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PROGRESS IN CARDIOLOGY

PAUL N. YU

JOHN F. GOODWIN



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Edited by

PAUL N. YU, M.D.

*Sarah McCort Ward Professor of Medicine,
University of Rochester School of Medicine and Dentistry,
Rochester, New York*

and

JOHN F. GOODWIN, M.D.

*Professor of Clinical Cardiology,
Royal Postgraduate Medical School,
London, England*



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PROGRESS IN CARDIOLOGY

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 in-

terpretation 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; anti-platelet 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 non-smokers. 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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Rochester, New York
London, England

Paul N. Yu
John F. Goodwin

CONTRIBUTORS

Peter C. Block, M.D.
Associate Professor of Medicine
Harvard Medical School
Associate Physician and Director
Cardiac Catheterization Laboratory
Massachusetts General Hospital
Boston, Massachusetts

Jeffrey S. Borer, M.D.
Professor of Medicine
Chief of Cardiac Catheterization Laboratory
Co-Director of Nuclear Cardiology
New York Hospital—Cornell Medical Center
New York, New York

Elias H. Botvinick, M.D.
Associate Professor of Medicine and Radiology
University of California
San Francisco, California

J. David Bristow, M.D.
Professor of Medicine
University of Oregon
Portland, Oregon

Michael Collins, M.D.
Associate
Cardiovascular Laboratory
Baptist Hospital
Miami, Florida

Henry J. Dargie, M.D.
Consultant in Cardiology
Western Infirmary
Glasgow, Scotland

Jeffrey Fisher, M.D.
Assistant Professor of Medicine
New York Hospital—Cornell Medical Center
New York, New York

Albert L. Hyman, M.D.
Professor of Research Surgery in Cardiology and
Pharmacology
Tulane University
Consultant in Cardiology
New Orleans Veterans Administration Hospital
New Orleans, Louisiana

Philip J. Kadowitz, Ph.D.
Professor of Pharmacology
Tulane University
New Orleans, Louisiana

John F. Goodwin, M.D.
Professor of Clinical Cardiology
Royal Postgraduate Medical School
London, England

Peter J. Lewis, M.D.
Senior Lecturer in Clinical Pharmacology
Royal Postgraduate Medical School
Honorary Consultant Physician
Hammersmith Hospital
London, England

Mark Libow, M.D.
Clinical Instructor in Cardiology
Emory University School of Medicine
Atlanta, Georgia

Barry M. Massie, M.D.
Assistant Professor of Medicine
University of California
Clinical Investigator
Veterans Administration
Director
Coronary Care Unit
Veterans Administration Hospital
San Francisco, California

Robert B. Mellins, M.D.
Professor of Pediatrics
Columbia University College of Physicians and Surgeons
Attending Pediatrician
Babies Hospital, Columbia-Presbyterian Medical Center
New York, New York

Stephen Scheidt, M.D.
Professor of Clinical Medicine
Assistant Dean for Continuing Medical Education
New York Hospital—Cornell Medical Center
New York, New York

Robert C. Schlant, M.D.
Professor of Medicine
Chief of Cardiology
Emory University School of Medicine
Atlanta, Georgia

Peter F. Semple, M.D.
Consultant Physician
MRC Blood Pressure Unit
Western Infirmary
Glasgow, Scotland

Ernst William Spannake, Ph.D.
Associate Professor of Physiology
Johns Hopkins University
Baltimore, Maryland

S. Alex Stalcup, M.D.
Assistant Professor of Pediatrics
Columbia University College of Physicians and Surgeons
Assistant Attending Pediatrician
Babies Hospital, Columbia-Presbyterian Medical Center
New York, New York

Gerald M. Turino, M.D.
Professor of Medicine
Columbia University College of Physicians and Surgeons
Attending Physician
Columbia-Presbyterian Hospital
New York, New York

CONTENTS

| | |
|--|-----------|
| 1. PERCUTANEOUS TRANSLUMINAL CORONARY ANGIOPLASTY . . . | 1 |
| <i>Peter C. Block</i> | |
| Technique | 2 |
| Pharmacologic Agents | 6 |
| Selection of Patients | 7 |
| Pathology of Arterial Stenoses Most Amenable to PTCA | 9 |
| Results from the NHLBI Registry | 10 |
| Complications | 11 |
| Long-term Follow-up | 13 |
| Mechanism | 13 |
| Current Status | 15 |
| 2. MYOCARDIAL PERFUSION SCINTIGRAPHY WITH THALLIUM-201: | |
| CURRENT STATUS AND FUTURE PROSPECTS | 19 |
| <i>Barry M. Massie, Elias H. Botvinick, and J. David Bristow</i> | |
| Background | 20 |
| Thallium-201 as an Imaging Agent | 20 |
| Physiologic Basis for Perfusion Scintigraphy | 20 |
| Rationale and Physiology of Exercise Scintigraphy | 22 |
| Physiologic Basis for Thallium-201 Redistribution | 23 |
| Methodology | 24 |
| Instrumentation and Acquisition | 24 |
| Acquisition of Delayed Postexercise Scintigrams | 25 |
| Scintigraphy following Pharmacologic Vasodilation | 26 |
| Normal Thallium-201 Scintigrams | 26 |

| | |
|--|--------|
| Exercise Thallium-201 Scintigraphy | 30 |
| Diagnosis of Coronary Artery Disease | 30 |
| Assessment of the Extent of Coronary Artery Disease | 35 |
| Factors Affecting Sensitivity | 39 |
| Patients with Known Coronary Artery Disease | 40 |
| Evaluation of Bypass Graft Patency | 43 |
| Evaluation of Lung and Right Ventricular Thallium-201 Uptake in Coronary Artery Disease | 44 |
| Comparison with Exercise Radionuclide Angiography in Coronary Artery Disease Diagnosis | 44 |
| Thallium-201 Scintigraphy at Rest | 45 |
| Diagnosis of Acute Myocardial Infarction | 45 |
| Prognostic Implications in Acute Myocardial Infarction | 46 |
| Other Ischemic Syndromes | 47 |
| Recent Advances and Future Prospects | 47 |
| Quantitation of Thallium-201 Scintigrams | 47 |
| Tomographic Reconstruction of Thallium-201 Scintigrams | 48 |
| Future Directions | 50 |
| 3. EFFECT OF PATHOPHYSIOLOGIC STATES OF THE LUNG ON VASOACTIVE SUBSTANCE METABOLISM | 57 |
| <i>Gerald M. Turino, S. Alex Stalcup, and Robert B. Mellins</i> | |
| Alveolar Hypoxia | 58 |
| Acid-Base Derangements and Acute Hyperoxia | 61 |
| Hypoxia and Converting Enzyme Activity in Endothelial Cell Culture | 62 |
| Oxygen Tension and ACE Activity in Fetus and Placenta | 64 |
| Concomitant Acidosis and Hypoxia and Circulating Concentrations of Bradykinin | 65 |
| Experimental Pulmonary Emphysema and Pulmonary ACE Activity | 66 |
| Hyperoxia | 68 |
| Changes in Pulmonary ACE Activity and Circulatory Homeostasis | 68 |
| Summary | 69 |
| 4. CURRENT POSITION OF PROSTAGLANDINS IN CARDIOVASCULAR DISEASE | 71 |
| <i>Peter J. Lewis</i> | |
| What Are Prostaglandins? | 71 |
| Biosynthesis of Eicosanoids | 72 |
| Prostanoids and the Interaction between Platelets and Blood Vessel Walls | 74 |
| Platelet Adhesion and Atheroma | 75 |
| Antiplatelet Drugs and the Thromboxane-Prostacyclin System | 76 |
| Dietary Fat and Prostaglandins | 77 |
| Prostaglandins and the Ductus Arteriosus | 78 |
| Prostaglandins and Pulmonary Hypertension | 79 |
| Prostaglandins and Arterial Hypertension | 79 |
| Prostacyclin and Pre-eclampsia | 80 |
| Summary | 81 |

| | |
|--|------------|
| 5. ANGIOTENSIN-CONVERTING ENZYME INHIBITORS IN HYPERTENSION AND HEART FAILURE | 83 |
| <i>Peter Semple</i> | |
| Development and Chemistry | 83 |
| Hypertension | 84 |
| Animal Models | 84 |
| Clinical Use | 85 |
| How Do Converting Enzyme Inhibitors Lower Blood Pressure? . . | 86 |
| Pharmacokinetics and Side Effects | 87 |
| Congestive Heart Failure | 87 |
| 6. CATECHOLAMINES, CARDIOMYOPATHIES, AND CARDIAC FUNCTION | 93 |
| <i>Henry J. Dargie and John F. Goodwin</i> | |
| Role of Catecholamines in Cardiomyopathy | 93 |
| Synthesis, Storage, and Release of Catecholamines | 93 |
| Plasma Norepinephrine as an Index of Sympathetic (Adrenergic) Activity | 94 |
| Hypertrophic Cardiomyopathy | 94 |
| Terminology and Concepts | 94 |
| Catecholamines as a Cause of Hypertrophic Cardiomyopathy . . | 95 |
| Chronic Catecholamine Excess | 95 |
| Effects of Beta-Adrenoceptor Stimulation and Blockade | 95 |
| Neural Crest Disorders | 96 |
| Embryology | 96 |
| Endocrine Disease | 97 |
| Plasma Norepinephrine | 97 |
| Congestive Cardiomyopathy | 98 |
| Terminology and Concepts | 98 |
| Catecholamines and Congestive Cardiomyopathy | 98 |
| Experimental Cardiomyopathy | 99 |
| Catecholamines and Heart Failure | 99 |
| Receptor Function | 100 |
| Neurotransmitter Synthesis | 100 |
| Parasympathetic Function | 100 |
| Catecholamines and Peripheral Circulation | 101 |
| Therapeutic Considerations | 101 |
| Beta-Adrenoceptor Blockade in Congestive Cardiomyopathy . . | 101 |
| 7. PHARMACOLOGY OF THE PULMONARY CIRCULATION | 107 |
| <i>Albert L. Hyman, Ernst W. Spannhaake, and Philip J. Kadowitz</i> | |
| Drugs Acting on Autonomic Receptors | 109 |
| Catecholamines and the Sympathetic Nerves | 109 |
| Cholinergic Mechanisms | 113 |
| Autacoids | 114 |
| Histamine | 114 |
| 5-Hydroxytryptamine (Serotonin) | 114 |

| | |
|---|----------------|
| Bradykinin | 115 |
| Angiotensins | 116 |
| Vasoactive Intestinal Peptide | 117 |
| Prostaglandins and Other Arachidonic Acid Metabolites | 118 |
| Effects of Vasodilator Drugs | 122 |
| Summary | 125 |
| 8. SMOKING AND HEART DISEASE | 131 |
| <i>Mark Libow and Robert C. Schlant</i> | |
| Pathogenesis of Atherosclerosis | 131 |
| Smoking, Platelets, and Coagulation | 134 |
| Angina | 137 |
| Myocardial Infarction | 138 |
| Sudden Death | 141 |
| Pathophysiologic Features | 142 |
| Therapeutic Techniques | 148 |
| 9. CARDIOGENIC SHOCK: PATHOPHYSIOLOGY AND THERAPY | 163 |
| <i>Jeffrey Fisher, Stephen Scheidt, Michael Collins, and Jeffrey S. Borer</i> | |
| Definition | 163 |
| Pathophysiology | 164 |
| Circulatory Derangement | 164 |
| Determinants of Myocardial Oxygen Supply | 164 |
| Determinants of Myocardial Oxygen Demand | 165 |
| Determinants of Left Ventricular Function | 166 |
| Associated Organ Changes | 167 |
| Microcirculatory Derangements | 167 |
| Etiology and Classification | 168 |
| Hemodynamic Subsets in Acute Myocardial Infarction | 168 |
| Pharmacologic Treatment | 169 |
| Diuretics | 171 |
| Vasodilator Agents | 171 |
| Sodium Nitroprusside | 172 |
| Phentolamine | 173 |
| Nitroglycerin and Other Nitrate Preparations | 173 |
| Salbutamol | 174 |
| Inotropic Agents and Vasopressors | 174 |
| Dopamine | 174 |
| Dobutamine | 176 |
| Norepinephrine | 177 |
| Isoproterenol | 178 |
| Digitalis | 178 |
| Combined Vasodilator and Inotropic-Vasopressor Therapy | 179 |
| Parasympathetic Blocking Agents | 179 |
| Antiarrhythmic Agents | 179 |
| Temporary Pacemakers | 180 |
| Anticoagulants | 180 |

| | |
|---|-----|
| Mechanical Therapeutic Devices | 181 |
| Counterpulsation with Intra-aortic Balloon Pump | 181 |
| External Counterpulsation | 184 |
| Abdominal Left Ventricular Assist Device | 185 |
| Surgical Treatment | 185 |
| Acute Ventricular Septal Defect | 185 |
| Acute Mitral Regurgitation | 186 |
| Acute Tricuspid Regurgitation | 186 |
| Cardiac Rupture | 186 |
| Left Ventricular Aneurysm | 186 |
| Prognosis | 186 |
| Prevention | 187 |

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. Staple⁴⁹ 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 Hopff²³ 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 an-

gioplasty, 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 Judkins¹⁵ 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 others²⁹ 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 cath-

eter 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 1978¹⁹ 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-

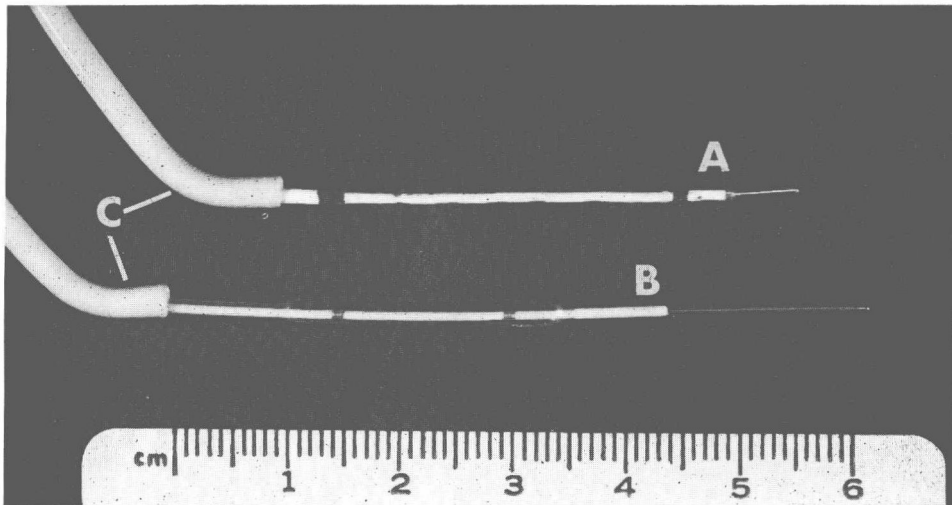


Fig. 1. Two dilating (A and B) and guiding (C) catheters. The upper dilating catheter (A) has a short wire guide, 0.018 cm in diameter, annealed to its tip. The two dark markers are radiopaque and lie proximal and distal to the balloon segment, which is 2 cm in length and 3.0 mm in diameter. The second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