11

PROGRESS IN CARDIOLOGY

PAUL N. YU

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PREFACE

Progress in Cardiology Volume 11 contains nine chapters that deal with a variety of cardiovascular problems.

Dr. Block gives a timely report on percutaneous transluminal coronary angiography (PTCA) in Chapter 1. The historical aspects, technique, selection of patients, use of pharmacologic agents, and complications are reviewed. Based upon a registry of 1,500 patients undergoing PTCA, more than 80% had single-vessel disease, and the overall success rate was 63%. Dr. Block states that, at the present time, PTCA is most suitable for patients who have severe angina pectoris but only one coronary arterial stenosis and that PTCA will not replace coronary revascularization for the majority of patients with symptomatic occlusive coronary artery disease.

The second chapter, by Dr. Massie and co-workers, focuses on the current status and future prospects of myocardial perfusion scintigraphy with thallium-201. The methodology, sensitivity, specificity, and interpretation of thallium-201 scintigrams are discussed in detail. Thallium-201 scintigraphy plays an important role in the assessment of location, extent, and severity of coronary artery disease and is also valuable in the diagnosis and quantitation of acute myocardial infarction. It is anticipated that new radiopharmaceuticals and new instrumentation will appreciably facilitate further studies in nuclear cardiology.

In Chapter 3, Dr. Turino and associates present an interesting review of the effects of disordered pulmonary physiology on the metabolism of vasoactive agents. Pulmonary endothelial cells are primarily responsible for the metabolic activity of the lung's circulation. The lungs clear, activate, or release a number of vasoactive agents. These metabolic activities are influenced by the level of alveolar oxygen tension and the degree of acid-base derangement.

The current role of prostaglandins in cardiovascular diseases is succinctly discussed by Dr. Lewis in Chapter 4. Prostaglandins have an important place in the interaction between platelets and the blood vessel wall, central to the problem of atheroma formation. They also have a potential involvement in blood pressure regulation. Thromboxane and prostacyclin have opposite effects; antiplatelet drugs may inhibit the effect of the former or may enhance the effects of the latter. The roles of prostaglandins in patent ductus arteriosus closure, in the pathophysiology of pulmonary and arterial hypertension, and in pre-eclampsia also are appraised.

In Chapter 5, Dr. Semple reviews the use of angiotensin-converting enzyme inhibitors (ACEI) in hypertension and heart failure. Historical aspects of the development of ACEI and their chemical structures are described. The mechanism of their antihypertensive effects in both animal models and in patients is still incompletely understood. ACEI improve various hemodynamic parameters and exercise tolerance in patients with congestive heart failure, particularly in those with increased plasma renin activity.

In the following chapter, Drs. Dargie and Goodwin cover the role of catecholamines in both hypertrophic and congestive cardiomyopathy. A number of chemical and experimental studies suggest chronic catecholamine stimulation or excess as a cause of hypertrophic cardiomyopathy; however, the catecholamine theory is persuasive, but not proved. Abnormalities in adrenergic nerve function observed in congestive cardiomyopathy probably are the consequences rather than the causes of cardiac failure.

In Chapter 7, Dr. Hyman and co-workers present an up-to-date review of the pharmacology of the pulmonary circulation. Pulmonary vessels are supplied with alpha- and beta-2-adrenoreceptors, which are innervated by sympathetic nerves. Stimulation of alpha-receptors causes vasoconstriction, whereas stimulation of beta-2-receptors induces vasodilation. The role of the parasympathetic nervous system in the pulmonary vascular bed is uncertain. A number of pharmacologic agents may induce either pulmonary vasoconstriction or vasodilation. Unfortunately, the interpretation of direct pulmonary vascular responses is complicated by indirect influences related to changes in pulmonary blood flow, left atrial pressure, blood Po_2, and airway resistance.

The relationship between smoking and heart disease is extensively discussed by Drs. Libow and Schlant in Chapter 8. Cardiovascular morbidity and mortality have been reported to be higher in smokers than in nonsmokers. The bulk of evidence has indicated that long-term smoking may influence the character and the function of the platelets and may contribute to the development of atherosclerosis. The mechanisms by which tobacco aggravates myocardial ischemia are still unknown, however. The overall mortality rate is lower and the incidence of recurrent myocardial infarction is reduced in smokers who stop smoking.

In the final chapter, Dr. Fisher and associates give an authoritative review of the pathophysiology and therapy of cardiogenic shock. The most common cause of cardiogenic shock is acute myocardial infarction involving more than 40% of the left ventricular myocardium and resulting in impaired vital organ perfusion and circulatory collapse. Determinants of myocardial oxygen supply, myocardial oxygen demand, and left ventricular function are clearly delineated. These authors discuss in detail the treatment of cardiogenic shock with pharmacologic agents, mechanical devices, and surgical procedures and conclude with a note on prognosis and prevention.

We are preparing the contents of *Progress in Cardiology,* Volume 12. This volume will feature a symposium on nuclear cardiology organized by Dr. George A. Beller, who will serve as the guest editor.

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CONTENTS

Chapter 1

PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY

Peter C. Block

The concept of treating atherosclerotic obstructions of arteries percutaneously using a catheter was first developed by Dotter and Judkins in 1964.[15] They devised a coaxial catheter system consisting of a size-12-French catheter introduced over an inner size-8-French catheter. Staple[49] modified their technique by developing a single, size-8-to-10-French catheter that gradually tapered to its tip. The catheter was introduced over a 0.038-cm wire guide and was advanced through an atherosclerotic stenosis. The advantage of this system over the coaxial system was that the gradual tapering of the catheter eliminated the "shoulder" of the outer catheter and possibly minimized damage to the vessel intima. Intimal damage to arteries at the site of stenosis and local complications such as hematoma formation at the percutaneous insertion site were common, however, and the technique never gained acceptance in the United States, although a number of European centers used the technique extensively in the treatment of large series of patients.[52]

The development of a balloon dilation catheter in 1974 by Gruntzig and Hopff[23] led to widespread acceptance of the concept of transluminal angioplasty. These researchers developed a noncompliant balloon made of polyvinyl chloride that could be inflated to a predetermined outer diameter at a pressure of 4 to 5 atmospheres. The balloon was inflatable, but not expandable, and could be attached to the tip of a size-7-French catheter. The smaller catheter size reduced the rate of complications at the catheter insertion site and allowed the catheter to traverse more stenotic segments of the peripheral vasculature. Once the balloon segment of the catheter was positioned within the stenotic arterial segment, the balloon was inflated. This inflation applied a lateral force against the atherosclerotic plaque, rather than the combination longitudinal and lateral force produced by a "wedge-shaped" tapered catheter. Gruntzig's reports in 1976 and 1977[20,21] of the short-term results and follow-up after 2 years in 200 patients established percutaneous transluminal angioplasty, using his modification of the balloon catheter, as an effective method of treatment for peripheral atherosclerotic disease.

In their first description of transluminal angioplasty, Dotter and Judkins[15] mentioned the possibility of using the angioplasty technique in areas other than the lower extremities and specifically mentioned the possibility of dilating proximal coronary artery stenoses. Technical problems arose in adapting transluminal angioplasty to small arteries, however. The most important drawback was that balloon-tipped dilating catheters were difficult to miniaturize to sizes that would allow passage through a stenotic coronary artery less than 1.5 mm in diameter. A "guiding" catheter was also needed to introduce the small dilating catheter into the appropriate artery.

In dogs in which the left anterior descending coronary artery had been partially ligated, Gruntzig was able to perform dilation of the stenotic arterial segments using the miniaturized catheter system.[22,28] From these experiments, it appeared that continuous distal coronary perfusion with oxygenated blood was necessary to avoid ischemia at the time that the dilating catheter traversed the coronary stenosis. Later experience in humans has shown that continuous perfusion of the coronary artery is not required during coronary angioplasty. The new technique was studied further in cadaver hearts[1,18,34,48] and in humans.

In a cooperative study, Gruntzig, Myler, and others[29] performed transluminal angioplasty of distal coronary artery stenoses in patients undergoing coronary bypass graft operations. These patients had coronary stenoses dilated in the operating room when a stenosis could not be bypassed because of its distal location. From those studies, it was concluded that it was possible to traverse coronary artery stenoses and to dilate coronary obstructions without producing myocardial infarction or peripheral embolization. "Sizing" of the balloon catheter was found to be important, so that the diameter of the undiseased portion of the vessel adjacent to the stenosis would not be exceeded by the diameter of the inflated balloon. This technique avoided overstretching and the risk of vessel rupture. It was also found that calcification in a coronary stenosis made the chances of successful transluminal angioplasty less likely.

In September of 1977, Gruntzig performed the first percutaneous transluminal coronary angioplasty (PTCA) in Zurich. His preliminary communication in *Lancet* in early 1978[19] reported that five patients with coronary artery atherosclerosis and angina pectoris had been treated successfully with this new technique. Follow-up studies, by serial stress testing with myocardial imaging and angiography, indicated that PTCA was an effective treatment in certain patients with severe coronary atherosclerosis and angina pectoris and initiated widespread investigation of the technique.

TECHNIQUE

PTCA is performed using techniques similar to those used in coronary angiography. In most centers, routine coronary cineangiography is performed at the beginning of any PTCA procedure to establish that there has been no change in the coronary circulation since the patient's last angiogram. In the Cardiac Catheterization Laboratory of the Massachusetts General Hospital, we insert a radial artery cannula for constant intra-arterial pressure monitoring throughout the procedure. A Swan-Ganz pulmonary artery catheter and a temporary transvenous pacemaker are also placed in the standard fashion, usually from a percutaneous subclavian puncture. These devices allow continuous hemodynamic monitoring throughout and after the procedure, as well as ventricular pacing if necessary. Following local anesthesia, a standard angiographic catheter is placed percutaneously from the groin or through a cutdown from the brachial artery. Coronary cineangiography is performed. Intravenous nitroglycerin is given during angiography to evaluate the stenotic area more completely and to exclude coronary spasm. This is important because patients receive continuous intravenous nitroglycerin during and after the procedure. It is therefore valid to compare coronary angiography performed before and after PTCA because the procedure is done under identical pharmacologic conditions.

Once coronary angiography has been completed, a size-8- or 8.5-French guiding catheter is introduced percutaneously from the groin, usually using an introducing sheath, or is introduced directly into the brachial artery cutdown using standard techniques.[14] The guiding catheter is positioned under fluoroscopic control at the appropriate coronary ostium. Angiography can be performed through the guiding catheter, and the patient is positioned so that the coronary stenosis is best seen in one or the other oblique view. One must take care that pressure monitored from the guiding catheter is nonocclusive, so that coronary flow is adequate as the dilating catheter is introduced subselectively into the coronary artery. The superimposition of the image of the Swan-Ganz line and the pacemaker over the region of coronary stenosis in the right side of the heart allows "marking" of the stenosis. This marking facilitates exact positioning of the balloon segment of the dilating catheter within the coronary stenosis at the time of dilation.

The coronary dilating catheter is a double-lumen balloon catheter with either a short, flexible wire guide, 0.018 cm in diameter, annealed to its tip or a standard-length wire guide that is movable within the catheter's central lumen (Fig. 1). The advantage of a movable wire guide is that it can be first advanced through the coronary stenosis. The dilating catheter then can be advanced over the wire guide; this technique minimizes the risk of plaque dissection or perforation. If balloon catheters must be changed when the coronary ste-

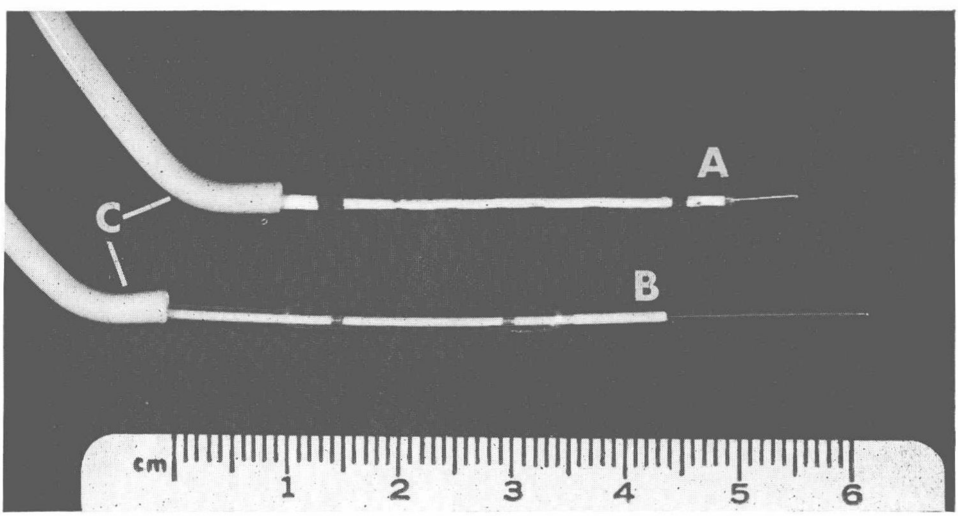

Fig. 1. Two dilating (A and B) and guiding (C) catheters. The upper dilating catheter (A) has a short wire guide, 0.018 cm in diameter, annealed to its tip. The two dark markers are radiopaque and lie proximal and distal to the balloon segment, which is 2 cm in length and 3.0 mm in diameter. The second dilating catheter (B) has a movable wire guide. Radiopaque markers lie within the balloon segment. The balloon segment and outer diameter of B are identical in size to catheter A.