant mesothelioma.

Complicating features of asbestos exposure include rounded atelectasis and bronchogenic carcinoma.

CT scanning has played an increasing role in the diagnosis of asbestos-related pleural and parenchymal disease. While no standardized method of applying CT scanning to the evaluation of asbestos-related diseases has been accepted (as has the ILO classification of PA chest radiographs), nevertheless CT scanning clearly demonstrates less severe parenchymal involvement than chest radiography, and at times can clarify the nature of pleural thickening seen on plain chest films. The extent and nature of bronchogenic carcinoma and malignant pleural mesothelioma can also be better discerned by CT scanning than plain radiography. The role of CT in regards to asbestos-related diseases will be discussed.



# Interventional Angiography in Trauma to Chest

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# Notes