POCKET CONSULTANT

Cardiology

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Preface to the second edition

It is hoped that this book will be of practical help to doctors and nurses confronted by typical management problems in the cardiac patient. As a practical guide it is necessarily dogmatic and much information is given in list format or in tables, especially in sections dealing with drug therapy.

Some subjects in cardiology are often not well covered in clinical training and it is intended that some sections will help fill any gaps in the doctors' or nurses' clinical course, e.g. sections on congenital heart disease, pacing, and cardiac investigations.

Practical procedures such as cardiac catheterisation cannot be learnt from a book and technical aspects of catheterisation are not covered here. However, interpretation of catheter laboratory data is discussed and it is hoped that the book will be of value to the doctor learning invasive cardiology. A practical subject like echocardiography cannot be covered in depth in a book of this size, but the fundamentals and common cardiac conditions are discussed.

Since the publication of the first edition there have been enormous advances in many aspects of cardiology, particularly in the management of ischaemic heart disease. New sections include thrombolysis, percutaneous transluminal coronary angioplasty, management of hyperlipidaemias, Doppler echocardiography, plus advances in pacing and electrophysiology. The sections dealing with drug therapy have been up-dated and the discussion of calcium antagonists and ACE inhibitors extensively revised. The chapter on congenital heart disease now includes a section on management problems of cyanotic congenital heart disease in the adult.

Nuclear cardiology has not been included. It is a vital and growing branch of cardiology but the subject cannot be covered without including many colour diagrams and pictures. This would greatly increase the expense of the book, and I have reluctantly decided to exclude it.

Acknowledgements

I would like to thank my wife Lindsay and my secretaries Mrs Evelyn Beinart and Mrs Jane West for their enormous help in typing the manuscript.

The work of a large number of authors has contributed to the body of knowledge in this book and it would be impossible to thank them individually or to provide detailed references to their work in a pocket book. The list of further reading incorporates references and my thanks to them all. My thanks also to my many colleagues who have helped with suggestions and alterations. I am also indebted to Dr R. Sutton and Medtronic Ltd for permission to modify their pacing code diagrams and to Dr P.E. Gower for permission to include the nomogram for body surface area.

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1 Cardiac symptoms and physical signs

1.1 Common cardiac symptoms

Angina

Typical angina presents as a chest tightness or heaviness brought on by effort and relieved by rest. The sensation starts in the retrosternal region and radiates across the chest. Frequently it is associated with a leaden feeling in the arms. Occasionally it may present in more unusual sites, e.g. pain in the jaws or teeth on effort, without pain in the chest. It may be confused with oesophageal pain, or may present as epigastric or even hypochondrial pain. The most important feature is its relation to effort. Unilateral chest pain (submammary) is not usually cardiac pain, which is generally symmetrical in distribution.

Angina is typically exacerbated by heavy meals, cold weather (just breathing in cold air is enough) and emotional disturbances. Arguments with colleagues or family and watching exciting television are typical precipitating factors.

Stable angina

Angina induced by effort and relieved by rest. Not increasing in frequency or severity, and predictable in nature. Associated with ST segment depression on ECG.

Decubitus angina

Angina induced by lying down at night or during sleep. It may be due to an increase in LVEDV (and hence wall stress) on lying flat, associated with dreaming, or getting into cold in sheets. Coronary spasm may occur in REM sleep. It may respond to a diuretic or calcium antagonist taken in the evening.

Unstable (crescendo) angina

Angina of increasing frequency and severity. Not only induced by effort but coming on unpredictably at rest. It may progress to myocardial infarction.

Variant angina (Prinzmetal angina)
Angina occurring unpredictably at rest associated with transient ST segment elevation on the ECG. It is not common. It is associated with coronary spasm often in the presence of additional arteriosclerotic lesions.

Other types of retrosternal pain
- *pericardial pain* is described in **7.1.** It is usually retrosternal or epigastric, lasts much longer than angina and is often stabbing in quality. It Is related to respiration and posture (relieved by sitting forward). Diaphragmatic pericardial pain may be referred to the left shoulder.
- *aortic pain* (p. 369). Acute dissection produces a sudden tearing intense pain retrosternally radiating to the back. Its radiation depends on the vessels involved. Aortic aneurysms produce chronic pain especially if rib or vertebral column erosion occurs.
- *non-cardiac pain*. May be oesophageal or mediastinal with similar distribution to cardiac pain but not provoked by effort. Oesophageal pain may be provoked by ergonovine, making it a useless test for coronary spasm. Chest wall pain is usually unilateral. Stomach and gall bladder pain may be epigastric and lower sternal and be confused with cardiac pain.

Dyspnoea
An abnormal sensation of breathlessness on effort or at rest. With increasing disability orthopnoea and paroxysmal nocturnal dyspnoea (PND) occur. Pulmonary oedema is not the only cause of waking breathless at night: it may occur in non-cardiac asthma. A dry nocturnal cough is often a sign of impending PND. With acute pulmonary oedema pink frothy sputum and streaky haemoptysis occur. With poor LV function Cheyne – Stokes ventilation makes the patient feel dyspnoeic in the fast cycle phase.

1 Cardiac symptoms and physical signs

1.1 Common cardiac symptoms

Effort tolerance is graded by New York Heart Association criteria as follows:

Class 1
Patients with cardiac disease but without resulting limitations of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation or angina.

Class 2
Patients with cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitation, dyspnoea or angina (e.g. walking up two flights stairs, carrying shopping basket, making beds, etc.). By limiting physical activity patients can still lead a normal social life.

Class 3
Patients with cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest, but even mild physical activity causes fatigue, palpitation, dyspnoea or angina (e.g. walking slowly on the flat). Cannot do any shopping or housework.

Class 4
Patients with cardiac disease who are unable to do any physical activity without symptoms. Angina or heart failure may be present at rest. They are virtually confined to bed or a chair and are totally incapacitated.

Syncope
Syncope may be due to several causes.
- *vasovagal* (vasomotor, simple faint) is the commonest cause. Sudden dilation of venous capacitance vessels associated with vagal-induced bradycardia. Induced by pain, fear, emotion.

- *postural hypotension*. Usually drug induced (by vasodilators). May occur in true salt depletion (by diuretics) or hypovolaemia.
- *carotid sinus syncope*. A rare condition with hypersensitive carotid sinus stimulation (e.g. by tight collars) inducing severe bradycardia.
- *cardiac dysrhythmias*. Commonest causes are sinus arrest, complete AV block, ventricular tachycardia. 24-hour ECG monitoring is necessary.
- *obstructing lesions*. Aortic or pulmonary stenosis, left atrial myxoma or ball valve thrombus, HOCM, massive pulmonary embolism. Effort syncope is commonly secondary to aortic valve or subvalve stenosis in adults and Fallot's tetralogy in children.
- *cerebral causes*. Sudden hypoxia, transient cerebral arterial obstruction, spasm or embolism.
- *cough syncope*. This may be due to temporarily obstructed cerebral venous return.
- *micturition syncope*. This often occurs at night, and sometimes in men with prostatic symptoms. It may be in part due to vagal overactivity and partly due to postural hypotension.

The commonest differential diagnosis needed is sudden syncope In the adult with no apparent cause. Stokes–Adams attacks and epilepsy are the main contenders.

Stokes–Adams attacks	Epilepsy
No aura or warning	Aura often present
Transient unconsciousness (often only a few seconds)	More prolonged unconsciousness
Very pale during attack	Tonic/clonic phases
Rapid recovery	Prolonged recovery. Very drowsy
Hot flush on recovery	Absent

A prolonged Stokes–Adams episode may produce an epileptiform attack from cerebral hypoxia. It is not always possible to distinguish the two clinically.

1 Cardiac symptoms and physical signs

1.1 Common cardiac symptoms

Cyanosis

Central cyanosis should be detectable when arterial saturation is less than 85% and when there is more than 5g reduced haemoglobin present. It is more difficult to detect if the patient is also anaemic. Cardiac cyanosis may be caused by poor pulmonary blood flow (e.g. Pulmonary atresia), by right-to-left shunting (e.g. Fallot's tetralogy) or common mixing situations with high pulmonary blood flow (e.g. TAPVD).

Cyanosis from pulmonary causes should be improved by increasing the FiO_2. This will not affect cyanosis from cardiac causes and is a useful test in the cyanosed neonate.

Peripheral cyanosis in the absence of central cyanosis may be due to peripheral vasoconstriction, poor cardiac output or peripheral sludging of red cells (e.g. polycythaemia).

Embolism

Both systemic and pulmonary embolism are common in cardiac disease. Predisposing factors in cardiology are:

Pulmonary emboli	Systemic emboli
Prolonged bed rest	Aortic stenosis (calcium)
High venous pressure	HOCM
Central lines	Mitral stenosis in AF
Femoral vein catheterisation	Infective endocarditis
Pelvic disease (tumour, inflammation)	LA myxoma
Tricuspid endocarditis	Prosthetic aortic or mitral valves
Deep vein thrombosis	Closed mitral valvotomy with a calcified valve (calcium)

Either or both

Myocardial infarction
Dilated cardiomyopathy
CCF
Polycythaemia
Diuretics
Pro-coagulable state
Eosinophilic heart disease

1 Cardiac symptoms and physical signs

1.1 Common cardiac symptoms

Oedema

Factors important in cardiac disease are: elevated venous pressure (CCF, pericardial constriction), increased extracellular volume (salt and water retention), secondary hyperaldosteronism, hypoalbuminaemia (liver congestion, anorexia and poor diet), venous disease and secondary renal failure.

Acute oedema and ascites may develop in pericardial constriction. Protein losing enteropathy can occur with a prolonged high venous pressure exacerbating the oedema.

Other symptoms

These are discussed under relevant chapters:
Palpitation: Principles of paroxysmal tachycardia diagnosis **6.10**.
Haemoptysis: Mitral stenosis **3.1**.
Cyanotic attack: Catheter complications **10.3**.

1.2 Physical examination

Hands

It is important to check for:

- dilated hand veins with CO_2 retention
- temperature (? cool periphery with poor flows, hyperdynamic circulation)
- peripheral cyanosis
- clubbing: cyanotic congenital heart disease, infective endocarditis
- capillary pulsation; aortic regurgitation, PDA
- Osler's nodes, Janeway lesions, splinter haemorrhages: infective endocarditis
- nail fold telangiectases: collagen vascular disease
- arachnodactyly: Marfan syndrome
- polydactyly, syndactyly, triphalangeal thumbs: ASD
- tendon xanthomata

Facial and general appearance

Down's syndrome (AV canal), elf-like facies (supravalvar aortic stenosis), Turner's (coarction, AS), moon-like plump facies (pulmonary stenosis), Noonan's syndrome (pulmonary stenosis, peripheral pulmonary artery stenosis)

Mitral facies with pulmonary hypertension

Central cyanosis.

Differential cyanosis in PDA + Pulmonary hypertension or interrupted aortic arch

Xanthelasma

Teeth: must be checked as part of general CVS examination

Dysphoea at rest. ? Accessory muscles of respiration

Jugular venous pulse (JVP)

Waveform examples are shown in Fig. 1.1. It should fall on inspiration. Inspiratory filling of the neck veins occurs in pericardial constriction (Kussmaul's sign). The waves produced are as follows:

'a' wave. Atrial systole. It occurs just before the carotid pulse. It is lost in AF. Large 'a' waves indicate a raised RVEDP (e.g. PS. PHT). Cannon 'a' waves occur in: junctional tachycardia, complete AV block, ventricular ectopics (atrial systole against a closed tricuspid valve).

'c' wave. Not visible with the naked eye. Effect of tricuspid valve closure on atrial pressure.

'x' descent. Fall in atrial pressure during ventricular systole due to downward movement of base of heart.

'v' wave. Atrial filling against a closed tricuspid valve.

'y' descent. Diastolic collapse following tricuspid valve opening.

1.2 Physical examination

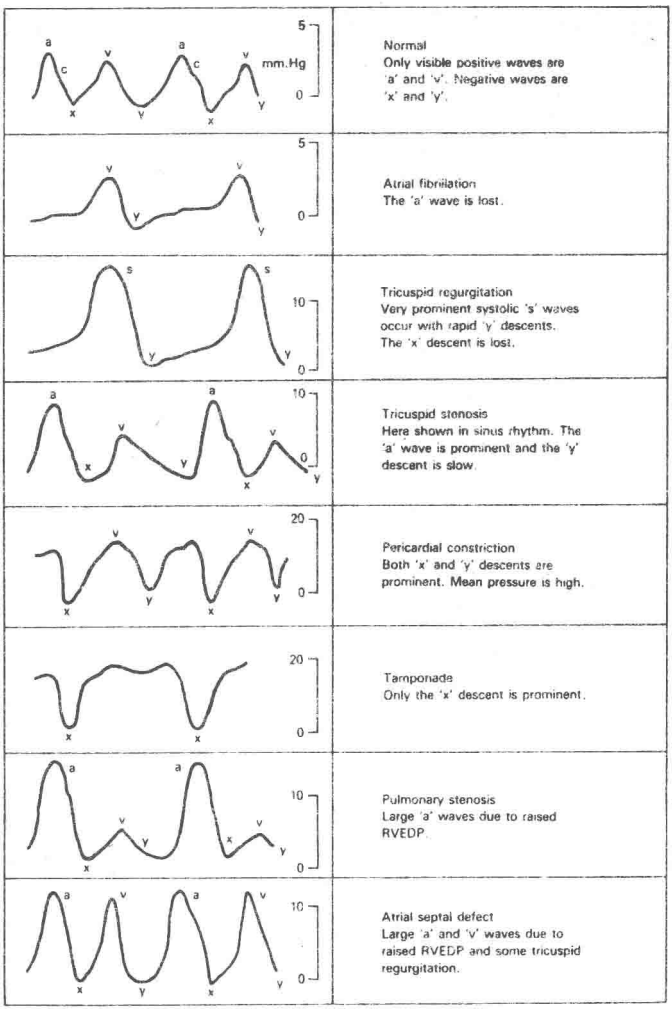

Fig. 1.1 Examples of waveforms seen on jugular venous pulse.

's' wave occurs in tricuspid regurgitation. Fusion of 'x' descent and 'v' wave into a large systolic pulsation with rapid 'y' descent

The normal range of jugular venous pressure is −7 to +3 mmHg. The patient sits at 45° and sternal angle is used as a reference point. Distinction of the JVP from the carotid pulse involves the following 5 features: timing, the ability to compress and obliterate the JVP, the demonstration of hepatojugular reflux, the alteration of the JVP with position and the site of the pulsation itself.

The carotid pulse

Waveform examples are shown in Fig. 1.2. There are three components to the carotid pulse: percussion wave, tidal wave and dicrotic notch.

Percussion wave: a shock wave transmitted up the elastic walls of the arteries. *Tidal wave*: reflection of the percussion wave with forward moving column of blood. It follows the percussion wave and is not usually palpable separately. *Dicrotic notch* times with aortic valve closure.

All the pulses are felt: radials and femorals simultaneously (coarctation). Any pulse may disappear with dissection of the aorta. Right arm and carotid pulses area stronger than left in supra-valve aortic stenosis p. 94).

Palpation

This checks for: thrills, apex beat, abnormal pulsation, and palpable sounds. Systolic thrill in the aortic area suggests aortic stenosis. Feel for thrills in other sites:

Left sternal edge: VSD or HOCM.

Apex: ruptured mitral chordae.

Pulmonary area: Pulmonary stenosis.

Subclavicular area: subclavian artery stenosis.

Diastolic thrills are less common: Feel for apical diastolic thrill in mitral stenosis with patient lying on left side and breath held