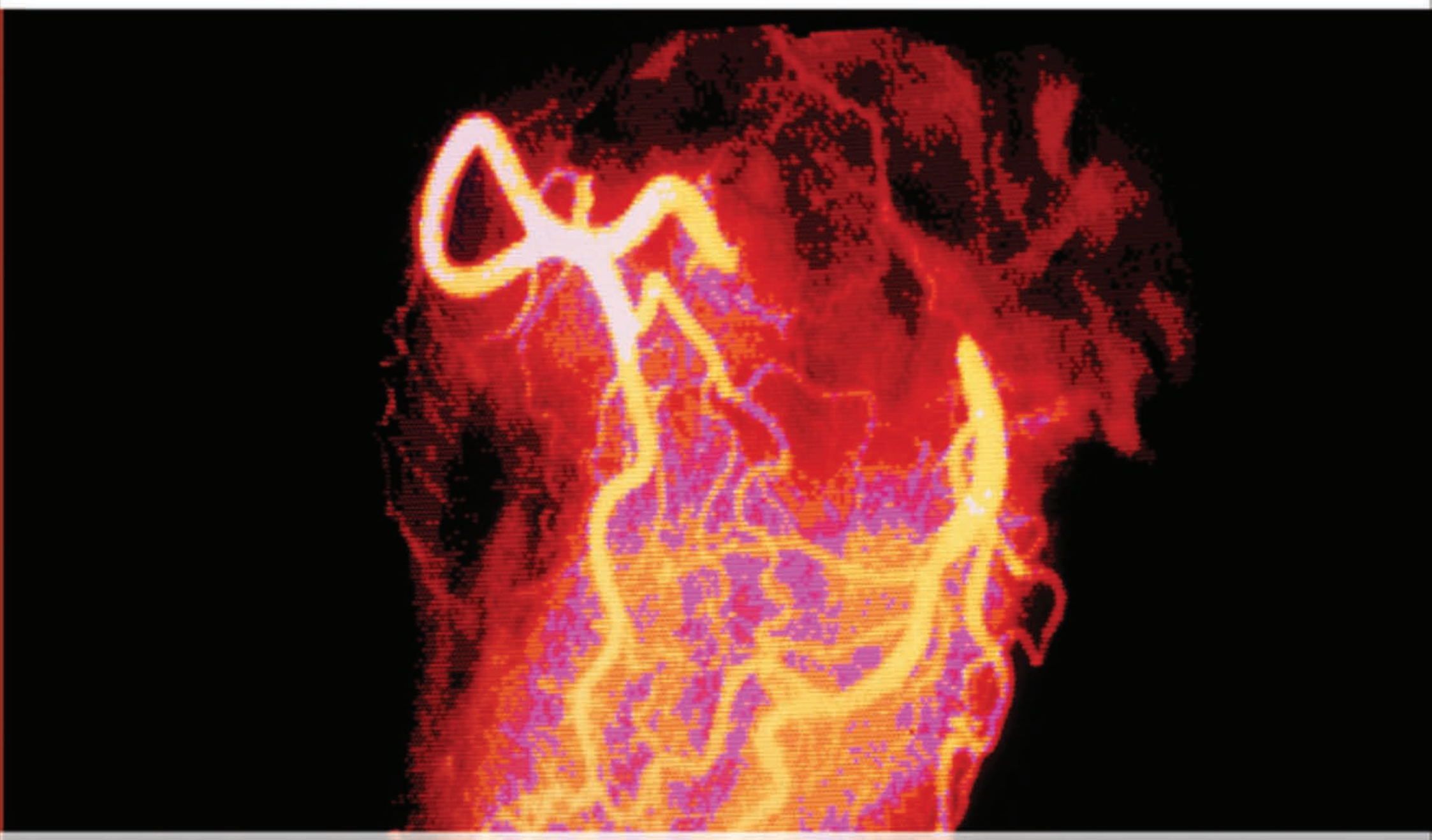
ABC of Interventional Cardiology

SECOND EDITION

Ever D. Grech



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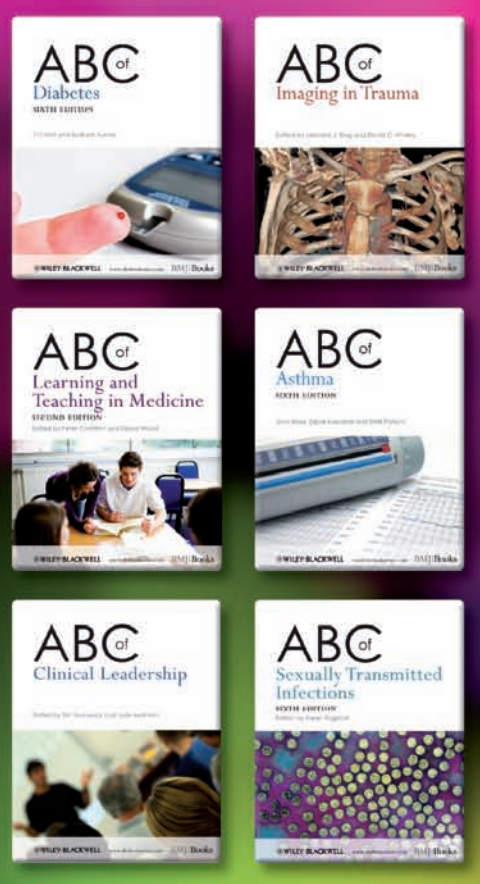

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Interventional Cardiology

Second Edition

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Preface

It is only 33 years since the first percutaneous transluminal coronary angioplasty (PTCA) was carried out by the pioneering Swiss radiologist Andreas Greuntzig in Zurich, heralding the dawn of interventional cardiology. In this short time, interventional cardiology has overcome many limitations and undergone major evolutionary changes – most notably the development of the intracoronary stent and more explicitly the drug-eluting stent. Across the world, many thousands of patients now safely undergo percutaneous coronary intervention everyday and the numbers continue to grow. In many countries, the numbers far exceed surgical bypass operations.

Although at first, PTCA was indicated only as treatment for chronic stable angina caused by a discrete, easily accessible lesion in a single coronary artery, this has now progressed enormously to encompass complex multi-lesion and multi-vessel disease. Moreover, percutaneous coronary intervention has now become widely used in the management of acute coronary syndromes (which principally include 'heart attacks') with definite benefits in terms of morbidity and mortality. The effectiveness and safety of these procedures has undoubtedly been enhanced by the adjunctive use of new anti-platelet and anti-thrombotic agents, and newer drugs are being evaluated. As drug-eluting stents address the Achilles' heel of angioplasty and stents – restenosis – the huge increase in percutaneous coronary procedures seen over recent years is likely to continue.

As the indications increase and more patients are treated, so inevitably do the demands on healthcare budgets. Although percutaneous intervention is expensive, this burden must be weighed against bypass surgery which is significantly more costly and multi-drug therapy which would be required over many years.

Although percutaneous coronary intervention has held centre stage in cardiology, major in-roads have also been made in non-coronary areas. Transcatheter valvular treatments – including actual new valve implantation, closure devices and ethanol septal ablation – have become effective and safe alternatives to surgery, as have paediatric interventional procedures. A greater understanding of cardiac electrophysiology and heart failure has led to important advances in the treatment of arrhythmias and resynchronisation therapy. Pacemakers, implantable cardioverter defibrillators (ICD) and cardiac resynchronisation therapy (CRT) are benefiting ever larger numbers of patients both in terms of life quality and mortality.

Where are we heading? This is perhaps the biggest question in the minds of many interventional cardiologists. New ideas and technology generated by industry, coupled with high levels of expertise, are fuelling advances in almost all areas of interventional cardiology. The next decade promises many new (and possibly unexpected) developments in this exciting and restless field of medicine.

In writing this book, I have endeavoured to present broad (and sometimes complex) aspects of interventional cardiology in a clear, concise and balanced manner. To this end, I have concentrated on an easy-to-read style of text, avoiding jargon and exhaustive detail where possible and supplemented with many images and graphics.

Ever D. Grech
Sheffield

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Finally, my enduring gratitude goes to my wife Lisa and our children Alexander and Frances for their unfailing encouragement, patience and love.

List of Abbreviations

CTO	Chronic total occlusion
HRT	Hormone replacement therapy
IVUS	Intravascular ultrasound
LAD	Left anterior descending (artery)
LCx	Left circumflex (artery)
Non-STEMI	Non-ST segment elevation myocardial infarction
PCI	Percutaneous coronary intervention
RCA	Right coronary artery
STEMI	ST segment elevation myocardial infarction

List of Trial Abbreviations

ACE	Abciximab and Carbostent Evaluation
ADMIRAL	Abciximab before Direct Angioplasty and Stenting in Myocardial Infarction Regarding Acute and Long-Term Follow-up
ASSENT-4	Assessment of the Safety and Efficacy of a New Treatment Strategy for Acute Myocardial Infarction
BARI	Bypass Angioplasty Revascularisation Investigation
CADILLAC	Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications
CAPITAL-AMI	Combined Angioplasty and Pharmacological Intervention Versus Thrombolytics Alone in Acute Myocardial Infarction
CAPTURE	C7E3 Antiplatelet Therapy in Unstable Refractory Angina
CARDia	Coronary Artery Revascularisation in Diabetes
CARE-HF	Cardiac Resynchronization – Heart Failure
CARESS-in-AMI	Combined Abciximab REteplase Stent Study in Acute Myocardial Infarction
CHAMPION	Cangrelor Versus Standard Therapy to Achieve Optimal Management of Platelet Inhibition
CHARISMA	Clopidogrel for High Atherothrombotic Risk and Ischemic Stabilization Management and Avoidance
CLARITY	Clopidogrel as Adjunctive Reperfusion Therapy
COMMIT	Clopidogrel and Metoprolol in Myocardial Infarction Trial
COMPANION	Comparison of Medical Therapy, Pacing, and Defibrillation in Chronic Heart Failure
COURAGE	Clinical Outcomes Utilising Revascularisation and Aggressive Drug Evaluation
CREDO	Clopidogrel for the Reduction of Events during Observation
CURE	Clopidogrel in Unstable Angina to Prevent Recurrent Events
ECSG	European Cooperative Study Group
EPIC	Evaluation of C7E3 for Prevention of Ischemic Complications
EPILOG	Evaluation in PICA to Improve Long-Term Outcome with Abciximab Glycoprotein IIb/IIIa Blockade
EPISTENT	Evaluation of Platelet IIb/IIIa Inhibitor for Stenting
ESPRIT	Enhanced Suppression of the Platelet Glycoprotein IIb/IIIa Receptor Using Integrilin Therapy
EUROPA	European Trial on Reduction of Cardiac Events with Perindopril in Stable Coronary Artery Disease
EVEREST	Endovascular Valve Edge-to-Edge Repair Study
FAME	FFR Versus Angiography for Multivessel Evaluation
FINESSE	Facilitated Intervention with Enhanced Reperfusion Speed to Stop Events
FREEDOM	Future Revascularisation Evaluation in Patients with Diabetes Mellitus: Optimal Management of Multivessel Disease
FRISC II	Fast Revascularisation during Instability in Coronary Artery Disease
GISSI	Gruppo Italiano per to Studio della Sopravvivenza nell'infarto miocardico
GUSTO	Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries
GUSTO IV ACS	Global Use of Strategies to Open Occluded Arteries IV in Acute Coronary Syndrome
HOPE	Heart Outcomes Prevention Evaluation
HORIZONS-AMI	Harmonizing Outcomes with Revascularization and Stents in Acute Myocardial Infarction
ICTUS	Invasive Versus Conservative Treatment in Unstable Coronary Syndromes Investigators

IMPACT II	Integrilin to Minimize Platelet Aggregation and Coronary Thrombosis
ISAR-COOL	Intracoronary Stenting with Antithrombotic Regimen Cooling Off
ISAR-REACT 2	Intracoronary Stenting and Antithrombotic Regimen – Rapid Early Action for Coronary Treatment 3
ISIS-2	Second International Study of Infarct Survival
JUPITER	Justification for the Use of Statins in Prevention: an Intervention Trial Evaluating Rosuvastatin
MADIT I and II	Multicenter Automatic Defibrillator Implantation Trials. The Use of Defibrillators in Primary Prevention
MIST	Migraine Intervention with Starflex Technology
MUSTT	Multicenter Unsustained Tachycardia Trial
On-TIME 2	Ongoing Tirofiban in Myocardial Infarction Evaluation
PARAGON	Platelet IIb/IIIa Antagonism for the Reduction of Acute Coronary Syndrome Events in the Global Organization Network
PEACE	Prevention of Events with Angiotensin-Converting Enzyme Inhibition
PLATO	Platelet Inhibition and Patient Outcomes
PRISM	Platelet Receptor Inhibition in Ischemic Syndrome Management
PRISM-PLUS	Platelet Receptor Inhibition in Ischemic Syndrome Management in Patients Limited by Unstable Signs and Symptoms
PROSPECT	Predictors of Response to Cardiac Resynchronization Therapy
PURSUIT	Platelet Glycoprotein IIb/IIIa in Unstable Angina: Receptor Suppression Using Integrilin Therapy
RAPPORT	Reopro and Primary PTCA Organization and Randomized Trial
RAVEL	Randomised Study with the Sirolimus-Eluting Velocity Balloon-Expandable Stent in the Treatment of Patients with De Novo Native Coronary Artery Lesions
RESTORE	Randomized Efficacy Study of Tirofiban for Outcomes and Restenosis
RITA 3	Randomised Intervention Treatment of Angina
SCD-Heft	Sudden Cardiac Death in Patients with Heart Failure
SHOCK	Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock
SIRIUS	Sirolimus-Coated Velocity Stent in Treatment of Patients with De Novo Coronary Artery Lesions Trial
Stent-PAMI	Stent Primary Angioplasty in Myocardial Infarction
SYNTAX	Synergy between PCI with Taxus and Cardiac Surgery
TACTICS-TIMI 18	Treat Angina with Aggrastat and Determine Cost of Therapy with an Invasive or Conservative Strategy – Thrombolysis in Myocardial Infarction
TAMI	Thrombolysis and Angioplasty in Myocardial Infarction
TIMI IIIB	Thrombolysis in Myocardial Infarction IIIB
TRANSFER-AMI	Trial of Routine Angioplasty and Stenting after Fibrinolysis to Enhance Reperfusion in Acute Myocardial Infarction
TRITON-TIMI 38	Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition with Prasugrel – Thrombolysis in Myocardial Infarction
TRUCS	Treatment of Refractory Unstable Angina in Geographically Isolated Areas without Cardiac Surgery
VANQWISH	Veterans Affairs Non-Q-Wave Infarction Strategies in Hospital
VINO	Value of First Day Coronary Angiography/Angioplasty in Evolving Non-ST Segment Elevation Myocardial Infarction
WHO MONICA	World Health Organisation: Monitoring Trends and Determinants in Cardiovascular Disease

CHAPTER 1

Modifying Risk Factors to Improve Prognosis

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OVERVIEW

- Certain personal characteristics and lifestyles point to increased likelihood of coronary heart disease and are called *risk factors*
- The three principal modifiable risk factors are smoking, hypercholesterolaemia and hypertension. Other modifiable factors linked to lifestyle include a saturated-fat-rich diet, obesity and physical inactivity
- Prevention strategies (primary or secondary prevention) aim to reduce the risk of developing or retard the progression of atheroma, to stabilise plaques and to reduce the risk of their erosion or rupture. These measures can collectively reduce the risk of future cardiovascular events (mortality, myocardial infarction and strokes) by as much as 75–80%
- Percutaneous coronary intervention (PCI) or coronary artery bypass graft (CABG) revascularisation is not a cure for coronary heart disease and they are predominantly carried out to improve symptoms. They may have little or no prognostic impact in chronic stable angina. However, CABG and PCI confer significant short- and long-term mortality benefit in acute coronary syndromes and, in particular, primary PCI for acute ST segment elevation myocardial infarction

In affluent societies, coronary artery disease causes severe disability and more deaths than any other disease including cancer. It manifests itself as silent ischaemia, angina, unstable angina, myocardial infarction, arrhythmias, heart failure and sudden death. Although this is the result of atheromatous plaque formation and its effect, the actual cause of this process is not known. However, predictive variables – known as *risk factors* – have been identified which increase the chance of its early development. Risk factors can be classified as modifiable and non-modifiable (Table 1.1).

It is clearly not possible to prevent the increased risk associated with ageing, a positive family history or male gender. However, there are many factors which can be usefully ameliorated by interventions. Moreover, there are some aspects of lifestyle that have been shown to reduce the risk of an acute myocardial infarction.

Table 1.1 Risk factors for the development of premature ischaemic heart disease and acute myocardial infarction.

Risk factor	RR*	Modifiable	Not modifiable	RR for AMI†	PAR for AMI (%)†
Smoking	5.1	√	–	2.87‡	35.7‡
Age	4.7	–	√	–	–
Abnormal lipids	3.1	√	–	3.25	49.2
Hypertension	3.1	√	–	1.91	17.9
Diabetes	2.0	√	–	2.37	9.9
Male sex	2.0	–	√	–	–
Obesity	1.8	√	–	1.12	20.1
Positive family history	1.5	–	√	–	–
Psychosocial factors	–	–	√	2.67	32.5
5× daily fresh fruits/vegetables	–	√	–	0.70	13.7
Regular alcohol	–	√	–	0.91	6.7
Regular exercise	–	√	–	0.86	12.2

Uncertain risk factors include: hypertriglyceridaemia, lipoprotien (a), microalbuminuria, uric acid, renin, fibrinogen, C-reactive protien and hyperhomocyteinaemia.
*From Steeds. RR, Relative risk.
†From INTERHEART case-control study. Yusuf S *et al. Lancet* 2004;**364**: 937–52.
‡For current and former smokers.
RR for AMI: Relative risk for acute myocardial infarction. PAR for AMI(%): Population attributable risk for acute myocardial infarction.
Notes: These 9 risk factors accounted for 90% of the population attributable risk in men and 94% in women. Psychosocial factors included depression, stress at work or at home, moderate/severe financial stress, one or more recent life events, low control score. The control population was drawn from hospital in-patients with non-cardiac conditions (58%) and community-based hospital visitors (36%). A minority were WHO MONICA controls (3%) and unknown (3%).

Risk factors are not simply additive but may be synergistically cumulative. Data from epidemiological surveys have shown for some time that combinations of risk factors generate exponential risks (Figures 1.1 and 1.2). This applies to both men and women. Risk factors are not static but increase with age – this may partly explain the independent effect of age. Blood pressure increases normally with age, so whatever definition is used for hypertension, the frequency of this condition will increase with age. Cholesterol and triglycerides increase with age as do insulin resistance and body mass index.

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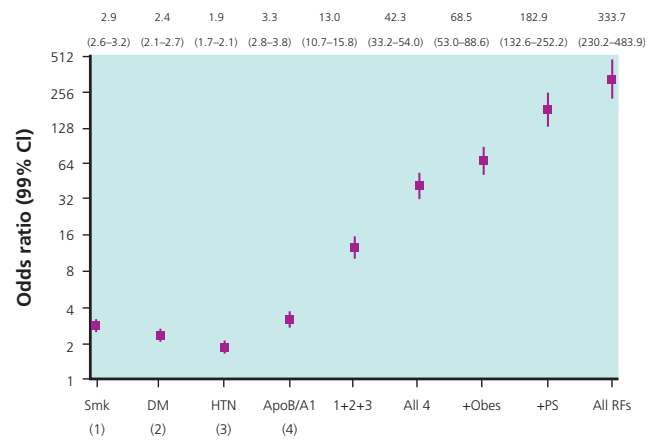

Figure 1.1 The adverse effect of single and combined risk factors on the risk of acute myocardial infarction. Smk, smoking; DM, diabetes mellitus; HTN, hypertension; ApoB/A1, lipid abnormalities; Obes, obesity; PS, psychosocial factors; RFs, risk factors. From INTERHEART case-control study. Yusuf S *et al. Lancet* 2004;**364**:937–52.

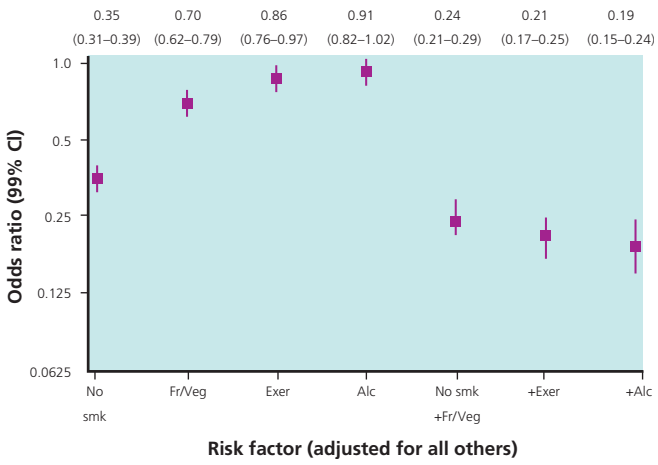

Figure 1.2 The beneficial effect of single and combined risk factors on the risk of acute myocardial infarction. No smk, no smoking; Fr/Veg, daily 5 fresh fruits/vegetables; Exer, regular exercise; Alc, regular alcohol. From INTERHEART case-control study. Yusuf S *et al. Lancet* 2004;**364**:937–52.

Impact of risk factors

Smoking

Smoking confers a fivefold relative risk for acute myocardial infarction and cardiovascular death. By comparison, stopping smoking has an almost immediate effect on reducing the cardiovascular risk by about 50%. Ex-smokers still have a higher risk than lifelong non-smokers. In one study, the survival rate of patients who stopped smoking after an acute myocardial infarction at 8 years of follow-up was about 75% compared with 60% for patients who continued to smoke. Similarly reinfarction is about twice as common in smokers than in those who stop smoking after a first infarction. At 8 years of follow-up, reinfarction was about 38% in smokers compared with 22% in quitters. Overall smoking increases mortality by about 2.5 times and reduces absolute survival by, on average, 10 years.

Hyperlipidaemia

High blood cholesterol is associated with an increased cardiovascular risk. However, as a single risk factor it is relatively weak – it becomes more important when associated with smoking, hypertension and diabetes. There is also an important interaction with age. In men, there is a doubling of risk from serum cholesterol in the lowest population quintile (<200 mg/dl; 5.2 mmol/l) to the highest (>260 mg/dl; >6.7 mmol/l).

Hypertension

Both diastolic and systolic hypertension have been shown to be risk factors for myocardial infarction and cardiovascular death. The relative risk of persistently elevated blood pressure of >160 mmHg systolic is 4 times the risk compared with systolic blood pressure of <120 mmHg.

The relative risk of persistently elevated diastolic blood pressure >100 mmHg is 3 times higher when compared with a diastolic pressure of <80 mmHg. Research data have shown that reduction in diastolic pressure of 5–6 mmHg and systolic pressure of 10–14 mmHg over 5 years with drug therapy does reduce cardiac mortality and non-fatal myocardial infarction in elderly people by about 20%, and in younger people by about 14%. Data from the longitudinal epidemiological study in Framingham showed that left ventricular hypertrophy diagnosed by echocardiography is associated with a twofold increased risk in death in women and a 1.5-fold increased risk in men over a 4-year period.

Diabetes mellitus

This is a major risk factor for premature vascular disease, stroke, myocardial infarction and death. Diabetes increases the risk of developing coronary heart disease by 1.5 times at age 40–49 and by 1.7 times at age 50–59 in men and by 3.7 times at age 40–49, and 2.4 times at age 50–59 in women. There are data that show that diabetic control is important for cardiovascular risk, with correlations between cardiovascular events, ischaemic heart disease and death rate and glycosylated haemoglobin. Much more effective risk reduction is associated with aggressive treatment of the commonly associated hypertension, lipid abnormalities and obesity in the diabetic patient.

Obesity

Obesity has been increasing in epidemic proportions and confers a prognostic disadvantage. Those with body mass index (weight/ht^2) of 25–29 kg/m^2 are considered to be overweight and those >32 are classified as obese. The latter have a twofold relative increase in mortality from all causes and a threefold increase in cardiovascular death. One study showed that a high body mass index was associated with an increase risk of death per se, especially when it was present in young people aged 30–44 years. More recent evidence suggests that waist circumference is an important independent risk factor as truncal or visceral obesity appears to be more atherogenic. An expanded waist circumference is a necessary criterion for the diagnosis of the metabolic syndrome, in addition to at least two of the other four criteria (Table 1.2).

Table 1.2 International Diabetes Federation definition of metabolic syndrome – focus on waist circumference.

Abdominal obesity plus at least two of the following:	>94 cm male, >80 cm female
Elevated triglycerides	≥1.7 mmol/l
Reduced HDL-cholesterol	<1.0 mmol/l male, 1.3 mmol/l female
Raised blood pressure	>130/80 mmHg
Raised fasting plasma glucose	≥5.6 mmol/l

HDL, High-density lipoprotein.

Despite the presence of the *obesity paradox* – overweight and obese patients with established cardiovascular disease seem to have a more favourable prognosis than leaner patients – there is data to support purposeful weight reduction in the prevention and treatment of cardiovascular diseases. Furthermore, interventional trials involving bariatric surgery for severe obesity have shown that significant weight reduction resulted in significantly reduced mortality.

Physical activity and fitness

There is a close inverse relationship between cardiorespiratory fitness and cardiac outcomes such as coronary disease and death. This can be readily assessed by exercise tolerance testing. Patients with a low level of cardiorespiratory fitness have a 70% higher risk for all-cause mortality and a 56% higher risk for coronary or cardiovascular events compared with those with a high level of fitness. Those with intermediate levels of fitness have a 40% higher mortality risk and a 47% higher coronary or cardiovascular event rate than those with higher fitness. Following acute myocardial infarction or coronary artery bypass graft (CABG), cardiac rehabilitation programmes that promote exercise and weight loss can improve cardiometabolic risk profiles of patients.

Gender

Men have twice the cardiovascular mortality as women at all ages and in all parts of the world. This was thought to be related to the beneficial effect of female sex hormones, especially oestrogens, as the cardiovascular risk in women increases after the menopause. However, two large randomised controlled trials showed that hormone replacement therapy (HRT) did not reduce the cardiovascular risk in women; rather, the thrombotic effects of oestrogens precipitated fatal and non-fatal cardiovascular events, especially in the early years of treatment. Women appear to possess differently weighted risk factors than men for reasons that are unclear.

More recent data have shown strong associations of accelerated atherosclerosis with low levels of testosterone in men followed up for 4–8 years. Low testosterone level in men has been shown to be linked with increased mortality. Male HRT has not yet been shown to reduce cardiovascular risk, although results from animal studies are encouraging.

Psychosocial factors

Some psychosocial factors double the risk of developing cardiovascular disease. Social class has an important effect on mortality from heart disease with people in low-income groups having an excess mortality compared with high-income earners. This is not simply related to deprivation. Within the same working cohort (e.g. Whitehall civil servants), cardiovascular events and mortality were found to be 2–3 times higher in those workers with low socioeconomic status compared with those with high socioeconomic status. In fact, there is little relationship between actual average income and life expectancy. It is not just a matter of money. Mortality is 2–3 times higher in people with poor social links than in those with good social support networks. The reasons are unclear but they are not explained by differences in other known risk factors such as smoking.

Depression

Depression carries an adverse prognosis, especially in association with coronary artery disease and is associated with an eightfold increase in cardiovascular death. Patients with depression have a fivefold increased mortality after acute myocardial infarction. There are no data to suggest that treatment of depression with any specific therapy reverses the excess mortality. Depression also influences the outcome after coronary artery bypass surgery. After controlling for age, sex, number of grafts, diabetes, smoking, left ventricular ejection fraction and previous myocardial infarction, moderate or severe depression at the time of surgery increased the risk of death by 2.4 times, and mild to moderate depression that persisted for 6 months conferred a 2.2 times increased risk of death, during a 5-year follow-up period.

How to assess cardiovascular risk

Cardiovascular risk stratification is carried out through clinical history, physical examination and serum biomarkers. Following extensive validation, tools such as the Framingham or Reynolds risk scores have been adopted in clinical practice by most primary care practitioners. These scores can identify patients with established risk factors who are at greater risk and would most likely benefit from primary prevention. There are also a number of risk estimates that can be provided electronically from the internet (www.riskscore.org.uk; www.bhsoc.org) that have used large populations on which to base risk assessment. They may have some limitations as they are spot estimates that are critically dependent on age as well as actual measurements of blood pressure and cholesterol – which can fluctuate.

More recently, non-invasive imaging of coronary plaque using cardiac magnetic resonance (CMR) and calcification with measurement of coronary calcium using multislice computed tomography (MSCT) scanning have also been used to identify higher risk populations. However, it is as yet uncertain whether treatment modification in this group will result in improved clinical outcome.

Effects of drug treatments

There are two distinct groups of patients who are treated with drug therapy. The first includes those with risk factors for the

development of premature vascular disease who do not as yet have overt disease, and is categorised as *primary prevention*. The second includes those patients who have overt cardiovascular disease, such as previous myocardial infarction, peripheral vascular disease and stroke, and is categorised as *secondary prevention*. The physician must weigh up the risks and benefits of treatment in each individual patient. For example, in patients with overt vascular disease the threshold for drug treatment is much lower because there is a higher benefit to risk ratio from the known drug treatment. In those patients who are at risk but who do not yet have overt disease, the risks may outweigh the benefits especially if the overall likelihood of a cardiovascular event is small. Age has a large effect here as the risk of developing vascular disease increases exponentially over the age of 65. Moreover, the absolute risk of an event increases with age, so decisions about the appropriateness of primary prevention need to be reviewed on a regular basis as the patient ages. There are risk calculators available to help the physician make treatment decisions.

Aspirin

Aspirin reduces platelet activation by the inhibition of cyclooxygenase-1 (COX-1) enzyme in platelets, blocking the synthesis of prostaglandin G2/H2 and thromboxane A_2. It is the most commonly prescribed drug for the prevention of atherothrombotic events. Its use in patients early after acute myocardial infarction is associated with a reduction in mortality of about 25% (ISIS-2 study). When used in patients with chronic stable angina, there is some evidence that myocardial infarction and sudden death as a combined end point is reduced by about 30%. The benefit is seen almost immediately on starting the drug. However, the benefit of aspirin is to postpone events and not to prevent them. By comparing the event rate in patients taking aspirin and placebo, it is possible to estimate the delay in events conferred by the drug. The average benefit is a delay in event rate of maximum 24 months with aspirin. Aspirin for primary prevention remains controversial as the relatively small benefit is offset by gastrointestinal problems such as bleeding.

Clopidogrel

A thienopyridine derivative, clopidogrel prevents adenosine diphosphate (ADP)-mediated activation of platelets, thereby blocking activation of the glycoprotein IIb/IIIa complex.

In terms of primary prevention, clopidogrel offers no benefit over aspirin and may even cause harm. In the CHARISMA study, a long-term trial of aspirin combined with clopidogrel versus aspirin alone, there was no significant benefit over aspirin alone and a suggestion of harm in those patients who had risk factors for cardiovascular disease compared with those who had overt disease. However, in patients with overt vascular disease, the drug has been shown to reduce cardiovascular events by about the same degree as aspirin.

In the setting of acute non-ST segment elevation acute coronary syndome, patients had fewer ischaemic end points when treated with the combination of clopidogrel and aspirin compared with aspirin alone, irrespective of whether percutaneous coronary intervention (PCI) was performed or not (CURE study). In the setting of acute ST segment myocardial infarction treated with aspirin and thrombolytic therapy, the addition of clopidogrel for 1 month conferred a small but significant benefit at 1 month (CLARITY and COMMIT studies).

Cholesterol-lowering drugs

Statins (3-hydroxy-3-methylglutaryl-coenzyme A (HMG CoA) inhibitors) have been shown to reduce all-cause mortality and cardiovascular events (acute myocardial infarction, angina, stroke) in both primary and secondary prevention of cardiovascular disease (Figure 1.3).

In a meta-analysis involving over 70,000 patients without established cardiovascular disease but with cardiovascular risk factors, statin therapy was associated with a significant risk reduction in all-cause mortality of 12%, in major coronary events of 30% and in major cerebrovascular events of 19%. Moreover, statin use was not associated with an increased risk of cancer.

Statins may have additional antiplatelet and anti-inflammatory benefits. Recently, the JUPITER study showed that rosuvastatin significantly reduced the incidence of major cardiovascular events in apparently healthy people without hyperlipidaemia, but elevated high-sensitivity C-reactive protein (hs-CRP). The proposal that an elevated hs-CRP may be a risk marker or risk factor remains uncertain. Statins have no proven benefit in patients with heart failure.

For patients with clinical evidence of cardiovascular disease (previous myocardial infarction or stroke), large-scale trials have indicated that the baseline annual risk of death is about 3%, which is reduced to 2.5% by taking simvastatin. Similarly, long-term registry studies of patients after coronary bypass surgery have shown that average (50%) survival is about 17 years, which is almost 3% per year. In these registry studies, it is also clear that other factors impact on survival after an event – especially the degree of left ventricular damage and the burden of coronary artery disease (number of diseased vessels). Similarly co-morbidity relating to disease in other organ systems adversely affects survival – especially the presence of diabetes and chronic renal dysfunction.

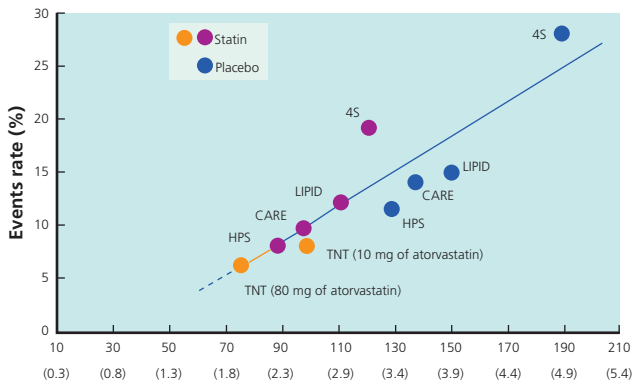

Figure 1.3 Cardiovascular event rates in secondary prevention studies. LIPID, Long-term intervention with pravastatin in ischaemic disease; 4S, Scandinavian simvastatin survival study; CARE, cholesterol and recurrent events; HPS, heart protection study; TNT, treating to new targets; LDL-C, low-density lipoprotein cholesterol.

Supplemental treatment with *n*-3 polyunsaturated fatty acids has also been shown to reduce mortality in patients after acute myocardial infarction, although the effect was small and only 5% of this study population were taking statins at baseline.

β-Adrenoceptor blocking drugs

These drugs reduce mortality by about 10–15% at the time of acute myocardial infarction and have also been shown to reduce late mortality after myocardial infarction by about 20–25%. However, in the setting of chronic stable angina there is no evidence that β-blockers reduce the incidence of myocardial infarction or prolong survival.

Angiotensin-converting enzyme (ACE) inhibitors

These drugs have proven benefit in reducing cardiovascular death both in heart failure and following acute myocardial infarction. In patients with stable coronary disease but without heart failure or left ventricular dysfunction, HOPE and EUROPA studies have shown that patients could gain additional cardiovascular protection with an angiotensin-converting enzyme (ACE) inhibitor. However, in the PEACE study, trandolapril failed to provide any further benefit in terms of death from cardiovascular causes, myocardial infarction or coronary revascularisation. These negative results could be explained by the fact that the study did not include patients with diabetes or high cardiovascular risk, 70% of patients were taking lipid-lowering therapies, more than 90% were treated with aspirin and many patients had undergone prior revascularisation. In a subsequent meta-analysis of these and other studies, ACE inhibitors conferred a significant benefit in reducing mortality, myocardial infarction, stroke and revascularisation. It is therefore currently recommended that ACE inhibitors should be considered for all patients with coronary artery disease. However, this is optional in lower risk patients in whom cardiovascular risk factors are well controlled and revascularisation has been performed.

There are data to show that hypertensive patients treated with ACE inhibitors develop atrial fibrillation less frequently compared with those taking other anti-hypertensive drugs including β-blockers, for example.

Effects of coronary artery revascularisation

In patients with chronic stable angina, CABG surgery may improve prognosis in some subgroups of patients, when compared to medical therapy. However, the benefit of this treatment is small. Evidence from rather dated randomised controlled trials of surgery versus medical treatment has shown the following:

- *Patients with significant left main stem stenosis*: Survival over a 10-year period was increased by an average of 19 months.
- *Patients with significant stenoses of three vessels*: Survival was extended by <6 months.
- *Patients with single or two significant coronary artery stenosis*: Survival was extended by only 1 month.
- *Patients with impairment of left ventricular function*: Survival was extended for about 8 months longer after surgery.

The recent SYNTAX trial of patients with severe coronary artery disease (including severe left main stem disease) showed that at 1 year, CABG was superior to PCI in terms of composite outcome of death, myocardial infarction, stroke and repeat interventions. Repeat revascularisation was significantly higher in the PCI group, and most of these patients were treated with PCI rather than CABG. However, mortality per se was similar in both groups (4.4% PCI vs 3.5% CABG) and the stroke rate was nearly 4 times higher in the CABG group (2.2% vs 0.6% PCI). Longer term follow-up may further clarify the relative benefits of these two procedures.

In patients with acute coronary syndromes (unstable angina, non-ST segment elevation myocardial infarction and ST segment elevation myocardial infarction), the combined end point of myocardial infarction and mortality is reduced by timely intervention by either early PCI or CABG. A review of the clinical trial evidence shows that those patients at highest risk benefit most. In the setting of acute ST segment elevation myocardial infarction, a meta-analysis has shown that primary PCI confers significant mortality and recurrent myocardial infarction benefits.

Conclusion

Although cardiovascular disease continues to exert major socio-economic consequences, there has been a substantive fall in death rates from coronary heart disease over the past decades. Recent evidence highlights the crucial impact of risk factor modification by way of primary and secondary prevention, revascularisation strategies, as well as the modern care of acute coronary syndromes, which incorporates early PCI/CABG policies. The dividing wall between secondary and primary prevention appears to be less significant than before as emerging data highlights the trend towards multiple risk factor modification for all groups. As lowering all risk factors simultaneously has a multiplicative effect in reducing risk, some groups are exploring the interesting potential of a single daily, multi-drug tablet (referred to as the *polypill*). This will include aspirin and a lower dose statin, an ACE inhibitor and a β-blocker for all those above 55 years, diabetics above 35 years and any ages in those with known coronary artery or cerebrovascular disease.

Further reading

Baigent C, Keech A, Kearney PM *et al*. Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of data from 90,056 participants in 14 randomised trials of statins. *Lancet* 2005;**366**: 1267–78.

Brugts JJ, Yetgin T, Hoeks SE *et al*. The benefits of statins in people without established cardiovascular disease but with cardiovascular risk factors: meta-analysis of randomised controlled trials. *Br Med J* 2009;**338**:b2376.

Channer KS, Jones TH. Cardiovascular effects of testosterone – implications for the ''male menopause''. *Heart* 2003;**89**:121–2.

Danchin N, Cucherat M, Thuillez C *et al*. Angiotensin-converting enzyme inhibitors in patients with coronary artery disease and absence of heart failure or left ventricular systolic dysfunction. An overview of long-term randomized controlled trials. *Arch Intern Med* 2006;**166**:787–96.

Kodama S, Saito K, Tanaka S *et al*. Cardiorespiratory fitness as a quantitative predictor of all-cause mortality and cardiovascular events in healthy men and women. *J Am Med Assoc* 2009;**301**:2024–35.

Lavie CJ, Milani RV, Ventura HO. Obesity and cardiovascular disease. Risk factor, paradox, and impact of weight loss. *J Am Coll Cardiol* 2009;**53**: 1925–32.

Lewington S, Whitlock G, Clarke R *et al*. Blood cholesterol and vascular mortality by age, sex and blood pressure: a meta-analysis of individual data from 61 prospective studies with 55,000 vascular deaths. *Lancet* 2007;**370**: 1829–39.

Sjöström L, Narbro K, Sjöström CD *et al*. Effects of bariatric surgery on mortality in Swedish obese subjects. *N Engl J Med* 2007;**357**:741–52.

Smith SC, Allen J, Blair SN *et al*. AHA/ACC guidelines for secondary prevention for patients with coronary and other atherosclerotic vascular disease: 2006 update. *J Am Coll Cardiol* 2006;**47**:2130–9.

Yusuf S, Hawken S, Ounpuu S *et al*. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case control study. *Lancet* 2004;**364**:937–52.

CHAPTER 2

Pathophysiology and Investigation of Coronary Artery Disease

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OVERVIEW

- Coronary artery disease is the leading cause of death in affluent societies and is usually caused by atheroma causing stenosis or total occlusion
- All patients with definite or possible angina should be referred to a cardiologist where possible
- Effective lifestyle and risk factor modification, as well as optimisation of medical therapy are important general practitioner (GP) roles
- Non-invasive and invasive investigations aim to confirm the diagnosis of angina, provide risk stratification and guide suitability for revascularisation (Figure 2.1)

Pathophysiology

Coronary artery disease is almost always due to atheromatous narrowing and subsequent occlusion of the vessel (Figure 2.2). Early atheroma – from the Greek *athera* (porridge) and *oma* (lump) – may be present from young adulthood onwards. A mature plaque is composed of two constituents, each associated with a particular cell population. The lipid core is mainly released from necrotic 'foam cells' – monocyte-derived macrophages – which migrate into the intima and ingest lipids. The connective tissue matrix is derived from smooth muscle cells, which migrate from the media into the intima, where they proliferate and change their phenotype to form a fibrous capsule around the lipid core (Figure 2.3).

When a plaque produces a >50% diameter stenosis (or >75% reduction in cross-sectional area), reduced blood flow through the coronary artery during exertion may lead to ischaemia and anginal symptoms. The degree of angina may vary considerably between individuals.

Acute coronary events usually arise when thrombus formation follows disruption of a plaque. Intimal injury causes denudation of the thrombogenic matrix or lipid pool and triggers thrombus formation causing subtotal occlusion of the artery, which may precipitate unstable angina. Downstream embolism of this thrombus may produce microinfarcts, resulting in acute non-ST segment elevation myocardial infarction. In acute ST segment elevation myocardial infarction, occlusion is more complete.

ABC of Interventional Cardiology, 2nd edition.

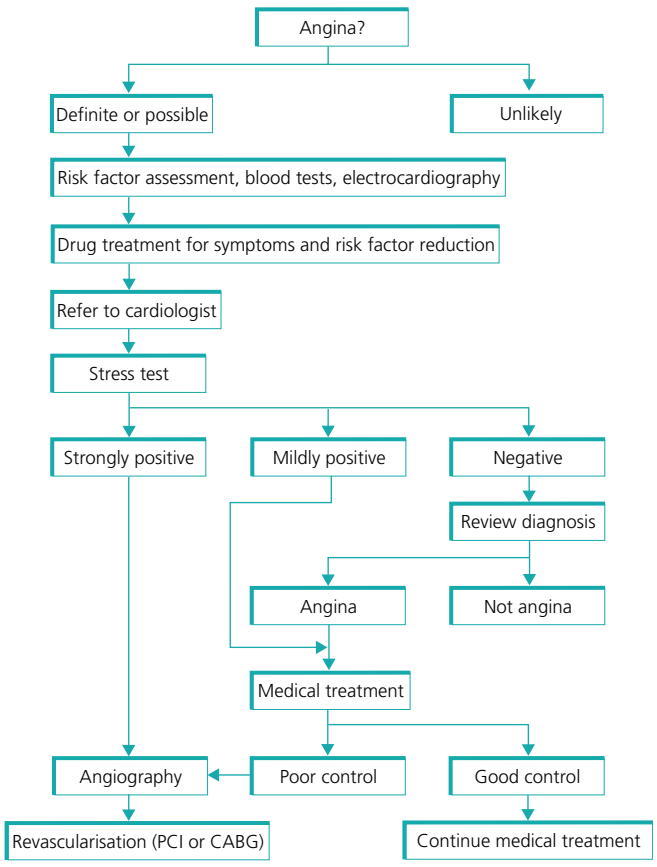


Figure 2.1 Algorithm for management of suspected angina. PCI, percutaneous coronary intervention; CABG, coronary artery bypass grafting.

Investigations

Patients presenting with chest pain may be identified as having definite or possible angina from their history alone. In the former group, risk factor assessment should be undertaken, both to guide diagnosis and because modification of some associated risk factors can reduce cardiovascular events and mortality. A blood count, biochemical screen and thyroid function tests may identify extra factors underlying the onset of angina. Initial drug treatment should include aspirin, a β-blocker and a nitrate. Anti-hypertensive and lipid-lowering drugs may also be given, in conjunction with advice on lifestyle and risk factor modification.

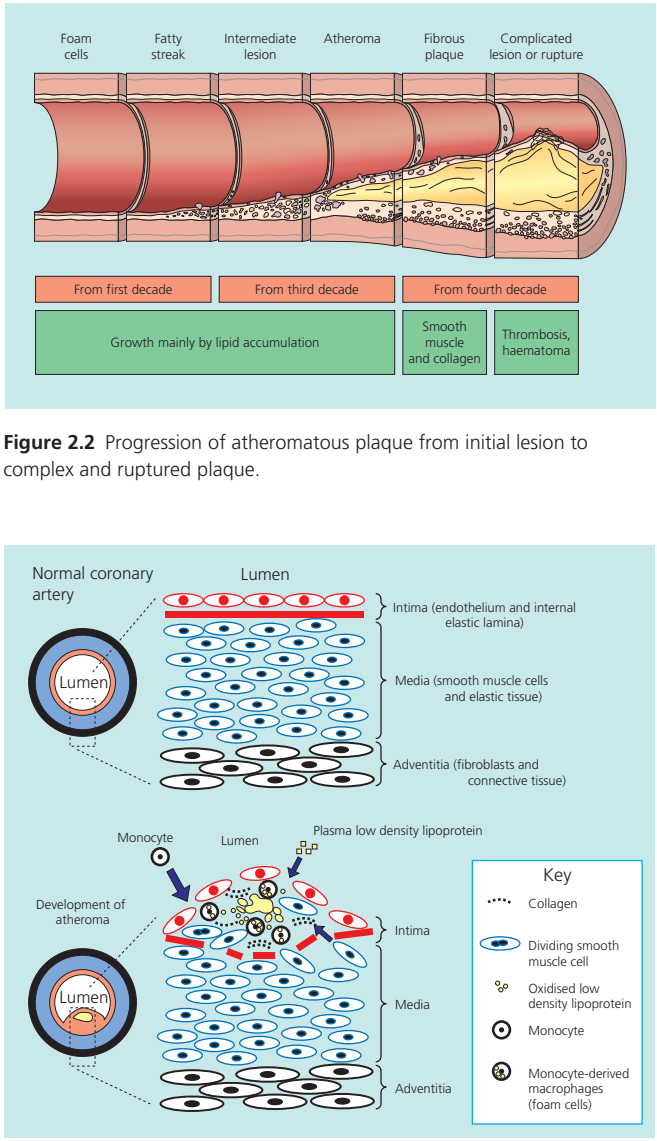


Figure 2.2 Progression of atheromatous plaque from initial lesion to complex and ruptured plaque.

Figure 2.3 Schematic representation of normal coronary artery wall (top) and development of atheroma (bottom).

All patients should be referred to a cardiologist to clarify the diagnosis, optimise drug treatment and assess the need and suitability for revascularisation (which can improve both symptoms and prognosis). Patients should be advised to seek urgent medical help if their symptoms occur at rest or on minimal exertion and if they persist for more than 10 minutes after sublingual nitrate has been taken, as these may herald the onset of an acute coronary syndrome (Table 2.1).

Table 2.1 Priorities for cardiology referral.

Recent onset of symptoms
Rapidly progressive symptoms
Possible aortic stenosis
Severe symptoms (minimal exertion or nocturnal angina)
Angina refractory to medical treatment
Threatened employment

Non-invasive investigations

Electrocardiography

An abnormal electrocardiogram increases the suspicion of significant coronary disease, but a normal result does not exclude it.

Chest X-ray

Patients with angina and no prior history of cardiac disease usually have a normal chest X-ray.

Exercise electrocardiography

This is the most widely used test in evaluating patients with suspected angina (Table 2.2). It is generally safe (risk ratio of major adverse events is 1 in 2500, and of mortality is 1 in 10 000) and provides diagnostic as well as prognostic information. The average sensitivity and specificity is 75%. The test is interpreted in terms of achieved workload, symptoms and electrocardiographic response. A 1-mm depression in the horizontal ST segment is the usual cut-off point for significant ischaemia (Figure 2.4). Poor exercise capacity, an abnormal blood pressure response and profound ischaemic electrocardiographic changes are associated with poor prognosis (Table 2.3a and 2.3b).

Stress echocardiography

Stress-induced impairment of myocardial contraction is a sensitive marker of ischaemia and precedes electrocardiographic changes and angina. Cross-sectional echocardiography can be used to evaluate regional and global left ventricular impairment during ischaemia, which can be induced by exercise or an intravenous infusion of drugs that increase myocardial contraction and heart rate (such as dobutamine) or dilate coronary arterioles (such as dipyridamole or adenosine). The test has a higher sensitivity and specificity than exercise electrocardiography and is useful in patients whose physical condition limits exercise.

Table 2.2 Exercise stress testing.

Indications	Contraindications
Confirmation of suspected angina	Acute cardiac failure
Evaluation of extent of myocardial ischaemia and prognosis	Any feverish illness
Risk stratification after myocardial infarction	Left ventricular outflow tract obstruction or hypertrophic cardiomyopathy
Detection of exercise-induced symptoms (such as arrhythmias or syncope)	Severe aortic or mitral stenosis
Evaluation of outcome of interventions (such as PCI or CABG)	Uncontrolled hypertension
Assessment of cardiac transplant	Pulmonary hypertension
Rehabilitation and patient motivation	Recent myocardial infarction
	Severe tachyarrhythmias
	Dissecting aortic aneurysm
	Left main stem stenosis or equivalent
	Complete heart block (in adults)

PCI, percutaneous coronary interventions; CABG, coronary artery bypass graft.

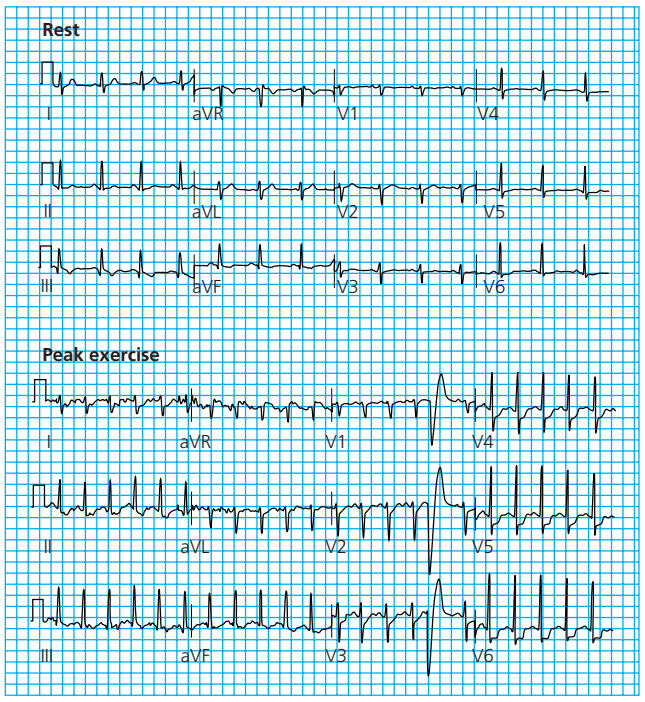


Figure 2.4 Example of strongly positive exercise stress test. After only 2 minutes and 24 seconds of exercise (according to Bruce protocol), the patient developed chest pain and electrocardiography showed marked ischaemic changes (maximum 3-mm ST segment depression in lead V6).

Table 2.3a Main end points for exercise electrocardiography.

Target heart rate achieved (>85% of maximum predicted heart rate)
ST segment depression >1 mm (downsloping or planar depression of greater predictive value than upsloping depression)
Slow ST recovery to normal (>5 min)
Decrease in systolic blood pressure >20 mmHg
Increase in diastolic blood pressure >15 mmHg
Progressive ST segment elevation or depression
ST segment depression >3 mm without pain
Arrhythmias (atrial fibrillation, ventricular tachycardia)

Table 2.3b Features indicative of a strongly positive exercise test.

Exercise limited by angina to <6 min of Bruce protocol
Failure of systolic blood pressure to increase >10 mmHg, or fall with evidence of ischaemia
Widespread marked ST segment depression >3 mm
Prolonged recovery time of ST changes (>6 min)
Development of ventricular tachycardia
ST elevation in the absence of prior myocardial infarction

Radionuclide myocardial perfusion imaging

Thallium-201 or technetium-99m (^{99m}Tc-sestamibi, ^{99m}Tc-tetrofosmin) is injected intravenously at peak stress, and its myocardial distribution relates to coronary flow. Images are acquired with a gamma camera (Figure 2.5). This test can distinguish between reversible and irreversible ischaemia (the latter signifying infarcted tissue). Although it is expensive and requires specialised equipment, it is useful in patients whose exercise test is non-diagnostic or whose exercise ability is limited.

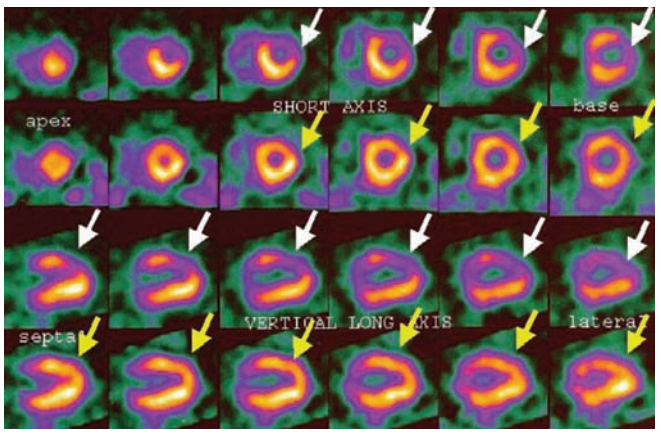


Figure 2.5 Tc^{99m} (tetrafosmin) perfusion scan showing reversible antero-lateral wall ischaemia, induced by intravenous dobutamine infusion (white arrows). Normal rest images are shown (yellow arrows).

A multigated acquisition (MUGA) scan assesses left ventricular function and can reveal salvageable myocardium in patients with chronic coronary artery disease. It can be performed with either thallium scintigraphy at rest or metabolic imaging with fluorodeoxyglucose by means of either positron emission tomography (PET) or single photon emission computed tomography (SPECT).

Cardiac magnetic resonance imaging (MRI)

Magnetic resonance imaging (MRI) is an imaging technique that uses a magnetic field and radio waves to image the body, and X-ray radiation is not required. It has emerged as an important modality to assess cardiac structure, wall motion and perfusion imaging. It is also able to differentiate viable myocardium from infarcted tissue, which may guide revascularisation strategy (Figure 2.6). Stress MRI may offer an alternative to SPECT and stress echocardiography in the functional evaluation of coronary artery disease.

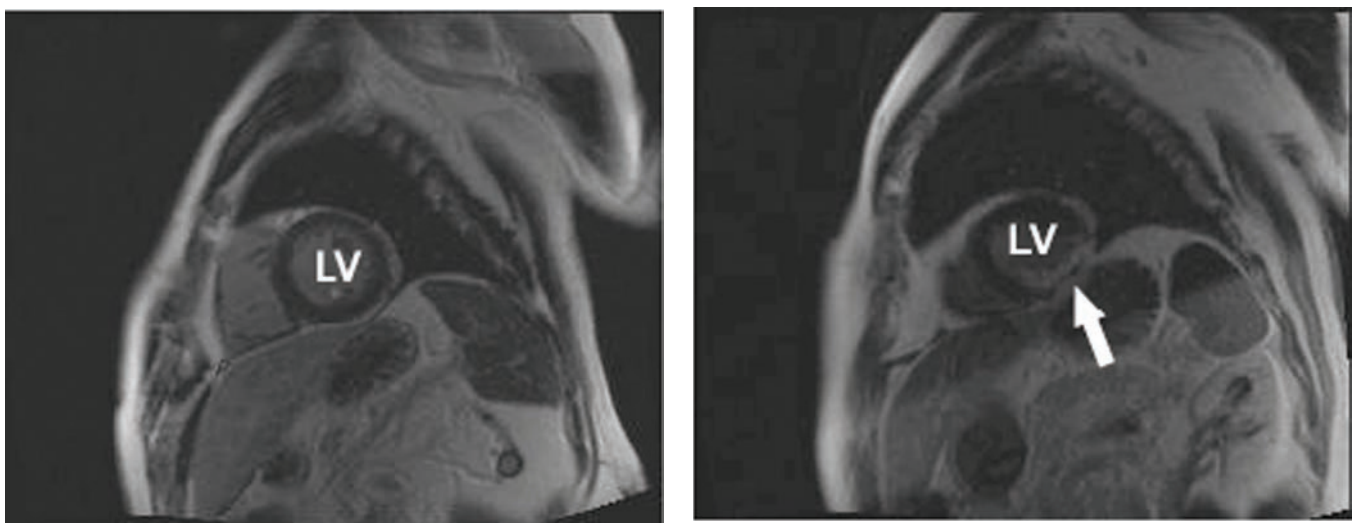
Cardiac multidetector computed tomography (MDCT)

Recent advances in multidetector computed tomography (MDCT) allow sufficient spatial resolution for direct non-invasive coronary artery imaging and has reasonably good diagnostic accuracy for detection of significant lesions in large coronary arteries. It is particularly useful in evaluating the origin, course and patency of anomalous coronary arteries and grafts (Figure 2.7). It may also detect calcium within an atheromatous plaque and has been used in 'calcium scoring', which may indicate the presence of significant coronary artery disease. Calcium scoring is probably best used as a risk factor rather than as a diagnostic test.

Invasive investigations

Coronary angiography

The only absolute way to evaluate coronary artery disease is by angiography (Figure 2.8, Table 2.4). However, as the initial response to atherosclerosis is a compensatory dilatation of the coronary



(a) (b)

Figure 2.6 (a) Cardiac magnetic resonance image of a normal heart showing circular LV in a short axis view. (b) Inferior and inferolateral LV scarring due to right coronary artery occlusion. Contrast enhancement of the infarcted myocardium is seen (arrow) following intravenous administration of a gadolinium chelate, which diffuses into interstitium (infarcted myocardium) but not myocardial cells. The increased concentration of gadolinium in infarcted myocardium results in hyper-enhancement. LV, left ventricle.

Table 2.4 Main indications for coronary angiography.

Uncertain diagnosis of angina (coronary artery disease cannot be excluded by non-invasive testing)
Assessment of feasibility and appropriateness of various forms of treatment (percutaneous intervention, bypass surgery, medical)
Class I or II stable angina with positive stress test or class III or IV angina without positive stress test
Angina not controlled by drug treatment
Unstable angina or non-ST segment elevation myocardial infarction (higher risk patients)
Acute ST segment elevation myocardial infarction – including cardiogenic shock, ineligibility for thrombolytic treatment, failed thrombolytic reperfusion, reinfarction or positive stress test
Life-threatening ventricular arrhythmia
Angina after bypass surgery or percutaneous intervention
Before valve surgery or corrective heart surgery to assess occult coronary artery disease

artery, angiography may underestimate the degree of generalised atherosclerosis.

It is usually performed as part of cardiac catheterisation, which includes left ventricular angiography and haemodynamic measurements, providing a more complete evaluation of an individual's cardiac status. Cardiac catheterisation is safely performed as a day case procedure.

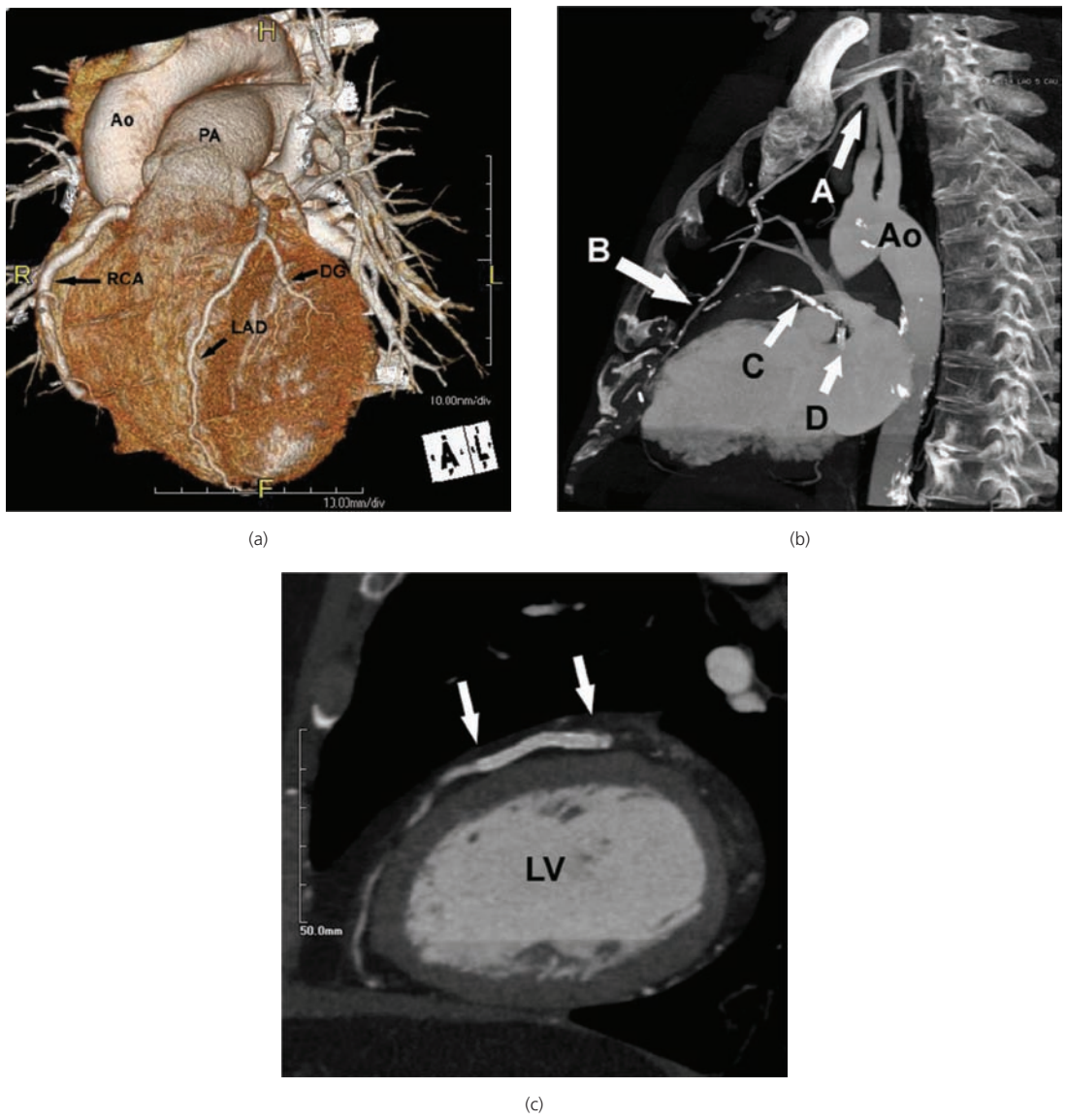
Patients must be fully informed of the purpose of the procedure as well as its risks and limitations. Major complications, though rare in experienced hands, include death (risk ratio 1 in 1500), stroke (1 in 1000), coronary artery dissection (1 in 1000) and arterial access complications (1 in 500). Risks depend on the individual patient, and predictors include age, coronary anatomy (such as severe left main stem disease), impaired left ventricular function, valvar heart disease, the clinical setting and non-cardiac disease. The commonest complications are transient or minor and include arterial access bleeding and haematoma, pseudoaneurysm, arrhythmias, reactions to the contrast medium and vagal reactions (during sheath insertion or removal).

Before the procedure, patients usually fast and may be given a sedative. Although a local anaesthetic is used, arterial access (femoral, brachial or radial) may be mildly uncomfortable. Patients do not usually feel the catheters once they are inside the arteries. Transient angina may occur during injection of contrast medium, usually because of a severely diseased artery. Patients should be warned that, during left ventricular angiography, the large volume of contrast medium may cause a transient hot flush and a strange awareness of urinary incontinence (and can be reassured that this does not actually happen). Modern contrast agents rarely cause nausea and vomiting.

Insertion of an arterial sheath with a haemostatic valve minimises blood loss and allows catheter exchange. Three types of catheter, which come in a variety of shapes and diameters, are commonly used (Figure 2.9). Two have a single hole at the end and are designed to facilitate controlled engagement of the distal tip within the coronary artery ostium. Contrast medium is injected through the lumen of the catheter, and moving X-ray images are obtained and recorded. Other catheters may be used for graft angiography. The 'pigtail' catheter has an end hole and several side holes and is passed across the aortic valve into the left ventricle. It allows injection of 30–40 ml of contrast medium over 3–5 seconds by a motorised pump, providing visualisation of left ventricular contraction over two to four cardiac cycles (Figure 2.10). Aortic and ventricular pressures are also recorded during the procedure.

Intravascular ultrasound (IVUS)

In contrast to angiography, which gives a two-dimensional luminal silhouette with little information about the vessel wall, intravascular



(a) (b) (c)

Figure 2.7 (a) Three-dimensional 64-slice cardiac computed tomography scan. (Ao, aorta; PA, pulmonary artery; RCA, right coronary artery; LAD, left anterior descending artery; DG, diagonal artery.) (b) Cardiac computed tomography scan showing course of patent left internal mammary artery (A and B) supplying the native distal LAD artery. The proximal and mid-LAD artery is heavily calcified (C) and there is a visible stent in the left circumflex artery (D). There is also calcification of the aortic arch and descending aorta (Ao). (c) Patent coronary stent (between two arrows) in left anterior descending artery.

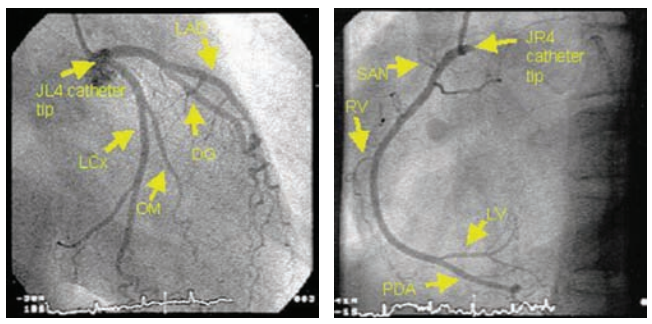


Figure 2.8 Angiogram of normal left and right coronary arteries. LAD, left anterior descending artery; DG, diagonal artery; LCx, left circumflex artery; OM, obtuse marginal artery; SAN, sino-atrial node artery; RV, right ventricular branch artery; LV, left ventricular branch artery; PDA, posterior descending artery; JL4, left 4 Judkins; JR4, right 4 Judkins.

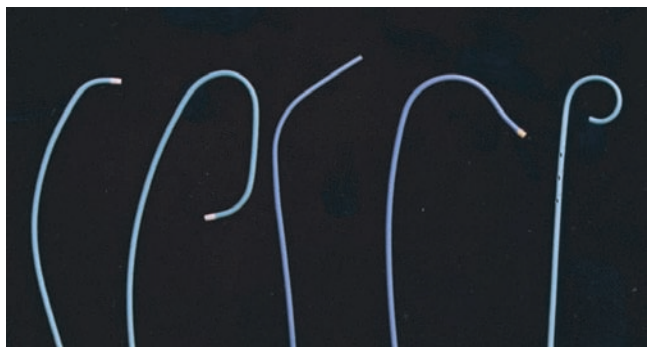


Figure 2.9 Commonly used diagnostic catheters (from left to right): right Judkins, left Judkins, multipurpose, left Amplatz and pigtail.