

# Thomas Pavillion March 15, 2000

2:15–3:00      Session 1: Fallacious Dogma in the Diagnosis of Pulmonary Embolism  
*Eric N. C. Milne, MD*

Session 2: Diagnosis and Management of Pleural Disease  
*Francine L. Jacobson, MD, MPH*

Session 3: ABCs of Blunt Chest Trauma  
*Jud W. Gurney, MD*

Session 4: Thoracic Manifestations of Lymphoma  
*David S. Schwartz, MD*

3:00–3:15      Break

3:15–4:00      Session 1: The Small Solitary Pulmonary Nodule  
*David F. Yankelevitz, MD*

Session 2: The Radiologic and Pathologic Spectrum of Bronchopulmonary Carcinoids  
*Kay H. Vydareny, MD*

Session 3: A Chest Radiologist's Approach to Abdominal CT in Chest Diseases  
*Daniel C. Rappaport, MD*

Session 4: Thoracic Sarcoidosis  
*Gordon L. Weisbrod, MD*

4:00–4:45      Session 1: Neuroendocrine Tumors of the Thorax  
*Gerald F. Abbott, MD*

Session 3: Tracheobronchial Stenting Techniques  
*Stephen T. Kee, MD*

Session 4: Radiologic Evaluation of the Diaphragm  
*Kook S. Oh, MD*

5:00–6:00

## Expert Film Reading Panel

*Moderator: Robert D. Tarver, MD*

Wednesday, cont.



# Fallacious Dogma in the Diagnosis of Pulmonary Embolism

*Eric N. C. Milne, MD, FRCR, FRCP*

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Dogma 1. That “infarct” means dead tissue. Infarction means literally to “stuff in” and has no connotation of death. In the heart and brain, where there is only one source of circulation, an embolus *does* lead to tissue death, but in the lung, which has two circulations, pulmonary and bronchial, infarction is rare and usually causes simple stuffing with blood. Death of tissue is even rarer, which is why it is extremely uncommon to find cavitation infarcts, (except following septic emboli which do cause tissue death).

Dogma 2. That abrupt mechanical blockage of a pulmonary artery causes elevated pulmonary artery pressure, pain and dyspnoea. A simple mechanical block, e.g., caused by abruptly inflating a large balloon within a pulmonary artery, causes no symptoms and in fact the patient is unaware that you have done anything. However, if a blood clot of the same size is used to block the artery the patient will immediately experience massive symptomatology. Clearly the effects are biochemical, not mechanical.

Dogma 3. That there is no shift in ventilation away from an embolised area. This erroneous belief has led to the insistence that all perfusion scans be accompanied by a ventilation scan. The teaching being that if there is a ventilation defect associated with the perfusion defect, the defect is probably not due to an embolus. This thinking has unfortunately led to a major drop in the accuracy of interpreting perfusion scans for embolisation.

Dogma 4. That hypoxia is caused by diminished perfusion to an area, (PE), with normal ventilation to that area. Diminished or absent perfusion to a portion of the lung, with no change in ventilation certainly causes an abnormal ventilation/perfusion (V/Q) ratio but diminishing perfusion while ventilation continues cannot cause hypoxia. It simply causes wasted ventilation. To cause hypoxia the *ventilation* must be absent or diminished in relation to the amount of perfusion. How is it possible for this to occur as a result of pulmonary embolism? The answer is that all of the perfusion from the blocked areas must now be redistributed to flow through the non embolised lung. This causes a very large increase in regional perfusion, (Q), with no change, (and possibly even diminution in ven-

tilation.) The result is an abnormal V/Q ratio with insufficient oxygen for the amount of perfusion...result, hypoxia.




Dogma 5. That the pulmonary artery on the side of an embolus enlarges. It used to be taught that a large pulmonary embolus could be progressively ‘forced’ down a pulmonary artery by each systolic contraction of the right ventricle, and that this kept the artery from collapsing during ventricular diastole, causing the artery to be permanently bigger. However the opposing view is that the redistributed flow from the blocked side to the ‘good’ side causes the artery to be bigger on the non-embolised side. Careful analysis of plain films in a hundred cases of P.E., proven by angiography leads to the conclusion that it is impossible from the plain film to say which side the embolus is on. The observer does better by simply guessing!

Dogma 6. That the lung is oligemic, (on the plain film), distal to a pulmonary embolus. The ‘Westermark’ sign, (oligemia distal to a pulmonary embolus), has been shown by many observers to be a rare, (6 to 12%) phenomenon in acute P.E. Our own results have shown that even with this small percentage of allegedly oligemic areas, many of those attributed to P.E. are in fact caused by pulmonary disease, (e.g. regional emphysema and McLeod syndrome). Oligemia is however commoner in chronic P.E. The Westermark sign is of no practical value for the diagnosis of acute P.E.

Dogma 7. That right heart failure due to a massive P.E. will cause pulmonary edema. Right heart failure most certainly can occur with a large P.E. and this will cause neckvein swelling and/or ankle edema, however right heart failure does not cause pulmonary edema. Left heart failure must also be present to cause this. It has been suggested that prior heart disease must be present before heart failure will occur in P.E., but we have not, in our series of over 800 cases found this to be true.

Dogma 8. That the beneficial effects of heparin are due to clot dissolution and prevention of further clot formation. When heparin is given to a patient gravely ill from a large P.E., the relief of hypoxia is rapid, in fact it occurs long before there is any reduction in the size of the clot. We believe that the





immediate improvement in hypoxia is due to bronchodilation. It appears to be widely forgotten that heparin is an excellent bronchodilator. The bronchodilation increases ventilation in all areas, including the non-embolised areas, (with their increase in blood flow), pushing the V/Q ratio back towards the normal 1/1.

(Note, it should be stressed that this is presently a *hypothesis* which fits well with observations and appears to be reasonable, but is not yet proven.)

Further erroneous dogma will be illustrated and discussed in the tutorial.



# Diagnosis and Management of Pleural Disease

Francine L. Jacobson, MD, MPH

## Objectives

- (1) Review pleural diseases, both benign and malignant.
- (2) Evaluate CXR, CT, MRI and US in the diagnosis and management of pleural diseases.
- (3) Share interventional strategies for image guided management of pleural disease.

## Pleural Diseases

The pleural space can be involved in a variety of disease processes including trauma, infection, inflammation and malignancy. Pleural processes may be primary as in diffuse mesothelioma, localized fibrous tumor of pleura or lipoma. Pleural processes very frequently accompany other abnormalities, both local and systemic.

Pneumothorax and pleural effusion may both follow traumatic event such as a rib fracture. The development of spontaneous pneumothorax most commonly reflects the equivalent of trauma with rupture of bleb or paraseptal emphysema. Pleural effusion may be associated with trauma directly, following rib fracture accompanied by vascular injury. Pleural effusion may occur later in the course of a pneumothorax due to loss of the intimate relationship between visceral and parietal pleural surfaces or blockage of pleuro-lymphatic communication. A chylous effusion results from trauma to the thoracic duct.

Pleural effusions are frequently encountered in the setting of pneumonia. Up to 50% of patients with pneumonia will develop a parapneumonic pleural effusion. Parapneumonic effusions may progress through 3 sequential stages—exudative, fibropurulent and organizing. In the exudative phase, fluid flows from the interstitium across visceral pleura into pleural space. The fluid is sterile with a high proportion of protein, normal pH and normal glucose. It will collect in dependent portions of the pleural space and is usually treated adequately by antibiotics for underlying pneumonia. During the fibropurulent phase, PMN leukocytes and bacteria accumulate. Fibrin facilitates the development of loculations; glucose and LDH increase while pH decreases. During the organization phase, fibroblasts produce a fibrotic reaction creating resistance to respiratory motion. At this time, decortication may be required. Empyema represents actual infection in the pleural space. Associated organisms include *Streptococcus pneumoniae*, *Staphylococcus aureus*, *H. influenzae*, *Streptococcus pyogenes*, *Bacteroides*, and *Fusobacterium* species.

Pleural effusions are also associated with a wide variety of non-infectious causes of inflammation and systemic diseases. Such effusions are most frequently transudative. Specific diagnosis may depend on recognition of other clinical and imaging features of the disease. Congestive heart failure, abdominal process with ascites and collagen vascular disease are often associated with pleural effusions. Pleural effusions in these processes tend to be small to moderate in size. Patients may be asymptomatic or present with pleuritic chest pain.

Large pleural effusions, presenting with dyspnea and cough, may be associated with malignancy.

Paramalignant effusions result from lymphatic obstruction and may themselves contain no malignant cells. Malignant cells are detected in examination of fluid from 50% of malignant pleural effusions on presentation. Carcinoma of the lung and breast are the most frequent tumors to invade the pleural space. Lung cancer can produce both parapneumonic and malignant pleural effusions. Lymphoma is the most frequent cause of chylous effusion. Carcinomas of lung, breast, ovary, stomach, and lymphoma account for approximately 80% of all malignant pleural effusions. Patients with malignancy may also have other causes contributing to accumulation of pleural effusion such as congestive heart failure, hypoalbuminemia, and parapneumonic effusion accompanying pneumonia. Concomitant disease may be suspected in cases where fluid is a transudate rather than an exudate. The fluid in a malignant pleural effusion is ordinarily an exudate with protein concentration of 4 g / dL. A large tumor burden or long-standing disease may be identified by low pH and low glucose. Primary treatment of malignant pleural effusion usually involves both drainage and sclerosis to prevent recurrence.

Pleural tumors arise from cells native to the pleural space. Fat may originate in the extrapleural space as well. Lipoma may occur anywhere but is most characteristically encountered in the region of the axilla. Thin septations do not indicate malignant degeneration into liposarcoma. Liposarcoma includes a large component of soft tissue and may contain fat only in microscopic quantities.

Localized fibrous tumor of the pleura has undergone several changes in name to more accurately describe it pathologically. Originally called benign mesothelioma, it is not related to diffuse malignant mesothelioma. It is no longer called benign fibrous tumor of the pleura because it is benign only 80% of the time. In 20% of



cases, it presents with mitotic figures and local invasion that limits successful resection. These tumors can be umbilicated resulting in movement of the tumor with changes in patient position. Paraneoplastic syndromes include Hypertrophic Pulmonary Osteoarthropathy (HPO) and hypoglycemia.

Diffuse malignant pleural mesothelioma is a rare tumor that develops 20-30 years following asbestos exposure. Three other manifestations of asbestos related pleural disease are recognized: plaque formation, pleurisy, and fibrosis. Pathologically, mesothelioma is subdivided into three microscopic subtypes: epithelial, sarcomatous, and mixed histologies. Although there is no widely accepted staging system for mesothelioma, the Butchart, TNM, and Brigham staging systems have been used most commonly. Resectability of tumor is more critical than the staging system. Diagnosis may be difficult as the pattern of disease has a strong overlap with pleural carcinomatosis. Diffuse malignant pleural mesothelioma is resistant to standard modes of therapy and, if untreated, results in death 4 to 12 months from the time of diagnosis. For selected patients, an aggressive approach combining radical surgery with chemotherapy and radiotherapy has demonstrated a long-term survival advantage. Epithelial cell type along with negative resection margins and negative extrathoracic lymph nodes has the best prognosis. Five-year survival in this sub-group is currently 25-30%. Prognosis is improving with aggressive tri-modality treatment including extrapleural pneumonectomy, chemotherapy and radiation therapy.

### **Chest Radiographs (CXR)**

Chest radiographs provide the most frequent imaging examination in the setting of pneumothorax and pleural effusion. Mobility may be demonstrated with changes in patient position. Volume estimation is approximate, particularly in bedside examinations. Pleural disease can be very subtle on radiographs and even confusing. Following evaluation with CT, follow-up may be performed at a higher level using serial radiographs. Results of pleural disease such as encasement of lung are apparent on radiographs.

### **Computed Tomography (CT)**

CT can be used to assess size and loculation of pneumothorax and pleural effusion. It demonstrates pleural calcification and is diagnostic of lipoma. The transaxial plane traditionally limits use of CT for staging of mesothelioma. Multiplanar reconstruction of multidetector CT scans may increase the contribution of CT to mesothelioma staging. CT may be helpful in detecting adjacent lung disease, such as pulmonary infarction, rounded atelectasis and pneumonia. Contrast enhancement of inflammatory pleural disease may reveal "split pleura sign" in fibropurulent and organizing phases pleural effusion.

## **Magnetic Resonance Imaging (MRI)**

MRI is used to solve problems related to the nature of substances in the pleural space. Since this is frequently determined by laboratory examination of fluid, MRI is infrequently performed to evaluate pleural effusions. It is helpful for differentiation of pleural fluid from solid tumor and currently provides the primary radiologic staging information required for treatment of mesothelioma. Tumor exhibits prominent enhancement while hypointensity is characteristic of benign pleural disease.

### **Ultrasound (US)**

Ultrasound can detect the normal motion of pleura, referred to as the "glide sign". Ultrasound is suitable for evaluation of pleural effusions including loculations and is frequently used to direct pleural therapy. It is also sensitive in demonstrating tumor nodules in the setting of a pleural effusion.

### **Interventional Radiology (IR)**

Ultrasound and CT are the most frequently used modalities to guide radiologists in performing diagnostic and therapeutic procedures for patients with pleural disease. All radiologists who perform transthoracic fine needle aspiration biopsy need to be skilled in emergency evacuation of a pneumothorax. Placement of a Cook catheter with a Heimlich valve or the equivalent integrated unit and placement of drainage catheters are the most frequent pleural interventions undertaken by radiologists.

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# ABCs of Blunt Chest Trauma

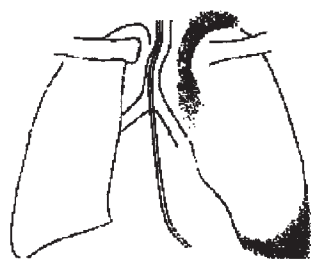
Jud W. Gurney, MD

## Objectives

1. Consider the evidence for CT angiography in aortic transection
2. Recite the ABC's used to screen the chest radiograph in patients with blunt chest trauma.
3. Identify the "P" sign of bronchial fracture
4. Identify each of the follow-up examinations if the screening ABC's are positive.

Trauma is a disease.(1,2) It is the leading cause of death in the first four decades of life and is the fourth leading cause of death overall. Motor vehicle accidents account for the majority of injuries. The portable chest is the initial and most important radiographic examination of the trauma patient. In the hectic and harried emergency room environment, rapid and accurate evaluation is important. Usually it is not the obvious lesion such as tension pneumothorax or right stem intubation that is missed but the second lesion with subtle findings, which is more life threatening. The following is a systematic approach to evaluate trauma patients. It is patterned after the ABC's of clinical management (Airway, Breathing, and Circulation) and includes all the major injuries seen in the trauma victim.

### A: Aortic Transection

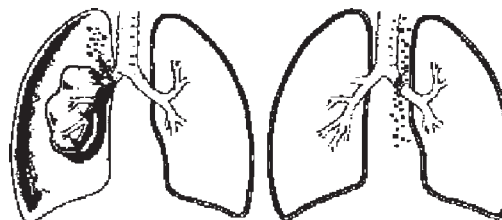


Aortic transection accounts for 16% of motor vehicle accident fatalities and is found in approximately 1% of major trauma victims. Most patients with aortic transection do not reach the hospital in time for treatment. Survivors need rapid treatment. Only 1% survive for a month untreated. Ninety percent of the transections occur at the level of the aortic isthmus. Unfortunately, chest radiographs do not image the transection directly. Only indirect signs (mediastinal widening, NG deviation, left apical cap, abnormal contour aortic arch) are evidence of possible injury.(3) Any of these signs should prompt aortography or CT angiography for definitive diagnosis.

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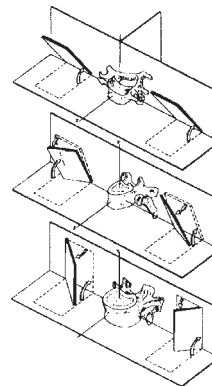
### B: Bronchial Fracture

Bronchial fractures are found in less than 1% of patients with major chest trauma.(4) Eighty percent of bronchial fractures occur within 2.5 cm of the carina. It is unusual to visualize directly the fractured



bronchus and detection requires awareness of the indirect "P" signs: persistent, progressive, pneumomediastinum and/or pneumothorax (especially if the pneumothorax is unrelieved by chest tube drainage). The fallen lung sign is specific but rare. Over 30% of bronchial fractures are initially missed and almost 50% of the fractures are detected on a delayed basis. If bronchial injury is suspected then bronchoscopy is recommended for further evaluation.

### C: Cord



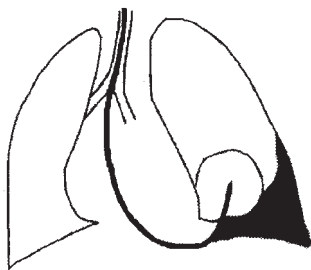
Thoracic spine fractures occur in 3% of patients with major chest trauma.(5) Only 12% of patients with thoracic spine fractures are neurologically intact. The high frequency of cord injury is due to two factors; the thoracic cord has a tenuous blood supply and the cord is large compared to the size of the spinal canal. Most commonly fractures occur at the functional

thoracolumbar junction (T9-T11), which is the region that the posterior spinal facets change from a thoracic orientation to a lumbar orientation. The signs of thoracic spine fracture may again be indirect and overlap those seen with aortic injury (paraspinal widening, mediastinal widening, left apical cap, and deviated NG tube).(6) If thoracic spine fractures are suspected, at minimal a lateral examination is indicated usually followed by CT and/or MRI.

### D: Diaphragmatic Rupture

Diaphragmatic ruptures occur in 3% - 7% of patients with blunt chest and abdominal trauma.(7) Up to 90% of these injuries occur on the left, the right hemidiaphragm being partially protected by the liver. Seventy percent of these injuries are initially missed, even though over 75% have abnormal chest radiographs. Many of these signs, however, are non-specific (elevation left hemidiaphragm, opacification





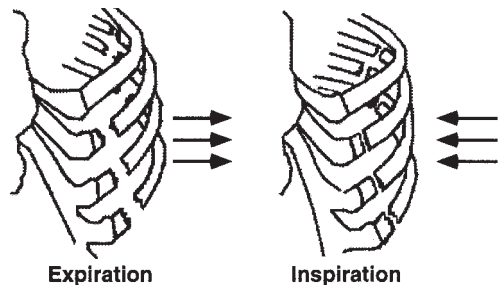
left lower lobe). For left-sided ruptures, barium examination of the stomach and colon usually suffice to make a diagnosis. Nuclear medicine liver/spleen scans have traditionally been utilized for right-sided ruptures.

Other modalities that have been successful in identifying ruptures include ultrasound, CT, and MRI.

### E: Esophageal Rupture

Esophageal ruptures occur in fewer than 1% of patients with blunt chest trauma.<sup>(1)</sup> The sudden increase in intraesophageal pressure from trauma ruptures the esophagus, the same pathophysiology that occurs in rupture from intense vomiting (Boerhave's syndrome). The ruptures usually occur in the left inferior posterolateral wall of the esophagus. Again the tear is not directly identified on chest radiographs and one must rely on indirect signs along with a high index of suspicion. These indirect signs include: pneumomediastinum, left pneumothorax, and left pleural effusion. Pneumomediastinal air along the inferior descending aorta and left cardiovertebral angle of the diaphragm (the "V" sign of Naclerio) should be a clue to esophageal tear. If this injury is suspected, an esophagram with non-ionic contrast is recommended. The morbidity and mortality from delayed treatment of this injury are high.

### F: Rib Fractures



Rib fractures occur in 60% of patients with major chest trauma.<sup>(1)</sup> The detection rate is lower, however, approximately 20%. The most important rib fractures are those that produce a flail chest. If the patient has more than five adjacent rib fractures, or if the patient has more than three segmental rib fractures (more than one fracture per rib), than flail chest should be suspected. If the rib fractures look like elephant trunks, a flail segment should be suspected (this sign is due to rotation of the segmental segments) Radiographic identification is important as this condition may not be apparent clinically. The flail chest moves paradoxically and impairs ventilation. Treatment usually requires long term mechanical ventilation with all the attendant problems inherent to prolonged mechanical ventilation.

The other important rib fracture is that of the first rib. Because this rib is protected by the clavicle, shoulder, and scapula, considerable force is required to break it. First rib fractures are a marker of severe trauma. Up to 18% of patients with aortic tears and 90% of patients with bronchial tears have first rib fractures.

### G: Gas (Pneumothorax)

Severely injured patients are often radiographed in the supine position. Small pneumothoraces may be difficult to detect. Air will accumulate in the nondependent portion of the thorax (antero inferior). A pleural line may not be evident. Subtle signs include abnormal sharpness of cardiac or mediastinal edges, deep sulcus sign, and visualization of pericardial fat tags. Upright, decubitus films or CT should be used to exclude suspected air collections, especially if the patient is being ventilated.

### H: Heart (Cardiac Injury)

Myocardial contusions are common injuries in the trauma victim and are diagnosed from blood chemistries. Rare injuries include laceration of pericardium or coronary artery, rupture of interventricular septum, or valve dislocation. Cardiac injury should be suspected with any abnormal cardiac size or shape and should also be suspected if the patient has or develops severe pulmonary edema. Again, the injuries cannot be diagnosed from the chest radiograph and when suspected require further evaluation with echocardiography, angiography, or CT.

### I: Iatrogenic (Malposition Tubes and Catheters)

Finally, malposition of the various tubes and catheters used to treat the trauma victim are common, especially in light of the haste and urgency with which they have been placed. Malposition may lead to significant morbidity and mortality. The NG tube, which is commonly placed in the field prior to arriving at the trauma center, is an aid in the diagnosis of both aortic transection and left diaphragmatic rupture.

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# Thoracic Manifestations of Lymphoma

David S. Schwartz, MD

As thoracic radiologists, there are a number of services that we provide to the patients with lymphoma and their physicians. We will examine our role in each of these functions. They include:

1. Initial evaluation of a previously undiagnosed patient
2. Providing radiographic staging of a patient with known lymphoma
3. Assessing the results of therapy and restaging the patient
4. Performing percutaneous biopsy in the patient with suspected lymphoma.

To better understand this disease, it is helpful to separate lymphoma into two main categories, as this relates to presentation, therapy, and prognosis. Hodgkin's Disease (HD) is considered distinct from non-Hodgkin's lymphoma (NHL), with the latter containing a number of subdivisions, including AIDS-related lymphoma. Clinically, the most common presentation of both HD and NHL is painless enlargement of any of the superficial lymph nodes. This may be accompanied by fever, night sweats, or weight loss. Back pain, radicular pain, and bone pain may reflect various areas of involvement.

## Hodgkin's Disease

HD represents about 1% (7000-8000) of new cancer cases each year in the United States, with two age peaks, one in the twenties and one in the fifties. Both males and females are affected, with male predominance among the younger patients. There are a number of forms of HD, with Nodular Sclerosis accounting for the majority (65-80%) of the cases. This is the one type with a female predominance, and the type most often seen with mediastinal or supraclavicular adenopathy. Other variations include Lymphocyte Predominant (5%), Mixed Cellularity (20-35%), and Lymphocyte Depletion (<5%).

HD almost always originates in a lymph node, and spreads via the lymphatics to contiguous nodes. Extranodal involvement is also by direct spread to adjacent tissues. Hematogenous dissemination may occur late in the course of the disease, especially in the lymphocyte depletion form of HD.

Peripheral lymph nodes are involved in at least 70% of patients with HD, particularly the cervical or supraclavicular regions. Anterior mediastinal involvement is typical of the nodular sclerosing form of HD. There may be spread to hilar lymph nodes, and contiguous extension into the lung parenchyma. The lung mani-

festations may include nodules, interstitial infiltrates, or appear as areas of consolidation. Pleural effusion occasionally results from impairment of lymphatic drainage. Superior vena cava syndrome is an unusual complication. Extra-thoracic disease includes the spleen, abdominal and retroperitoneal lymph nodes, and, rarely, the bone or bone marrow. Other extranodal sites are extremely rare, and put the diagnosis of HD in doubt.

Diagnosis of HD depends on the identification of Reed-Sternberg cells in a biopsy specimen with the correct cellular background.

Therapy for HD consists of radiation, chemotherapy, or a combination of the two. Surgery is utilized for staging or managing complications, such as hypersplenism or spinal cord compression. Following treatment, restaging is usually performed. This takes place one to two months after radiation, or after three or four cycles of chemotherapy. For thoracic lesions, restaging usually includes CT scans, and evaluation of these will be discussed later. Follow-up is continued on a frequent basis (every 2-3 months) for 4 years, and then every 6-12 months. Most relapses occur within the first 3-4 years, but may occasionally be seen later.

## Non-Hodgkin's Lymphoma

NHL includes a very varied group of tumors, and is much more common than HD (40,000 new cases each year). A number of etiologies have been associated with various forms of NHL. HTLV-I virus is associated with adult T-cell leukemia-lymphoma (ATLL). AIDS patients with the HIV virus develop high-grade B-cell lymphoma. Epstein-Barr virus has been implicated in Burkitt's lymphoma, organ transplant-related lymphoma, AIDS-related lymphoma, and some T-cell lymphomas. Other immunodeficiency syndromes and autoimmune disorders have also shown a relationship to NHL. Occasional HD patients develop NHL following therapy, especially after radiation. The exact relationship has not been delineated.

The Working Formulation classification of NHL includes differentiation of Low-grade, Intermediate-grade, and High-grade types. The Clinical Classification includes T-cell or B-cell status. While the Ann Arbor classification of involved sites is used in NHL, the histopathology is more critical in predicting survival: Low-grade lymphoma Rarely curable; survival ranges from <5 to 10 years Intermediate, high-grade A number of early deaths, but overall high rate of cure





Staging includes bone marrow biopsy with flow cytometry to define clonality. Diagnostic spinal tap may be performed, and UGI series is indicated if there are GI symptoms or if there is involvement of Waldeyer's ring. Restaging following therapy is important, especially with potentially curable patients.

Therapy for NHL includes radiation and chemotherapy. Experimental monoclonal antibody treatment may also be used.

### Initial Evaluation of a Previously Undiagnosed Patient

Often, the first imaging study obtained is a chest radiograph. In many cases, this appears normal, due either to absence of thoracic involvement or findings that are not apparent on plain film studies. Computed tomography of the chest usually follows, in order to find occult lesions or better delineate abnormalities already identified.

**Mediastinum and Hila**—The classic finding in HD (especially the nodular sclerosing form) is an anterior mediastinal mass. On chest radiograph, this is usually readily apparent, extending on both sides of the midline. CT examination defines the amount of involvement more accurately, and permits measurement of the mass, useful in initial staging and follow-up studies. Other nodes in the mediastinum, including the prepericardiac region, are occasionally involved. Hilar adenopathy may also occur, but almost always in the presence of mediastinal disease.

The nodal involvement in NHL is more variable, with other areas of the mediastinum frequently involved. Pulmonary parenchymal disease may be present in the absence of enlarged mediastinal or hilar lymph nodes. In both HD and NHL, low-density regions may be present within affected nodes, representing areas of necrosis. Some Hodgkin's nodes may calcify following treatment, either radiation or chemotherapy.

**Lung** — In HD, pulmonary disease almost always represents direct extension from hilar lymph nodes. This is often seen as a coarse reticular pattern branching outward from the hila. There may also be more focal areas within the lungs, with nodules or regions of air-space consolidation. When the interlobular septa are involved, Kerley lines are visible, and lymphatic obstruction may result in pleural effusion.

In NHL, pulmonary parenchymal involvement may be evident in the absence of lymphadenopathy. The abnormality may be seen as a coarse reticulo-nodular interstitial process, with this pattern more commonly associated with hilar lymph node enlargement. One or more nodules or masses may be present within the lungs. These tend to be poorly defined, and air bronchogram may be present within

them. Areas of segmental or patchy airspace consolidation are another manifestation of lymphomatous involvement. The term "Primary Pulmonary Lymphoma" is utilized to describe NHL of the lung with no evidence of lymphadenopathy or distant disease. This is usually of the small lymphocyte variety.

**Chest Wall** — Direct extension from mediastinal masses to the chest wall may occur. These involve both the soft tissue and the bone, with destruction of underlying rib or spine. There may also be isolated areas of chest wall lymphoma, which suggest an extrapleural origin. Chest wall lesions are usually identified on CT, but MRI may prove more useful in defining the exact extent of disease. Particularly following radiation, it may be difficult to distinguish residual tumor from edema or fibrosis.

### Staging

Staging Hodgkin's Disease is the most important factor in determining the prognosis and appropriate therapy, and the role of radiology is apparent in defining the areas of involvement. For staging lymphoma, we will primarily be analyzing chest radiographs or CT examinations. Rarely, MRI will be employed, usually to evaluate growth into neighboring structures or tissues. Abdominal and pelvic CT, UGI series, and CNS studies may also be requested. Lymphangiogram has largely been replaced by cross-sectional imaging modalities. The Cotswolds staging classification is now used, a modification of the original Ann Arbor system:

<u>Classification</u>	<u>Description</u>
Stage I	Involvement of a single lymph node region or lymphoid structure
Stage II	Involvement of two or more lymph node regions on the same side of the diaphragm (mediastinum=1, hilar regions are lateralized). The number of anatomical sites is indicated by a subscript (e.g., II <sub>3</sub> )
Stage III	Involvement of lymph node regions or structures on both sides of the diaphragm
Stage IV	Involvement of one or more extranodal sites in addition to a site for which "E" has been used
Designations	A No symptoms B Fever, night sweats, or weight loss X Bulky disease (mediastinal mass >1/3 the maximum diameter of the chest or a nodal mass greater than 10cm maximum diameter) E Involvement of a single extranodal site that is contiguous or proximal to a known nodal site CS Clinical stage PS Pathologic stage (Biopsy)



## Restaging

Following radiation or chemotherapy, we are often called upon to reevaluate the extent of remaining disease. Particularly in bulky disease of the mediastinum, the tumor may not completely regress, even with successful treatment. Fibrous tissue may be difficult to differentiate from residual tumor. CT scan may be of limited value in resolving this question, depending primarily on decreasing size of the mass. MRI signal characteristics may be nonspecific. Gallium-67 scintigraphy is frequently used to monitor the results of therapy, particularly with HD. It is very sensitive for residual tumor, and a positive scan usually indicates the need for further treatment (although thymic hyperplasia or infection may give false-positive results). In addition, gallium scans are excellent for identifying recurrent disease on follow-up studies. Thallium-201 is also being utilized for this problem, with the advantage that scanning can be done almost immediately after injection.

## Biopsy for Lymphoma

Another major role of the thoracic radiologist is to provide biopsy information when peripheral lymph nodes are not amenable to sampling. Often, a mediastinal mass, chest wall mass, or pulmonary parenchymal lesion is the most accessible tissue. CT-guided needle biopsy may be the best source of diagnostic material, and we are often called upon to provide tissue for the definitive diagnosis of suspected lymphoma. Sampling any of these areas is well within our capabilities, but there are certain requirements for tissue handling that must be met. The specimen should be placed in saline (not formalin) and it should not be refrigerated. A fairly large amount of tissue is required, and we try to send at least 4-5 cores.

The pathologists prepare routine histopathology slides for analysis, as this provides the most basic information about the lesion. These microscopic sections are where the Reed-Sternberg cells will be identified, an indicator of HD. Touch preps for cytology are also prepared, providing additional information.

T-cell or B-cell monoclonality can be determined by molecular diagnostic studies utilizing flow cytometry, and this also aids in the classification of NHL. A fairly large amount of tissue is required for this analysis, which determines the features of the entire population of cells. If more detailed analysis is required, DNA hybridization or other studies may be performed. These examine the nuclear composition of single cells, and also require a fairly large piece of tissue.

## Other Lymphoproliferative Disorders

**Posttransplant Lymphoproliferative Disorder (PTLD)** – The result of immunosuppression, PTLD is linked to the Epstein-Barr virus. It varies from a benign process to a B-cell NHL. This process develops in about 2% of organ transplant recipients, and is related to the suppressive therapy, particularly cyclosporine, rather than the organ involved. Radiographically, PTLD is usually manifested as single or multiple pulmonary nodules. Hilar or mediastinal adenopathy may also be present. The disease frequently responds to the cessation of immunosuppression therapy.

**Lymphocytic Interstitial Pneumonitis (LIP)** – LIP is characterized by interstitial infiltration with lymphocytes and plasma cells. There may be nodular aggregations as well. The disease is associated with AIDS, Sjogren's syndrome, and a number of autoimmune disorders. Reticular, nodular or reticulonodular opacities may be present on chest radiographs, and airspace consolidation may also be seen. Lymphadenopathy is rare, but pleural effusions are seen.

**Pseudolymphoma** – This rare disease may appear as pulmonary nodules or infiltrates. Histologically, it is similar to lymphoma, but the cells are polyclonal, rather than monoclonal. The patients are usually asymptomatic, with the incidental finding of a pulmonary nodule or mass on chest radiograph. The diagnosis is frequently made after surgical removal, and the patients usually develop no other abnormalities.

**Mycosis Fungoides** – This is a T-cell NHL, originating in the skin. Spread to multiple sites may occur, with nodules or infiltrates in the lungs.

**Lymphomatoid Granulomatosis** – Pulmonary lesions of this disease are nodules (which may cavitate) or irregular infiltrates. Histologically, there are lymphocytes, plasma cells, histiocytes and atypical lymphoreticular cells. There also may be involvement of the kidneys, skin or CNS. Lymphomatoid granulomatosis is angiocentric, with vascular occlusion. The microscopic findings are those of both a vasculitis and lymphoproliferative disorder. Most patients are middle-aged, with males affected twice as often as females. The prognosis is poor.

**Waldenstrom's Macroglobulinemia** – Patchy cellular infiltrates with sheets of lymphocytes result in reticulonodular changes in the lungs, either focal or diffuse. Focal consolidations may also occur, and pleural effusion is not uncommon. Lung parenchyma is often destroyed in this disease. An atypical immunoglobulin, similar to that seen in multiple myeloma, is the characteristic feature. There are usually constitutional symptoms as well as symptoms related to

multi-system involvement. This is a disease of adults aged 50 or older, and it has a male preponderance.

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# The Small Solitary Pulmonary Nodule

*David F. Yankelevitz, MD*

The small solitary pulmonary nodule continues to be source of controversy despite being one of the most commonly detected radiographic abnormalities. While the incidence of new chest x-ray detected solitary pulmonary nodules is estimated to be approximately 150,000/year in the United States, the incidence of small solitary pulmonary nodules seen using CT scanning is not known.

The solitary pulmonary nodule is defined by two words, solitary and nodule. As for the latter, it is usually considered to be less than 3 cm in diameter and according to most, surrounded by aerated lung. Lesions larger than 3 cm are usually described as masses, as their prevalence of malignancy is quite high. As for the solitary aspect, traditional definitions were derived from radiographic images and allowed for only one nodule, as 2 or more which were visible on radiographs had different clinical implications. With CT scanning, additional very small nodules may be evident, and the concept of the dominant nodule must be considered. With a view towards the small solitary pulmonary nodule, we now must define the concept of small. Using CT as the standard, the lower limit for detection is probably around 2-3 mm, however, there currently is no strict definition for what is considered small.

Whenever a solitary nodule, or small solitary nodule is detected the primary concern is to exclude malignancy, preferably without having to resort to major surgery for diagnostic purposes. Thus, the ultimate goal of the diagnostic evaluation is to establish the probability of malignancy and make management decisions accordingly. With renewed interest in CT screening for lung cancer along with the increasing availability of CT scanners, there is an increasing need to develop appropriate protocols for diagnosis of small nodules.

The use of decision theoretic approaches has progressed since the time Ledley and Lusted first introduced Bayes Theorem to medicine in 1959. Variations using likelihood ratios have been advocated. More recently multivariate techniques such as logistic regression and even neural nets have been proposed. All of these decision approaches attempt to combine clinical features about the patient along with radiographic descriptions of the nodule. These are then jointly used to estimate the probability of malignancy.

Radiographic assessment, including CT, is still the key for evaluating solitary pulmonary nodules. Since

the 1950's it has been recognized that only two features are sufficiently predictive of malignancy to exclude further evaluation. These include calcification in a benign pattern and stability in size for 2 years. With the advent of CT, recognition of fat deposits in a nodule is also acceptable in confirming a benign diagnosis. Other features used to describe nodule morphology such as shape and edge characteristics are not sufficiently predictive to be able to exclude malignancy. There has recently been renewed interest in developing computer aided image processing techniques to further describe morphologic features of small pulmonary nodules, however it is uncertain how useful these will ultimately become.

An area of great promise using CT is the assessment of contrast enhancement. A recent multicenter study has confirmed a difference in enhancement between benign and malignant nodules. It has been shown that malignant nodules show a greater degree of enhancement due to their increased vascularity. Analysis of time density curves of the nodule enhancement may even provide further information than the single measure of the maximum amount of enhancement. The role of this technique particularly for small nodules is yet to be defined.

Transthoracic needle biopsy is a very useful technique, as this can provide for pathologic diagnosis. In nodules greater than 1.0 cm the yield is quite high, with some investigators reporting a sensitivity for obtaining diagnostic material in patients with a malignancy of 95%. However, with nodules less than 1.0 cm the accuracy of this procedure rapidly declines. This primarily relates to being able to accurately position the needle within the nodule. Without careful documentation of needle tip location within the nodule, acceptance of a benign or non-specific diagnosis is not recommended. The use of cutting needles or core biopsy is quite useful especially when considering the possibility of a benign diagnosis.

The role of nuclear medicine has been rapidly expanding. PET FDG has shown great promise in distinguishing benign from malignant nodules. Although PET scanners are not widely available, a technique known as coincidence scanning, which uses FDG and has images obtained on a modified dual headed gamma camera may provide analogous information. One of the principle limitations of PET scanning is the difficulty in characterizing nodules less than 1.0 cm. This primarily relates to technical as-

pects of the imaging device and not necessarily to lack of uptake of radioactivity by the nodule. Nevertheless, it can pose a significant problem. There has also been the development of new imaging compounds, including radiolabeled peptides. These have primarily been useful in the investigation of carcinoid tumors, but certain classes have shown an affinity for adenocarcinomas.

Finally, the development of video assisted thoracoscopy (VAT's) allows for definitive diagnosis in a less invasive manner than open thoracotomy. Wire localization techniques are similar to that performed in breast biopsy, can be applied in conjunction with VAT's to help localize small deep nodules.

Thus while at first glance, the small solitary pulmonary nodule seems like an entity which should be relatively easy to diagnose, this is not the case. The choice of diagnostic procedures is large and dependent on many factors. It may be that no single approach will be optimal and may even be different for different institutions where availability and expertise

are different. Additionally, a lung cancer is considered one of the most lethal forms of cancer, a reasoned approach is imperative as the consequences of a wrong diagnosis can be quite severe.

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# The Radiologic and Pathologic Spectrum of Bronchopulmonary Carcinoids

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## Objectives

Following this workshop, the audience will:

1. Be able to describe the radiologic appearance of bronchopulmonary carcinoids.
2. Know the histologic differences between typical and atypical pulmonary carcinoids.
3. Know the prognostic difference between typical and atypical carcinoids.

Bronchopulmonary carcinoids are uncommon pulmonary malignancies representing 1-2% of all lung tumors. They are neuro-endocrine tumors, felt to arise from a pluripotential stem cell. Although they present with a common clinical and radiologic pattern, they have a spectrum of pathologic patterns which show varying degrees of malignancy ranging from the low grade typical carcinoid, to the intermediate grade atypical carcinoid, to the higher grade large cell neuroendocrine tumor and small cell carcinoma.

## Clinical Presentation

Bronchopulmonary carcinoids present in a wide age range, predominantly in the third to seventh decades. The average age at presentation is 45.5 years, younger than the usual age for bronchogenic carcinoma. Patients with atypical carcinoids tend to present at an older age than do those with typical carcinoids. The male:female ratio is nearly 1:1. Whites are affected more than blacks and there is no known association with cigarette smoking.

Up to 50% of the patients, typically those with peripheral tumors, are asymptomatic. The others, especially those with central tumors, present with a variety of symptoms typically caused by endobronchial obstruction. Thus, recurrent pneumonia or atelectasis and wheezing in one lung are common. Although the tumors usually have an intact overlying mucosa, they are very vascular and hemoptysis is common. Sputum cytology is rarely useful in the diagnosis.

Paraneoplastic syndromes are uncommon. The carcinoid syndrome, which includes flushing, diarrhea, abdominal cramping, wheezing and cardiac disease, almost always occurs in patients with liver metastasis and is uncommon with thoracic carcinoids. When it does occur, it is more frequent in patients

with typical carcinoids. Up to 2% of patients can present with Cushing's syndrome due to production of ectopic ACTH or ACTH releasing hormone by the tumor. Indeed, bronchial carcinoids are the most common source of ectopic ACTH production.

## Radiologic Presentation

There is no difference in the radiologic presentation of typical and atypical carcinoid tumors. Carcinoids occur both centrally (85% of tumors arise in the main or, most commonly, the lobar bronchi) and in the periphery of the lung. Most carcinoids are central and present as a hilar or perihilar mass. On chest x-ray, the mass is smooth or lobulated and may have a notched contour. A spiculated mass, typical of bronchogenic carcinoma, is rarely seen. Because of the endobronchial component of the mass, there is often associated atelectasis, post-obstructive consolidation, or mucoid impaction. Peripheral tumors are also sharply demarcated, usually ovoid nodules surrounded by aerated lung.

On CT, calcification or ossification can be seen in nearly 40% of the central lesions. Peripheral tumors, which are more likely to represent atypical carcinoid, are less likely to be calcified. Most tumors are round. When they are ovoid, they tend to be oriented with their long axis parallel to the nearest bronchus or pulmonary artery. Because of their vascularity, carcinoids usually show marked enhancement following injection of contrast material. Many carcinoids are both endobronchial and extrabronchial; in fact, the endobronchial component may be very small relative to the extrabronchial component. CT allows visualization of the entire lesion and of its effects (e.g. mucus plugging, atelectasis or post-obstructive consolidation).

Carcinoids typically have a large number of somatostatin binding sites. <sup>111</sup>Indium octreotide, a somatostatin analog, can be used for scintigraphic localization both of the primary tumor and lymph node metastases, and can be useful in detecting functionally active small tumors not visible on CT.

Metastatic disease occurs more frequently in atypical carcinoids than in typical carcinoids (46.4% vs. 23.1%) (4). The most frequent sites of metastases



are to lymph nodes and liver. Metastases may also involve bone, lung and adrenals.

### **Pathology**

Typical carcinoids are composed of uniform cells in a rich fibrovascular stroma. There are fewer than two mitoses per 10 high power fields and there may be dystrophic calcification or ossification. They are highly vascular. Electron microscopy reveals neurosecretory granules which are more common and larger than in atypical tumors.

Atypical carcinoids comprise approximately 10% of all carcinoids and demonstrate more active mitoses (2-19 per 10 high power fields). There may be architectural distortion and tumor necrosis. Although both typical and atypical carcinoids metastasize to regional lymph nodes and may invade blood or lymph vessels, atypical tumors do so more frequently and more aggressively.

Typical and atypical carcinoids cannot be distinguished by their gross anatomical appearance.

### **Treatment and Prognosis**

The treatment for the patient with any type of carcinoid is surgical excision. The prognosis of patients

with typical carcinoid is excellent with studies showing 84-95% ten-year survival for typical carcinoids and 44-60% ten-year survival for atypical tumors. Tumor size larger than 3 cm. and nodal metastases adversely affect survival.

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# A Chest Radiologist's Approach to Abdominal CT in Chest Diseases

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## Learning Objectives

1. To enable the chest radiologist to understand the technique of abdominal CTs.
2. To learn to recognize common pitfalls and approach to incidental findings in the abdomen found at the time of thorax CTs.
3. The learner will use the information to critically appraise their CT technique and perhaps implement changes.
4. The learner will heighten their sensitivity to the diseases that occur in the abdomen in association with the diseases of the chest.

This workshop is intended as an overview of the utility of abdominal CT and the diagnosis and management of chest diseases. Too often as subspecialist radiologists focus only on the thorax, potentially de-emphasizing the patient as a whole. Ironically most disease processes that occur in the thorax are systemic diseases.

All chest CT's include portions of the abdomen because of the concavity of the diaphragm, in many cases this is only portions of the liver and spleen and perhaps portions of the adrenal glands, stomach and pancreas. Findings may be incidental or coincidental to a disease process in the chest. In either case, the CT radiologist must note the findings and manage the patients with the same vigor as the thorax findings.

This workshop will be divided into two parts. The first part will focus on technique, contrast pharmacokinetics in abdominal organs, and pitfalls commonly seen on chest CT's. In addition, incidental findings will also be highlighted. Newer techniques of scanning using multi-detector CT's will be highlighted. The second portion will focus on abdominal manifestations of a few common thorax related diseases, which may occur when the abdomen is scanned for diagnostic purposes.

## Technique

Prescription of CT's will be tailored primarily to the disease process and clinical implications. Contrast rates and patterns will vary significantly by both institutional bias and vendor specific applications of various CT scanners. Helical technique is no longer cutting edge, but rather the standard of care. There is little role for combined thorax and abdomen scanning

on axial scanners except for high resolution CT and perhaps limited unenhanced adrenals. Technique for these studies is of utmost importance. This involves good positioning, appropriate technique (MA, KV) one breath-hold, optimal collimation, good oral contrast, good venous opacification (dependent on rate, volume and delays), appropriate reconstruction algorithms and slice profiles all taken in conjunction with methodical interpretation.

When scanning chest, abdomen at the same, the following should apply. As a general rule, minimum collimation should be 8 mm. or less. On multi-slice CT because of the ability to scan faster, we have gone routinely to 5 mm. slices. A 50% overlap is ideal. Pitch should be adjusted to be no higher than 1.3-1.5 on single detector scanners. Any increase beyond this level results in significant broadening of the slice profile. In the situation of limited breath hold, clustered back to back spiral examinations could be performed. Venous opacification should include appropriate delays for the chest (20-30 sec.) at least 2 cc/sec., and delays at the top of the liver. The liver should be scanned at 60 sec. when injecting a 3 cc/sec. or greater, whereas 70 sec. when injecting at slower rates than less than 3 cc/sec.

This delay between chest and abdomen spiral acquisitions allows for a repeat breath-hold prior to the commencement the liver scanning. Volumes of contrast vary dependent on institutional bias, but probably a minimum of 100 cc. when liver scanning is to be performed. A good rule of thumb is good opacification of both portal vein and hepatic venous system with a minimum of 40 HU rise from unenhanced state (ideally greater than 50 HU). Many factors play a role in the quality of the CT of the liver including venous access, cardiac output, patient positioning, and disease states. Oral contrast is used routinely in our institution when abdominal CT is performed. Oral contrast is safe, cheap, and can be one of the radiologist's greatest friends.

One thousand cc. starting at least 60 min. prior and finishing at least one cup just prior to CT commencement is ideal. When pelvic CT is to be performed at the same time, oral contrast administration should be begin 75-90 min. prior to the study. Any commercially available oral contrast will suffice.



Scanning of the abdomen should go to the iliac crest and when the pelvis is specifically scanned and scanning should be continued through the ischial spines. Delays between the abdomen and pelvic acquisitions are unnecessary but can be done if iliac venous opacification is desired, or tube heating is an issue.

If hard copy images are desired, maximum 20 on one film using an optimal window and level setting which may be the same as the mediastinal window settings. Liver windows obtained with narrow window and slightly higher levels can be also filmed. Techniques may vary from routine, for example when the chest is scanned because of a specific abdominal investigations, perhaps arterial phase livers might be required which would change the timing of the chest CT. When vascular pathology is in question, also single dynamic runs during arterial phase through the chest and abdomen may be required.

The liver is the absolute critical organ and the hardest organ to scan optimally in the body. The liver should be emphasized when setting up protocols, and liver should always be optimized. The only time one would want to sacrifice appropriate portal venous phase imaging of the liver is when vascular pathology is in question such as aortic dissection. Now with multi-slice CT, it is possible to obtain full run aortic dissections from the top of the arch to the ischial spine, and then following an appropriate delay, portal venous phase imaging of the liver could be performed.

## Contrast Kinetics

There still remains much controversy on the optimal scan parameters of the liver except for certain rules:

- a) Rapid injection is ideal.
- b) Scanning the liver during peak enhancement before the onset equilibrium.
- c) Degree of liver enhancement is related to the volume of contrast administered.

What remains controversial is when peak enhancement of the liver occurs, how to predict when peak enhancement is, whether contrast should be injected uniphasic or biphasic, low flow or high flow states, and how much enhancement is needed for lesion detection. At the same time, the chest radiologist must realize that we want to optimize the chest CT at the same time.

To understand the controversy, one must realize that there are three phases of liver enhancement:

- 1) Bolus phase
- 2) Non-equilibrium phase
- 3) Equilibrium phase.

Bolus phase occurs during peak and increasing aortic enhancement and thus occurs in the first 50-60 sec.

Non-equilibrium phase occurs when there is increasing hepatic venous opacification and lasts anywhere up to 110-120 sec. Equilibrium phase occurs when the aortic and hepatic enhancement curves become parallel. The liver must be scanned during non-equilibrium phase, because during contrast equilibration later on, lesion detection becomes diminished. Also, ideally one would like to scan the liver during peak enhancement (ideally greater than 50 HU above baseline). Some authors have suggested using "bolus tracking" method available on many vendor's scanners, to predict peak enhancement. Unfortunately, this is not a viable option during combined chest abdomen scanning, because these monitoring phases occur during the ideal time period in which to scan the thorax. Although many chest CT's can be performed unenhanced, abdominal CT's cannot. Therefore, even though the indication for the chest may be unenhanced, enhancement becomes "free", when the abdomen is being scanned. This will almost never cause a problem in the thorax; although if it does invoke a potential area of concern, then delayed imaging during late equilibrium phase could also be obtained through the area of concern.

## Other Organ Pharmacokinetics

### Spleen

The ideal time to scan the spleen is not well documented but ideally when splenic pathology is a concern, then minimum portal venous phase imaging is the best time. As a result, if the study timing is optimized for the liver, the spleen rarely is an issue.

### Kidneys

Renal imaging can be at times confusing, but again the minimal standard would be approximately 80 sec. delay when primary renal pathology is not being sought. If there is a concern of a primary surgical renal disease, then unenhanced kidney should also be obtained prior to commencement of the study.

### Adrenals

Although enhancement of adrenal can be helpful in the differential diagnosis of adrenal masses, often contrast is not necessary. Our standard abdominal CT is only to do enhanced studies of the abdomen except when staging bronchogenic carcinoma. In this situation we obtain, unenhanced thin sections of the adrenals just prior to contrast injection. Alternatively a delayed 15 or 30 min. can be helpful (see below under bronchogenic carcinoma).

### Aorta

Aortic pathology is one area where liver opacification should be sacrificed. Ideally, higher pitch breath-hold non-paused helical acquisitions are ideal. Peak arterial enhancement with rapid 3 cc/sec. or greater



rates is appropriate. Common indications include staging longitudinal extent of aortic dissection or aneurysms. When three-dimensional imaging is to be done, it is imperative that no pauses be built for fear of respiratory misregistration artifact.

## Incidental Lesions

### Liver

The three most common focal lesions seen incidentally in the liver are cysts, hemangiomas and bile duct hamartomas.

Cysts are seen in 2.5-7% of the population. CT criteria for a cyst include homogeneous low attenuation indiscernible wall, no enhancement.

Hemangiomas are seen in 0.4%-7.3% of the population. CT shows well-demarcated lesions with thick globular peripheral enhancement and of "vascular" attenuation. This finding alone on a single pass CT is very specific for hemangioma. The sensitivity of this sign may be fairly high also and should be sought in all liver lesions.

Bile Duct Hamartoma occurs in 0.2-3% of the pathology specimens. They appear as non-specific low attenuation liver lesions less than 1 cm. in size.

Other liver abnormalities seen commonly include diffuse changes of cirrhosis, focal or diffuse fatty changes. Cirrhosis shows a lobular contour with lobar redistribution and secondary changes of portal hypertension.

### Spleen

Lesions include granuloma, cysts (false, epidermoid and hydatid), hemangioma, lymphangiomas and hamartomas. Most focal lesions in the spleen are non-specific but fortunately benign. Other diseases include infarcts, lymphoma, metastases and primary tumors. Diffuse disease may cause splenomegaly.

### Adrenal

There is a plethora in the literature concerning the incidentally found adrenal masses. The work-up will address both the patient with and without lung cancer. When a lesion is found incidentally in the adrenal, size, density, and enhancement are all important characteristics to study. Specific attention to the adrenal incidentaloma will be provided in the workshop.

Common chest diseases that have abdominal findings:

(Some of these will be illustrated in the workshop)

1. Congenital—all congenital lesions generally have a higher incidence of other associated anomalies and should be sought on chest diseases. These may be as simple as an aberrant arterial supply to a sequestered lung segment towards complex vascular and situs problems. Congenitally ac-

quired disease such as cystic fibrosis or Kartageners disease may also have abdominal findings.

2. Infections- such as TB, fungal and illnesses related to AIDS may have findings in the liver spleen or peritoneum. At times the findings in the thorax may be non-specific but when combined with the abdominal findings will lend higher level of specificity.
3. Immunologic- such as the vasculitides and scleroderma often has abdominal findings.
4. Neoplastic- it is imperative that the chest radiologist be able to differentiate benign from malignant disease when staging bronchogenic carcinoma. The liver and adrenal sites are common locations for diseases to spread. Other disease because of their systemic nature often involves both sides of the diaphragm.
5. Granulomatous- diseases such as sarcoidosis commonly have abdominal findings in the liver, spleen, and adrenals.
6. Trauma
7. Metabolic- anemias, Gauchers, Amyloid, Gauchers etc.
8. Vascular- aortic dissections, and aneurysms
9. Idiopathic- LAM, Tuberos sclerososis, Neurofibromatosis, Castleman's
10. Drugs- Amiodarone

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# Thoracic Sarcoidosis

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## Objectives:

The objectives of this workshop include:

1. showing the common and less common imaging features of thoracic sarcoidosis;
2. discussing the most important differential diagnoses of the different patterns of involvement;
3. and emphasizing that sarcoidosis is a combined clinical, radiological and pathological diagnosis.

Sarcoidosis is a clinical, radiological and pathological disease associated with the presence of noncaseating granulomata in lymph nodes and other tissues. The pathology is not pathognomonic and may be seen with tuberculous and fungal infection, exposure to beryllium, and in lymph nodes draining malignant diseases. Therefore, the presence of noncaseating granulomata pathologically requires close correlation with clinical and radiological findings for a diagnosis to be made.

The radiology of thoracic sarcoidosis is quite variable and one must be aware of the common and more unusual presentations and particularly radiological signs which would mitigate against a diagnosis of sarcoidosis. The common radiological signs include bilateral paratracheal and hilar lymphadenopathy with or without diffuse interstitial reticulo-nodular pulmonary opacities.

The main differential diagnosis of the lymphadenopathy includes malignant diseases such as lymphoma and metastatic carcinoma, and infectious diseases such as tuberculosis and fungal infection. The lymphadenopathy in these diseases tends to be asymmetrical and regional and is also accompanied by significant clinical findings. Occasionally a sarcoid reaction will develop in thoracic lymph nodes following treatment of certain neoplasms. Tissue biopsy may be required to rule out recurrent tumor.

The main differential diagnosis of the interstitial lung disease includes conditions which affect the mid and upper lung zones predominantly. Langerhans cell histiocytosis, extrinsic allergic alveolitis, and lymphangitic carcinomatosis can look similar. It is very unusual for interstitial sarcoidosis to predominantly affect the lower lung zones and therefore idiopathic pulmonary fibrosis, collagen vascular disease and asbestosis are not usually a diagnostic problem. Sili-

cosis may have similar interstitial and lymph node features but clinical history usually resolves the problem.

Unusual radiological findings include bony lesions; pleural lesions including pleural effusion, chylothorax, pneumothorax, pleural thickening, and pleural apical cap; mediastinal lesions including isolated middle and anterior mediastinal lymphadenopathy, posterior mediastinal lymphadenopathy, calcification in lymph nodes (egg shell), mediastinal emphysema, and tracheal involvement; hilar lesions including unilateral hilar lymphadenopathy; lung lesions including bronchostenosis with lobar atelectasis, post-obstructive bronchiectasis, cavitary parenchymal lesions, mycetomata, "alveolar" sarcoid, and "vanishing lung"; unilateral parenchymal lesions including alveolar, interstitial, and solitary pulmonary nodule; vascular lesions including pulmonary arterial hypertension, superior vena caval obstruction, pulmonary artery obstruction.

"Alveolar" sarcoidosis usually presents as multiple focal ill-defined areas of consolidation often occurring in a patient with few if any symptoms. The differential diagnosis would include acute processes such as pneumonia and more prolonged air space processes such as bronchoalveolar carcinoma, lymphoma, exogenous lipid pneumonitis, and chronic infection. Occasionally the air space process is peripherally situated in the lung and the differential diagnosis then includes eosinophilic pneumonia, bronchiolitis obliterans organizing pneumonia, and thromboembolic disease. The presence of lymphadenopathy would favour sarcoidosis.

Areas of mass-like conglomeration are not uncommon in sarcoidosis. I stress the site of these lesions in the perihilar, mid and upper lung zones often extending posterior to the hila. The main differential diagnosis of conglomerate disease includes silicosis, granulomatous infection such as tuberculosis, and intravenous talc granulomatosis. Calcifications are commonly present in the conglomerate opacities and also in mediastinal lymph nodes, sometimes in an eggshell distribution. Eggshell calcification is most commonly seen in silicosis but can occur in sarcoidosis.

Uncommonly, sarcoidosis can cause bullae within the lungs. These bullae are usually in the mid and



upper lung zones. The differential diagnosis would include bullous emphysema, and post-infectious pneumatoceles. These bullae may harbour mycetomas. Sarcoidosis is the second commonest cause of mycetoma formation after tuberculosis. The mycetomas may be suggested by the development of progressive apical pleural thickening.

Thoracic CT and high resolution CT have added more information regarding the distribution of lymphadenopathy within the chest and the character of the parenchymal lung abnormality. CT improves sensitivity for the detection of all types of lesions. The high resolution CT findings include: nodules which occur predominantly in a perilymphatic distribution along bronchovascular bundles, adjacent to interlobular septa, and subpleurally, including the fissural pleura; ground-glass attenuation; "air space opacities" often in a peripheral distribution; reticular opacities; distortion of lung architecture; cystic air spaces; conglomerate areas of fibrosis resembling progressive massive fibrosis; traction bronchiectasis; air-trapping on expiratory CT; and mycetoma formation. Predominant sites of lesions are in upper and mid-lung zones and often perihilar.

Necrotizing sarcoid granulomatosis should be regarded as a variant of classical sarcoidosis in which there is a vasculitis affecting the arteries and veins with either a round cell infiltration of the wall or a granulomatous vasculitis. Pathologically, the granulomas tend to be more ill-defined and confluent than in classical sarcoidosis and exhibit variable amounts of necrosis. Radiologically, opacities or nodules may be seen.

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# Neuroendocrine Tumors of the Thorax

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## Objectives

This presentation will review the demographic, pathologic, and radiologic features of thoracic neuroendocrine tumors. At the completion of this workshop, attendees will be understand the current classification of thoracic neuroendocrine tumors and be aware of their pathologic and radiologic features.

## Introduction

Neuroendocrine (NE) differentiation is a relatively common histologic finding in pulmonary neoplasms. Some cases present with a clinical hormone syndrome; others reveal a neuroendocrine nature by their morphologic or immunohistochemical characteristics.

Thoracic tumors with NE features include those involving the lung (typical bronchial carcinoid, atypical bronchial carcinoid, large cell NE carcinoma, small cell carcinoma) and thymus (thymic carcinoid). Thoracic NE neoplasms have ultrastructural and immunohistochemical features that resemble NE cells found in normal lung. Their neuroendocrine properties include the ability to synthesize, store and secrete chemical messenger substances such as neuroamines and neuropeptides.

In the normal lung, NE cells occur as solitary cells along the basement membrane of the bronchial and bronchiolar epithelium. They also occur as nodular cell clusters called neuroepithelial bodies and form part of the diffuse neuroendocrine system, a concept introduced by Feyrter in 1938. Despite extensive research efforts using electron microscopy, immunohistochemical studies and molecular probes for analyses of gene expression, the physiologic roles of NE cells in pulmonary physiology remain unclear. Various concepts of pulmonary NE cell hyperplasia, dysplasia, and neoplasia have been proposed.

Some investigators propose that NE tumors derive from normal NE cells; others hypothesize that they arise from pluripotent stem cells which differentiate with neuroendocrine features. The classification and terminology of these tumors has generated considerable discussion and some confusion. Pulmonary NE lung tumors are often considered within a spectrum of differentiation, from the least malignant typical carcinoid, which follows a relatively indolent clinical course, to the most aggressive and malignant small cell carcinoma.

## Carcinoid Tumors

The majority of carcinoid tumors occur in the gastrointestinal tract (90%). The respiratory tract is the next most common site of involvement; carcinoids occur less commonly in the thymus.

## Typical Bronchial Carcinoid

Typical bronchial carcinoids are uncommon neoplasms, representing 1% - 2% of all lung tumors. They affect females slightly more often than males (52:48 in one large series), with an average age at presentation of 45.5 years. Affected patients may be asymptomatic or present with symptoms related to bronchial obstruction. Systemic hormonal manifestations are rare, even though hormonal products can be demonstrated within cellular neurosecretory granules in all carcinoids. Approximately 2% of cases of bronchial carcinoids develop Cushing syndrome. Carcinoid syndrome is less commonly seen, usually in patients with carcinoid metastases to the liver.

Macroscopically, bronchial carcinoids are well-defined nodules or masses that may be lobulated. They often involve central airways, but may be peripheral and subpleural. Tumors may grow within the bronchial lumen exclusively but invasion through the bronchial wall is common. Often, the bulk of tumor is extraluminal, with a smaller intraluminal component – a so-called “iceberg” tumor. Central tumors may partially or completely occlude the bronchial lumen and produce distal atelectasis and pneumonitis.

Radiographically, bronchial carcinoids manifest most frequently as a well-defined hilar or perihilar mass, with or without distal parenchymal disease, and without lobar predilection. In some cases, the tumor is obscured by distal effects of the tumor (atelectasis, obstructive pneumonitis, recurrent pneumonia, and mucoid impaction). CT demonstrates the well-defined tumor nodule or mass and often shows its bronchial relationship. Approximately 30% of bronchial carcinoids exhibit calcification that may be punctate or diffuse. Marked homogeneous contrast enhancement occurs frequently, reflecting the rich vascular stroma of bronchial carcinoids, and may help to differentiate the central enhancing carcinoid from adjacent atelectatic or consolidated lung. CT may demonstrate regional lymph node enlargement which may represent metastases or hyperplasia resulting from associated recurrent or chronic distal infection.



Carcinoids have numerous high-affinity, somatostatin-binding sites which may be useful in detecting occult tumors producing ACTH-dependent Cushing syndrome. Octreotide, a somatostatin analogue, has been used for scintigraphic localization of carcinoids.

The treatment for bronchial carcinoids is complete surgical excision. Patients may require lobectomy, bilobectomy, or pneumonectomy because of long-standing distal obstructive changes in the parenchyma.

### Atypical Bronchial Carcinoid

In 1972, Arrigoni and colleagues recognized the more malignant character of atypical bronchial carcinoids. Atypical carcinoids share some morphologic features of typical carcinoids but are distinguished by the presence of nuclear pleomorphism, increased cellularity and mitotic activity, and areas of coagulative necrosis. They affect patients over a wide age range, but patients with atypical lesions are, on average, ten years older than those with typical carcinoid neoplasms.

Atypical carcinoids are indistinguishable from typical carcinoids by their imaging features and by their gross pathologic appearance. Atypical lesions are larger, on average, than typical carcinoids and metastasize more frequently to regional lymph nodes.

### Large-Cell Neuroendocrine Carcinoma

Large-cell neuroendocrine carcinoma (LCNEC) affects patients with a median age of 59 years (range, 35 to 75 years). The tumor is highly associated with cigarette smoking. Microscopically, LCNEC is characterized by large cells with a neuroendocrine appearance by light microscopy, a high mitotic rate (higher than seen in atypical carcinoid), necrosis, and neuroendocrine features by immunohistochemistry. They are commonly peripheral tumors greater than 3 cm in diameter. In the recent WHO International Histological Classification of Tumors, LCNEC is considered a variant of large cell carcinoma.

### Small Cell Carcinoma

Small cell carcinoma (SCC) is the most aggressive pulmonary neuroendocrine tumor. Although commonly discussed as a type of bronchogenic carcinoma, it is also categorized as a neuroendocrine tumor of the lung. SCC is strongly associated with cigarette smoking and typically affects males more often than females. All SCC are high grade malignant lesions. Microscopically, SCC is characterized by small, uniform oval cells with scant cytoplasm. Most are located centrally within lobar or main stem bronchi. The tumor grows rapidly with early and widespread metastases. SCC typically manifests radiographically as a hilar or perihilar mass, often with associated mediastinal widening. The tumor may be obscured by hilar or medias-

tinal lymphadenopathy, by distal atelectasis or post-obstructive pneumonitis. SCC is the most common primary pulmonary malignancy causing superior vena cava obstruction. Approximately 10% of SCC are located peripherally and are usually associated with spread to hilar or mediastinal lymph nodes.

While neuroendocrine differentiation is a hallmark of SCC, it is not found in every case. SCC may cause a clinical hormone syndrome by secreting ectopic hormones. The most commonly seen syndromes are Cushing syndrome and inappropriate secretion of antidiuretic hormone.

### Thymic Carcinoid

Thymic carcinoids are rare primary malignant neuroendocrine neoplasms that are histologically similar to atypical bronchial carcinoids. Necrosis and local invasion are common histologic findings. In general, thymic carcinoids are more aggressive than their bronchial counterparts.

Affected patients have a mean age of 53 years, but tumors occur across a wide age range with a male:female ratio of 3:1. Most patients are symptomatic (chest pain, cough, weakness, pulmonary infection). Approximately 20% of patients present with a clinical hormone syndrome (MEN-Type 1 or 2, Cushing, hypercalcemia).

Radiologically, thymic carcinoid manifests as an anterior mediastinal mass that mimics thymoma. Tumors are characteristically well-defined, lobulated, and heterogeneous on cross-sectional imaging. Lymphadenopathy is present in 30% of cases. Evidence of local invasion and mass effect are present in 69% of cases. Pleura effusion is uncommon.

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# Tracheobronchial Stenting Techniques

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## Tracheobronchial Stenting

Stenting may be performed in both benign and malignant strictures of upper and lower airways that are unsuitable for surgical reconstruction. The general condition of the patient may preclude open reconstruction due to the presence of co-morbidity, recent thoracic surgery, or limited lifespan. Certain characteristics of the stricture may prohibit reconstructive surgery: these include active disease, ongoing active airway inflammation, extensive length or multifocality, as well as poor underlying quality of the residual airway. Strictures of the trachea are most commonly due to prior prolonged intubation or neoplasm. Less common causes include radiation stenosis, polychondritis, tracheomalacia, and in the pediatric age group extrinsic strictures secondary to vascular malformations.

Prior to the advent of lung transplantation as an accepted therapy for end stage lung disease, bronchial stenoses were almost invariably due to underlying cancer, and were usually amenable to surgical resection or dilatation. Stenting was reserved for cases of advanced malignancy in whom the patient's overall condition precluded reconstructive surgery. Bronchial stenosis is a relatively common complication of lung transplantation, occurring in single lung, double lung, and heart-lung transplant recipients. The complication is felt to occur secondary to the lack of bronchial artery supply with resulting airway ischemia. These ischemic stenoses occur at the bronchial suture line and in the more distal airway, and have been reported to occur in approximately 10% of patients undergoing transplantation.

## Stent Types

Two main types of airway stents are available: Silicone based devices and metallic stents. Both bare and covered metallic prostheses are available.

### Silicone Stents

A number of silicone based devices are in widespread use in both benign and malignant tracheobronchial strictures. Straight short tubes as well as bifurcated Y-shaped devices are available. The straight stents are flanged on both ends to prevent dislodgement, and can remain in place in patients for extended periods. The selection of the correct size and length is critical. The stent must be long enough to enable its flanges to anchor the stent within the

stricture; short enough to avoid compromise of a lobar bronchus distally or the trachea proximally; and of satisfactory diameter to maintain the caliber of the airway. Early versions were prone to dislodgement and in 1990 Dumon introduced a Silastic stent with four rows of studs oriented at 90 degree angles on the external surface to prevent migration.

The main advantage of silicone based stents is that they are easily removed. They can therefore be placed while a patient is in respiratory difficulty and removed either when the patient's ventilatory status has recovered sufficiently, or when reconstructive surgery is possible. Unlike currently available metallic stents it is possible to obtain devices which can accommodate a bifurcation, such as the carina. When placed in an appropriate fashion a single device can be used to maintain patency of the distal trachea and both mainstem bronchi. At the present time a metallic device which will perform this function is not widely available, however, a system is undergoing evaluation in Europe.

Silicone based (Silastic) stents do have some disadvantages. Stenotic airways need to be predilated prior to stent insertion, whereas metallic stents can be placed within a narrow airway lumen and subsequently dilated. The relatively thick wall of the stents results in a smaller internal diameter than that achieved by metallic devices, and the stents frequently become occluded with mucus plugs. In addition granulation tissue and tumor overgrowth develop at the ends of the stent, therefore regular bronchoscopic examination and treatment are necessary to keep the airway clear. Great care must be taken in placing these devices at airway branch points as they will occlude side branches. Also of note, in general silicone stents must be placed under general anesthesia because of the need for rigid bronchoscopic instrumentation.

### Metallic Stents

A variety of metallic stents are now on the market, of which the Gianturco Z-stent (Cook Inc., Bloomington, In.) was the first to obtain FDA approval for use in the tracheobronchial tree. The Wallstent prosthesis (Schneider Inc., Minneapolis, Mn.) and the Palmaz stent (Johnson and Johnson, Warren, N.J.) have also been utilized extensively. A large number of newer devices, mostly nitinol based, are also available and are becoming more popular.



The main advantages of metallic stents are the ease of insertion of low profile systems, and the thin low surface area of the devices. The procedure can be performed using flexible bronchoscopy in the interventional room, under deep sedation. The bare metal stents have an extremely thin wall which rapidly becomes embedded in the airway, and have large gaps in the wall which allows for normal ciliary function and reduced mucus impaction.

The main disadvantage of metallic stents is the inability to remove or reposition these devices once deployed. Stents become firmly embedded in the wall of the airway and become incorporated in the epithelium in less than 6 weeks. Removal has been reported using pincers to grip the wall of the stent, and then applying a twisting motion to pull the stent away from the wall. Potential complications from this maneuver are catastrophic and in our experience once these devices are placed they are permanent. Another problem associated with metallic stents is the development of granulation tissue either at the ends of the stents or through the interstices. This requires careful follow-up by repeat bronchoscopy and may require subsequent procedures such as bronchoplasty, restenting and laser tissue ablation.

Gianturco Z-stents are constructed in a single or tandem arrangement of cylindrically fashioned steel wires. Outward-protruding hooks are incorporated into the wall of the stent to inhibit migration following deployment. The device is introduced in a sheath, over-the-wire, and released when in position. The device is self-expanding, and therefore exerts a continuous force on the airway wall, which has been reported to cause airway erosion. Stent fracture and migration has also been reported with a frequency of up to 30%. The relatively high incidence of complications with this device has decreased its popularity in this application.

The Wallstent, also a self-expanding device, is deployed in a similar fashion. The stent has less intrinsic radial force, and has not been associated with long-term erosive complications. The main advantages of this stent are the availability of long, large diameter devices, which makes it suitable for use in tracheal strictures, and the flexibility of the device which conforms to curved structures such as distal airways. The main disadvantage is an inherent function of the deployment system. The Wallstent foreshortens substantially from both distal and proximal ends when released from its sheath which makes precise positioning difficult.

The Palmaz stent is a balloon expandable prosthesis which is crimped onto a appropriate balloon and dilated when in position. The device has a higher radial strength which makes it more suitable than the

Wallstent to maintain patency of strictures with a high degree of elastic recoil. The stents are somewhat shorter and less flexible than the Wallstents and can be positioned very accurately. This is beneficial in the treatment of those cases with short segment lesions adjacent to branch airways.

Because of the flexibility and low-profile of the deployment systems used for the metal stents it is feasible to place these devices under conscious sedation without the need for general anesthesia. Our practice is to utilize general anesthesia for tracheal stent placement in order to reduce patient motion due to the coughing stimulus that occurs with tracheal irritation. Most bronchial interventions can be performed without full anesthesia.

## Preprocedure Evaluation

All patients have extensive preprocedural workup including objective measurements of oxygen saturation and pulmonary function.

## Imaging Studies

Plain radiographs can be useful in patient follow-up to ensure device stability, however, they have little role to play in the initial workup. Computed tomography using thin-section contiguous scans and three-dimensional reconstructions are extremely helpful in the planning of interventions in patients with strictures, particularly when the trachea is involved. It is possible to measure the expected normal diameter of the airway, the length of the region involved, and the relationship of the lesion to the vocal cords superiorly and the carina inferiorly. In our institution spiral CT scans using 3 mm collimation of the airways from the tracheal cartilage to the distal mainstem bronchi are performed before and after stenting. In patients clinically suspected of having tracheomalacia, scans are performed at maximal inspiration and maximal expiration in order to unmask subtle areas of narrowing exacerbated by the increased intrathoracic pressure on inspiration.

## Bronchoscopy

Most patients will have had bronchoscopy prior to consideration for endoluminal treatment, however, in certain cases the initial scope is performed at the time of intervention.

In general a flexible bronchoscope is used for insertion of metallic stents and for balloon dilatation of pre-existing stents. Rigid bronchoscopy is required for the insertion of silicone devices.

## Anesthesia

All patients under consideration for the placement of a tracheal-bronchial stent should undergo a complete examination of their airway. This includes the



state of the unaffected airways as well as broncoscopic evaluation of the stricture, including the length, distance below the cords, and relationship to airway branch points. Endoscopic evaluation of the airway must be performed in an anesthetized spontaneously breathing patient. Muscular paralysis while advantageous for vigorous dilatation and stent placement, must not be instituted until a detailed examination is completed and the operator is certain a airway can be subsequently maintained. Vocal cord paralysis, which can mimic tracheal stenosis, can be obscured by instituting muscular paralysis too early. An endotracheal tube is placed in most patients, either by the anesthesiologist in cases of general anesthesia, or by the bronchoscopist in cases utilizing deep sedation. This allows for easy airway maintenance and repeated bronchoscopic evaluation. General anesthesia is performed by our colleagues using propafol and muscle relaxants; sedation is instituted by a bronchoscopy nurse using midazolam and morphine sulphate.

## Insertion Technique

### Silicone Stents

Silicone devices have low inherent radial force, and strictures should be dilated before stenting. Rigid bronchoscopy is necessary to allow dilation and subsequent stent placement. Dilatation can be performed using the Holinger bronchoscope which is insinuated into the stricture and advanced with a corkscrew motion. Gum-tipped Jackson dilators can also be used as can various angioplasty balloons.

In patients with tracheal stomas a T-tube stent can be inserted either via the stoma or the mouth. This extends up to the vocal cords and down as far as the carina. In patients without tracheal stomas either Y-tubes or straight short stents are inserted in a similar fashion. The stent is mounted on the rigid scope, the scope advanced across the stricture, and then withdrawn, leaving the stent in place. A biopsy forceps is then used to advance a limb of the Y-tube into the other bronchus. Placement of stents above the carina in this fashion is relatively straightforward, and the stents can be removed and repositioned until a satisfactory result is achieved. With more distal bronchial stents, the operators vision is somewhat obscured and deployment is more difficult.

Because silicone stents are not anchored to the airway they are prone to movement. Overdilation of a stricture prior to stent placement or the use of an undersized stent may prevent the waist of the stricture holding the stent in place. Inaccurate placement as well as stent dislodgement can lead to lobar obstruction. In patients with benign disease most operators will perform follow-up bronchoscopy at 6 months, and remove the stent. The airway is in-

spected and if patency maintained the stent need not be reinserted.

## Insertion of Metallic Stents

### Tracheal Stenoses

When tracheal stenting is planned the pre-procedural imaging is reviewed and determinations of the various lengths and optimal diameters obtained. The patients is then positioned supine on the fluoroscopy table. General anesthesia is administered and the anesthesiologist performs a visual assessment of the upper airway and vocal cords. At this stage the patient is paralyzed and intubated. Care has to be taken when performing endotracheal (ET) intubation in these patients as the stricture is often close to the vocal cords, and either intubation will be difficult to achieve, or the tube may be placed distal to the stricture. Optimally the ET tube is positioned approximately 1 cm above the lesion. Flexible bronchoscopy of the distal airways is then performed through the stricture, the distal extent of the stricture relative to the carina identified and the position marked with a radiopaque marker on the patients skin. At this stage a guidewire with a soft tip is advanced through either the scope or the ET tube into the distal airway. A pre-stent dilatation can be performed or in most cases the lesion primarily stented.

In the rare case where the stricture is too narrow and tight to allow passage of a small flexible bronchoscope a guidewire is passed and the lesion stented based on the reconstructed CT images. The air column in the trachea and proximal bronchi can be useful in positioning the distal end of the stent above the carina. In certain cases two guidewires can be used to identify the position of the carina, by passing a wire into each mainstem bronchus.

The length, large diameter and flexibility of the Wallstent make it the device of choice for tracheal stenting. A downside of the stent is the degree of shortening on deployment. Because of this the device is opened in one or other proximal mainstem bronchi and withdrawn until in position in the distal trachea. Using fluoroscopy the stent is positioned across the stricture with the proximal extent below the vocal cords, and the distal extent above the carina. The overall length of the stent used depends on the pre-procedural imaging studies and the bronchoscopic assessment. Most stents used are 4-8 cm in length. The mean diameter of an adult trachea varies with size and sex, but is approximately 14-16 mm in diameter.

The Wallstent used can be either uncovered or covered by a polyethylene membrane. In benign strictures an uncovered device is used, whereas in neoplastic strictures a covered stent deployed in an attempt to reduce tumor ingrowth.



### **Bronchial Stenoses**

Depending on operator preference either deep sedation or general anesthesia is employed for this procedure. Either way the patient undergoes airway assessment and intubation. The scope is then used to identify the stricture(s) and the position marked on the skin. Most bronchial stenoses in our practice are due to post-transplantation ischemia, and are short focal lesions. A guidewire is passed through the stricture and into the distal airway. In most cases a balloon angioplasty is performed both to more accurately identify the position of the stricture and to determine the optimal diameter of the airway. The waist of the inflating balloon assists in marking the center of the stricture, and the result of balloon angioplasty as seen on repeat bronchoscopy allows for accurate determination of the diameter of the bronchial lumen. In most cases repeat bronchoscopy identifies some improvement in bronchial lumen, and may occasionally obviate the need for stenting.

Assuming a degree of residual stenosis a Palmaz stent is crimped onto a balloon and deployed across the stenosis. The length of stent used is determined by the bronchoscopic appearance of the stricture and the balloon waist. The diameter of balloon used to deploy the stent is taken from the previous angioplasty. For the mainstem bronchi the stent is usually dilated to 10 mm, and to 8 mm in more distal airways.

In patients undergoing palliative treatment for bronchial strictures due to underlying neoplasia the lesions are usually longer and may involve branch airways. In these cases a longer stent such as a Wallstent can be used, and the branch airways covered, but not necessarily occluded by the stent. If maintenance of branch patency is of critical importance due to compromised respiratory status, a wire can be passed through the interstices of a deployed stent, and a balloon dilatation can be performed to open a channel into the branch bronchus. In benign strictures attempts are made to prevent covering of a branch airway by the stent. Airways are marked on the skin during bronchoscopy and a guidewire can be placed in an airway to mark its location. Bronchography can also be performed using either water-soluble or lipid-soluble contrast to mark airway anatomy

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# Radiologic Evaluation of the Diaphragm

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In addition to radiography, fluoroscopy, and injection of positive and negative (gas) contrast media into the peritoneal or pleural cavity, there are newer modalities that are available for better radiologic evaluation of the diaphragm. As we discuss the radiologic approach, special emphasis will be placed upon a specific modality in the evaluation of specific problems of the diaphragm.

## The Level of the Diaphragm

The left hemidiaphragm is one-half of an intercostal space lower than the right hemidiaphragm in 89% of normal population [1]. This is due to depression by the cardiac apex. Because the right-sided cardiac apex with dextrocardia depresses the right hemidiaphragm, the right hemidiaphragm is lower than the left hemidiaphragm irrespective of abdominal situs. In patients with levocardia and abdominal situs inversus, the left hemidiaphragm is lower than the right hemidiaphragm despite the position of the liver under the left hemidiaphragm.

## The Distance between the Gastric Air Bubble and the Left Hemidiaphragm

The distance between the top of the gastric air bubble and the top of the dome of the left hemidiaphragm is less than 1 cm in about 88% of the normal population on the upright posteroanterior radiograph of the chest. However, the true distance must be evaluated on the upright lateral radiograph of the chest because the two anatomic structures may not be in the same anatomic plane. Gastric fundus must be reasonably distended with gas for accurate assessment of the distance. Eventration away from the gastric fundus may easily cause a false increase of the distance.

## Functional Evaluation of the Diaphragm

Inspiratory and expiratory radiographs are the simplest methods to evaluate normal and abnormal movement of the diaphragm. The paralyzed hemidiaphragm and severe unilateral eventration may be detected because of abnormal movement. Without deep inspiratory effort, abnormal movement of the

diaphragm may not be evident. Continuous positive airway pressure can easily push down the paralyzed hemidiaphragm to a normal level [2]. We must be aware of these two pitfalls. Fluoroscopy is less often used because of ultrasonography that can easily detect normal and abnormal movement of the diaphragm in infants and children. Ultrasonography can also be performed at the bed side [3]. CT fluoroscopy and cine MRI may be useful with further technical advancement in the near future.

## Leaky Diaphragm

When the peritoneal sac is grossly distended with fluid or gas for a prolonged period of time, the diaphragm can become leaky because of an “acquired” defect permeable to fluid or air between the peritoneal cavity and the pleural cavity [4]. Hydrothorax caused by ascites is usually on the right side, or is usually larger in amount on the right side than on the left side because the transdiaphragmatic lymphatic vessels are more abundant on the right side than on the left side (5).

## Acute Rupture of the Diaphragm

Acute rupture of the diaphragm was detected in 16 (5.2%) of 307 patients with multiple injuries who were dead on arrival (6). In a recent series of 25 cases of traumatic diaphragmatic injury, penetrating trauma was the cause in 15 cases and blunt trauma in 10 cases (7). The sensitivity and specificity of helical CT in the diagnosis of diaphragmatic trauma in a swine model were 92% and 87% respectively for sagittal reconstruction images (8). Although CT is more readily available and convenient in an acute trauma setting, MRI can be used for uncertain cases after CT or nonacute selected cases. In a recent series, MRI confirmed acute rupture of the diaphragm in all seven patients and demonstrated an intact diaphragm in all nine patients (9).

## Diaphragmatic and Juxtadiaphragmatic Lesions

MRI and helical CT are commonly used. MRI has advantage over helical CT because of direct multiplanar imaging ability, better tissue characterization, and no use of radiation. I prefer MRI to helical CT or

ultrasonography because MRI depicts the origin and extent of the lesions better in most cases.

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# Notes