PREFACE

Arterial diseases are the leading causes of morbidity and mortality in all industrialized countries, and their incidence is increasing in non-industrial countries. Among the industrialized countries, the United States in particular is in the midst of an epidemic of obesity and diabetes. One in four Americans meets the criteria for obesity and the number keeps growing.

Lower Extremity Arterial Disease (LEAD) is a common disease entity for men older than 40 and women older than 50 years of age. The prevalence of LEAD continues to increase with age, from less than 3% in the population younger than the age of 60 to more than 20% at age 75 and older. The majority of patients older than 75 with LEAD are asymptomatic. Prevention of arterial disease is key to reducing morbidity and mortality. LEAD is associated with specific risk factors, namely hypertriglyceridemia, homocysteinema, very low HDL cholesterol, physical inactivity, and above all cigaret smoking and diabetes mellitus, alone or in tandem.

LEAD, symptomatic or not, particularly when it coexists with Coronary Artery Disease (CAD) (Chapter 9), calls for polypharmacy. Polypharmacy should no longer have a bad connotation in treating patients with LEAD and CAD. In patients with metabolic syndrome and LEAD, a short-acting statin in the evening and triglyceride-reducing fenofibrate during breakfast can improve time to claudication significantly, improve endothelial function, improve the lipid profile, and at the same time decrease the probability of a coronary event. Fenofibrate and rosuvastatin or simvastatin should be given twelve hours apart to avoid an overlap of their half-lives. In treating LEAD, aggressive risk factor modification should be implemented, which includes: smoking cessation, euglycemic control of diabetes, ideal control of both systolic and diastolic pressure, dramatic improvement of the lipid profile, low calorie Mediterranean diet rich in antioxidants, and, equally important, exercise therapy, either community based or supervised (Chapter 11).

Percutaneous interventions with balloon angioplasty, bare metal stents, and the more preferable for the femoral and intrafemoral arteries, drug-diluting stents, offer dramatic improvement of the stenosed or occluded lumen (Chapter 12). Blood flow is restored and great symptomatic relief is achieved. Arterial grafting techniques have also pro-

vided tremendous advances in reinstituting peripheral blood flow with the lowest possible periprocedural complication rate. Today's therapeutic armamentarium also includes the most promising approach for advanced and distal disease (i.e., therapeutic angiogensis). The vascular growth factors can be administered intra-arterially or intramuscularly in the ischemic muscle. Therapeutic angiogenesis combined with the other current therapeutic options and aggressive risk-factor modification can remarkably improve claudication, prevent limb loss, and prolong life (Chapters 10 and 13).

The presence of LEAD, as defined by an ankle brachial index (ABI) of less than one, adversely influences the prognosis of coronary heart disease. The lower the ABI (the lowest in either ankle), the worse the prognosis. The morbidity and mortality from coronary artery bypass grafting is higher in patients with LEAD. The same increased morbidity and mortality also occurs during or after percutaneous coronary interventions, short or long term. LEAD differs from other peripheral arterial diseases by its specific medical therapy as well; the drug currently approved by the Food and Drug Administration to treat the symptoms of claudication, the phosphodiesterase III inhibitor Cilostazol, has no effect on other arterial beds like the renal or the carotid systems (Chapter 10).

Intermittent claudication can be caused by an abdominal aortic aneurysm (AAA), which is a totally different disease entity with different pathophysiology and distinct genetic mechanisms. AAAs cannot be stopped from increasing in diameter with either blood pressure control or antilipid therapy.

Lower Extremity Arterial Disease provides a comprehensive state-of-the-art review of LEAD. A detailed review of its cardinal symptom, intermittent claudication, is presented in Chapter 1. The book provides a thorough and detailed description of noninvasive and accurate assessment of LEAD with special emphasis on the ABI and its diagnostic and prognostic significance. Modern diagnostic methods, such as vascular flow patterns and magnetic resonance angiography, are eloquently presented for the educational benefit of the clinician in Chapter 2. The known risk factors for LEAD and CAD—smoking, diabetes mellitus, dislipidemia, systemic hypertension, and physical inactivity—are presented in the chapters on epidemiology and risk factors (Chapters 3–8).

The question "What do claudiants die from?" is reviewed and analyzed in the chapter on LEAD coexisting with CAD. The presence of LEAD increases significantly the probability of coexisting CAD. In patients who cannot exercise much because of claudication or in asymptomatic patients with LEAD, the preferred approach to diagnose coex-

isting CAD is dual isotope pharmacological stress testing either with adenosine IV or dipyridomole IV. Adenosine should not be given if bronchial asthma, hypotension, or profound bradycardia is present. Alternatively, dobutamine can be utilized as a pharmacologic stressor. Dobutamine should not be used in patients with LEAD and atrial fibrillation; dobutamine remarkably accelerates atrioventricular conduction. Dobutamine should be avoided as a pharmacologic stressor if blood pressure is elevated; it may precipitate a hypertensive crisis. If the clinical index of suspicion is high, then the cardiovascular physician may recommend coronary angiography (luminography is a more accurate term). Overall, single photon emission computed tomographic images of the myocardium are preferable because they can accurately assess myocardium at risk in patients with LEAD.

In morbidly obese patients (those weighing more than 350 lbs) with LEAD, neither myocardial perfusion studies nor coronary angiography can be performed for technical reasons. These patients can be evaluated with contrast echocardiography (Chapter 9). The management of patients with LEAD and CAD is the same as in every patient who has myocardial ischemia, silent or symptomatic.

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