

RUHANI COLLEGE · REGISTERED MASSAGE THERAPY DIPLOMA PROGRAM

Final Examination — Student Study Guide*Days 1 to 6 · The whole course, from the foundations of pathology to clinical integration*

Course	RU RMT 10 Pathology 1: Skin, Connective Tissue and Skeletal Pathologies
Assessment	Final Examination — 60 multiple-choice items, 2 hours, closed book, 50% of the final grade
Administered	Day 6, in the final two hours
Coverage	The whole course, Days 1 to 6
Structure	Six sections, one per instructional day: A Foundations and the Integument (8 marks); B Skin Pathology (10); C Connective Tissue Pathology I (10); D Connective Tissue Pathology II (10); E Skeletal Pathology I (11); F Skeletal Pathology II and Clinical Integration (11)
This guide	21 topics, 79 review questions, and a full answer key with discussion

How to use this guide

1. Sections A, B and C are written as revision. That material was examined on Quiz 1, so this guide summarises it and tests it rather than teaching it again from the beginning. If any of it is shaky, go back to the Quiz 1 Study Guide, which covers the same ground in full.
2. Sections D, E and F are new material that has not been examined before. They are written at full depth and they carry 32 of the 60 marks. Give them the majority of your revision time.
3. Work through the topic explanation, then close the guide and answer the review questions in writing from memory. Check the answer key at the back afterwards, not alongside.
4. Pay particular attention to the topics marked as recurring across days. The examination is written to reward candidates who can connect material — the healing timelines, the inflammatory versus degenerative distinction, and the contraindication classes all appear in more than one section.
5. Section F carries the clinical integration content, which is where all seven course outcomes converge. Those items give you a presentation and ask what it indicates or what you would do. They cannot be answered by recall alone.
6. This guide contains no examination questions and no question in it appears on the paper. It teaches the material the paper is drawn from.
7. Use the final revision checklist the evening before. Anything you cannot explain aloud without notes is not yet learned.

What this guide covers

This guide covers all six instructional days. Sections A to C revise the foundations of pathology and terminology, infection prevention and control, the integumentary system and its pathologies, and connective tissue pathology I. Sections D to F cover the material examined here for the first time: soft-tissue injury, overuse and the stages of healing; bone tissue, fracture, metabolic bone disease and postural deviation; and joint disease, degenerative spinal conditions, bone infection and neoplasm, together with the clinical reasoning framework that draws the whole course into a single treatment decision.

Section A — Foundations of Pathology and the Integument — revision

Eight marks. Examined previously on Quiz 1, so this section is written as consolidation. Work the questions; if the explanations feel thin, return to the Quiz 1 Study Guide for the full treatment.

A1. The disease process, terminology and cellular response

Disease unfolds in a fixed sequence: etiology, the cause; pathogenesis, the mechanism; manifestation, the signs and symptoms; and resolution, the outcome. The order is examinable and reversing pathogenesis and manifestation is the usual error.

The vocabulary of origin: idiopathic means of unknown cause, iatrogenic means caused by medical treatment, nosocomial means acquired in a health care facility, congenital means present from birth, and hereditary means genetically transmitted. The vocabulary of outcome: a prognosis is the predicted course, a sequela is a condition remaining after the original disease has resolved, and a complication arises during the course of the disease.

A sign is objective and observable by you; a symptom is subjective and reported by the client. Cellular adaptations: hypertrophy is an increase in cell size, hyperplasia an increase in cell number, atrophy a decrease in either, metaplasia the replacement of one mature cell type by another, and dysplasia disordered growth that may be pre-malignant.

Suffixes: "-itis" inflammation, "-osis" abnormal or degenerative condition, "-oma" tumour or swelling, "-pathy" disease of, "-penia" deficiency, "-malacia" softening. This last pair returns in Section E, where osteopenia and osteomalacia are examined directly and are easily confused with each other and with osteoporosis.

Review questions

1. Give the four stages of the disease process in order.
2. Distinguish hypertrophy, hyperplasia and atrophy.
3. Decode "osteopenia", "osteomalacia" and "osteoporosis", and state how they differ.

A2. Infection, infection control and inflammation

Five classes of pathogen: bacteria, viruses, fungi, parasites and prions. The chain of infection has six links — infectious agent, reservoir, portal of exit, mode of transmission, portal of entry, susceptible host — and breaking any one prevents transmission.

Standard precautions are applied to every client regardless of disclosure, because infectious status is frequently unknown and a person may be infectious before any symptom appears. Hand hygiene is the single most effective preventive measure. Cleaning removes soil, disinfection destroys most organisms, and sterilisation destroys all including spores; treatment tables are cleaned and disinfected, never sterilised.

Following a sharps or body fluid exposure, first aid comes before paperwork: wash with soap and running water allowing free bleeding, irrigate any mucous membrane exposure, seek urgent medical assessment because prophylaxis is time-critical, then report and document.

The five cardinal signs of inflammation are redness, heat, swelling, pain and loss of function. Contraindications are classified as absolute, local or conditional; the conditional class is much the largest in practice, and this classification is used in every section of the examination.

Review questions

4. Why do standard precautions apply to every client rather than only to those who disclose an infection?
5. State the correct order of actions following a sharps injury in the clinic.
6. Define the three classes of contraindication and explain why the conditional class matters most in practice.

A3. Skin structure, function and lesion terminology

Epidermal layers from deep to superficial: stratum basale, spinosum, granulosum, lucidum (thick skin only) and corneum. Mitosis occurs in the basale; the corneum is the barrier. The epidermis is avascular and contains no appendages.

The dermis contains the blood vessels, sensory receptors, hair follicles and glands. Because the appendages extend into it, a partial-thickness wound can re-epithelialise from within; a full-thickness wound cannot, and heals by scarring from the margins.

Six functions: protection, thermoregulation, sensation, excretion, vitamin D synthesis, and prevention of water loss. The vitamin D function connects directly to osteomalacia in Section E.

Ageing skin thins, loses elasticity, loses vascularity, produces less sebum and sweat, loses subcutaneous fat, turns over more slowly, senses less reliably and defends less effectively. Every change points in the direction of more caution, and examination distractors are usually built by reversing one of them.

Primary lesions arise from the disease process — macule, patch, papule, plaque, nodule, vesicle, bulla, pustule, wheal, cyst. Secondary lesions result from change to a primary lesion — scale, crust, erosion, ulcer, fissure, excoriation, lichenification, atrophy, scar. Describe what you find; never name a condition in a record.

Review questions

7. Where does mitosis occur in the epidermis, and how long does a keratinocyte take to reach the surface?
8. Why can a partial-thickness burn re-epithelialise from within the wound bed while a full-thickness burn cannot?
9. Classify as primary or secondary: wheal, fissure, nodule, lichenification, bulla, excoriation.

Section B — Skin Pathology — revision

Ten marks. Also examined on Quiz 1. Consolidation and testing rather than first teaching.

B1. Infections of the skin

Bacterial: impetigo produces honey-coloured crusts and is highly contagious; folliculitis is follicular pustules; a furuncle is a deeper follicular abscess; cellulitis is a spreading infection of dermis and subcutaneous tissue with a poorly defined advancing border, often with fever, and requires same-day referral.

Viral: warts are caused by human papillomavirus; herpes simplex produces grouped vesicles and remains latent in sensory ganglia; herpes zoster is reactivation of latent varicella-zoster virus from the dorsal root ganglia, producing a painful unilateral dermatomal eruption that stops at the midline, infectious to those without varicella immunity; molluscum contagiosum produces umbilicated papules.

Fungal: the tinea group are dermatophyte infections named by body region — corporis, pedis, cruris, capitis, unguium. Contagious by contact and by fomites. Candidiasis affects warm moist skin folds.

Parasitic: scabies produces intense itching worse at night with burrows in finger webs and flexor wrists, is highly contagious, and is an absolute contraindication until treated.

Antibiotics act only on bacteria. They have no effect on warts, cold sores, shingles or tinea.

Review questions

10. What features distinguish cellulitis, and what action does it require?
11. Explain why the eruption of herpes zoster stops at the midline.
12. Why does a client on methotrexate or long-term corticosteroids warrant a lower threshold for referral of a skin infection?

B2. Inflammatory conditions, wounds, burns and scars

Atopic dermatitis favours flexural surfaces with itching, weeping and lichenification, associated with asthma and hay fever. Contact dermatitis follows the distribution of the contact and is an occupational condition for massage therapists. Psoriasis favours extensor surfaces with well-demarcated plaques and silvery scale, is immune-mediated, is not contagious, and is not a contraindication over intact plaques.

Anaphylaxis is indicated by facial or lip swelling, difficulty breathing, wheeze, throat tightness or a sense of impending doom. Call emergency services, assist with the client's own auto-injector, lie them flat with legs raised unless breathing is easier upright, and do not leave them.

Wound healing proceeds through haemostasis, inflammation, proliferation and maturation. Note that this is a four-phase sequence for wounds specifically, distinct from the three stages of soft-tissue healing in Section D and the four stages of fracture healing in Section E. Confusing the three is one of the most reliable ways to lose marks on this paper.

A hypertrophic scar stays within the original wound margins; a keloid extends beyond them. Burns: superficial involves epidermis only and does not blister; partial thickness blisters and is severely painful; full thickness destroys the dermis and is characteristically painless in the burned area because the nerve endings are gone.

Neoplasms: basal cell carcinoma is the most common and least aggressive, a pearly papule with rolled borders that rarely metastasises. Squamous cell carcinoma is rough and scaly and may metastasise late. Melanoma is least common and most dangerous, metastasising early. The ABCDE rule is Asymmetry, Border, Colour, Diameter and Evolving, with evolution the most significant.

Review questions

13. Name the three healing sequences taught in this course, with the number of stages in each, and state which body of material each belongs to.
14. What single feature distinguishes a hypertrophic scar from a keloid, and why is that clinically useful?
15. Why is a full-thickness burn painless in the burned area, and what is the danger in that?
16. A client asks whether their psoriasis means you cannot treat them. What is your answer, and why?

Section C — Connective Tissue Pathology I — revision

Ten marks. Examined on Quiz 1. Consolidation, with particular attention to rheumatoid arthritis, which returns in Section F for direct comparison with osteoarthritis.

C1. Connective tissue and autoimmunity

Collagen provides tensile strength, elastin provides recoil, ground substance permits diffusion and lubricates fibre movement, and fibroblasts produce and maintain the matrix. Creep is gradual deformation under sustained low load, which is why connective tissue responds to sustained moderate pressure rather than brief force.

Autoimmune disease results from loss of self-tolerance. Shared features: a relapsing and remitting course, systemic involvement, chronic inflammation, constitutional symptoms that are often more disabling than the local ones, a female predominance in most, onset in early to middle adulthood, and treatment with immunosuppressive medication.

The relapsing and remitting course is the single most important feature for a therapist, because the appropriate treatment is determined by what the disease is doing today, not by its name.

The medication history is frequently more informative than the diagnosis. Long-term corticosteroid use predicts fragile skin, easy bruising, reduced bone density, muscle weakness, delayed healing and immunosuppression, whether or not any of those has been formally diagnosed.

Review questions

17. Why does the relapsing and remitting course of autoimmune disease matter more to treatment planning than the diagnosis?
18. What does a history of several years of oral corticosteroids tell you before you have examined anything?

C2. The systemic autoimmune conditions

Lupus is a multi-system disease affecting skin, joints, kidneys, blood and serous membranes, with a photosensitive malar rash sparing the nasolabial folds and a symmetrical non-erosive arthritis.

Systemic sclerosis produces excessive collagen deposition with skin thickening and hardening. The limited form is CREST: Calcinosis, Raynaud phenomenon, Esophageal dysmotility, Sclerodactyly, Telangiectasia.

Raynaud phenomenon is episodic digital artery vasospasm with triphasic colour change — pallor, cyanosis, rubor — triggered by cold or stress. Cold applications and contrast therapy are contraindicated; keep the room and the whole client warm.

Rheumatoid arthritis targets the synovial membrane; the pannus that forms erodes cartilage and bone secondarily.

Symmetrical polyarthritis of the small joints, morning stiffness over an hour, warm boggy swelling, and constitutional symptoms. It involves the metacarpophalangeal and proximal interphalangeal joints and spares the distal interphalangeal joints, which is the reverse of osteoarthritis.

Sjögren syndrome damages the exocrine glands, producing dry eyes and dry mouth. Heritable disorders: Marfan syndrome affects fibrillin with a risk of aortic dissection; osteogenesis imperfecta affects type I collagen with a risk of fracture; Ehlers-Danlos syndrome affects collagen with hypermobility and, in the vascular type, arterial rupture.

Fibromyalgia is centrally mediated. Central sensitisation amplifies sensory processing, producing allodynia and hyperalgesia. Begin conservatively, progress by the response over the following days, and do not apply tissue-directed deep work to a problem that is not in the tissue.

Review questions

19. What tissue is the primary target in rheumatoid arthritis, and what follows from that?
20. Give the hydrotherapy implications of Raynaud phenomenon.
21. Define allodynia and hyperalgesia and state the treatment consequence.
22. Why is stretching usually the wrong approach for a hypermobile client who reports feeling tight?

Section D — Connective Tissue Pathology II — new material

Ten marks, examined here for the first time. Soft-tissue injury, overuse conditions, and the stages of healing that govern technique selection. The stages of healing material is the backbone of this section and reappears in Section F.

D1. The three stages of tissue healing

Soft tissue heals in three overlapping stages. Knowing which stage a client is in matters more than knowing the name of their injury, because the stage determines what you may safely do.

Acute (inflammatory)	Roughly the first three to five days. Vasodilation, increased permeability, oedema, heat, redness and pain. The tissue is fragile and actively inflamed. Goals: control oedema and pain, protect the repair.
Sub-acute (proliferative)	From roughly day five to around three weeks. Fibroblasts lay down new collagen, capillaries grow in, granulation tissue forms. The new collagen is disorganised and weak. Goals: support organised repair, restore pain-free range, prevent excessive adhesion.
Chronic (remodelling)	From around three weeks onward, continuing for months. Collagen is remodelled, realigned along lines of stress, and strengthened. Goals: remodel scar, restore full range and strength, return to function.

Technique by stage

Acute	Work proximal to the injury. Lymphatic drainage. Pain-free movement within tolerance. Cold. No frictions, no deep work, no stretching into pain, no heat.
Sub-acute	Gentle work approaching the site as tolerance allows. Careful transverse work. Pain-free active and passive range. Contrast therapy may be introduced. Progressive loading begins.
Chronic	Deeper work, cross-fibre frictions, myofascial release, full-range work and stretch. Heat before treatment. Progressive strengthening.

Two errors are common and both are examinable. Applying acute-stage management to a chronic problem, which leaves the tissue unremodelled and the client no better after months; and applying chronic-stage technique to an acute injury, which increases bleeding and swelling and prolongs recovery.

Factors delaying healing: age, poor nutrition, diabetes, poor circulation, infection, smoking, corticosteroids, repeated mechanical stress on the healing tissue, and inadequate protection early on.

Review questions

- Name the three stages of soft-tissue healing in order, with an approximate timeframe and the principal goal of each.
- A client presents four months after a gradual-onset elbow problem. What stage are they in, and what does that permit?
- Why is heat contraindicated in the acute stage?
- List the factors that delay soft-tissue healing.

D2. Strain, sprain and contusion

The terms describe which tissue is injured, not how badly.

Strain	Injury to muscle or tendon, usually from a sudden forceful contraction or from being loaded while lengthening. Most often at the musculotendinous junction.
Sprain	Injury to a ligament, from a force taking the joint beyond its normal range.
Contusion	A crush injury to tissue from direct blunt force, with bleeding into the tissue.

Grading, applied to both strain and sprain

Grade 1	Microscopic tearing. Mild pain, minimal swelling, strength and function essentially retained.
Grade 2	Partial tearing. Moderate pain, swelling and bruising, definite loss of strength or of joint stability, functional impairment.
Grade 3	Complete rupture. Marked swelling, possible palpable defect, major loss of function. In a grade 3 sprain the joint is unstable, and pain may paradoxically be less than grade 2 because the structure is no longer under tension. Refer.

Ligament healing

Ligament is dense regular connective tissue with a poor blood supply. It heals slowly, heals with scar, and never fully regains its pre-injury properties. Critically, the proprioceptive receptors within the ligament are damaged too, so joint position sense remains impaired long after pain has resolved. This is why re-injury rates after ankle sprain are so high, and why proprioceptive retraining rather than manual therapy is the decisive element of rehabilitation.

Contusion and myositis ossificans

A deep contusion produces bleeding within the muscle, marked loss of range early, and bruising that tracks distally under gravity. The important complication is myositis ossificans — the formation of bone within the muscle following significant contusion or repeated trauma, classically in the quadriceps or brachialis. Aggressive massage or forced stretching of an acute deep contusion is a recognised contributing factor, which is exactly why deep work early after a contusion is contraindicated.

Delayed onset muscle soreness

Not an injury. Diffuse, usually bilateral soreness appearing twelve to twenty-four hours after unaccustomed activity, peaking at twenty-four to seventy-two hours and resolving within a week. Follows eccentric loading. There is no single point of injury and no palpable defect. Distinguishing it from a strain is examinable: a strain has a definite moment of onset, focal tenderness and pain on resisted contraction.

Review questions

27. State the essential difference between a strain and a sprain.
28. Describe the three grades of strain and sprain, and explain the paradox of pain in a grade 3 sprain.
29. Why are re-injury rates after ankle sprain so high?
30. What is myositis ossificans, and why does it make early deep work on a contusion contraindicated?
31. How would you distinguish delayed onset muscle soreness from a grade 1 muscle strain?

D3. Tendinopathy and overuse conditions

The terminology has changed and the change carries real clinical weight. "Tendinitis" implies active inflammation. Research has shown that chronic tendon problems typically show little inflammation and instead show disorganised collagen, increased ground substance and neovascularisation — a failed healing response. "Tendinosis" describes that degenerative state. "Tendinopathy" is now preferred as an umbrella term, because it makes no claim about which process is present.

This is not pedantry. If the problem is inflammation, rest and anti-inflammatory measures make sense. If the problem is a failed repair, rest allows the tissue to weaken further, and the treatment is progressive loading. The change in terminology reflects a complete reversal in management.

Presentation

- Gradual onset over weeks to months, without a single injuring event.
- Pain localised to the tendon, often at its bony attachment.
- Characteristically eased by warming up and worse afterwards, and worst the morning after loading.
- Pain on palpation of the tendon and on resisted contraction of the muscle.
- Possible thickening of the tendon, and possible crepitus.

Common sites

Lateral and medial elbow at the common extensor and flexor origins, the rotator cuff and long head of biceps, the gluteal tendons at the greater trochanter, patellar tendon, Achilles tendon, and tibialis posterior.

Bursitis and friction syndromes

A bursa is a synovial sac reducing friction where a tendon or muscle passes over bone. Bursitis presents with localised swelling, warmth and tenderness directly over the bursa, with pain on the movement that compresses it. The discriminator from tendinopathy is that bursitis hurts on passive movement compressing the bursa, whereas tendinopathy hurts on resisted contraction. Septic bursitis — hot, red, exquisitely tender, often febrile — requires same-day referral.

Iliotibial band syndrome produces lateral knee pain from compression of tissue beneath the distal band. De Quervain tenosynovitis affects the abductor pollicis longus and extensor pollicis brevis at the radial styloid. Trigger finger is nodular thickening of a flexor tendon catching at the A1 pulley.

Compartment syndrome

Acute compartment syndrome is rising pressure within a fascial compartment compromising perfusion. It presents with pain out of proportion to the injury, a tense compartment, pain on passive stretch of the enclosed muscles, and paraesthesia. It is a surgical emergency requiring fasciotomy. Massage is absolutely contraindicated, and the limb must not be elevated above heart level, because that further reduces perfusion pressure.

Chronic exertional compartment syndrome produces predictable pain at a predictable point in exercise, which resolves within an hour of stopping. It is not an emergency, but it should be assessed medically.

Review questions

32. Explain why the term "tendinopathy" is preferred to "tendinitis", and what that changes about management.
33. How would you distinguish bursitis from tendinopathy at the same joint?
34. Describe the presentation of acute compartment syndrome and state why the limb must not be elevated.
35. How does chronic exertional compartment syndrome differ from the acute form?

D4. Assessment: selective tissue tension testing

Selective tissue tension testing distinguishes contractile from non-contractile structures by loading them differentially. This is one of the most useful assessment frameworks in the course and is directly examinable.

8. Active free range of motion — the client moves alone. Tests willingness and ability to move, and screens overall function. It loads both contractile and non-contractile structures, so it localises but does not differentiate.
9. Passive range of motion — you move the client, who stays relaxed. This loads the non-contractile structures: capsule, ligament, bursa, cartilage, fascia. Pain here implicates inert tissue. The end feel is assessed here.
10. Resisted isometric testing — the client contracts against your resistance with no movement occurring. This loads the contractile structures: muscle and tendon, with minimal load on inert tissue. Pain here implicates muscle or tendon.

The logic follows: pain on resisted isometric testing without movement points to contractile tissue; pain on passive movement at end range points to non-contractile tissue; pain on both may indicate either a joint problem with associated muscular guarding, or a lesion involving both.

End feel

The quality of the resistance felt at the end of passive range.

Normal end feels Soft tissue approximation (knee flexion), tissue stretch (ankle dorsiflexion), bone to bone (elbow extension).

Abnormal end feels Empty — the client stops you before any resistance is felt, suggesting serious pathology or acute inflammation. Springy block — a rebound, suggesting an internal derangement such as a meniscal tear. Muscle spasm — a sudden guarded stop. Capsular — a firm restriction earlier than expected, suggesting capsular tightening. Boggy — a soft mushy stop, suggesting effusion.

An empty end feel and a springy block both warrant caution and, in most cases, referral.

Myofascial pain and trigger points

A trigger point is a hyperirritable spot within a taut band of skeletal muscle, tender on compression and capable of producing a characteristic referred pain pattern. Referral means the client's pain may be some distance from the muscle generating it, which is why the site of pain is frequently not the source of the problem.

The corollary runs through the whole course: assess the joint above and the joint below every painful region as a matter of routine, and treat the chain as well as the site if you want the result to last.

Review questions

36. Explain how selective tissue tension testing distinguishes contractile from non-contractile structures.
37. What does an empty end feel indicate, and what should you do?
38. Give three normal and three abnormal end feels.
39. Why should you assess the joint above and the joint below a painful region as a matter of routine?

Section E — Skeletal Pathology I — new material

Eleven marks, the joint largest section. Bone as living tissue, fracture and its healing, metabolic bone disease, and postural deviation. The osteoporosis material has the most direct effect on how you actually work of anything in this course.

E1. Bone as living tissue and Wolff's law

Bone is not inert scaffolding. It is a highly vascular, metabolically active connective tissue that rebuilds itself continuously.

Composition

About a third organic matrix, predominantly type I collagen, providing tensile strength and flexibility. About two thirds inorganic mineral, principally calcium hydroxyapatite, providing compressive strength and rigidity. The combination matters: collagen without adequate mineral gives bone that bends, which is the mechanism of rickets; mineral without adequate collagen gives bone that shatters, which is the mechanism of osteogenesis imperfecta.

The cells

Osteoblasts	Build bone. They synthesise osteoid and control its mineralisation. Stimulated by mechanical loading.
Osteocytes	Mature osteoblasts trapped within the matrix. They sense mechanical strain and signal the need for remodelling — the mechanotransducers of bone.
Osteoclasts	Resorb bone by dissolving mineral and digesting matrix.

Compact and spongy bone

Compact or cortical bone forms the dense outer shell and the shafts of long bones. Spongy, trabecular or cancellous bone forms the interior of the epiphyses, the vertebral bodies and the flat bones, with trabeculae aligned along lines of stress. Trabecular bone has a far higher surface-to-volume ratio and turns over faster, which is precisely why osteoporosis affects the vertebrae, the femoral neck and the distal radius first.

Wolff's law

Bone adapts its architecture in response to the mechanical loads placed upon it. Loaded bone becomes denser and stronger along lines of stress; unloaded bone is resorbed. The whole adult skeleton is replaced roughly every ten years.

The clinical consequences are direct. Weight-bearing and resistance exercise increase bone density; swimming and cycling, excellent as they are for other purposes, do not load bone appreciably. Immobilisation, bed rest and wheelchair use produce measurable bone loss within weeks, and it is regained slowly and not always fully. Progressive loading after fracture is not merely rehabilitation of muscle — it is what rebuilds the bone.

Calcium regulation

Parathyroid hormone raises blood calcium by increasing bone resorption, renal reabsorption and vitamin D activation. Calcitonin lowers it by inhibiting osteoclasts. Vitamin D is essential for calcium absorption from the gut, so deficiency produces osteomalacia regardless of calcium intake. Oestrogen restrains osteoclast activity, which is why bone loss accelerates sharply after menopause.

Review questions

40. State Wolff's law and give two clinical consequences.
41. Name the three bone cell types and the function of each.
42. Why does osteoporosis affect the vertebrae, femoral neck and distal radius before other sites?
43. Explain the roles of vitamin D and oestrogen in bone health.

E2. Fracture classification and healing

Classification

By severity	Closed (simple) — skin intact. Open (compound) — bone breaches the skin; high infection risk, a surgical emergency.
By direction	Transverse, oblique, spiral (from a twisting force), longitudinal.
By completeness	Complete; incomplete; greenstick (incomplete, in the flexible bone of a child); comminuted (three or more fragments).
By mechanism	Traumatic; stress (repetitive submaximal overload); pathological (through bone weakened by disease); avulsion (tendon or ligament pulls off its attachment).

By fragment position	Non-displaced, displaced, angulated, impacted.
Special types	Compression — vertebral body collapse, common in osteoporosis. Salter-Harris — through a growth plate in children.

Named fractures worth knowing: Colles, distal radius with dorsal displacement from a fall on an outstretched hand, the commonest upper limb fragility fracture; scaphoid, presenting with snuffbox tenderness, often missed on early imaging and at high risk of avascular necrosis; femoral neck, in older adults after a fall, with high morbidity and mortality; vertebral compression, often with no recalled trauma at all.

Stress fractures

From repetitive submaximal loading where resorption outpaces remodelling. Common at the metatarsals, tibia, fibula, femoral neck, navicular and pars interarticularis. Recognition matters because massage over one is contraindicated and delay risks progression to a complete fracture. Look for focal point tenderness directly on bone rather than the diffuse tenderness of medial tibial stress syndrome; pain that begins later in activity then progressively earlier, eventually persisting at rest and at night; and pain on percussion or hopping. Femoral neck and navicular stress fractures are high-risk and require prompt referral.

The four stages of fracture healing

1. Haematoma formation — hours to days. Ruptured vessels bleed and a clot forms; inflammation follows and debris is cleared.
2. Fibrocartilaginous (soft) callus — days to weeks. Fibroblasts and chondroblasts bridge the gap with collagen and cartilage. Capillaries grow in. The callus splints the fracture but has little mechanical strength, so this is the phase in which displacement most easily occurs.
3. Bony (hard) callus — weeks to months. Osteoblasts replace soft callus with trabecular bone. Clinical union is typically declared at six to twelve weeks in an adult.
4. Remodelling — months to years. Excess callus is removed, trabecular bone is replaced by compact bone, and the shaft is reshaped along lines of stress in accordance with Wolff's law.

Bone is one of the few tissues that heals with true tissue rather than with scar. Remodelling in a child can be so complete that no trace remains.

Review questions

1. Name the four stages of fracture healing in order, with approximate timeframes.
2. Define greenstick, pathological, avulsion and comminuted fractures.
3. What features suggest a stress fracture rather than medial tibial stress syndrome, and what do you do?
4. Why does bone heal differently from most other tissues?

E3. Fracture complications and management

Complications

Acute compartment syndrome	Commonest after tibial and forearm fractures. Pain out of proportion, tense compartment. Surgical emergency.
Deep vein thrombosis	From immobility and vessel injury. Unilateral calf pain, swelling and warmth. Absolute contraindication; risk of pulmonary embolism; urgent referral.
Fat embolism syndrome	Marrow fat entering the circulation after long bone or pelvic fracture. Breathlessness, confusion, petechial rash, typically 24 to 72 hours after injury. Medical emergency.
Non-union, delayed union and mal-union	Failure to heal, slow healing, or healing in a poor position. Causes include poor blood supply, movement at the site, infection and smoking.
Avascular necrosis	Bone death from interrupted blood supply. Femoral head, scaphoid, talus and lunate are the classic sites.
Complex regional pain syndrome	Disproportionate persistent pain with swelling, colour and temperature change, altered sweating and hair growth, and marked allodynia. Early recognition improves outcome.

The five Ps

Screened distal to a suspected acute fracture: Pain (intense and disproportionate), Pallor, Paraesthesia, Pulselessness, Paralysis. Pulselessness and paralysis indicate a vascular or neurological emergency. Pain out of proportion to the injury is the earliest warning of compartment syndrome.

The action for a suspected fracture: stop treatment; immobilise in the position found, supporting above and below; do not allow weight bearing; check and record distal circulation, sensation and movement; arrange emergency medical care; control bleeding and cover an open fracture without pushing anything back in; keep the client warm and still; document fully and complete an incident report.

Massage through fracture recovery

This is where the massage therapist contributes most, and it is examinable.

Immobilisation phase	The fracture site and cast are an absolute local contraindication. Reduce oedema proximal to the cast with lymphatic drainage. Treat the compensatory tension immobilisation always produces — neck and shoulder from crutch or sling use, contralateral overload, lumbar strain from altered gait. Maintain range in every joint not immobilised. Screen at every visit for deep vein thrombosis. Educate on elevation and warning signs.
Post-cast phase	Expect trophic changes: thin, dry, hypersensitive skin; muscle atrophy; joint stiffness; reduced proprioception; oedema. Begin very gently — the skin may not tolerate normal pressure or lubricant initially. Gentle fascial work and skin mobilisation. Graded work to atrophied muscle. Restore range progressively within pain-free limits. Frictions to the callus only once union is consolidated and medical clearance is given. Reinforce the progressive loading programme. Screen for complex regional pain syndrome.

Review questions

5. Give the five Ps and state which indicate an emergency.
6. What can a massage therapist safely do during the immobilisation phase of fracture recovery?
7. What changes should you expect when a cast is removed, and how does that shape the first treatments?
8. What is complex regional pain syndrome and why does early recognition matter?

E4. Metabolic bone disease

Osteoporosis

Reduced bone mineral density with deterioration of bone microarchitecture, producing bone that is fragile and disproportionately prone to fracture. The bone that remains is normally mineralised — there is simply less of it. Osteopenia describes reduced density short of the diagnostic threshold.

Primary type 1 is postmenopausal, driven by the fall in oestrogen, with accelerated trabecular loss and predominantly vertebral and distal radius fractures. Primary type 2 is age-related, affecting both cortical and trabecular bone in both sexes, with femoral neck fractures predominating. Secondary osteoporosis arises from corticosteroid therapy, hyperthyroidism, hyperparathyroidism, malabsorption, chronic kidney disease, prolonged immobility and eating disorders.

Osteoporosis is silent. There is no pain from low bone density itself, and the first indication is often a fragility fracture — one occurring from a fall from standing height or less, or from no recalled trauma at all. Progressive height loss and increasing thoracic kyphosis follow vertebral compression fractures. Diagnosis is by dual-energy X-ray absorptiometry reported as a T-score.

Treatment adaptation — the clearest example in the course

- Light to moderate pressure only. No deep work over the thoracic cage, spine, pelvis or any bony prominence.
- No joint mobilisation, no spinal traction, no rapid or forceful technique, no end-range passive movement.
- Avoid heavy compression through the rib cage in prone. Rib fracture from prone pressure is a documented event.
- Position with generous pillow support. A client with marked hyperkyphosis may be unable to lie prone or flat supine at all; side-lying or semi-reclined is often necessary.
- Assist on and off the table — the transfer is the highest-risk moment of the visit.
- Adapt on risk factors, not only on a stated diagnosis. Many clients with reduced bone density have never been assessed.

Sudden onset of severe mid-back pain in an older client, with or without recalled trauma, should raise the possibility of a vertebral compression fracture. Do not treat; refer.

Osteomalacia and rickets

Softening of bone from defective mineralisation of the osteoid matrix. The quantity of matrix is normal; the mineral content is not. This is the key distinction from osteoporosis. Caused by vitamin D deficiency, and also by malabsorption, chronic kidney disease and some medications. In children, with open growth plates, weight-bearing bones bow — this is rickets. In

adults: diffuse bone pain and tenderness, proximal muscle weakness, waddling gait, and increased fracture risk. Managed with vitamin D and calcium, generally effectively.

Paget disease of bone

A chronic disorder of remodelling in which osteoclastic resorption is excessively rapid and the compensatory osteoblastic formation is disorganised. The result is bone that is enlarged and dense in places but structurally weak and deformed. Usually over fifty-five, more common in men, often familial. Affects pelvis, lumbar spine, skull, femur and tibia, typically asymmetrically. Frequently asymptomatic and found incidentally. When symptomatic: deep aching bone pain often worse at night, bone enlargement and bowing, and increased skin warmth over the affected bone from increased vascularity. Complications include pathological fracture, secondary osteoarthritis and nerve compression.

Review questions

9. Define osteoporosis and distinguish it from osteomalacia and Paget disease.
10. Why is osteoporosis described as silent, and what is the practical consequence for a massage therapist?
11. List six specific modifications for a client with known or suspected osteoporosis, and justify each.
12. A client of seventy reports sudden severe mid-thoracic pain that began two days ago while lifting a bag. What is your concern and your action?

E5. Postural deviations

The spine has alternating curves: cervical and lumbar lordosis are secondary curves developing after birth with head control and standing; thoracic and sacral kyphosis are primary curves present from fetal life. The alternation acts as a spring, distributing load and increasing resistance to compression.

The distinction that governs everything

Functional	Soft tissue is pulling the spine out of alignment — muscle imbalance, fascial restriction, habitual posture, compensation for a leg length difference or for pain elsewhere. Bone and joint architecture is normal. Reduces or disappears with position change, active correction, or forward bending. Most treatable by manual therapy and exercise, particularly if addressed early.
Structural	Bones and joints have changed shape, or were abnormal from the outset. Does not correct with position change or effort. May be associated with underlying disease. Difficult or impossible to reverse; treatment aims at comfort, function and prevention of progression.

The types

- Hyperkyphosis — exaggerated thoracic curve. Associated with osteoporosis and vertebral compression fracture in older adults, and with Scheuermann disease in adolescents. Shortened pectorals and anterior chest; lengthened thoracic extensors. May restrict rib excursion and breathing.
- Hyperlordosis — exaggerated lumbar curve, commonly with anterior pelvic tilt. Shortened hip flexors and lumbar extensors; lengthened abdominals and gluteals. Often responds well to manual therapy and exercise.
- Flatback — reduced or absent lumbar lordosis with posterior pelvic tilt. Shortened hamstrings and abdominals. Reduces shock absorption.
- Scoliosis — lateral curvature, S- or C-shaped. Affects roughly one to three per cent of adolescents, more common and more progressive in girls, idiopathic in around eighty per cent of cases.
- Rotoscoliosis — lateral curve with vertebral rotation. The rotation produces the rib hump on forward bending and is the defining feature of a structural scoliosis.
- Forward head posture — head anterior to the line of gravity, greatly increasing load on the cervical extensors. Shortened suboccipitals and upper trapezius; lengthened deep neck flexors.

Assessment

Observe from posterior, lateral and anterior. The Adams forward bending test — the client bends forward with feet together and knees straight — reveals a rib hump or paraspinal prominence where vertebral rotation is present, indicating a structural scoliosis. Ask the client to actively correct: a functional deviation reduces, a structural one does not.

Treatment and honest goals

Treat both the shortened and the lengthened tissue. The lengthened side is frequently the painful one, held under sustained eccentric load, and it is commonly neglected. Adapt pressure for any bone fragility risk. Reinforce postural education and remedial exercise.

Two cautions carry marks. First, posture is a weak predictor of pain: many people with marked deviation have no symptoms and many with excellent alignment have severe pain, so treat the symptomatic tissue and the functional limitation, not the appearance. Second, never claim to straighten a structural curve. For a functional deviation addressed early, meaningful change is achievable; for a structural one, the honest goals are comfort, maintained mobility, improved respiratory ease and slowed progression. Claiming otherwise is untrue and a professional misconduct risk.

Review questions

13. Distinguish a functional from a structural postural deviation, and state how you would test which is present.
14. Describe the Adams forward bending test and state what a positive result indicates.
15. For a client with hyperlordosis, which muscle groups are likely to be short and which lengthened, and why does treating both matter?
16. A client asks whether you can correct her scoliosis. What do you say?

Section F — Skeletal Pathology II and Clinical Integration — new material

Eleven marks, and the section where all seven course outcomes converge. Joint disease, degenerative spinal conditions, bone infection and neoplasm, then the clinical reasoning framework. Many of these items give you a presentation and ask what it indicates or what you would do; they cannot be answered by recall alone.

F1. The synovial joint and articular cartilage

A synovial joint comprises articular cartilage covering the bone ends, a fibrous capsule, a synovial membrane producing synovial fluid, ligaments, and associated structures such as menisci, labra, bursae and fat pads. Muscles crossing the joint provide the dynamic stability on which the passive structures depend.

Articular cartilage — the critical tissue

Hyaline cartilage composed of chondrocytes within a matrix of type II collagen, proteoglycans and a high water content. It is avascular, aneural and alymphatic — no blood supply, no nerve supply, no lymphatic drainage. Three consequences follow, and all three are examinable.

5. Cartilage cannot signal its own damage. Early degeneration is entirely painless, which is why osteoarthritis is well advanced by the time it hurts. The pain of an arthritic joint comes from subchondral bone, capsule, synovium and surrounding muscle — never from the cartilage itself.
6. Cartilage is nourished by diffusion of synovial fluid, driven by intermittent compression and decompression during movement. Immobility starves it. This is the physiological argument for keeping arthritic joints moving.
7. Repair capacity is minimal. Defects fill with fibrocartilage, which is mechanically inferior and wears faster. Damage is, for practical purposes, permanent.

Inflammatory versus degenerative — the central distinction

Inflammatory	Morning stiffness over an hour. Better with movement, worse with rest. Warm, soft, boggy swelling, often red. Symmetrical and polyarticular. Small joints of hands and feet early. Systemic features — fatigue, malaise, fever, weight loss. Onset at any age. Raised inflammatory markers.
Degenerative	Morning stiffness under thirty minutes. Worse with use, better with rest. Little warmth; swelling is bony and hard. Asymmetrical, few joints. Weight-bearing and previously injured joints. No systemic features. Onset generally after the fourth decade. Normal inflammatory markers.

The clinical use is direct: an inflammatory pattern in a client without a diagnosis warrants referral; a degenerative pattern usually warrants treatment together with a suggestion to have it assessed.

Review questions

17. Why does articular cartilage heal so poorly, and what are the three clinical consequences of its being avascular and aneural?
18. Give six features that distinguish an inflammatory from a degenerative joint presentation.
19. Where does the pain of osteoarthritis actually come from, and why does that matter to your treatment?

F2. Osteoarthritis, and its comparison with rheumatoid arthritis

Osteoarthritis is the most common joint disease in the world. The description of it as simple wear and tear is inaccurate: it is an active process involving the whole joint, with an inflammatory component.

Pathophysiology

8. Chondrocyte metabolism shifts toward matrix degradation; proteoglycan content falls and cartilage loses its water-binding capacity and resilience.
9. The cartilage surface fibrillates and fissures, then thins. Joint space narrows.
10. Subchondral bone, now bearing increased load, becomes sclerotic. Subchondral cysts may form.
11. Osteophytes develop at the joint margins, attempting to increase the load-bearing surface.
12. Cartilage debris provokes secondary synovitis, producing effusion and inflammatory pain.
13. The capsule thickens and contracts, range falls, and surrounding muscle weakens from disuse, which increases joint load and accelerates the cycle.

Presentation and distribution

Deep aching pain, initially only with use and later at rest; morning stiffness characteristically under thirty minutes with gelling after inactivity; progressive loss of range; crepitus; bony hard enlargement with Heberden nodes at the distal interphalangeal joints and Bouchard nodes at the proximal; joint instability from muscle weakness and capsular laxity; and functional loss. Commonly affects the knee, hip, first carpometacarpal joint, interphalangeal joints, cervical and lumbar facets, and the first metatarsophalangeal joint.

Radiographic findings are joint space narrowing, subchondral sclerosis, osteophytes and subchondral cysts — but radiographic severity correlates poorly with symptoms. Treat the person and their function, not the image.

Management and the therapist's role

Exercise has the strongest evidence of any intervention. Weight management, education and pacing, analgesia, injection for flares, walking aids and bracing, and arthroplasty for advanced disease follow. The massage therapist contributes by reducing the secondary muscular pain and guarding that accompanies every arthritic joint, which is frequently the larger part of what the client actually feels; by treating compensation above and below; by gentle pain-free range of motion supporting cartilage nutrition; and by reinforcing the exercise programme at every visit.

During an acute flare with a warm swollen joint, work proximally and distally and leave the joint alone. Do not force range. Set honest goals: symptom and function improvement, not cartilage regeneration.

Osteoarthritis compared with rheumatoid arthritis

This is the single most examinable comparison in the course.

- Nature — degenerative whole-joint failure versus systemic autoimmune inflammatory disease.
- Primary tissue — articular cartilage versus synovial membrane.
- Onset age — over forty, or earlier if post-traumatic, versus thirty to fifty but any age including childhood.
- Symmetry — asymmetrical and few joints versus symmetrical and polyarticular.
- Hand joints — distal interphalangeal and first carpometacarpal versus metacarpophalangeal, proximal interphalangeal and wrist, with the distal interphalangeal joints characteristically spared.
- Morning stiffness — under thirty minutes versus over sixty and often several hours.
- Effect of activity — worse with use, better with rest, versus better with movement, worse after rest.
- Local signs — bony hard enlargement and crepitus with little warmth, versus warm, soft, boggy swelling with visible redness.
- Systemic features — absent versus fatigue, fever, weight loss, anaemia and nodules.
- Deformity — Heberden and Bouchard nodes versus ulnar deviation, swan neck and boutonnière.
- Laboratory findings — normal inflammatory markers versus raised markers with rheumatoid factor and anti-CCP.
- Massage priority — support movement and treat surrounding muscle, versus adapt to disease activity and never treat an active flare.

Review questions

20. Set out the six steps of osteoarthritis pathophysiology.
21. Compare osteoarthritis and rheumatoid arthritis across at least eight parameters.
22. A client with knee osteoarthritis asks what massage can do for her. What is your answer?
23. Why does radiographic severity correlate so poorly with symptoms in osteoarthritis?

F3. Crystal and inflammatory arthropathies

Gout

An inflammatory arthritis caused by deposition of monosodium urate crystals in a joint following prolonged elevation of blood uric acid. Risk factors include male sex, increasing age, obesity, hypertension, chronic kidney disease, diuretic therapy, alcohol, fructose-sweetened drinks and diets high in red meat and shellfish.

Presentation is abrupt, often overnight, with intense pain in a single joint, frequently described as the worst pain the client has experienced. The joint is hot, red, grossly swollen and exquisitely tender, often intolerant even of bedding. The first metatarsophalangeal joint is affected in about half of first attacks — podagra. Untreated attacks settle over one to two weeks; recurrence leads to chronic tophaceous gout with joint destruction.

Pseudogout

Calcium pyrophosphate deposition rather than urate. Affects larger joints, most commonly knee and wrist, typically in older adults. Clinically resembles gout but is generally less intensely painful; distinguished definitively by joint aspiration and crystal analysis.

For both: an acute attack is an absolute local contraindication — the joint must not be touched, compressed, mobilised or heated. Crucially, a hot red swollen single joint cannot be reliably distinguished from septic arthritis without investigation, so any first presentation of this kind requires same-day medical referral.

Septic arthritis — the emergency

Infection within the joint space. A hot, red, swollen, exquisitely painful joint with marked loss of range, the client resisting all movement, accompanied by fever and systemic illness. It destroys cartilage within days. Absolute contraindication and same-day emergency referral.

Ankylosing spondylitis and the spondyloarthropathies

A chronic inflammatory arthritis principally affecting the sacroiliac joints and axial skeleton, in which enthesial inflammation is followed by new bone formation and, over years, fusion. Typically begins in late adolescence or early adulthood, more often in men, strongly associated with HLA-B27.

The inflammatory back pain pattern is examinable and clinically important: onset before forty-five, gradual; duration over three months; morning stiffness over thirty to sixty minutes; improvement with exercise and no improvement with rest; night pain characteristically in the second half of the night, driving the client to get up and move; and alternating buttock pain. Recognising this in a young adult and referring matters, because average diagnostic delay is measured in years and early treatment alters outcome.

Progression brings loss of spinal mobility in all planes, reduced chest expansion from costovertebral involvement, and in advanced disease a fixed kyphotic posture. Extra-articular features include anterior uveitis, inflammatory bowel disease and psoriasis. A fused spine is osteoporotic and brittle, so fracture risk is markedly increased and any fracture is unstable.

Treatment: maintain mobility and comfort, treat paraspinal and chest wall musculature, support the exercise programme. No forceful spinal technique, no mobilisation, no traction. Adapt positioning.

The related conditions are psoriatic arthritis, occurring with psoriasis and capable of affecting the distal interphalangeal joints; reactive arthritis, following gastrointestinal or genitourinary infection; and enteropathic arthritis, with inflammatory bowel disease.

Review questions

24. Describe the presentation of acute gout, name the joint most commonly involved first, and state the action required.
25. Why must a hot, red, swollen single joint with fever be referred the same day?
26. Give the six features of inflammatory back pain and explain why recognising them matters.
27. Why does a client with established ankylosing spondylitis require particular caution with spinal technique?

F4. Degenerative spinal conditions, bone infection and bone tumours**Degenerative spine**

- Degenerative disc disease — age-related loss of disc water content and height. Near-universal with age and not truly a disease. Axial pain, worse with sitting and flexion. Poor correlation with imaging.
- Spondylosis — the general term for degenerative change: facet osteoarthritis plus disc degeneration with osteophyte formation.
- Facet joint syndrome — osteoarthritis of the zygapophyseal joints. Pain on extension and rotation toward the affected side, eased by flexion.
- Spondylolysis and spondylolisthesis — a defect in the pars interarticularis, and anterior slip of one vertebra on another. Common in adolescent athletes in extension sports. Pain on extension; avoid extension and forceful technique.
- Spinal stenosis — narrowing of the central canal or foramina, producing neurogenic claudication: leg pain on walking, relieved by sitting and by flexion. Typically older adults.
- Disc herniation — nucleus displacing through the annulus, producing radicular pain in a dermatomal distribution with possible weakness, numbness and reflex change. Most resolve conservatively over weeks. Refer any progressive neurological deficit.

Cauda equina syndrome — the spinal emergency

Compression of the nerve root bundle below the termination of the spinal cord, most often by a large central disc herniation. Delayed decompression risks permanent bladder, bowel and sexual dysfunction.

Screen for: saddle anaesthesia — numbness or altered sensation in the perineum, buttocks and inner thighs; bladder dysfunction — difficulty initiating, retention, reduced awareness of filling, or incontinence; bowel incontinence or loss of sphincter control; recent sexual dysfunction; bilateral leg pain, weakness or numbness; and progressive neurological deficit.

The action: stop treatment, arrange emergency care — this is an emergency department presentation, not a family physician appointment — and document what you found, what you said and what you did.

Osteomyelitis

Infection of bone and bone marrow, most commonly by *Staphylococcus aureus*. Routes are haematogenous spread, direct inoculation through an open fracture or surgery, or contiguous spread from adjacent soft-tissue infection or ulcer. Risk factors include diabetes, particularly with foot ulceration, peripheral vascular disease, immunosuppression, intravenous drug use, recent orthopaedic surgery, prosthetic joints and open fracture.

Presents with deep, constant, localised bone pain unrelieved by rest or position; local warmth, swelling and tenderness; fever, chills and malaise; and reluctance to use or bear weight on the limb. Absolute contraindication and urgent referral.

Bone tumours

Primary bone tumours are uncommon. Benign: osteoma, osteoid osteoma, osteochondroma, giant cell tumour. Malignant: osteosarcoma, typically in adolescents and young adults around the knee; Ewing sarcoma in children and young adults; chondrosarcoma in older adults.

Metastatic bone disease is far more common. Breast, prostate, lung, kidney and thyroid cancers commonly metastasise to bone, favouring the vertebrae, pelvis, ribs, proximal femur and proximal humerus — axial and proximal rather than distal. Pathological fracture through weakened bone is a real risk.

The pain pattern that must not be missed: deep, constant, boring pain unrelated to movement or position, not relieved by rest, characteristically worse at night, progressive rather than fluctuating, and accompanied by unexplained weight loss, fatigue, fever or a history of cancer of any kind at any time. Do not treat. Refer promptly and document.

Review questions

28. Name the features of cauda equina syndrome and state the required action.
29. Describe the pain pattern suggestive of bone malignancy and contrast it with mechanical back pain.
30. In whom should osteomyelitis be suspected, and how does it present?
31. How does spinal stenosis present, and what distinguishes it from vascular claudication?

F5. Clinical reasoning, red flags and the treatment decision

This is the material that draws the whole course together, and it carries a disproportionate share of the marks in Section F because the items are applied rather than recall.

The four-stage framework

14. History — the presenting complaint in the client's words; onset, mechanism, location and radiation; character and severity; aggravating and easing factors, which is frequently where the answer is; the twenty-four hour pattern including night pain and the duration of morning stiffness; whether it is improving, static or worsening; previous episodes; medical history, medications and surgeries; red flag screening; and the client's occupation, activity and goals.
15. Observation and palpation — does the client look well; gait and functional movement; posture in all three views; skin colour, integrity and temperature; whether swelling is soft and boggy or bony and hard; deformity and muscle bulk; temperature compared side to side; tissue texture and restriction; whether tenderness is focal or diffuse and over which structure. Always compare with the unaffected side.
16. Testing — active free range first, then passive with end feel, then resisted isometric, then special tests and neurological screening as indicated.
17. Impression and plan — what tissue, what stage, what is driving it; any red flags or contraindications; treat, adapt or refer; goals agreed with the client; techniques, positioning, pressure and duration; home care; informed consent obtained and recorded; and the reassessment and documentation plan.

Red flags across all six days

- Constitutional — unexplained weight loss, fever, chills, night sweats, profound fatigue, a client who simply looks unwell.

- Pain pattern — constant pain unrelated to movement or position, severe night pain that wakes the client, progressive rather than fluctuating, unrelieved by any position.
- Neurological — progressive weakness or numbness, bilateral limb symptoms, saddle anaesthesia, bowel or bladder change.
- Vascular — unilateral calf pain with swelling and warmth, sudden breathlessness or chest pain, absent or diminished distal pulse, pallor or dusky discolouration of a limb.
- Joint — a single hot, red, swollen, exquisitely painful joint, particularly with fever.
- Integumentary — a lesion meeting ABCDE criteria, spreading erythema with warmth and fever, a non-healing ulcer, a rash with systemic illness.

A single red flag in isolation is a prompt for careful questioning rather than alarm. A cluster, or any neurological or bladder finding, means you do not treat and you refer.

Treat, adapt or refer

Treat as planned	No red flags, the condition is within scope, tissue and stage identified, standard technique appropriate.
Treat with adaptation	A condition or risk factor constrains pressure, position, technique, duration or region, but treatment remains appropriate and beneficial. This is by far the most common outcome in real practice.
Withhold and refer	An absolute contraindication, a red flag cluster, or an undiagnosed presentation requiring medical assessment. Referral is not a failure; it is the clearest demonstration of professional competence.

Documenting and communicating

Record the findings that led to your decision, not only the decision. Where you adapted, record what you changed and why. Where you withheld treatment, record the findings, what you advised, the urgency you conveyed and the client's response. Record consent specifically.

When referring: describe what you found rather than what you think it is; be clear about urgency — today, this week, or at their convenience; do not diagnose and do not speculate aloud about serious diagnoses; offer to provide a written summary for the physician; and follow up at the next contact.

Review questions

32. Describe the four stages of the clinical reasoning framework and what each contributes.
33. Give six red flags spanning at least three body systems.
34. Distinguish treat, adapt and refer, and give a clinical example of each from this course.
35. Why is it said that referral is a demonstration of competence rather than a failure?
36. What must a chart note record when you withhold treatment and refer?

Answer Key and Discussion

Answers to all 79 review questions, with discussion. Work through the questions first and write your own answer before reading these. An answer you have retrieved from memory is remembered; an answer you have read is not.

Where an answer says more than you wrote, that is the point. The discussion explains why the answer is what it is, which is what the examination rewards — it is written to test whether you can apply a principle to a presentation you have not seen before, not whether you can recall a list.

A1. The disease process, terminology and cellular response

1. Give the four stages of the disease process in order.

Etiology (the cause), pathogenesis (the mechanism by which the cause produces disease in the tissue), manifestation (the resulting signs and symptoms), and resolution (the outcome, whether recovery, chronicity, disability or death).

2. Distinguish hypertrophy, hyperplasia and atrophy.

Hypertrophy is an increase in the size of existing cells in response to increased demand, such as muscle enlarging with resistance training. Hyperplasia is an increase in the number of cells, such as callus forming on repeatedly loaded skin. Atrophy is a decrease in cell size or number from reduced demand, disuse, denervation or poor nutrition, such as muscle wasting under a cast.

3. Decode "osteopenia", "osteomalacia" and "osteoporosis", and state how they differ.

Osteopenia is bone deficiency — reduced density that has not yet reached the diagnostic threshold for osteoporosis. Osteomalacia is bone softening — a normal quantity of bone matrix that is inadequately mineralised, from vitamin D deficiency. Osteoporosis is reduced bone mineral density with deterioration of microarchitecture — the bone present is normally mineralised, there is simply less of it. The three are examined directly in Section E and the distinction between the last two is a reliable discriminator.

A2. Infection, infection control and inflammation

4. Why do standard precautions apply to every client rather than only to those who disclose an infection?

Because infectious status is frequently unknown to both parties. A person may be infectious during the incubation period before any symptom appears, or during the non-specific prodrome, and some blood-borne infections remain asymptomatic for years. A precaution triggered by disclosure fails in precisely the situations where it is most needed.

5. State the correct order of actions following a sharps injury in the clinic.

Wash immediately with soap and running water, allowing the site to bleed freely, without scrubbing or squeezing. Irrigate any mucous membrane or eye exposure with water or saline. Seek urgent medical assessment, since post-exposure prophylaxis is time-critical. Report and document under clinic policy. Follow up with recommended testing. First aid precedes both the incident report and the application of any antiseptic dressing.

6. Define the three classes of contraindication and explain why the conditional class matters most in practice.

Absolute — no massage anywhere today. Local — the affected area is not treated but the rest of the body may be. Conditional or relative — treatment proceeds with specified modifications to pressure, position, technique or duration. The conditional class matters most because it is by far the largest: osteoporosis, controlled autoimmune disease, anticoagulant therapy, pregnancy, fibromyalgia and advanced age all sit there. Most pathology changes how you work rather than whether you work.

A3. Skin structure, function and lesion terminology

7. Where does mitosis occur in the epidermis, and how long does a keratinocyte take to reach the surface?

In the stratum basale, the deepest layer. A keratinocyte takes roughly four weeks to migrate to the surface and be shed from the stratum corneum. In psoriasis this transit is compressed to days, so cells arrive immature and accumulate as the characteristic thick silvery scale.

8. Why can a partial-thickness burn re-epithelialise from within the wound bed while a full-thickness burn cannot?

Because a partial-thickness injury spares some dermis and therefore spares the hair follicles and glands within it, which act as islands of epithelial cells from which the surface regenerates. A full-thickness injury destroys the dermis and all its appendages, leaving no internal source of epithelium, so the wound can only close by contraction and migration from the margins — slowly, and with scarring.

9. Classify as primary or secondary: wheal, fissure, nodule, lichenification, bulla, excoriation.

Primary — wheal, nodule and bulla, all arising directly from the disease process. Secondary — fissure, lichenification and excoriation, all resulting from change to skin already affected. Note that lichenification and excoriation both arise from scratching, which is the commonest route by which a primary lesion becomes a secondary one.

B1. Infections of the skin

10. What features distinguish cellulitis, and what action does it require?

A warm, red, tender, swollen area with poorly defined and advancing borders, most often on the lower leg, frequently with fever and malaise. It requires withholding treatment and arranging same-day medical assessment. The discriminating features are the indistinct advancing edge and the systemic symptoms; a sharply demarcated lesion without systemic illness is more likely something else.

11. Explain why the eruption of herpes zoster stops at the midline.

Because the virus reactivates in a single dorsal root ganglion and travels along that one sensory nerve to the skin it supplies. A nerve root supplies one dermatome, and dermatomes do not cross the midline. The pathogenesis therefore predicts the presentation exactly, which is why understanding the mechanism removes the need to memorise the appearance.

12. Why does a client on methotrexate or long-term corticosteroids warrant a lower threshold for referral of a skin infection?

Because both suppress the immune response. The infection may progress faster, present with fewer of the classic inflammatory signs because the response producing them is blunted, and deteriorate more quickly toward systemic illness. The same appearance therefore carries greater urgency, and waiting to see whether it settles is not appropriate.

B2. Inflammatory conditions, wounds, burns and scars

13. Name the three healing sequences taught in this course, with the number of stages in each, and state which body of material each belongs to.

Wound healing has four phases — haemostasis, inflammation, proliferation, maturation — and belongs to the skin material on Day 2. Soft-tissue healing has three stages — acute or inflammatory, sub-acute or proliferative, and chronic or remodelling — and belongs to Day 4. Fracture healing has four stages — haematoma, soft callus, hard callus, remodelling — and belongs to Day 5. They describe related biology in different tissues, and the examination tests whether you can keep them apart.

14. What single feature distinguishes a hypertrophic scar from a keloid, and why is that clinically useful?

Whether the scar extends beyond the boundaries of the original wound. Hypertrophic scars remain within the original margins and often improve over time; keloids extend into surrounding uninjured skin and do not regress spontaneously. It is clinically useful because it predicts the course and guides expectations — and because keloid tissue may be aggravated by heavy mechanical stimulus, so scar work should be conservative.

15. Why is a full-thickness burn painless in the burned area, and what is the danger in that?

Because full-thickness injury destroys the dermis and with it the sensory nerve endings. The danger is that absence of pain leads to underestimation of severity, by clients and by inexperienced assessors. A typical presentation is a painless leathery central area surrounded by an intensely painful partial-thickness margin, and it is the painless part that is the more serious injury.

16. A client asks whether their psoriasis means you cannot treat them. What is your answer, and why?

You can treat them. Psoriasis is an immune-mediated condition and is not contagious. Intact plaques may be worked over with adequate lubricant, avoiding any technique that would abrade or crack them; cracked, bleeding or secondarily infected plaques are a local contraindication. Because psoriasis worsens with stress, massage may plausibly help the condition itself and not only the symptoms. It is worth saying clearly that it is not contagious, since many clients with visible psoriasis have been treated as though it were.

C1. Connective tissue and autoimmunity

17. Why does the relapsing and remitting course of autoimmune disease matter more to treatment planning than the diagnosis?

Because the same client with the same diagnosis may require completely different treatment on different visits. In remission, normal technique may be entirely appropriate; during a flare, the affected structures are a local

contraindication. A therapist who plans from the label rather than from the current presentation will eventually treat an active flare as though it were remission and make the client worse.

18. What does a history of several years of oral corticosteroids tell you before you have examined anything?

To expect thin, fragile skin that bruises and tears easily; reduced bone density with raised fracture risk; muscle weakness; delayed healing; and suppressed immunity, so infections progress faster and present less obviously. It justifies limiting pressure, taking care with positioning and shear, avoiding joint mobilisation, and lowering the referral threshold for anything that looks infected — without waiting for a formal diagnosis of osteoporosis.

C2. The systemic autoimmune conditions

19. What tissue is the primary target in rheumatoid arthritis, and what follows from that?

The synovial membrane. Autoimmune synovitis produces a proliferative pannus that erodes cartilage and then subchondral bone, so cartilage damage is secondary. From this single fact follow the joint distribution — joints rich in synovium, meaning the small joints of the hands and feet and the wrists — the warm boggy swelling rather than hard bony enlargement, the systemic nature of the disease, and the characteristic deformities arising from damage to capsule, ligament and tendon.

20. Give the hydrotherapy implications of Raynaud phenomenon.

Cold applications are contraindicated, and contrast therapy is contraindicated, because both provoke the vasospasm the condition consists of. Keep the treatment room warm, warm your hands before contact, and advise the client to keep the whole body warm rather than only the extremities, since cooling of the core triggers peripheral vasospasm.

21. Define allodynia and hyperalgesia and state the treatment consequence.

Allodynia is pain in response to a stimulus that would not normally be painful, such as light touch or the pressure of clothing. Hyperalgesia is an exaggerated response to a stimulus that is painful. The consequence is that pressure tolerance may be far below what the tissue itself would require: begin lighter and shorter than you otherwise would, and progress according to how the client felt over the following one to three days rather than how they felt in the room.

22. Why is stretching usually the wrong approach for a hypermobile client who reports feeling tight?

Because the problem is instability rather than shortness. The client already has excessive passive range, and the muscular tension they feel is frequently protective, compensating for absent passive restraint. Releasing it wholesale can leave the joint less supported and the client more symptomatic. The appropriate emphasis is stability, strengthening and proprioceptive retraining.

D1. The three stages of tissue healing

23. Name the three stages of soft-tissue healing in order, with an approximate timeframe and the principal goal of each.

Acute or inflammatory, roughly the first three to five days — goal is to control oedema and pain and to protect the repair. Sub-acute or proliferative, from around day five to three weeks — goal is to support organised collagen deposition, restore pain-free range and limit excessive adhesion. Chronic or remodelling, from around three weeks onward and continuing for months — goal is to remodel and realign the scar and restore full range and strength. The stages overlap rather than switching cleanly.

24. A client presents four months after a gradual-onset elbow problem. What stage are they in, and what does that permit?

The chronic or remodelling stage. That permits deeper work, cross-fibre frictions to reorganise collagen, myofascial release, full-range work and stretch, and heat applied before treatment. It also means progressive loading is the appropriate primary management. Treating this as an acute problem — rest, ice and avoidance — is a common error that leaves the tissue unremodelled and the client no better.

25. Why is heat contraindicated in the acute stage?

Because heat produces vasodilation, which increases blood flow into tissue that is already bleeding and oedematous. The result is more swelling, more pain and a longer recovery. Heat also masks the tissue response, making it harder to judge how the injury is progressing. It becomes appropriate once the acute inflammatory phase has passed, where it increases tissue extensibility before stretching or deeper work.

26. List the factors that delay soft-tissue healing.

Advancing age; poor nutrition, particularly protein deficiency; diabetes; impaired arterial supply or venous return; infection; smoking, which impairs oxygen delivery; corticosteroid and some immunosuppressive medication; repeated

mechanical stress or movement across the healing tissue; and inadequate protection in the early stage. Several commonly coexist, and recognising them lets you set realistic expectations about recovery time.

D2. Strain, sprain and contusion

27. *State the essential difference between a strain and a sprain.*

A strain is injury to muscle or tendon; a sprain is injury to a ligament. The terms describe the tissue injured, not the severity or the timeframe. They are frequently reversed, including by students who otherwise perform well, and precision matters when communicating with other practitioners and when documenting.

28. *Describe the three grades of strain and sprain, and explain the paradox of pain in a grade 3 sprain.*

Grade 1 is microscopic tearing with mild pain and retained function. Grade 2 is partial tearing with moderate pain, swelling, bruising and definite functional loss or joint laxity. Grade 3 is complete rupture with marked swelling, possible palpable defect and major functional loss. The paradox is that a grade 3 sprain may hurt less than a grade 2, because a completely ruptured ligament is no longer under tension and therefore no longer generating pain from stretch. Joint instability, not pain severity, is the indicator of grade 3.

29. *Why are re-injury rates after ankle sprain so high?*

Because the proprioceptive receptors within the ligament are damaged along with the collagen, so joint position sense remains impaired long after pain and swelling have resolved. The client feels recovered and returns to activity with degraded positional feedback, and the joint is caught in the same vulnerable position again. This is why proprioceptive retraining, rather than manual therapy alone, is the decisive element of rehabilitation.

30. *What is myositis ossificans, and why does it make early deep work on a contusion contraindicated?*

It is the formation of heterotopic bone within muscle tissue following a significant contusion or repeated trauma, classically in the quadriceps or brachialis. Aggressive massage and forced stretching of an acute deep contusion are recognised contributing factors, presumably by disturbing the healing haematoma. Early deep work is therefore contraindicated, and the acute management of a deep contusion is protection and gentle proximal work rather than attempts to disperse the bruise.

31. *How would you distinguish delayed onset muscle soreness from a grade 1 muscle strain?*

Delayed onset muscle soreness appears twelve to twenty-four hours after unaccustomed activity, particularly eccentric loading; it is diffuse, usually bilateral, has no single point of injury, peaks at twenty-four to seventy-two hours and resolves within a week. A strain has a definite moment of onset that the client can identify, focal tenderness usually at the musculotendinous junction, and pain reproduced on resisted contraction of that muscle. Onset pattern and focality are the discriminators.

D3. Tendinopathy and overuse conditions

32. *Explain why the term "tendinopathy" is preferred to "tendinitis", and what that changes about management.*

Because chronic tendon problems typically show little active inflammation, and instead show disorganised collagen, increased ground substance and neovascularisation — a failed healing response rather than an inflammatory one. "Tendinopathy" makes no claim about which process is present. It changes management fundamentally: if the problem were inflammation, rest and anti-inflammatory measures would be appropriate, whereas for a failed repair, rest allows further weakening and progressive loading is the primary treatment.

33. *How would you distinguish bursitis from tendinopathy at the same joint?*

Bursitis produces localised swelling, warmth and tenderness directly over the bursa, with pain on passive movement that compresses it. Tendinopathy produces pain on resisted contraction of the associated muscle and on palpation of the tendon itself, with pain that eases on warm-up and is worst the morning after loading. The discriminator is resisted contraction versus passive compression.

34. *Describe the presentation of acute compartment syndrome and state why the limb must not be elevated.*

Pain out of proportion to the injury, a tense and swollen compartment, pain on passive stretch of the muscles within the compartment, and paraesthesia in the distribution of the nerve traversing it. Pulselessness is a late and ominous sign. The limb must not be elevated above heart level because elevation reduces arterial perfusion pressure, and in a compartment where tissue pressure is already high this worsens the ischaemia. It is a surgical emergency and massage is absolutely contraindicated.

35. How does chronic exertional compartment syndrome differ from the acute form?

The chronic exertional form produces predictable pain and tightness beginning at a predictable point during exercise, which resolves within an hour of stopping and recurs on the next comparable effort. It is not an emergency. The acute form arises after trauma or fracture, is progressive rather than self-limiting, is characterised by pain out of proportion to the injury, and is a surgical emergency. The distinguishing features are the relationship to exercise and whether the symptoms resolve on rest.

D4. Assessment: selective tissue tension testing**36. Explain how selective tissue tension testing distinguishes contractile from non-contractile structures.**

Resisted isometric testing loads the contractile structures — muscle and tendon — while producing no movement and therefore minimal load on the inert tissues, so pain there implicates muscle or tendon. Passive movement loads the non-contractile structures — capsule, ligament, bursa, cartilage — while the muscle is relaxed, so pain there implicates inert tissue. Active movement loads both and therefore localises without differentiating. The sequence is active, then passive, then resisted.

37. What does an empty end feel indicate, and what should you do?

An empty end feel is one where the client stops the movement before you feel any tissue resistance, because of pain. It suggests significant pathology — acute inflammation, infection, fracture or neoplasm — rather than a mechanical restriction. Do not push through it. It is a finding that warrants withholding aggressive treatment and, in most cases, referring for assessment.

38. Give three normal and three abnormal end feels.

Normal — soft tissue approximation, as in knee flexion where the calf meets the thigh; tissue stretch, a firm elastic resistance as in ankle dorsiflexion; and bone to bone, an abrupt hard stop as in elbow extension. Abnormal — empty, where the client stops you before resistance is felt; springy block, a rebound suggesting internal derangement such as a meniscal tear; and boggy, a soft mushy stop suggesting joint effusion. Muscle spasm and an early capsular feel are also abnormal.

39. Why should you assess the joint above and the joint below a painful region as a matter of routine?

Because the site of pain is frequently not the source. Fascial continuity transmits load between structures that are not directly attached; a restricted joint forces a neighbour to absorb more load than it is built for; weakness in one group produces compensatory overwork in another; and trigger points refer pain to sites distant from the muscle harbouring them. Treating only the painful tissue reduces symptoms; treating the chain reduces recurrence.

E1. Bone as living tissue and Wolff's law**40. State Wolff's law and give two clinical consequences.**

Bone adapts its architecture in response to the mechanical loads placed upon it: loaded bone becomes denser and stronger along the lines of stress, and unloaded bone is resorbed. Consequences: weight-bearing and resistance exercise increase bone density and are the intervention that actually changes it in osteoporosis, whereas swimming and cycling do not load bone appreciably; and immobilisation produces measurable bone loss within weeks, which is why progressive loading after a fracture is what rebuilds the bone rather than merely restoring muscle.

41. Name the three bone cell types and the function of each.

Osteoblasts build bone, synthesising osteoid and controlling its mineralisation, and are stimulated by mechanical loading. Osteocytes are mature osteoblasts trapped within the matrix that sense mechanical strain and signal the need for remodelling. Osteoclasts resorb bone by dissolving mineral and digesting matrix. The balance between osteoblast and osteoclast activity determines whether bone mass is maintained, gained or lost.

42. Why does osteoporosis affect the vertebrae, femoral neck and distal radius before other sites?

Because those sites are rich in trabecular bone, which has a far higher surface-to-volume ratio than cortical bone and therefore turns over much faster. Where resorption exceeds formation, the tissue with the highest turnover loses mass first. This is why the characteristic fragility fractures of osteoporosis are vertebral compression fractures, femoral neck fractures and Colles fractures of the distal radius.

43. Explain the roles of vitamin D and oestrogen in bone health.

Vitamin D is essential for the absorption of calcium from the gut; deficiency produces defective mineralisation — osteomalacia in adults, rickets in children — regardless of dietary calcium intake. Oestrogen restrains osteoclast activity,

so the fall in oestrogen at menopause removes that restraint and bone loss accelerates sharply, which is the mechanism of postmenopausal (primary type 1) osteoporosis.

E2. Fracture classification and healing

44. Name the four stages of fracture healing in order, with approximate timeframes.

Haematoma formation, hours to days. Fibrocartilaginous or soft callus, days to weeks. Bony or hard callus, weeks to months, with clinical union typically at six to twelve weeks in an adult. Remodelling, months to years. The soft callus stage is the one of greatest mechanical vulnerability, because the callus splints the fracture without providing real strength.

45. Define greenstick, pathological, avulsion and comminuted fractures.

Greenstick — an incomplete fracture in the flexible, more porous bone of a child, which bends and cracks rather than breaking through. Pathological — a fracture through bone already weakened by disease such as osteoporosis, metastatic deposit or Paget disease, often from trivial force. Avulsion — a fragment pulled off by the tendon or ligament attached to it. Comminuted — three or more fragments.

46. What features suggest a stress fracture rather than medial tibial stress syndrome, and what do you do?

Focal point tenderness directly on the bone rather than the diffuse tenderness along the posteromedial border seen in medial tibial stress syndrome; pain that begins progressively earlier in activity and eventually persists at rest and at night; and pain reproduced by percussion or hopping. Withhold local treatment and refer for imaging. Massage over a stress fracture is contraindicated and delay risks progression to a complete fracture; femoral neck and navicular sites are particularly high-risk.

47. Why does bone heal differently from most other tissues?

Because it heals with true bone rather than with scar. Most soft tissues repair by laying down fibrous scar that never matches the original in structure or properties. Bone proceeds through haematoma, soft callus and hard callus to a remodelling phase in which the callus is resorbed and the bone is reshaped along lines of stress, so the healed bone can be structurally equivalent to the original — and in children, indistinguishable from it.

E3. Fracture complications and management

48. Give the five Ps and state which indicate an emergency.

Pain — intense and disproportionate to the mechanism; Pallor; Paraesthesia; Pulselessness; Paralysis, all screened distal to the injury. Pulselessness and paralysis indicate a vascular or neurological emergency requiring immediate care. Pain out of proportion to the apparent injury is the earliest warning sign of compartment syndrome and should not be dismissed because the other four are absent.

49. What can a massage therapist safely do during the immobilisation phase of fracture recovery?

Reduce oedema in the limb proximal to the cast using lymphatic drainage; treat the compensatory tension immobilisation produces in the neck and shoulder from crutch or sling use, in the contralateral limb from overload, and in the lumbar spine from altered gait; maintain range of motion in every joint not immobilised, above and below the cast; screen at each visit for complications, particularly deep vein thrombosis; and educate the client on elevation, movement of free joints and the warning signs requiring medical contact. The fracture site and the cast itself are an absolute local contraindication.

50. What changes should you expect when a cast is removed, and how does that shape the first treatments?

Trophic changes: skin that is thin, dry, scaly and hypersensitive; muscle atrophy; joint stiffness; markedly reduced proprioception; and oedema. The first treatments must therefore be very gentle — the skin may not tolerate normal pressure or even normal lubricant initially. Work progresses through gentle fascial and skin mobilisation, graded work to atrophied muscle, and progressive restoration of range within pain-free limits. Frictions to the callus wait until union is consolidated and medical clearance is given.

51. What is complex regional pain syndrome and why does early recognition matter?

A condition of persistent pain disproportionate to the original injury, accompanied by swelling, colour and temperature change, altered sweating, hair and nail growth, and marked allodynia so that very light touch is painful. Early recognition matters because outcomes are substantially better when it is identified and managed early. For the therapist it means using only very light contact, not pushing through pain, and referring promptly rather than assuming the client is simply not progressing.

E4. Metabolic bone disease

52. Define osteoporosis and distinguish it from osteomalacia and Paget disease.

Osteoporosis is reduced bone mineral density with deterioration of microarchitecture — less bone, normally mineralised. Osteomalacia is a normal quantity of bone matrix that is inadequately mineralised, from vitamin D deficiency — the bone is soft rather than scarce. Paget disease is disordered, accelerated and disorganised remodelling producing bone that is enlarged and dense in places but structurally weak, typically focal to one or a few bones. Quantity, mineralisation, and remodelling quality respectively.

53. Why is osteoporosis described as silent, and what is the practical consequence for a massage therapist?

Because reduced bone density produces no pain of itself. The first indication is frequently a fragility fracture — from a fall from standing height or less, or from no recalled trauma at all. The practical consequence is that a substantial proportion of clients in any general practice have reduced bone density and do not know it, so pressure, positioning and joint technique should be adapted on the basis of risk factors — age, sex, corticosteroid use, height loss, kyphosis, family history — rather than waiting for a stated diagnosis.

54. List six specific modifications for a client with known or suspected osteoporosis, and justify each.

Light to moderate pressure only, because the bone cannot tolerate deep compression. No deep work over the thoracic cage or spine, because rib and vertebral fracture from prone pressure is documented. No joint mobilisation, traction or end-range passive movement, because forceful technique risks fracture. Positioning with generous pillow support, and side-lying or semi-reclined where a marked kyphosis prevents lying flat. Assistance on and off the table, since the transfer is the highest-risk moment of the visit. And adaptation based on risk factors rather than only a stated diagnosis, since most reduced bone density is undiagnosed.

55. A client of seventy reports sudden severe mid-thoracic pain that began two days ago while lifting a bag. What is your concern and your action?

A vertebral compression fracture. In an older client, and particularly one with risk factors for reduced bone density, a sudden onset of severe mid-back pain from a trivial mechanism — or from none at all — raises this possibility. The action is not to treat and not to mobilise, and to refer for medical assessment. Continuing with a booked treatment on the assumption of a muscular strain would be a significant error.

E5. Postural deviations

56. Distinguish a functional from a structural postural deviation, and state how you would test which is present.

A functional deviation arises from soft tissue — muscle imbalance, fascial restriction, habit or compensation — with normal bone and joint architecture, and it reduces or resolves with a change of position, with active correction, or on forward bending. A structural deviation involves changed bone or joint shape and does not correct. To test: ask the client to actively correct the posture, compare standing with sitting, and perform the Adams forward bending test looking for a rib prominence indicating vertebral rotation.

57. Describe the Adams forward bending test and state what a positive result indicates.

The client stands with feet together and knees straight and bends forward while you observe from behind at eye level with the spine. A unilateral rib hump or paraspinal prominence indicates rotation of the vertebral bodies, which is the defining feature of a structural scoliosis. A functional curve reduces on forward bending and produces no rib prominence.

58. For a client with hyperlordosis, which muscle groups are likely to be short and which lengthened, and why does treating both matter?

Likely shortened: hip flexors and lumbar extensors. Likely lengthened: abdominals and gluteals. Treating both matters because the lengthened tissue is frequently the painful one — it is held under sustained eccentric load and is commonly neglected in favour of whatever feels tight. Treating only the short side addresses half the problem and often does not change the pattern.

59. A client asks whether you can correct her scoliosis. What do you say?

You explain honestly what is and is not achievable. If the curve is structural, involving rotation and changed vertebral shape, it cannot be straightened by manual therapy, and claiming otherwise is both untrue and a professional misconduct risk. What can genuinely be offered is reduction of the muscular pain and tension that accompanies sustained asymmetry, maintenance of mobility, improved ease of breathing where rib excursion is restricted, and support of an exercise programme. For a functional curve addressed early, meaningful change in alignment is achievable, and it is fair to say so.

F1. The synovial joint and articular cartilage

60. *Why does articular cartilage heal so poorly, and what are the three clinical consequences of its being avascular and aneural?*

It is avascular, aneural and alymphatic, so there is no blood supply to deliver repair cells and no inflammatory repair response; defects fill with mechanically inferior fibrocartilage. The three consequences: early degeneration is painless because there are no nerve endings, so osteoarthritis is advanced before it hurts; cartilage is nourished by synovial fluid driven by intermittent loading, so immobility starves it and movement is genuinely therapeutic; and damage is effectively permanent, so no manual therapy can regenerate it.

61. *Give six features that distinguish an inflammatory from a degenerative joint presentation.*

Duration of morning stiffness — over an hour versus under thirty minutes. Response to activity — inflammatory improves with movement and worsens with rest, degenerative does the reverse. Swelling quality — warm, soft and boggy versus hard and bony with little warmth. Symmetry — symmetrical and polyarticular versus asymmetrical and few joints. Joint distribution — small joints of hands and feet early versus weight-bearing and previously injured joints. Systemic features — present versus absent. Onset age and inflammatory markers also differ.

62. *Where does the pain of osteoarthritis actually come from, and why does that matter to your treatment?*

From the subchondral bone, the joint capsule, the synovium where secondary synovitis has developed, and the surrounding musculature — never from the cartilage, which is aneural. It matters because it explains why treating the muscles crossing and supporting the joint is genuinely useful rather than merely palliative, and why radiographic severity correlates so poorly with symptoms. It also sets the honest limit on what you can claim: you can change the muscular and capsular contribution, not the cartilage.

F2. Osteoarthritis, and its comparison with rheumatoid arthritis

63. *Set out the six steps of osteoarthritis pathophysiology.*

Chondrocyte metabolism shifts toward matrix degradation and proteoglycan content falls, so cartilage loses water-binding capacity and resilience. The surface fibrillates and fissures, then thins, narrowing the joint space. Subchondral bone bearing increased load becomes sclerotic and may form cysts. Osteophytes develop at the joint margins. Cartilage debris provokes secondary synovitis with effusion. The capsule thickens and contracts, range falls, and surrounding muscle weakens from disuse, increasing joint load and accelerating the cycle.

64. *Compare osteoarthritis and rheumatoid arthritis across at least eight parameters.*

Nature — degenerative versus systemic autoimmune. Primary tissue — cartilage versus synovium. Onset age — over forty versus thirty to fifty but any age. Symmetry — asymmetrical versus symmetrical polyarticular. Hand distribution — distal interphalangeal and first carpometacarpal versus metacarpophalangeal, proximal interphalangeal and wrist with distal joints spared. Morning stiffness — under thirty minutes versus over sixty. Activity effect — worse with use versus better with movement. Local signs — hard bony enlargement with little warmth versus warm boggy swelling. Systemic features — absent versus prominent. Deformities — Heberden and Bouchard nodes versus ulnar deviation, swan neck and boutonnière. Laboratory — normal versus raised markers with rheumatoid factor.

65. *A client with knee osteoarthritis asks what massage can do for her. What is your answer?*

That it can reduce the muscular pain, guarding and stiffness that accompany the arthritic joint, which is frequently the larger part of what she actually feels day to day; support range of motion within pain-free limits; address the compensation the knee is causing at the hip, the back and the other leg; and help her stay active and comfortable. It cannot regenerate cartilage or reverse the joint changes, and it would be dishonest to imply otherwise. The most useful thing you can tell her is that exercise has the strongest evidence of any intervention for osteoarthritis, and that your work supports rather than replaces it.

66. *Why does radiographic severity correlate so poorly with symptoms in osteoarthritis?*

Because the tissue that is visibly damaged on the image — cartilage and bone — is not the tissue generating the pain. Pain comes from the subchondral bone, capsule, synovium and surrounding muscle, and the contribution of each varies between people. Many people have marked radiographic change and no symptoms; others have severe pain with modest change. The clinical consequence is to treat the person and their function rather than the image, and not to let a client's report be discounted because their scan looks unimpressive.

F3. Crystal and inflammatory arthropathies

- 67. Describe the presentation of acute gout, name the joint most commonly involved first, and state the action required.**

Abrupt onset, often overnight, of intense pain in a single joint, commonly described as the worst pain the client has experienced. The joint is hot, red, grossly swollen and exquisitely tender, often intolerant even of the weight of bedding. The first metatarsophalangeal joint is affected in around half of first attacks — podagra. The action is to withhold local treatment entirely and, on a first presentation, arrange same-day medical assessment, because acute gout cannot be reliably distinguished from septic arthritis without joint aspiration.

- 68. Why must a hot, red, swollen single joint with fever be referred the same day?**

Because septic arthritis cannot be excluded on clinical appearance alone, and septic arthritis destroys articular cartilage within days. The differential includes acute gout and pseudogout, which are far more common but are distinguished definitively only by aspiration and crystal analysis. Given that the consequence of missing the infection is irreversible joint destruction, the presentation is treated as an emergency until proven otherwise.

- 69. Give the six features of inflammatory back pain and explain why recognising them matters.**

Onset before the age of forty-five and gradual in nature; duration exceeding three months; morning stiffness lasting over thirty to sixty minutes; improvement with exercise and no improvement with rest; night pain characteristically in the second half of the night that drives the client to get up and move; and alternating buttock pain. Recognising the pattern matters because the average delay to diagnosis of ankylosing spondylitis is measured in years, early treatment materially alters the course of the disease, and a massage therapist frequently sees these clients before anyone else does.

- 70. Why does a client with established ankylosing spondylitis require particular caution with spinal technique?**

Because a fused spine is both rigid and osteoporotic. The fusion removes the segmental movement that normally distributes load, and the disuse and inflammation together reduce bone density, so fracture risk is markedly increased. A fracture through a fused spine is unstable and dangerous. Forceful technique, mobilisation and traction are therefore contraindicated, and positioning must be adapted since a client with fixed kyphosis may be unable to lie flat.

F4. Degenerative spinal conditions, bone infection and bone tumours

- 71. Name the features of cauda equina syndrome and state the required action.**

Saddle anaesthesia in the perineum, buttocks and inner thighs; bladder dysfunction including difficulty initiating urination, retention, reduced sensation of filling or incontinence; bowel incontinence or loss of sphincter control; recent-onset sexual dysfunction; bilateral leg pain, weakness or numbness; and progressive neurological deficit. The action is to stop treatment and arrange emergency care — an emergency department presentation, not a routine referral — then document the findings, the advice and the actions taken. Delay risks permanent bladder, bowel and sexual dysfunction.

- 72. Describe the pain pattern suggestive of bone malignancy and contrast it with mechanical back pain.**

Malignant bone pain is deep, constant and boring; unrelated to movement or position; not relieved by rest; characteristically worse at night; and progressive rather than fluctuating. It is frequently accompanied by unexplained weight loss, fatigue, fever, or a history of cancer at any time. Mechanical pain by contrast varies with position and load, is eased by rest or by a particular posture, fluctuates over days and weeks, and is not accompanied by constitutional symptoms. The discriminating question is whether anything the client does changes the pain.

- 73. In whom should osteomyelitis be suspected, and how does it present?**

Suspect it in clients with diabetes, particularly with foot ulceration; peripheral vascular disease; immunosuppression; intravenous drug use; recent orthopaedic surgery or a prosthetic joint; or an open fracture or penetrating wound. It presents with deep, constant, localised bone pain unrelieved by rest or position, local warmth, swelling and tenderness, fever, chills and malaise, and reluctance to use or bear weight on the limb. It is an absolute contraindication requiring urgent medical referral.

- 74. How does spinal stenosis present, and what distinguishes it from vascular claudication?**

Spinal stenosis produces neurogenic claudication: leg pain, heaviness or paraesthesia brought on by walking and by standing upright, and relieved by sitting and by lumbar flexion. Clients often report they can push a shopping trolley or cycle much further than they can walk upright, because both involve flexion. Vascular claudication is relieved by standing still regardless of spinal position and is related to the metabolic demand of the muscle rather than to posture. The postural relationship is the discriminator.

F5. Clinical reasoning, red flags and the treatment decision

75. Describe the four stages of the clinical reasoning framework and what each contributes.

History — establishes onset, mechanism, pattern, course, medical and medication background, red flags and the client's goals; aggravating and easing factors are frequently the most informative part. Observation and palpation — establishes whether the client looks well, and what is visible and palpable, always compared with the unaffected side. Testing — active, then passive with end feel, then resisted isometric, then special and neurological tests, differentiating contractile from non-contractile structures. Impression and plan — identifies tissue, stage and driver, screens for contraindications, determines treat, adapt or refer, and sets goals, technique, consent and documentation.

76. Give six red flags spanning at least three body systems.

Constitutional — unexplained weight loss, and fever with night sweats. Pain pattern — constant pain unrelated to movement or position, and severe night pain that wakes the client. Neurological — saddle anaesthesia, and new bladder or bowel dysfunction. Vascular — unilateral calf pain with swelling and warmth. Joint — a single hot, red, exquisitely painful joint with fever. Integumentary — spreading erythema with an advancing edge and systemic illness. A single flag prompts careful questioning; a cluster, or any neurological or bladder finding, means withhold and refer.

77. Distinguish treat, adapt and refer, and give a clinical example of each from this course.

Treat as planned — no red flags and standard technique appropriate; for example a chronic-stage lateral elbow tendinopathy in an otherwise well client. Treat with adaptation — a condition constrains pressure, position, technique or duration but treatment remains beneficial; for example a client with osteoporosis, treated at light to moderate pressure in side-lying with no joint mobilisation. Withhold and refer — an absolute contraindication or red flag cluster; for example a spreading warm erythematous lesion with fever in an immunosuppressed client. Adaptation is much the most common outcome in practice.

78. Why is it said that referral is a demonstration of competence rather than a failure?

Because recognising the limits of your scope, and acting on that recognition promptly and clearly, is one of the most consequential skills a therapist has. A therapist who identifies a red flag, withholds treatment, conveys the appropriate urgency, and documents the finding and the advice has done more for that client than any technique would have. Failing to refer, by contrast, delays the care the client needs and exposes both of you to harm. Referral also does not usually mean losing the client — most conditions requiring referral are later treatable with adaptation.

79. What must a chart note record when you withhold treatment and refer?

The findings that led to the decision, described rather than diagnosed; the decision itself and its rationale; what you advised the client, including the urgency you conveyed — today, this week, or at their convenience; the client's response to that advice; whatever treatment you did provide and any modifications made, with the reason for each; and consent for whatever was done. The record must be sufficient for a colleague who has never met the client to understand what happened and why.

Final revision checklist

Tick each line only when you could explain it aloud to someone else without notes. Reading a line and recognising it is not the same as being able to produce it.

- The four stages of the disease process, and the vocabulary of cause, course and outcome.
- Sign versus symptom; hypertrophy, hyperplasia, atrophy, metaplasia and dysplasia.
- The six links in the chain of infection, and which link a given control measure breaks.
- Standard precautions, the three levels of decontamination, and the sharps exposure protocol in order.
- The five cardinal signs of inflammation, and the three classes of contraindication.
- Epidermal layers, where mitosis occurs, and what the dermis contains.
- The six functions of skin, including the vitamin D link to osteomalacia.
- The changes of ageing skin, and the direction of each.
- Primary versus secondary lesions, with the full terminology.
- Cellulitis, impetigo, herpes zoster, the tinea group, scabies — presentation and action for each.
- Atopic dermatitis versus psoriasis; why psoriasis is not a contraindication over intact plaques.
- The three healing sequences — wound (4 phases), soft tissue (3 stages), fracture (4 stages) — kept apart.
- Hypertrophic scar versus keloid; burn classification and why full thickness is painless.
- The three skin cancers, and the ABCDE rule with evolution as the most significant criterion.
- Collagen, elastin, ground substance, fibroblasts; creep and why sustained load matters.
- Autoimmunity, the shared features, and what long-term corticosteroid use predicts.
- Lupus, CREST in full, Raynaud phenomenon and its hydrotherapy implications.
- Rheumatoid arthritis — synovium as primary target and everything that follows.
- Marfan, osteogenesis imperfecta, Ehlers-Danlos; the Beighton score and why not to stretch.
- Central sensitisation, allodynia, hyperalgesia and the fibromyalgia treatment approach.
- The three stages of soft-tissue healing with timeframes, goals and permitted technique.
- Strain versus sprain; the three grades; the pain paradox in a grade 3 sprain.
- Why proprioceptive loss explains ankle sprain recurrence.
- Myositis ossificans and why early deep work on a contusion is contraindicated.
- Delayed onset muscle soreness versus strain — onset pattern and focality.
- Tendinopathy versus tendinitis, and why the change in terminology reverses the management.
- Bursitis versus tendinopathy — passive compression versus resisted contraction.
- Acute compartment syndrome, why not to elevate, and how the chronic exertional form differs.
- Selective tissue tension testing — what each of the three tests loads.
- Normal and abnormal end feels, and what an empty end feel means.
- Bone composition, the three cell types, and Wolff's law with its consequences.
- Why trabecular sites fail first in osteoporosis.
- Fracture classification by severity, direction, completeness and mechanism.
- The four stages of fracture healing, and the stress fracture pattern with its referral.
- Fracture complications, the five Ps, and which indicate emergency.
- Massage during immobilisation and after cast removal.
- Osteoporosis versus osteomalacia versus Paget disease.
- The six osteoporosis modifications, and adapting on risk factors rather than diagnosis.
- Functional versus structural deviation; the Adams forward bending test.
- Short and lengthened groups in hyperkyphosis, hyperlordosis and flatback.
- Why posture is a weak predictor of pain, and what you must never promise.
- Articular cartilage — avascular, aneural, alymphatic — and the three consequences.
- The inflammatory versus degenerative pattern, on at least six parameters.
- Osteoarthritis pathophysiology in six steps, and where the pain actually comes from.

- Osteoarthritis versus rheumatoid arthritis across at least eight parameters.
- Gout, pseudogout, and why a hot red single joint with fever is a same-day referral.
- The six features of inflammatory back pain and why recognising them matters.
- Degenerative spinal conditions, and how stenosis differs from vascular claudication.
- Cauda equina syndrome — the features and the emergency action.
- Osteomyelitis — who is at risk and how it presents.
- The pain pattern of bone malignancy, contrasted with mechanical pain.
- The four-stage reasoning framework, the red flag screen, and treat / adapt / refer.
- What a chart note must record when you withhold treatment and refer.

A note on how to revise

- Weight your time by the marks. Sections D, E and F carry 32 of the 60 marks and have never been examined before. Sections A to C carry 28 and you have already been tested on them once.
- Learn the three healing sequences side by side, on one page, in one sitting. Wound healing has four phases, soft-tissue healing has three stages, fracture healing has four stages. Confusing them is one of the most reliable ways to lose marks on this paper.
- Learn the osteoarthritis versus rheumatoid arthritis comparison cold. It is the single most examinable table in the course and it draws on both Section C and Section F.
- For every red flag, be able to say which system it belongs to, what it might indicate, and how urgently you would act. The Section F items ask exactly that.
- Practise applying, not reciting. Take any condition and ask: what stage is this, what class of contraindication, what would I change, and what would I say? The applied items are built on those four questions.
- Retrieval beats rereading, and spaced practice beats a single long session. Write your answers out rather than recognising them on the page.
- If a topic will not stick, teach it to somebody. Explaining aloud exposes gaps immediately.