Communications to the Editor

Communications for this section will be published as space and priorities permit. The comments should not exceed 350 words in length, with a maximum of five references; one figure or table can be printed. Exceptions may occur under particular circumstances. Contributions may include comments on articles published in this periodical, or they may be reports of unique educational character. Please include a cover letter with a complete list of authors (including first and last names and highest degree), corresponding author's address, phone number, fax number, and email address (if applicable). Specific permission to publish should be cited in the cover letter or appended as a postscript. CHEST reserves the right to edit letters for length and clarity.

Spontaneous Regression of Lung Metastases

Possible BOOP Connection?

To the Editor:

In our practice in an inner city hospital over the last 2 years, we diagnosed three cases of bronchiolitis obliterans with organizing pneumonia (BOOP). The brief case reports are as follows:

Case 1: A 51-year-old man with history of hypertension and Hodgkin’s lymphoma was receiving radiotherapy and mechlorethamine, vincristine, procarbazine, and prednisone (MOPP) chemotherapy regimen. He had received three cycles of MOPP when he presented to us with a history of weight loss and low grade fever. Chest radiograph showed left lower lobe infiltrate. He was started on antibiotics for suspected pneumonia. Despite a prolonged course of antibiotics, the patient continued to have low grade fever. Bronchoscopy biopsy results confirmed a diagnosis of BOOP.

Case 2: A 41-year-old man with acute myeloid leukemia and a history of recent bone marrow transplant presented to our hospital with supraventricular tachycardia. He was treated for dysrhythmia, and his chest radiograph showed left lower lobe infiltrate which did not respond to antibiotics. Bronchoscopy biopsy results led to a diagnosis of BOOP.

Case 3: A 70-year-old woman with known asthma, presented with cough. Chest radiograph revealed left lower lobe infiltrate. After antibiotics, repeat radiographs showed waxing and waning of the pulmonary lesions. Needle biopsy results were suspicious for malignancy. However, open lung biopsy results led to a diagnosis of BOOP.

Although not a new disease, the diagnosis of BOOP was popularized by an article by Epler and colleagues in 1985. The diversity of radiologic and clinical presentations of BOOP has prompted consideration of BOOP in patients with persisting radiologic abnormality. This, and better awareness of this entity, could be responsible for the increased number of patients diagnosed with BOOP in recent years.

Idiopathic BOOP affects men and women equally, usually in the 40 to 60 year age range. Some patients may give history of toxic fume or mineral dust exposure, infection (viral, mycobacterial, and Legionella), bone marrow or heart lung transplant, rheumatoid arthritis, or connective tissue disorders. Cordier and associates described different radiologic presentations of BOOP, including patchy migrating pneumatic foci, diffuse interstitial lung disease, and solitary foci of pneumonia resected because of concern over malignancy. Histologically, bronchi may have mild mononuclear cell infiltrate with foci of goblet cell metaplasia and metaplastic cuboid epithelium. Pulmonary lesions of BOOP may have a waxing and waning character and sometimes may show spontaneous regression. Needle aspiration biopsy is limited in its ability to prove a diagnosis of BOOP; therefore, open lung biopsy or bronchoscopy biopsy is the mainstay of the diagnosis.

Due to the patients’ age, comorbid conditions, and characteristic radiologic findings, we had entertained the possibility of malignancy in all three of these patients. Indeed, in Case 3, CT-guided fine needle aspiration biopsy was read as “suspicious for malignancy.” We recognized that BOOP has frequently been confused with malignancy and vice versa. In addition, the medical literature has focused in the past on the phenomenon of spontaneous regression of pulmonary metastatic lesions, which was reported for the first time by Bumpus in 1928. Since then, there have been periodic reports of spontaneous regression of lung metastases from the renal cell carcinoma. But Holland observed in 1973 that histologic proof that the lung lesions, reported regressed, were indeed metastatic renal cancer was obtained in only 6 of the 37 patients. Kavoussi and colleagues, in a case report of metastatic renal cell cancer and review of the literature, stated that only 20% of reported cases of spontaneous regression of renal cell cancer had histologic documentation. Since 1990, there have been only nine reported cases of spontaneous regression of renal cell cancer that have met the criteria of spontaneous regression. Of these nine, only five had biopsy of the metastatic lesions. Various mechanisms, including psychogenic mechanisms, have been suggested as a possible basis for spontaneous regression of cancer.

BOOP can present radiologic-like multiple or single nonresolving lung nodules. It occurs more frequently in older patients where malignancy will always be a possibility, especially for those patients with lesions on the chest radiograph that resemble malignant metastatic lesions. These lesions may wax or wane or even spontaneously regress. Since BOOP became well known only after 1985, we hypothesize that some of the earlier reports of spontaneous regression of pulmonary metastatic lesions likely represented the BOOP syndrome, particularly in cases where the metastatic lesions that regressed later were never proven histologically.

Therefore, we performed a MEDLINE search with words “spontaneous regression” and “metastasis” and found 1,550 papers in the literature before 1985 and only 900 papers after 1985. We chose 1985 as a cut-off date because the scientific community became better aware of BOOP after the publication by Epler and coworkers.

We observed that over the recent times, reports of spontaneous regression of lung metastasis have markedly decreased, likely due to the following three reasons: (1) better awareness of BOOP along with the awareness that BOOP can radiologically mimic pulmonary metastasis; (2) awareness of the limitation of fine needle aspiration cytology for the diagnosis of BOOP; and (3)
emphasis on obtaining tissue for definitive diagnosis of lung metastasis or BOOP. Spontaneous regression has been reported in other malignancies (eg, melanoma), but the purpose of this letter is to suggest a possible explanation for the reports of spontaneous regression of lung metastasis, especially in “pre-BOOP awareness” era.

Keshav Chander, MD
Alton Ochsner Medical Foundation
New Orleans, LA

Correspondence to: Keshav Chander, MD, Department of Cardiology, Alton Ochsner Medical Foundation, 1514 Jefferson Highway, New Orleans, LA 70121; e-mail: thebngle@hotmail.com

REFERENCES
4 Bumpus HC. The apparent disappearance of pulmonary metastasis in a case of hypernephroma following nephrectomy. J Urol 1928; 20:185–191

Clinical Significance of Cough as a Defense Mechanism or a Symptom in Elderly Patients With Aspiration and Diffuse Aspiration Bronchiolitis

To the Editor

Although cough is the most common symptom for adult patients seeking medical attention from primary care physicians in the United States and is associated with deterioration in patients’ quality of life,1 the concise and distinct guideline is not available. Thus, the recent consensus report, “Managing Cough as a Defense Mechanism and as a Symptom”2 is very helpful and meaningful for chest physicians. However, age-related changes in cough reflex and the protective roles of cough as the defense mechanism of aspiration in older patients are not argued in the report. Owing to the increasing number of the aged population, many pulmonologists and geriatricians recognized that silent aspiration might be very important for the pathogenesis of aspiration pneumonia and nosocomial pneumonia in older patients.3–9 Because pneumonia is in principle prevented by the defense mechanisms, such as upper airway reflexes, mucociliary clearance, and phagocytosis by alveolar macrophages, age-dependent declines of upper airway reflexes may be one of the pathophysiologic features of aspiration pneumonia in older sub-

Shinji Teramoto, MD, FCCP
Takeshi Matsuse, MD
Yasuyoshi Ouchi, MD
Department of Geriatric Medicine
Tokyo University Hospital, Japan

Correspondence to: Shinji Teramoto, MD, FCCP, Department of Geriatrics, University of Tokyo, 7-3-1 Hongo Bunkyo-ku, Tokyo University Hospital 113–8655, Japan

REFERENCES
5 Teramoto S, Matsuse T, Ouchi Y. Foreign body aspiration into the lower airways may not be unusual in older adults [letter]. Chest 1998; 116:1733–1734

To the Editor:

Because of space constraints, the Consensus Panel Report of the American College of Chest Physicians on “Managing Cough as a Defense Mechanism and as a Symptom,”1 while comprehensive, could not be as exhaustively complete as the panel members would have liked. Consequently, although (1) the protective role of cough as a defense mechanism against aspiration, (2) acute and
chronic cough caused by a variety of aspiration syndromes in adults and children, and (3) the importance of having a high index of suspicion for aspiration in the elderly patient with cough because the classic signs and symptoms of aspiration may be minimal or nonexistent. Issues are mentioned in multiple places throughout the document, Drs. Teramoto, Matsuse, and Ouchi have correctly noted that the age-related changes in the cough reflex were not specifically mentioned. I thank them for doing so.

Given the opportunity to expand upon the discussion of aspiration and taking the lead of Drs. Teramoto, Matsuse, and Ouchi, I believe the possibility that an aspiration syndrome is causing any pulmonary problem should be considered in every patient, but especially in elderly, debilitated, or sedated patients with unexplained deterioration in pulmonary status and in any patient presenting with a potential aspiration syndrome. While a compromised cough reflex places patients at risk for an aspiration syndrome, I believe that one should not solely consider respiratory defense mechanisms against aspiration. The situation is much more complicated. For instance, there are other nonrespiratory, airway-protective mechanisms that also may be dysfunctional and play an equal or more important role.

Shaker has divided airway protective mechanisms into the following two categories: (1) protective mechanisms against anterograde aspiration (ie, aspiration during swallowing); and (2) protective mechanisms against retrograde aspiration (ie, aspiration during reflux of gastric contents). In managing our patients with aspiration syndromes, I believe that it is important to consider and assess the adequacy of mechanisms against deglutitive aspiration and to consider that airway protective mechanisms against gastroesophageal, esophagopharyngeal, and pharyngolaryngeal reflux may be inadequate. In regard to the latter, it appears, based upon experimental studies in human volunteers, that these mechanisms are multiple and involve delicate interaction between upper gastrointestinal and upper respiratory tracts.

Shaker divides airway protective mechanisms against retrograde aspiration into the following two groups: (1) basal mechanisms that include competence of lower and upper esophageal sphincters; and (2) response mechanisms that include secondary esophageal peristalsis, esophaugoophagopharyngeal sphincter contractile reflex, esophagohemoral closure reflex, pharyngo-upper esophageal contractile reflex, pharyngolaryngeal adduction (closure) reflex, and pharyngeal (secondary) swallow. The roles and relative importance of these protective mechanisms in patients with aspiration syndromes await future investigation.

I also believe that it is important to stress that clinically significant aspiration can be entirely silent, including the absence of cough. This issue has been prospectively assessed using videofluoroscopic tapes of modified barium swallow procedures in two studies in stable patients receiving long-term mechanical ventilation via tracheostomy. Both studies showed that bedside evaluations to exclude swallowing dysfunction were insensitive and should only be counted on as a screening procedure to detect gross disturbances. In one of the studies, aspiration occurred in 50% of patients and was silent in 77% of these. Choking, coughing, or respiratory distress occurred in the minority of patients who aspirated.

While the gag reflex is assessed by many to predict the adequacy of swallowing and mental alertness, theoretical considerations, and the relatively few studies do not support this practice. I do not believe that it should be assumed that testing for a gag reflex helps assess the risk of aspiration during swallowing for the following reasons: (1) the stimuli and the neuromuscular processes involved in gagging and swallowing are different; (2) many normal individuals who do not have a gag reflex can swallow normally; and (3) I am aware of no studies that show the presence or absence of a gag can predict adequacy of swallowing. Moreover, in a small study comparing obtunded patients and alert medical staff members, it was determined that the gag reflex poorly and unreliably predicted the level of consciousness.

---

**Table 1—The Spectrum of Aspiration Syndromes**

<table>
<thead>
<tr>
<th>Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute respiratory distress syndrome (Mendelson’s syndrome)</td>
</tr>
<tr>
<td>“Asthma”</td>
</tr>
<tr>
<td>Bacterial pneumonia and lung abscess</td>
</tr>
<tr>
<td>Bronchiectasis</td>
</tr>
<tr>
<td>Bronchorrhea</td>
</tr>
<tr>
<td>Café coronary</td>
</tr>
<tr>
<td>Chronic interstitial fibrosis</td>
</tr>
<tr>
<td>Cough</td>
</tr>
<tr>
<td>Diffuse aspiration bronchiolitis</td>
</tr>
<tr>
<td>Exacerbations of COPD (when frequent)</td>
</tr>
<tr>
<td>Exogenous lipoid pneumonia</td>
</tr>
<tr>
<td>Foreign body aspiration</td>
</tr>
<tr>
<td><em>Mycobacterium fortuitum or chelonae</em> pneumonia</td>
</tr>
<tr>
<td>Recurrent pneumonia</td>
</tr>
</tbody>
</table>

* This listing is not meant to be all inclusive.

---

Richard S. Irwin, MD, FCCP
Division of Pulmonary, Allergy, and Critical Care Medicine
University of Massachusetts Medical School
Worcester, MA

Correspondence to: Richard S. Irwin, MD, FCCP, Department of Medicine, University of Massachusetts Medical School, 53 Lake Avenue North, Worcester, MA 01655-0330

REFERENCES

Mortality of Untreated Deep Vein Thrombosis Following Knee Arthroscopy

To the Editor:

It was recently stated that the mortality of untreated deep venous thrombosis (DVT) is between 1 and 5%; because of this, anticoagulant therapy for this disorder is obligatory in all cases.\(^1\) Mortality is a fraction that requires a denominator (the incidence), and for DVT, this figure has been mostly unknown. To avoid generalizations, it is instructive to examine the problem in a specific context. Demers et al\(^2\) have recently provided a very accurate figure for the incidence of venographically proven DVT following knee arthroscopy. They found DVT in 17.9% of cases postoperatively. 4.9% of which represented proximal vein thrombosis. These data are concordant with the findings of another prospective study which, using compression ultrasound, found a 3.5% incidence of proximal DVT following the same type of surgery.\(^3\) According to Demers et al\(^2\), 20,000 arthroscopies are performed in Canada each year. Consequently, there must be approximately 980 cases of proximal DVT each year following such surgery, most of which are unrecognized and therefore untreated.

The incidence of such thrombosis is considerable, but what is the clinical significance? A study of 10,262 such procedures determined an overall complication rate of 1.68%, 6.9% of which were clinically recognized thromboembolic disease.\(^4\) This represents an incidence of recognized thromboembolic disease of approximately 0.0012%. There are a few reports of fatal pulmonary air embolism following arthroscopy, but compared to the incidence of proximal DVT, the mortality from pulmonary thromboembolism appears to be unmeasurably small.\(^5\) By comparison, the mortality associated with the treatment of thromboembolic disease with 3 months of warfarin is approximately 1 in 1,000, with a 1% incidence of major hemorrhage; the use of IV heparin in this situation causes approximately 5 in 1,000 deaths and major bleeding in 5% of cases.\(^6\)

This is not to say that proximal DVT is unimportant. In the setting of diminished cardiopulmonary function, it may be life-threatening. But for proximal DVT following arthroscopic surgery, this is clearly not the case. In this case, the risks associated with treatment appear to be greater than those of the underlying condition.

Paul Egermayer, MBChB
Research Fellow
Christchurch Hospital
Christchurch, New Zealand

Correspondence to: Paul Egermayer, MBChB, Hagley Building, Christchurch Hospital, Private Bag 4710, Christchurch, New Zealand

REFERENCES


Continuous IV Sedation and Prolonged Mechanical Ventilation

To the Editor:

In the article published in the August 1998 issue of CHEST, Kollef and colleagues\(^1\) came to the conclusion that continuous IV sedation is associated with prolongation of mechanical ventilation. We do not agree with that conclusion.

As can be seen from the baseline characteristics of the study groups (Table 1 of their article),\(^1\) the continuous IV sedation group had significantly more patients with acute lung injury or ARDS, and their PaO\(_2\)/FiO\(_2\) was significantly lower than the noncontinuous sedation group. The authors do not present data on the total amount of sedatives given in each group, but they show that (Table 2 of their article)\(^2\) despite receiving continuous IV sedation (72.1% are on lorazepam), the continuous group received additional IV bolus sedation in significantly higher percentage, and in the form of a relatively longer-acting sedative (lorazepam). In their linear regression model, the authors do not control for the degree of lung injury nor for the amount of the long-acting sedative that was administered; both factors affect the duration of mechanical ventilation.

We conclude that the observed differences in the duration of mechanical ventilation may be entirely related to the fact that the continuous IV sedation group had worse lung injury and received more long-acting sedatives than the bolus IV sedation groups and may have nothing to do with the way sedatives were administered.

Tacla A. Sfeir, MD
Tihomir Stefanec, MD
Fellows Critical Care Medicine
Saint Vincents Hospital and Medical Center
New York, NY

Correspondence to: Tacla A. Sfeir, MD, New York Medical College, St. Vincents Hospital and Medical Center, Coleman 1050 East, 153 West 11th Street, New York, NY 10011

REFERENCE


To the Editor:

Drs. Sfeir and Stefanec raises the point that we did not control adequately for the degree of lung injury in our linear regression model, which determined that the use of continuous IV sedation was associated with prolongation of mechanical ventilation. I agree with this conclusion. It is extremely difficult in a cohort study to prove causality between a potential risk factor and the outcome of interest. Such studies are primarily used to generate hypotheses or other questions for future investigation. Because of the results we obtained in this study, we subsequently performed a randomized controlled study examining the influence of protocolized sedation for the management of patients with acute respiratory failure. The main goal of this study was to determine
Role of Chest Pain in Aortic Dissection

Is It Enough for a Predictive Diagnosis?

To the Editor:

We read with interest the pattern that Rosman and colleagues (September 1998) have developed to take histories in patients with aortic dissection (AD). They have hypothesized that the collection of anamnestic data could contribute to a more accurate diagnosis. Moreover, they have scored some features of the typical “ripping and tearing” chest pain as quality, radiation, and sudden intensity at onset to compare, in a retrospective study, effective diagnosis and initial clinical suspicion.

This interesting and debated topic has attracted our attention due to the high number of patients with AD that we have managed in the last 5 years (176 cases between October 1993 and August 1998). This huge number of cases with a clinical or imaging diagnosis (CT scan, MRI, angiography, transthoracic or transesophageal echocardiography), which is often confirmed by either surgical inspection or autopsy examination, has contributed to a general increase in our experience both in diagnosis and management. More than 12% of these patients (Table 1) have been sent to our attention with an abnormal clinical scenario (mild dizziness or chest discomfort, lower limb paresthesia, hoarseness, transient ischemic attack, etc) or symptoms more suggestive of myocardial ischemia. Most of these patients did not complain about sudden chest pain, but described symptoms slowly worsening in the previous days. The contribution of technology in all of these situations has addressed our therapy and allowed us to save more people.

This is the main reason why we do not feel completely at ease with their hypothesis. It is hard to judge a clinical emergency, like AD, only by familiar signs and symptoms. Many vascular areas can be involved by progression of the intimal tear and the false lumen along the aortic and major vessel walls can show symptoms that might easily mislead a physician.

We agree both with the general importance of the initial history in raising a clinical suspicion and with the diffuse tendency to quickly turn to an imaging diagnosis instead of trusting our deductive instincts. Nevertheless, due to the high mortality rate and unpredictability of this disease, we strongly believe in the interaction between physicians and machines. We do not feel comfortable in treating patients, medically or surgically, without a clear definition of this entity. A quick scanning of the patient when possible, even if risky, allows a more accurate knowledge of extension and prognosis.

The result of AD treatment is often a question of time; it does not allow a thorough analysis of the patient history and it obligates the physician to apply the right procedure in a few hours. Although it is reasonable that a clinical suspicion should have the lead in decision making, today we have resources that were not available to Morgagni, the Italian anatomist and pathologist, in the 18th century.

In the last decade, both surgical and pharmacologic therapies for AD have improved. This goal has been achieved with the participation of an even more accurate diagnosis performed by cardiologists or radiologists and their excellent skills in rapidly detecting the aortic wall defect. In most of the cases, according to our experience, this would have not been possible without coupling clinical suspicion and diagnostic imaging.

Stefano Schena, MD
Alfonso Agnino, MD
Luigi de Luca Tapputi Schinosa, MD
Institute of Cardiac Surgery
Medical School, University of Bari
Bari, Italy

Correspondence to: Stefano Schena, MD, Via delle Murge 59/A, 70124 Bari, Italy; e-mail: s.schena@usa.net

REFERENCES

Table 1—Characteristics of Patients With AD Seen at the Institute of Cardiac Surgery, University of Bari

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>176</td>
</tr>
<tr>
<td>Age (yr ± SD)</td>
<td>61.04 ± 14.46</td>
</tr>
<tr>
<td>Sex (male:female)</td>
<td>122:54</td>
</tr>
<tr>
<td>Dissection type (De Bakey’s classification)</td>
<td>94:43:39</td>
</tr>
<tr>
<td>BP</td>
<td></td>
</tr>
<tr>
<td>Systolic (mm Hg ± SD)</td>
<td>130.20 ± 37.22</td>
</tr>
<tr>
<td>Diastolic (mm Hg ± SD)</td>
<td>76.42 ± 18.39</td>
</tr>
<tr>
<td>Chest pain*</td>
<td>154</td>
</tr>
<tr>
<td>Other complaints*</td>
<td></td>
</tr>
<tr>
<td>Chest oppression</td>
<td>3†</td>
</tr>
<tr>
<td>Dizziness</td>
<td>2</td>
</tr>
<tr>
<td>Hoarseness</td>
<td>1</td>
</tr>
<tr>
<td>Lower limb ischemia</td>
<td>4</td>
</tr>
<tr>
<td>Lower limb paresthesia</td>
<td>1</td>
</tr>
<tr>
<td>Renal failure</td>
<td>4</td>
</tr>
<tr>
<td>Transitory ischemic attack</td>
<td>7</td>
</tr>
</tbody>
</table>

*As main symptom.
†Pressure sensation.
To the Editor:

We appreciate the comments and the interesting case series described by Drs. Schena, Agnino, and Schinosa in response to our recent report.1 In their series, 154 of 176 patients (88%) with aortic dissection had severe chest pain, while the remaining 22 patients had other symptoms, so that technology played an even more critical role in diagnosis. They conclude that the diagnosis requires “interaction between physicians and machines.” We agree. Our point is that a careful, not lengthy, history is what enables the clinician first to consider and then to order the appropriate test in this catastrophic disorder. Their large series corroborates our finding that the initial history is useful in the vast majority of patients. Even in patients in whom the symptoms were not striking, it was still the history that guided the clinician to the appropriate, often lifesaving, diagnostic test and treatment.

We all agree that both physicians and machines are essential in every case for rapid diagnosis of aortic dissection.

Howard S. Rosman, MD, FCCP
Steven Borzak, MD
Sarine Patel, MD
Henry Ford Heart and Vascular Institute
Detroit, MI

Correspondence to: Howard S. Rosman, MD, FCCP, Henry Ford Hospital, Cardiovascular Medicine Division, 2799 West Grand Boulevard, Detroit, MI 48202

REFERENCE