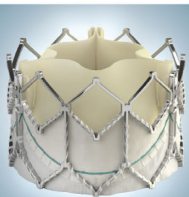


Cardiology

A Practical Handbook

David Laflamme

Foreword by Dr Paul Dorian



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CRC Press
Taylor & Francis Group

Cardiology

A Practical Handbook



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There has been a near exponential increase in the amount of information available on the pathophysiology and management of heart diseases over the past decades. Meanwhile, our understanding of the underlying pathology and physiology has deepened and broadened with new methodologies to monitor cardiac structure and function. These developments have led to an overwhelming amount of information available to students, trainees, and physicians on all aspects of cardiac disease. What is in short supply is a comprehensive yet concise and clear description of the important cardiac conditions and disorders, an approach to their management, and an easily consulted and well indexed summary to be used at the bedside or in the clinic.

The Cardiology Handbook fills an extremely important, not well occupied niche in providing junior and senior practitioners alike with a brief yet detailed summary of “what you need to know” about virtually all important cardiovascular conditions.

This handbook does not aim to be a comprehensive review of all of the evidence pertaining to pathophysiology, investigation, and treatment of cardiovascular disorders. It does however wonderfully clearly get “straight to the point” and I think will be a frequently used and extremely effective resource for a brief “look up” in the clinic, in the emergency room, and on the wards.

I anticipate this handbook will be a treasured resource for senior medical students, trainees in internal medicine, emergency medicine, anesthesia, and other specialties where cardiovascular disorders are commonly seen. The effective use of figures and tables makes this appealing both visually and cognitively, and for those unfamiliar with the complex and varied terrain of cardiovascular medicine, will prove an outstanding trail map to allow successful navigation of unfamiliar terrain. In an era where there is often “too much information”, this handbook provides just the right amount.

Sincerely,



Paul Dorian, MD, FRCPC, FHRS
Dexter H.C. Man Chair in Cardiology
Director Division of Cardiology, University of Toronto
Past President, Canadian Heart Rhythm Society



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*To my parents, Diane and Marcel,
for their constant support,
and to Anne-Sophie, for
her extraordinary patience.*

Cardiology is a fascinating medical specialty. This vast discipline combines various fields of expertise, including research, prevention, clinical evaluation, diagnostic examinations, therapeutic management, invasive interventions and rehabilitation.

As a result of the spectacular progress over the last two decades, cardiology now comprises sophisticated and advanced diagnostic and therapeutic tools (validated by evidence-based medicine) allowing cardiologists to more effectively manage their patients, who have greatly benefited in terms of survival and/or quality of life.

In relation to such an effervescent and increasingly complex discipline, physicians who have the privilege to practice cardiology must have access to resources that enable them to acquire, update, refine and organize their knowledge.

The purpose of this book is to provide a concise overview of modern cardiology. More than 10,000 pages of references have been condensed and organized into less than 350 pages. This pocket book provides practising clinicians with specific and accurate information on a particular subject. It is also designed to allow students, interns and all other personnel working in the field of cardiology to acquire and organize their valuable knowledge.

The design of this cardiology handbook was a captivating but colossal task. The information presented in this book is derived from numerous valid and up-to-date sources and has been verified by various reviewers, experts in their respective fields. The various recommendations are also derived from American, European or Canadian learned society evidence-based guidelines.

I sincerely hope that this book will inspire or maintain your passion for cardiology and I wish you an enjoyable read.

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February 2016

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Legal statement

Every physician is responsible for his or her acts. Although the recommendations and management presented in this book are based on valid, reliable and up-to-date references at the time of writing, the author and publisher decline all responsibility and remind the reader that every physician must practice medicine according to current medical and scientific guidelines, taking into account his or her own capacities and limitations, and, if necessary, seeking advice from more experienced specialists.





Cardiac diagnostic assessment

01

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1.1/ CARDIAC PHYSICAL EXAMINATION

EXAMINATION OF THE PATIENT

GENERAL INSPECTION: Diaphoresis; Signs of hypoperfusion; Cachexia; Mitral facies; Cheyne-Stokes breathing

CUTANEOUS INSPECTION: Cyanosis (central vs peripheral); Pallor; Telangiectasias (Osler-Weber-Rendu; scleroderma); Tanned skin (hemochromatosis); Jaundice (liver disease); Ecchymoses (coagulopathy); Petechiae (thrombocytopenia); Purpura (vasculitis; endocarditis); characteristic axillary skin fold (pseudoxanthoma elasticum); Lentiginosis (LEOPARD; Carney); Lupus pernio - erythema nodosum (sarcoidosis); blue sclera (osteogenesis imperfecta); nicotine stains (smoking)

- > **Familial hypercholesterolemia:** Arcus senilis; pathognomonic tendinous xanthomas (extensor tendons; MCP; Achilles tendon); Xanthelasma
- > **Familial hypertriglyceridemia (LPL deficiency):** Eruptive xanthomas; Lipemia retinalis
- > **Dysbetalipoproteinemia:** Tuberos xanthomas (elbows; knees); Palmar xanthomas

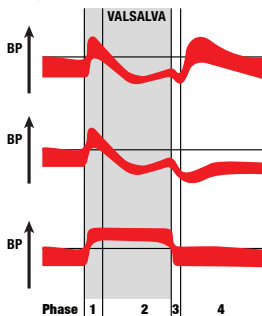
SIGNS OF ENDOCARDITIS: Roth's spots; Janeway lesions; Osler's nodes; splinter hemorrhages; mucosal petechiae

FUNDOSCOPY: hypertensive retinopathy (arteriovenous nicking; exudates; hemorrhages; cotton-wool spots; papilledema); diabetic retinopathy; endocarditis (Roth's spot); Hollenhorst plaque (cholesterol embolism); Lipemia retinalis

VITAL SIGNS

BLOOD PRESSURE: in both arms (\pm legs)

- > **Large pulse pressure (> 50% of SBP):** Age - HTN; AR; Patent ductus arteriosus; Ruptured aneurysm of the sinus of Valsalva; Fever; Anemia; Hyperthyroidism; Pregnancy; AV fistula; Paget's disease
- > **Narrow pulse pressure (< 25% of SBP):** Cardiac tamponade; Heart failure; Cardiogenic shock; Aortic stenosis
- > **BP difference between the two arms > 10 mmHg:** Normal variant; PAD; Inflammatory vascular disease (Takayasu; giant cell arteritis); Supravalvular aortic stenosis; CoA; Aortic dissection
- > **BP difference between the arms and legs > 20 mmHg:** Hill's sign (significant AR); CoA; severe PAD
- > **Pulsus paradoxus:** \searrow SBP > 10 mmHg on inspiration
- > **Orthostatic hypotension:** \searrow SBP > 20 mmHg or \searrow DBP > 10 mmHg during the first 3 minutes after standing up
- > **Valsalva response**



NORMAL RESPONSE (SINUSOIDAL)

1. \uparrow BP (\uparrow intrathoracic pressure)
2. \downarrow venous return; \downarrow BP; \uparrow SVR; Reflex tachycardia
3. Release: \downarrow BP (brief)
4. Overshoot: \uparrow venous return; \uparrow BP; \downarrow SVR; Reflex bradycardia

NO OVERSHOOT (PHASE 4)

Systolic dysfunction

SQUARE WAVE

\uparrow filling pressure (little impact of \downarrow venous return)

OTHER VITAL SIGNS: Pulse; heart rate regularity; Respiratory rate; Oxygen saturation; Temperature; Weight; Height; Waist

> **Body surface area (m²) = 0.007184 x weight (kg) x height (cm)**

+

NECK

CAROTID PULSE

- > **Shape:** ▶▶ Hemodynamic assessment (arterial recording)
- > **Carotid sinus massage:** abnormal response if **asystole > 3 seconds (sinus arrest or AV block) and/or significant and symptomatic fall in SBP**

+

JUGULAR VEINS

- > **Jugular vein vs carotid artery:** Biphasic; Height modified by inspiration and position and hepatojugular reflux (HJR); Impalpable; Compressible
- > **Height:** distance between the sternal angle and the summit of venous pulsation; **normal < 3 cmH₂O**
 - **CVP (cmH₂O):** height above the sternal angle + 5 cmH₂O
 - **Normal CVP:** < 8 cmH₂O (< 6 mmHg)
 - **Conversion:** 1.36 cmH₂O = 1 mmHg
- > **Waves:** ▶▶ Hemodynamic assessment (atrial recording)

+

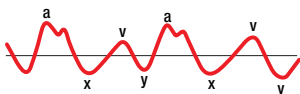
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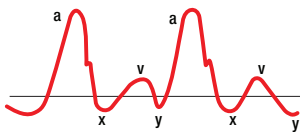
Normal pattern

a wave > v wave



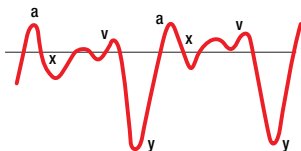
Tricuspid stenosis (or RVH)

Predominant a wave



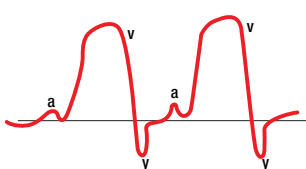
Constrictive pericarditis

Predominant y descent / W (or M) pattern



Tricuspid regurgitation

Predominant v wave



- > **Kussmaul's sign:** inspiratory increase (or absence of decrease) of CVP (constriction; RCM; RV infarction; pulmonary embolism; TS; RA tumor; right heart failure)
- > **Hepatojugular reflux (HJR):** Right upper quadrant (RUQ) compression (25 mmHg) x 15 seconds
 - **Abnormal response:** sustained \nearrow of CVP > 3 cm throughout compression (patient breathing normally); reflects right heart failure and/or wedge pressure > 15 mmHg

+

PRECORDIUM

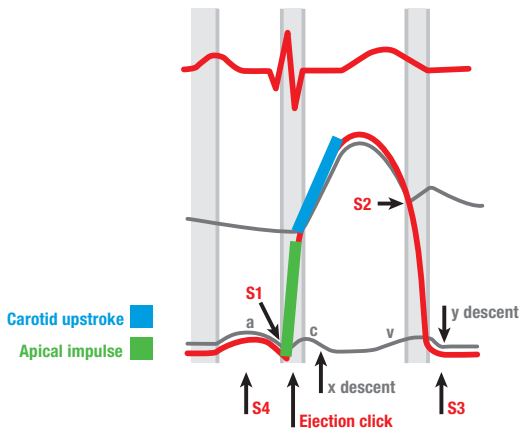
INSPECTION OF THE RIB CAGE: pectus excavatum; pectus carinatum; kyphoscoliosis; barrel chest; surgical scars; breathing; PPM / ICD

INSPECTION OF THE PRECORDIUM: position and dimensions of the apical impulse

PALPATION: patient in 30° supine position

- > **Apical impulse:** with the fingertips; left lateral supine position as required; **medial to the midclavicular line; 4th or 5th intercostal space; diameter < 2 cm** +
- **Apical pulsation:** corresponds to **isovolumic contraction of the LV**
- **Hyperdynamic apex:** increased amplitude of apical impulse but of normal duration; AR; patent ductus arteriosus; MR; VSD; hyperthyroidism; anemia; pregnancy
- **Sustained apical impulse:** **the impulse persists during or after the carotid upstroke;** associated with **pressure overload** (AS; HCM; HTN) +
- **Enlarged apical impulse:** dilatation > 2 cm and/or shift downwards and to the left; associated with **LV volume overload** +
- **Palpable S3:** LV volume overload
- **Palpable S4:** noncompliant LV / ↑ end-diastolic pressure
- **Triple apical impulse:** HCM (early-systole; end-systole due to dynamic LVOT obstruction; S4)
- > **Ectopic pulsation:** LV aneurysm (mid-precordial or anterior axillary)
- > **Left parasternal heave:** associated with RVH
- > **Thrill:** palpate the 4 areas (palm of the hand at the level of the MCP joints)
- > **Pulsation in the 2nd right intercostal space:** ascending aortic aneurysm
- > **Pulsation in the 2nd left intercostal space:** PA dilatation

AUSCULTATION - SYSTOLIC HEART SOUNDS



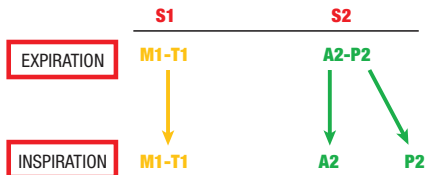
LOOK FOR: S1; Ejection click; Mid-systolic click; S2

S1: M₁-T₁ (interval: 20-30 ms); maximum at the apex; T₁ mainly in the 5th left intercostal space; precedes the carotid upstroke

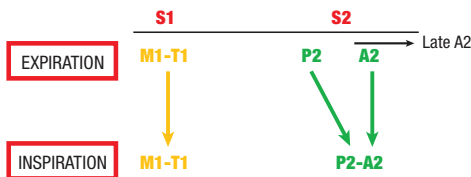
- > ↗ **Intensity of S1 (S1 ≥ S2 in the 2nd left intercostal space)**: Rheumatic MS (early stage); Hyperdynamic state (↗ dP/dt); PR < 120 ms; ↗ flow through mitral valve (VSD; patent ductus arteriosus)
- > ↘ **Intensity of S1**: Calcified MS (↘ mobility); Systolic dysfunction; PR > 200 ms; acute AR
- > **Variable intensity of S1**: AF; AV dissociation; Tamponade
- > **Split S1 (split sound in the 5th left intercostal space)**: RBBB; ASD (delayed T1); ST (delayed T1); Ebstein's anomaly

S2: A2-P2; maximum in the 2nd left intercostal space

- > **Normal physiological splitting**: on inspiration; 2nd left intercostal space; interval: 20-60 ms



- > **Normal but narrow inspiratory splitting**: PHT (associated with ↗ P2)
- > **Increased splitting**: RBBB; severe MR (early A2); VSD (early A2); RVOT obstruction (late P2)
- > **Fixed splitting (A2-P2 variation < 20 msec)**: ASD; right heart failure (absence of variation of ejection volume according to RV preload)
- > **Paradoxical splitting**: LBBB; RV PPM; AS; HCM; LV systolic dysfunction; AR (prolonged ejection)



- > ↗ **intensity of A2**: HTN; CoA; Ascending aortic aneurysm; Transposition of the great arteries; supravulvar AS
- > ↘ **intensity of A2**: Valvular AS; AR
- > ↗ **intensity of P2**: P2 > A2 in the 2nd left intercostal space or P2 heard at the apex or palpable P2; PHT; Supravulvar RVOT obstruction
- > ↘ **intensity of P2**: Valvular PS; pulmonary regurgitation (except if secondary to PHT); Transposition of the great arteries
- > **Single S2**: ↘ A2 (AS) or ↘ P2 (PS); Transposition of the great arteries

VALVULAR EJECTION CLICK: coincides with carotid upstroke (120-140 ms after QRS); high-pitched sound; diffuse radiation; best heard in lower left parasternal region; associated with bicuspid aortic valve (valve still pliable) or congenital PS (↘ click on inspiration; valve still pliable) +

- > **Vascular ejection click**: Aortic root dilatation; PA dilatation (idiopathic; post-stenotic; PHT)

MID-SYSTOLIC CLICK OF MVP: non-ejection click (after the carotid pulsation); high-pitched sound; earlier if the patient stands up; ± MR murmur

AUSCULTATION - DIASTOLIC HEART SOUNDS

LOOK FOR: S2; Opening snap; Pericardial knock; S3; Tumor plop; S4; Friction rub

MITRAL OPENING SNAP: High-pitched sound; stethoscope diaphragm at the apex; **A2-opening snap interval inversely proportional to the severity of MS** (40-120 ms after S2); interval decreases with tachycardia; ↘ intensity of opening snap when the valve is calcified

PERICARDIAL KNOCK: Constrictive pericarditis; early diastolic sound (at the end of the y descent); 100-120 ms after S2

S3: left lateral supine position; stethoscope bell at the apex; 140-160 ms after S2; during rapid ventricular filling (at the end of the y descent); associated with **ventricular volume overload** +

> **Etiologies:** Dilated cardiomyopathy; heart failure; MR; AR; VSD; patent ductus arteriosus; diastolic dysfunction; young subjects in good health; normal pregnancy

> **Right S3:** lower left parasternal region; ↗ on inspiration; TR; right heart failure; PHT

S4: left lateral supine position; stethoscope bell at the apex; occurs during atrial kick (after the P wave); associated with a **poorly compliant ventricle** and ↗ **filling pressure** +

> **Etiologies:** HTN; AS; HCM; LVH; Ischemia; Acute AR; Acute MR; Age

> **Right S4:** lower left parasternal region; ↗ on inspiration; RVOT obstruction; PHT

SUMMATION GALLOP: fusion of S3 and S4 during tachycardia

TUMOR PLOP: prolapse of the tumor through the AV valve; sound varies with position

PERICARDIAL FRICTION RUB: 1 or 2 or 3 components (rapid ventricular filling; atrial kick; ventricular systole); forced expiration while leaning forward; stethoscope diaphragm in left parasternal region

AUSCULTATION - MURMURS

IDENTIFY: Moment of the cycle; Configuration (crescendo; decrescendo; crescendo-decrescendo; plateau); Site; Radiation; Tone; Intensity; Modifiers (breathing; special maneuvers)

> **1/6:** Very faint murmur (barely perceptible)

> **2/6:** Faint murmur but heard immediately

> **3/6:** Moderate murmur

> **4/6:** Palpable thrill

> **5/6:** Very loud; heard even when only part of the stethoscope is in contact with the chest

> **6/6:** Heard even when the stethoscope is not in contact with the chest

BENIGN MURMUR: 1-2/6 in left parasternal region; Ejection murmur; S2 of normal intensity with normal physiological splitting; No other heart sounds or murmurs; No LVH (on examination or ECG); Murmur not increased by Valsalva maneuver or standing

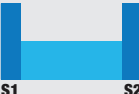





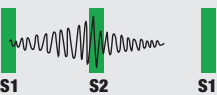
INDICATIONS FOR TTE: Diastolic or continuous or holosystolic or end-systolic or early systolic murmur or associated with ejection click or ≥ 3/6 with mid-systolic peak or radiating to the neck or back or signs or suspicion of MR, MVP, HCM or VSD on dynamic auscultation

SYSTOLIC MURMUR

Mid-systolic
(often diamond-shaped)



- Benign
- Ejection murmur: High output state (pregnancy; hyperthyroidism; anemia; AV fistula; AR; PR; ASD)
- Aortic stenosis (supra-avalvular; valvular; subvalvular)
- Aortic sclerosis
- HCM
- PS (supra-avalvular; valvular; subvalvular)
- CoA
- Functional / ischemic MR

<p>Holosystolic</p>  <p>S1 S2</p>	<ul style="list-style-type: none"> • MR • TR • VSD (restrictive)
<p>Early systolic</p>  <p>S1 S2</p>	<ul style="list-style-type: none"> • Acute MR • Primary TR (without PHT) • VSD: small muscular VSD or large VSD with significant PHT
<p>End-systolic</p>  <p>S1 S2</p>	<ul style="list-style-type: none"> • MVP • Functional / ischemic MR (↗ on exercise) • Tricuspid valve prolapse
DIASTOLIC MURMUR (ALWAYS INVESTIGATE)	
<p>Early diastolic</p>  <p>S2 S1</p>	<ul style="list-style-type: none"> • AR • Graham-Steell: PR secondary to PHT (decrecendo; high-pitched; signs of associated PHT) • PR without PHT (faint murmur)
<p>Mid-diastolic</p>  <p>S2 S1</p>	<ul style="list-style-type: none"> • MS • TS • Austin-Flint (absence of opening snap) • Myxoma • ↗ diastolic flow through AV valve (MR; TR; VSD; patent ductus arteriosus; ASD; abnormal pulmonary venous return) • Carey Coombs murmur (mitral valvulitis during acute rheumatic fever)
<p>End-diastolic</p>  <p>S2 S1</p>	<ul style="list-style-type: none"> • MS (presystolic accentuation) • TS • Austin-Flint • Myxoma • Ryland's murmur: diastolic mitral regurgitation in a context of complete AV block
CONTINUOUS MURMUR (OFTEN PATHOLOGICAL)	
<p>Starts at systole and continues uninterrupted during diastole</p>  <p>S1 S2 S1</p>	<ul style="list-style-type: none"> • Patent ductus arteriosus (Gibson's murmur; machinery murmur) • Aortopulmonary window • Coronary arteriovenous fistula • Ruptured aneurysm of the sinus of Valsalva • Neck venous hum • Mammary murmur of pregnancy • Stenosis of peripheral branch of pulmonary artery • Lutembacher's syndrome: MS + ASD • CoA / Intercostal collateral vessels • Pulmonary or systemic AV fistula • Bronchial collateral vessels

DYNAMIC AUSCULTATION

MANEUVERS	AS	HCM	MVP	MR	OTHER
Valsalva (↘ preload)	↘	↗	↗ duration of murmur	↘	↘ AR
Standing up (↘ preload)	↘	↗	↗ duration of murmur	↘	
Squatting or leg raising (↗ preload)	unchanged or ↗	↘	↘ duration of murmur	↗	↗ AR - ↗ VSD
Hand grip (↗ afterload)	unchanged or ↘	↘	↘ duration of murmur	↗	↗ AR - ↗ VSD ↗ MS (↗ HR)
Amyl nitrate (↘ afterload)	↗	↗	↗ duration of murmur	↘	↗ MS (↗ HR) ↘ AR - ↘ VSD ↘ Austin Flint
Post-PVC (↗ contractility)	↗	↗	↘ duration of murmur (↗ LV volume)	un- changed	

INSPIRATION: louder right heart sounds/murmurs (except for pulmonary valve ejection click);
↗ splitting of S2

PHYSICAL EXAMINATION - OTHER EXAMINATIONS

LUNGS: crackles; effusion; wheezing; pleural friction rub

ABDOMEN: Liver (pulsation); Ascites; Splenomegaly; Aorta; Murmurs

LOOK FOR PRESACRAL EDEMA

UPPER LIMBS: clubbing; arachnodactyly; signs of endocarditis; nicotine stains; sclerodactyly; pulse in both arms

LOWER LIMBS: murmurs; lower limb edema; pulse in both legs; capillary refill; discoloration; ulcer; coldness; atrophic changes; hair loss

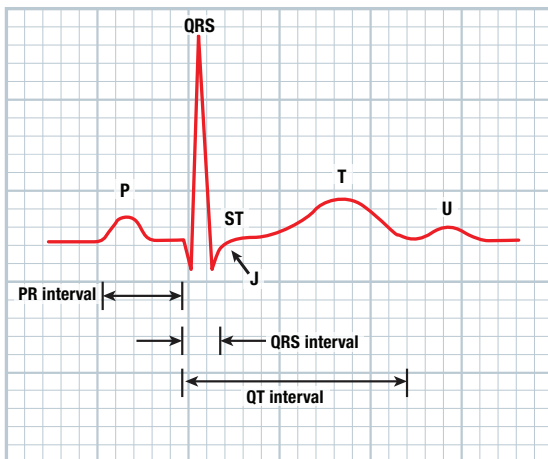
> **Radiofemoral delay:** CoA

> **Pulse:** 0 = absent; 1 = decreased; 2 = normal; 3 = bounding

BRIEF NEUROLOGICAL EXAMINATION**1.2/ ELECTROCARDIOGRAM (ECG)**

SYSTEMATIC APPROACH

- HEART RATE:** 1500 / number of small squares between 2 QRS or 300-150-100-75-60-50-43-38
- RHYTHM** (▶▶ Chapter 6 - Arrhythmias)
- P WAVE:** morphology
- AV CONDUCTION**
- QRS:** frontal axis; intraventricular conduction; precordial QRS transition; voltage / chamber hypertrophy
- REPOLARIZATION:** ST segment - T wave - QT interval - U wave
- MYOCARDIAL INFARCTION / Q WAVE**



NORMAL VALUES	
Calibration	<ul style="list-style-type: none"> • Vertical: 10 mm = 1 mV • Horizontal: 1 mm = 40 msec
P wave duration and amplitude	< 120 ms and < 2.5 mm in amplitude
P wave axis	60° (positive I-II-aVL-aVF; negative aVR) Normal axis: 0-90°
PR interval	120-200 ms
QRS duration	≤ 110 ms
QRS axis	-30° to +90°
Precordial QRS transition	R = S in V3 or V4
QRS amplitude	<ul style="list-style-type: none"> • Limbs: > 5 mm • Precordial: > 10 mm
J Point / ST segment	<p>Elevation</p> <ul style="list-style-type: none"> • V2-V3: < 2 mm (men > 40 years); < 2.5 mm (men < 40 years); < 1.5 mm (women) • Other leads: < 1 mm <p>Depression: < 0.5 mm</p>
T wave	<ul style="list-style-type: none"> • Positive: I-II-V3-V4-V5-V6 > V5-V6: T wave inversion < 1 mm in 2% • Negative: aVR • Variable: aVL-III-V1-V2 • Maximum amplitude V2: < 14 mm (men) and 10 mm (women)
QTc	<ul style="list-style-type: none"> • Men: < 450 ms • Women: < 460 ms

TECHNICAL CONSIDERATIONS

ARM LEAD REVERSAL: P and QRS and T wave inverted in I and aVL but not in V6

PRECORDEAL LEAD MALPOSITION: abnormal precordial QRS transition

RIGHT HEART OR POSTERIOR LEADS: V3R-V4R or V7-V8-V9

ARTIFACTS: tremor; Parkinson's disease (pseudo-flutter)

SINUS RHYTHM

►► Chapter 6 - Arrhythmias

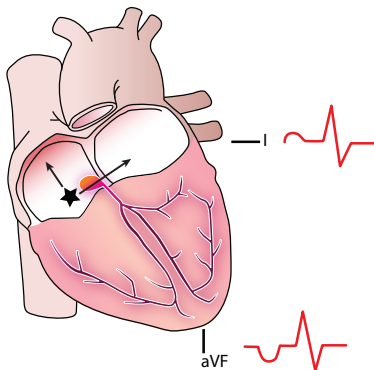
SINUS RHYTHM (SR): origin of sinoatrial node; HR 60 -100 bpm

> **Sinus tachycardia:** HR > 100 bpm

> **Sinus bradycardia:** HR < 60 bpm

> **Normal atrial activation:** RA to AV node and to LA; P axis 0° to +90° (positive P wave I-II-aVL-aVF); biphasic P wave in V1-V2; duration < 120 ms

> **Retrograde atrial activation:** retrograde AV conduction or ectopic atrial pacemaker close to AV node; negative P wave in II and aVF



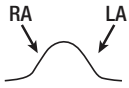
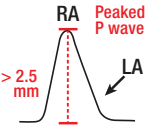
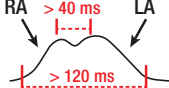

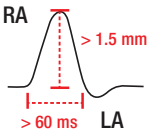
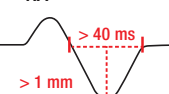
SINUS ARRHYTHMIA: phasic respiratory variation of the duration of the PP interval; variation ≥ 120 ms or $PP_{max} - PP_{min} / PP_{min} > 10\%$

SUPRAVENTRICULAR ARRHYTHMIAS

►► Chapter 6 - Arrhythmias

VENTRICULAR ARRHYTHMIAS

►► Chapter 6 - Arrhythmias

	NORMAL	RA ABNORMALITY	LA ABNORMALITY
II			
V1			
	<ul style="list-style-type: none"> • Axis 60° • Duration < 120 ms 	<ul style="list-style-type: none"> • Axis $> 75^\circ$ 	<ul style="list-style-type: none"> • Axis terminal portion -30° to -90° (negative in III)

INTRA-ATRIAL CONDUCTION BLOCK: Duration of P wave > 120 ms; biphasic P wave in inferior leads; does not satisfy the criteria for RA or LA anomaly

- > **Abnormal activation:** RA activation \rightarrow Bachmann's bundle block \rightarrow LA activation from coronary sinus (from inferior to superior)

AV CONDUCTION

►►| Chapter 6 - Arrhythmias

NORMAL PR INTERVAL: 120-200 ms

LONG PR INTERVAL: PR > 200 ms; 1st degree AV block

SHORT PR INTERVAL: PR < 120 ms in sinus rhythm; rule out accessory pathway

- > **Lown-Ganong-Levine syndrome:** atrio-His accessory pathway (James fibers) short-circuiting the AV node; narrow QRS

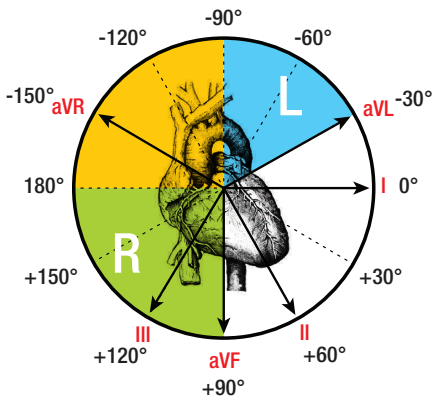
X:Y AV CONDUCTION: failure of AV node to conduct certain atrial impulses to the ventricle; **the refractory period of the AV node is longer when it is stimulated more rapidly (decremental conduction)** +

- > **AV block with variable conduction:** 2:1 3:1...; frequent in atrial flutter

AV DISSOCIATION: independent atrial and ventricular rhythms; 3 situations:

- 1) 3rd degree AV block; RR interval $>$ PP interval
- 2) Accelerated junctional rhythm or junctional tachycardia or VT (without retrograde VA conduction); RR $<$ PP; \pm fusion or capture beats
- 3) Sinus bradycardia with junctional or ventricular escape (without retrograde VA conduction); RR $<$ PP; \pm isorhythmic dissociation

FRONTAL QRS AXIS

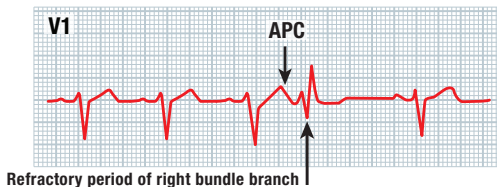


Normal axis	-30° to +90°	I + aVF + (if aVF - → II +)
Right axis deviation	+90° to +180°	I - aVF + DDx: RVH; LPHB; dextrocardia; lateral infarction; secundum ASD; vertical heart (COPD); pulmonary embolism
Left axis deviation	-30° to -90°	I + aVF - and II - DDx: LVH; LAHB; primum ASD; complete AV canal defect; Tricuspid atresia (under-developed RV); pregnancy; ascites; inferior infarction
Extreme "North-West" axis deviation	-90° to 180°	I - aVF -

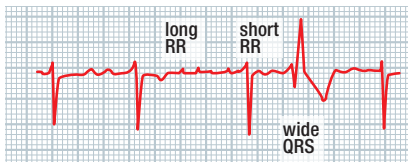
INDETERMINATE AXIS: Equiphasic QRS in all frontal leads (with no dominant QRS); axis perpendicular to the frontal plane

QRS: INTRAVENTRICULAR CONDUCTION

ABERRANT CONDUCTION OF SUPRAVENTRICULAR BEATS: arrival of a supraventricular beat during the relative refractory period of intraventricular conduction tissue; conduction with wide QRS (RBBB > LBBB morphology)



ASHMAN PHENOMENON: Long RR then short RR → wide QRS (frequent RBBB morphology); long RR associated with ↗ refractory period of His-Purkinje tissue



LAHB (LEFT ANTERIOR HEMIBLOCK)

- 1) Left axis deviation -45° to -90°
 - 2) qR in I and aVL
 - 3) rS in III and aVF
 - 4) QRS duration < 120 ms
 - 5) R wave peak time in aVL > 45 ms
 - > Late precordial QRS transition
- > **Rule out differential diagnoses:** LVH; COPD; inferior infarction

LPHB (LEFT POSTERIOR HEMIBLOCK)

- 1) Right axis deviation $+90^\circ$ to $+180^\circ$
 - 2) rS in I and aVL
 - 3) qR in III and aVF
 - 4) QRS duration < 120 ms
- > **Rule out differential diagnoses:** RVH; COPD; lateral infarction; dextrocardia; arm lead reversal

LAHB		LPHB	
I – aVL	III – aVF	I – aVL	III – aVF
<p>aVL: R wave peak time > 45 ms</p>			

LBBS (LEFT BUNDLE BRANCH BLOCK)

- 1) QRS ≥ 120 ms
- 2) Wide monophasic R wave in I-aVL-V5-V6
- 3) Absence of septal q wave in I-V5-V6
- 4) R wave peak time > 60 ms in V5-V6
- 5) ST and T in opposite direction to QRS (appropriate discordance)

<p>Acute myocardial infarction with a LBBS (Sgarbossa criteria)</p>	<ol style="list-style-type: none"> 1) ST elevation ≥ 1 mm in leads with positive QRS (inappropriate concordance) 2) ST depression ≥ 1 mm V1-V2-V3 (inappropriate concordance) 3) ST elevation ≥ 5 mm in leads with negative QRS (extreme discordance)
<p>Old myocardial infarction in LBBS</p>	<ol style="list-style-type: none"> 1) Cabrera's sign: Notch of the upslope of the S wave in V2-V3-V4 2) Chapman's sign: Notch of the upslope of the R wave in V5-V6-I-aVL

INCOMPLETE LEFT BUNDLE BRANCH BLOCK

- 1) QRS 110-119 ms
- 2) LVH pattern
- 3) R wave peak time > 60 ms in V5-V6
- 4) Absence of septal q wave in I-V5-V6

RBBB (RIGHT BUNDLE BRANCH BLOCK)

- 1) QRS \geq 120 ms
- 3) rsr' , rsR' , or rSR' in V1 or V2 (width of R' or r' > width of r)
- 4) S > 40 ms in I and V6 (S larger than R in V6)
 - > In a minority of patients, wide monophasic R wave in V1 and/or V2 (with R wave peak time > 50 ms in V1)
 - > **If axis deviation:** consider bifascicular block (RBBB with LAHB or RBBB with LPHB)
 - > **If bifascicular block with \nearrow PR:** consider lesion of 3 branches with prolonged HV interval

INCOMPLETE RIGHT BUNDLE BRANCH BLOCK

- 1) QRS 110-119 ms
- 2) Other criteria similar to RBBB

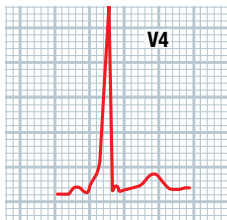
	NORMAL	RBBB	LBBB
V1 V2		<p>QRS > 120 ms</p> <p>r</p> <p>R'</p> <p>rsr' or rsR' or rSR'</p> <p>S</p>	<p>QRS > 120 ms</p> <p>Deep S wave</p> <p>r wave < 30 ms or absent</p>
V5 V6		<p>R</p> <p>I and V6: S > 40 ms</p> <p>q</p> <p>S</p>	<p>Prominent wide R wave</p> <p>Absence of septal Q wave</p> <p>R wave peak time > 60 ms</p>

NONSPECIFIC INTRAVENTRICULAR CONDUCTION DISORDER

- 1) QRS > 110 ms
- 2) Absence of criteria of RBBB or LBBB

VENTRICULAR PRE-EXCITATION

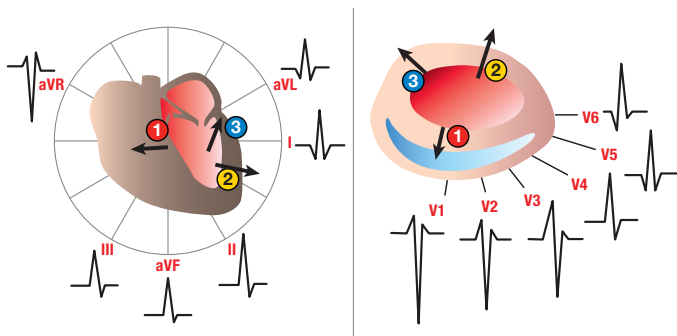
- 1) PR interval < 120 ms (in sinus rhythm)
- 2) **Delta Wave:** slow rise of the initial portion of the QRS
- 3) QRS \geq 120 ms
- 4) Secondary ST and T anomalies
 - > Possible pseudo-infarction (Q waves)
 - > **Concertina effect:** the degree of pre-excitation can vary according to conduction and the refractory period of the accessory pathway and AV node



SITE OF ACCESSORY PATHWAY	V1	aVF	aVL
Left lateral	+	+	-
Posterior or left septal	+	-	+
Posterior or right septal	-	-	+
Anterior or right lateral	-	+	+

QRS: PRECORDIAL R WAVE TRANSITION

NORMAL VENTRICULAR ACTIVATION: 1) Left-to-right septal activation (septal q wave in I-aVL-V5-V6; septal r wave in aVR and V1); 2) Anterior LV then lateral LV activation; 3) Posterobasal LV activation



NORMAL PRECORDIAL TRANSITION: rS V1 → qR V6; R = S in V3 or V4

- **DDx of abnormal precordial transition:** lead malposition; dextrocardia; anterior or anteroseptal or posterior myocardial infarction; LVH; RVH; LAHB; LBBB; RBBB; dilated or infiltrative cardiomyopathy; pre-excitation (right or anteroseptal accessory pathway); COPD; pneumothorax; chest wall anomaly...

LOW VOLTAGES: QRS amplitude < 5 mm on frontal leads and < 10 mm on precordial leads

- **DDx:** COPD; obesity; cardiomyopathy; infiltrative disease - amyloidosis - tumor; myocarditis; extensive MI; pericardial effusion; constrictive pericarditis; pleural effusion; myxedema; anasarca; calibration; left pneumothorax