Thomas Ischinger

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Preface

After 7 years of clinical use, percutaneous transluminal coronary angioplasty (PTCA) has now found worldwide acceptance, and its basic techniques are well standardized. A growing number of invasive cardiologists are acquainting themselves with the procedure in order to be in a position to offer new therapeutic options to their patients. However, the transition from the diagnostic to the therapeutic intervention is not always easily accomplished. Certain prerequisites concerning technique, the physician, technical equipment, and program organization are necessary for safe and effective performance.

The consequences of unsuccessful coronary angioplasty range from waste of time and money to severe complications for the patient; the decision to perform PTCA needs to be based on sound indications and techniques according to the best current scientific evidence.

Techniques and technology for coronary angioplasty continue to evolve. Scientific evidence of its short- and long-term value continues to accumulate, and applications of the procedure are still being extended.

This volume is obviously no substitute for well-guided hands-on experience with the procedure; it is intended rather to present current policies of patient selection and patient management; to describe standard techniques (femoral and brachial approaches) and potential applications; to point out problem areas and limitations of the procedure; and to provide tips that may be useful even for the more experienced angioplasty operator.

I believe the aim of presenting different experts' views and practical approaches to PTCA, although unavoidably resulting in some overlap, has helped to create a state-of-the-art report as well as a useful practical reference.

I thank my former colleagues from St. Louis University Division of Cardiology, in particular Harold L. Kennedy (Chief of Division), for their support and their enthusiasm for my work as director of interventional cardiology, which helped encourage me to create this book. I thank my former teacher Andreas R. Grüntzig of Emory University, for sharing with me his outstanding abilities and his experience.

St. Louis/Munich, November 1985 THOMAS SCHLEPPER

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冠状动脉血管成形术的临床应用

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Chapter 1. Introduction and Historical Background

THOMAS ISCHINGER

The feasibility of percutaneous transluminal balloon dilatation of coronary stenoses was not an accidental finding such as often do occur in medicine, but the result of several years of dedicated work: inspired by the telescope-like catheter system introduced by Charles Dotter and Melvin Judkins in 1964 [1, 2], in the early 1970s Dr. Andreas Gruentzig developed an inflatable balloon for dilatation of localized atherosclerotic obstructions in human peripheral arteries [3]. While gathering experience and learning from the results of peripheral angioplasty in a large number of patients, Gruentzig worked toward the goal of modifying the technique for intracoronary use. Animal experiments, postmortem studies, and intraoperative dilatations were performed [4, 5] before the dream came true on 16 September 1977 in the catheterization laboratory at the University Hospital of Zürich, Switzerland, when he tackled the LAD stenosis of his first PTCA patient:

... "The dilatation catheter was advanced through the stenosis with no difficulties. The stenosis was severe but the catheter slipped through it without resistance. The catheter wedged the stenosis and the distal coronary pressure was very low. To the surprise of all of us, no ST elevation, ventricular fibrillation or even extra-systole occurred and the patient had no chest pain. At this moment I decided not to start coronary perfusion with the roller pump. After the first balloon inflation the distal coronary pressure rose nicely. Encouraged by this positive response I inflated the balloon a second time to relieve the residual gradient ... I removed the catheter and immediate angiography in several oblique projections revealed a marked reduction of the LAD stenosis. There was no peripheral spasm or embolization. We therefore declared success."

This is part of the account of the first percutaneous transluminal coronary angioplasty (PTCA) performed by Dr. Andreas Gruentzig [6]. Not until 5 months later, in February 1978, were the results obtained in the first 5 patients published in *The Lancet* [7]. The results in a larger series of 50 patients treated with coronary angioplasty were published in 1979 in the *New England Journal of Medicine* [8]. By then, the potential of this new treatment was more widely recognized, and the success story of PTCA took its course. To date, some centers have treated more than 4000 patients with PTCA. Only recently, the results obtained with coronary angioplasty in 3079 patients enrolled in the National Heart Lung and Blood Institute's PTCA Registry were analyzed and published in a special issue of the *American Journal of Cardiology* [9]. There is no doubt that current results have met – if not exceeded – the expections and hopes stimulated by Gruentzig's original report.

Yet, we still share some of Gruentzig's original concerns: Complications of PTCA do occur, and we do not yet have a safe means of preventing myocardial infarction should dilatation result in irreversible coronary occlusion. Gruentzig's original concept of perfusing the distal coronary artery through the dilatation catheter is attracting increasing interest.

On the other hand, the relative ease with which successful PTCA can be carried out is often surprising and may seduce us into too uncritical use of the technique, as Gruentzig describes in his report, when he – "seduced by the enthusiasm" of his colleagues – recrossed the successfully dilated LAD stenosis for dilatation of an insignificant stenosis in a diagonal branch.

The procedure has remained essentially unchanged, and it is amazing how similar Gruentzig's first report is to a PTCA report prepared in 1985. However, there has been no stagnation in techniques and catheter technology. Catheters of the latest generation are becoming consistently smaller (low-profile balloon catheters), more versatile (steerable balloon catheter systems) [10], and stronger (balloons tolerating more than 12 atm). Other improvements include larger central lumina of the dilatation catheters for improved pressure recordings and/or dye injection, more flexible yet steerable guidewires [11], and guide catheters with better torque control. Some procedural modifications have been developed, such as longer inflation times [12] and higher dilatation pressures [13] to give greater initial dilatation and a better long-term outcome [14, 15]. High-resolution fluoroscopy and still-frame disk capacity have further improved the procedure.

Analyses of the vast experiences from large centers and from the NHLBI PTCA Registry have provided us with the necessary feedback: We have learned how to select patients more individually and how to interpret the results of PTCA. Good results have encouraged us to extend the applications of PTCA, but we have been held back by the low but constant incidence of complications. There is still no way of definitely predicting the outcome of PTCA, but we are better at calculating the risks. Today coronary angioplasty is considered the method of choice in some patients with coronary artery disease, and an equal alternative to coronary artery bypass surgery in others.

After its introduction into clinical practice the greatest improvement in PTCA technology was the development of the so-called steerable dilatation catheters. These catheters marked the beginning of a new era and are responsible for the still expanding potential of the procedure. Passage of tight, distal, or diffuse stenoses could not have been safely accomplished without these catheters. "Kissing balloon" techniques and complex procedures [16] involving multiple dilatations in multiple vessels in a single session have become feasible due to these technological advances. Coronary angioplasty now seems ready to accept a major challenge: comparison with coronary bypass surgery in patients with multiple vessel disease.

Whatever the outcome of this comparison, PTCA will remain a desirable palliative measure, one that can help many patients at various stages of their disease process. Furthermore, coronary angioplasty has taught us that it is possible to work successfully inside the human coronary artery. Coronary angioplasty has given birth to the field of interventional cardiology and continues to stimulate the search for new methods of arterial revascularization using techniques other than mechanical stenosis dilatation.

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Chapter 2. The Mechanism of Transluminal Angioplasty Pathology of the Arterial Stenoses that Are Most Amenable to PTCA

PETER C. BLOCK

Normal coronary arteries are compliant, and their lumens can be easily stretched. They have considerable reactivity, and variations in coronary artery lumen size are common due to changes in coronary vascular tone. Microscopically, there are three distinct layers in the coronary arteries: the intima, consisting of endothelial cells attached to a thin layer of extracellular matrix; the media, which is made up of variable layers of circumferentially oriented smooth muscle cells with surrounding elastin and collagen; and the adventitia, which consists of loosely arranged connective tissue, fibroblasts, and a few muscle cells. The internal elastic lamina separates the intima from the media.

There are two major types of atherosclerotic plaques which can cause stenoses of the coronary arteries: fibromuscular plaques and complicated plaques. Fibromuscular plaques are formed when medial smooth muscle cells proliferate, probably in response to focal injury, and variable amounts of intracellular and extracellular lipid are deposited. Any atherosclerotic plaque can produce a loss of normal arterial wall elasticity and reactivity because of a splinting action of the abnormal intima and media on the artery. Though the plaque itself may infringe upon the lumen by virtue of its volume, it has been shown that in arterial segments with experimentally induced fibromuscular plaques, less than 10% of the stenosis is due to the actual volume of the plaque. The remaining stenosis is due mainly to a splinting action and contraction of the underlying arterial wall [11].

A complicated plaque is most likely to be a degenerated fibromuscular plaque altered by necrosis, more lipid infiltration, hemorrhage, and finally calcification. A complicated plaque is usually surrounded by a thin, fibrous cap on the luminal surface, which surrounds a core of necrotic debris and lipid. The media is frequently fragmented, and there may be fraying of the internal elastic membrane with medial destruction. Calcium deposits are common. It is this kind of atherosclerotic plaque that is most often responsible for the clinical manifestations of atherosclerotic coronary artery disease in man. Mural thrombus, which may be formed due to exposure to circulating blood of thrombogenic material secondary to ulceration of the fibrous cap, may become organized and be incorporated further into the plaque. This results in a thicker and more fibrous cap, which compromises the lumen further and increases coronary insufficiency. Ultimately, this type of plaque may ulcerate or rupture due to intraplaque hemorrhage. Sudden mural thrombus formation over the exposed plaque elements leads to vascular thrombosis and possibly to myocardial infarction.

The nature of the atherosclerotic plaque undergoing transluminal angioplasty almost certainly has a direct bearing on the pathophysiologic response of the artery to balloon inflation. For example, passage of a guidewire and a dilating catheter through a long, ulcerated plaque could theoretically involve a high

chance of microembolism of loose plaque material distally. Conversely, dilation of a "young" fibromuscular plaque would be less likely to have the same consequences.

Atherosclerotic plaques are mostly focal and produce localized stenoses only. In fact, most stenoses occur in the more proximal segments of muscular arteries. About 70% of coronary atherosclerotic lesions occur within the first 4-cm section of one of the three major coronary arteries [14]. This anatomic distribution favors the use of transluminal angioplasty for the treatment of coronary stenoses, since it is technically easier to reach stenoses that are in the proximal portions of the coronary vasculature.

Experimental Data Concerning the Mechanism of Angioplasty

The mechanism of transluminal angioplasty has been elucidated from four major sources:

1. Angioplasty performed experimentally in autopsied human hearts [1].
2. The study of pathological specimens studied after transluminal angioplasty in normal dog coronary arteries [2, 5, 8, 9, 18].
3. Pathological study of transluminal angioplasty performed in atherosclerotic rabbit models [4, 5, 12, 20].
4. Pathological specimens studied after successful transluminal angioplasty both in femoral vessels and in coronary arteries in humans [6, 21].

Studies of Angioplasty Performed in Autopsied Human Hearts

Transluminal angioplasty performed in vitro in autopsied human hearts shows that the atheromatous plaque is split, usually at its thinnest point. The splits frequently extend from the intima down to the internal elastic membrane. These findings do not support the concept that atheromas are compressed by the expanding angioplasty balloon. Rather, they show that successful angioplasty involves splitting of the plaque, resulting in a larger lumen [1, 5]. These findings initially raised fears that angioplasty might be associated with the high incidence of coronary artery rupture. Since in vitro studies on autopsied arteries are necessarily done using nonviable tissue, the early reports raised questions as to whether their findings could be extrapolated to the effect of angioplasty in man.

Studies in Normal Dog Coronary Arteries

Electron microscopic studies of normal dog coronary arteries after angioplasty do not show as much disruption of the arterial wall as is found in autopsied human hearts. However, a normal dog coronary artery and the size of the inflated angioplasty balloon are approximately equal – a situation not found during

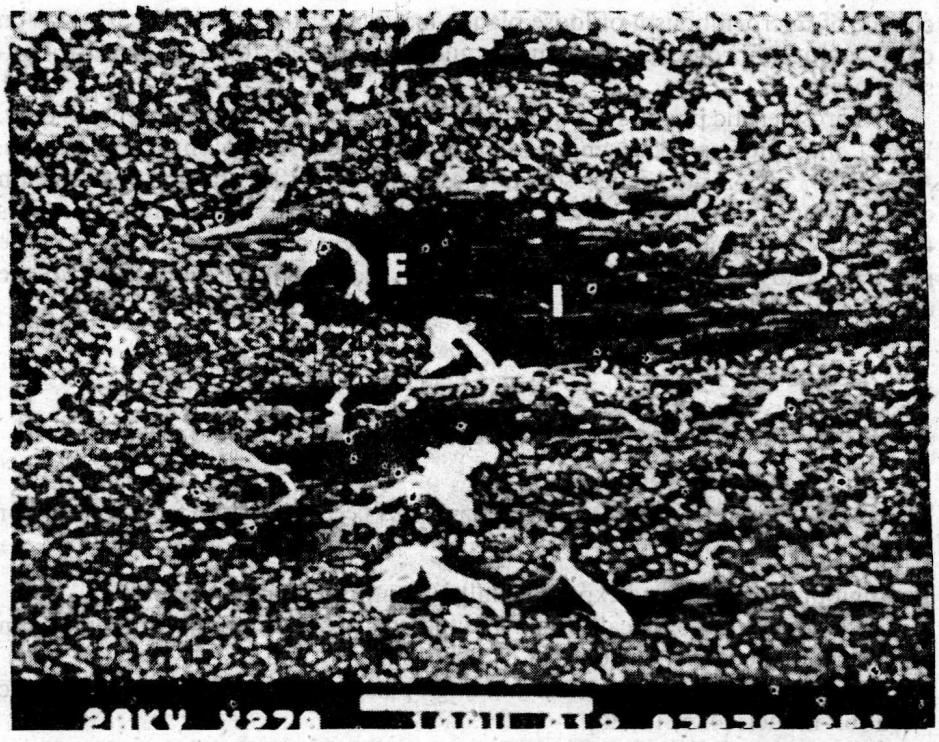

Fig. 1. Electron micrograph of a normal dog coronary artery after angioplasty. Partially adherent endothelial cells (*E*) lie adjacent to small islands of normal endothelium (*I*). A carpet of platelets (*P*) covers all areas denuded of endothelial cells

angioplasty of tightly stenosed coronary arteries in vitro or clinically in man. In the normal dog coronary artery, inflation of the balloon stretches the outer arterial wall only slightly. Contact of the endothelial cells with the inflated angioplasty balloon produces severe desquamation of endothelial cells and platelet deposition in the area of angioplasty. Superfical shearing of endothelial elements enlarges the arterial lumen only slightly. However, the demonstration by these studies that the inflated balloon causes severe desquamation of endothelial cells in the area of angioplasty is important. This highlights the marked platelet deposition that always occurs in the area of angioplasty (Fig. 1) [17, 18]. In the dog, the use of "antiplatelet" agents such as aspirin or low-molecular-weight dextran does not inhibit platelet deposition in the area of angioplasty. Segments of the arteries adjacent to the dilated segment show no endothelial disruption.

Studies in Atherosclerotic Rabbits

An animal model of atherosclerosis can be produced in rabbits by feeding them a 2% cholesterol diet and injuring the arterial endothelium. Arterial lesions pro-

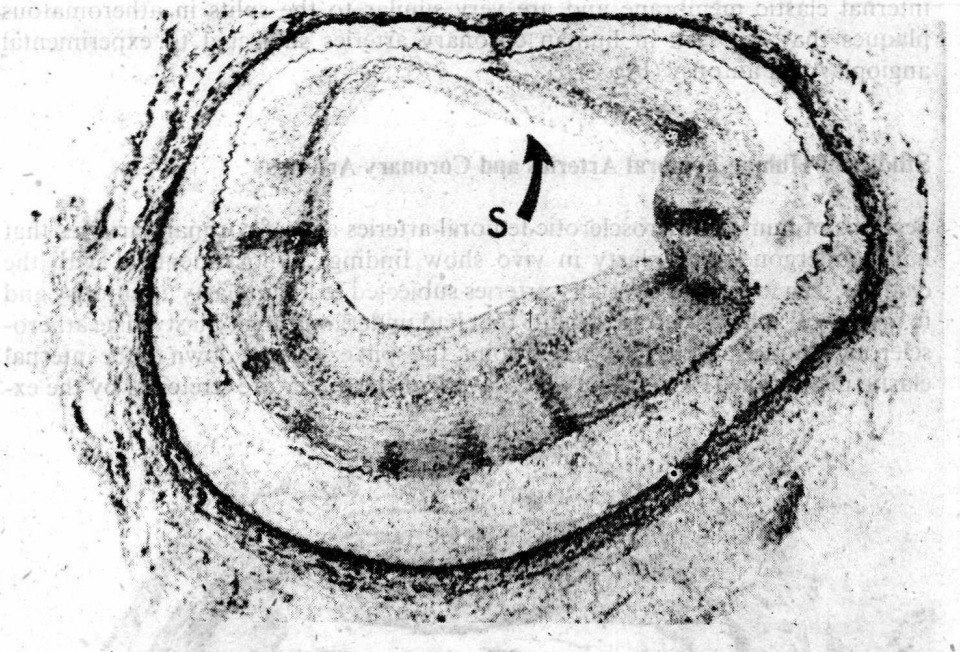

Fig. 2. Section of an atherosclerotic iliac artery of a rabbit after transluminal angioplasty. A split (*S, arrow*) extends through the plaque and continues circumferentially at the internal elastic lamina. X55 [4]. (By permission of the American Heart Association, Inc.)

duced in rabbits tend to be rich in cholesterol-laden macrophages, unlike the fibromuscular plaques seen in man. The response of these plaques to angioplasty may be different than that of the atherosclerotic plaques in man. Studies of angioplasty in this experimental model give clues to the mechanism of angioplasty in human atherosclerosis, since in both instances the size of the constricted atherosclerotic lumen and the size of the inflated angioplasty balloon are quite disparate. This is the situation which is common in angioplasty of human coronary arteries in vivo. The rabbit model for atherosclerosis also produces focal encroachment on the arterial lumen, which allows both angiographic and pathological study of the effects of angioplasty. Angiographic studies in atherosclerotic rabbits show that in areas where the balloon is inflated there is an increase in lumen size as evaluated by measurement of the width of the contrast column before and after angioplasty [4]. Some arteries also show an irregularity of the edge of the contrast medium, a finding frequently seen in human coronary arteries after successful angioplasty. This is most probably due to splitting of the atherosclerotic plaque (see below). Pathological study of the same arteries shows severe endothelial desquamation. A carpet of platelets adheres to the subendothelial collagen matrix, just as in the dog studies. In addition, in almost all instances, the atheromatous plaque splits at its thinnest point. Such splits frequently reach the

internal elastic membrane and are very similar to the splits in atheromatous plaques that are seen in human coronary arteries subjected to experimental angioplasty at autopsy (Fig. 2).

Studies of Human Femoral Arteries and Coronary Arteries

Sections of human atherosclerotic femoral arteries and of coronary arteries that have undergone angioplasty in vivo show findings almost identical with the changes seen in human coronary arteries subjected to angioplasty at autopsy and in arteries of atherosclerotic rabbits that had undergone angioplasty. The atherosclerotic plaque splits at its weakest point, the split extending down to the internal elastic membrane (Fig. 3). The outer layers of the artery are stretched by the ex-

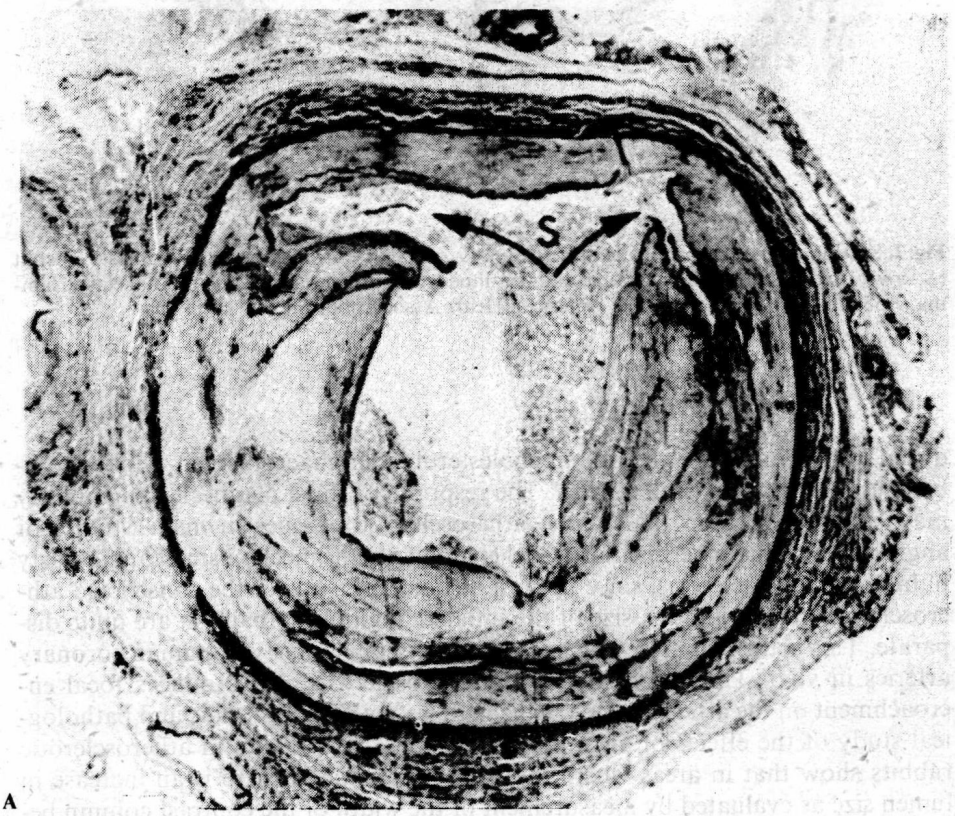

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Fig. 3. A Section of femoral artery at site of angioplasty, revealing splits (*S*) of the fibrous intimal plaque extending to the internal elastic lamina and following its circumference (*arrows*). The original lume has been enlarged considerably by the splits in the plaque [6]. (Reprinted by permission of the New England Journal of Medicine) **B** Section of left anterior descending coronary artery at site of angioplasty, showing a split (*arrow*) in the thinnest portion of a fibrous atherosclerotic plaque. The split extends into the media with circumferential extension. The lumen is partly occluded by a small thrombus (*dark area*). [6] (Reprinted by permission of the New England Journal of Medicine)