

Endocrine and Metabolic Disorders for Surgeons

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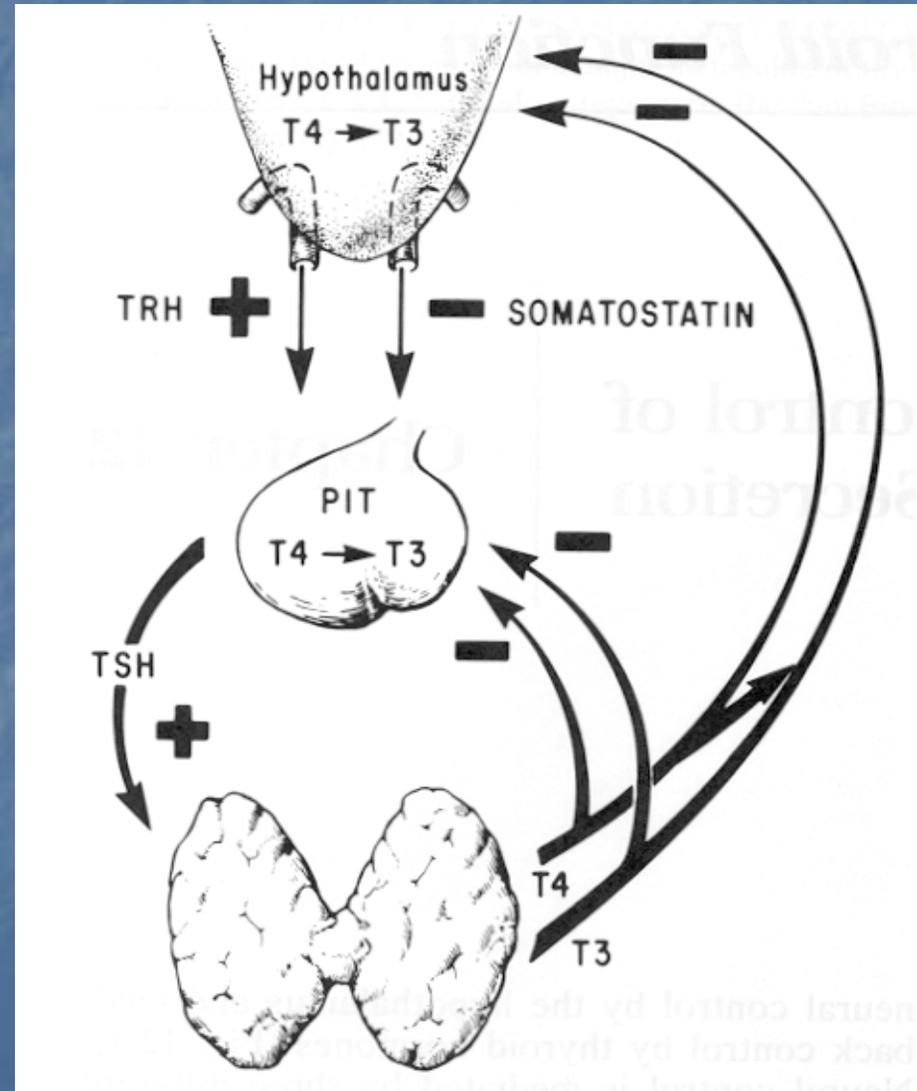
NNUH

The Syllabus says

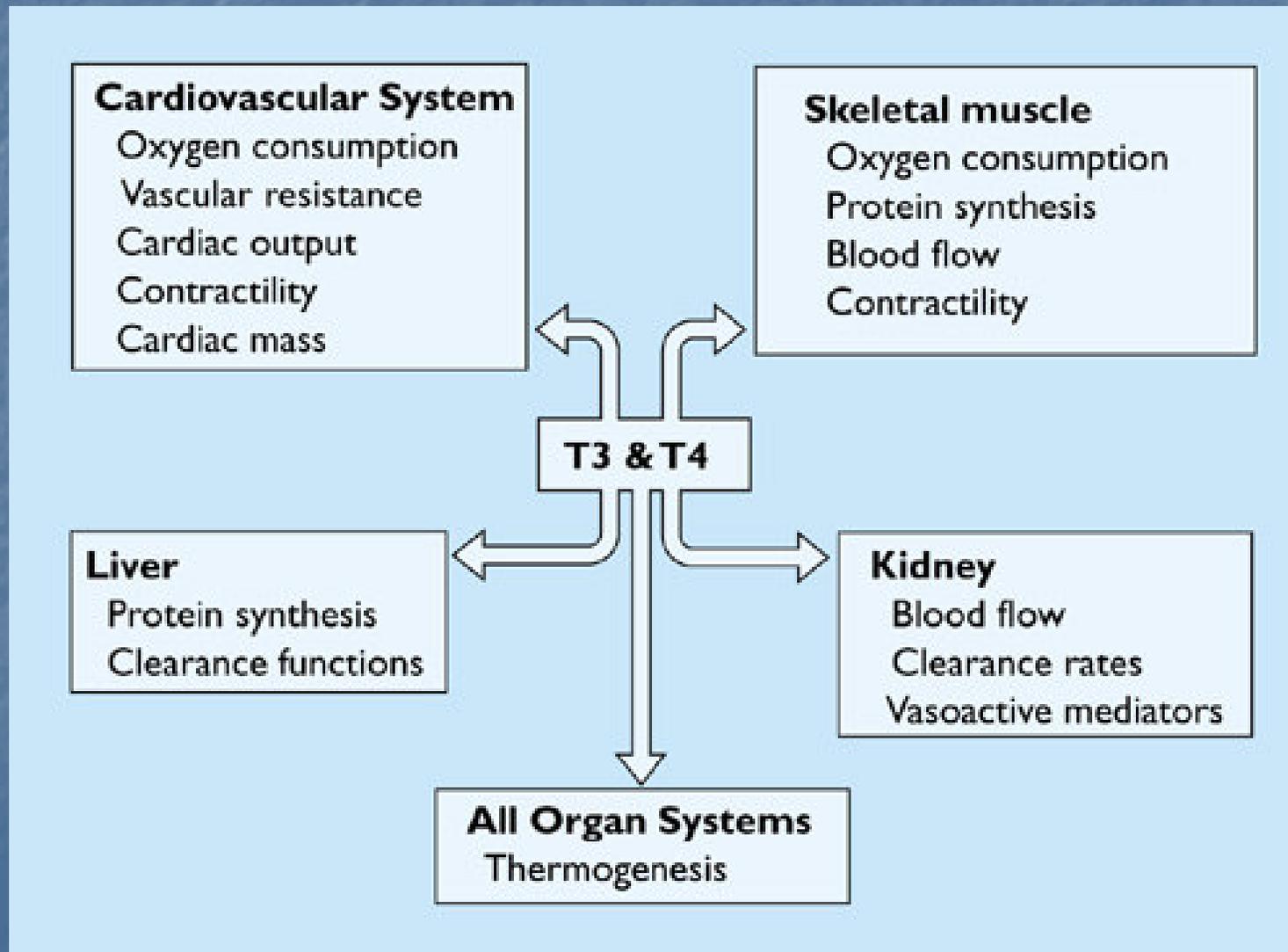
- To identify, investigate and manage surgical patients with common metabolic disorders
 - thyrotoxicosis and hypothyroidism
 - hypercalcaemia
 - corticosteroid therapy
 - diabetes mellitus
 - hyponatraemia

Thyroid Problems

- The hypothalamic-pituitary-thyroid axis is a classic feedback loop

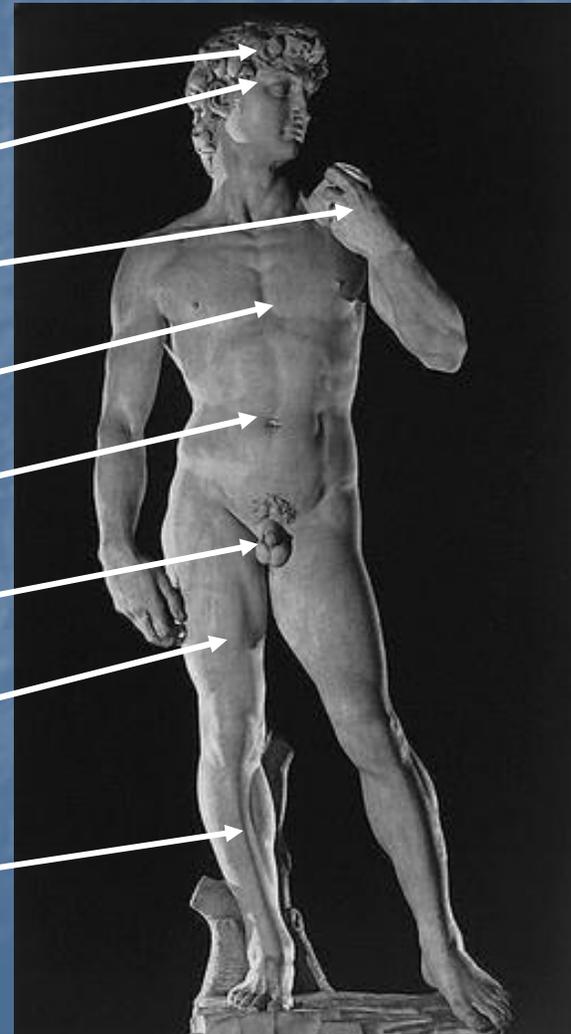


What does Thyroid Hormone Do?



Symptoms of Hyperthyroidism

- Neuro-psychiatric
- Thermoregulatory
- Dermatological
- Cardio-pulmonary
- Gastroenterological
- Endocrine / reproductive
- Muscular
- Skeletal



Symptoms of Hyperthyroidism

- Hyperactivity, irritability, altered mood (99%)
- Heat intolerance, sweating, (90%)
- Palpitations (85%)
- Fatigue, weakness (85%)
- Weight loss with increased appetite (85%)
- Diarrhoea (33%)
- Eye complaints (55%)

Signs of Hyperthyroidism

- Sinus tachycardia (100%) or AF (10%)
- Fine tremor (97%)
- Warm, moist skin (97%)
- Goitre (100% in Graves')
- Palmer erythema, onycholysis, pruritus (35%)
- Alopecia
- Muscle weakness and wasting, proximal myopathy
- Lid lag and retraction (71%)
- Gynaecomastia (10%)
- Chorea, periodic paralysis, psychosis (<1%)

Causes of Hyperthyroidism

- Graves' disease – TSH stimulating Ab's
- Hyperfunctioning nodule – autonomous adenoma
- Toxic MNG – multiple nodules
- Iodine load with underlying Graves'
- Hyperemesis gravidarium
- Hydatidiform mole
- Choriocarcinoma
- Pituitary adenoma

Symptoms of Hypothyroidism

- Tired, lethargy, fatigue, weight gain
- Depression / low mood
- Cold intolerance
- Dry skin, hair / hair loss
- Constipation
- Cardiac failure
- Hypercholesterolaemia / vascular disease
- Hoarse voice
- Menstrual changes (menorrhagia)

Signs of Hypothyroidism

- Dry skin, thin hair
- Cool peripheries
- Puffy face hands feet
- Yellow skin
- Bradycardic
- Peripheral oedema
- Slow relaxing reflexes
- Carpal tunnel syndrome
- Serous cavity effusions
- Galactorrhoea
- Ataxia, dementia, psychosis, coma



Causes of Hypothyroidism

■ Primary

- Iodine deficiency
- Autoimmune hypothyroidism (Hashimoto's)
- Iatrogenic: I^{131} , thyroidectomy, DXT
- Drugs: I containing contrast media, amiodarone, lithium
- Congenital: absent or ectopic glands, or dyshormonogenesis, TSH receptor mutation
- Destructive thyroiditis: postpartum, silent, subacute
- Infiltrative disorders: amyloid, sarcoid, haemochromatosis, etc.

Causes of Hypothyroidism

■ Secondary

- Hypopituitarism: tumours, trauma, surgery or DXT, infiltration, infarction
- Isolated TSH deficiency or inactivity
- Hypothalamic disease: tumours, trauma, infiltration, idiopathic

Surgical Considerations

- Complications abound in those operated on with hypo or hyperthyroidism
- If it is an elective procedure get an endocrinologists to fix their thyroid function if possible
- If it is an emergency then get senior help sooner rather than later
 - use T3 for those underactive
 - try and avoid operating on patients with uncontrolled hyperthyroidism at all costs

Hypercalcaemia

- General mechanisms
 - increased bone resorption
 - increased intestinal absorption of calcium
 - decreased renal excretion of calcium
- Symptoms of elevated calcium
 - stones, bones, abdominal groans and psychiatric overtones or asymptomatic

Hypercalcaemia - Causes

- Primary Hyperparathyroidism
 - benign tumor making PTH disregards feedback
- Malignancy
 - tumor making PTHrP (acts just like PTH)
- Extra-renal 1α hydroxylase activity
 - unregulated (not regulated by PTH)
 - lymphoid tissue and macrophages, granulomas
 - intestinal hyperabsorption of calcium

Hypercalcaemia - Causes 2

- Familial Benign Hypocalciuric Hypercalcaemia
 - inactivating mutation of CaR
 - autosomal Dominant - lifelong
 - typically mild hypercalcaemia and asymptomatic
 - hypocalciuria as CaR is in distal nephron also
- Vitamin D intoxication

Corticosteroid Therapy

■ Cautions

- adrenal suppression and infection, children and adolescents (growth retardation possibly irreversible), elderly (close supervision required particularly on long-term treatment); frequent monitoring required if history of tuberculosis (or X-ray changes), hypertension, recent myocardial infarction (rupture reported), congestive heart failure, liver failure, renal impairment, diabetes mellitus including family history, osteoporosis (post-menopausal women at special risk), glaucoma (including family history), corneal perforation, severe affective disorders (particularly if history of steroid-induced psychosis), epilepsy, peptic ulcer, hypothyroidism, history of steroid myopathy; pregnancy and breast-feeding

Corticosteroid Therapy

■ Complications

- *gastro-intestinal effects* include dyspepsia, peptic ulceration (with perforation), abdominal distension, acute pancreatitis, oesophageal ulceration and candidiasis; *musculoskeletal effects* include proximal myopathy, osteoporosis, vertebral and long bone fractures, avascular osteonecrosis, tendon rupture; *endocrine effects* include adrenal suppression, menstrual irregularities and amenorrhoea, Cushing's syndrome (with high doses, usually reversible on withdrawal), hirsutism, weight gain, negative nitrogen and calcium balance, increased appetite; increased susceptibility to and severity of infection; *neuropsychiatric effects* include euphoria, psychological dependence, depression, insomnia, increased intracranial pressure with papilloedema in children (usually after withdrawal), psychosis and aggravation of schizophrenia, aggravation of epilepsy; *ophthalmic effects* include glaucoma, papilloedema, posterior subcapsular cataracts, corneal or scleral thinning and exacerbation of ophthalmic viral or fungal disease; *other side-effects* include impaired healing, skin atrophy, bruising, striae, telangiectasia, acne, myocardial rupture following recent myocardial infarction, fluid and electrolyte disturbance, leucocytosis, hypersensitivity reactions (including anaphylaxis), thromboembolism, nausea, malaise, hiccups

Primary Adrenal Insufficiency

- Functional Status
 - primary “damage” to adrenal cortices
 - low cortisol
 - low aldosterone
 - high ACTH

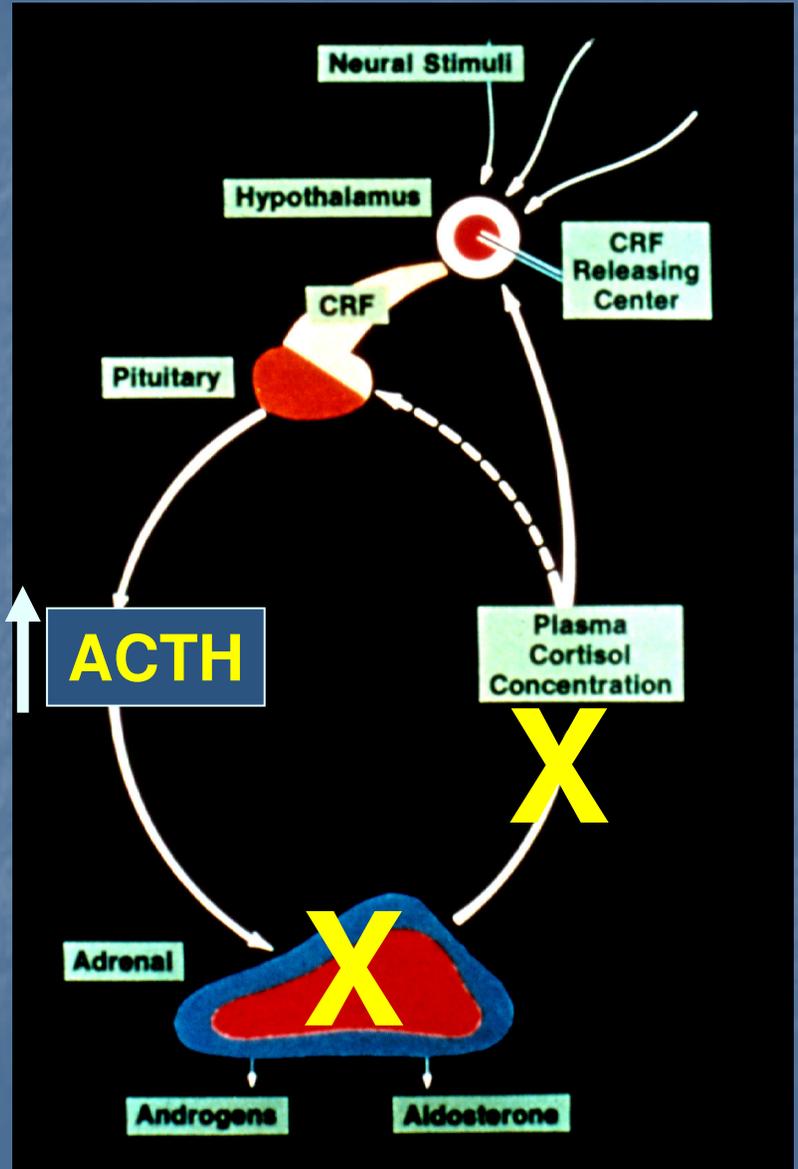
Primary Adrenal Insufficiency

Aetiologies:

- Autoimmune
 - Antibody(s) directed toward cellular components
 - Most common to 21-hydroxylase
 - Can be with combination immune injury to other target organs
 - skin (vitiligo), Thyroid, Gonad, Gastric parietal cells (Vit B₁₂)
- Infectious
 - Tuberculosis most common in past (5% of people with TB have adrenal involvement)
 - Fungal most common now (histoplasmosis, etc.)
 - Opportunistic infections (immune compromised patients, AIDS)
- Metastatic malignancies
 - Lung, breast, melanoma, etc.
 - Lymphoma
- Ischaemic injury
 - Haemorrhage
 - Coagulation disorders
 - Disseminated infections with intravascular coagulation

Primary Adrenal Insufficiency

- Diagnosis
 - low random cortisol
 - low blood glucose and low blood pressure
 - hyponatraemia and hyperkalaemia (mild)
 - elevated ACTH
 - blunted / absent short synacthen test





Hyperpigmentation
& Vitiligo



Hyperpigmented
scars

Management

- Call the endocrine team
- DO NOT OPERATE ON A SUSPECTED ACUTE ADDISONIAN PATIENT
 - glucocorticoid replacement with hydrocortisone
 - mineralocorticoid replacement with fludrocortisone

Diabetes

- How long do we have to talk?

Two Main Types

- Type 1
 - Autoimmune destruction of the β cells of the Islets of Langerhans in the pancreas. This leads to an absolute insulin deficiency. Insulin treatment is therefore mandatory
 - Previously known as IDDM or juvenile onset diabetes

Two Main Types

- Type 2
 - Impaired insulin action (insulin resistance) and eventually, impaired insulin secretion as well
 - Usually treated with oral medication initially, then may move onto insulin
 - Formerly known as NIDDM or maturity onset diabetes

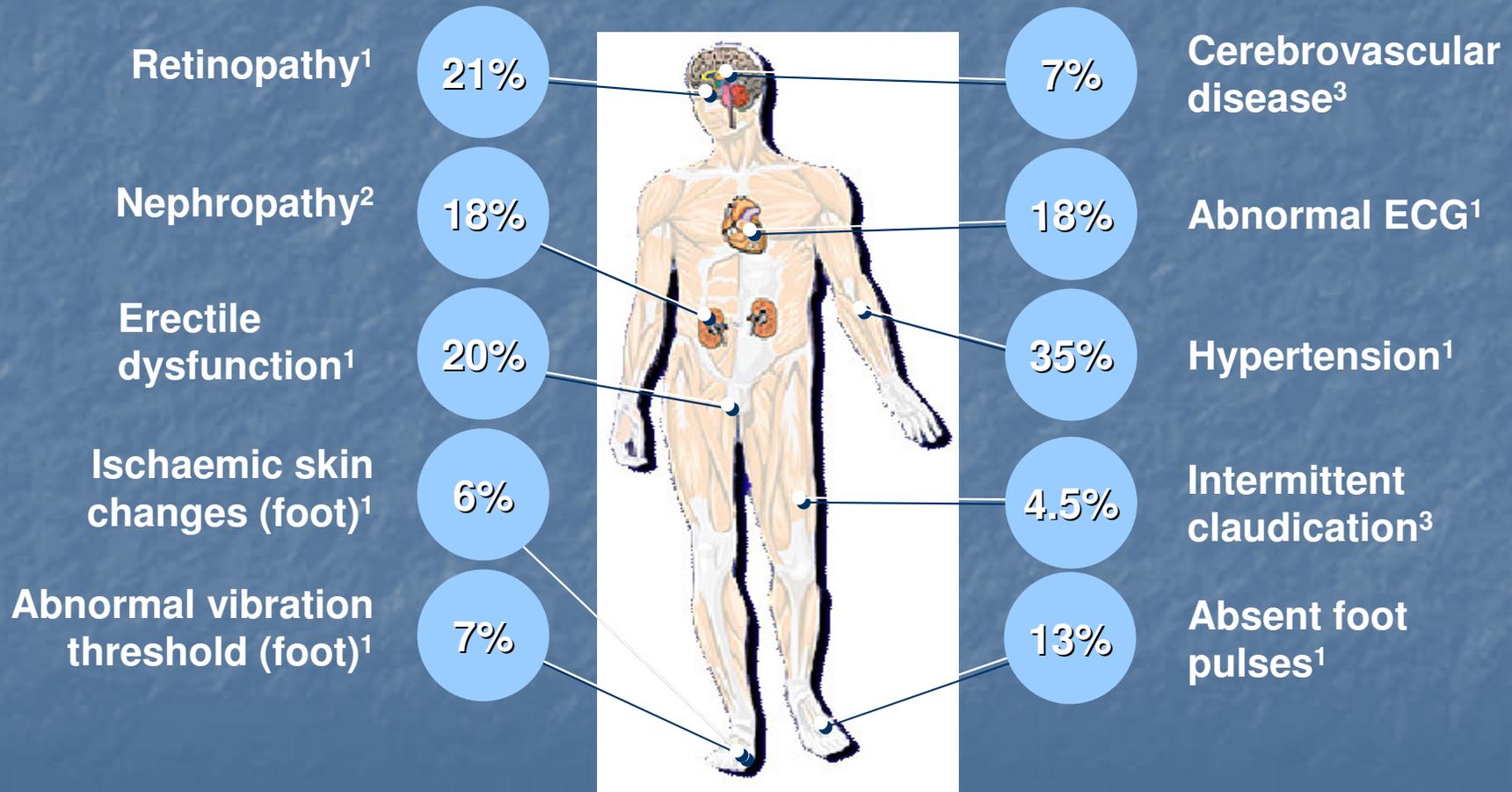
Why is it Important?

- Poorly controlled diabetes leads to accelerated cardiovascular morbidity and mortality
- A combination of microvascular and macrovascular disease

How Do You Make The Diagnosis?

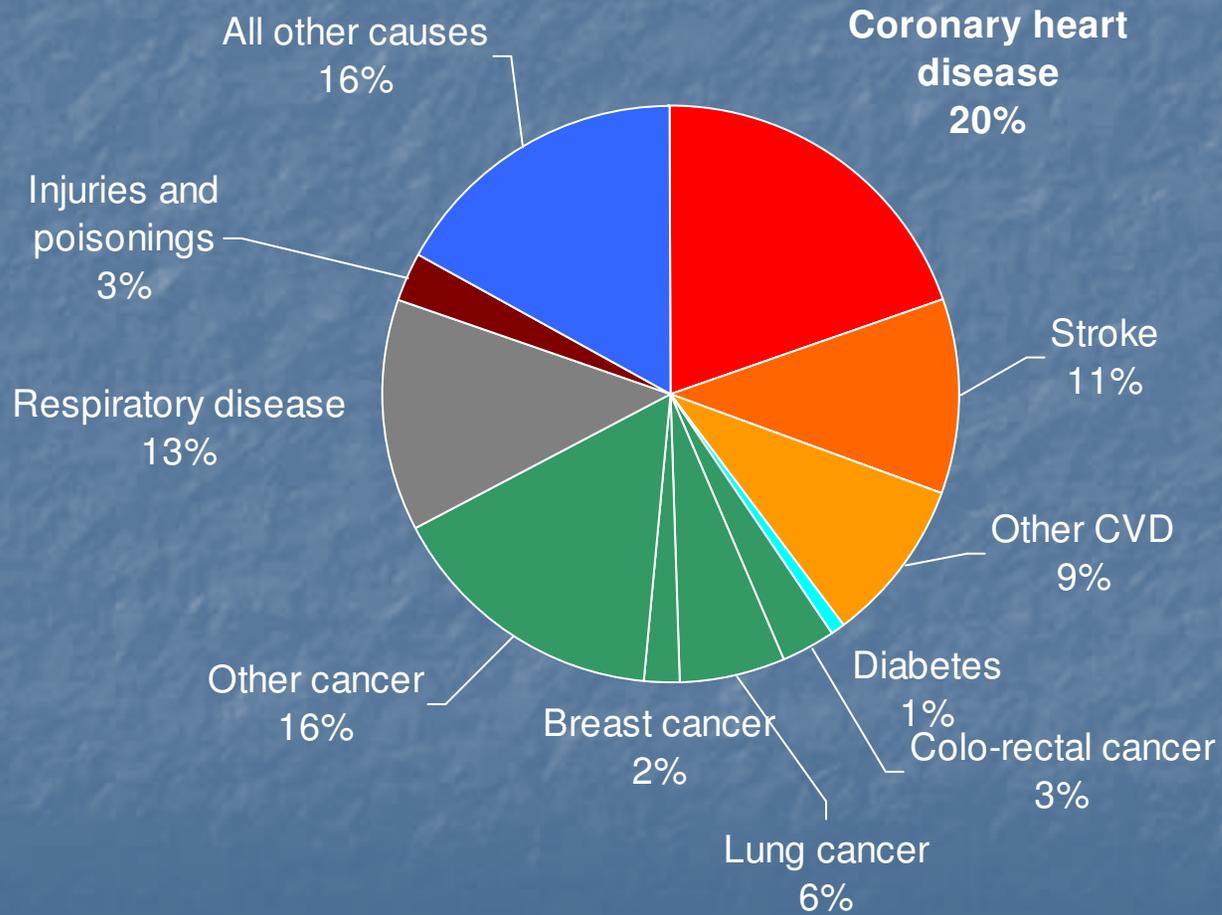
Plasma glucose concentration 2 h following a 75 g oral glucose test (mmol/l)	Fasting plasma glucose concentration (mmol/l)		
	< 6.1	≥ 6.1–6.9	≥ 7.0
< 7.8	Normal	Impaired fasting glycaemia	Diabetes
≥ 7.8–11.0	Impaired glucose tolerance	Impaired glucose tolerance	Diabetes
≥ 11.1	Diabetes	Diabetes	Diabetes

Vascular Complications Of Type 2 Diabetes At The Time Of Diagnosis



1. UKPDS Group. *Diabetes Res* 1990; **13**: 1–11. 2. The Hypertension in Diabetes Study Group. *J Hypertension* 1993; **11**: 30–17. 3. Wingard DL *et al.* *Diabetes Care* 1993; **16**: 1022–5.

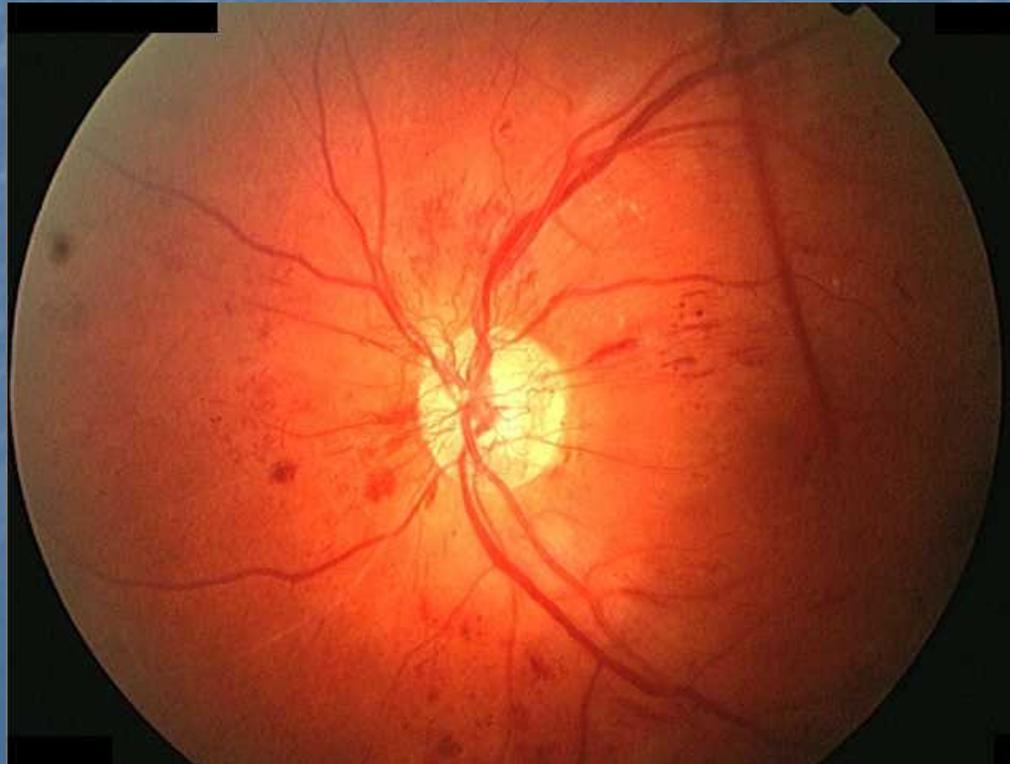
Causes of Death in the UK (2001)



1. BHF Coronary Heart Disease Statistics 2003. Available on www.heartstats.org

Microvascular Disease

- Diabetic retinopathy – the commonest cause of blindness in the developed world



Microvascular Disease

- Combinations of neuropathy and ischaemia



What Would Be Ideal?

- That they did not have diabetes
- If they had diabetes, for the HbA₁C to be <7% and blood glucose levels between 6 and 8 all the time
- If they had diabetes for it to be diet controlled
- If they were not diet controlled then for someone else to operate on them

What About Insulin and Surgery?

- Complex but can be worked out

Once Daily

- Usually a long acting analogue
- If taken in the morning, half the dose and have blood glucose tested on admission
- If >12 mmol/l give 6 units of Human Actrapid and repeat blood glucose level 1 hour later, giving another 6 units Human Actrapid if it was still higher than 12 mmol/l
- Resume normal insulin the next day, and be warned that their blood glucose would be high for a day or so

Once Daily

- If taken in the evening, then no dose change required

Twice Daily Insulin

- Usually a mixture
- Half morning dose and have blood glucose tested on admission
- If >12 mmol/l give 6 units of Human Actrapid and repeat blood glucose level 1 hour later, giving another 6 units Human Actrapid if it was still higher than 12 mmol/l
- Resume normal insulin that afternoon, and be warned that their blood glucose would be high for a day or so

3, 4 or 5 Injections Per Day

- If on a morning list
- Half morning dose and have blood glucose tested on admission
- If >12 mmol/l give 6 units of Human Actrapid and repeat blood glucose level 1 hour later, giving another 6 units Human Actrapid if it was still higher than 12 mmol/l
- Restart normal insulin later that afternoon if eating and drinking normally

3, 4 or 5 Injections Per Day

- If on an afternoon list
- Have their usual morning insulin doses in then omit their lunchtime dose
- Have blood glucose tested on admission
- If >12 mmol/l give 6 units of Human Actrapid and repeat blood glucose level 1 hour later, giving another 6 units Human Actrapid if it was still higher than 12 mmol/l
- Restart normal insulin later that afternoon if eating and drinking normally

For Non Day Case or Emergencies

- Not difficult
- Intravenous insulin from the time of admission to the time they are eating and drinking normally

When in Doubt – Ask for Help

- There is a Diabetes Inpatient Specialist Nurse available 9 to 5 Monday to Friday on bleep 0407
- There is an inpatient consultant diabetes ward round almost every weekday
- There are 2 consultant or SpR led triage rounds every single day

Hyponatraemia

- The ideal scenario is that everyone on intravenous fluids should have their U&E's checked every day – and the results should be looked at

Hyponatraemia

- Symptoms – mainly due to CNS dysfunction
 - headache, nausea, vomiting, muscle cramps, lethargy, restlessness, disorientation, and depressed reflexes
 - severe and rapidly evolving hyponatraemia include seizures, coma, permanent brain damage, respiratory arrest, brain-stem herniation, and death

Treatment

- Depends on cause
- Call for help sooner rather than later

Any Questions?