decisions about the book's focus, include the paucity of references, the lack of discussion about performing PFTs, and the superficial treatment of the concept of "normal" values and the sources of normal variability. I expect to refer to my copy of **Interpretation of Pulmonary Function Tests: A Practical Guide** both to reinforce my own understanding of the concepts and to help me present this material more clearly when teaching.

David R Park MD
Division of Pulmonary and
Critical Care Medicine
Harborview Medical Center
University of Washington
Seattle, Washington

Coronary Circulation and Myocardial Ischemia. Michael R Pinsky MD, Antonio Artigas MD PhD, Jean-Francois Dhainaut MD PhD, editors. (Update in Intensive Care Medicine series, Jean-Louis Vincent MD PhD, series editor.) New York: Springer-Verlag. 2002. Soft cover, illustrated, 193 pages. $49.95.

Coronary Circulation and Myocardial Ischemia is a succinct collection of reports that illustrate the basic physiology, applied physiology, functional assessment, and therapeutic and clinical applications involved in understanding the complexities of coronary circulation and the current rapidly evolving treatments for myocardial ischemia. Each of the 15 chapters were written by research contributors in the field and constitute terse summaries of basic science, physiology, and clinical applications pertaining to coronary circulation and myocardial ischemia. I will discuss the merits and limitations of the book's 4 sections.

Section I, entitled "Basic Physiology," discusses the local control of coronary blood flow and the basic cellular mechanisms involved in the development of atherosclerosis. The coronary vasculature is unique in that basal myocardial tissue oxygen extraction is very high (coronary *sinus* venous oxygen saturation is approximately 25%), so coronary blood flow must increase to meet that high myocardial metabolic demand. The authors support the hypothesis that adenosine is the locally released mediator that augments coronary blood flow, but they also present the limitations of the adenosine hypothesis, suggesting that other yet-to-be-identified mediators of coronary blood flow probably act in concert with adenosine to enhance coronary blood flow during increased myocardial metabolic demand. The authors focused their discussion of atherosclerosis on the currently "in vogue" hypothesis that inflammation and vessel infection play a key role in the development of atherosclerotic plaque. They nicely summarized the recent sero-epidemiology, animal models, and plaque detections studies that support the role of herpes cytomegalovirus, *Chlamydia pneumoniae*, and *Helicobacter pylori* in the formation of atherosclerotic plaque. However, their enthusiasm for *Chlamydia pneumonia* as a cause of atherosclerosis is based on small, positive, randomized macrolide-antibiotic trials with patients suffering coronary artery disease, and it should be tempered by the recent negative results from the larger ACADEMIC (Azithromycin in Coronary Artery Disease: Elimination of Myocardial Infection With Chlamydia[1]) and WIZARD (Weekly Intervention with Zithromax for Atherosclerosis and Related Disorders[2]) antibiotic trials, the results of which were published after the hardback version of **Coronary Circulation and Myocardial Ischemia** came out in 2000.

Section II, "Applied Physiology," discusses concepts such as ischemic preconditioning, coronary circulation in sepsis, and the importance of plaque thrombus generation and pharmacologic fibrinolysis of occluded vessels. Ischemic preconditioning, first described by Murray et al in 1986,[3] is the observation that myocardium exposed to antecedent brief sublethal ischemia and reperfusion has smaller subsequent infarct size than myocardium not exposed to ischemic preconditioning. The chapter author describes possible cellular mechanisms and clinical scenarios involved in ischemic preconditioning and alludes to the possible role of the adenosine-triphosphate-sensitive potassium channel (K_{ATP}) in ischemic preconditioning. Not mentioned, however, were recent clinical results from the large IONA study in Europe,[4] in which nicorandil (a K_{ATP} agonist) improved myocardial ischemia and unstable angina in patients with symptomatic coronary artery disease; these results support direct pharmacologic activation of ischemic preconditioning as a novel treatment for atherosclerotic heart disease. The next chapter in this section nicely illustrates the effect of sepsis on coronary circulation. Although early sepsis is often associated with high cardiac output the authors discuss, from their own research, the observation of sepsis-mediated increased coronary blood flow and, hence, impaired vasodilator reserve. The 2 final chapters in this section nicely summarize the role of inflammation, tissue factor, and altered shear stress in thrombosis generation and the utility of fibrin-selective and non-fibrin-selective fibrinolytic agents in clot dissolution.

Section III, "Functional Assessment of the Coronary Circulation," discusses assessment of coronary circulation via echocardiography, myocardial viability imaging with positron emission tomography, magnetic nuclear resonance imaging, and intracoronary ultrasound (although this final section chapter is misplaced, in section IV). This section of the book is fairly solid and the information provides an important and succinct update for both novices and seasoned practitioners on the new and evolving cutting-edge technologies for evaluating coronary circulation.

Section IV, "Therapeutic and Clinical Applications," deals with nonthrombotic pharmacologic therapy, fibrinolytic therapy, percutaneous coronary intervention, and adjunctive therapy in the treatment of myocardial ischemia. The main fault with this section is that although the authors provide a nice overview of ischemic heart disease therapies available in the year 2000, the field has rapidly advanced and there are many new and expanded standard therapies available for the treatment of atherosclerotic heart disease, including (1) angiotensin-converting enzyme inhibitors to prevent ischemic events (the Heart Outcomes Prevention Evaluation [the HOPE study]),[5] (2) 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CO-A) reductase inhibitors (statins) to reduce ischemic events in patients with atherosclerosis independent of low-density-lipoprotein cholesterol (the Heart Protection Study),[6] (3) clopidogrel in addition to aspirin to reduce ischemic cardiovascular events in patients with non-ST elevation myocardial infarction/unstable angina (the CURE [clopidogrel in unstable angina to prevent recurrent events] study)[7] or after percutaneous coronary intervention (the CREDO [clopidogrel for the reduction of events during observation] study),[8] and (4) rapamycin-coated intracoronary stents to reduce restenosis following percutaneous coronary intervention (the RAVEL [randomized study with the sirolimus-eluting bx velocity balloon-expandable stent] study and the SIRIUS study).[9,10]

In summary, **Coronary Circulation and Myocardial Ischemia** is an elegant, compact summary of the basic science and physiology, applied physiology, functional assessment, and treatment of coronary circulation and myocardial ischemia as known at the time of the initial (hard-cover) publication in 2000. Since then cardiovascular research has substantially advanced our understanding and treatment of ischemic heart disease, but those new findings were not included in the 2002 paperback edition of **Coronary Circulation and Myocardial Ischemia**. Certain of the new treatments are major advances, including the widespread use of angiotensin-converting enzyme inhibitors, statins, clopidogrel, novel anti-angina agents such as nicorandil (in Europe), and rapamycin-coated stents. These new advancements greatly benefit our patients but, unfortunately, make **Coronary Circulation and Myocardial Ischemia** dated. One additional note: the book suffers from many irritating errors in English usage, grammar, and spelling, which should have been corrected by the copy editor.

Arnold S Baas MD
Department of Medicine
Cardiology Division
University of Washington
Harborview Medical Center
Seattle, Washington

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Inhalation Therapy for Pulmonary Hypertension: The Proceedings of a Symposium Held at the Annual Congress of The European Respiratory Society, Berlin, September 2001. Timothy Higenbottam and Celia Emery, editors. New York: Parthenon. 2003. Soft cover, illustrated, 110 pages, $99.95.

Twenty years ago, severe forms of pulmonary hypertension were considered a "fate", rather than a "challenge," by physicians, nurses, and respiratory therapists. Interest in this field of pulmonology has grown, mainly because of encouraging new therapy options, one of which is inhalation therapy, using either gases (eg, nitric oxide) or aerosolized drugs (eg, prostanoids). The enormous progress on this subject gave reason for a special symposium, which was held in Berlin, Germany, in September 2001, during the Annual Congress of the European Respiratory Society. The presentations at the conference are now compiled in this soft-cover book.

Unlike usual textbooks, which try to give a complete review, this book discusses only the latest advances in inhalation therapy for pulmonary hypertension, for pharmacologists, toxicologists, physicians, respiratory physiologists, and graduate and medical students who are interested in these discipline. The editors, Timothy Higenbottam and Celia Emery, successfully maintained the logical structure of the symposium and included the transcribed post-presentation discussions between the presenters and the audiences, which is a charming way to make problems and criticisms visible that might be easily bypassed in reading the text.

Based on the table of contents, the reader might assume that this book refers exclusively to inhaled prostanoids. Actually, the book addresses many clinical questions in pharmacology, pathophysiology, and new therapeutic possibilities for severe pulmonary hypertension.

Higenbottam wrote Chapter 1, which presents the historical background and scientific rationale for the use of aerosolized prostanoids. The chapter describes the main advantages of the prostanoid substance group (vasodilation and inhibition of platelet aggregation), as well as the disadvantage of physiological prostacyclin for clinical use. The rapid hydrolyzation and inactivation of the molecule after contact with oxygenated blood fluids led to the development of similar, more stabile substances. In addition, the author refers to the World Health Organization's classification of pulmonary hypertension, to demonstrate which patients are responding to inhalation therapy. This leads to the pathophysiology of pulmonary arterial hypertension, which is the topic of Chapter 2, written by Robert Naeije. The author, who is a world expert in this field, points out that the mechanisms of pulmonary hypertension development are incompletely understood. The text presents the genetic patterns of pulmonary hypertension patients as well as additional aspects of specific pathological changes (so-called "plexiform lesions"), which are speculated to be related to genetic mutations. Then the text describes inflammatory pathways of the pulmonary vasculature, with spe-