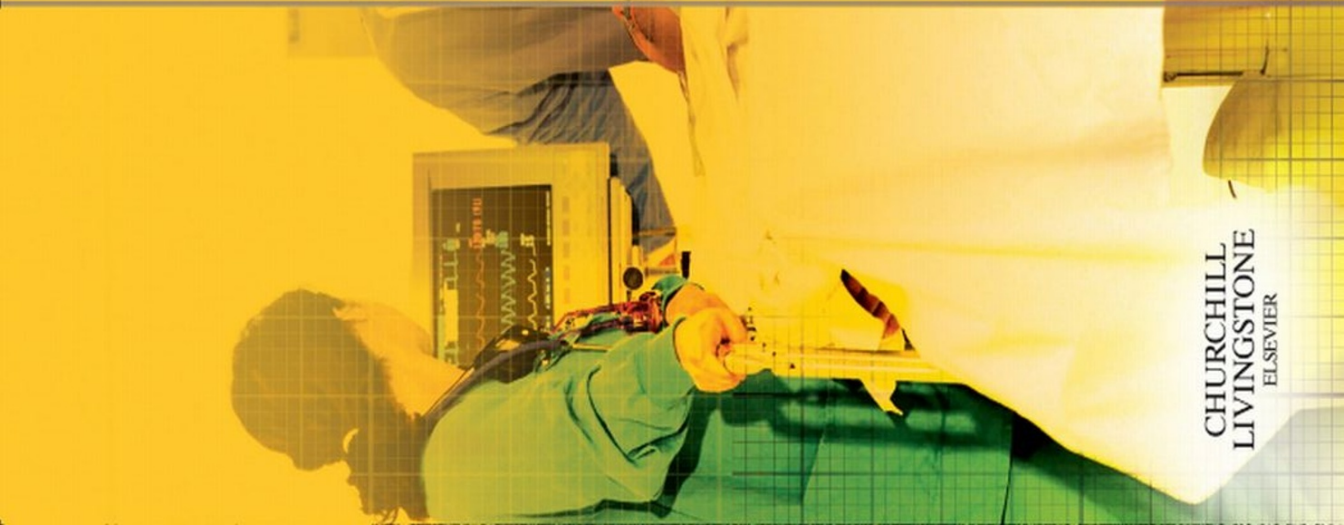
Churchill's Ready Reference

Cardiology

Alexander R. Lyon Glyn Thomas Vanessa Cobb Jamil Mayet



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Preface

'To study the abnormal is the best way of understanding the normal'
William James, 1842–1910, American Psychologist

Management of patients in medical practice depends critically upon identification of abnormality, whether this be anatomical, physiological or biochemical. Knowledge of normality is therefore crucial, both to identify when disease is present, and to prevent overdiagnosis when it is not. This book is designed to provide a concise source of normal ranges for cardiovascular physiology and pathophysiology relevant to clinical cardiology practice in the first decades of the 21st century. In addition to covering routine investigations such as coronary angiography, two-dimensional echocardiography and electrocardiography, we have included many of the novel cardiac investigations now available, such as T wave alternans, intravascular coronary ultrasound and cardiac magnetic resonance imaging. This text provides the reader with normal ranges for a wide variety of cardiovascular tests at his or her fingertips, facilitating correct interpretation of clinical results. We hope this will be a useful reference text to the reader encountering patients with cardiovascular disease as part of his or her working life as a healthcare professional.

Defining normality is not straightforward, and in many situations abnormality is specified in terms of clinical significance for outliers from the normal range. For many parameters with a Gaussian distribution the normal range is defined as the range of values that ninety-five per cent of healthy individuals would fall within. The extrapolation of this is that five per cent of healthy individuals may have a value which lies in the 'abnormal' range, without any clinical significance attached to this outlying value.

A second issue is that there is frequently a grey zone of overlap between normality and clinically significant abnormality, and this reflects a continuous spectrum in the transition between the two states. For example, if the upper cutoff for systolic blood pressure is 140 mmHg, does the individual with a value of 141 mmHg have a diagnosis of hypertension with associated risk, whilst a second individual with a systolic blood pressure value of

139 mmHg has a label of normal blood pressure with low risk? Add dynamic temporal variation in parameters, and the potential for inaccuracy associated with all tests, and the scenario may become more complex.

Our message is that all clinical readings and measurements should always be placed in context, and clinical judgement is critical to extrapolate the findings of any investigation to diagnosis and treatment.

The authors would like to acknowledge the contribution of the late Philip Poole-Wilson, who was involved in the initial conception and design of this text, and whose teaching and influence have been a tremendous influence to all involved in research and care of patients with cardiovascular disease. We would also like to thank Dr Sanjay Prasad, Consultant Cardiologist at the Royal Brompton Hospital, London and Dr Vinit Sawhney, Clinical Fellow in Cardiology at St Bartholomew's Hospital, London for their valuable contributions in the preparation of this book.

AL, GT, VC and JM

TOPIC 1

Coronary circulation

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Coronary blood flow

Oxygen consumption of myocardium at rest = 8–10 ml/min/100 g.

The heart receives ~5% of total cardiac output.

Myocardial oxygen extraction is high (~75%), and therefore there is little extraction reserve. Furthermore, the myocardium does not have great capacity for anaerobic glycolysis.

During exercise myocardial oxygen consumption can increase to > 40 ml/min/100 g.

Increased demand is met by increasing coronary blood flow.

Coronary blood flow is subject to autoregulation which is closely coupled to and driven by myocardial oxygen consumption (mVO_2). Autoregulation is lost when coronary perfusion pressure drops below 60 mmHg.

There is local metabolic control of coronary blood flow, thought to be largely mediated by adenosine and nitric oxide. There is additionally sympathetic innervation of alpha and beta adrenoceptors.

Coronary blood flow varies within the cardiac cycle and most flow occurs during diastole. During systole, extravascular compression reduces intramyocardial flow. The greatest resistance to perfusion is the subendocardial layer where the extravascular compressive forces are greatest and vascular pressure is reduced. Compressive forces are lower in the right ventricle and the drop in flow during systole is less pronounced.

Wave intensity analysis, using pressure- and flow-sensitive wires in human coronary arteries, has identified six predominant waves influencing phasic flow. It is postulated that a dominant backward-propagating 'suction' wave, generated by a fall in resistance of the coronary microvasculature with myocardial relaxation, is largely responsible for diastolic flow.

Coronary anatomy

Right coronary artery (RCA) (see Figure 1.1)

Arises from the right coronary sinus.

The first branch is the conus (infundibular) branch, which passes anteriorly to supply the right ventricular outflow tract. The conus branch may instead arise from the aorta.

In 55% of people, the proximal RCA gives off a small branch to the sinoatrial node. In 45% of cases it is supplied by the left circumflex vessel (LCx).

The RCA then passes along the right atrioventricular (AV) groove and gives off acute marginal branches to the free wall of the right ventricle.

At the crux (junction of AV groove and posterior interventricular sulcus) it supplies the inferior left ventricular (LV) wall.

The posterior descending branch supplies blood to the posterior third of the interventricular septum.

The posterolateral branch supplies the basal posterolateral LV wall.

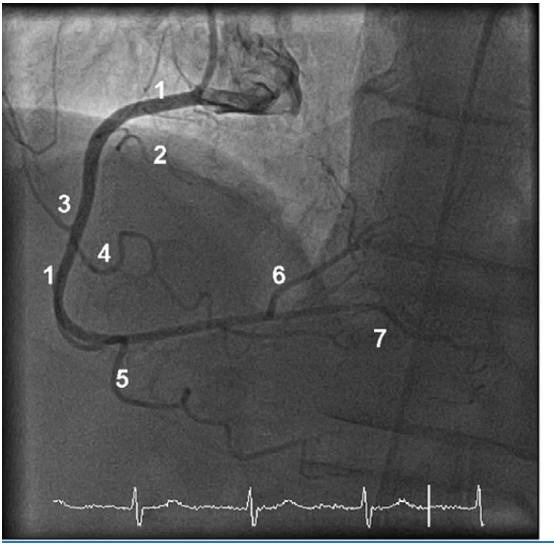

Figure 1.1 LAO view of right coronary artery.

1. Right coronary artery
2. Conus branch artery
3. Sinoatrial nodal artery
4. Right ventricular artery
5. Acute marginal artery
6. Posterolateral artery
7. Posterior descending artery

Left coronary artery (see Figure 1.2)

Arises from the left coronary sinus as the left main stem and bifurcates into the left anterior descending artery (LAD) and circumflex artery.

The LAD passes along the anterior interventricular groove. It gives off septal branches to the anterior 2/3 of the interventricular septum and diagonal branches which supply the anterolateral free LV wall. Terminal branches supply the apex.

The circumflex passes in the left AV groove and supplies the lateral LV. It gives off branches to the left atrium and obtuse marginal branches to supply the posterolateral LV.

- In some cases the left main stem trifurcates into LAD, circumflex and ramus intermedius branches. The ramus intermedius arises between the other two. It supplies the anterior LV free wall.
- In some cases the left main stem is absent and the LAD and LCx arise from separate ostia in the left coronary sinus.
- In some cases the circumflex artery arises from the right coronary sinus.
- Left or right dominance: The dominant vessel supplies the AV node and gives off the posterior descending artery (PDA) which supplies the posterior third of the interventricular septum. The RCA is dominant in 85%, the LCx is dominant in 10% and codominance is present in 5%.

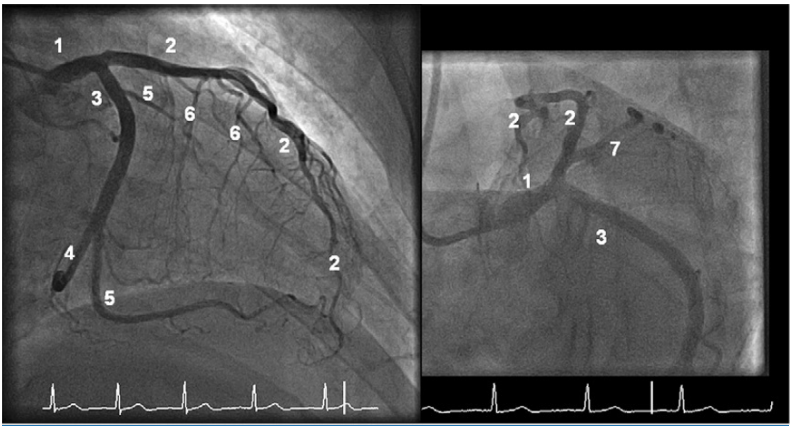

Figure 1.2 Left coronary artery anatomy. Left – RAO view, Right – LAO caudal ('spider') view.

1. Left main stem coronary artery
2. Left anterior descending coronary artery
3. Circumflex coronary artery
4. AV circumflex coronary artery
5. Obtuse marginal coronary arteries
6. Septal coronary arteries
7. Diagonal coronary artery

Coronary venous anatomy

Most venous drainage of the heart passes into the right atrium via the coronary sinus, the ostium of which lies in the posteroinferior interatrial septum. It receives blood from the middle cardiac vein (which runs in the posterior interventricular groove, alongside the posterior descending artery) and it is in continuity with the great cardiac vein (which runs parallel to the left circumflex artery). The anterior interventricular vein runs with the left anterior descending artery and drains into the great cardiac vein. The great cardiac vein also receives tributaries from the left marginal vein and left posterior vein. The small cardiac vein receives tributaries draining the right ventricle and also drains into the coronary sinus. The anterior cardiac vein returns blood separately to the right atrium and there are additional small veins that open directly into the cardiac chambers. The coronary venous anatomy when viewed in the LAO and RAO configurations is displayed in Figure 1.3.

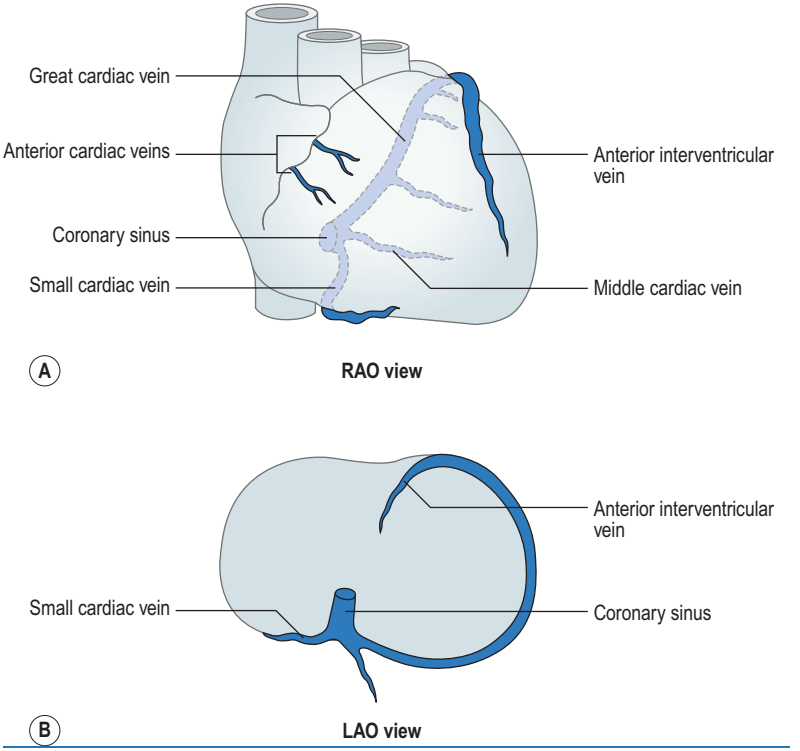

Figure 1.3 Normal coronary venous anatomy. (A) RAO view. (B) LAO view.

Coronary artery anomalies

Congenital coronary artery anomalies include:

1. Anomalous origin/course.
2. Intramyocardial (myocardial bridging). Part of the coronary artery takes an intramyocardial course.