mation can be found within the guidelines, it is useful to have them succinctly provided in book form. In the medication section, cromolyn and nedocromil are mentioned as second-line drugs, but Clark failed to mention that these medications in some of their forms are not available in the United States and are rarely prescribed. The title of chapter 7, "Stepwise Management, Asthma Action Plans, and Patient Education," is a bit misleading, as it barely touches on the stepwise approach to asthma management. Chapters 8–11 summarize the NHLBI age-based recommendations and strategies for asthma management. They are well organized and provide an easy way to review the guidelines. I would have liked to have seen a discussion on the potential risks of long-acting β agonists. While they are still recommended, concerns about the increased risk of death with β agonists and how that affects their place in the asthma-medication hierarchy warrants a more detailed discussion. One nice aspect of the book is its glossary, which will be especially useful for students.

Overall this is an easy to read, informative primer on asthma. The book is nicely laid out with clear tables and figures. Clark has done her research thoroughly and provides excellent references. The information is up to date and provides a good overview on asthma management and the current asthma guidelines. Some of the chapters have misleading titles, and some materials (eg, the discussions on allergens) are placed in chapters where they do not fit well. The latter half of the book is a distillation of information that can readily be obtained from the NHLBI guidelines,[2] though it is still useful to have that information in book form. This book is best suited for students in the medical fields, but it would also be useful for nurses, respiratory therapists, and primary care physicians who may not have the time to digest the guidelines, which can be accessed via the Internet.

Edward Carter MD
Pulmonary Division
Seattle Children's Hospital
Seattle, Washington

REFERENCES

1. The Global Initiative for Asthma (GINA). GINA report, global strategy for asthma management and prevention. http://www.ginasthma.com/guidelineitem.asp??l1=2&l2=1&intId=1561. *Accessed January 18, 2011.*
2. National Asthma Education and Prevention Program. Expert Panel Report 3: Guidelines for the diagnosis and management of asthma. 2007. http://www.nhlbi.nih.gov/guidelines/asthma/asthgdln.pdf. *Accessed February 12, 2008.*

The author has disclosed no conflicts of interest.

DOI: 10.4187/respcare.01233

Molecular Basis of Pulmonary Disease: Insights From Rare Lung Disorders. Francis X McCormack MD, Ralph J Panos MD, Bruce C Trapnell MD, editors. *Respiratory Medicine* series. Sharon R Rounds MD, series editor. New York: Humana/Springer. 2010. Hard cover, 434 pages, $219.

Understanding how a CD player works doesn't make a disc's music sound better. But if it's your job to fix CD players or tell customers when to give them up, knowing how they work helps a lot. **Molecular Basis of Pulmonary Disease: Insights From Rare Lung Disorders** is not an easy read, and it's not for every pulmonary physician or student of lung disease. It is a 2010 compendium of 19 chapters about 17 different rare lung diseases. Some are quite rare (eg, pulmonary capillary hemangiomatosis), but some are seen with some frequency by consulting pulmonary physicians (eg, pulmonary Langerhans cell histiocytosis), and some are relatively common (sarcoidosis).

This book's greatest appeal to me was the chapters on diseases one sees in clinical practice or presented at a conference. For several of them the book gives useful and sometimes intriguing information that provides the gratifying sensation that one is thoroughly and articulately updated about what the patient and caregivers need to know. An example is Vassallo's chapter about pulmonary Langerhans cell histiocytosis. This and other chapters dutifully adopt the latest nomenclature to keep one current in "lung-disease speak" (eg, "eosinophilic granuloma of the lung" and "histiocytosis X" are apparently passé nomenclature), but this chapter's 20 pages on this still puzzling disease update the reader about what really matters. Is the disease related to proliferative histiocytic disorders that may be prescribed chemotherapy? Yes and no: the same cell lineage is involved, but pulmonary Langerhans cell histiocytosis is nearly always polyclonal; it appears to be a reactive disorder.

In olden days, bronchoalveolar lavage fluid was collected in electron-microscopy medium for fixation and examination for cells with Birbeck granules—those intracellular tennis-racket-mimicking organelles. Then, briefly, it was CD1a antigen staining for diagnosis. And now S-100 staining is most commonly used. Are these 3 techniques similarly informative? No, they aren't: S-100 is found on many types of cells, and 5% positivity for CD1a in cells in bronchoalveolar lavage fluid, together with a consistent clinical picture, is the most secure and convenient way to make the diagnosis when electron microscopy and electron-microscopy media are not available.

Does pulmonary Langerhans cell histiocytosis occur only in cigarette smokers? No, but young adults who smoke make up over 90% of the cases in existing case series. Will smoking cessation make the disease go away? No, not in everyone. Are corticosteroids worth trying? Vassallo thinks so, when the disease is progressive (a 15% decline during monitoring of the FEV_1 or diffusing capacity of the lung for carbon monoxide), and he presents his sensible approach: 0.5 mg/kg/d of prednisone for 3 months, then repeat pulmonary function testing, with rapid steroid tapering to zero if no benefit is seen. Will he try corticosteroid treatment even if a smoking patient is unable to quit cigarettes? Yes he will, and so should we.

Does pulmonary Langerhans cell histiocytosis present with both restrictive and obstructive physiology on pulmonary function testing? Yes it does, perplexing test-taking fellows and the rest of us, and Vassallo explains why: although often called an interstitial lung disease, small airways are usually involved, especially early. Progressive disease leads to fibrosis and restrictive physiology, and cysts lead to air-trapping and further obstruction.

What is the molecular basis of pulmonary Langerhans cell histiocytosis? Vassallo and other experts don't know, and he doesn't spend time "hand-waving" or describing obscure animal experiments of questionable relevance. Genetic factors are probably not involved, he writes, because the disease is sporadic and doesn't run in families. Cigarette smoke is a reliable recruiter and increaser of the population of dendritic cells in the lungs of rodents and humans, but that is not enough to commonly provoke pulmonary Langerhans cell histiocytosis. Granulocyte macrophage colony stimulating fac-

tor expression is induced by cigarette smoke and is commonly abundant on airway epithelial cells in early pulmonary Langerhans cell histiocytosis lesions, but not elsewhere in lung biopsies of uninvolved tissue in the same patient—but this may be an epiphenomenon.

Another fascinating chapter describes new information about lymphangioleiomyomatosis, which occurs clinically as a sporadic disease in which histologically benign-appearing smooth-muscle cells infiltrate all structures throughout the lungs and result in cystic changes and disabling pleural effusions. There is no explanation at present for the cystic changes. Since symptomatic lymphangioleiomyomatosis occurs solely in women during the third to fifth decade of life, empirical antagonism of estrogen action with progestins is the usual therapy tried. Its success is unproven. Perhaps this is not surprising, since lymphangioleiomyomatosis usually worsens during pregnancy, a progestational hormonal state.

In their chapter, Henske and McCormack describe the new but incomplete understanding that lymphangioleiomyomatosis is related to tuberous sclerosis, an autosomal dominantly inherited disease of benign tumors in multiple organs. Thirty to forty percent of tuberous sclerosis patients have identifiable (but usually asymptomatic and rarely progressive) areas of lymphangioleiomyomatosis in their lungs. While tuberous sclerosis complex occurs in men and women, lymphangioleiomyomatosis is found solely in women. Moreover, the current conventional wisdom is that pulmonary lymphangioleiomyomatosis represents metastasis of histologically benign smooth-muscle cells to the lung—but only in women. Why? There are 2 theories: the particular smooth-muscle cell that participates is found solely in women (this seems unlikely); or (the more favored explanation) the hormonal milieu of young adult women favors metastasis through the thoracic duct. Lymphangioleiomyomatosis cells migrate up the thoracic duct into the internal jugular vein and are distributed throughout both lungs by the pulmonary arteries.

As for other links of lymphangioleiomyomatosis to tuberous sclerosis: 80% of tuberous sclerosis cases result from a de novo mutation in one of 2 genes, leading (most often) to angiolipomas in both kidneys, and possibly to large benign tumors ("tubers") in the brain and angiofibromas on the face. Angiolipomas's cell of origin is a somewhat immature smooth-muscle cell indistinguishable from those found in the lungs of affected lymphangioleiomyomatosis patients. The most exciting news about lymphangioleiomyomatosis is that, as in tuberous sclerosis complex, the mammalian target of rapamycin (mTOR) is over-expressed. In tuberous sclerosis this is due to a mutation in TSC1 or TSC2. Failure of inhibition of mTOR results in exuberant protein synthesis and cell proliferation in numerous cell types. Rapamycin (sirolimus), a potent immunosuppressive drug used after organ transplant, has now been tested in clinical trials in lymphangioleiomyomatosis patients and the results were promising, but without the stunning success of, for example, Gleevec in chronic myelogenous leukemia.

Some of the other chapters were less appealing to me. However, a great strength of this collection is that the writing is by experts and quite good: what is known is there in black-and-white text, diagrams, tables, color photomicrographs, reproduced computed tomography slices, and plain radiographs. This stands in contrast to reviews one finds too often these days, in which a junior author presents what resembles a book report with a senior author's name attached.

Most of this book's chapters are around 20 pages and include what you'd expect: disease epidemiology; available evidence on mechanism, including human and animal studies; clinical information on presentation, diagnosis, management, and prognosis; and abundant references. From Hermansky-Pudlak syndrome to cystic fibrosis, this compendium summarizes nearly all the uncommon lung diseases. For interested readers who prefer that approach to searching PubMed for a recent review (possibly of iffy quality), this would be a good book to have. However, since so much information about disease mechanism is a "work in progress," and where little is known, this book's authors resort to describing complex animal experiments that may be of dubious relevance in the long term, this book may not be a good choice for everyone in the field. Moreover, it does not pretend to be a clinically useful guide.

Bruce L Davidson MD MPH
Division of Pulmonary and
Critical Care Medicine
University of Washington
School of Medicine
Seattle, Washington

The author has disclosed no conflicts of interest.

DOI: 10.4187/respcare.01227

Thoracic Imaging. Michael Galanski MD. *Direct Diagnosis in Radiology* series. Stuttgart: Thieme. 2010. First published in German. Soft cover, 368 pages, $59.95.

The *Direct Diagnosis in Radiology* series comprises 12 pocket-sized books covering the main diagnostic imaging subspecialties. These texts cover the essential diagnoses a practicing radiologist should be aware of, in a well organized way, and are ideal for quick reference during a busy work day.

Thoracic Imaging is a 368-page soft cover-book, divided into 13 chapters, which covers the most commonly encountered disorders in chest radiology. The text follows the standard format of the series, including a brief definition (epidemiology, etiology, pathophysiology, pathogenesis) of each included disease; descriptions of imaging signs (modality of choice, radiographic, computed tomography, and magnetic resonance imaging findings, pathognomonic findings); review of clinical aspects (typical presentation, therapeutic options, course and prognosis, and "what does the clinician want to know"); and discussion of differential diagnosis. Each section is rounded out with tips, pitfalls, and selected references for those seeking a more in-depth review.

The authors hail from Germany, and in fact the book was first published in German and later translated to English, which explains the subtle differences in terminology from what is commonly used in the United States. However, this does not detract from what is overall a very successful attempt to provide an easily accessible, comprehensive review of more common diseases in thoracic radiology, leaving the more esoteric conditions for other more detailed texts. Throughout the text, the writing is clear and concise, and sections are bulleted and highly structured. Perhaps most importantly for the radiologists who will be referencing this book, an appropriately large number of high-quality diagnostic images illustrate the key findings within each section.

The opening chapter is dedicated to congenital disorders, including arteriovenous malformation, shunt, scimitar syndrome, pulmonary sequestration, pulmonary artery hypoplasia and atresia, and bronchial atresia. The disease processes are outlined in a