

RUHANI COLLEGE — Registered Massage Therapy Diploma Program

QUIZ 2 STUDENT STUDY GUIDE

RU RMT 24 — Neurology 3 | 10 Days | 70 Hours

Issued to all students in both the in-person and the live Zoom cohort. This guide is a study aid, not the examination. Everything taught in class, in the lecture decks, in the student handouts, in the laboratory sessions and in the Course Worksheet remains examinable whether or not it appears here.

1. What This Guide Covers

Assessment	Quiz 2
Format	40 multiple-choice items, 40 marks, 60 minutes, closed book.
Weight	20% of the final grade.
When	Day 8, final block.
Coverage	Lectures 5 to 7, Modules 21 to 35 — neurological assessment for the RMT; degenerative, demyelinating, movement and infectious disorders; and nervous system injuries and congenital conditions.
Structure of this guide	A numbered topic for each block of taught content. Each topic gives an explanation followed by short-answer review questions. The answers, with a discussion of why each is correct, are gathered in the answer key at the end. There are no multiple-choice questions in this guide — answering in your own words is a better preparation for the paper than recognising a right option.

2. How to Use This Guide

1. Read the topic explanation first, with the lecture deck and your student handout beside you. This guide summarises and connects; the deck is the taught content and the handout carries the detail.
2. Answer every review question in writing, in your own words, before you look at the answer key. Recognition feels like knowing and is not. Writing an answer exposes the gap.
3. Then read the answer and the discussion. Where your answer differed, go back to the topic and the deck rather than simply memorising the model answer.
4. Work through the Course Worksheet again. It is ungraded but everything in it is examinable, and its matching and short-answer sections cover the factual detail this guide summarises.
5. Revise in short spaced sessions rather than one long one, and sleep between them. That is not study advice borrowed from elsewhere — it is the neuroplasticity and consolidation material of this course applied to your own learning.
6. Bring questions to the examination review session. It is scheduled precisely so that gaps can be closed before the paper rather than discovered during it.
7. Practise the screen itself, not only the facts about it. Roughly a third of this paper is drawn from Lecture 5, and the items are written for a student who has performed the tests in the laboratory rather than only read about them. Test a partner, say the grading aloud, and write the finding as you would in a record.

3. Where the Marks Sit

The paper samples the taught content in proportion to teaching time. Use this to decide where to spend your revision, not to decide what to skip — every section carries items.

Section	Items	Weight of the paper
A — Lecture 5, Modules 21 to 25 (neurological assessment)	15	37.5%
B — Lecture 6, Modules 26 to 30 (degenerative, demyelinating, movement, infectious)	13	32.5%
C — Lecture 7, Modules 31 to 35 (injuries and congenital conditions)	12	30.0%
Total	40	100%

4. Topics

Topic 1 — Screening is not diagnosing: where the neurological screen sits in scope

Modules M21.

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The CMTO Standard of Practice on Client-centred Care requires the RMT to assess the client to determine their condition and whether massage therapy is indicated. Assessment is therefore not merely permitted — it is expected. What is not permitted is communicating a diagnosis, which is a controlled act under the Regulated Health Professions Act, 1991 and is not authorised to massage therapists.

The working distinction is this. Screening asks whether there is a neurological problem, roughly where it sits, whether it is safe to treat and whether referral is needed. Diagnosing asks what disease is causing it and names that disease to the client. The RMT does the first, records it objectively, and refers. Naming a condition — including ruling one out — is the second and is outside scope.

The neurological screen is not a separate examination bolted onto the assessment. It is a set of tests added when the history or observation gives a reason to add them: numbness, tingling, burning or altered sensation; weakness, clumsiness, dropping objects or tripping; radiating pain into a limb; recent head or neck trauma; dizziness or visual change; and any cranial nerve symptom. In every low back client, saddle sensation and bladder and bowel function are asked about regardless.

Review questions — Topic 1

- 1.1. State the difference between screening and diagnosing, and cite the authority for each.
- 1.2. List five findings in the history that should prompt you to add a neurological screen.

Topic 2 — The twelve cranial nerves — type, function and the standard bedside test

Modules M22.

Twelve paired nerves, numbered I to XII from rostral to caudal. They emerge from the brain and brainstem but belong to the peripheral nervous system. Most supply the head and neck; the vagus is the major exception, reaching the thorax and abdomen. Each carries one or more fibre types — sensory, motor, parasympathetic — and four carry parasympathetic fibres: III, VII, IX and X.

The twelve, with the test

- I Olfactory — smell. Sensory. Test each nostril separately with a familiar non-irritant odour.
- II Optic — vision. Sensory. Test acuity and confrontation visual fields.
- III Oculomotor — four extraocular muscles, eyelid elevation, pupil constriction, accommodation. Motor and parasympathetic.
- IV Trochlear — superior oblique; the eye moves down and in. Motor.
- V Trigeminal — facial sensation in three divisions and the muscles of mastication. Both. Test light touch over forehead, cheek and jawline, and resisted jaw clench.
- VI Abducens — lateral rectus; abduction. Motor. Mnemonic LR6 SO4.
- VII Facial — muscles of facial expression, taste from the anterior two-thirds of the tongue, lacrimal and salivary glands. Test raised eyebrows, tight eye closure, smile and cheek puff.
- VIII Vestibulocochlear — hearing and balance. Sensory. Gross hearing, then Rinne and Weber if indicated.
- IX Glossopharyngeal — pharyngeal sensation, taste posteriorly, parotid gland, carotid body and sinus afferents. Tested with X.
- X Vagus — pharynx, larynx and the viscera to the splenic flexure; roughly 75% of all parasympathetic fibres. Test palate elevation and voice quality.
- XI Accessory — trapezius and sternocleidomastoid. Motor. Resisted shoulder shrug and resisted head rotation.
- XII Hypoglossal — tongue muscles. Motor. Protrusion; deviation is toward the weak side.

The two rules that are always examined

- The forehead rule — the forehead receives bilateral cortical innervation, so a central (upper motor neuron) lesion spares it and a peripheral facial nerve lesion such as Bell's palsy does not. A new facial droop that spares the forehead is treated as a possible stroke.
- Accessory nerve relevance — XI supplies the two muscles most frequently treated in this profession. Weakness or wasting of upper trapezius is a neurological finding, not tightness.

Review questions — Topic 2

- 2.1. Which cranial nerves are sensory only, motor only, and mixed? Which four carry parasympathetic fibres?
- 2.2. Explain the forehead rule and state what it distinguishes.
- 2.3. On protrusion the tongue deviates to the left. Where is the lesion and why?

Topic 3 — Deep tendon reflexes — technique, grading, roots and interpretation

Modules M23.

The tendon is tapped to briefly stretch the muscle spindle, producing a monosynaptic reflex contraction. Technique matters: the limb must be relaxed and positioned symmetrically, because position materially changes amplitude, and the muscle should be slightly stretched. Where a reflex appears absent, reinforcement — the Jendrassik manoeuvre, or clenching the jaw — is used before recording it as absent.

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The scale runs 0 to 4+. Zero is absent even with reinforcement and is abnormal. 1+ is diminished, or present only with reinforcement. 2+ is normal. 3+ is brisker than average and may be normal in an anxious client or may be an early upper motor neuron sign. 4+ is very brisk with clonus and is abnormal. Some sources extend to 5+ for sustained clonus; the four-point scale above is the one used in this course.

The single most important interpretive rule is that symmetry matters more than the absolute grade. Amplitude varies widely between individuals and with position, relaxation, anxiety and examiner technique. A symmetrically brisk pair is usually normal; an asymmetrical pair is a finding.

Reflexes and their principal roots

- Biceps — C5 and C6, principally C5. Musculocutaneous nerve.
- Brachioradialis — C5 and C6, principally C6. Radial nerve.
- Triceps — C6, C7 and C8, principally C7. Radial nerve.
- Patellar — L2, L3 and L4, principally L4. Femoral nerve.
- Achilles — S1. Tibial nerve.
- There is no routinely testable L5 reflex — L5 is confirmed by dermatome and myotome instead.

What diminishes a reflex

- Lower motor neuron lesions at root, plexus or peripheral nerve.
- Sensory neuron problems, since the afferent limb is required.
- Neuromuscular junction disease such as myasthenia gravis.
- Primary muscle disease.
- Acute upper motor neuron lesions during spinal shock — absent initially, brisk later.
- Chronic upper motor neuron lesions and anxiety make reflexes brisker, not weaker.

Review questions — Topic 3

- 3.1. State the deep tendon reflexes tested in this course and their principal nerve roots.
- 3.2. Why does symmetry matter more than the absolute grade?
- 3.3. A client's reflexes are absent below the level of an acute cord injury sustained four days ago. Does this exclude an upper motor neuron lesion?

Topic 4 — Sensory testing, dermatomes, myotomes and the grading of strength

Modules M24.

Sensory testing is explained and demonstrated on an unaffected area first, so the client knows what normal feels like, then performed with the eyes closed. Compare side to side at the same point, and ask “does this feel the same on both sides?” rather than “can you feel this?” — because a reduced sensation is still a sensation and the second question returns a yes. Move from an area of reduced sensation outward to normal; the boundary is easier to detect in that direction.

Light touch and sharp-versus-dull test the two main ascending pathways. Vibration is a dorsal column modality, tested with a 128 Hz tuning fork on a bony prominence, and is often the earliest modality lost in peripheral neuropathy. Proprioception is tested by moving a distal joint through small ranges with the client's eyes closed and asking for the direction.

Myotome testing holds each contraction against steady resistance for about five seconds, because fatigable weakness only appears with sustained effort. Strength is graded 0 to 5: 0 no contraction; 1 a flicker; 2 movement with gravity eliminated; 3 movement against gravity but not resistance; 4 movement against gravity and some resistance, often subdivided; 5 normal power. Most clinically relevant weakness in massage therapy practice sits at grade 4 and is detected by comparing sides.

Upper limb myotomes

- C1–C2 neck flexion · C3 lateral neck flexion · C4 shoulder elevation · C5 shoulder abduction · C6 elbow flexion and wrist extension · C7 elbow extension and wrist flexion · C8 thumb extension and finger flexion · T1 finger abduction and adduction.

Lower limb myotomes

- L1–L2 hip flexion · L3 knee extension · L4 ankle dorsiflexion · L5 great toe extension · S1 ankle plantarflexion and eversion, and hip extension · S2 knee flexion · S3–S4 pelvic floor and bladder, assessed by history not by manual testing.
- Plantarflexion is very strong — test it functionally with repeated single-leg heel raises, not by manual resistance.

Review questions — Topic 4

- 4.1. Why is “does this feel the same on both sides?” a better question than “can you feel this?”
- 4.2. How is the S1 myotome tested, and why is manual resistance inadequate?
- 4.3. A client can move the shoulder through full range against gravity but not against your resistance. What grade is this?

Topic 5 — Putting it together — the limb screens, pattern interpretation and referral

Modules M25.

A complete upper limb screen takes about five minutes and is appropriate for any client with neck pain radiating into the arm: observe muscle bulk at the thenar eminence, first dorsal interosseous, deltoid and shoulder girdle; test dermatomes C4 to T1 by light touch comparing sides; test myotomes C4 to T1 against resistance; test the biceps, brachioradialis and triceps reflexes; and add neurodynamic testing if indicated. The lower limb screen observes gait, calf and quadriceps bulk and the ability to rise from a chair without the arms; asks directly about saddle sensation and bladder and bowel function; tests dermatomes and myotomes L1 to S2; and tests the patellar and Achilles reflexes plus the plantar response.

Three patterns must be told apart. A peripheral nerve lesion confines sensory loss to that nerve's cutaneous territory and weakness to the muscles it supplies. A nerve root lesion produces a dermatomal sensory band, myotomal weakness crossing several peripheral nerves, and diminution of the one reflex that root carries. A central lesion produces upper motor neuron signs below the level — spasticity, hyperreflexia, a positive Babinski — rather than a single dermatome and myotome.

Documentation records what was tested, how, and what was found, including negative findings, using the standard scales and without a diagnosis. The referral letter carries the same content plus the reason for referral. The referral conversation is calm and plain: say what you found, say that it needs a doctor's opinion, and do not name a condition.

The consolidated emergency findings

- Saddle anaesthesia, new bladder or bowel change, or bilateral leg symptoms — cauda equina syndrome. Same-day emergency assessment.
- Sudden facial droop, arm weakness or speech difficulty — suspected stroke. Call emergency services and note the time of onset.
- Worsening headache with vomiting, increasing drowsiness or unequal pupils after head injury — rising intracranial pressure. Emergency services.
- Pounding headache with hypertension in a client injured at or above T6 — autonomic dysreflexia. Sit upright and call for help.
- Fever with severe headache and neck stiffness — suspected meningitis. Emergency assessment.
- Before cervical treatment, screen the five D's and three N's: dizziness, drop attacks, diplopia, dysarthria, dysphagia, nausea, numbness, nystagmus.

Review questions — Topic 5

- 5.1. A client has numbness in the thumb and index finger, worse at night, with neck pain. Which findings would point to a C6 root problem rather than carpal tunnel syndrome?
- 5.2. Write an acceptable record of a reduced L5 sensory finding.

Topic 6 — Categorising pathology, and the general principle for progressive disease

Modules M26.

The category a condition belongs to predicts the shape of the treatment plan before you know anything else about the individual. Progressive degenerative conditions lose neurons that will not be recovered — ALS, Alzheimer's, Huntington's. Demyelinating conditions damage myelin, often with relapse and remission — multiple sclerosis, Guillain-Barré syndrome. Movement disorders arise from the basal nuclei and cerebellum — Parkinson's disease, dystonia, tremor. Infectious conditions are acute, may be transmissible, and are contraindicated while active. Traumatic and congenital conditions produce a static lesion with changing musculoskeletal consequences.

For progressive disease the general principle is that massage therapy is supportive and palliative. It will not alter the disease course, and the plan must say so honestly. What it genuinely contributes is comfort, maintenance of range, prevention or delay of contracture, support for circulation and skin integrity, and the value of consistent, unhurried care. An honest, in-scope statement is: "I can't change the condition, but I can make you more comfortable, help keep your joints moving, and give you an hour where something is being done for you."

Overstating the benefit is a professional misrepresentation; dismissing it is inaccurate and denies the client legitimate care. Both are examinable errors.

Review questions — Topic 6

- 6.1. Name the five categories of neurological pathology used in this course and give one example of each.
- 6.2. What may an RMT honestly claim for a client with a progressive degenerative condition?

Topic 7 — Amyotrophic lateral sclerosis, Huntington's disease and the dementias

Modules M27.

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Amyotrophic lateral sclerosis degenerates both upper and lower motor neurons — the only common condition to do both simultaneously. Corticospinal loss produces spasticity and hyperreflexia; anterior horn loss produces flaccidity, atrophy and fasciculations, and the two coexist in the same client. Bulbar involvement brings dysarthria, dysphagia and eventually respiratory failure. Sensation and cognition are typically preserved, which matters clinically and humanly: the client feels and understands everything.

Huntington's disease is autosomal dominant from a trinucleotide repeat expansion, so each child of an affected parent carries a fifty per cent risk. Degeneration of the basal nuclei, particularly the striatum, produces chorea — rapid, involuntary, irregular movements — with cognitive decline and psychiatric change.

Alzheimer's disease is the most common cause of dementia. Gross changes are diffuse cortical atrophy, widened sulci and enlarged ventricles, with the hippocampus and medial temporal lobe affected earliest — which is exactly why the first clinical feature is difficulty forming new memories. Early presentation includes short-term memory loss, repeated questions, word-finding difficulty and trouble with complex tasks; insight is often partially retained and the awareness is distressing. The other dementias are worth distinguishing: vascular dementia declines stepwise after small strokes with focal signs; dementia with Lewy bodies has fluctuating cognition, visual hallucinations and parkinsonian features, with antipsychotic medication dangerous in this group; frontotemporal dementia begins earlier with personality change and disinhibition.

Communication technique with a client who has dementia is examinable: approach from the front within their field of vision; say your name and why you are there at every visit; one idea per sentence in ordinary words; allow substantially more time for a response than feels comfortable; and obtain consent from the client wherever they are capable of the decision.

Review questions — Topic 7

- 7.1. Which condition produces both upper and lower motor neuron signs, and why does that matter on assessment?
- 7.2. Why is Alzheimer's disease characterised by early loss of new memory formation specifically?
- 7.3. State four elements of correct communication technique with a client who has moderate dementia.

Topic 8 — Multiple sclerosis, Guillain-Barré syndrome and the demyelinating pattern

Modules M28.

Multiple sclerosis is an immune-mediated attack on central myelin producing plaques of demyelination that slow or block conduction. Because plaques can occur anywhere in the central nervous system, the presentation is variable. Fatigue is the single most commonly reported and most disabling symptom. Optic neuritis — painful loss of vision in one eye — is often the first presentation. Sensory disturbance includes numbness, tingling, band-like sensations and Lhermitte's sign, an electric sensation down the spine on neck flexion. Spasticity and weakness usually follow an upper motor neuron pattern. Diagnosis is made medically on lesions disseminated in space and in time.

Uhthoff's phenomenon is a temporary worsening of neurological symptoms with a rise in core temperature, because raised temperature further slows conduction in already demyelinated fibres. Heat is therefore the single most important treatment modification: keep the room cool, avoid hot packs, hot hydrotherapy, saunas and general heating, and consider cool cloths if the client warms during the session. Fatigue governs session length and timing; treatment is scheduled at the client's better time of day and aims not to leave them depleted.

Guillain-Barré syndrome is an acute immune-mediated demyelinating polyneuropathy of the peripheral nervous system, typically following a gastrointestinal or respiratory infection by one to three weeks. It presents with rapidly progressive, symmetrical, ascending flaccid weakness beginning in the legs, with areflexia as a hallmark. The acute or progressive phase is a medical emergency because of the threat to respiratory function. In recovery, massage therapy is valuable for maintaining range, comfort and circulation.

Review questions — Topic 8

- 8.1. Explain Uhthoff's phenomenon and the treatment modifications that follow from it.
- 8.2. Contrast multiple sclerosis and Guillain-Barré syndrome on three points.

Topic 9 — Parkinson's disease, parkinsonism, dystonia and tremor

Modules M29.

Parkinson's disease is progressive degeneration of dopamine-producing neurons in the substantia nigra pars compacta, removing dopaminergic input to the striatum. The cardinal features are bradykinesia with rigidity and, in most clients, a resting tremor, with postural instability later. Associated features are a flexed stooped posture with forward head, a shuffling festinating gait with reduced arm swing and freezing in doorways, masked facies, micrographia, hypophonia and autonomic involvement including constipation and orthostatic hypotension.

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Rigidity is uniform regardless of the speed of passive movement — lead-pipe, or cogwheel where tremor is superimposed. That is what distinguishes it from spasticity, which is velocity-dependent and rises with the speed of movement. Testing at two speeds separates them at the table.

Parkinsonism describes the syndrome of bradykinesia with rigidity or tremor. Parkinson's disease is the most common cause but not the only one: drug-induced parkinsonism, most often from antipsychotic and antiemetic medication, is common, usually symmetrical and may reverse on withdrawal of the drug.

Dystonia is sustained or intermittent involuntary contraction producing twisting movements and abnormal postures; it may be focal, segmental or generalised, and a sensory trick — light touch to a specific point — may temporarily reduce it. Tremor types are distinguished by when they occur: essential tremor is postural and action, often bilateral and familial, and is the most common movement disorder; parkinsonian tremor is present at rest and reduces with voluntary movement; cerebellar intention tremor worsens as the hand approaches a target.

Review questions — Topic 9

- 9.1. Contrast the tremor of Parkinson's disease with cerebellar intention tremor.
- 9.2. How is rigidity distinguished from spasticity at the treatment table?

Topic 10 — Infections of the nervous system, peripheral neuropathy, and the contraindication summary

Modules M30.

One principle covers every acute infection of the nervous system and is not negotiable: acute infection with fever or systemic illness is an absolute contraindication to massage therapy. Massage increases circulation and can spread infection, it adds physiological demand to a body already under load, and where the infection is transmissible it endangers the therapist and subsequent clients.

Encephalitis is inflammation of the brain tissue itself, most commonly viral, presenting with flu-like symptoms, fever, headache and confusion, progressing to altered consciousness and seizures. Meningitis is inflammation of the meninges; viral meningitis is more common and usually self-limiting, bacterial meningitis less common and potentially rapidly fatal. Fever with severe headache and neck stiffness is a medical emergency.

Herpes zoster is reactivation of latent varicella-zoster virus from the dorsal root ganglia of spinal nerves or from cranial nerve ganglia, travelling down one sensory nerve — which is why the rash is strictly dermatomal and unilateral. A prodrome of burning, itching or deep aching precedes the rash by two to four days. Active vesicles contain live virus and transmit chickenpox, not shingles, to a person without varicella immunity; the client is infectious until every lesion has crusted. Post-herpetic neuralgia is neuropathic pain persisting in the affected dermatome beyond about three months from rash onset, with allodynia characteristic.

Poliomyelitis attacks the anterior horn cells, producing a pure lower motor neuron picture. Post-polio syndrome is new or worsening weakness, muscle and joint pain and profound fatigue developing typically fifteen to thirty years later, thought to result from failure of the enlarged motor units created during the original recovery — so fatiguing work is avoided. Peripheral neuropathy is most commonly caused by diabetes in Canada, with a symmetrical stocking-and-glove distribution beginning distally and vibration sense often lost earliest.

Review questions — Topic 10

- 10.1. For which conditions in this topic is massage an absolute contraindication, and what is the response?
- 10.2. Why is the rash of herpes zoster strictly dermatomal and unilateral?
- 10.3. A non-immune colleague asks whether they can catch shingles from a client with active lesions. What is the correct answer?

Topic 11 — Localising the lesion and reading spinal cord injury

Modules M31, M32.

Before planning treatment, establish where in the nervous system the problem sits. Four patterns: a peripheral nerve lesion follows the territory of one named nerve, motor and sensory; a nerve root lesion follows a dermatome and a myotome with one diminished reflex; a cord lesion produces a level below which function changes, with upper motor neuron signs below and possible autonomic involvement; a brain lesion produces contralateral signs, often with cranial nerve, speech, vision or cognitive features.

Three crossings decode every cord lesion. The corticospinal tract crosses at the pyramids, so a cord lesion produces ipsilateral weakness and a brain lesion contralateral weakness. The dorsal columns ascend ipsilaterally and cross in the medulla, so a cord lesion produces ipsilateral loss of vibration and proprioception. The spinothalamic tract crosses within one to two segments of entry, so a cord lesion produces contralateral loss of pain and temperature.

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Spinal cord injury level predicts function. C1 to C3 removes respiratory function, since the phrenic nerve arises from C3, C4 and C5. C4 largely preserves the diaphragm but not the limbs. C5 preserves shoulder abduction and elbow flexion. C6 preserves wrist extension and therefore a tenodesis grip — a major independence threshold. C7 adds elbow extension and with it transfers and effective wheelchair propulsion. T1 to T6 gives paraplegia with limited trunk control and continuing dysreflexia risk; T6 to T12 progressively better trunk control, sitting balance and cough. Below L1 to L2 the canal contains roots, not cord, so injury there is a lower motor neuron injury producing flaccidity.

The incomplete syndromes are distinguished by pattern. Central cord syndrome — the most common — affects the upper limbs disproportionately, classically after a hyperextension injury in an older adult with cervical spondylosis. Brown-Séquard syndrome is a hemisection with the dissociated pattern described above. Anterior cord syndrome follows occlusion of the anterior spinal artery, losing motor function and pain and temperature while sparing vibration and proprioception, and carries the poorest prognosis. Spinal shock abolishes all reflex activity below the lesion for days to weeks, producing flaccidity regardless of the anatomical lesion.

Secondary complications the RMT must screen for

- Pressure injury — the leading preventable complication, over the sacrum, ischial tuberosities, greater trochanters, heels and scapulae in insensate immobile areas.
- Deep vein thrombosis and pulmonary embolism, particularly in the first months.
- Osteoporosis below the level of injury, developing rapidly.
- Heterotopic ossification, contracture, and shoulder overuse from propulsion and transfers.
- Autonomic dysreflexia at or above T6.

Review questions — Topic 11

- 11.1. Why does injury at C6 represent a major functional threshold?
- 11.2. A client is four days post cord injury with flaccid paralysis and absent reflexes below the lesion. What phase is this and what will change?
- 11.3. How is a spastic limb handled during passive movement, and why?

Topic 12 — Radiculopathy, cauda equina, stroke, brain injury and complex regional pain syndrome

Modules M33, M34.

In disc herniation the nucleus pulposus displaces through a weakened or torn anulus fibrosus — bulge, protrusion, extrusion, sequestration — most commonly at L4-L5 and L5-S1. Position for comfort rather than convention: side-lying with support is frequently best tolerated in acute lumbar herniation, and the client's preferred position is clinical information. The cauda equina red flags are screened at every visit.

Cervical radiculopathy by level: C5 lateral shoulder pain, weak abduction, diminished biceps reflex; C6 lateral forearm, thumb and index finger, weak elbow flexion and wrist extension, diminished brachioradialis reflex; C7 posterior arm and middle finger, weak elbow extension and wrist flexion, diminished triceps reflex; C8 medial forearm and little finger with weak finger flexion. Lumbar radiculopathy: L3 and L4 anterior thigh and medial leg with weak knee extension and a diminished patellar reflex; L5 lateral leg and dorsum of foot with weak great toe extension and no reliable reflex change; S1 posterior leg, lateral foot and heel with weak plantarflexion and a diminished Achilles reflex.

Stroke is ischaemic in roughly four cases in five — thrombotic, embolic, lacunar or watershed — and haemorrhagic in the remainder. FAST is the recognition tool: face droop, arm drift, speech difficulty, time to call. Note the time symptoms began, because treatment eligibility depends on it. Hypertension is the single largest modifiable risk factor. In rehabilitation, massage therapy fits from the subacute phase onward: reduce spasticity-related discomfort, maintain range, prevent contracture, and support the subluxed or at-risk hemiplegic shoulder — never pull on, hang from or forcefully abduct that arm.

Traumatic brain injury has primary injury at impact and secondary injury over hours to days from oedema, raised intracranial pressure, ischaemia and inflammation. Coup-contrecoup and diffuse axonal injury are the mechanisms named in this course. The deterioration red flags are worsening headache, repeated vomiting, increasing drowsiness, unequal pupils, new weakness or seizure — emergency services, not a referral.

Complex regional pain syndrome is chronic pain grossly out of proportion to the inciting event, usually in a limb after fracture, sprain, surgery or immobilisation, with sensory, vasomotor, sudomotor and trophic changes. Type 1 has no identifiable nerve injury; type 2 follows a defined nerve injury. Treatment begins proximally or on the contralateral limb, well away from the painful region, with very light contact, progressing toward the affected area across sessions rather than within one.

Review questions — Topic 12

- 12.1. A client has weak great toe extension and reduced sensation on the dorsum of the foot, with a normal Achilles reflex. Which root is involved and why is the reflex normal?
- 12.2. State the FAST signs and the one additional piece of information that must be recorded.

12.3. Why does treatment for complex regional pain syndrome begin away from the painful limb?

Topic 13 — Spina bifida and cerebral palsy

Modules M35.

Spina bifida is a neural tube defect. Occulta is a vertebral arch defect without protrusion of neural tissue, often asymptomatic and sometimes marked by a tuft of hair or a dimple. Meningocele protrudes the meninges without the cord. Myelomeningocele includes neural tissue in the sac and produces the most significant deficit, with motor and sensory loss below the lesion and frequently bladder and bowel involvement and associated hydrocephalus.

The precaution specific to this population is latex allergy, whose rate is markedly elevated — attributed to repeated early exposure through multiple surgeries and catheterisation. Reactions range from contact dermatitis to anaphylaxis, so latex-free gloves and equipment are used and the allergy is confirmed and recorded at intake. Insensate skin and pressure-area risk are the other core considerations.

Cerebral palsy is a group of permanent disorders of movement and posture arising from a non-progressive injury to the developing brain, before, during or shortly after birth. The brain lesion does not progress — but its musculoskeletal consequences do change over a lifetime: contracture, joint deformity, pain and the effects of compensatory movement. Classification is by motor type, most commonly spastic, and by distribution. Treatment goals are reduction of hypertonicity and its accompanying pain, maintenance of range and the delay or limitation of contracture, which is a genuinely valuable long-term contribution.

Review questions — Topic 13

13.1. Which precaution is specific to clients with spina bifida, and why does it arise?

13.2. The brain lesion in cerebral palsy is non-progressive. Why does the clinical picture nevertheless change over a lifetime?

5. Answer Key and Discussion

Work each question before reading the answer. The discussion explains why the answer is correct and, where relevant, why the obvious alternative is not — which is what the examination items are written to test.

Topic 1 — Screening is not diagnosing: where the neurological screen sits in scope

1.1 State the difference between screening and diagnosing, and cite the authority for each.

Screening establishes whether there is a neurological problem, approximately where it sits, whether treatment is safe and whether referral is required; the authority is the CMTO Standard on Client-centred Care, which requires assessment. Diagnosing identifies and names the causative disease; communicating a diagnosis is a controlled act under the Regulated Health Professions Act, 1991 and is not authorised to RMTs. Ruling a condition out is also a diagnostic statement.

1.2 List five findings in the history that should prompt you to add a neurological screen.

Any of: numbness, tingling, burning or altered sensation anywhere; new weakness, clumsiness, dropping objects or tripping; pain radiating into a limb; recent head or neck trauma; dizziness, visual change or balance disturbance; any cranial nerve symptom; and in low back presentations, any saddle or sphincter change — which is asked about whether or not the client volunteers it.

Topic 2 — The twelve cranial nerves — type, function and the standard bedside test

2.1 Which cranial nerves are sensory only, motor only, and mixed? Which four carry parasympathetic fibres?

Sensory only: I, II and VIII. Motor only: III, IV, VI, XI and XII. Mixed: V, VII, IX and X. Parasympathetic fibres are carried by III (ciliary ganglion), VII (pterygopalatine and submandibular ganglia), IX (otic ganglion) and X.

2.2 Explain the forehead rule and state what it distinguishes.

The forehead has bilateral cortical innervation, so an upper motor neuron lesion above the facial nucleus leaves it working and the client can still raise that eyebrow. A lower motor neuron lesion of the facial nerve itself weakens the whole half of the face including the forehead. It therefore distinguishes a central cause such as stroke from a peripheral cause such as Bell's palsy — and the central one is an emergency.

2.3 On protrusion the tongue deviates to the left. Where is the lesion and why?

A left hypoglossal lower motor neuron lesion. Each genioglossus pushes the tongue to the opposite side; when one is weak, the intact muscle pushes unopposed and the tongue deviates toward the weak side. Look also for wasting and fasciculation of the tongue at rest.

Topic 3 — Deep tendon reflexes — technique, grading, roots and interpretation

3.1 State the deep tendon reflexes tested in this course and their principal nerve roots.

Biceps C5, brachioradialis C6, triceps C7, patellar L4 and Achilles S1. Testing all three upper limb reflexes localises the cervical level in a client with arm symptoms; note that L5 has no reliably testable reflex.

3.2 Why does symmetry matter more than the absolute grade?

Because reflex amplitude varies widely between individuals and is influenced by limb position, relaxation, anxiety and examiner technique. A difference between the two sides of the same person, tested in the same position at the same session, is far more informative than the grade taken alone.

3.3 A client's reflexes are absent below the level of an acute cord injury sustained four days ago. Does this exclude an upper motor neuron lesion?

No. During spinal shock all reflex activity below the lesion is temporarily abolished, producing flaccidity and areflexia regardless of whether the lesion is anatomically an upper motor neuron one. Reflexes return over days to weeks and then become brisk. Interpreting the acute picture as a lower motor neuron lesion is a classic error.

Topic 4 — Sensory testing, dermatomes, myotomes and the grading of strength

4.1 Why is "does this feel the same on both sides?" a better question than "can you feel this?"

Because reduced sensation is still sensation, so the second question returns a yes even where the deficit is marked. Inviting a comparison detects the partial reduction that is typical of early sensory change.

4.2 How is the S1 myotome tested, and why is manual resistance inadequate?

By ankle plantarflexion and eversion. Manual resistance is inadequate because plantarflexion is very strong and a genuinely weak calf will still overcome the examiner's hand and be graded normal; repeated single-leg heel raises expose the weakness functionally.

4.3 A client can move the shoulder through full range against gravity but not against your resistance. What grade is this?

Grade 3. Grade 4 would allow some resistance, and grade 2 would require gravity to be eliminated.



Topic 5 — Putting it together — the limb screens, pattern interpretation and referral

- 5.1** *A client has numbness in the thumb and index finger, worse at night, with neck pain. Which findings would point to a C6 root problem rather than carpal tunnel syndrome?*

Sensory change extending proximally up the lateral forearm rather than stopping at the wrist; weakness of elbow flexion and wrist extension, which crosses more than one peripheral nerve; and a diminished brachioradialis reflex. Carpal tunnel syndrome confines change to the median territory distal to the wrist, produces thenar weakness only, and does not alter reflexes.

- 5.2** *Write an acceptable record of a reduced L5 sensory finding.*

Something in the form: "Light touch reduced over the L5 dermatome, right dorsum of foot and first web space, compared with left. Great toe extension 4/5 right, 5/5 left. Achilles reflex 2+ bilaterally. Saddle sensation and bladder function reported normal." It states the test, the method, the finding and the negatives, on the standard scales, with no diagnosis.

Topic 6 — Categorising pathology, and the general principle for progressive disease

- 6.1** *Name the five categories of neurological pathology used in this course and give one example of each.*

Progressive degenerative — ALS. Demyelinating — multiple sclerosis. Movement disorder — Parkinson's disease. Infectious — meningitis. Traumatic or congenital — spinal cord injury or cerebral palsy. The category predicts the shape of the plan before any individual detail is known.

- 6.2** *What may an RMT honestly claim for a client with a progressive degenerative condition?*

Comfort and pain relief; maintenance of joint range and delay of contracture; support for circulation, skin condition and sleep; and the therapeutic value of regular, attentive care. Not modification of the disease course and not restoration of lost function.

Topic 7 — Amyotrophic lateral sclerosis, Huntington's disease and the dementias

- 7.1** *Which condition produces both upper and lower motor neuron signs, and why does that matter on assessment?*

Amyotrophic lateral sclerosis. It matters because the usual interpretive rule — that spasticity and hyperreflexia mean upper motor neuron, and atrophy with fasciculations means lower — breaks down: both appear in the same client and sometimes in the same limb. Recognising the combination is what identifies the pattern.

- 7.2** *Why is Alzheimer's disease characterised by early loss of new memory formation specifically?*

Because the hippocampus and medial temporal lobe are affected earliest, and the hippocampus is required for the formation of new declarative memories. Older memories, already consolidated into distributed neocortical networks, and procedural memories, which depend on the basal nuclei and cerebellum, are comparatively preserved.

- 7.3** *State four elements of correct communication technique with a client who has moderate dementia.*

Approach from the front and within their field of vision; reintroduce yourself and your purpose at every visit; use one idea per sentence in short ordinary sentences; and allow substantially more time for a response than feels natural. Consent is sought from the client wherever capacity for that decision exists.

Topic 8 — Multiple sclerosis, Guillain-Barré syndrome and the demyelinating pattern

- 8.1** *Explain Uhthoff's phenomenon and the treatment modifications that follow from it.*

A rise in core temperature temporarily worsens neurological symptoms in multiple sclerosis, because heat further slows conduction in demyelinated fibres. Modifications: keep the treatment room genuinely cool; avoid hot packs, hot hydrotherapy, saunas and general heating; consider cool cloths; and advise the client that a hot bath or hot weather may transiently worsen their symptoms without indicating a relapse.

- 8.2** *Contrast multiple sclerosis and Guillain-Barré syndrome on three points.*

Location — MS demyelinates the central nervous system, Guillain-Barré the peripheral. Course — MS is typically relapsing and remitting over years, Guillain-Barré is acute and monophasic with recovery over weeks to months. Motor pattern — MS usually produces upper motor neuron signs with spasticity and hyperreflexia, Guillain-Barré produces flaccid weakness with areflexia.

Topic 9 — Parkinson's disease, parkinsonism, dystonia and tremor

- 9.1** *Contrast the tremor of Parkinson's disease with cerebellar intention tremor.*

Parkinsonian tremor is a resting tremor that reduces or disappears when the limb is used. Cerebellar intention tremor is absent at rest and worsens as the hand approaches its target, demonstrated on finger-to-nose testing. Essential tremor, by contrast, is a postural and action tremor, often bilateral and familial.

- 9.2** *How is rigidity distinguished from spasticity at the treatment table?*

By moving the limb passively at two different speeds. Spasticity is velocity-dependent, so resistance rises with speed and may show a clasp-knife give-way. Rigidity is uniform through range and at any speed, felt as lead-pipe resistance or as cogwheeling where a tremor is superimposed.

Topic 10 — Infections of the nervous system, peripheral neuropathy, and the contraindication summary

10.1 *For which conditions in this topic is massage an absolute contraindication, and what is the response?*

Acute encephalitis, acute meningitis, acute poliomyelitis, the acute or progressive phase of Guillain–Barré syndrome, and any acute infection with fever or systemic illness. Local massage over active herpes zoster lesions is also contraindicated. The response is: do not treat, and refer — urgently, since several of these are medical emergencies.

10.2 *Why is the rash of herpes zoster strictly dermatomal and unilateral?*

Because the virus lies latent in the dorsal root ganglion of a single spinal nerve, or in a cranial nerve ganglion, and on reactivation travels down that one sensory nerve to the skin it supplies. The distribution of the rash is therefore the distribution of that nerve.

10.3 *A non-immune colleague asks whether they can catch shingles from a client with active lesions. What is the correct answer?*

They cannot catch shingles, but they can catch chickenpox. Active vesicles contain live virus; in a person without immunity to varicella the primary infection is chickenpox. Shingles only arises from reactivation of the person's own latent virus. The client is infectious until all lesions have crusted.

Topic 11 — Localising the lesion and reading spinal cord injury

11.1 *Why does injury at C6 represent a major functional threshold?*

Because wrist extension is preserved, which permits a tenodesis grip — passive finger flexion produced by extending the wrist. That is the difference between dependence and a substantial degree of self-care and transfer independence. C7 adds elbow extension and with it effective transfers and manual wheelchair propulsion.

11.2 *A client is four days post cord injury with flaccid paralysis and absent reflexes below the lesion. What phase is this and what will change?*

Spinal shock. All reflex activity below the lesion is temporarily abolished, so the presentation is flaccid and areflexic regardless of the anatomical lesion. Over days to weeks reflex activity returns, and where the lesion is an upper motor neuron one, spasticity and hyperreflexia then develop.

11.3 *How is a spastic limb handled during passive movement, and why?*

Slowly, with sustained positioning and warmth, in a calm environment. Spasticity is a velocity-dependent increase in tone from loss of descending inhibition of the stretch reflex, so fast passive movement increases resistance and slow handling reduces it. Anxiety also raises tone, which is why the environment is part of the technique.

Topic 12 — Radiculopathy, cauda equina, stroke, brain injury and complex regional pain syndrome

12.1 *A client has weak great toe extension and reduced sensation on the dorsum of the foot, with a normal Achilles reflex. Which root is involved and why is the reflex normal?*

L5. The Achilles reflex is principally S1, and there is no reliably testable L5 deep tendon reflex — which is why an L5 radiculopathy is confirmed by the dermatome at the dorsum of the foot and first web space together with weakness of great toe extension, rather than by a reflex change.

12.2 *State the FAST signs and the one additional piece of information that must be recorded.*

Face — is it drooping on one side; Arms — does one drift down when both are raised; Speech — is it slurred, jumbled or absent; Time — call emergency services immediately. The additional information is the time the symptoms began, because eligibility for reperfusion treatment depends on it.

12.3 *Why does treatment for complex regional pain syndrome begin away from the painful limb?*

Because allodynia means light touch is genuinely painful and the sensitised system responds to intense or direct input with more pain rather than more relief. Beginning proximally or on the contralateral limb allows a therapeutic input the nervous system reads as safe, and progression toward the affected area occurs across sessions rather than within one.

Topic 13 — Spina bifida and cerebral palsy

13.1 *Which precaution is specific to clients with spina bifida, and why does it arise?*

Latex allergy. Its rate is markedly elevated in this population, attributed to repeated early exposure through multiple surgeries and catheterisation, and reactions range from contact dermatitis to anaphylaxis. Only latex-free gloves and equipment are used, and the allergy status is confirmed and recorded at intake.

13.2 *The brain lesion in cerebral palsy is non-progressive. Why does the clinical picture nevertheless change over a lifetime?*

Because the musculoskeletal consequences progress even though the lesion does not: sustained hypertonicity produces contracture and joint deformity, growth and ageing alter the mechanics, and compensatory movement patterns generate their own pain and overuse. That is exactly why maintenance of range and delay of contracture is a valuable long-term contribution.

6. Final Checklist Before the Paper

- You can state each item of taught content in your own words, not merely recognise it.
- You can explain a mechanism, not only name it — most items on the paper ask why, not what.
- You know the emergency red flags and the required response for each, without hesitation.
- You know the numbers: reflex roots and grading, strength grading, dermatome and myotome landmarks, autonomic outflow levels, and the thresholds that appear in this course.
- You can distinguish the pairs that are easily confused — upper and lower motor neuron, spasticity and rigidity, radicular and peripheral, obstructive and central apnea, anterograde and retrograde amnesia, allodynia and hyperalgesia.
- You know what is inside the scope of practice and what is a controlled act.
- You have re-read the Course Worksheet and your own laboratory notes.
- You have slept. The sleep and memory material in this course applies to you as well: consolidation happens overnight, and a night of sleep after revision outperforms an equivalent period awake.