

HOSPITAL PHYSICIAN®

CARDIOLOGY BOARD REVIEW MANUAL

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The *Hospital Physician Cardiology Board Review Manual* is a peer-reviewed study guide for fellows and practicing physicians preparing for board examinations in cardiology. Each bimonthly manual reviews a topic essential to the current practice of cardiology.

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Coronary Physiology and Intravascular Ultrasound

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Coronary Physiology and Intravascular Ultrasound

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INTRODUCTION

Angina is a major source of morbidity and a harbinger of mortality. The most common etiology of this condition is coronary artery disease (CAD), but angina also can be caused by microvascular disease, cardiac valvular disease, severe anemia or hypoxia, and high cardiac-output states. A variety of techniques available in the cardiac catheterization laboratory can assist cardiologists in the diagnosis of these conditions and help guide treatment decisions.

In recent years, the evaluation of patients with angina has focused on studying the anatomy of the epicardial coronary arteries for evidence of atherosclerosis using angiography. However, simply discovering the existence of atherosclerosis is insufficient to determine the etiology of angina and its treatment course. Conversely, the lack of angiographic evidence of atherosclerosis does not exclude its presence and the risks associated with it.^{1,2} Therefore, further evaluation is sometimes needed.

Some degree of coronary atherosclerosis exists in almost all adults in Western society. Angina occurs only when an atherosclerotic lesion restricts blood flow and causes myocardial ischemia or infarction. Revascularization therapy in the form of percutaneous coronary intervention or coronary artery bypass grafting (CABG) is directed only at such ischemia-producing lesions. Therefore, an understanding of the functional significance of a coronary artery lesion is critical in determining the lesion's role in the patient's clinical presentation and in selecting treatment.

CORONARY PHYSIOLOGY

The coronary arteries supply the myocardium with blood to serve its metabolic needs. Myocardial oxygen demand reflects the myocardium's metabolic activity and is determined by heart rate, inotropic state (contractility), and intramyocardial wall stress (which is directly

related to chamber size and pressure as well as inversely related to wall thickness). Oxygen extraction from the coronary blood is near maximal even in the resting state, with coronary sinus oxygen saturations typically measuring 30% or less. Therefore, any increases in myocardial oxygen demand generally require parallel increases in coronary artery blood flow. Blood flow is regulated primarily through changes in coronary vascular resistance. Reduced resistance increases blood flow, while increased resistance reduces blood flow as predicted by Ohm's law. Small intramyocardial coronary arterioles serve as "resistance" vessels that can increase and decrease blood flow through vasodilation and vasoconstriction, respectively, varying blood flow up to five-fold.³⁻⁵ In contrast, normal epicardial coronary arteries serve as conduits or "conductance" vessels, allowing blood to flow freely without any significant resistance (**Figure 1A**).

Atherosclerotic epicardial coronary arteries that develop intraluminal lesions can limit blood flow, in effect making the large artery a "resistance" vessel (**Figure 1B**). Since these lesions are relatively fixed and do not vary with myocardial oxygen demand, they can cause ischemia and infarction when myocardial oxygen demand increases beyond a certain level. In dogs, experimental concentric epicardial coronary artery stenoses of 50% lumen diameter or greater can limit maximal coronary blood flow, and stenoses of 90% lumen diameter or greater can limit resting blood flow as well.⁶⁻¹⁰

However, as predicted by fluid dynamics theory and demonstrated in vivo, the resistance of an epicardial coronary artery lesion on blood flow is not determined simply by the degree of luminal diameter stenosis it causes. A lesion resists blood flow by converting the flowing blood's energy into heat, primarily by inducing both frictional energy losses and separation energy losses. Frictional energy losses are related to the degree of lumen diameter stenosis present and the length and roughness of the lesion.^{11,12} Separation energy losses are caused by the flow turbulence the lesion produces and are related to the lesion's morphology, including its entrance and exit angles, and its eccentricity (**Figure 2**).¹³⁻¹⁵ All these geometric factors must be taken into account in order to