

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 (Ca²⁺ release, ATP hydrolysis, motor-unit recruitment) to measurable clinical parameters such as MVC, fatigue index and EMG pattern.

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 \rightarrow 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 llx first

Energy Cost: 70 % of ATP during cycling (detach/re-cock); 30 % on Ca²⁺ pumps—explains high VO₂ during sustained tetany.

3 Muscle-Fibre Types

Feature	Type I (Slow-oxidative)	type ita (Fast oxidative-	Type IIx (Fast-glycolytic)
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Feature	Type I (Slow-oxidative)	Type IIa (Fast oxidative- glycolytic)	Type IIx (Fast-glycolytic)
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 1, larger Type II units are recruited. **Clinical:** low-load BFR training recruits Type II at lighter loads → joint-friendly strength gains.

4 Plasticity & Lifespan Changes

Factor	Fibre-type Shift	Mechanism	Practice Impact
Endurance training	$IIx \rightarrow IIa \rightarrow I$	↑ PGC-1α, mitochondrial biogenesis	Programme longer sets for metabolic health
Resistance / power	I/lla → 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	$I \rightarrow IIx$ relative \uparrow but atrophy overall	Unloading ↓ AMPK, ↓ protein synthesis	Early mobilisation & NMES preserve I fibres

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 \times 15 reps, RPE \leq 13.
- 4. Rationale: Low mechanical & cardiorespiratory stress yet metabolic stimulus; aligns with fibre physiology.

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 Ca²⁺ 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 Ca2+-ATPase); it uses ATP hydrolysis.
- 4. Dense mitochondria, ample myoglobin & capillaries sustain aerobic ATP, preventing metabolite accumulation.
- Endurance training (continuous or high-volume intervals) and high-repetition resistance training with short rest.

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.

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 \rightarrow periosteal bone collar \rightarrow primary marrow cavity, vascular invasion \rightarrow secondary ossification centres in epiphyses \rightarrow epiphyseal plate growth	Long bones (femur, humerus), vertebrae	Growth-plate injuries threaten limb length; respect in paediatric rehab

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

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Regulator **Effect on Balance** PT Implication Mechanical load (Wolff's ↑ Strain → ↑ osteoblast activity Progressive resistance, WBV counteracts osteopenia law)

PTH (intermittent) Anabolic (stimulates formation) Teriparatide patients tolerate higher loading

Post-menopause loss → prescribe impact + strength

Inhibits osteoclasts exercise

Vit D / Ca²⁺ Nutrition counselling integral to fracture rehab Mineral supply

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

4 Joint Physiology

Estrogen

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 \uparrow viscosity \downarrow \rightarrow 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)

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.

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 (≈

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9 mo).

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 Ca²⁺/PO₄³⁻ supersaturation and alkaline pH generated by osteoblast activity.

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.
- Integrate knowledge of neuromuscular, metabolic and hormonal changes into safe, progressive physiotherapy programmes.

2 Muscle Hypertrophy vs. Endurance - Mechanistic Snapshot

Feature	Hypertrophy / Strength (≥ 65 % 1-RM)	Endurance / Fatigue-Resistance (40-60 % VO2max)
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 O₂ extraction

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Feature	Hypertrophy / Strength (≥ 65 % 1-RM)	Endurance / Fatigue-Resistance (40-60 % VO₂max)
Chronic gains (≥ 6 wk)	Myofibrillar hypertrophy: • mTOR → protein synthesis • Satellite-cell fusion • Type IIx → IIa CSA ↑	Mitochondrial biogenesis : • PGC- 1α ↑ • Type IIx \rightarrow IIa quality shift • Myoglobin ↑ • Capillarisation ↑
Hormonal milieu	Acute ↑ GH, testosterone, IGF-1, mechanogrowth factor	Modest catecholamine & cortisol rise; chronic ↑ insulin sensitivity
Structural change	Pennation angle 1, tendon CSA 1, connective-tissue stiffness 1	Mitochondria volume ↑ 40 – 100 %, glycogen stores ↑, oxidative enzymes ↑
Functional outcome	10-30 % strength gain per 8 wk (novice); RFD ↑	↑ VO₂max, ↑ 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 1.
- 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 1; 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, 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-1a/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 % VO₂max.
- **Autonomic change:** Resting HR ↓, vagal tone ↑—important when using HR zones.

Clinical pearl: Four weeks of HIIT (4 \times 4 min @ 90 % HRmax) can raise VO₂max ~10 % in cardiac-rehab patients when tolerated.

5 Programming Parameters - Translating Physiology to Practice

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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	$3-5 \text{ sets} \times 6-12 \text{ reps}$	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 % VO₂max	30-60 min	continuous	4-5×/wk	Treadmill jog
HIIT	85-95 % HRmax	$4-8 \times 30 \text{ s-4 min}$	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.

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 Q reserve	Interval walking 1:1 work-rest, Borg RPE 11-13
Diabetes	Impaired GLUT-4 translocation	Combine aerobic + resistance same session; foot care

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-1**α 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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