Chapter 3. Musculoskeletal Physiology

Part 1 | Muscle Physiology

1 Learning Objectives

After completing this part you will be able to …

1. **Describe the sliding-filament mechanism** from electrical excitation to cross-bridge cycling and relaxation.
2. **Distinguish Type I, Type IIa and Type IIx muscle fibres** in terms of structure, metabolism and functional output.
3. **Predict how training, ageing and pathology shift fibre-type distribution** and adapt physiotherapy programmes accordingly.
4. **Link molecular events (Ca^{2+} release, ATP hydrolysis, motor-unit recruitment)** to measurable clinical parameters such as MVC, fatigue index and EMG pattern.

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2 Muscle-Contraction Mechanism (Sliding-Filament Theory)

Step	Cellular Event	Key Molecules	Physiotherapy Angle
1 Neuromuscular transmission	ACh released → binds nicotinic receptors → sarcolemma depolarises (EPP)	ACh, Na^+ channels	NMES uses depolarisation to activate fibres directly
2 Action-potential propagation	AP travels along sarcolemma → down T-tubules	Voltage-gated Na^+/K^+ channels	Myelin loss (MS) slows conduction → earlier fatigue
3 Excitation–contraction coupling (ECC)	T-tubule DHPR triggers RyR on SR → Ca^{2+} flood cytosol	Dihydropyridine & ryanodine receptors, Ca^{2+}	Malignant hyperthermia (faulty RyR) CI for certain modalities (heat)
4 Cross-bridge cycling	a) Ca^{2+} binds troponin-C; tropomyosin shifts b) **Attach:** Energised myosin head binds actin c) **Power-stroke:** ADP + Pi released, myosin pivots pulling actin ~10 nm d) **Detach:** New ATP binds myosin, head detaches e) **Re-cock:** ATP hydrolysed, head re-energised	Actin, myosin, ATP, troponin, tropomyosin	Static holds ↑ time under tension → more cross-bridge cycles → hypertrophy
5 Relaxation	SERCA pumps resequester Ca^{2+} into SR; tropomyosin covers sites	SERCA, ATP	Spasticity meds enhance Ca^{2+} reuptake—combine with stretching
6 Force summation	↑ Firing rate & ↑ motor-unit recruitment (size principle)	Type I → IIa → IIx	Plyometrics demand high MU synchrony; older adults lose IIx first

Energy Cost: 70 % of ATP during cycling (detach/re-cock); 30 % on Ca^{2+} pumps—explains high VO_2 during sustained tetany.

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3 Muscle-Fibre Types

Feature	Type I (Slow-oxidative)	Type IIa (Fast oxidative-glycolytic)	Type IIx (Fast-glycolytic)

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Colour / Myoglobin	Red / high	Pink / moderate	White / low
Mitochondria & Capillary density	High	Medium-high	Low
ATPase isoform	Slow	Fast	Fastest
Contraction speed ($T_{1/2}$)	~100 ms	~50 ms	~25 ms
Fatigue resistance	Excellent	Intermediate	Poor
Primary fuel	β-oxidation (fat)	Fat + glycogen	Glycogen / PCr
Typical location	Postural (soleus, erector spinae)	Quads, deltoid	Gastrocnemius lateral head, biceps brachii
Training adaptation	↑ mitochondrial density, angiogenesis	Converts to IIx with disuse, to I with endurance	Hypertrophies most with high-load power training
PT Relevance	Balance & endurance tasks	Mixed-sport conditioning	Explosive, quick tasks; atrophy early post-immobilisation

Motor-Unit Recruitment – Henneman's Size Principle

Low-threshold (Type I) units fire first; as force demand ↑, larger Type II units are recruited.
Clinical: low-load BFR training recruits Type II at lighter loads → joint-friendly strength gains.

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4 Plasticity & Lifespan Changes

Factor	Fibre-type Shift	Mechanism	Practice Impact
Endurance training	IIx → IIa → I	↑ PGC-1α, mitochondrial biogenesis	Programme longer sets for metabolic health
Resistance / power	I/IIa → IIx hypertrophy (not conversion)	mTOR activation, satellite-cell fusion	Cycle heavy-load blocks for sarcopenia
Ageing (Sarcopenia)	Preferential loss of IIx, MU denervation	Motor-neuron apoptosis	Use explosive concentric cues with safety
Disuse / Bed rest	I → IIx relative ↑ but atrophy overall	Unloading ↓ AMPK, ↓ protein synthesis	Early mobilisation & NMES preserve I fibres

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5 Applied Example – Designing an Exercise Set

1. **Goal:** Improve stair-climb power in 70-y-o COPD patient.
2. **Analysis:** Needs Type IIa recruitment without excessive ventilatory load.
3. **Prescription:**
• 30 % 1-RM sit-to-stands with **blood-flow restriction** (BFR) → earlier IIa activation.
• 2 s down, 1 s up (temporal overload).
• 3 sets × 15 reps, RPE ≤ 13.
4. **Rationale:** Low mechanical & cardiorespiratory stress yet metabolic stimulus; aligns with fibre physiology.

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6 Self-Check Quiz (answers below)

1. **During the power-stroke, which molecules leave the myosin head?**
2. **Which fibre type exhibits the highest peak power output?**
3. **Name the pump responsible for Ca^{2+} resequestration and state its energy source.**
4. **Why does a β-oxidation-dominant fibre resist fatigue?**
5. **List two training methods most effective at converting Type IIx to Type IIa fibres.**

Answers

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1. **ADP and inorganic phosphate (Pi).**
2. **Type IIx**—fast-glycolytic fibres.
3. **SERCA (sarcoplasmic-reticulum Ca^{2+}-ATPase);** it uses **ATP hydrolysis**.
4. Dense mitochondria, ample myoglobin & capillaries sustain aerobic ATP, preventing metabolite accumulation.
5. **Endurance training** (continuous or high-volume intervals) and **high-repetition resistance training** with short rest.

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7 Key Take-Home Points

- **Sliding-filament mechanics** convert chemical energy (ATP) into force; ECC defects produce weakness or spasm.
- **Fibre-type composition dictates speed, power and fatigue behaviour**—vital information for specific, safe exercise prescription.
- **Physiological plasticity** means training, detraining or disease can shift fibre profiles; PTs must reassess and adapt.

Part 2 | Skeletal-System Physiology

1 Learning Objectives

On completing this part you will be able to …

1. **Describe intramembranous and endochondral ossification** and outline the bone-remodelling cycle.
2. **Explain mechanical, hormonal and nutritional regulation** of bone mass and strength, linking them to physiotherapy interventions.
3. **Classify joints into fibrous, cartilaginous and synovial categories,** listing sub-types, structural components and functional roles.
4. **Relate joint physiology—cartilage biomechanics, synovial fluid dynamics and ligament behaviour—to movement, injury and rehabilitation.

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2 Bone Formation

Pathway	Developmental Steps	Representative Bones	Clinical / PT Note
Intramembranous ossification	Mesenchymal cells → osteoblast clusters (ossification centres) → osteoid deposition → trabecular fusion → compact bone formation	Flat bones of skull, mandible, clavicle	Rapid healing; clavicle mid-shaft fractures unite faster
Endochondral ossification	Hyaline cartilage model → periosteal bone collar → primary marrow cavity, vascular invasion → secondary ossification centres in epiphyses → epiphyseal plate growth	Long bones (femur, humerus), vertebrae	Growth-plate injuries threaten limb length; respect in paediatric rehab

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3 Bone-Remodelling Cycle

1. **Activation** – osteoclast precursors recruited (RANKL ↑, OPG ↓)
2. **Resorption** – osteoclasts digest mineral & matrix (≈ 2 weeks)
3. **Reversal** – macrophage-like cells prepare surface
4. **Formation** – osteoblasts lay osteoid, mineralise (≈ 3 months)
5. **Quiescence** – lining cells cover new lamellae

Regulator	Effect on Balance	PT Implication
Mechanical load (Wolff's law)	↑ Strain → ↑ osteoblast activity	Progressive resistance, WBV counteracts osteopenia
PTH (intermittent)	Anabolic (stimulates formation)	Teriparatide patients tolerate higher loading
Estrogen	Inhibits osteoclasts	Post-menopause loss → prescribe impact + strength exercise
Vit D / Ca^{2+}	Mineral supply	Nutrition counselling integral to fracture rehab

Remodelling rate: cortical ≈ 3 % yr^{-1}; trabecular ≈ 20 % yr^{-1}—hence vertebral bodies fracture early in osteoporosis.

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4 Joint Physiology

4.1 Classification & Structure

Class	Sub-type & Example	Connecting Tissue	Mobility
Fibrous	Sutures (skull), Syndesmosis (distal tib-fib)	Dense CT	Synarthrosis (immobile) → slight
Cartilaginous	Synchondrosis (1st rib-sternum), Symphysis (pubic)	Hyaline / fibrocartilage	Amphiarthrosis (slight move)
Synovial (Diarthrosis)	Plane, Hinge, Pivot, Condyloid, Saddle, Ball-socket	Capsule + synovial membrane, cartilage, ligaments, bursae	Freely movable

4.2 Synovial-Joint Components & Function

Component	Composition	Biomechanical Role	Rehab Insight
Articular cartilage	70 % water, type II collagen, proteoglycans	Low-friction, load distribution; viscoelastic creep	Cyclic compression (cycling) nourishes cartilage via fluid flow
Synovial fluid	Hyaluronan, lubricin, plasma filtrate	Viscous lubricant; nutrient medium	Warm-up ↑ viscosity ↓ → smoother motion
Ligaments	Dense reg. CT, crimped collagen	Passive restraint, proprioceptors	Early protected ROM encourages fibre realignment after sprain
Meniscus / Labrum	Fibrocartilage	Deepen socket, shock absorption	Meniscectomy ↓ contact area → emphasise quad-ham co-contraction
Capsule	Fibrous + synovial layers	Encloses, guides movement	Capsular pattern informs mobilisations (ER > Abd > IR in shoulder)

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5 Cartilage & Lubrication Mechanics

- **Boundary lubrication** (lubricin) dominates at low speeds / high loads—important in weight-bearing stance.
- **Fluid film lubrication** (pressurised synovial fluid) during dynamic movement—reason for gentle range exercises post-injury.

Viscoelastic behaviour: **stress-relaxation & creep** make prolonged low-load stretch (LLPS) effective for capsular tightness.

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6 Integrative Example – ACL-Reconstructed Knee

- **Phase 1 (0-4 wks):** Graft avascular; protect with inner-range quads (≤ 60°) → respects ligament viscoelasticity.
- **Phase 2:** Controlled closed-chain loads stimulate ligament mechanoreceptors & osteoligamentous tunnel healing.
- **Phase 3:** Plyometrics harness elastic energy storage of tendon-bone complex—requires full graft incorporation (≈

9 mo).

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7 Self-Check Quiz (answers below)

1. **Which bone cell expresses RANKL and what is its role?**
2. **Give one example of a synchondrosis and state whether it permits movement.**
3. **Why does immobilisation lead to rapid peri-articular osteoporosis?**
4. **Explain how lubricin deficiency might present clinically.**
5. **Name the primary stimulus for conversion of osteoid to mineralised bone.**

Answers

1. **Osteoblasts**; RANKL binds RANK on osteoclast precursors, promoting differentiation and bone resorption.
2. **Epiphyseal growth plate** of developing long bone; it is immobile (synarthrosis).
3. Lack of mechanical strain ↓ osteoblast activity and ↑ osteoclast dominance, accelerating trabecular resorption around joints.
4. Increased friction → early-onset osteoarthritis, joint pain and crepitus on motion.
5. Adequate **local Ca^{2+}/PO_4^{3-} supersaturation** and alkaline pH generated by osteoblast activity.

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8 Key Take-Home Points

- **Bone is a dynamic tissue**; mechanical load, hormones and nutrition steer the resorption–formation balance.
- **Joint health relies on movement-dependent lubrication and nutrient diffusion**—"motion is lotion."
- Physiotherapists leverage these principles through **graded loading, weight-bearing, joint mobilisation and patient education** to optimise skeletal integrity.

Part 3 | Muscular Adaptations to Exercise

1 Learning Objectives

On completing this part you will be able to …

1. **Explain the cellular and systems-level adaptations** that produce muscle hypertrophy, strength gains and endurance improvements.
2. **Contrast acute (within-session) physiological responses** with chronic (training) adaptations for both resistance and aerobic exercise.
3. **Apply the SAID principle** (Specific Adaptation to Imposed Demand) to choose sets, reps, intensity and rest that match patient goals.
4. **Integrate knowledge of neuromuscular, metabolic and hormonal changes** into safe, progressive physiotherapy programmes.

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2 Muscle Hypertrophy vs. Endurance – Mechanistic Snapshot

Feature	Hypertrophy / Strength (≥ 65 % 1-RM)	Endurance / Fatigue-Resistance (40–60 % VO_2max)
Primary stimulus	High mechanical tension, micro-trauma	Sustained metabolic stress, mitochondrial demand
Early gains (0–4 wk)	↑ Neural drive, MU synchrony, ↓ antagonist co-activation	↑ Capillary recruitment, improved O_2 extraction

Feature	Hypertrophy / Strength (≥ 65 % 1-RM)	Endurance / Fatigue-Resistance (40–60 % VO_2max)
Chronic gains (≥ 6 wk)	**Myofibrillar hypertrophy**: • mTOR → protein synthesis • Satellite-cell fusion • Type IIx → IIa CSA ↑	**Mitochondrial biogenesis**: • PGC-1α ↑ • Type IIx → IIa quality shift • Myoglobin ↑ • Capillarisation ↑
Hormonal milieu	Acute ↑ GH, testosterone, IGF-1, mechano-growth factor	Modest catecholamine & cortisol rise; chronic ↑ insulin sensitivity
Structural change	Pennation angle ↑, tendon CSA ↑, connective-tissue stiffness ↑	Mitochondria volume ↑ 40 – 100 %, glycogen stores ↑, oxidative enzymes ↑
Functional outcome	10–30 % strength gain per 8 wk (novice); RFD ↑	↑ VO_2max, ↑ lactate threshold, ↓ HR sub-max, fatigue time ↑

3 Resistance-Exercise Responses & Adaptations

3.1 Acute Session

- **Neural:** High-frequency MU firing, synchrony, H-reflex amplitude ↑.
- **Endocrine:** GH & testosterone peak 15 min post-set (compound lifts, short rest).
- **Metabolic:** ATP-PCr depletion, lactate ↑, pH ↓), cell swelling.

3.2 Chronic Training (≥ 8 wk)

- **Myofibrillar protein synthesis** exceeds breakdown (net accretion).
- **Satellite-cell activation** doubles myonuclei pool, expanding transcriptional capacity.
- **Connective-tissue reinforcement** – collagen cross-links align with fibre tension axis.
- **Neural plasticity** – corticospinal excitability ↑; motor-cortex map enlarges.

Practical cue: Early (first 2–3 wk) strength jump is neural—teach technique before adding load.

4 Aerobic-Exercise Responses & Adaptations

4.1 Acute Bout

- **Cardio-respiratory:** HR, SV, $\dot{Q}$ ↑; redistribution of blood flow to Type I fibres.
- **Metabolic:** Rapid ↑ in mitochondrial ATP turnover; catecholamine-driven lipolysis.

4.2 Chronic Endurance Training

- **Mitochondrial biogenesis** via PGC-1α/Nrf1 signalling → citrate synthase, cytochrome-c oxidase ↑.
- **Angiogenesis:** VEGF-mediated capillary-to-fibre ratio climbs from ~1.5 to > 2.0.
- **Substrate shift:** ↑ Fat oxidation, glycogen sparing; lactate threshold moves from 55 % to 75 % VO_2max.
- **Autonomic change:** Resting HR ↓, vagal tone ↑—important when using HR zones.

Clinical pearl: Four weeks of HIIT (4 × 4 min @ 90 % HRmax) can raise VO_2max ~10 % in cardiac-rehab patients when tolerated.

5 Programming Parameters – Translating Physiology to Practice

Goal	Intensity	Volume	Rest	Frequency	Example
Max strength	80–90 % 1-RM	3–6 sets × 3–5 reps	2–3 min	2–3×/wk	Dead-lift, leg-press
Hypertrophy	65–80 % 1-RM	3–5 sets × 6–12 reps	60–90 s	2–4×/wk	Squat + accessory
Power	30–60 % 1-RM high-velocity	3–5 sets × 3–6 reps	2 min	2×/wk	Medicine-ball toss
Endurance	50–70 % VO_2max	30–60 min	continuous	4–5×/wk	Treadmill jog
HIIT	85–95 % HRmax	4–8 × 30 s–4 min	equal rest	2–3×/wk	Cycle 4 × 4 protocol

Elderly or post-op patients may use **blood-flow-restriction (BFR)** at 20–30 % 1-RM to evoke hypertrophy with low joint stress.

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6 Special Considerations

Condition	Adaptation Issue	PT Strategy
Sarcopenia	↓ Type IIx fibres, anabolic resistance	High-velocity resistance; protein 25–30 g/meal
Tendinopathy	Collagen turnover lagging	Eccentric-heavy slow-resistance 3×/wk; 12-week block
Chronic HF	Limited $\dot{Q}$ reserve	Interval walking 1:1 work-rest, Borg RPE 11–13
Diabetes	Impaired GLUT-4 translocation	Combine aerobic + resistance same session; foot care

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7 Self-Check Quiz (answers below)

1. **Which signalling pathway (mTOR or AMPK) predominates after a 10-RM squat set?**
2. **State two cellular markers of mitochondrial biogenesis following endurance training.**
3. **Why is lactate not a waste product in muscle metabolism?**
4. **Give the approximate time-frame when neural adaptations plateau and hypertrophy predominates in a novice lifter.**
5. **Explain why fast-eccentric loading elicits more DOMS than concentric loading.**

<details> <summary>Answers</summary>

1. **mTOR** dominates, driving protein synthesis.
2. ↑ **PGC-1α** expression and ↑ **citrate-synthase activity** (or cytochrome-c oxidase).
3. Lactate is an **energy shuttle**—it is oxidised by heart and Type I fibres or recycled to glucose in liver (Cori cycle).
4. Around **3–4 weeks** of consistent training.
5. Eccentric contractions cause **greater sarcomere strain & micro-damage**, activating inflammatory and repair cascades leading to soreness.

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8 Key Take-Home Points

- **Hypertrophy ≠ strength ≠ endurance**—each arises from distinct molecular triggers; match load, velocity and metabolic stress to the desired outcome.
- Early gains are **neural**, later gains **structural**. Periodise accordingly.
- **Endurance adaptations** hinge on mitochondrial & vascular expansion, improving fatigue resistance and metabolic health.
- Physiotherapists leverage these principles to **restore function, prevent injury and optimise performance** across the lifespan.

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