CORONARY HEART DISEASE

Coronary heart disease (CHD) is the most common form of heart disease and the single most important cause of premature death in Europe, the Baltic states, Russia, North and South America, Australia and New Zealand. By 2020 it is estimated that it will be the major cause of death in all regions of the world. In the UK (population 59 million), 1 in 3 men and 1 in 4 women die from CHD, an estimated

in the last 10 years CHD mortality has fallen by 42% among UK men and women aged 16-64. Disease of the coronary arteries is almost always due to atheroma and its complications, particularly thrombosis;

STABLE ANGINA

Angina pectoris is the symptom complex caused by transient myocardial ischaemia and constitutes a clinical syndrome rather than a disease; it may occur whenever there is an imbalance between myocardial oxygen supply and demand (Box 18.54). Coronary atheroma is by far the most common cause of angina; however, the symptom may also be a manifestation of other forms of heart disease, particularly aortic valve disease and hypertrophic cardiomyopathy. This section describes the features of 'stable' angina pectoris which occurs when coronary perfusion is impaired by fixed or stable atheroma of the coronary arteries

The clinical spectrum of coronary heart disease ranges from

silent (asymptomatic) ischemia to chronic stable angina, unstable angina, acute myocardial infarction (AMI), ischemic cardiomyopathy, and sudden cardiac death

Risk Factors…atheroschlerosis

Uncontrollable

- Sex
- Hereditary
- Race
- Age

Controllable

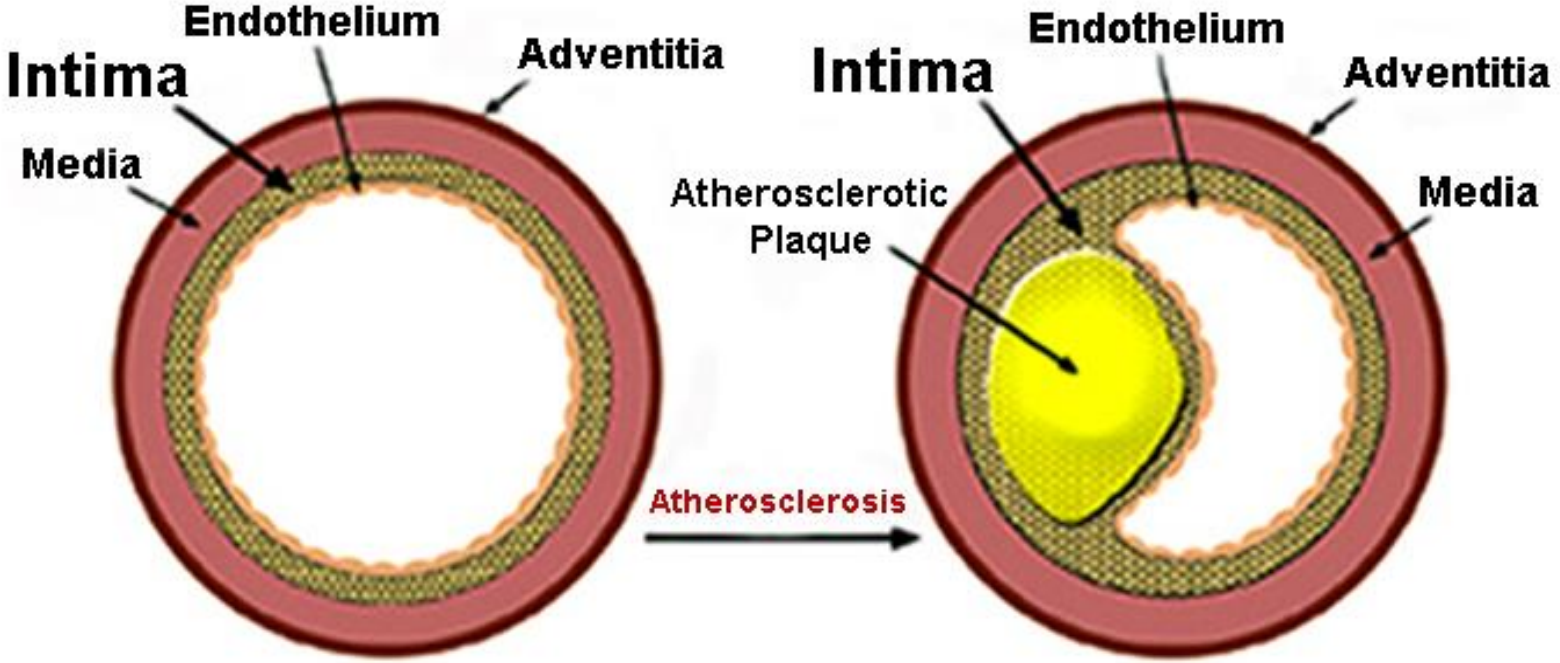
- High blood pressure
- High blood cholesterol
- Smoking
- Physical activity
- Obesity
- Diabetes
- Stress and anger

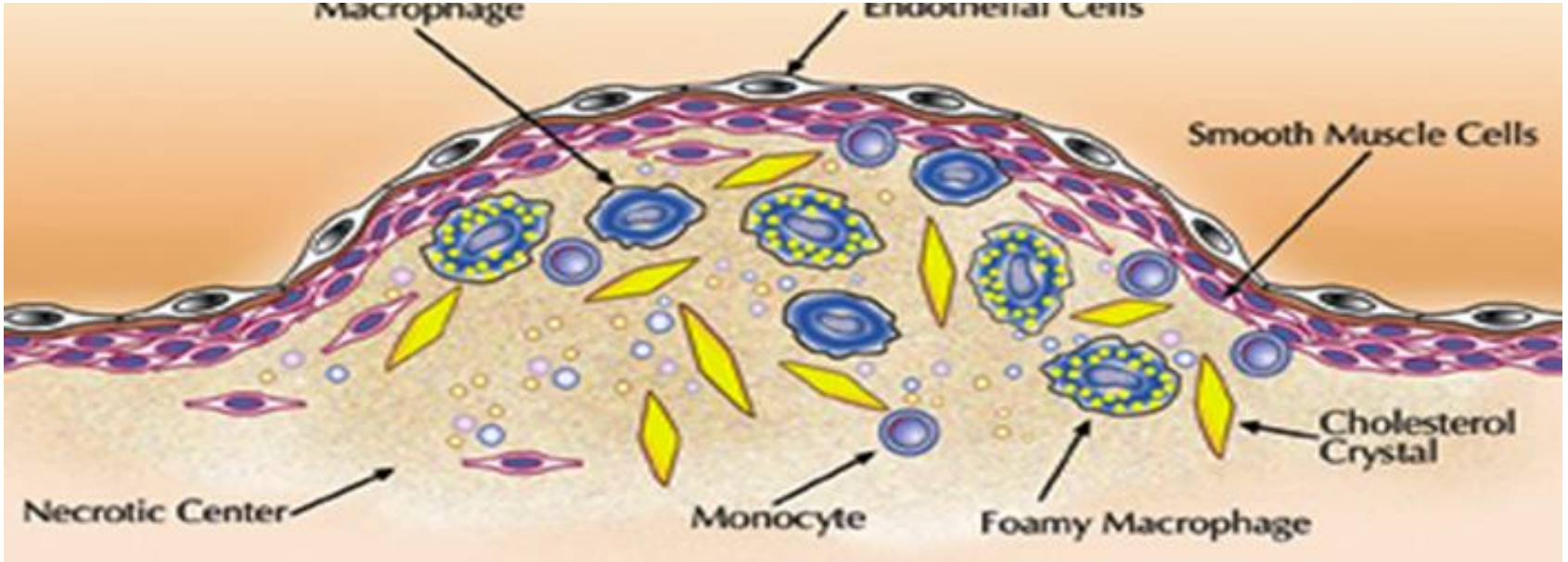
SEVERAL NONATHEROSCLEROTIC PROCESSES

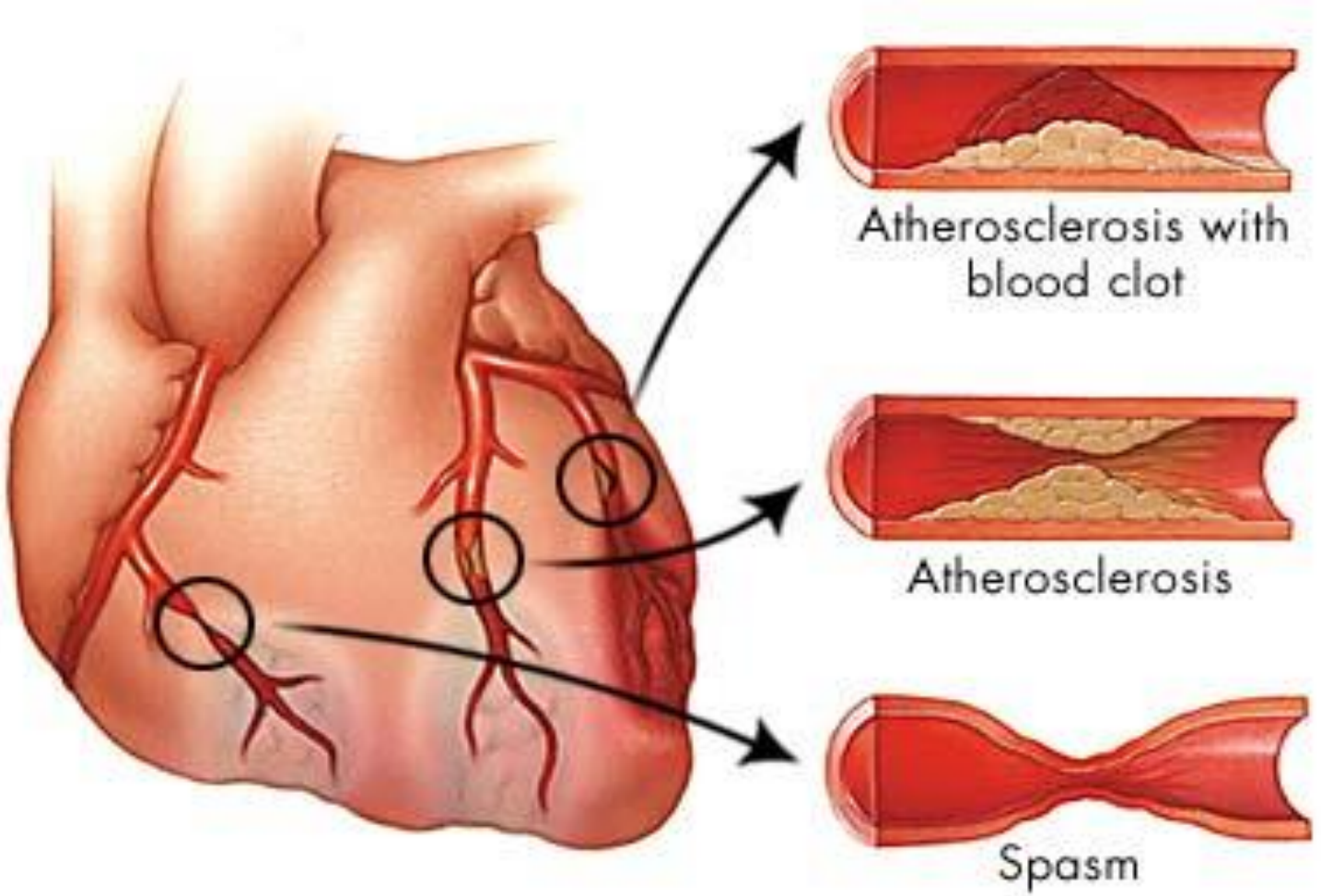
MAY AFFECT THE CORONARY ARTERIES AND PRODUCE ACUTE CORONARY INSUFFICIENCY. ACUTE EMBOLIZATION DOWN A CORONARY ARTERY MAY OCCLUDE BLOOD FLOW AND PRODUCE MYOCARDIAL ISCHEMIA AND INFARCTION. THE MOST COMMON SOURCES OF SUCH EMBOLI INCLUDE INFECTIOUS ENDOCARDITIS, MURAL THROMBI IN THE LEFT ATRIUM OR VENTRICLE, THROMBI FROM PROSTHETIC VALVES, INTRACARDIAC TUMORS, AND PARADOXICAL EMBOLI FROM THE VENOUS SYSTEM ACROSS AN ATRIAL SEPTAL DEFECT OR VENTRICULAR SEPTAL DEFECT (VSD). CHEST WALL TRAUMA MAY RESULT IN CORONARY INJURY AND IN SITU THROMBOSIS. MEDIASTINAL RADIATION MAY RESULT IN FIBROSIS OF THE CORONARY ARTERIES AND PRODUCE MYOCARDIAL INFARCTION. AORTIC DISSECTION MAY PROPAGATE TO THE AORTIC ROOT AND OCCLUDE A CORONARY ARTERY AT ITS ORIGIN. CORONARY ARTERY DISSECTION MAY OCCUR DURING CARDIAC CATHETERIZATION AND RARELY MAY BE A SPONTANEOUS EVENT. SEVERAL FORMS OF ARTERITIS MAY INVOLVE THE CORONARY ARTERIES, INCLUDING SYPHILIS, TAKAYASU'S ARTERITIS, POLYARTERITIS NODOSA, SYSTEMIC LUPUS ERYTHEMATOSUS, AND GIANT CELL ARTERITIS. THESE SYNDROMES MAY RESULT IN OBSTRUCTION, OCCLUSION, OR THROMBOSIS OF THE CORONARY ARTERIES

Atherosclerotic Plaque: Definition and Formation

- Focal accumulation of smooth muscle cells, foam cells, cholesterol crystals and lipid under the endothelium of the artery (within the Tunica Intima)
- Given time, this plaque can protrude into the lumen of the vessel reducing blood flow
- Often develops at branch points or curves within the vasculature → blood is slowed and/or turbulent


Endothelium
Intima
Adventitia
Media
Endothelium
Intima
Adventitia
Atherosclerotic
Plaque
Media
Atherosclerosis


Macrophage
Smooth Muscle Cells
Cholesterol
Crystal
Necrotic Center
Monocyte
Foamy Macrophage


Atherosclerosis with
blood clot
Atherosclerosis
Spasm

IN 10% TO 20% OF PATIENTS WITH SUSPECTED ANGINA AND 3% OF PATIENTS WITH MYOCARDIAL INFARCTION, NORMAL EPICARDIAL CORONARY ARTERIES ARE DOCUMENTED AT ARTERIOGRAPHY (SYNDROME X). THESE PATIENTS TEND TO BE YOUNG, ARE MORE COMMONLY WOMEN, HAVE MINIMAL CARDIAC RISK FACTORS, AND HAVE A RELATIVELY GOOD PROGNOSIS. THE ANGINAL PAIN TENDS TO BE ATYPICAL. CORONARY SPASM, THROMBOSIS, OR EMBOLISM HAS BEEN SUGGESTED AS THE CAUSE IN MANY OF THESE PATIENTS

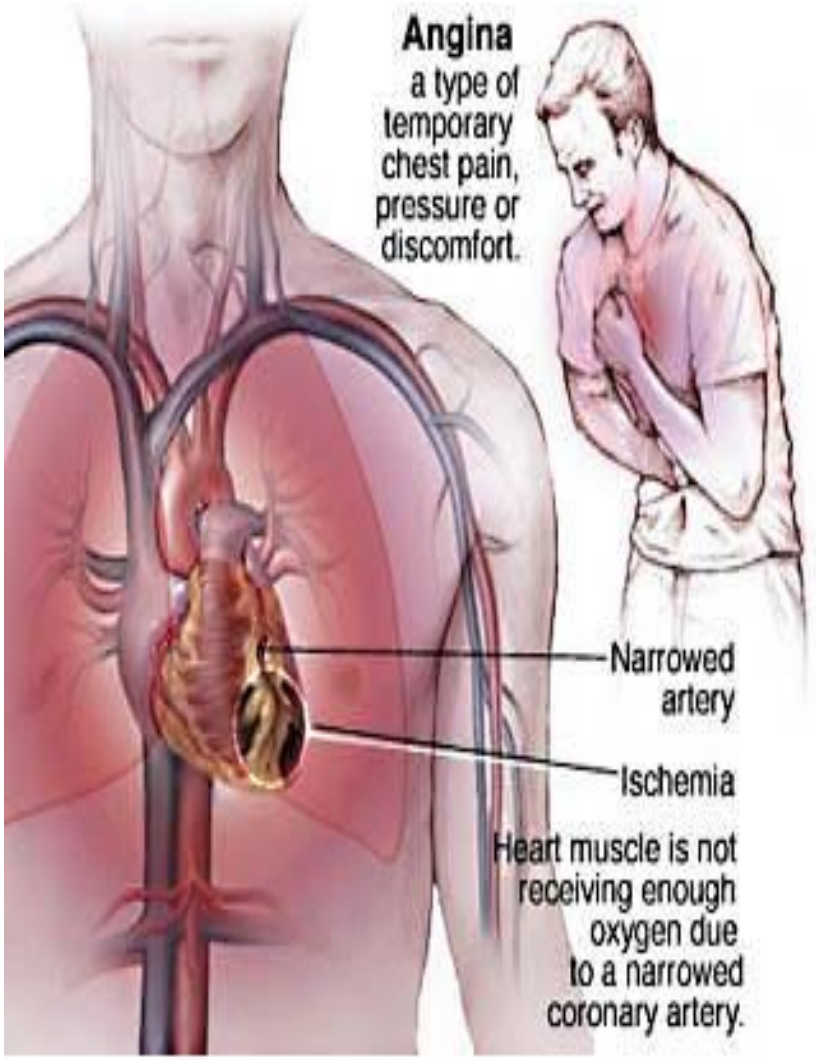
Table 9-2. Angina Pectoris

Type	Pattern	ECG	Usual Coronary Abnormality	Medical Therapy
Stable	Chronic unchanged pattern of precipitation and relief Induced by physical activity or emotional stress; lasts 5-10 min, relieved by rest or sublingual nitroglycerin	Baseline often normal or nonspecific ST-T changes, or signs of prior myocardial infarction ST segment depression or T wave inversion during angina	≥ 70% stenosis resulting from atherosclerotic plaque in one or more coronary arteries	Aspirin Sublingual nitroglycerin Anti-ischemic medications*
Unstable	Recent increase in angina frequency or severity, especially with rest pain; new-onset angina if at low activity level; angina after a myocardial infarction May last longer and be less responsive to sublingual nitroglycerin	As with stable angina, although changes during discomfort may be more pronounced Occasionally, ST segment elevation during discomfort	Fissured plaque with platelet and fibrin thrombus contribute to stenosis	Aspirin, heparin (e.g., aPTT 1.5-2 × normal) Anti-ischemic medications Glycoprotein IIb/IIIa inhibitors
Prinzmetal's or variant angina	Typically unpredictable rest pain, often in early morning hours	Transient ST segment elevation during pain (ST segment depression and/or T wave inversion can also occur)	Coronary artery spasm at a region of fixed but often nonstenotic lesion; can also occur in angiographically normal vessel	Calcium-channel blockers Nitrates Aspirin

*Long-acting nitrates, β-adrenergic blocking drugs, calcium channel-blocking drugs-see text.
aPTT = activated partial thromboplastin time.

Angina Pectoris

- **At least 70% occlusion of coronary artery resulting in pain. What kind of pain?**
  - **Chest pain**
  - **Radiating pain to:**
    - **Left shoulder**
    - ***Jaw***
    - **Left or Right arm**
- **Usually brought on by physical exertion as the heart is trying to pump blood to the muscles, it requires more blood that is not available due to the blockage of the coronary artery(ies)**
- **Is self limiting→ usually stops when exertion is ceased**

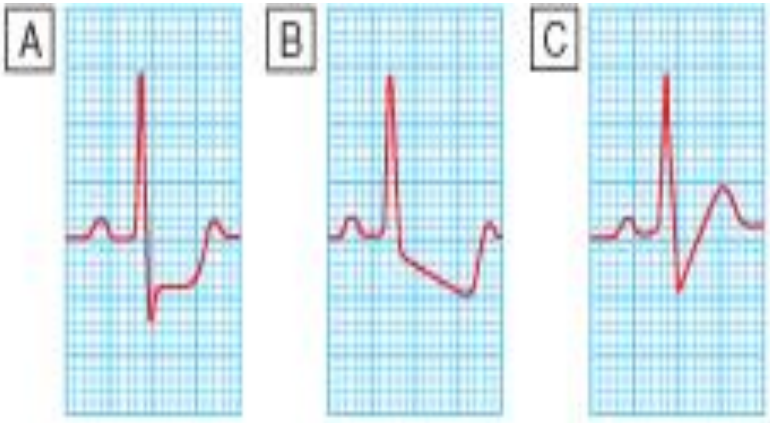


Angina Pectoris Continued

- Angina Pectoris can be Stable or Unstable:
- Stable:
  - The pain and pattern of events is unchanged over a period of time (months→ years)
- Unstable:
  - The pain and pattern is changing, be it in duration, intensity or frequency
  - A Myocardial Infarction waiting to happen

Investigations

Resting ECG The ECG may show evidence of previous myocardial infarction but is often normal even in patients with left main or severe three-vessel coronary artery disease. Occasionally, there is T-wave flattening or inversion in some leads, providing non-specific evidence of myocardial ischaemia or damage



ECG

Exercise ECG=For patients in whom the etiology of chest pain is not clear, exercise or pharmacologic stress testing may clarify the diagnosis

ECHO AND Stress echocardiography

This is an alternative to myocardial perfusion scanning and can achieve similar predictive accuracy (superior to exercise ECG). The technique uses transthoracic echocardiography to identify ischaemic segments of myocardium and areas of infarction. The former characteristically exhibit reversible defects in contractility during exercise or pharmacological stress with a dobutamine infusion; the latter typically do not contract at rest or during stress.

CT SCAN AND Coronary arteriography In contrast to the functional information provided by stress testing, coronary arteriography provides detailed anatomical information about the extent and nature of coronary artery disease (Fig. 18.63), and is usually performed with a view to coronary bypass grafting or percutaneous coronary intervention (PCI-p. 586). In some patients, diagnostic coronary angiography may be indicated when non-invasive tests have failed to elucidate the cause of atypical chest pain. The procedure is performed under local anaesthesia and requires specialised radiological equipment, cardiac monitoring and an experienced operating team. Management

Management of Ischemic Heart Disease

The approach to the management of angina involves risk factor modification, lifestyle changes, pharmacotherapy, and revascularization:

- **1-Lifestyle:**

Diet –

Exercise Preventive treatment –

- **Low fat, low cholesterol diet**

Cessation of smoking

2-LIPID LOWERING AGENT

3-ANTIPLATLETS

4-DRUGS

Table 9-5. Medications for Angina Pectoris

Drug Class	Examples	Physiologic Antianginal Effect	Side Effects	Comments
Organic nitrates	Available in sublingual, topical, intravenous, and oral preparations	↓Preload >afterload Coronary vasodilation	Headache, flushing, orthostasis	Tolerance develops with continuous use
β-Adrenergic blocking agents	Metoprolol, atenolol, propranolol, nadolol	↓Heart rate ↓Blood pressure ↓Contractility	Bradycardia, hypotension, bronchospasm, depression	May worsen heart failure and AV conduction Avoid in vasospastic angina
Calcium-channel-blocking agents				
Phenylalkylamines Benzothiazepines	Verapamil Diltiazem	Both classes produce: ↓Heart rate ↓Blood pressure ↓Contractility Coronary vasodilation	Bradycardia, hypotension; constipation with verapamil	May worsen heart failure and AV conduction
Dihydropyridines	Nifedipine, amlodipine	↓Blood pressure Coronary vasodilation	Hypotension, reflex tachycardia	Short-acting formulations may aggravate angina

VARIANT ANGINA

PAGE 97 PAGE 98 IN ADDITION TO FIXED CORONARY STENOSES, ANGINA MAY ALSO BE PRECIPITATED BY DYNAMIC CORONARY OBSTRUCTION. THIS IS THE RESULT OF CORONARY ARTERY SPASM, WHICH MAY OCCUR EITHER AT THE SITE OF AN ATHEROSCLEROTIC PLAQUE (PRINZMETAL'S ANGINA) OR IN THE SETTING OF ANGIOGRAPHICALLY NORMAL CORONARY ARTERIES (PURE VASOSPASTIC ANGINA). THE SPASM TENDS TO INVOLVE A PROXIMAL CORONARY ARTERY BUT MAY BE MORE DIFFUSE. THE CLINICAL SYNDROME IS SIMILAR TO USUAL ANGINA; HOWEVER, PATIENTS DESCRIBE THE DISCOMFORT AS A PAIN, AND EPISODES TEND TO OCCUR AT REST, FREQUENTLY IN THE MORNING HOURS, AND ARE ASSOCIATED WITH PROFOUND, TRANSIENT ST ELEVATION ON THE ECG (FIG. 9-4). MARKED ISCHEMIA MAY DEVELOP AND MAY PRECIPITATE VENTRICULAR TACHYARRHYTHMIAS AND SUDDEN CARDIAC DEATH; HOWEVER, PROGRESSION TO MYOCARDIAL INFARCTION IS RELATIVELY UNCOMMON. PATIENTS MAY NOT HAVE THE USUAL CARDIAC RISK FACTORS, ALTHOUGH SMOKING IS FREQUENT AND COCAINE USE MAY PRECIPITATE AN ATTACK. DURING CARDIAC CATHETERIZATION, CORONARY VASOSPASM MAY BE PROVOKED AFTER INTRACORONARY INFUSION OF ERGOT ALKALOIDS (ERGONOVINE) OR ACETYLCHOLINE. HYPERVENTILATION MAY ALSO BE USED AS A PROVOCATIVE TEST FOR CORONARY VASOSPASM, WITH A SENSITIVITY OF MORE THAN 90%. VARIANT ANGINA CAN BE TREATED WITH VASODILATORS, SPECIFICALLY NITRATES AND VASODILATING CALCIUM-CHANNEL BLOCKERS. NONSELECTIVE B-BLOCKERS ARE CONTRAINDICATED IN TRUE VASOSPASTIC ANGINA BECAUSE BLOCKADE OF THE VASODILATORY EFFECTS OF B_2-RECEPTOR STIMULATION MAY RESULT IN UNOPPOSED A-ADRENERGIC VASOCONSTRICTION. SIMILARLY, ASPIRIN MAY EXACERBATE VASOSPASTIC ANGINA BY INHIBITING THE PRODUCTION OF NATURALLY OCCURRING VASODILATORY PROSTAGLANDINS