

Chapter 4. Cardiovascular Physiology

Part 1 | Heart Anatomy & Function

1 Learning Objectives

After finishing this part, you will be able to ...

1. **Trace the events of a complete cardiac cycle** and relate pressure-volume changes to the Wiggers diagram.
2. **Define and calculate cardiac output (CO)** and its determinants (HR × SV), explaining how exercise and pathology modify each variable.
3. **Identify the components of a normal electrocardiogram (ECG)**, measure key intervals, and recognise the electrical basis of selected arrhythmias.
4. **Apply cardiac-cycle and ECG knowledge to physiotherapy practice**—vital-sign monitoring, exercise prescription, emergency recognition.

2 Cardiac Cycle Overview

Phase	Mechanical Event	Valve Status	Pressures*	Heart Sound
Atrial systole (~0.1 s)	Atria contract—"atrial kick" (≈ 20 % EDV)	AV open; SL closed	Atrial P ↑; Ventricular P slight ↑	—
Isovolumetric ventricular contraction	Ventricles contract; all valves closed	AV snap shut	LV P ↑ rapidly to >80 mm Hg	S₁ ("lub")
Ventricular ejection (rapid & reduced)	SL valves open; blood expelled	SL open	LV P peaks 120 mm Hg; Ao P follows	—
Isovolumetric relaxation	Ventricles relax; all valves closed	SL close	LV P ↓ below Ao; AV still closed	S₂ ("dub")
Passive filling (rapid + diastasis)	AV valves open; ventricles fill 80 %	AV open	Vent P low; atria refill	Possible S₃ (normal youth / HF)

*Pressures given for left heart at rest.

Physio Pearl: Orthostatic hypotension occurs when baroreflex fails to boost HR & SVR during transition from isovolumetric relaxation to passive filling—instruct slow positional changes with ankle pumps.

3 Cardiac Output (CO)

$$\text{CO} = \text{Heart Rate (HR)} \times \text{Stroke Volume (SV)}$$

Variable	Determinants	Exercise Effect	PT Implication
HR	SA-node rate ± autonomic tone	Linear ↑ to HR _{max} (≈ 220 – age)	β-blockers blunt HR—use RPE 11-13
SV	EDV (preload), contractility, afterload	↑ 40-60 % VO ₂ max then plateaus	Upright cycling: calf pump ↑ preload; avoid valsalva (↑ afterload)
Ejection Fraction (EF)	SV / EDV (normal ≥ 55 %)	Slight ↑ during exercise	HFpEF vs HFrEF guides intensity

Fick equation (indirect VO₂ → CO): $\text{CO} = \frac{\text{VO}_2}{\text{a-v O}_2 \text{ diff}}$

4 Wiggers Diagram Snapshot

Synchronises electrical (ECG), mechanical (pressure-volume), and acoustic (heart sounds) events.

LV Pressure: /\ /
Aortic Pressure: / \ /
LA Pressure: _/_ _/_
Heart Sounds: S1 S2
ECG: P QRS T

Understand timing to position stethoscope and interpret murmurs (e.g., systolic ejection in aortic stenosis between S₁-S₂).

5 Electrocardiogram (ECG) Basics

Wave / Interval	Electrical Event	Normal Duration	Clinical Clue
P wave	Atrial depolarisation	≤ 0.12 s	Tall P—RA enlargement
PR interval	AV nodal delay	0.12-0.20 s	> 0.20 s = 1° AV block
QRS complex	Ventricular depolarisation	≤ 0.10 s	Wide QRS—bundle-branch block
ST segment	Ventricular plateau	Isoelectric	Elevation -> acute MI
T wave	Ventricular repolarisation	—	Peaked T—hyperkalaemia
QTc	Vent depol+repol	≤ 0.44 s (rate-corrected)	Long QT—torsades risk

Axis Quick-Check

Lead I & aVF both positive → **Normal axis** (-30° to +90°). Deviation may signal hypertrophy or conduction block.

Arrhythmia Nuggets

Rhythm	ECG Hallmark	PT Action
Sinus tachycardia	Normal P, HR > 100	Expected in exercise; monitor if HR > HR _{max}
Atrial fibrillation	No P, irregular RR	Check radial pulse irregularity; RPE for intensity
Ventricular tachycardia	Wide QRS ≥ 3 beats	Emergency—stop rehab, activate code

Safety Rule: Terminate exercise if ST depression ≥ 2 mm, drop in SBP > 10 mm Hg, or symptomatic arrhythmia.

6 Integration: From ECG to Cardiac Output During Exercise

- Warm-up:** HR ↑ via sympathetic drive; P-R shortens, SV rises from Frank-Starling preload.
- Steady-state aerobic:** CO plateaus; ST should remain at baseline.
- High-intensity:** If ST drifts or frequent PVCs appear, reduce intensity.

7 Self-Check Quiz (answers below)

- Which cardiac phase follows closure of the semilunar valves?**
- Calculate CO if HR = 90 bpm and SV = 80 mL.**

3. **What ECG interval lengthens in first-degree AV block?**
4. **List two mechanisms that increase stroke volume during aerobic exercise.**
5. **Why might beta-blockers mask early signs of myocardial ischaemia on an exercise ECG?**

1. **Isovolumetric relaxation.**
2. $CO = 90 \times 0.08 \text{ L} = 7.2 \text{ L min}^{-1}$.
3. **PR interval** ($> 0.20 \text{ s}$).
4. Enhanced **preload (venous return)** and increased **contractility** via sympathetic activation.
5. They blunt sympathetic HR and contractility rise, reducing demand and attenuating ischaemic ST changes.

8 Key Take-Home Points

- The **cardiac cycle links pressure, volume, sound and electricity**—master the timeline to interpret vitals correctly.
- **Cardiac output is adjustable** via HR and SV; physiotherapists use graded exercise, position, and hydration to influence both.
- A **systematic ECG review (rate-rhythm-axis-intervals-ST-extras)** enables rapid detection of unsafe patterns before or during therapy.

Part 2 | Blood Vessels & Circulation

1 Learning Objectives

By the end of this part you will be able to ...

1. **Compare the histological layers and mechanical properties** of arteries, arterioles, capillaries, venules and veins.
2. **Trace blood flow through the systemic and pulmonary circuits**, noting pressure changes and velocity profiles.
3. **Explain short- and long-term mechanisms that regulate arterial blood pressure (BP)** and why they matter during physiotherapy.
4. **Apply vessel physiology to patient scenarios** such as orthostatic hypotension, intermittent claudication, chronic venous insufficiency and edema control.

2 Vessel Structure & Function

Layer (inside → out)	Arteries	Capillaries	Veins
Tunica intima	Endothelium + internal elastic lamina (IEL)	Endothelium only (~1 μm)	Endothelium, sparse IEL
Tunica media	Elastic arteries: 40-70 elastic lamellae Muscular arteries/arterioles: 1-40 smooth-muscle layers	—	Thin; few muscle cells
Tunica externa (adventitia)	Collagen & vasa vasorum (in large vessels)	—	Dominant layer; collagen + valves (infoldings of intima) in limbs
Wall : lumen ratio	High (thick wall)	1 : 1	Low (thin wall, large lumen)
Compliance ($\Delta V/\Delta P$)	Low (except elastic aorta)	N/A	High - ~ 60 % blood volume reservoir
Function	Pressure reservoir & distribution; arterioles = resistance control	Exchange of gases, nutrients, wastes	Capacitance; one-way return; reservoir for mobilization during exercise

Poiseuille's Law $R = \frac{8\eta L}{\pi r^4}$ $R = \frac{8\eta L}{\pi r^4}$

→ **Arteriolar radius (r) is the biggest determinant of systemic vascular resistance (SVR).**

3 Microcirculation - Capillary Exchange

- **Continuous capillaries:** Tight junctions; muscle, brain → precise control.
- **Fenestrated:** Pores; kidney, intestine → rapid filtration.
- **Sinusoidal:** Large gaps; liver, marrow → cell movement.

Starling Forces (mm Hg) $J_v = K_f[(P_c - P_i) - \sigma(\pi_c - \pi_i)]$ $J_v = K_f[(P_c - P_i) - \sigma(\pi_c - \pi_i)]$

Symbol	Meaning
P_c	Capillary hydrostatic pressure (outward)
π_c	Capillary oncotic pressure (inward, albumin)
K_f	Filtration coefficient (permeability × surface)

Physio link: Manual lymph drainage & muscle pump ↑ interstitial negative pressure and lymph flow → reduce edema.

4 Systemic vs Pulmonary Pressures

Site	Systolic/Diastolic (mm Hg)	Mean	Velocity
Aorta	120 / 80	100	~30 cm s ⁻¹
Arterioles	80 → 35	50	rapid drop
Capillaries	—	25 (arterial end) → 10 (venous end)	slowest (~0.1 cm s ⁻¹) - exchange rises again
Vena cava	—	2-5	low-pressure circuit
Pulmonary artery	25 / 8	15	

5 Blood-Pressure Regulation

5.1 Short-Term (Seconds - Minutes)

Sensor	Pathway	Effector	Example in PT
High-pressure baroreceptors (carotid sinus, aortic arch)	CN IX, X → medulla (NTS)	Vagus ↓ HR; SNS ↓ SVR	Orthostatic training—baroreflex adapts in 5-7 days
Low-pressure (volume) receptors (atria, pulmonary)	Vagal afferents	ADH & sympathetic modulation	Aquatic therapy ↑ central volume → diuresis
Chemoreceptors (carotid & aortic bodies)	↑ CO ₂ , ↓ O ₂	↑ SNS, ventilation	COPD rehab—avoid severe hypoxia triggers

5.2 Intermediate

System	Trigger	Action
RAAS	↓ Renal perfusion / SNS β ₁	Renin → Ang II → vasoconstriction + aldosterone → Na ⁺ /H ₂ O retention
ADH (vasopressin)	↑ Osmolality or ↓ BP	V ₂ receptors ↑ H ₂ O reabsorption; V ₁ vasoconstriction

5.3 Long-Term (Days - Weeks)

- **Renal-body fluid mechanism:** Pressure-natriuresis shifts; ultimately sets arterial pressure.
- **Structural vascular adaptation:** Chronic exercise ↓ arterial stiffness (elastin maintenance).

6 Clinical & Physiotherapy Implications

Scenario	Physiological Basis	Intervention
Orthostatic hypotension post-bedrest	↓ Blood volume & baroreflex sensitivity	Gradual tilt-table, compression stockings, hydration
Intermittent claudication (PAD)	Atherosclerotic narrowing; ↓ flow	Graded walking to near-pain—induces collateral growth
Chronic venous insufficiency	Valve incompetence; high venous P	Calf-pump exercises, graduated compression 30-40 mm Hg
Resistance training BP spikes	Valsalva ↑ intrathoracic P → ↑ afterload	Teach exhale on effort; monitor SBP < 220 mm Hg

7 Self-Check Quiz (answers below)

1. Which vessel type is the primary determinant of systemic vascular resistance and why?
2. Explain how skeletal-muscle contraction aids venous return.
3. What baroreceptor reflex change occurs during sustained endurance training?
4. Give two reasons capillaries are ideal for exchange.
5. Calculate mean arterial pressure (MAP) if BP = 130/80 mm Hg.

Answers

1. **Arterioles**—their lumen radius is small and highly adjustable; resistance $\propto 1/r^4$ (Poiseuille).
2. Contraction compresses veins, pushing blood toward the heart; valves prevent backflow—the “muscle pump.”
3. Set-point shifts slightly lower; baroreflex curve resets, allowing lower resting HR/BP without triggering reflex tachycardia.
4. Single endothelial layer (short diffusion distance) and enormous total cross-sectional area (low flow velocity).
5. $MAP \approx DBP + \frac{1}{3}(SBP - DBP) \rightarrow 80 + (50/3) \approx 93 \text{ mm Hg}$.

8 Key Take-Home Points

- **Arteries withstand pressure; arterioles regulate it; capillaries exchange; veins store and return.**
- Blood pressure is kept within tight limits by **rapid neural reflexes and slower hormonal-renal systems**—exercise challenges both.
- Physiotherapists manipulate **position, muscle pump, graded activity and external compression** to optimise circulation and control BP-related risks.

Part 3 | Hemodynamics & Cardiovascular Disorders

1 Learning Objectives

After this section you should be able to ...

1. **Interpret the physical laws that govern blood flow** (pressure, resistance, compliance, inertia, viscosity).
2. **Predict how changes in vessel radius, length or viscosity alter flow and shear stress**—the foundations of

many pathologies.

3. **Relate the hemodynamic consequences** of key cardiovascular disorders to the clinical signs you monitor in physiotherapy.
4. **Adjust exercise and positioning** based on each disorder’s physiological limitations and risk profile.

2 Blood-Flow Dynamics—Core Principles

Law / Concept	Key Equation	Practical Meaning
Poiseuille’s Law (laminar flow)	$Q = \Delta P \cdot \pi \cdot r^4 / 8 \eta L$ $Q = 8 \eta L \Delta P \cdot \pi \cdot r^4$	Radius (r) is the “volume knob”—a 16 % ↑ r doubles flow.
Resistance	$R = 8 \eta L / \pi r^4$ $R = \pi r^4 / 8 \eta L$	Arteriolar tone sets systemic vascular resistance (SVR).
Flow Velocity	$v = Q / A$ $v = Q / \pi r^2$	Capillaries: huge A → very slow v → exchange time.
Reynolds Number	$Re = \rho \cdot v \cdot D$ $Re = \eta \rho \cdot v \cdot D$	>2000 → turbulent → murmurs, bruit in stenoses.
Compliance	$C = \Delta V / \Delta P$ $C = \Delta P / \Delta V$	Veins highly compliant; aging arteries lose compliance → ↑ pulse pressure.
Shear Stress	$\tau = 4 \eta Q / \pi r^3$ $\tau = 4 \eta Q / \pi r^3$	Moderate laminar shear releases NO (atheroprotection); oscillatory shear promotes plaque.

Physio Pearl: Slow rhythmic diaphragmatic breathing lowers intrathoracic pressure swings, boosting venous return and stroke volume—useful in hypotensive clients.

3 Common Cardiovascular Disorders & Hemodynamic Impact

Disorder	Primary Lesion / Change	Hemodynamic Consequence	Physiotherapy Considerations
Systemic Hypertension	↑ SVR (arteriolar constriction + stiffness)	LV after-load ↑ → concentric hypertrophy, ↓ compliance	Gradual aerobic conditioning ↓ SVR; avoid Valsalva during strength sets
Atherosclerosis / Coronary Artery Disease	Intimal plaque → radius ↓, turbulence ↑	↓ Coronary flow reserve; risk of ischemia with modest ↑ HR	Use RPE & angina scale; interval progression only if symptom-free & ECG stable
Heart Failure (HFrEF)	↓ Contractility → ↓ SV, ↑ EDV	Pulmonary & systemic congestion; low perfusion at rest/exercise	Interval or continuous exercise at 40–60 % VO ₂ peak; monitor weight & edema daily
Aortic Stenosis	Fixed outflow obstruction → pressure gradient >40 mm Hg	Severe LV pressure load; CO can’t rise with exercise	CONTRA high-intensity; terminate exertion if SBP drop or dizziness
Aneurysm (Abdominal Aorta)	Medial degeneration → ↑ diameter ↓ wall shear	Law of Laplace: Tension = P·r → risk rupture if >5.5 cm	Avoid heavy lifting & spikes in BP; emphasize breathing control
Peripheral Arterial Disease (PAD)	Plaque in limb arteries; ↓ r → critical drop in Q	Claudication pain at low workloads	Supervised walking to near-pain threshold 3–5 d·wk ⁻¹ stimulates collaterals
Deep-Vein Thrombosis / CVI	Stasis + valve failure; ↑ venous P	Edema, ulcer, embolus risk	Early mobilisation, ankle pumps; class II–III compression; contraindicate vigorous massage over DVT
Orthostatic Hypotension	Baroreflex delay / volume loss	↓ MAP on standing ≥20 mm Hg SBP	Tilt-table, compression hosiery, gradual positional changes



Disorder	Primary Lesion / Change	Hemodynamic Consequence	Physiotherapy Considerations
Shock (septic, hypovolemic, cardiogenic)	Profound ↓ effective arterial blood volume or contractility	MAP <65 mm Hg; organ hypoperfusion	PT limited to positioning & gentle limb movement until hemodynamics stabilise

4 Interactive Example—Why Radius Rules

Scenario: Femoral artery narrowed 50 % by plaque (r from 4 mm → 2 mm). $\text{Relative Flow} = \left(\frac{2}{4}\right)^4 = \frac{1}{16}$ $\text{Relative Flow} = \left(\frac{4}{2}\right)^4 = 16$

→ **94 % drop** in maximal flow, explaining rapid leg fatigue.

Therapy: Interval walking promotes collateral dilation (radius ↑), partially restoring Q.

5 Blood-Pressure Regulation Recap (Applied)

- **Exercise Pressor Response:** ↑ HR & SV, local arteriole dilation in active muscle, systemic SNS constriction elsewhere → MAP rises modestly.
- **Valsalva:** ↑ Intrathoracic P → ↓ venous return → Phase II drop in SV → baroreflex tachycardia—avoid in aneurysm, CHF.
- **Cold Immersion:** Cutaneous vasoconstriction ↑ SVR; watch hypertensive clients in hydrotherapy.

6 Self-Check Quiz (answers below)

1. Calculate the percentage change in resistance if arteriole radius decreases 30 %.
2. Which phase of the Valsalva manoeuvre risks syncope and why?
3. Name two endothelial factors: one vasodilator and one vasoconstrictor.
4. Explain how chronic aerobic training affects pulse pressure.
5. Why does an aortic stenosis patient often have a slow rising (anacrotic) pulse?

Answers:

1. $R_{\text{new}}/R_{\text{old}} = (1/0.7)^4 \approx 4.16$ $R_{\text{new}}/R_{\text{old}} = (1/0.7)^4 \approx 4.16$ → **Resistance ↑ 316 %**.
2. **Phase IV (release):** sudden ↓ intrathoracic P → venous surge, reflex bradycardia → transient cerebral hypoperfusion.
3. **NO (nitric oxide)** dilates; **Endothelin-1** constricts.
4. Arterial compliance ↑, so **pulse pressure narrows** (SBP less steep, DBP slightly higher).
5. Fixed narrow valve delays systolic ejection → prolonged upstroke and reduced amplitude of arterial pulse.

7 Key Take-Home Points

- **Radius is king:** small changes create huge shifts in flow and pressure.
- **Disorders alter hemodynamics through radius, compliance or pump failure**—identify the primary defect to tailor interventions.
- Physiotherapists must adjust **intensity, posture, temperature and compression** to work with, not against, each patient's cardiovascular limitations.