

RUHANI COLLEGE OF MASSAGE THERAPY

RU RMT 24 · NEUROLOGY 3 · 10 DAYS / 70 HOURS

Lecture 1

Neurology Review and the Neurological Basis of Massage Therapy

Consolidating Neurology 1 and Neurology 2 · Why the nervous system governs every treatment decision

- Reviews the neuroanatomy and neurophysiology taught in RU RMT 11 (Neurology 1) and RU RMT 16 (Neurology 2)
- Establishes the five neurological mechanisms through which massage therapy produces its effects
- Introduces the regulatory frame for neurological treatment planning: Massage Therapy Act, 1991 and CMTO Standards of Practice
- Primary clinical reference for the course: Rattray & Ludwig, Clinical Massage Therapy (Talus, 2000)

Ten instructional days of seven hours — 70 hours in total. Delivered in person at Ruhani College, Brampton, and live online via Zoom; all hands-on laboratories are on campus and mandatory.

Days 1–5

- Day 1 (7 h) — Lecture 1. Neurology review and the neurological basis of massage therapy; receptor and technique laboratory.
- Day 2 (7 h) — Lecture 2. Integrative cerebral functions; relaxation-response and home-care laboratory.
- Day 3 (7 h) — Lecture 3. The autonomic and enteric nervous systems; autonomic observation and abdominal massage laboratory.
- Day 4 (7 h) — Lecture 4. Autonomic reflexes and disorders; dysreflexia drill. Quiz 1, Lectures 1–4.
- Day 5 (7 h) — Lecture 5 part 1. Scope, history, observation and the cranial nerves; cranial nerve laboratory.

What you are expected to do

- Complete the worksheet progressively across all ten days rather than in one sitting.
- Attend on campus for every hands-on laboratory session, whichever cohort you are enrolled in.
- Bring the assigned Rattray condition entries to each pathology day.
- Treat every condition studied as a treatment-planning problem, not a memorisation list.
- Use the CMTO Standards of Practice as the test of whether a proposed plan is defensible.

Days 6–10

- Day 6 (7 h) — Lecture 5 part 2. Reflexes, sensation, dermatomes, myotomes and strength; limb screen practicum. Assignment released.
- Day 7 (7 h) — Lecture 6. Degenerative, demyelinating, movement and infectious disorders; spasticity handling laboratory.
- Day 8 (7 h) — Lecture 7. Injuries and congenital conditions; positioning and hemiplegic shoulder laboratory. Quiz 2, Lectures 5–7.
- Day 9 (7 h) — Lecture 8. Neurological treatment planning; treatment-planning practicum. Assignment due.
- Day 10 (7 h) — Integrated case practicum, worksheet due, examination review, and the Final Written Examination.

Assessment components

- Quiz 1 — 30 multiple-choice items, Lectures 1–4, Day 4. Worth 20%.
- Quiz 2 — 40 multiple-choice items, Lectures 5–7, Day 8. Worth 20%.
- Assignment — neurological treatment plan, released Day 6, due Day 9. Worth 10%.
- Final Written Examination — 50 multiple-choice items, Lectures 1–8, Day 10. Worth 40%.
- Professional Practice, Participation and Attendance — continuous observation. Worth 10%. Carries the ungraded but required Course Worksheet.
- Passing standard: 70% of the weighted total, and 70% of the 70 scheduled contact hours.

The Neurology Series: 11 → 16 → 24

Neurology 3 is the final course of the series. It does not introduce a new region of anatomy; it converts what you already know into clinical decisions.

What came before

- RU RMT 11 Neurology 1 — nervous system organisation, the neuron, the action potential, synapses and neurotransmitters, peripheral nerves, the four plexuses and their lesions.
- RU RMT 16 Neurology 2 — the twelve cranial nerves, the spinal cord and its tracts, the brain and its coverings, cortical organisation, somatic sensory and motor pathways.

What Neurology 3 adds

- Integrative brain functions: consciousness, sleep, learning and memory.
- The autonomic and enteric nervous systems in clinical depth.
- Neurological assessment and screening performed by the RMT.
- The major neurological pathologies, each worked through to a treatment plan.
- The reasoning framework that ties assessment, pathology, scope and documentation together.

By the end of Lecture 1, the student will be able to:

- Recall and apply the core neuroanatomy and neurophysiology taught in Neurology 1 and Neurology 2, at the level required to reason about pathology.
- Explain the organisation of the nervous system into central and peripheral, somatic and autonomic, and enteric divisions.
- Distinguish upper motor neuron from lower motor neuron presentations and state why this distinction changes treatment.
- Describe the five neurological mechanisms through which massage therapy produces its therapeutic effects.
- Locate neurological screening within the massage therapy assessment and identify the findings that require referral.
- State the Scope of Practice of Massage Therapy under the Massage Therapy Act, 1991, and the CMTO Standards that govern treatment planning.
- Use the prescribed texts — in particular Rattray and Ludwig — as working clinical references rather than background reading.



The Texts You Will Work From

Primary clinical reference

- Rattray, F., & Ludwig, L. (2000). Clinical Massage Therapy: Understanding, Assessing and Treating Over 70 Conditions. Talus Inc.
- Written for the Ontario 2,200-hour curriculum; 1,178 pages, over 300 illustrations, over 650 cited sources.
- Organised by condition. Each entry gives anatomy and pathology, assessment protocol, a specific treatment plan, and client self-care.
- This structure is the model for the treatment planning you will be assessed on.

Supporting references

- Werner, R. A Massage Therapist's Guide to Pathology — indications and contraindications.
- Salvo, S. G. Mosby's Pathology for Massage Professionals.
- Tortora, G. J., & Derrickson, B. Principles of Anatomy and Physiology — the anatomy and physiology backbone.
- Lowe, W. Orthopedic Assessment in Massage Therapy — assessment technique.
- Fritz, S. Mosby's Fundamentals of Therapeutic Massage.
- CMTO Standards of Practice — the regulatory test applied to every plan.

Rattray, F., & Ludwig, L. (2000). Clinical Massage Therapy: Understanding, Assessing and Treating Over 70 Conditions. Talus Inc. ISBN 978-0-9698177-1-0.

Organisation of the Nervous System

Structural division

- Central nervous system (CNS): the brain and spinal cord — integration and command.
- Peripheral nervous system (PNS): cranial and spinal nerves, ganglia and receptors — the input and output cabling.
- Grey matter is cell bodies, dendrites and synapses; white matter is myelinated axons.
- A ganglion is a collection of cell bodies in the PNS; a nucleus is the CNS equivalent.

Functional division

- Sensory (afferent) — carries information toward the CNS.
- Motor (efferent) — carries commands away from the CNS.
- Somatic motor — voluntary control of skeletal muscle.
- Autonomic motor — involuntary control of smooth muscle, cardiac muscle and glands; sympathetic and parasympathetic divisions.
- Enteric — the intrinsic nervous system of the gastrointestinal tract.

MESSAGE THERAPY APPLICATION

- Every massage technique enters this system at one point only: peripheral sensory receptors. Everything else that follows is the nervous system's own response.

Tortora, G. J., & Derrickson, B. (2020). Principles of Anatomy and Physiology (16th ed.). Wiley.



The Neuron and the Neuroglia

Neuron

- Dendrites receive; the cell body integrates; the axon conducts; the axon terminal transmits.
- Structural classes: multipolar, bipolar, unipolar (pseudounipolar).
- Functional classes: sensory, motor, interneuron — interneurons are the great majority.
- Neurons are amitotic: with limited exceptions they do not divide, which is why nervous tissue damage is so consequential.

Neuroglia

- CNS: astrocytes (blood–brain barrier, metabolic support), oligodendrocytes (myelin), microglia (immune), ependymal cells (CSF).
- PNS: Schwann cells (myelin and the neurilemma) and satellite cells.
- The neurilemma exists only in the PNS — this is the single anatomical reason peripheral nerves can regenerate and central axons largely cannot.

Membrane Potential and the Action Potential

- Resting membrane potential is approximately -70 mV, maintained by the sodium–potassium pump moving three sodium ions out for every two potassium ions in.
- Graded potentials are local, variable in size and decremental; they summate toward threshold.
- Threshold is approximately -55 mV. Reaching it opens voltage-gated sodium channels and the action potential fires.
- Depolarisation is sodium influx; repolarisation is potassium efflux; a brief hyperpolarisation follows.
- The action potential is all-or-none: stimulus intensity is coded by firing frequency and by how many fibres recruit, never by a larger impulse.
- During the absolute refractory period no second impulse is possible; during the relative refractory period only a stronger-than-normal stimulus succeeds.

MESSAGE THERAPY APPLICATION

- All-or-none conduction is why increasing pressure does not produce a proportionally larger neural signal — it recruits more receptors and higher-threshold fibres, including nociceptors.

Tortora, G. J., & Derrickson, B. (2020). Principles of Anatomy and Physiology (16th ed.). Wiley.

Conduction Speed, Myelin and Fibre Types

Conduction

- Continuous conduction in unmyelinated fibres is slow; saltatory conduction jumps node to node in myelinated fibres and is fast.
- Speed rises with axon diameter and with myelination, and falls with cooling.
- Demyelination slows or blocks conduction without severing the axon — the mechanism in multiple sclerosis and Guillain-Barré syndrome.

Fibre classes

- A fibres — large, myelinated, fastest. Carry proprioception, discriminative touch, and fast sharp pain (A-delta).
- B fibres — medium, lightly myelinated; autonomic preganglionic.
- C fibres — small, unmyelinated, slowest. Carry dull aching pain, temperature and autonomic postganglionic traffic.

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- Large-diameter A-beta touch and pressure fibres conduct faster than C-fibre pain. This speed difference is the physical basis of the gate control effect described later in this lecture.



The Synapse and Neurotransmission

- Chemical synapses dominate: the impulse reaches the terminal, calcium enters, vesicles fuse and release transmitter into the synaptic cleft.
- Transmitter binds receptors on the postsynaptic membrane, producing either an excitatory postsynaptic potential or an inhibitory one.
- Spatial summation adds input from many terminals at once; temporal summation adds rapid input from one terminal.
- Transmitter action is terminated by diffusion, enzymatic degradation, or reuptake — the target of many medications clients take.
- Circuits diverge to amplify a signal and converge to integrate many signals onto one output neuron.

Neurotransmitters That Matter Clinically

Principal transmitters

- Acetylcholine — the neuromuscular junction, all autonomic ganglia, all parasympathetic targets.
- Norepinephrine and epinephrine — most sympathetic targets and the adrenal medulla.
- Glutamate — the main excitatory transmitter of the CNS; central to sensitisation.
- GABA and glycine — the main inhibitory transmitters.
- Dopamine — motor control via the basal nuclei; depleted in Parkinson's disease.

Modulators of pain and mood

- Serotonin — mood, sleep and descending pain inhibition.
- Endorphins and enkephalins — endogenous opioids; presynaptic inhibition of substance P release.
- Substance P — a nociceptive transmitter in the dorsal horn.
- Each of these appears again in Lecture 8 when technique selection for chronic pain is discussed.

Nerve Injury, Degeneration and Repair

- Chromatolysis: the cell body swells and Nissl bodies disperse after axonal injury — the metabolic shift toward repair.
- Wallerian degeneration: the axon distal to the injury disintegrates and is cleared by macrophages and Schwann cells.
- In the PNS, surviving Schwann cells and the neurilemma form a regeneration tube; axons regrow at roughly one millimetre per day.
- In the CNS there is no neurilemma, oligodendrocytes inhibit regrowth, and astrocytic scarring blocks the path — functional recovery depends on plasticity rather than regrowth.
- Recovery time therefore has an anatomical basis you can explain to a client, and a realistic ceiling you must respect when setting goals.

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- Prognosis language belongs to the referring physician. What the RMT can state is the general biology and the plan: maintain range, protect insensate tissue, support circulation, and reassess.

Sensory Receptors and Proprioceptors

Receptor classes

- Mechanoreceptors — touch, pressure, vibration, stretch.
- Thermoreceptors — warmth and cold.
- Nociceptors — free nerve endings responding to actual or threatened tissue damage.
- Chemoreceptors and photoreceptors complete the set.
- Cutaneous receptors: Merkel discs, Meissner corpuscles, Ruffini endings, Pacinian corpuscles.

Proprioceptors

- Muscle spindle — intrafusal fibres sensing length and rate of change; drives the stretch reflex.
- Golgi tendon organ — at the musculotendinous junction, sensing tension; produces autogenic inhibition.
- Joint kinaesthetic receptors — position and movement sense at the capsule.
- Together these give the position sense that is lost in dorsal column pathology.

Why Receptors Determine Technique Selection

Technique choice is not a matter of preference. Each technique preferentially loads a different receptor population, and therefore produces a different neural response.

Technique and receptor

- Static holds and slow effleurage — Ruffini endings and slowly adapting mechanoreceptors; parasympathetic bias.
- Rhythmic petrissage — broad mechanoreceptor recruitment; tone reduction.
- Vibration and rapid tapotement — Pacinian corpuscles; stimulating and sympathetically arousing.
- Sustained tension at the musculotendinous junction — Golgi tendon organ; autogenic inhibition.
- Passive stretch applied quickly — muscle spindle; provokes the stretch reflex and resistance.

What this means in practice

- A client who needs down-regulation is treated slowly, predictably and without sudden stimulus.
- A client who needs activation before exercise tolerates faster, more varied input.
- Stretch applied fast recruits the very reflex you are trying to quiet; apply it slowly.
- Pressure that recruits nociceptors produces guarding, not relaxation, regardless of intent.

Spinal Nerves, Roots and Rami

- Thirty-one pairs of spinal nerves: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, 1 coccygeal.
- Each nerve forms from a dorsal (sensory) root and a ventral (motor) root; the dorsal root ganglion holds the sensory cell bodies.
- Every spinal nerve is mixed. Within about a centimetre it divides into a dorsal ramus and a ventral ramus.
- Dorsal rami supply the deep back muscles and the skin of the back; ventral rami supply everything else and form the plexuses.
- Cervical nerves C1–C7 exit above their same-numbered vertebra; C8 exits between C7 and T1; from T1 down, nerves exit below their same-numbered vertebra.

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- Herpes zoster travels the dorsal root ganglion of one spinal nerve — which is why its rash is strictly dermatomal and unilateral. Lecture 6 returns to this.

Dermatomes — landmarks worth knowing

- C4 — top of the shoulder. C5 — lateral arm. C6 — thumb. C7 — middle finger. C8 — little finger. T1 — medial forearm.
- T4 — nipple line. T10 — umbilicus. T12 — inguinal region.
- L3 — medial thigh and knee. L4 — medial malleolus. L5 — dorsum of the foot and great toe. S1 — lateral foot and heel.
- S2–S4 — perineum and saddle area; numbness here is a cauda equina red flag.

Myotomes

- C5 shoulder abduction. C6 elbow flexion and wrist extension. C7 elbow extension and wrist flexion. C8 thumb extension. T1 finger abduction.
- L2 hip flexion. L3 knee extension. L4 ankle dorsiflexion. L5 great toe extension. S1 ankle plantarflexion and eversion. S2 knee flexion.
- Dermatomes overlap substantially; myotomes are usually the more reliable of the two on screening.

Structure

- Formed from the ventral rami of C1–C4, lying deep to the sternocleidomastoid.
- Cutaneous branches: lesser occipital, great auricular, transverse cervical and supraclavicular nerves.
- Motor branches supply the infrahyoid muscles through the ansa cervicalis.
- The phrenic nerve arises from C3, C4 and C5 and is the sole motor supply to the diaphragm.

Clinical points

- "C3, 4 and 5 keep the diaphragm alive" — spinal cord injury above C3 threatens respiration.
- Diaphragmatic irritation refers pain to the shoulder tip through the phrenic nerve.
- Cutaneous branches emerge along the posterior border of sternocleidomastoid; deep sustained pressure there can produce referred sensory symptoms.

Roots C5–T1 → trunks → divisions → cords → branches. Randy Travis Drinks Cold Beer.

Organisation

- Trunks: superior (C5–C6), middle (C7), inferior (C8–T1).
- Each trunk splits into anterior and posterior divisions.
- Cords are named for their relation to the axillary artery: lateral, posterior, medial.
- Terminal branches: musculocutaneous, axillary, radial, median, ulnar.

Terminal branch functions

- Musculocutaneous (C5–C7) — anterior arm flexors; lateral forearm sensation.
- Axillary (C5–C6) — deltoid and teres minor; regimental badge sensation.
- Radial (C5–T1) — all elbow, wrist and finger extensors.
- Median (C6–T1) — most forearm flexors, thenar muscles, lateral three and a half digits.
- Ulnar (C8–T1) — flexor carpi ulnaris, most intrinsic hand muscles, medial one and a half digits.

Named presentations

- Erb's palsy — upper trunk C5–C6; the waiter's tip position.
- Klumpke's palsy — lower trunk C8–T1; clawed hand, sometimes with Horner's syndrome.
- Thoracic outlet syndrome — compression of the plexus or subclavian vessels at the scalenes, first rib or pectoralis minor.
- Cervical rib — a supernumerary rib compressing the inferior trunk.

Peripheral nerve signs

- Median at the carpal tunnel — thenar wasting, LOAF weakness, hand of benediction.
- Ulnar at the elbow or wrist — claw hand, loss of finger abduction, Froment's sign.
- Radial in the spiral groove — wrist drop.
- Long thoracic — winged scapula from serratus anterior paralysis.
- Axillary after shoulder dislocation — deltoid weakness with badge-area numbness.

MASSAGE THERAPY APPLICATION

- Thoracic outlet syndrome is one of the most commonly treated conditions in this list — Rattray gives a full assessment and treatment protocol for it.

Rattray, F., & Ludwig, L. (2000). Clinical Massage Therapy: Understanding, Assessing and Treating Over 70 Conditions. Talus Inc. ISBN 978-0-9698177-1-0 — condition entry: Thoracic Outlet Syndrome.

Structure and branches

- Ventral rami L1–L4, formed within the psoas major.
- Iliohypogastric and ilioinguinal (L1) — lower abdominal wall.
- Genitofemoral (L1–L2) and lateral femoral cutaneous (L2–L3).
- Femoral nerve (L2–L4) — iliacus, sartorius, quadriceps; sensation to the anterior thigh and, via the saphenous branch, the medial leg.
- Obturator nerve (L2–L4) — the adductor group and medial thigh sensation.

Clinical points

- Meralgia paraesthetica — entrapment of the lateral femoral cutaneous nerve at the inguinal ligament; burning lateral thigh numbness with no motor loss.
- Femoral nerve involvement abolishes or diminishes the patellar reflex (L4).
- The psoas relationship means deep anterior hip work can affect these nerves; palpate with care and consent.



The Sacral Plexus and the Sciatic Nerve

Branches

- Ventral rami L4–S4.
- Sciatic nerve (L4–S3) — the largest nerve in the body; two nerves in one sheath.
- Tibial division — hamstrings and all posterior leg muscles; branches into medial and lateral plantar nerves.
- Common fibular division — anterior and lateral leg; superficial and deep fibular branches.
- Superior and inferior gluteal, posterior femoral cutaneous, pudendal.

Clinical points

- Common fibular injury at the fibular head produces foot drop and loss of dorsiflexion and eversion.
- Tibial nerve entrapment at the tarsal tunnel gives plantar burning and numbness.
- Superior gluteal nerve injury weakens gluteus medius, producing a Trendelenburg sign.
- Piriformis syndrome and true radicular sciatica must be distinguished on assessment — the treatment plans differ.

Cauda Equina Syndrome — the Emergency You Must Never Miss

Below the conus medullaris at approximately L1–L2, the canal contains only nerve roots. Compression here is a surgical emergency.

Red flags

- Saddle anaesthesia — numbness of the perineum, inner thighs and buttocks (S2–S4).
- New bladder retention, overflow incontinence or loss of bowel control.
- Bilateral leg weakness, numbness or radicular pain.
- New sexual dysfunction.
- Rapidly progressive neurological deficit.

Required response

- Stop the treatment. Do not continue and do not book a review appointment.
- Direct the client to emergency care the same day; time to decompression determines the outcome.
- Document objectively what the client reported and what you observed, and the referral you made.
- Screen for these findings on every low back client, every visit — not only on the first.



The Spinal Cord: Grey and White Matter

Grey matter

- Butterfly-shaped and centrally placed.
- Posterior (dorsal) horn — sensory interneurons receiving afferent input.
- Anterior (ventral) horn — cell bodies of lower motor neurons.
- Lateral horn — present only at T1–L2 and S2–S4; holds autonomic preganglionic cell bodies.

White matter and landmarks

- Organised into posterior, lateral and anterior columns (funiculi) carrying the ascending and descending tracts.
- Cervical and lumbar enlargements supply the limbs.
- The cord ends at the conus medullaris around L1–L2; the filum terminale anchors it.
- Three meninges and the epidural and subarachnoid spaces provide protection; lumbar puncture is performed below the conus.

Dorsal column–medial lemniscus

- Carries discriminative touch, vibration, two-point discrimination and conscious proprioception.
- First-order neuron ascends ipsilaterally in the cord; decussation occurs in the medulla.
- Damage produces loss of position and vibration sense with a positive Romberg's sign — the pattern seen in tabes dorsalis.

Spinothalamic and spinocerebellar

- Spinothalamic (anterolateral) carries pain, temperature, crude touch and pressure.
- It decussates within one or two segments of entry, inside the cord itself.
- Spinocerebellar tracts carry unconscious proprioception to the cerebellum for coordination.
- The different decussation levels explain the crossed findings of Brown-Séquard syndrome in Lecture 7.

- Direct (pyramidal) pathways: lateral corticospinal, anterior corticospinal and corticobulbar. These carry precise voluntary movement.
- Roughly ninety per cent of corticospinal fibres decussate at the pyramids of the medulla — which is why a cortical lesion produces contralateral weakness.
- Indirect (extrapyramidal) pathways: rubrospinal, tectospinal, vestibulospinal and reticulospinal. These govern posture, tone, balance and gross automatic movement.
- The basal nuclei and cerebellum do not project directly to the cord; they modulate the motor cortex and the brainstem nuclei.
- This is why basal nuclei disease produces disordered movement rather than weakness — the deficits in Parkinson's and Huntington's disease.

Upper Motor Neuron vs Lower Motor Neuron

This is the single most useful distinction in clinical neurology for a massage therapist, because it predicts what you will find and what you can change.

Upper motor neuron lesion

- Lesion in the brain or spinal cord, above the anterior horn cell.
- Spastic paralysis with increased tone, velocity-dependent.
- Hyperreflexia; clonus may be present.
- Positive Babinski sign.
- Little or no atrophy early; disuse atrophy later. No fasciculations.
- Causes: stroke, spinal cord injury, multiple sclerosis, cerebral palsy, traumatic brain injury.

Lower motor neuron lesion

- Lesion at the anterior horn cell, root, plexus or peripheral nerve.
- Flaccid paralysis with decreased tone.
- Hyporeflexia or areflexia.
- Marked atrophy, and fasciculations.
- Sensory loss follows the distribution of the damaged nerve or root.
- Causes: peripheral nerve injury, radiculopathy, poliomyelitis, Guillain-Barré syndrome.

MASSAGE THERAPY APPLICATION

- Amyotrophic lateral sclerosis is the important exception: it damages both, so spasticity and atrophy with fasciculations appear together in the same client.

The arc

- Receptor → sensory neuron → integrating centre → motor neuron → effector.
- Monosynaptic reflexes such as the stretch reflex have no interneuron; polysynaptic reflexes such as the withdrawal reflex do.
- Reciprocal inhibition relaxes the antagonist as the agonist contracts.
- Crossed extensor reflexes maintain balance during withdrawal.

Clinically tested reflexes

- Biceps C5–C6; brachioradialis C6; triceps C7.
- Patellar L3–L4; Achilles S1–S2.
- Plantar response — a Babinski sign in an adult indicates upper motor neuron involvement.
- Reflex testing localises the level, and asymmetry matters more than absolute briskness. Lecture 5 covers technique in detail.



The Brain: Lobes and Functional Areas

Lobes

- Frontal — primary motor cortex in the precentral gyrus, premotor and prefrontal areas, Broca's area for speech production.
- Parietal — primary somatosensory cortex in the postcentral gyrus, and the somatosensory association area for stereognosis.
- Temporal — auditory cortex, Wernicke's area for language comprehension, hippocampus for memory.
- Occipital — visual cortex.
- Insula — interoception and visceral sensation.

Organisation

- Both the motor and sensory cortices are somatotopically mapped as a homunculus; the hands and face occupy disproportionate area.
- Lateralisation: language is left-hemisphere dominant in most people; spatial and non-verbal processing is right-dominant.
- The corpus callosum connects the hemispheres.
- Cortical mapping is plastic and reorganises with use, disuse and injury.

Brainstem, Cerebellum and Diencephalon

Brainstem

- Midbrain — cerebral peduncles, corpora quadrigemina, cranial nerves III and IV.
- Pons — bridges the cerebellar hemispheres; cranial nerves V–VIII; pontine respiratory centres.
- Medulla oblongata — pyramidal decussation; cardiovascular, respiratory rhythmicity, vomiting and swallowing centres; cranial nerves IX–XII.
- The reticular formation runs the length of the brainstem and gives rise to the reticular activating system.

Cerebellum and diencephalon

- Cerebellum — coordination, timing, balance and motor learning; lesions produce ipsilateral ataxia and intention tremor.
- Thalamus — the relay for all sensory input except olfaction.
- Hypothalamus — autonomic control, temperature, hunger and thirst, circadian rhythm, endocrine control via the pituitary.
- Epithalamus — the pineal gland and melatonin.

Coverings, Cerebrospinal Fluid and Blood Supply

- Meninges: dura mater, arachnoid mater and pia mater; the subarachnoid space carries cerebrospinal fluid.
- Cerebrospinal fluid is produced by the choroid plexuses, circulates through the ventricles and subarachnoid space, and is reabsorbed at the arachnoid granulations.
- Obstruction produces hydrocephalus and raised intracranial pressure — headache, vomiting, altered consciousness, papilloedema.
- The brain receives blood from the internal carotid and vertebral arteries, which anastomose in the circle of Willis.
- The brain has almost no stored energy reserve: it depends on continuous glucose and oxygen delivery, and tolerates interruption very poorly.
- The blood–brain barrier, formed by astrocyte end-feet and tight junctions, restricts what reaches neural tissue — including many drugs.

MASSAGE THERAPY APPLICATION

- Vertebral artery anatomy underlies the cervical safety cautions: avoid sustained end-range extension with rotation, and screen for the five D's and three N's before neck work.



The Twelve Cranial Nerves at a Glance

I to VI

- I Olfactory — smell (sensory).
- II Optic — vision (sensory).
- III Oculomotor — four extraocular muscles, pupil constriction, accommodation (motor, parasympathetic).
- IV Trochlear — superior oblique (motor).
- V Trigeminal — facial sensation, muscles of mastication (both).
- VI Abducens — lateral rectus (motor).

VII to XII

- VII Facial — expression, taste anterior two-thirds, lacrimal and salivary glands (both).
- VIII Vestibulocochlear — hearing and balance (sensory).
- IX Glossopharyngeal — posterior tongue, gag, parotid gland (both).
- X Vagus — pharynx, larynx, thoracic and abdominal viscera (both).
- XI Accessory — trapezius and sternocleidomastoid (motor).
- XII Hypoglossal — tongue muscles (motor).

The Four Cranial Nerves an RMT Uses Most

Directly treatment-relevant

- Accessory (XI) — motor supply to trapezius and sternocleidomastoid, the two muscles most frequently treated in this profession. Weakness or wasting here is a neurological finding, not tightness.
- Trigeminal (V) — sensation to the whole face and motor supply to masseter, temporalis and the pterygoids; the nerve behind all temporomandibular work.

Screening and effect

- Facial (VII) — the forehead rule distinguishes Bell's palsy from a central lesion such as stroke; new facial droop is always referred.
- Vagus (X) — the principal parasympathetic pathway. The rest-and-digest shift you produce during treatment is largely vagally mediated.
- Full testing protocols for all twelve are covered in Lecture 5.



Five Mechanisms — an Overview

Massage therapy does not act on tissue in isolation. Its effects are produced through the nervous system by five identifiable mechanisms.

- Mechanoreceptor stimulation and segmental pain modulation — the gate control effect.
- Autonomic modulation — a shift from sympathetic toward parasympathetic dominance.
- Alteration of muscle tone through the muscle spindle, the Golgi tendon organ and reciprocal inhibition.
- Descending pain inhibition and the modulation of central sensitisation.
- Neuroplasticity — the structural basis of motor re-learning, postural change and habit formation through home care.
- Each mechanism is examined on the next five slides, and each returns in Lecture 8 when treatment plans are constructed.

Mechanoreceptor Stimulation and Gate Control

- Large-diameter A-beta fibres carrying touch and pressure converge with small C and A-delta nociceptive fibres in the dorsal horn.
- A-beta input excites inhibitory interneurons in the substantia gelatinosa, reducing the onward transmission of nociceptive signals.
- The effect is segmental: input must enter at or near the same spinal level as the pain to close the gate.
- It is also transient. Gate control explains relief during and shortly after treatment; it does not explain lasting change.
- The clinical consequence is that non-painful, broad, rhythmic contact is more effective at modulating pain than painful deep pressure.

MESSAGE THERAPY APPLICATION

- If a client reports that pressure only helps while your hands are on them, that is the gate control effect behaving exactly as expected — and a signal to add the other four mechanisms to the plan.

Rattray, F., & Ludwig, L. (2000). Clinical Massage Therapy: Understanding, Assessing and Treating Over 70 Conditions. Talus Inc. ISBN 978-0-9698177-1-0.

Autonomic Modulation and the Relaxation Response

What is observed

- Reduced heart rate and blood pressure.
- Slower and deeper respiration.
- Reduced resting muscle tone and generalised warmth from vasodilation.
- Improved reported sleep quality and reduced anxiety.
- Clients frequently drift toward sleep — a normal sign of parasympathetic dominance.

How it is produced

- Slow, predictable, rhythmic input with minimal novelty reduces reticular activating system arousal.
- A quiet, warm, dimly lit room removes the sensory triggers that sustain sympathetic tone.
- Pain, cold, sudden contact and unpredictability all reverse the effect immediately.
- Lectures 3 and 4 develop the anatomy and pathology behind this mechanism in full.

Muscle Tone: Spindle, Golgi Tendon Organ and Reciprocal Inhibition

- Resting muscle tone is neurally maintained; a hypertonic muscle is usually a muscle receiving excessive motor drive, not a shortened piece of tissue.
- Rapid stretch excites the muscle spindle and provokes reflex contraction — the opposite of the intended effect.
- Slow, sustained stretch allows spindle adaptation and permits lengthening.
- Sustained tension at the musculotendinous junction loads the Golgi tendon organ and produces autogenic inhibition of that muscle.
- Contracting the antagonist inhibits the agonist through reciprocal inhibition — the basis of muscle energy technique and proprioceptive neuromuscular facilitation.
- Guarding and splinting are protective reflexes; they resolve when the perceived threat resolves, not when force is applied.

Descending Inhibition and Central Sensitisation

Descending inhibition

- The periaqueductal grey and rostral ventromedial medulla project down to the dorsal horn.
- Serotonergic and noradrenergic pathways, and endogenous opioids, suppress nociceptive transmission at the segment.
- This system is influenced by expectation, safety, context and the therapeutic relationship — which is why how you communicate is part of the treatment.

Central sensitisation

- Prolonged nociceptive input increases the excitability of dorsal horn neurons.
- The result is allodynia (pain from non-painful stimuli) and hyperalgesia (exaggerated pain from painful stimuli).
- Pain becomes disproportionate to tissue findings, and spreads beyond the original site.
- This is the shared mechanism of fibromyalgia and complex regional pain syndrome, both revisited later in the course.

MESSAGE THERAPY APPLICATION

- In a sensitised client, more pressure produces more sensitisation. Start well below tolerance, progress across sessions rather than within one, and treat flares as information rather than failure.

Neuroplasticity, Motor Re-learning and Home Care

- The nervous system reorganises structurally in response to use: synapses strengthen, cortical maps shift, and new connections form.
- This is the mechanism by which post-stroke and post-injury rehabilitation works when regeneration is impossible.
- Plastic change requires repetition, attention and relevance — a passive treatment once a week does not supply any of the three.
- The home care programme is therefore not an optional extra; it is the part of the plan that produces durable change.
- Plasticity is not always beneficial: maladaptive plasticity underlies persistent pain, learned guarding patterns and phantom limb pain.

MESSAGE THERAPY APPLICATION

- Design home care for retention: two or three items at most, demonstrated, written down, and reviewed at the next visit. Lecture 2 explains the memory limits behind that rule.

Where the Neurological Screen Fits

Neurological screening is not a separate examination. It is a set of tests you add to the assessment when the history or observation gives you a reason to.

Screen when the client reports

- Numbness, tingling, burning or altered sensation anywhere.
- Weakness, clumsiness, dropping objects or tripping.
- Radiating pain into a limb.
- Recent head or neck trauma.
- Dizziness, visual change, speech change or facial change.
- Bladder, bowel or saddle changes — screen immediately and urgently.

What the screen contains

- Dermatome testing for sensation.
- Myotome testing for strength.
- Deep tendon reflexes for the level and for upper versus lower motor neuron pattern.
- Cranial nerve screening where indicated.
- Balance and coordination where indicated.
- Full technique is taught in Lecture 5.



Scope of Practice of Massage Therapy

Section 3 of the Massage Therapy Act, 1991 defines the practice of massage therapy as the assessment of the soft tissue and joints of the body and the treatment and prevention of physical dysfunction and pain of the soft tissues and joints by manipulation to develop, maintain, rehabilitate or augment physical function, or relieve pain.

What follows from this

- Assessment is explicitly within scope — an RMT is expected to assess, not merely to treat.
- The object of treatment is soft tissue and joints; neurological conditions are treated through their soft tissue and functional consequences.
- Communicating a diagnosis is a controlled act under the Regulated Health Professions Act, 1991 and is not authorised to RMTs.
- You may describe findings; you may not name a disease as the client's diagnosis.

Saying it correctly

- Not: "You have carpal tunnel syndrome."
- Instead: "You have altered sensation in the distribution of the median nerve and weakness of thumb abduction. I am going to write to your physician and ask for an assessment."
- The second version is accurate, useful, documentable and within scope.
- The same construction works for every condition in this course.

This Standard is the direct authority for how a treatment plan is made, changed and ended. Several of its requirements are examinable in this course.

Requirements that govern planning

- Obtain informed consent before assessing, treating, or modifying a treatment plan.
- Assess the client, including health history, to determine the condition and whether massage therapy is indicated.
- Develop a treatment plan based on the assessment and the client's own goals; monitor response and modify accordingly.
- Integrate an evidence-informed approach: professional knowledge, external research, client perspective and practice context.
- Only treat conditions within your competence and within the Scope of Practice.
- Refer, with consent, to the professional whose expertise best addresses the client's needs.

The six elements of consent

- The nature of the treatment.
- The expected benefits.
- The risks and side effects.
- Alternative courses of action.
- The likely consequences of not having treatment.
- The client's right to ask questions, and to have treatment stopped or modified at any time on request.

Safety, Records and the Limits of the Role

Safety and Risk Management

- The practice setting must be maintained safely and sanitarily, and appropriately lit and arranged for privacy and safety.
- Recognise and manage situations that place clients, yourself or others at risk.
- Respond effectively to safety incidents, document them, and disclose to the relevant authorities.
- For neurological clients this covers table safety, transfers, positioning, seizure response and emergency recognition.

Records and referral

- Record keeping requirements cover health history, assessment findings, the treatment plan, treatment provided, consent and outcomes.
- Client health records are retained for ten years after the last visit, or ten years after the client turns eighteen.
- Objective documentation of a neurological finding is what makes a referral credible and defensible.
- Refer with the client's consent, and document what you referred, to whom and why.

Every neurological presentation in this course can be worked through with the same three questions, in this order.

- One — Is this safe to treat today? Screen for the emergencies: cauda equina, acute stroke, rising intracranial pressure, autonomic dysreflexia, acute central nervous system infection, thunderclap headache. If any is present, you refer; you do not treat.
- Two — Where is the lesion, and what does that predict? Central or peripheral; upper or lower motor neuron; which level, root, plexus or nerve. This determines what you will find and what tissue is at risk.
- Three — What can massage therapy legitimately change? Secondary muscular consequences, joint range, circulation, autonomic load, pain modulation and function. It cannot change the lesion itself, and the plan should not imply that it can.
- The answers give you the contraindications, the goals, the techniques, the dosage and the referral — in other words, the treatment plan.

- Neurology 3 converts the anatomy of Neurology 1 and 2 into clinical decisions; the review in this lecture is the working vocabulary for the remaining seven.
- The upper versus lower motor neuron distinction predicts presentation, prognosis and treatment approach more reliably than any other single finding.
- Massage therapy acts on the nervous system through five mechanisms: gate control, autonomic modulation, tone regulation, descending inhibition, and neuroplasticity.
- Technique selection is a receptor-selection decision, not a matter of therapist preference.
- Scope is defined by the Massage Therapy Act, 1991; communicating a diagnosis is a controlled act and is not authorised to RMTs.
- The CMTO Standard of Practice on Client-centred Care sets the requirements for consent, assessment, planning, modification and referral.
- Every case in this course is worked through the same three questions: is it safe, where is the lesion, and what can massage therapy legitimately change.



Review Questions and References

Review questions

- List the six features that distinguish an upper motor neuron lesion from a lower motor neuron lesion.
- Explain the gate control mechanism and state why its effect is segmental and transient.
- Name the five red flags of cauda equina syndrome and state the required response.
- Why can peripheral nerves regenerate when central axons largely cannot?
- State the six elements of informed consent required by the CMTO Standard of Practice.
- Give an in-scope wording for reporting a suspected median nerve compression to a client.

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