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.

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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	**S_1** ("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	**S_2** ("dub")
Passive filling (rapid + diastasis)	AV valves open; ventricles fill 80 %	AV open	Vent P low; atria refill	Possible S_3 (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.

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3 Cardiac Output (CO)

CO=Heart Rate (HR)×Stroke Volume (SV)\boxed{\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 HRmax (≈ 220 − age)	β-blockers blunt HR—use RPE 11-13
SV	EDV (preload), contractility, afterload	↑ 40-60 % VO_2max 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 VO_2 → CO): CO=VO_2a-v O_2 diff\text{CO} = \frac{\text{VO_2}}{\text{a-v O_2 diff}}

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4 Wiggers Diagram Snapshot

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

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LV Pressure:      /\        /
Aortic Pressure:  / \_____/
LA Pressure:    __/  \____
Heart Sounds:   S1  S2
ECG:       P    QRS  T
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Understand timing to position stethoscope and interpret murmurs (e.g., systolic ejection in aortic stenosis between S_1-S_2).

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 > HRmax
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

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

7 Self-Check Quiz (answers below)

1. **Which cardiac phase follows closure of the semilunar valves?**
2. **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 × 0.08 L = **7.2 L min^{-1}**.
3. **PR interval** (> 0.20 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.

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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.

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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 (ΔV/ΔP)	**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

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Poiseuille's Law R=8ηLπr4R = \frac{8ηL}{πr^{4}}R=πr48ηL

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

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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) Jv=Kf[(Pc−Pi)−σ(πc−πi)]J_v = K_f[(P_c-P_i) – σ(π_c-π_i)]Jv=Kf[(Pc−Pi)−σ(πc−πi)]

Symbol	Meaning
PcP_cPc	Capillary hydrostatic pressure (outward)
πcπ_cπc	Capillary oncotic pressure (inward, albumin)
KfK_fKf	Filtration coefficient (permeability × surface)

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

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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
Vena cava	—	2–5	rises again
Pulmonary artery	25 / 8	15	low-pressure circuit

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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_2O retention
ADH (vasopressin)	↑ Osmolality or ↓ BP	V_2 receptors ↑ H_2O 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).

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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

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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 ≈ DBP + ⅓(SBP − DBP) → 80 + (50/3) ≈ **97 mm Hg**.

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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

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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.

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2 Blood-Flow Dynamics—Core Principles

Law / Concept	Key Equation	Practical Meaning
Poiseuille's Law (laminar flow)	Q=ΔP·π·r48ηLQ = \dfrac{ΔP·π·r^{4}}{8ηL}Q=8ηLΔP·π·r4	Radius (r) is the "volume knob"—a 16 % ↑r doubles flow.
Resistance	R=8ηLπr4R = \dfrac{8ηL}{πr^{4}}R=πr48ηL	Arteriolar tone sets systemic vascular resistance (SVR).
Flow Velocity	v=QAv = \dfrac{Q}{A}v=AQ	Capillaries: huge A → very slow v → exchange time.
Reynolds Number	Re=ρ·v·DηRe = \dfrac{ρ·v·D}{η}Re=ηρ·v·D	>2000 → turbulent → murmurs, bruit in stenoses.
Compliance	C=ΔVΔPC = \dfrac{ΔV}{ΔP}C=ΔPΔV	Veins highly compliant; aging arteries lose compliance → ↑ pulse pressure.
Shear Stress	τ=4ηQ/πr3τ = 4ηQ / πr^{3}τ=4ηQ/πr3	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.

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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_2peak; 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

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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). Relative Flow=(24)4=116\text{Relative Flow} = \left(\frac{2}{4}\right)^{4} = \frac{1}{16}Relative Flow=(42)4=161

→ **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. Rnew/Rold=(1/0.7)4≈4.16R_{new}/R_{old} = (1/0.7)^{4} ≈ 4.16Rnew/Rold=(1/0.7)4≈4.16 → **Resistance ↑ 316 %**.
2. **Phase IV (release):** sudden ↓ intrathoracic P → venous surge, reflex bradycardia → transient cerebral hypo-perfusion.
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.