



DIABETIC EMERGENCIES

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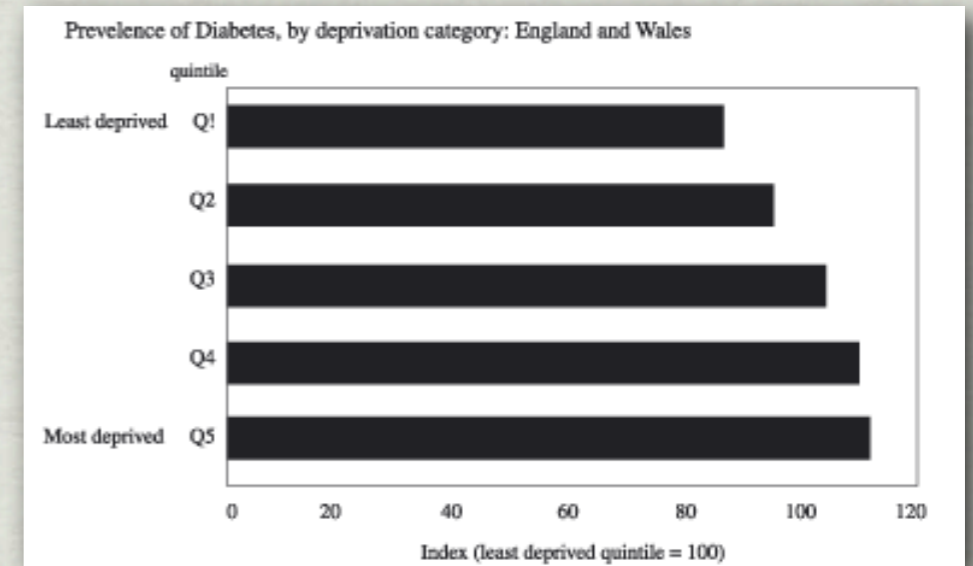
Objectives

Understand:

- * Pathophysiology
- * Clinical presentation
- * Hypoglycaemia
- * DKA
- * HONK
- * Complications



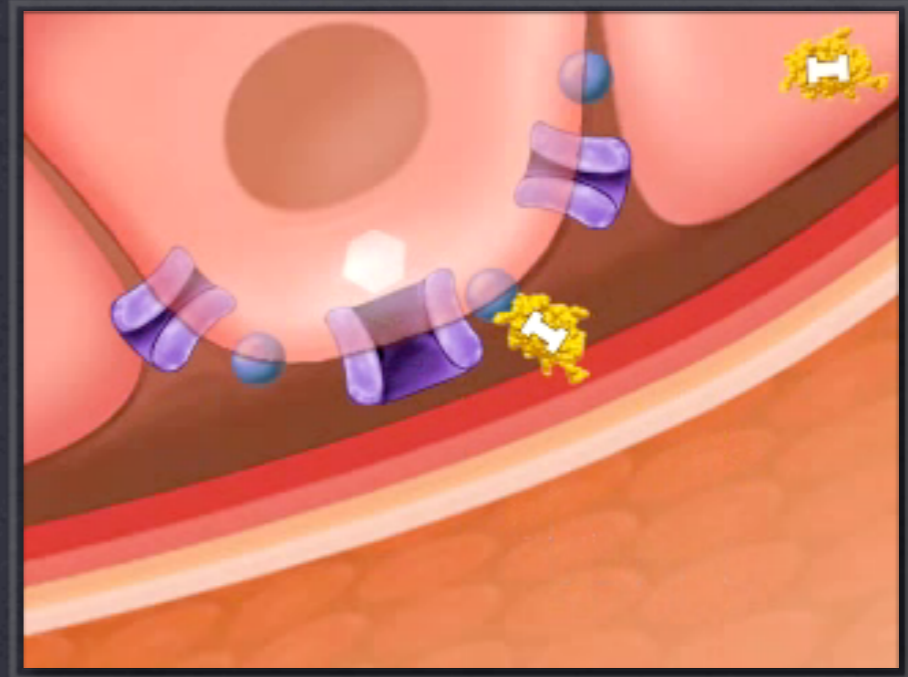
Introduction



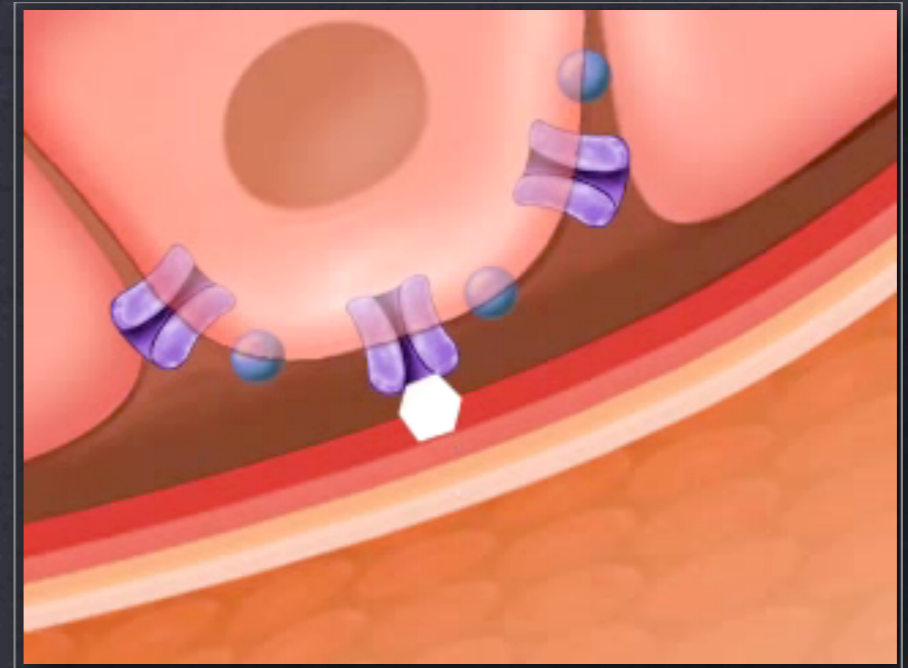
- * Common, affects 1.3 million people and increasing, 2-3% population, 25-30/100000 kids, 9% of hospital costs
- * Life threatening if not acted on promptly and correctly
- * More common in ethnic minorities/older age groups, in less affluent, men
- * There is a NSF for diabetes

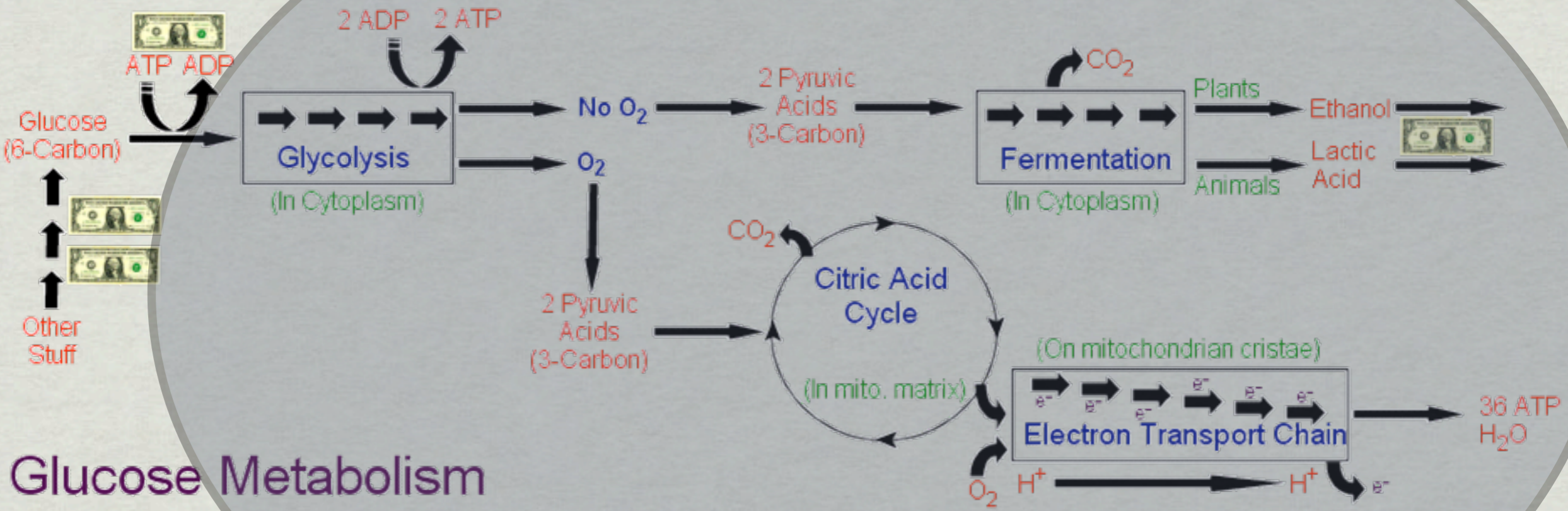
Pathophysiology

With Insulin



Without Insulin





Glucose Metabolism

Insulin Effects

- * Reduced release of glucose by liver
- * Increased uptake of glucose eg in skeletal muscle
- * Inhibit triglyceride breakdown to glycerol and free fatty acids (& stimulate its formation)
- * Inhibits protein breakdown to free amino acids (which can themselves increase hepatic production)

- * Type 1: IDDM, usually juvenile onset, damage to pancreatic β cells, always need insulin, more prone to DKA. 30% twin concordance
- * Type 2: NIDDM, usually adults >40yrs, more common in men, asians, obesity, lack of exercise, high calorie diet. Decreased insulin and insulin resistance. 80% twin concordance.
- * Lack of insulin results in failure of uptake and utilisation of glucose in cells causing rise in blood glucose. Long term risks of CVS/renal/eye disease.
- * Acute complications DKA, HONK, hypoglycaemia. Long term in DM: IHD, Blindness, renal failure, arterial dz-amputations



Clinical Presentation

- * History polydipsia, polyuria, weight loss with increased appetite
- * May have reduced GCS, sweaty, clammy, tachycardia and fits (hypo)
- * May be acidotic, shocked, coma (DKA). Coma without acidosis, raised osmolarity- HONK
- * fasting BS ≥ 7 mmol/l, random ≥ 11.1 mmol/l

Hypoglycaemia

=glucose <3mmol/l

- * Neonates or DM missed meals or overdosed insulin/oral hypoglycaemic agents, increased activity
- * EXPLAIN
 - * EXogenous drugs
 - * Pituitary insufficiency
 - * Liver failure
 - * Addison's disease
 - * Islet cell tumours
 - * Non-pancreatic neoplasms

Hypoglycaemia

- * Think of in any coma, may have confusion, personality change appear drunk, sweaty, tachycardia, fits, focal signs
- * Check BM and formal sugar but treat
- * Oral if awake
- * SC/IV/IM glucagon 1mg NOT in liver disease, alcohol or malnourished or sulphonyl urea OD. Doesn't work if on beta blockers
- * IV dextrose 50ml 50%, 250ml 10% (5ml/kg)

Hypoglycaemia

- * If cause found, corrected and patient well, discharge
- * If not responding further investigate eg CT and admit
- * If OD eg sulphonyl ureas, admit (long duration of action). May need continuous infusion

DKA: Introduction

- * More common in children, life threatening. May have developed over 2-3 days. Often compliance issues.
- * Excess glycogenolysis with high glucagon levels and low insulin and raised sugar levels results in acidosis, fluid loss (osmotic diuresis), hypotension and shock
- * Caused by four 'I's (Infection, Infarction, Insufficient insulin and Intercurrent illness)
- * Death from hypokalaemia, cerebral oedema, & aspiration pneumonia

DKA: Features

- * S/S of DM, plus D&V and abdominal pain
- * Hyperventilation (Kussmaul) and acetone breath
- * Altered GCS, dehydration and +/- shock
- * Urinary glucose/ketones
- * ↓pH, ↑K⁺, ↓Na, ↑Glucose, ↑urea/creatinine
- * High anion gap: $(\text{Na}^+ + \text{K}^+ - \text{HCO}_3^- - \text{Cl}^-) \{ >14-18 \}$
- * Check urine, CXR, blood cultures for infection

DKA: Management I

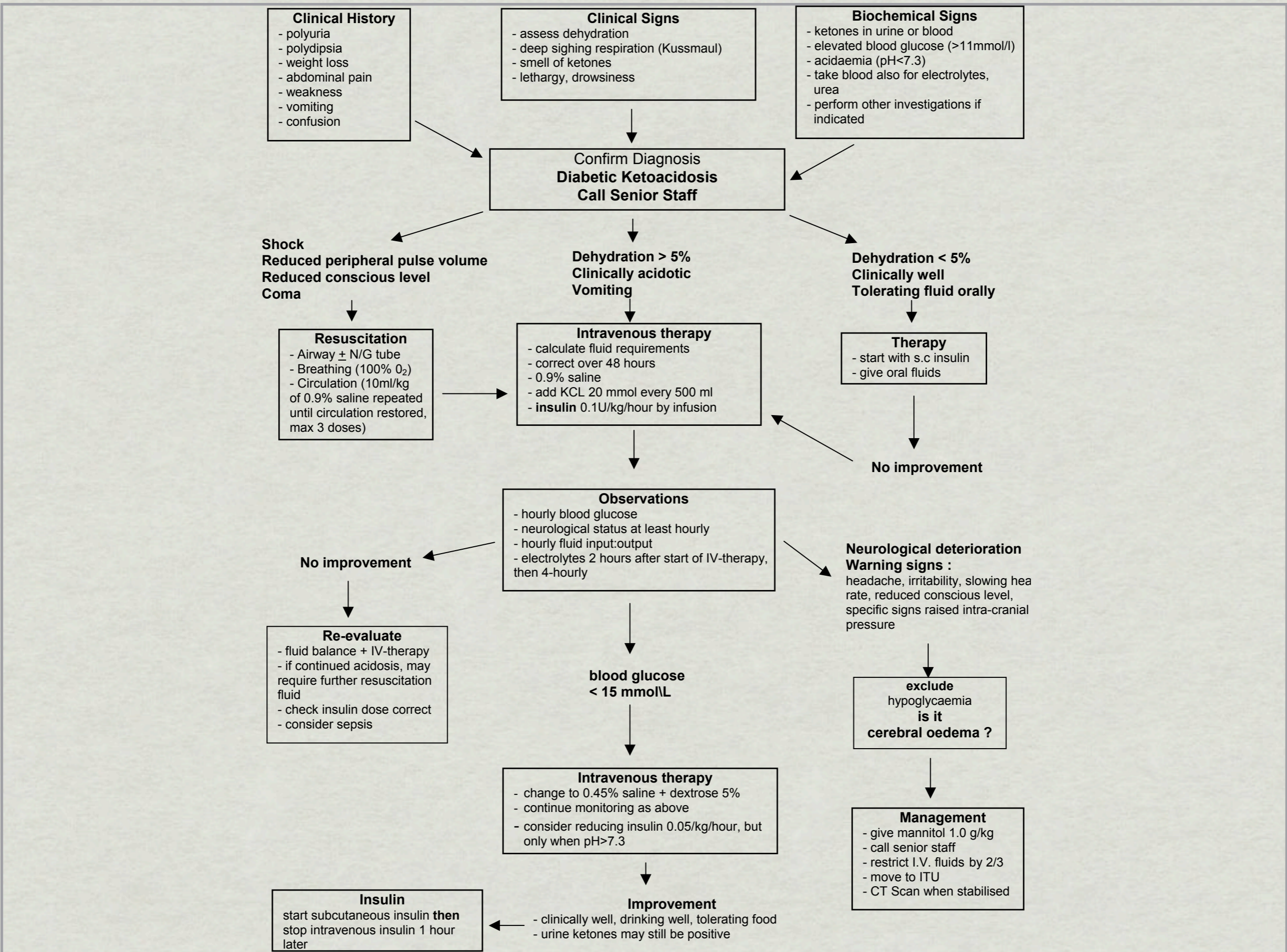
- * **A:** may need RSI if airway risk from coma/vomiting etc
- * **B:** O₂ 100% by mask
- * **C:** IV access and initial resuscitation bolus fluids of Normal saline 1-2 litres (10ml/kg aliquots up to 30ml/kg). Get routine bloods/cultures/gases. May need CVP.
- * **D:** RSI if less than 8, (also may need CT)

DKA: Management II

- * Add potassium after bolus resuscitation fluid, 40mmol/l unless anuric
- * Strict fluid record, Paeds: Maintenance plus deficit correction **over 48 hours** once resuscitated.

| Maintenance requirements | Age | Requirements |
|--------------------------|-------------|-----------------|
| | 0 - 2 yrs | 80 ml/kg/24 hrs |
| | 3 - 5 | 70 ml/kg/24 hrs |
| | 6 - 9 | 60 ml/kg/24 hrs |
| | 10 - 14 | 50 ml/kg/24 hrs |
| | adult (>15) | 30 ml/kg/24 hrs |

- * Adults: start NS 2L/2hrs, then 2 → 4 hourly. 5% dextrose when BM<11
- * Insulin 6u/hr (0.1u/kg/hr) IV. NO loading dose



HONK

Hyperosmolar non-ketotic

- * Usually in older patients
- * Calculate osmolality: $[2(\text{Na}^+ + \text{K}^+) + \text{Urea} + \text{Glucose}]$,
Osmolar Gap = Calculated - Actual (NR: 15-20)
- * Similar treatment to DKA, (initially NS). Consider 0.45% NS if osmolality $>320\text{mmol/l}$

Complications

- * Cerebral Oedema
- * Shock
- * Hypokalaemia
- * Aspiration pneumonia
- * Pulmonary oedema





Questions?



Summary

- * Insulin lack causes hyperglycaemia, high triglycerides and protein breakdown
- * There is Type I (IDDM) & Type II (NIDDM)
- * Hypoglycaemia can mimic drunkenness
- * Glucagon not in malnourished for hypo
- * DKA, look for infection and focus on fluids/potassium and then insulin. Avoid rapid drop in glucose