



RU RMT 17 Endocrinology

Adrenal Glands, the Stress Axis and Pancreatic Endocrinology

Lecture 05 of 06

Registered Massage Therapy Diploma Program · RU RMT 17 Endocrinology

Aligned to FOMTRAC Inter-Jurisdictional Practice Competencies (2016), CMTO Standards of Practice, and the Regulated Health Professions Act, 1991

Prerequisites: Lectures 01 to 04 — feedback control, gland survey, the pituitary axes and calcium regulation



- **Why these glands share a lecture:**
- Cortisol, epinephrine, glucagon and insulin all act on the same variable — available blood glucose — and all four are central to the body's response to stress and to feeding.
- **Clinical weight:**
- This is the single most important lecture in the course for day-to-day safety. Diabetes mellitus is the endocrine condition you will meet most often, and hypoglycemia and adrenal crisis are the two endocrine emergencies most likely to occur in a clinic.
- **Structure:**
- Adrenal anatomy and hormones, the stress response, adrenal pathology, pancreatic islets, diabetes mellitus, then emergency recognition and treatment planning.
- **Evidence thread:**
- This lecture also addresses what the research does and does not support about massage therapy and cortisol. You are expected to be able to state this accurately to a patient.
- **Safety expectation:**
- By the end of the day you must be able to recognize hypoglycemia and state your immediate response without hesitation.



Learning Objectives (1 of 2)

- By the end of this session, the student will be able to:
 - Describe the location and structure of the adrenal gland and name the three cortical zones and their secretions.
 - Explain the actions of aldosterone and the renin-angiotensin-aldosterone system in blood pressure and electrolyte regulation.
 - Describe the metabolic, anti-inflammatory, cardiovascular and connective tissue effects of cortisol, and its diurnal rhythm.
 - Contrast the acute catecholamine response of the adrenal medulla with the sustained cortisol response of the adrenal cortex.
 - Trace the hypothalamic-pituitary-adrenal axis and explain allostatic load.
 - State accurately what the current evidence does and does not support regarding massage therapy and serum cortisol.



Learning Objectives (2 of 2)

- Describe the clinical features of Cushing's syndrome, Addison's disease, adrenal crisis and pheochromocytoma.
- Explain the effects of exogenous corticosteroid therapy on skin, connective tissue, bone, blood glucose and the patient's own stress axis.
- Describe the cell types of the pancreatic islets and the antagonistic actions of insulin and glucagon.
- Differentiate type 1, type 2 and gestational diabetes mellitus by mechanism, onset and management.
- Identify the acute and chronic complications of diabetes mellitus and their consequences for massage treatment.
- Recognize hypoglycemia and state the correct in-clinic response, and identify when to call emergency services.
- Construct a treatment plan for a patient with diabetes addressing timing, site precautions, pressure, foot care and documentation.

Adrenal and stress

- **Zona glomerulosa:**
 - The outer cortical zone; secretes mineralocorticoids.
- **Zona fasciculata:**
 - The middle and thickest cortical zone; secretes glucocorticoids.
- **Zona reticularis:**
 - The inner cortical zone; secretes adrenal androgens.
- **Glucocorticoid:**
 - A steroid hormone raising blood glucose and suppressing inflammation; cortisol is the principal example.
- **Mineralocorticoid:**
 - A steroid hormone regulating sodium, potassium and water; aldosterone is the principal example.
- **Catecholamine:**
 - Epinephrine or norepinephrine.
- **Allostatic load:**
 - The cumulative physiological cost of repeated or chronic activation of the stress response.

Pancreas and glucose

- **Islets of Langerhans:**
 - The endocrine clusters within the pancreas.
- **Gluconeogenesis:**
 - Formation of glucose from non-carbohydrate sources such as amino acids.
- **Glycogenolysis:**
 - Breakdown of stored glycogen to release glucose.
- **Insulin resistance:**
 - Reduced target-cell response to insulin despite normal or raised insulin levels.
- **Hypoglycemia:**
 - Blood glucose below the normal range; an acute emergency.
- **Diabetic ketoacidosis:**
 - Severe insulin deficiency with acidosis; a medical emergency.
- **Peripheral neuropathy:**
 - Nerve damage producing altered or absent sensation, commonly in the feet.



Section 1

The Adrenal Glands

- Anatomy and the three cortical zones
- Aldosterone and the renin-angiotensin-aldosterone system
- Cortisol and its effects on every tissue you treat
- Adrenal androgens
- The adrenal medulla and the acute stress response



Anatomy of the Adrenal Gland

- **Position:**
- One gland sits on the superior pole of each kidney, retroperitoneally, at approximately the level of the twelfth thoracic vertebra. The alternative name suprarenal reflects this position.
- **Size:**
- Each weighs about four to five grams in the adult; the right is pyramidal and the left more crescent-shaped.
- **Two organs in one:**
- The outer cortex and the inner medulla have different embryological origins, different tissue types, different hormones and different control mechanisms. They happen to occupy the same capsule.
- **The cortex:**
- Derived from mesoderm; glandular tissue producing steroid hormones from cholesterol; makes up about 80 to 90 per cent of the gland; controlled hormonally by ACTH from the anterior pituitary and, for aldosterone, by the renin-angiotensin system.
- **The medulla:**
- Derived from neural crest tissue; functionally a modified sympathetic ganglion; produces catecholamines; controlled neurally by sympathetic preganglionic fibres.
- The three cortical zones, from superficial to deep: zona glomerulosa, zona fasciculata, zona reticularis. A common mnemonic is that the deeper you go the sweeter it gets — salt, sugar, sex.
- **Terminology caution:**
- The outer zone is the zona glomerulosa. The word granulosa refers to the cells surrounding a developing ovarian follicle and belongs to Lecture 06. The two are frequently confused.



Aldosterone and the Renin-Angiotensin-Aldosterone System

- **Source:**
- Zona glomerulosa of the adrenal cortex.
- **Principal action:**
- Increases sodium reabsorption in the distal tubule and collecting duct of the kidney, with water following osmotically, and increases potassium excretion.
- **Net effect:**
- Raises blood volume and blood pressure while lowering plasma potassium.
- **Triggers for release:**
- Falling blood pressure or blood volume, rising plasma potassium, and angiotensin II. ACTH has only a minor influence.
- **The RAAS cascade:**
- Low renal perfusion causes the kidney to release renin, which converts angiotensinogen to angiotensin I, which is converted by angiotensin-converting enzyme to angiotensin II, a powerful vasoconstrictor that also stimulates aldosterone release.
- Pharmacological link you will see on intake forms: ACE inhibitors such as ramipril and angiotensin receptor blockers such as losartan interrupt this cascade to lower blood pressure. Both are extremely common.
- **Practical consequence:**
- Patients on antihypertensive medication are at increased risk of orthostatic hypotension when they sit up or stand after a treatment that has already lowered sympathetic tone. Rise slowly, sit before standing, and supervise the transition.

- **Source and control:**
- Zona fasciculata, under the control of ACTH from the anterior pituitary, which is itself driven by hypothalamic CRH.
- **Metabolic action:**
- Raises blood glucose by stimulating gluconeogenesis in the liver, reducing glucose uptake by peripheral tissues, mobilizing fatty acids from adipose tissue and breaking down muscle protein for amino acid substrate.
- Anti-inflammatory and immunosuppressive action: Reduces production of inflammatory mediators, stabilizes lysosomal membranes, reduces capillary permeability and suppresses lymphocyte activity. This is the basis of therapeutic corticosteroid use.
- **Cardiovascular action:**
- Maintains vascular responsiveness to catecholamines; without cortisol, blood pressure cannot be sustained.
- **Connective tissue action:**
- Inhibits fibroblast activity and collagen synthesis. Sustained elevation produces thin fragile skin, easy bruising, poor wound healing, weakened tendon and ligament, and reduced bone density.
- **Diurnal rhythm:**
- Highest shortly after waking, declining through the day to a trough around midnight. Superimposed on this rhythm is an acute rise with physical or psychological stress.
- **Direct relevance to your work:**
- Every tissue you place your hands on — skin, fascia, muscle, tendon, bone — is altered by sustained cortisol excess. This is why the corticosteroid question belongs on your intake form.

Adrenal androgens

- **Source:**
- Zona reticularis, under ACTH control.
- **Products:**
- Weak androgens, principally dehydroepiandrosterone, converted in peripheral tissue to testosterone or estrogen.
- **Significance in men:**
- Minor, since the testes produce far more testosterone.
- **Significance in women:**
- More important; they are a principal source of androgen, contributing to libido and to axillary and pubic hair.
- **In children:**
- Contribute to the early stages of puberty.
- **After menopause:**
- Become a significant source of the estrogen produced by peripheral conversion in adipose tissue.
- **Pathological excess:**
- Congenital adrenal hyperplasia or an androgen-secreting adrenal tumour can produce virilization in a woman — deepening voice, facial hair, male-pattern hair loss. This is a referral finding.

Adrenal medulla

- **Products:**
- Epinephrine, about 80 per cent, and norepinephrine, about 20 per cent.
- **Classification:**
- Amine hormones; water-soluble, acting through membrane receptors and second messengers, so the response is almost immediate.
- **Control:**
- Neural. Sympathetic preganglionic fibres synapse directly on the secretory cells; the medulla is effectively a modified sympathetic ganglion.
- **Effects:**
- Increased heart rate and contractility, bronchodilation, blood diverted from gut and skin to skeletal muscle, glycogenolysis and lipolysis, pupil dilation, reduced digestive activity.
- **Duration:**
- Brief, seconds to a few minutes, in contrast with the sustained cortisol response.
- **Pathology:**
- **Pheochromocytoma**, a catecholamine-secreting tumour, produces episodes of severe hypertension, pounding headache



Section 2

The Stress Response and the Evidence on Massage

- Short-term and long-term stress responses
- The hypothalamic-pituitary-adrenal axis
- Allostatic load and chronic stress
- What the research on massage and cortisol actually shows
- The problem with the term adrenal fatigue



Short-Term and Long-Term Stress Responses Compared

Feature	Short-term (sympathetic and medullary)	Long-term (cortical)
Pathway	Sympathetic nerves to the adrenal medulla	Hypothalamus to pituitary to adrenal cortex
Chemical mediator	Epinephrine and norepinephrine	Cortisol
Onset	Seconds	Minutes to hours
Duration	Seconds to minutes	Hours to days, or continuous if the stressor persists
Fuel strategy	Rapid glycogen breakdown and fat mobilization	Gluconeogenesis, including breakdown of muscle protein
Cardiovascular effect	Immediate rise in heart rate and cardiac output	Maintenance of vascular tone and blood pressure
Immune effect	Brief enhancement	Suppression if sustained
Purpose	Immediate physical response to threat	Sustained adaptation to a continuing demand



The HPA Axis and Allostatic Load

- **The axis:**
- A stressor activates the hypothalamus to release CRH; the anterior pituitary releases ACTH; the adrenal cortex releases cortisol; rising cortisol inhibits both upper tiers, closing the negative feedback loop.
- **Adaptive purpose:**
- The response is protective. It makes fuel available, sustains blood pressure and limits an excessive inflammatory reaction.
- The problem is duration, not the response itself: The system evolved for stressors lasting minutes to hours, not months to years.
- **Allostatic load:**
- The cumulative physiological cost of repeated or prolonged activation of the stress response, proposed to explain how chronic stress translates into physical disease (McEwen, 1998).
- **Consequences described in the literature:**
- Impaired glucose tolerance, hypertension, central fat deposition, reduced bone density, impaired wound healing, reduced immune competence, sleep disturbance and altered pain sensitivity (Chrousos, 2009).
- **Relevance to a massage practice:**
- Chronic pain, chronic fatigue and fibromyalgia populations show altered stress-axis regulation. This provides a plausible physiological framework for the persistent muscular tension, poor sleep and slow recovery these patients describe.
- **The limit of the framework:**
- A plausible mechanism is not proof that treatment corrects it. Keep the explanation and the claim separate.



Massage Therapy and Cortisol — What the Evidence Shows

- **The common claim:**
- That massage therapy reliably lowers serum cortisol. It appears in a great deal of promotional material and in some textbooks.
- **Where the claim came from:**
- A body of research, including frequently cited work reporting reductions in cortisol together with increases in serotonin and dopamine after massage (Field et al., 2005).
- **The critical appraisal:**
- A comprehensive quantitative review of the cortisol literature found the reduction attributable to massage to be small, inconsistent across studies, and not reliably distinguishable from the change expected simply from resting quietly (Moyer et al., 2011).
- **The methodological problems identified:**
- Small samples, absence of adequate control conditions, variability in sampling time relative to the cortisol diurnal rhythm, and selective reporting.
- **What is better supported:**
- Reductions in self-reported anxiety, perceived stress and pain, and improvements in reported sleep quality — outcomes with more consistent support across the massage therapy literature.
- **What you may say to a patient:**
- That many people find treatment reduces their experience of stress and improves how they sleep, and that reduced sympathetic arousal is a reasonable expected effect.



The Problem With the Term 'Adrenal Fatigue'

- **The claim:**
- That prolonged stress exhausts the adrenal glands so that they can no longer produce sufficient cortisol, producing fatigue, poor stress tolerance, cravings and body aches.
- **The evidence:**
- A systematic review examined the studies offered in support of the concept and concluded that adrenal fatigue is not a substantiated medical condition, and that the tests used to diagnose it do not reliably distinguish affected from unaffected people (Cadegiani & Kater, 2016).
- **The clinical danger:**
- The symptoms attributed to adrenal fatigue overlap substantially with hypothyroidism, anemia, sleep apnea, depression, diabetes and genuine adrenal insufficiency — all of which are diagnosable and treatable. Accepting the label can delay a real diagnosis.
- **What is real:**
- Adrenal insufficiency, both primary (Addison's disease) and secondary (from pituitary failure or corticosteroid withdrawal), is a genuine, dangerous and treatable condition.
- **What is also real:**
- Altered regulation of the HPA axis in chronic stress and chronic pain states is an active research area described in the physiological literature. Altered regulation is not the same claim as glandular exhaustion.
- **How to handle it in practice:**
- If a patient tells you they have been diagnosed with adrenal fatigue, do not argue and do not endorse. Record what they report in their own words, treat their presenting complaint appropriately, and encourage medical assessment of persistent



Section 3

Adrenal Pathology

- Cushing's syndrome and the effects of corticosteroid therapy
- Addison's disease and adrenal crisis
- Congenital adrenal hyperplasia and pheochromocytoma
- Treatment planning for the adrenal patient



- **Definition:**
- The clinical picture produced by prolonged excess of glucocorticoid, from any cause.
- **Commonest cause by far:**
- Prescribed corticosteroid therapy for asthma, inflammatory bowel disease, rheumatoid arthritis, lupus, transplant immunosuppression or other conditions. This is called exogenous or iatrogenic Cushing's syndrome.
- **Endogenous causes:**
- An ACTH-secreting pituitary adenoma, which is specifically called Cushing's disease; an adrenal tumour; or ectopic ACTH production by a tumour elsewhere, typically in the lung.
- **Body shape changes:**
- Central weight gain with relatively thin limbs, rounding of the face, and a fat pad over the lower cervical and upper thoracic region, historically called a buffalo hump.
- **Skin and connective tissue:**
- Thin fragile skin that tears and bruises easily, purple abdominal and thigh striae, poor wound healing, and increased susceptibility to infection.
- **Musculoskeletal:**
- Proximal muscle wasting and weakness, reduced bone density with vertebral fracture risk, and avascular necrosis of the femoral head in some patients on high-dose therapy.
- **Other features:**
- Hypertension, raised blood glucose or frank diabetes, low mood or irritability, and menstrual disturbance.



Cushing's Syndrome and Corticosteroid Therapy — Treatment Planning

- **Skin comes first:**
- The skin can tear under force that would be unremarkable in another patient. Use light pressure, generous lubricant, no friction or shearing, careful draping and gentle handling of limbs.
- **Inspect and record:**
- Look at the skin before and after treatment and document any bruising or fragility. This protects both the patient and you.
- **Bone:**
- Apply the full osteoporosis precaution set from Lecture 04 — no joint mobilization, no rib springing, no percussion over the trunk, supported positioning and supervised transfers.
- **Muscle:**
- Proximal weakness is genuine muscle loss, not deconditioning alone. Do not challenge it with resisted work, and expect reduced tolerance for position changes.
- **Blood glucose:**
- Corticosteroids raise blood glucose. If the patient is also diabetic, apply the full set of diabetes precautions from later in this lecture.
- **Infection risk:**
- Immune suppression raises infection risk. Maintain strict hygiene and reschedule if you have any transmissible illness yourself.
- **The suppressed axis:**
- A patient on long-term corticosteroids has a suppressed HPA axis and cannot mount a normal cortisol response to stress.



Addison's Disease and Adrenal Crisis

- **Definition:**
- Primary adrenal insufficiency, in which the adrenal cortex fails to produce adequate cortisol and, usually, aldosterone.
- **Commonest cause in Canada:**
- Autoimmune destruction of the adrenal cortex, often occurring together with other autoimmune endocrine disease. Tuberculosis remains an important cause worldwide.
- **Presentation:**
- Chronic fatigue and weakness, weight loss, poor appetite, nausea, abdominal discomfort, dizziness on standing, low blood pressure, salt craving, muscle and joint aching, and low mood.
- **The distinctive sign:**
- Darkening of the skin, most visible in skin creases, over pressure points, on scars and inside the mouth. It occurs because loss of cortisol feedback raises ACTH, whose precursor molecule also stimulates melanocytes. This sign occurs in primary but not in secondary adrenal insufficiency.
- **Secondary adrenal insufficiency:**
- Caused by pituitary failure or, far more commonly, by abrupt withdrawal of long-term corticosteroid therapy. Aldosterone output is preserved and skin darkening does not occur.
- **Management:**
- Lifelong replacement with hydrocortisone, and fludrocortisone where mineralocorticoid is also deficient. Patients are taught to increase their dose during illness, injury or surgery — commonly called stress dosing (Bornstein et al., 2016).

- **Adrenal crisis:**

- Acute severe cortisol deficiency precipitated by infection, injury, vomiting, surgery or missed medication. It presents with



Adrenal Insufficiency — Practical Management in Clinic

Before and during treatment

- Confirm the diagnosis, the medication and whether the patient has taken it today.
- Ask whether they are currently unwell, have vomited, or have had a recent injury or infection. Any of these raises crisis risk.
- Keep treatment moderate in intensity and duration. A patient who cannot raise their own cortisol tolerates physiological stress poorly.
- Monitor for pallor, sweating, nausea, unusual weakness or confusion during the session.
- Manage position changes carefully because of orthostatic hypotension; allow time sitting before standing and stay with the patient.
- Avoid heat applications that cause vasodilation and worsen hypotension.

If a crisis is suspected

- Stop treatment immediately.
- Call emergency services. Adrenal crisis is a medical emergency with a real mortality risk.
- Lay the patient flat with the legs elevated if they are conscious and it is safe to do so.
- Tell the emergency responders that the patient has known adrenal insufficiency. Many patients carry a medical alert bracelet or an emergency injection kit.
- Do not administer any medication. Administering an injection is not within the massage therapy scope of practice.
- Stay with the patient until help arrives, then document the event fully and promptly.



Section 4

The Pancreatic Islets and Glucose Homeostasis

- Islet cell types and their hormones
- Insulin and glucagon as an antagonistic pair
- The complete blood glucose feedback loop
- The counter-regulatory hormones



Cells of the Islets of Langerhans

Cell type	Approximate proportion	Hormone	Principal action
Beta cell	About 70 per cent	Insulin	Lowers blood glucose: promotes uptake into muscle and adipose tissue, glycogen storage in liver and muscle, protein and fat synthesis
Alpha cell	About 20 per cent	Glucagon	Raises blood glucose: stimulates hepatic glycogenolysis and gluconeogenesis
Delta cell	About 5 to 10 per cent	Somatostatin	Inhibits both insulin and glucagon release and slows nutrient absorption
PP (F) cell	A small minority	Pancreatic polypeptide	Regulates pancreatic exocrine secretion and appetite

The islets make up only about 1 to 2 per cent of pancreatic mass but receive a disproportionately large share of its blood supply — a recurring feature of endocrine tissue.



Insulin — The Only Hormone That Lowers Blood Glucose

- **Stimulus for release:**
- A rise in blood glucose detected directly by the beta cell — a humoral stimulus. Release is amplified by gut hormones released during a meal and by parasympathetic activity.
- Action on skeletal muscle and adipose tissue: Inserts glucose transporters into the cell membrane, allowing glucose to enter. Without insulin these tissues cannot take up glucose efficiently.
- **Action on the liver:**
- Promotes conversion of glucose to glycogen for storage and inhibits gluconeogenesis.
- **Anabolic actions beyond glucose:**
- Promotes amino acid uptake and protein synthesis, and promotes fat storage. Insulin is the body's principal storage hormone.
- **The critical point:**
- Insulin is the only hormone that lowers blood glucose. At least five hormones raise it — glucagon, cortisol, epinephrine, growth hormone and thyroid hormone.
- **Why the asymmetry exists:**
- The brain depends almost entirely on a continuous glucose supply and has minimal storage capacity. Hypoglycemia threatens the brain within minutes, whereas hyperglycemia causes harm over years. The body therefore defends far more strongly against low glucose.
- **Direct clinical consequence:**
- **An insulin overdose or a missed meal after a dose is an acute emergency. A single high reading is not.**



The Blood Glucose Feedback Loop

- **Rising glucose after a meal — stimulus:**
- Blood glucose rises above the set point.
- **Receptor and control centre:**
- The beta cell senses the rise directly.
- **Effector and response:**
- Insulin is released; muscle, adipose tissue and liver take up glucose; the liver stores glycogen.
- **Feedback:**
- Blood glucose falls toward the set point; the stimulus is removed; insulin secretion declines.
- **Falling glucose during fasting — stimulus:**
- Blood glucose falls below the set point.
- **Receptor, effector and response:**
- The alpha cell senses the fall; glucagon is released; the liver breaks down glycogen and performs gluconeogenesis; glucose is released into blood.
- **Feedback:**
- Blood glucose rises toward the set point; glucagon secretion declines.
- **The counter-regulatory reinforcement:**
- If glucose continues to fall, epinephrine, cortisol and growth hormone are added to the response. Epinephrine produces the sweating, shaking and anxiety that warn a patient of hypoglycemia — which is why patients with long-standing diabetes and blunted warning symptoms are at higher risk.



Section 5

Diabetes Mellitus

- Type 1, type 2 and gestational diabetes
- **Acute complications:**
- hypoglycemia and hyperglycemic emergencies
- Chronic complications and their treatment implications
- Treatment planning and the diabetic foot



Types of Diabetes Mellitus

Feature	Type 1	Type 2	Gestational
Mechanism	Autoimmune destruction of beta cells; absolute insulin deficiency	Insulin resistance with progressive beta cell failure; relative deficiency	Insulin resistance produced by placental hormones
Typical onset	Childhood or young adulthood, but may occur at any age	Usually adulthood; increasingly seen in younger people	Second or third trimester
Proportion of cases	About 5 to 10 per cent	About 90 per cent	Affects a minority of pregnancies
Body habitus	Often normal or low weight at diagnosis	Often, though not always, associated with excess weight	Variable
Onset of symptoms	Rapid, over days to weeks	Gradual; often silent for years before diagnosis	Usually detected by routine screening
Management	Insulin, essential and lifelong	Diet, activity, oral medication, and insulin when required	Diet, activity, and insulin if required; usually resolves after delivery
Acute emergency risk	Diabetic ketoacidosis; hypoglycemia	Hyperosmolar hyperglycemic state; hypoglycemia if on insulin or certain oral agents	Generally lower, but hypoglycemia if treated with insulin

Gestational diabetes resolves after delivery, but confers a substantially increased lifetime risk of type 2 diabetes (Diabetes Canada 2018).



Hypoglycemia — The Emergency You Are Most Likely to Meet

- **Definition:**
- Blood glucose below the normal range, most often caused by too much insulin, a missed or delayed meal, unusual physical exertion, or alcohol.
- **Early autonomic warning signs:**
- Sweating, shakiness, pallor, palpitations, hunger, tingling around the mouth and anxiety. These are produced by the epinephrine response.
- **Later neurological signs:**
- Confusion, difficulty concentrating, slurred speech, unusual behaviour, irritability, drowsiness and, if untreated, seizure and loss of consciousness.
- **The clinical trap in a massage setting:**
- A patient who becomes vague, uncooperative, irritable or difficult to rouse may be assumed to be deeply relaxed or asleep. Consider hypoglycemia first in a patient with diabetes.
- Immediate response if the patient is conscious and able to swallow: Stop treatment, sit them up, and give a fast-acting carbohydrate such as fruit juice, regular soft drink or glucose tablets. Follow with a longer-acting snack once they improve.
- Response if the patient cannot swallow safely, is unconscious or is seizing: Do not put anything in the mouth. Place them on their side, call emergency services immediately, and stay with them.
- **Clinic preparation:**
- Keep juice or glucose tablets accessible and check the expiry date. Know where the patient keeps their own supplies. Document any episode fully.



Chronic Complications and What They Change

The complications

- **Peripheral neuropathy:**
 - Reduced or absent sensation, usually beginning in the feet in a stocking distribution; also burning pain and numbness.
- **Autonomic neuropathy:**
 - Orthostatic hypotension, impaired sweating and thermoregulation, delayed gastric emptying, and blunted hypoglycemia warning symptoms.
- **Peripheral vascular disease:**
 - Reduced circulation to the lower limb, with cold feet, absent pulses, hair loss and slow healing.
- **Impaired wound healing and infection risk:**
 - A minor break in the skin can progress to serious infection.
- **Retinopathy:**
 - A leading cause of vision loss; relevant to fall risk and to written aftercare.
- **Nephropathy:**
 - A leading cause of kidney failure, bringing anemia, hypertension and renal bone disease with it.
- **Cardiovascular disease:**
 - Substantially increased risk of heart attack and stroke.

What each changes in your plan

- Reduced sensation means the patient cannot reliably report pressure. Their feedback is not a safe guide; use your own judgement and observe tissue response.
- Reduced circulation and impaired healing mean no vigorous friction, no aggressive technique on the lower limb and no heat applications to the feet or legs.
- Autonomic neuropathy means slow, supervised transitions from lying to sitting to standing.
- Infection risk means strict hygiene, clean linens, and no treatment over any break in the skin.
- **Foot inspection at every visit:**
 - look for redness, blisters, cracks, ulcers, ingrown nails, discoloration or unusual warmth, and refer promptly if anything is found.
- Retinopathy and neuropathy together create a genuine fall risk; supervise the transfer on and off the table.
- Never use hot packs on an insensate limb. This is one of the most common avoidable injuries in clinical practice.



Building the Treatment Plan for a Patient With Diabetes

- **Intake:**
- Type of diabetes, duration, medication including insulin type, most recent control, presence of neuropathy, retinopathy, kidney disease, foot problems and cardiovascular disease.
- **Timing:**
- Establish when they last ate and last dosed. Avoid booking at the peak action time of their insulin where possible, and keep appointment times consistent so that the routine is predictable.
- **Site precautions:**
- Avoid pressure over insulin injection sites and over an insulin pump or continuous glucose monitor. Altered absorption at a treated site is a real, though variable, concern.
- **Pressure:**
- Moderate overall. Reduce further where sensation is impaired, since the patient cannot warn you accurately.
- **Hydrotherapy:**
- Avoid heat on the feet, lower legs and any area of reduced sensation or circulation. Neutral or cool applications are safer.
- **Duration:**
- Standard duration is usually appropriate for a well-controlled patient. Shorten it where control is poor, complications are advanced or the patient tires quickly.
- **Aftercare:**
- Advise gentle movement, adequate hydration and, where relevant, that they check their glucose after treatment as their own care team has instructed. Do not advise on medication or dose.



Case Application — The Patient Who Went Quiet

- **Presentation:**
- A 61-year-old woman with type 2 diabetes on insulin books a 60-minute treatment for hip and low back pain. Forty minutes into the session, prone, she stops answering your questions clearly and her replies are slurred. Her skin is damp and pale.
- **What you must consider first:**
- Hypoglycemia. In a patient with insulin-treated diabetes, altered consciousness is hypoglycemia until proven otherwise. Do not assume she has fallen asleep.
- **Immediate action:**
- Stop treatment, help her to turn and sit up safely, cover her, and stay with her. Ask directly whether she feels low and whether she has her glucose meter or tablets.
- **If she can swallow safely:**
- Give fruit juice, a regular soft drink or glucose tablets, then a longer-acting snack once she improves. Do not send her home until she is fully alert and someone knows she is unwell.
- If she cannot swallow safely or does not improve: Call emergency services. Place her in a side-lying recovery position and do not put anything in her mouth.
- **Afterwards:**
- Document the time, the signs observed, what you did, the response, whether emergency services were involved and the advice you gave.
- **Preventing recurrence:**
- Adjust her appointment time relative to meals and insulin, ask about food and dosing at every visit, and record the arrangement in her plan.



Review Questions — Lecture 05

- 1. Name the three zones of the adrenal cortex from superficial to deep and the hormone class each produces.
- 2. Explain why the adrenal medulla is described as a modified sympathetic ganglion.
- 3. Outline the renin-angiotensin-aldosterone system and name two medication classes that interrupt it.
- 4. List the effects of cortisol on metabolism, inflammation, connective tissue and bone.
- 5. Describe the diurnal rhythm of cortisol and give one practical consequence for a massage practice.
- 6. Compare the short-term and long-term stress responses across five features.
- 7. Define allostatic load and give three physiological consequences described in the literature.
- 8. State accurately what the current evidence supports about massage therapy and serum cortisol, and give one sentence you could say to a patient.
- 9. Explain why the term adrenal fatigue is problematic and how you would respond to a patient who uses it.
- 10. List six features of Cushing's syndrome and give a treatment modification for each.
- 11. Explain why skin darkening occurs in Addison's disease but not in secondary adrenal insufficiency.
- 12. Describe adrenal crisis and state exactly what you would do if it occurred during a treatment.
- 13. Name the four islet cell types and their hormones.
- 14. Explain why only one hormone lowers blood glucose while several raise it.
- 15. Differentiate type 1 from type 2 diabetes across five features.
- 16. List the early and late signs of hypoglycemia and state the in-clinic response for a conscious and an unconscious patient.
- 17. Name five chronic complications of diabetes and give one specific treatment modification for each.
- 18. Explain why hot packs must never be applied to an insensate limb.

- The adrenal gland is two organs in one capsule: a steroid-secreting cortex under pituitary control, and a catecholamine-secreting medulla under direct sympathetic control.
- Aldosterone defends blood volume and pressure; cortisol mobilizes fuel, suppresses inflammation, maintains vascular tone and, when sustained, degrades skin, connective tissue, muscle and bone.
- The HPA axis is adaptive over minutes to hours; the harm attributed to chronic stress arises from sustained activation, described as allostatic load.
- The claim that massage reliably lowers cortisol is not well supported. Reductions in reported anxiety, stress and pain are better supported and are what you should describe to patients.
- Adrenal fatigue is not a substantiated diagnosis; adrenal insufficiency is real, dangerous and treatable, and adrenal crisis is a medical emergency.
- Corticosteroid therapy is the commonest cause of cortisol excess and produces fragile skin, reduced bone density, muscle wasting, raised blood glucose, infection risk and a suppressed stress axis.
- The islets defend blood glucose through the antagonistic pair insulin and glucagon; insulin is the only hormone that lowers glucose, which is why hypoglycemia is the acute emergency.
- Diabetes changes timing, site precautions, pressure, hydrotherapy, foot inspection, fall precautions and documentation. Recognizing hypoglycemia and responding correctly is a core professional competency.

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