What is a Primary Care Physician?

Yesterday, I received a call from a representative of the University of Massachusetts who was working on a survey about attitudes toward specialty training which was supported by a grant from the Robert Wood Johnson Foundation. Her first question to me was, "Are you a specialist or a primary care physician?"

I explained that I was the Chief of the Pulmonary Division at the University of Florida and the Gainesville Veterans Administration Medical Center, and she thought that made me a specialist. I then explained what I do when I attend in the medical intensive care unit (MICU) and when I see patients in the outpatient clinic, and she became confused. Frankly, so am I.

If a pulmonary physician also practices critical care, he or she takes care of the whole patient. I would not consider recruiting an endocrinologist to take care of diabetic acidosis in the MICU, nor do I need an infectious disease specialist to treat sepsis. If the attending physician in the intensive care unit is not a primary care physician, who is? After the patients improve and are followed in the pulmonary clinic, this same physician can certainly manage outpatient diabetes if he or she can manage diabetic acidosis, etc.

This "schizophrenic situation" was illustrated to me vividly when I closed one of my outpatient clinics to find time to edit this journal. I spent 6 months finding other physicians to provide follow-up of my patients. Often, I asked the patients if they already had another physician. Frequently, they indicated they had a family physician. I was surprised, since they always called me for anything that was wrong with them. I asked when they had last consulted their family physician, and rarely had they done so for years. Why did they not consult their family physicians? The answer may offend some primary care physicians, but this was the answer I received. They did not consult their family physicians because when they did, they were always referred to a specialist for the complaint of the day. Not only do we need more primary care physicians, but those that exist must learn to take care of more illnesses without consultation.

Why should a primary care physician need consultative help to provide care for a patient with routine chronic obstructive pulmonary disease? Despite all the furor about attitudes toward the treatment of asthma, the vast majority of asthmatic patients do not need a specialist's care. Yet many patients with these diseases are routinely referred to and followed by pulmonologists. Yes, I am suggesting that fewer patients really need the pulmonologist, and yes, I am suggesting that we cut our own fiscal throats by admitting that what we do is often simple. But some of what we do is not simple, ie, ICU care, transbronchial lung biopsies, management of immunocompromised hosts, transplants, etc. These are a few of the activities that should occupy the time of pulmonary physicians.

In any event, what we render now is primary care. Any artificial distinction decreed by politicians and bureaucrats does not alter the reality that pulmonary and critical care physicians do render primary care and should be acknowledged for doing so.

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Primum Non Nocere
Is The Therapy Worse Than The Disease?

For nearly half a century, we have known that there is an increased incidence of supraventricular tachycardia (SVT) after lung surgery. These arrhythmias are more common in patients after pneumonectomy than after lobectomy or wedge resections. Although the exact mechanisms generating these arrhythmias are not known, Krowka et al speculated that right ventricular dysfunction may contribute to their genesis.

A variety of antiarrhythmic agents have been proposed as a prophylactic measure to suppress these arrhythmias. All of these agents, however, have predictable side effects that may occur more readily than the arrhythmias themselves. In this issue of Chest (see page 1642), Van Mieghem and colleagues report an alarmingly high incidence of adult respiratory distress syndrome (ARDS) associated with amiodarone therapy after lung surgery. They prospectively evaluated amiodarone and verapamil as prophylactic agents for SVT after lung surgery. This study was terminated because of life-threatening respiratory distress in three patients (9.37 percent) in the amiodarone group, two of whom ultimately died from complications of ARDS. This editorial opinion will focus on the link between amiodarone and lung injury, physiologic perturbations that occur after pneumonectomy, and current treatment options for SVT.

Amiodarone causes lung injury by both direct and
indirect mechanisms.\textsuperscript{5,6} Postulated mediators involved in amiodarone-associated lung injury (AALI) include reactive oxygen species, phospholipids, and the iodide molecule of amiodarone. Venet and associates\textsuperscript{7} were the first to demonstrate that amiodarone may modulate lung injury by amplifying immune or inflammatory responses within the lung. In patients with AALI, CD8 lymphocytosis with or without polymorphonuclear leukocytes has been demonstrated in the bronchoalveolar lavage fluid. This cytochemical profile supports the view that AALI may be a form of hypersensitive pneumonitis. Further, certain pharmacokinetic characteristics of amiodarone, such as its high accumulation in lung tissue and its prolonged elimination half-life (average 45 days), make the lung the target organ for lung injury. These pharmacokinetic characteristics may have an important clinical bearing on the occurrence of lung injury after amiodarone therapy: lung injury may progress despite discontinuation of the drug, or it may occur weeks after discontinuation of the drug if steroid therapy is withdrawn or tapered prematurely.\textsuperscript{5,6}

Several lines of experimental evidence suggest that ambient oxygen concentration may represent an important determinant in the propagation of AALI.\textsuperscript{5,6,8} This finding is similar to oxygen-associated bleomycin- and nitrofurantoin-induced pulmonary endothelial and parenchymal lung injury. The common practice of administration of high FiO\textsubscript{2} (100 percent) during one-lung anesthesia and during refractory hypoxemia could have played a role in AALI in the patient population described by Van Mieghem et al. In a recent retrospective analysis, Greenspon and associates\textsuperscript{9} reported that ARDS developed in 50 percent of the patients who had received a short course of amiodarone prophylaxis before receiving an automatic implantable cardioverter-defibrillator or who had a subendocardial resection. However, in another group of patients who did not receive amiodarone, none developed ARDS despite undergoing similar operative procedures. The most intriguing finding in this study was that several patients in whom postoperative ARDS was successfully treated were subsequently restarted on amiodarone months after surgery without recurrence of ARDS. This finding again re-emphasizes that the mechanism of AALI involves an interaction between amiodarone and intraoperative factors such as inspired oxygen concentration, and that these factors could play a pivotal role in amplifying AALI.

A number of physiologic perturbations occur during and after pneumonectomy that make the remaining lung vulnerable to injury. These include surgical trauma, the lateral decubitus position, and release of vasoactive mediators such as cytokines.\textsuperscript{10-14} After pneumonectomy, lymphatic reserve is reduced and blood flow through the remaining lung augmented. The increased pulmonary blood flow recruits more endothelial surface area, increases in shearing forces and mean pulmonary capillary pressure (Pc), all of which increase the Starling forces favoring net filtration out of the vasculature into the pulmonary parenchyma.\textsuperscript{10-12} In a recent study, Mathur et al\textsuperscript{10} demonstrated that the ratio of edema fluid (EF) to serum protein (SP) was 0.6 or greater in a small group of patients with pulmonary edema after lung resection. The high EF/SP ratio with normal filling pressure and high cardiac output in these patients suggests that observed pulmonary edema resulted in part from loss of structural integrity of the alveolar capillary membrane. Other contributing elements include hyperinflation of the remaining lung, resulting in increased afterload and reduced perimicrovascular pressure, which exaggerates the filtration of fluid into the interstitium of the remaining lung.\textsuperscript{12} Therefore, in the presence of these physiologic perturbations, drugs associated with pulmonary toxicity, such as amiodarone, are contraindicated during the perioperative period.

What are the available therapeutic options for the treatment of SVT after lung injury? In stable patients, simple vagal maneuvers, such as the Valsalva or carotid sinus massage, should be tried. Pharmacologically, the calcium channel blockers have been the mainstay of acute conversion, but are associated with significant hypotension. In addition, calcium channel blocker use is not advised in wide complex QRS tachycardia not known to involve the AV nodes. There is emerging evidence that adenosine may be a useful agent to control SVT.\textsuperscript{4} The advantages of adenosine include short half-life, faster response time, and fewer adverse effects. Further, adenosine can be safely used as a diagnostic tool to differentiate between SVT and ventricular tachycardia. Digitalis is a poor choice because it is not effective in terminating sympathetically mediated tachycardia. Furthermore, the use of digitalis in patients with accessory pathways may be dangerous, as conduction may be enhanced.\textsuperscript{4,15}

To summarize, atrial arrhythmias after lung surgery may be best treated only if they are symptomatic and accompanied by hemodynamic instability. Because patients have increased Pc after pneumonectomy, altered endothelial integrity, and limited lymphatic reserve, drugs with serious potential for lung injury (such as amiodarone) are contraindicated during the perioperative period. Measures to control Pc, such as fluid restriction, maintaining good oxygenation, and providing pain relief by thoracic epidural analgesia, may reduce morbidity and mortality associated with both postoperative arrhythmias and postpneumonectomy pulmonary edema.\textsuperscript{10,12}
Further research should include identifying early markers of lung injury after lung secretion, the link between intraoperative \( \text{FIO}_2 \) and lung injury, immunomodulation of circulating mediators, and the effect of selective pulmonary vasodilators, such as nitric oxide, on pulmonary hemodynamics and gas exchange.

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Pulmonary Rehabilitation of Early COPD
COPD as a Systemic Disease

Pulmonary rehabilitation (PR) is an established method of care for patients with advanced COPD and related pulmonary disorders. It goes beyond ordinary care and involves patient and family education, breathing training and breathing exercises, systemic exercises, and patient support groups. All patients enrolled in a PR program also require the systematic use of both bronchoactive and strategic drugs. Oxygen is lifesaving in selected patients with chronic stable hypoxemia. Pulmonary rehabilitation improves the quality of life and probably extends useful life. It should be considered the standard of care for patients who wish to receive more than ordinary care.

Many healthcare workers with extensive experience in pulmonary rehabilitation have begun to ask the question, "Why not begin pulmonary rehabilitation before the late and often irreversible stages of disease? Why not begin pulmonary rehabilitation at a time when a major impact on disease prognosis is possible?" In support of the notion that pulmonary rehabilitation techniques should be initiated much earlier in the course of COPD, a recent study shows that significant exercise limitation is present in patients with only modest degrees of airflow obstruction. This important study presents important new data which suggest that a global impact of airflow obstruction begins in the early natural history of COPD. Thus, it is certainly possible that a vicious cycle of mild airflow limitation with resultant reduced activities of daily living begins and creates a state of premature morbidity well before advanced states of airflow obstruction and COPD occur. Couple this with the psychological counterpart of COPD which includes anxiety, depression, and somatic preoccupation, and we have the scenario of COPD as a systemic disease, from its basic origins.

Smoking, rightfully considered the root cause of COPD, may be used by patients to counter anxiety and depression. Smoking may also decrease food intake. Now a nutritional component becomes part of the puzzle of emerging COPD. Reduced caloric intake, protein restriction, failure to consume appropriate and protective amounts of antioxidant vitamins and insidiously emerging, but subtle multiple organ system dysfunction begins to dominate the tranquility and personal adaptations necessary for every human being to cope with an emerging and ultimately life-threatening chronic disease state. Undernutrition in COPD is associated with reduced immune responses, and this factor may create the opportunity for an increased number of bacterial and viral chest infections. Repeated infections cause additional metabolic and nutritional stresses on the patient. The stage is now set for an inexorable course of progressive functional decline.

To counter this conspiracy of events, patients with only mild degrees of airflow obstruction as identified...