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The child with polyuria

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Outline

- Define polyuria
- Differential diagnosis of polyuria
- Able to manage a patient presenting with polyuria

Case

- 2 years old girl previously healthy, presented with frequent urination & drinking water for 2 weeks
- What further history you want to obtain?

- Imp to ask about the **volume of the urine** and the frequncey of the urine.

What is too much ?

- **1 cup** **1L** **2L** **5L**

Polyuria

=

Urine output it depends **on body size**.

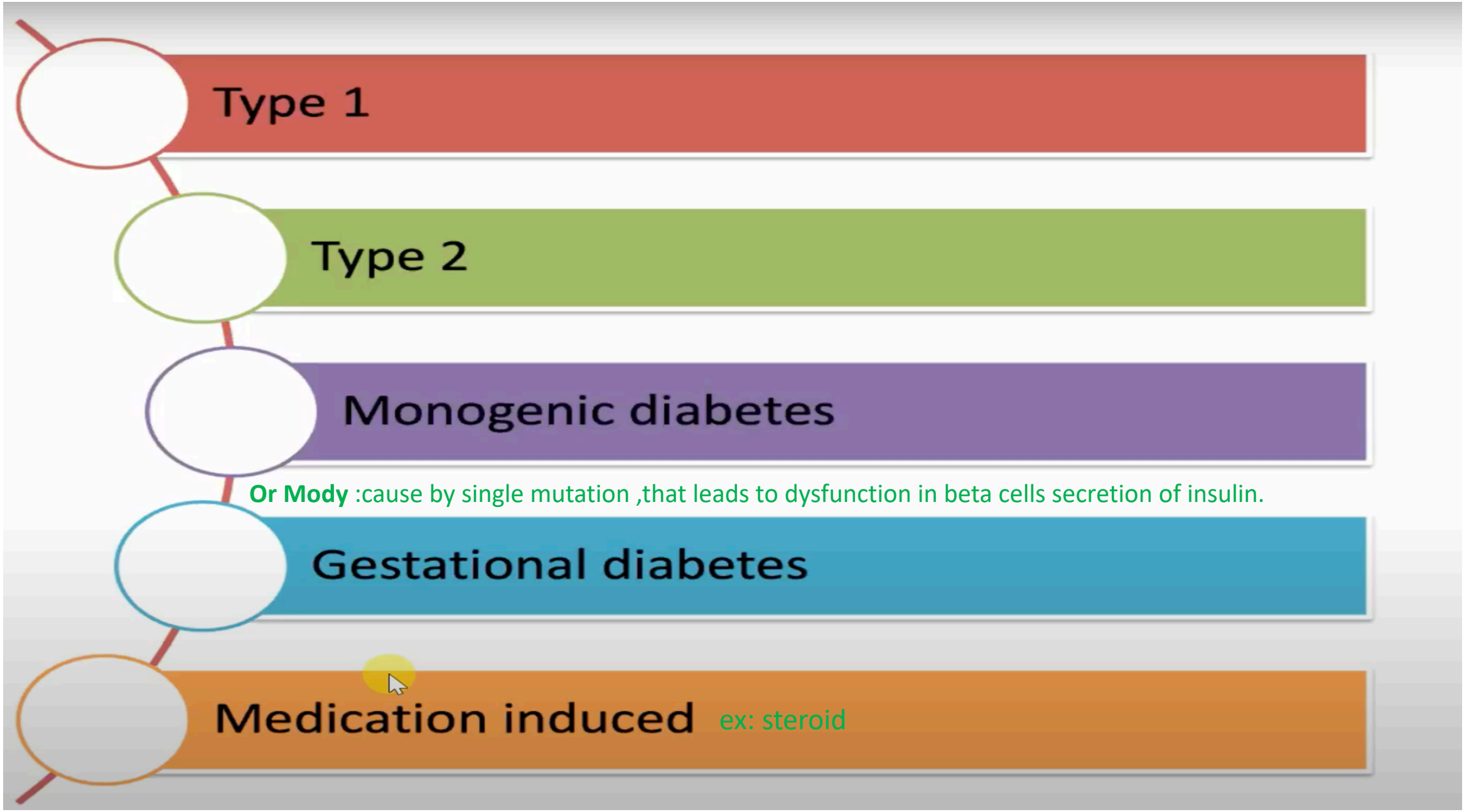
Urine output >2L/m²/day

What is your differential diagnosis?

Metabolic	• **Diabetes mellitus** • Hypercalcemia • Hypokalemia
Renal	• Renal tubular acidosis • Barter syndrome • Nephrognic diabetes insipidus
CNS	• **Central diabetes insipidus**
Psychogenic	
Medications	• Diuretics

DIABETES MELLITUS

TYPES OF DIABETES ?



Or Mody :cause by single mutation ,that leads to dysfunction in beta cells secretion of insulin.

ex: steroid

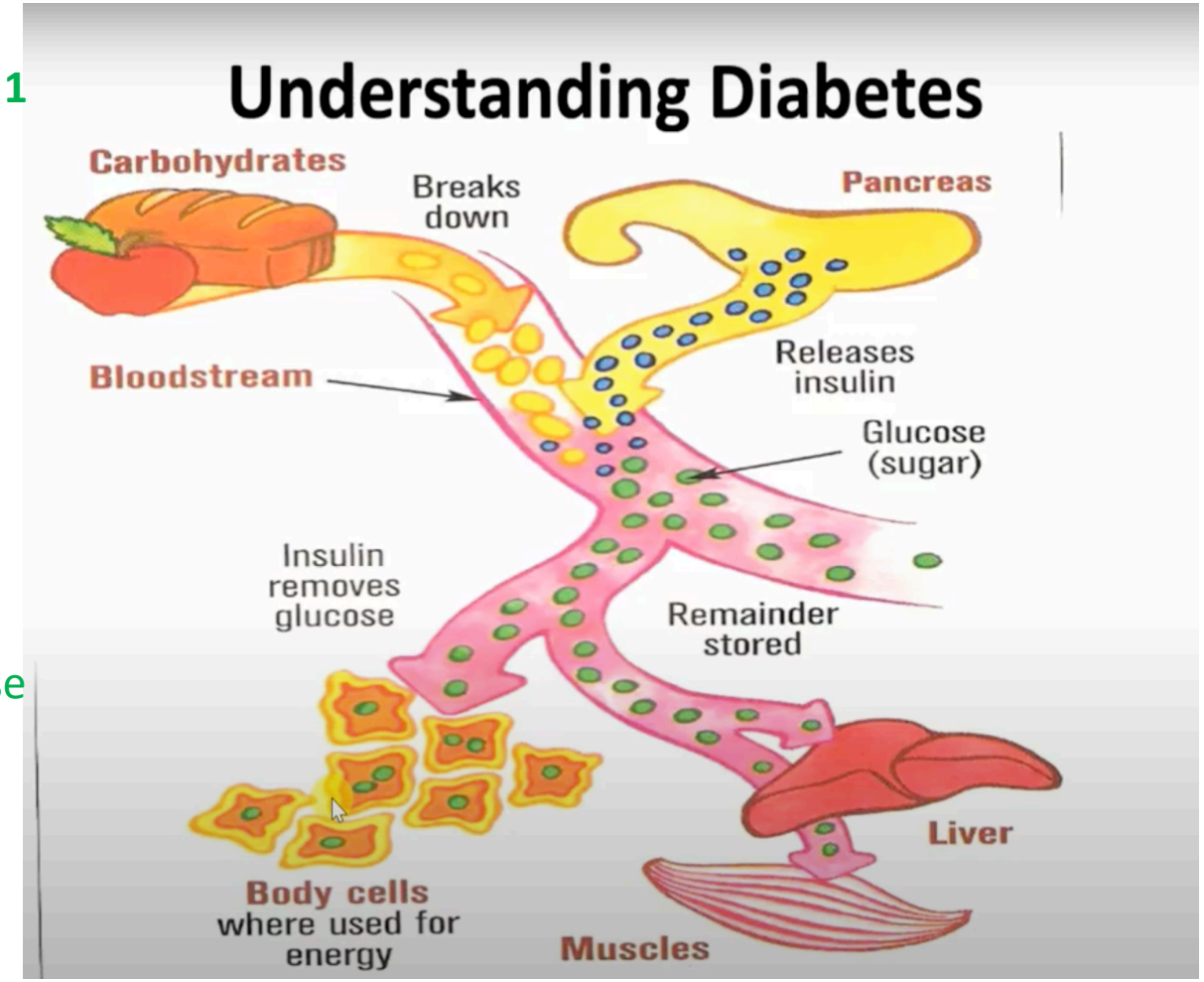
- Diabetes secondary to disease (endocinopathy)such as cushing or acromegaly .

To understand diabetes mainly type 1(معلومات نعرفها من قبل)

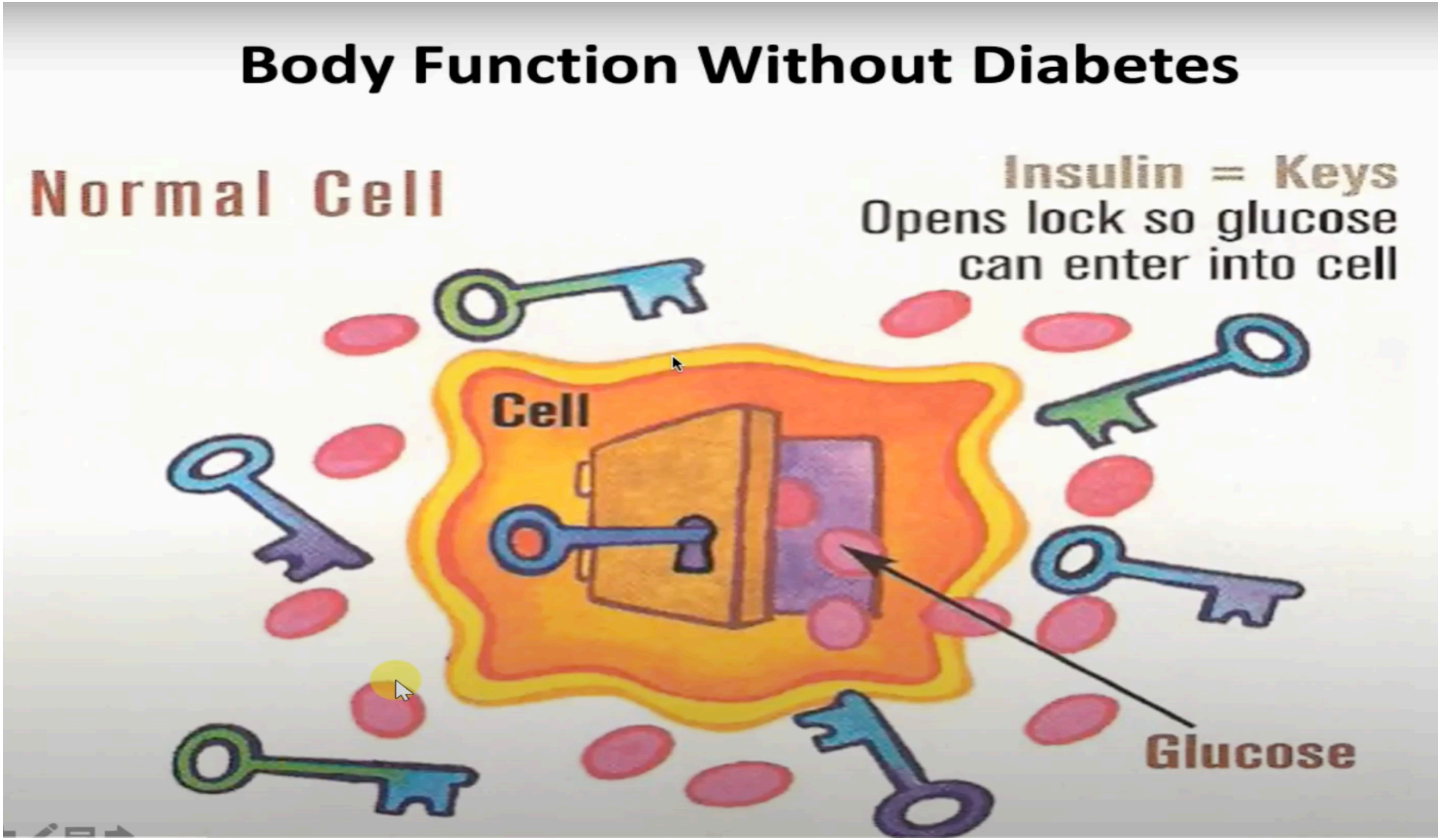
-When the child eats a meal , it will break down to 3 main things :
(glucose , amino acid and fatty acid).
But the main fuel of the body is glucose (the only substrate that will feed the cells).

- If the body needs glucose and there is glucose depletion , then the body will break protein +fats and converts them to glucose, to increase amount of glucose in the blood , regardless how much they were eating .

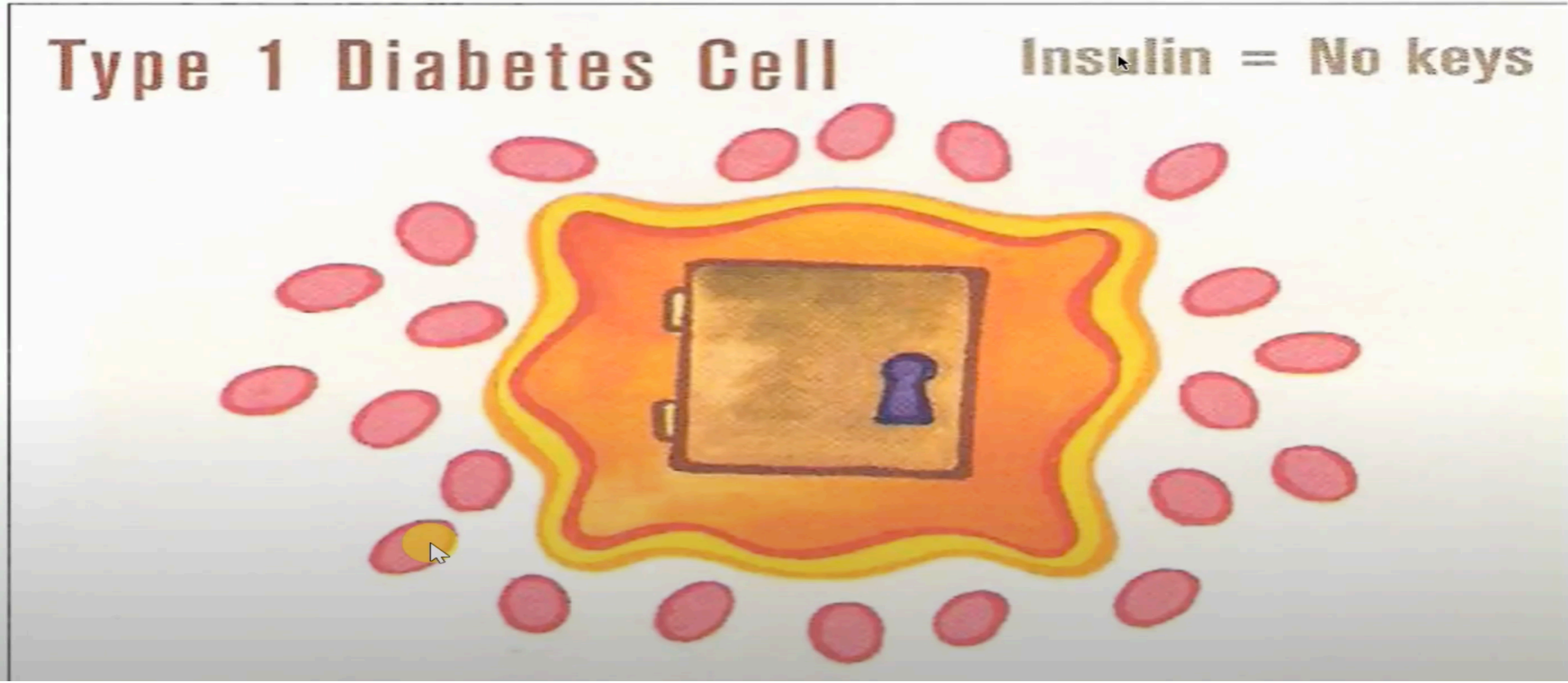
- Glucose needs insulin to enter the cells.



Body Function Without Diabetes



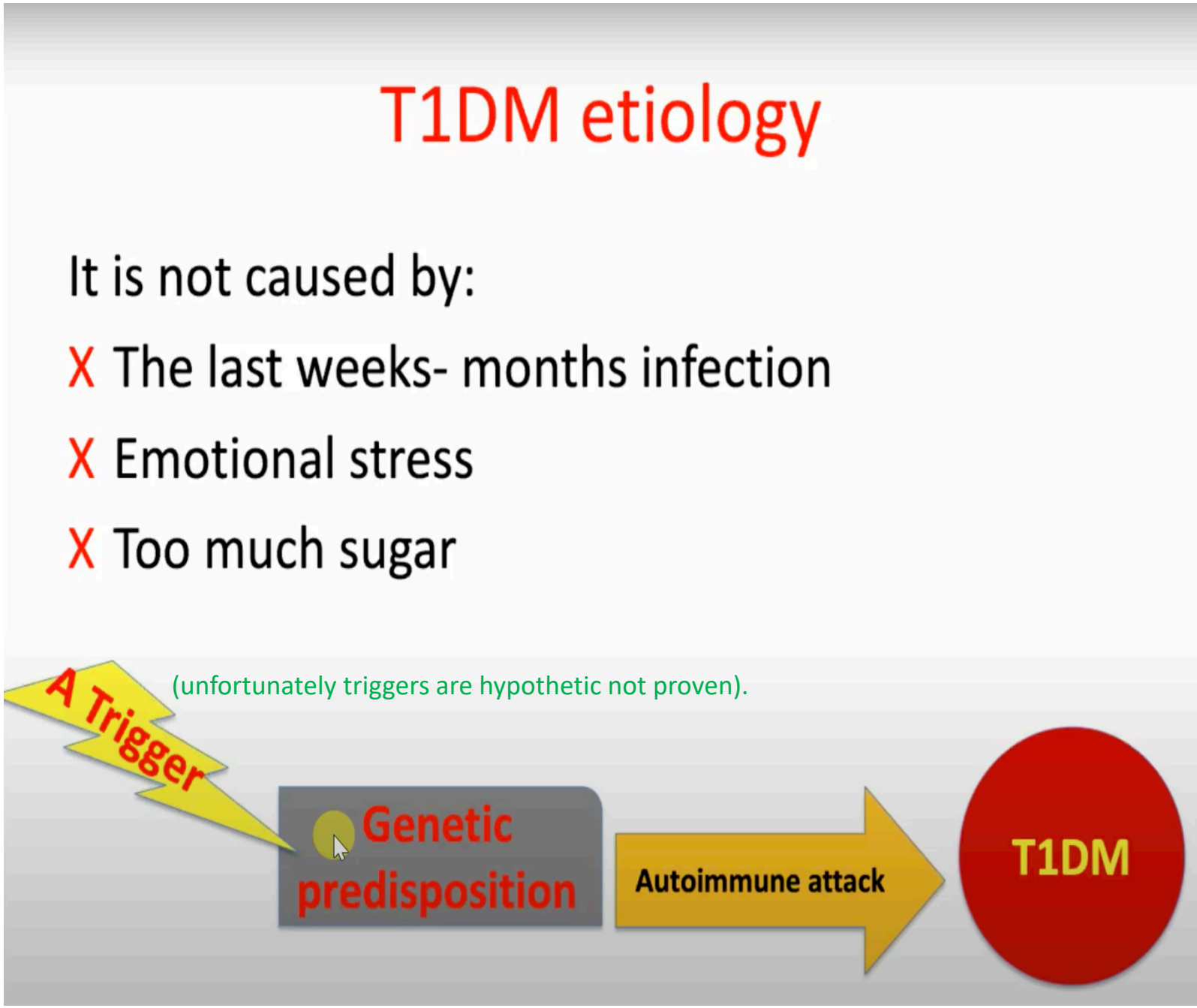
Type 1 diabetes



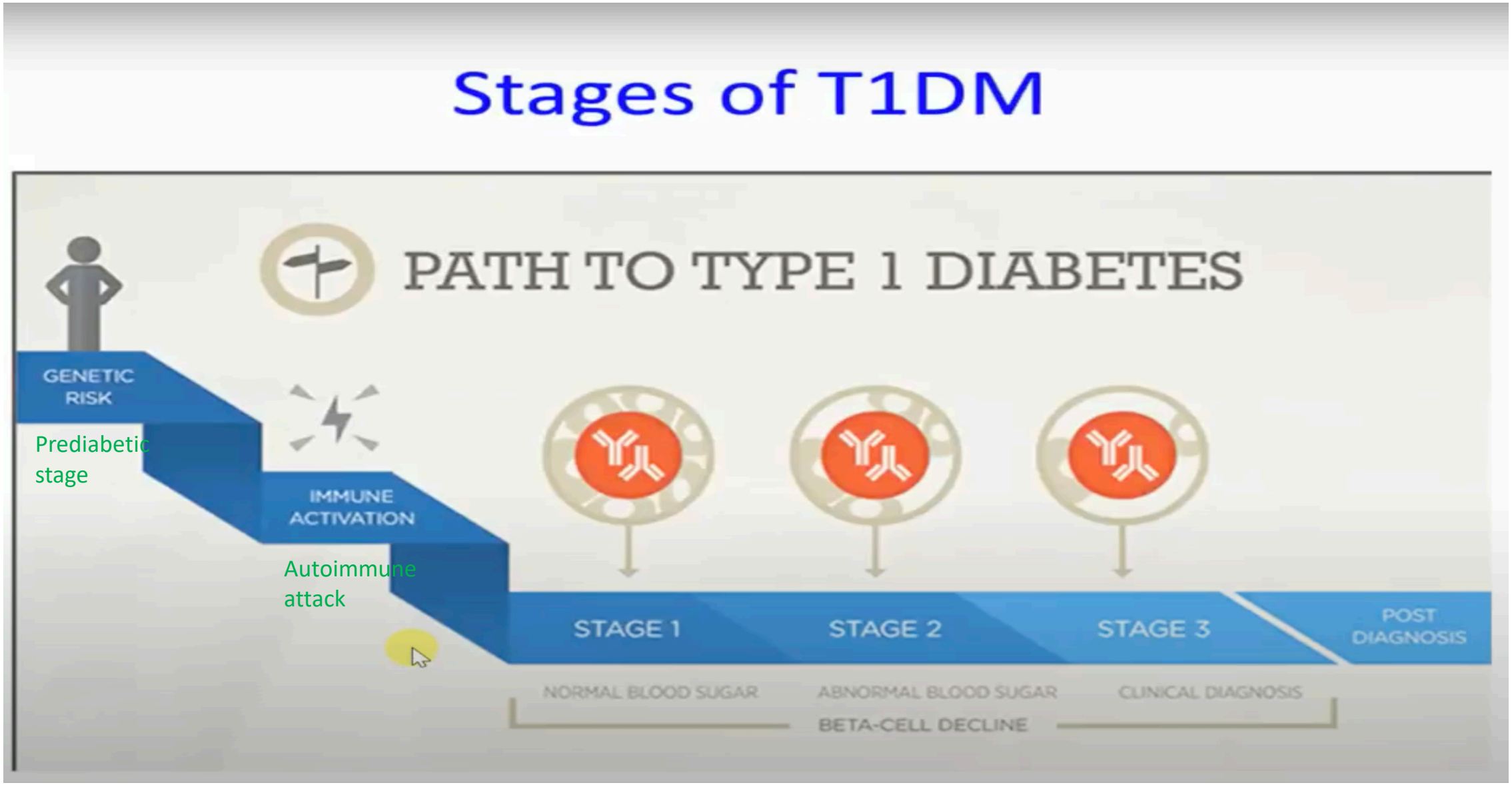
DM1: caused by autoimmune destruction of the pancreas,(how?):

-it happens in those who are genetically predisposed to DM1 ,
once they expose to a trigger (this will trigger an autoimmune attack against the pancreas, and with the destruction that will happen to pancreas over months to years -> type 1 DM eventually will happen once we only have around 5% of beta cells (intact) in pancreas.

- The glucose level will remain steady (NORMAL GLUCOSE LEVEL) until we have only 5% of beta cells(intact).



Stages of T1DM



Stage1: normal blood sugar but you can detect the AB in blood

Stage2: normal blood sugar and AB without symptoms.

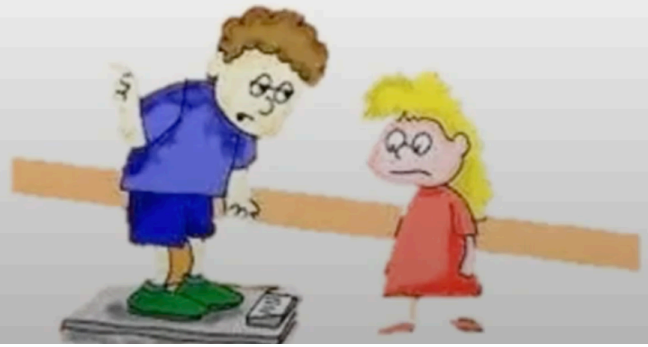
Stage 3: symptoms start clinical diagnosis and AB

- Classical symptoms : polyuria, polydipsia, polyphagia and weight loss

Post dx: chronic long term phase.

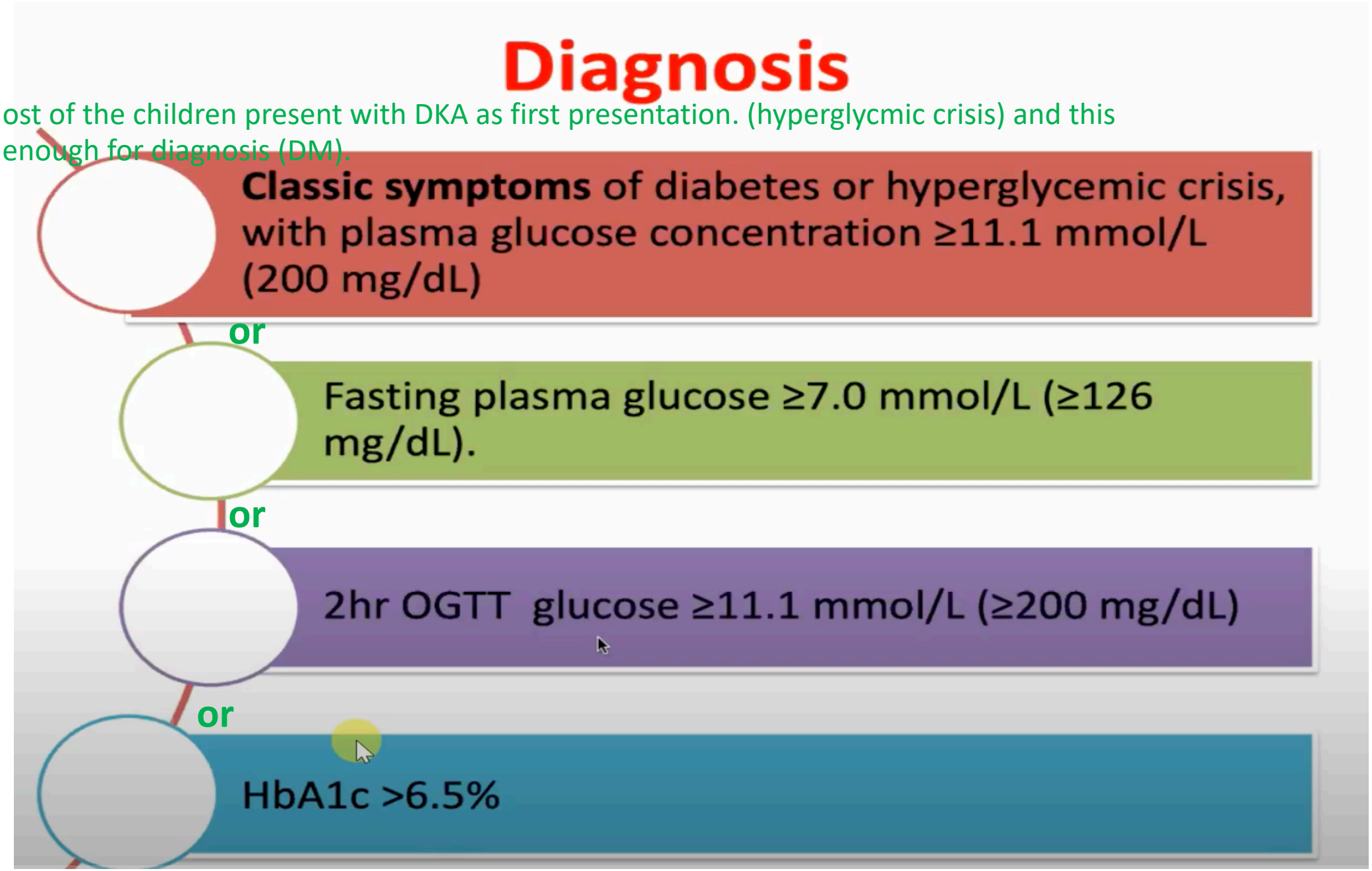
- for all the stage you can detect antibodies (AB) in blood

Symptoms

Polyuria (Frequent Urination) 	Polydipsia (Excessive Thirst) 
Polyphagia (Excessive Hunger/Increased Appetite) 	Involuntary Weight Loss 

Diagnosis

Most of the children present with DKA as first presentation. (hyperglycmic crisis) and this is enough for diagnosis (DM).



TREATMENT

Glucose monitoring

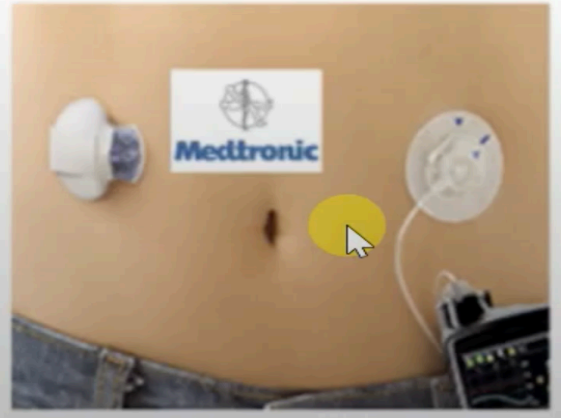
We can measure the glucose level by **glucometer**

Continuous glucose monitoring

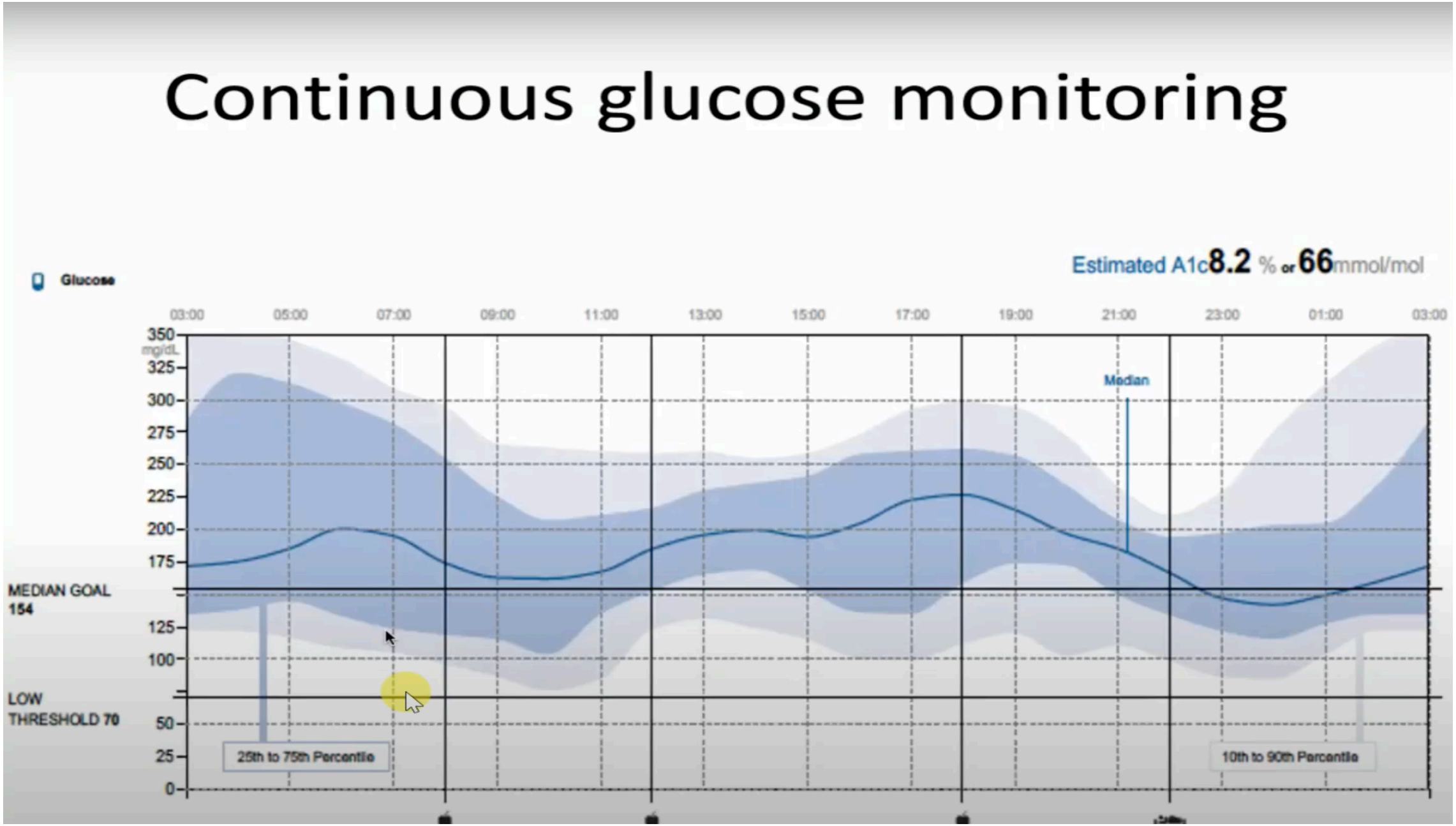
for your information

Or measure it by one of new devices called continuous glucose monitoring device

dexcom | G5™ mobile

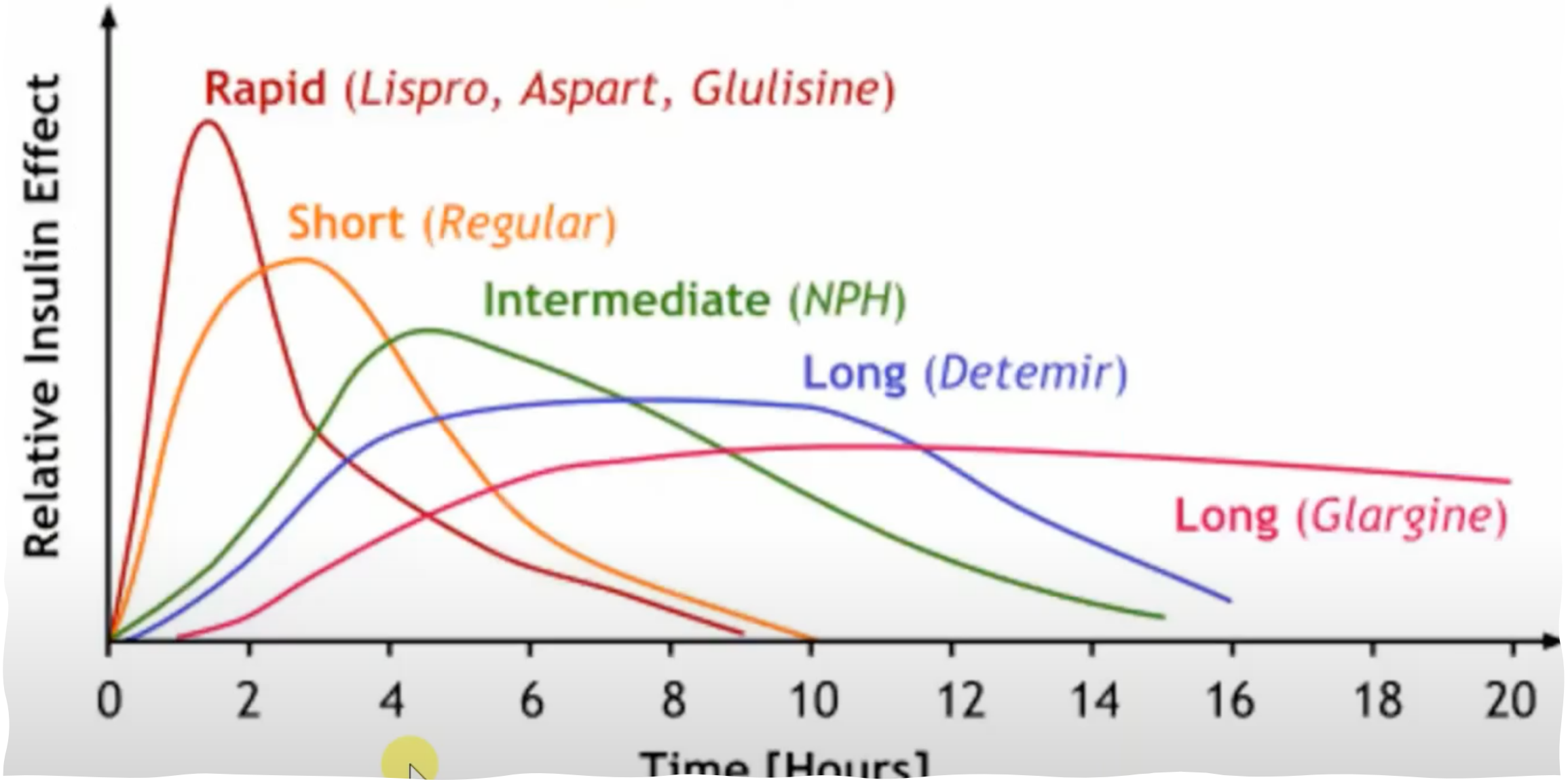


Continuous glucose monitoring



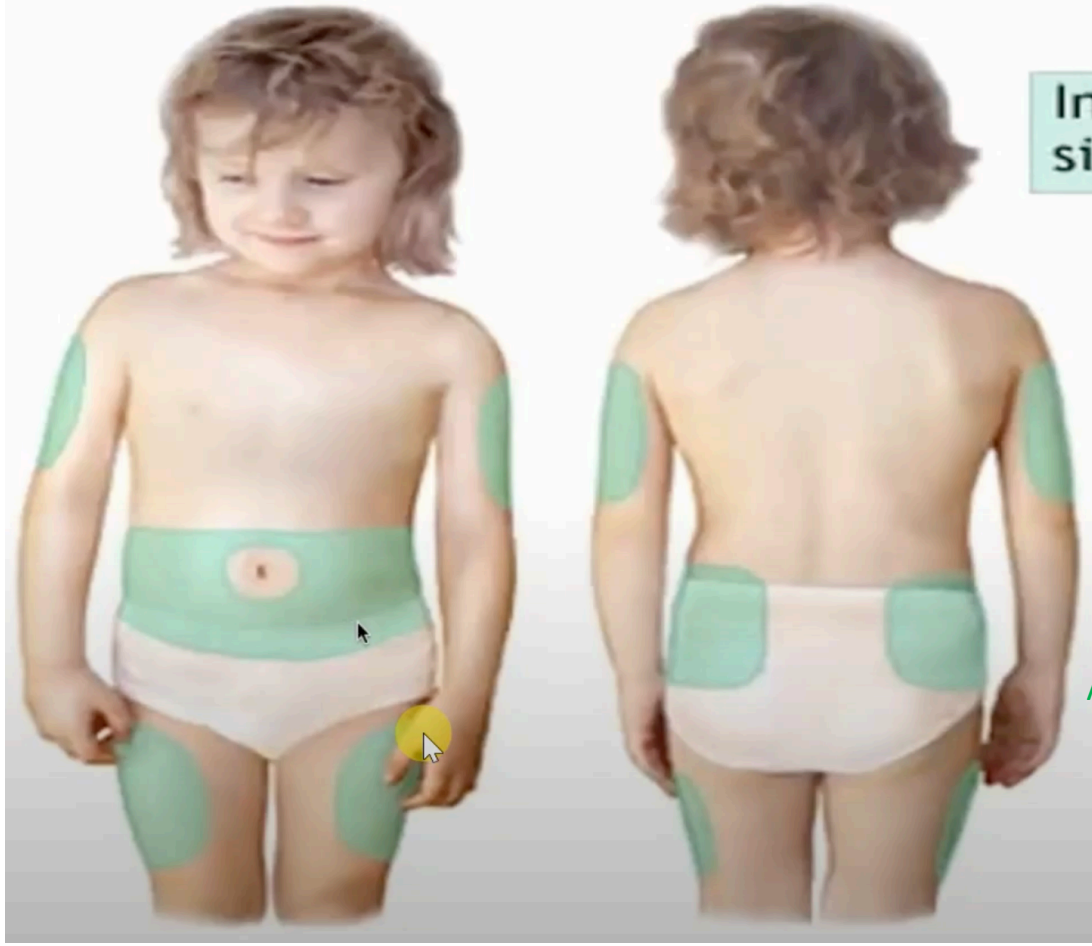
الدكتورة مرت على كل الجدول

Insulin type (trade name)	Onset	Peak	Duration
Bolus (prandial) insulins			
Rapid-acting insulin • Aspart (novorapid®) • Glulisine (apidra™) •Lispro (humalog®)	You have to inject insulin 10- 15min before the meal. 10 - 15 min 10 - 15 min 10 - 15 min	1 - 1.5 h 1 - 1.5 h 1 - 2 h	3 - 5 h 3 - 5 h 3.5 - 4.75 h
Short-acting insulins •Regular (humulin®-r) • Regular (novolin®getoronto)	30 min	2 - 3 h	6.5 h
Basal insulins	Inject it daily (at the same time everyday)		
Intermediate-acting insulins • NPH (humulin®-n) يسمونه العكر لان لونه مو صافي •NPH (novolin®ge NPH)	1 - 3 h	5 - 8 h	Up to 18 h
Long-acting basal insulin • Detemir (levemir®) • Glargine (lantus®) •Degludec (Tersiba)	90 min	Not applicable	Up to 24 h (glargine 24 h, detemir 16 - 24 h) Degludec up to 72hr


Relative Insulin Effect
Rapid (Lispro, Aspart, Glulisine)
Short (Regular)
Intermediate (NPH)
Long (Detemir)
Long (Glargine)
0
2
4
6
8
10
12
14
16
18
20
Time [Hours]

حاولوا تعرفوا اشكالها والوانها لان المرضى كذا يعرفوها

Injection sites



Insulin injection sites: Subcutaneous

- Outer arm
- Abdomen

Avoid 1cm from umbilicus

- Hip area

Buttocks

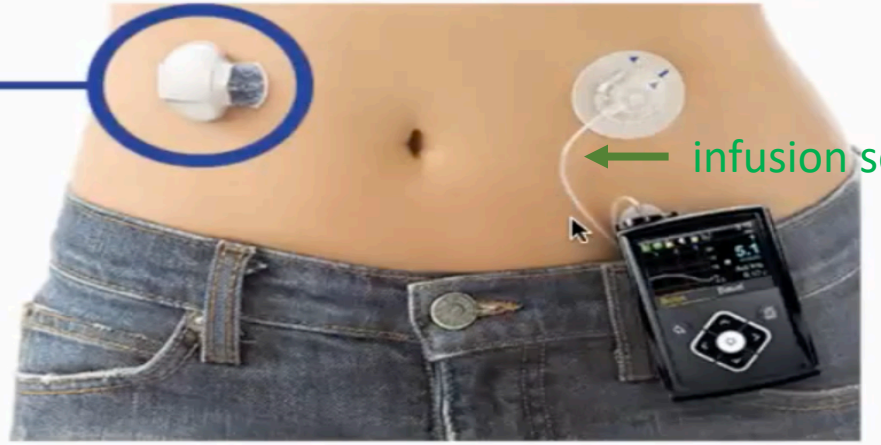
- Thigh

At upper outer side of the thigh

Insulin pump

Sensor for CGM
optional extra

هذا السينسور يحدد اذا الجسم يحتاج انسولين
او لا وعلى أساسه تشتغل البمب



Insulin vial
to fill
reservoir

Reservoir

Insulin Pump

Delivers insulin continuously and
subcutaneous through the catheter
(infusion set).

Infusion set
before insertion

Infusion set
after insertion

Diabetes Management 24/7

Constant Juggling:

Insulin/medication

with:

Exercise

&

Food intake

BG

BG

BG

DIABETES COMPLICATIONS

ACUTE

-hypoglycemia
-DKA

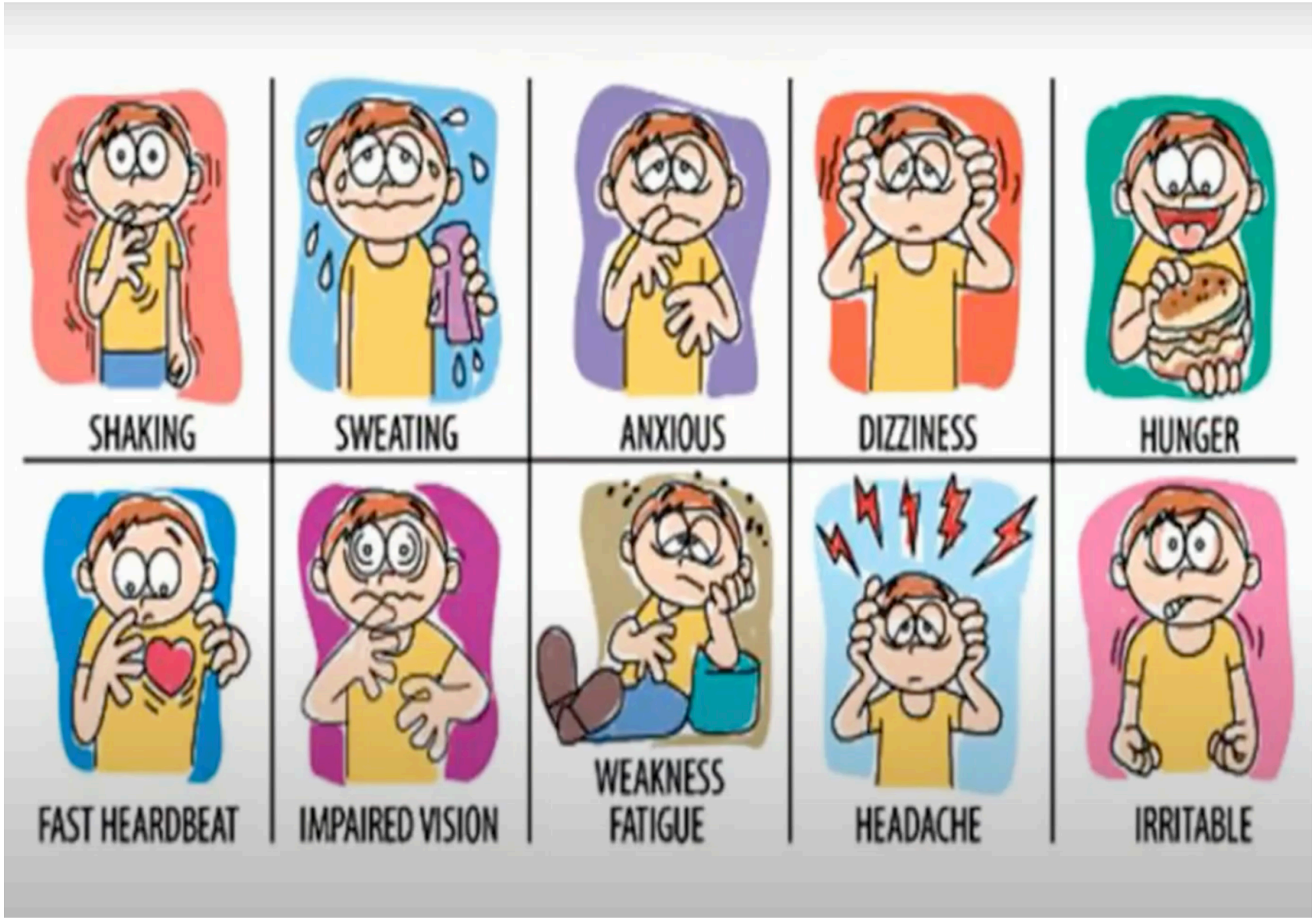
Hypoglycemia

imp numbers hypoglycemia in diabetic patient Less than

70 mg/dl هذي الوحدة تكون موجودة في اجهزة القياس اللي يستخدموها المرضى

4 mmol/l الوحدة اللي يستخدموها المستشفيات

-Hypoglycemia in normal person:
Less than 50 mg/dl
Or 2.8 mmol/l


SHAKING
SWEATING
ANXIOUS
DIZZINESS
HUNGER
FAST HEARDBEAT
IMPAIRED VISION
WEAKNESS
FATIGUE
HEADACHE
IRRITABLE

complications of Hypoglycemia

In case of Recurrent episodes

- Cognitive, psychological changes (eg, confusion, irritability)
- Accidents, Falls
- Low health related quality of life
- hypoglycemia unawareness

  Dangerous

  المريض مايحس ان عنده اعراض الهايبوقلاسيميا ف مايعالج نفسه

  Risk of coma or seizure
- Dementia (elderly)
- CV events: Cardiac autonomic neuropathy, MI, arrhythmia

Treatment of Hypoglycemia

- 1 cup of Juice
- Glucose tablets
- Glucagon s.c

In case of coma , unconscious , seizure , can not swallow.

لما يكون صاحي اعطيه ملعقة عسل او ثلاث حبات تمر كوب عصير ، لكن لما يفقد الوعي اعطيه قلوكاجون او لو عندي اي في اكسس اعطيه محلول سكري على طول

١٢٥ ملل من عصير البرتقال أو التفاح

ملعقة طعام عسل أو مربى

٢ مكعب سكر

٣ حبات تمر

Acute complications

DIABETIC KETOACIDOSIS

DKA

- leading cause of morbidity and mortality in children
- **Risk factors :**
  - new-onset diabetes
  - Children with poor control
  - previous episodes of DKA
  - Adolescent
  - Children on insulin pumps or long-acting insulin analogs
  - Children with psychiatric disorders, and those with difficult family circumstances
  - Poor sick day management

Losses of fluids and electrolytes in diabetic ketoacidosis

	Average (range) losses per kg	
Water	70 mL (30–100)	≤10 kg* 11–20 kg >20 kg
Sodium	6 mmol (5–13)	
Potassium	5 mmol (3–6)	
Chloride	4 mmol (3–9)	
Phosphate	(0.5–2.5) mmol	

+ insulin deficiency status.

Clinical manifestations of DKA

Dehydration

Tachypnea; deep, sighing (Kussmaul) respiration

Sign indicates sever DKA

Nausea, vomiting, and abdominal pain that may mimic an acute abdominal condition

Confusion, drowsiness, progressive obtundation and loss of consciousness

Late stage of DKA

Diagnosis

Hyperglycemia BG > 11 mmol/L (≈ 200 mg/dL)

Ketonemia and ketonuria

Venous pH< 7.3 or bicarbonate < 15 mmol/L

Change in PH by 1
Change in bicarb by 5

DKA severity

- Mild: venous pH<7.3 or bicarbonate <15 mmol/L
- Moderate: pH<7.2, bicarbonate <10 mmol/L
- Severe: pH<7.1, bicarbonate <5 mmol/L.

Management

- Airway-breathing-circulation
- If shock ……..> give fluid bolus

Fluid replacement

DKA patient has deficit in both total body water and electrolytes.

- Deficit + maintenance

DKA severity

For 24 hr

Role : 4, 2, 1

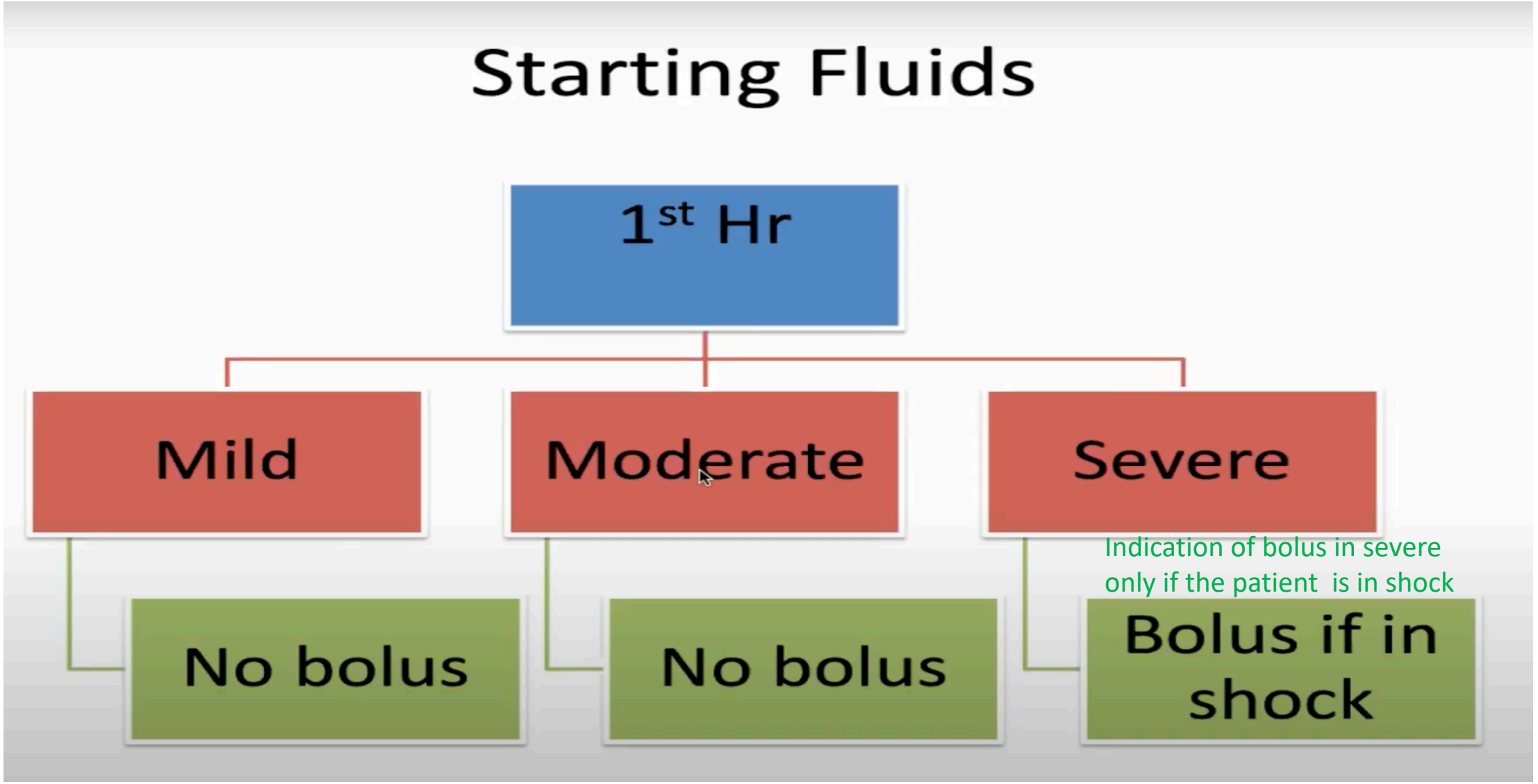
For fluid replacement in DKA,we have to take two factors in consideration : **1- fluid deficit from DKA 2- amount of maintenance fluid that they need**.

-With increase of DKA severity we need to replace them slowly over long time peroid to avoid complications.

Deficit: Weight X deficit %(based on DKA severity) X 10

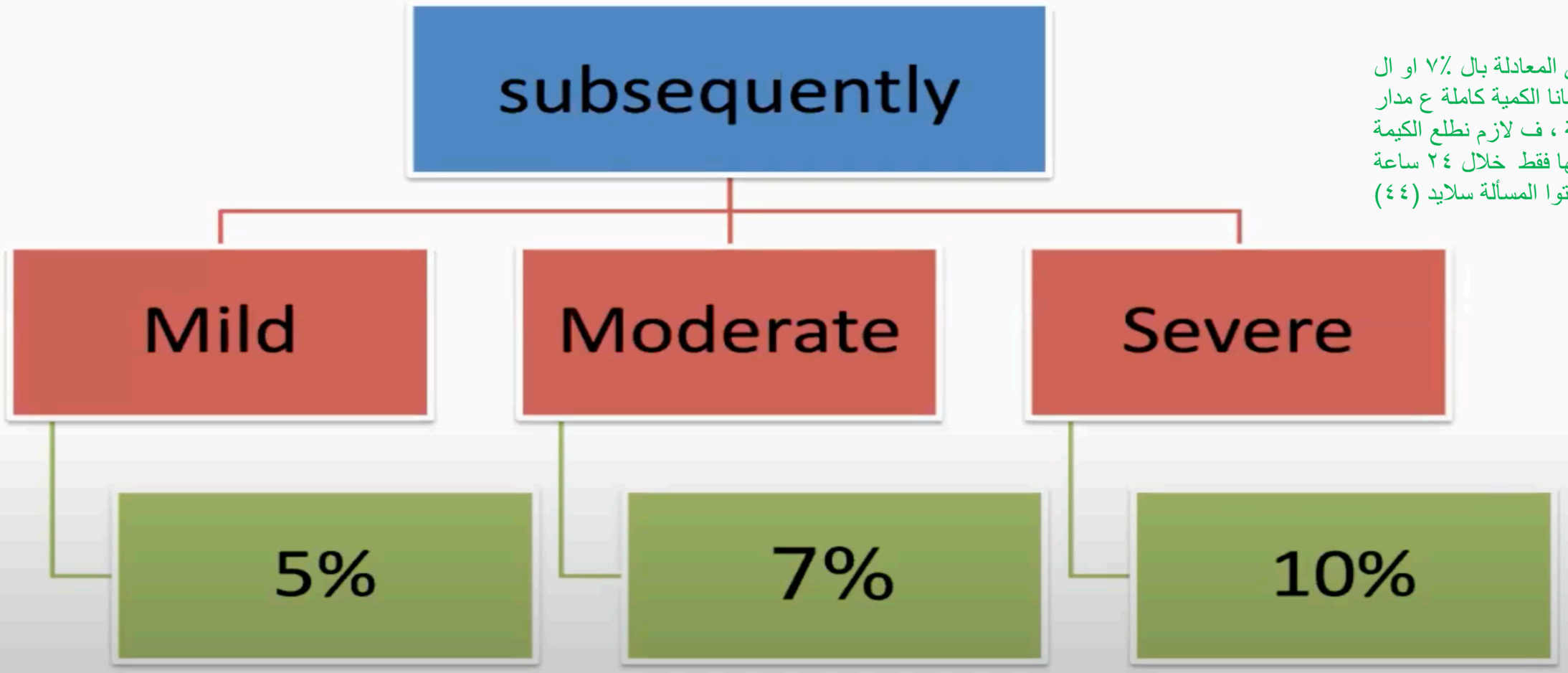
Calculate the severity: (IMP NUMBERS)
- Mild :5% (GIVE FLUID over 24H)
- Moderate :7% (Give fluid over 48 - 72h)
-Severe: 10% Give fluid over 48- 72h)

Starting Fluids



Deficit replacement

IMP



- لما نعوض في المعادلة بال ٧٪ او ال ١٠٪ حيطلع معانا الكمية كاملة ع مدار يومين الى ثلاثة ، ف لازم نطلع الكيمة اللي يحتاجها فقط خلال ٢٤ ساعة حتفهموا اذا حليتوا المسألة سلايد (٤٤)

DKA severity

IMP

لما يصير فيه اختلاف بين PH and Bicarb ناخذ الأسوأ و نحدد السيفرتي

- Mild: venous pH<7.3 or bicarbonate <15 mmol/L
- Moderate: pH<7.2, bicarbonate <10 mmol/L
- Severe: pH<7.1, bicarbonate <5 mmol/L.

Your turn !

IMP for exam

Severity of DKA:
It is severe
حسب السلايد اللي قبل

- 10 years old girl, has 2 weeks history of polyuria, polydepsia, vomiting for 1 day.
- Glucose 30 mmol/l
- Urine Ketones +4
- Gas: PH= 7.1, Hco3 =4, Co2 =12
- Weight 30 kg

- **Calculate the required fluids in the first 24hr?**

Wt 30kg, severe DKA

- Maintenance= 1680ml/ 24hr

4,2,1
الناتج رح يطلع معانا ٧٠ لكن هذا احتياجه في الساعة ف لازم نضرب في ٢٤ ساعة =١٦٨٠ في اليوم

Deficit: Weight X deficit %(based on DKA severity) X 10

- Deficit= 30 X 10 X 10= 3000ml/ 48-72hr
  = 1500 ml/24hr

هنا ال١٠ اللي عوضناها لانه طلع Severe

- Required fluids in the 1st 24hr= 1680+ 1500
  = 3180ml/24hr
  = **132ml/hr**

Type of fluids

NACL	KCl	Glucose
0.9 % NS Start with	40 mmol/l	D 5 W When glucose drop below 50 mmol\L we add D5 to the fluids then D10 then D12.5 ,Depend on the case
0.45 % NS then	60 mmol/l	D 10 W
We start with NS in all patients then we change after 12hr, especially when we start to deal with hyperkalemia	Add kcl gradually (START WITH 40 THEN 60)especially when we give insulin (cause intracellular shift of K+)	D 12.5 W

- Aim to keep glucose more the 7 mmol\l

Fluid Basics

- Avoid hypervolemia / rapid osmo correction
  Avoid ripid correction (risk of cerebral edema)
- No insulin 1st hr
  (because risk of cerebral edema)
- No insulin bolus
  (because risk of cerebral edema)
- No $NaHCO_3$
  (because risk of cerebral edema)
- Replace all electrolyte deficit

Monitoring

- Gas Every 2 hr
- Electrolytes Every 4 hr
- Glucose Every 1 hr
- Renal function Every 4 hr
- Urine ketones With every urine
- Others Ex: Mg, phosphorus

DKA compilations

- Acute: Cerebral edema –electrolytes imbalance.
- Chronic

Risk Factors for Developing Cerebral Edema

- 0.7 to 3.0%
- Younger age (<5 years)
- New-onset diabetes
- High initial serum urea
- Low initial partial pressure or arterial carbon dioxide (pCO_2)
- Rapid administration of hypotonic fluids
- IV bolus of insulin
- Early IV insulin infusion (within 1st hour of fluids)
- Failure of serum sodium to rise during treatment
- Use of bicarbonate

Signs and symptoms of cerebral edema

- Headache and slowing of heart rate (Typical)
- Change in neurological status (restlessness,
- irritability, increased drowsiness, and incontinence)
- Specific neurological signs (e.g., cranial nerve palsies, papilledema)
- Rising blood pressure
- Decreased O2 saturation

Diagnostic criterion:

No need to do imaging , it is clincal diagnose.

- 1 Diagnostic criteria
- **or**
- 2 major criteria
- **or**
- 1 major and 2 minor criteria
- Signs that occur before treatment should not be considered in the diagnosis of cerebral edema.

Diagnostic Criteria

Having 1 (+) diagnostic criteria is enough
to diagnose with cerebral edema.

- Abnormal motor or verbal response to pain
- Decorticate or decerebrate posture
- Cranial nerve palsy (especially III, IV, and VI)
- Abnormal neurogenic respiratory pattern (e.g., grunting, tachypnea, Cheyne–Stokes respiration, apnea)

2 major criteria

or

1 major + 2 minor criteria

- **Major Criteria**
  - Altered mentation/fluctuating level of consciousness
  - Sustained heart rate deceleration (decrease more than 20 beats/min) not attributable to improved intravascular volume or sleep state
  - Age-inappropriate incontinence
- **Minor criteria**
  - Vomiting
  - Headache
  - Lethargy or not easily arousable
  - Diastolic blood pressure >90mmHg
  - Age < 5 years

Treatment

- Initiate treatment as soon as the condition is suspected.
- Reduce the rate of fluid administration by one-third.
- Give mannitol, 0.5–1 g/kg IV over 10–15 min, and repeat if there is no initial response in 30 min to 2 h.
- Hypertonic saline (3%), suggested dose 2.5–5 mL/kg over 10–15 min
- Move patient to PICU. /Consult neurosurgery.
- Elevate the head of the bed to 30◦.

DIABETES CHRONIC COMPLICATIONS

Diabetes Complications – Key Messages

- Nephropathy, retinopathy, neuropathy and hypertension are relatively rare in pediatric diabetes

- Start Screening after pubertal and 5 years of DM duration. Then the screening will be every year.

Screening for:

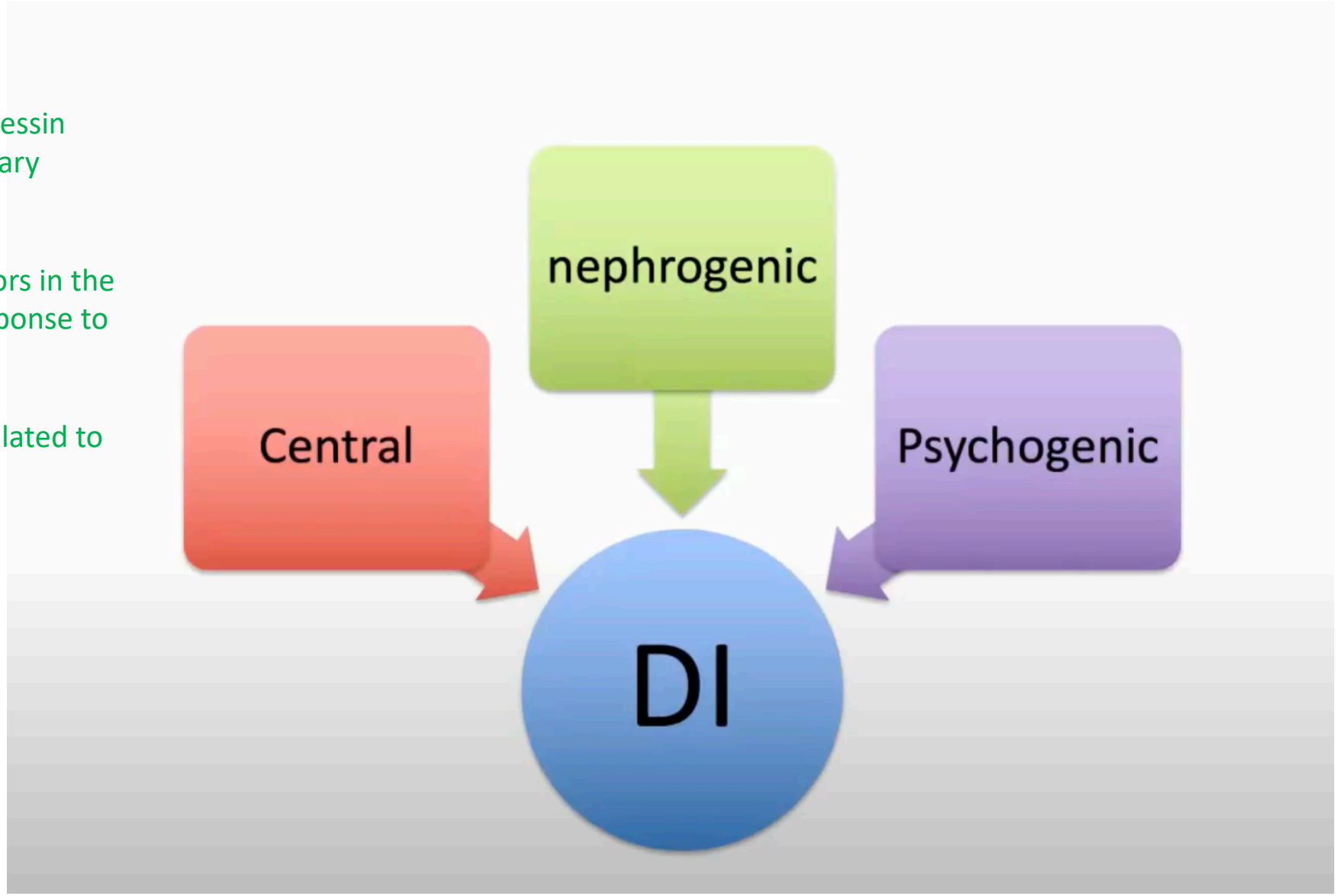
- Nephropathy -> alb/cr ratio.
- Retinopathy -> send patient to optha for ophthalmoscope or retinal imaging (more accurate).
- Neuropathy : physical ex.
- HTN : checking BP.

Diabetes insipidus

- Central: deficiency in secretion of desmopressin (ADH) from the pituitary gland.

- Nephrogrnic: receptors in the kidney have poor response to desmopressin (ADH)

- Psychogenic: is not related to true pathology.



As you remember:

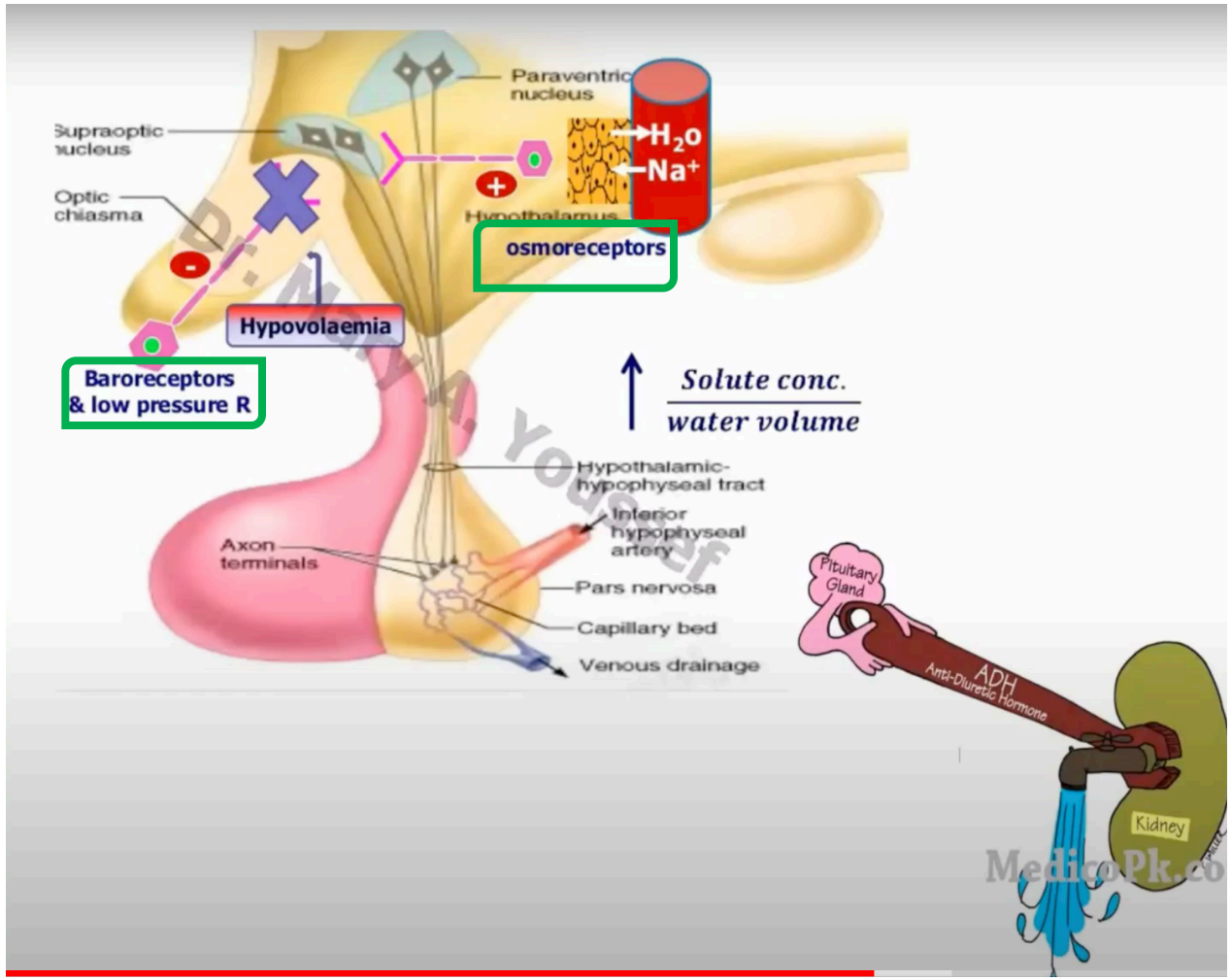
Central DI

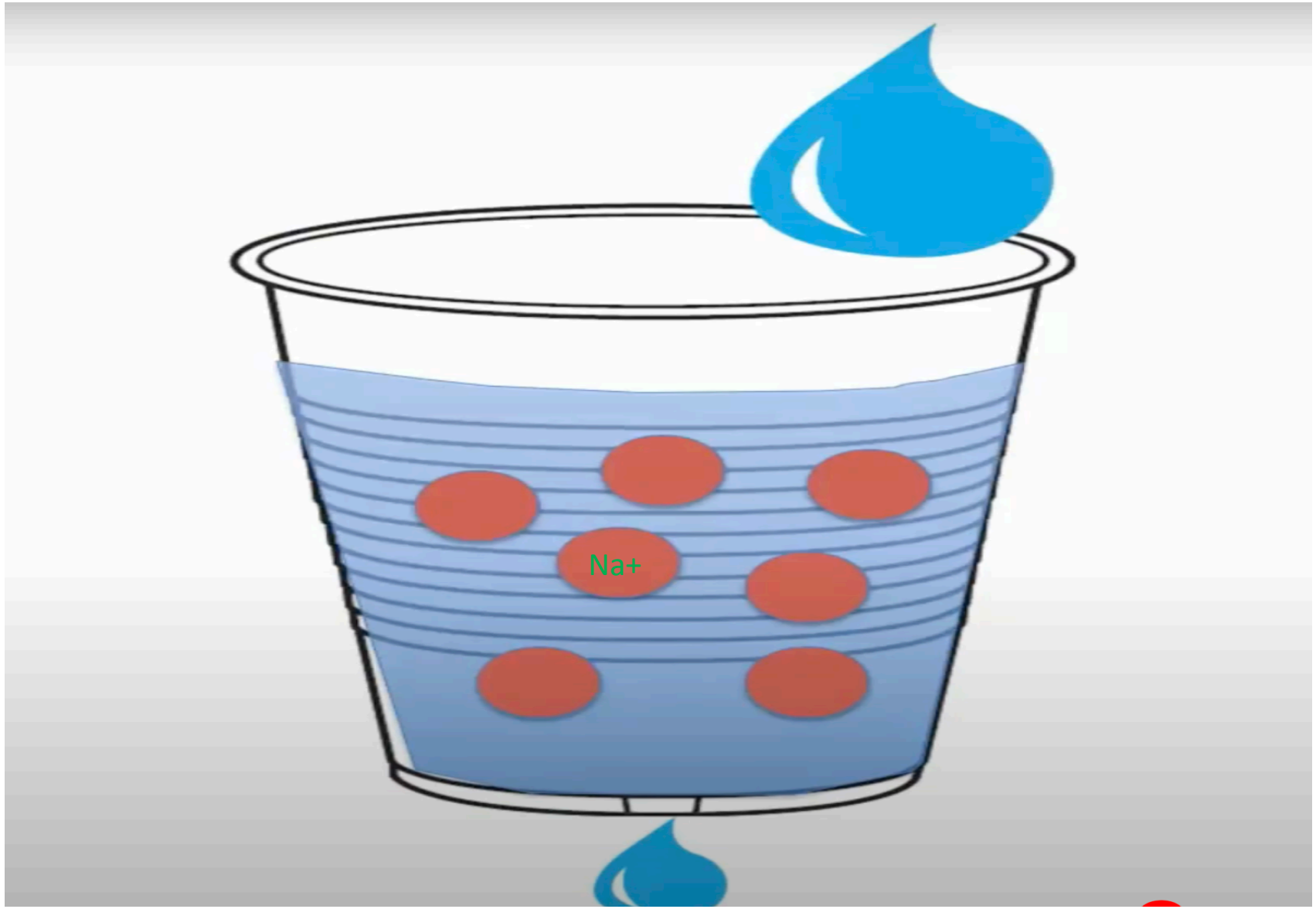
- Pathophysiology: ADH (desmopressin) manufacture in hypothalamus and stored in posterior pituitary, ADH controlled
how much water reabsorbed from renal tubules, so if you don't have ADH(desmopressin) you will loss water كانها حنفية
مفتوحة) uncontrolled) = polyuria then develop polydipsia because of this loss، then the Na concentration
will become high due to this loss (hypernatremia)

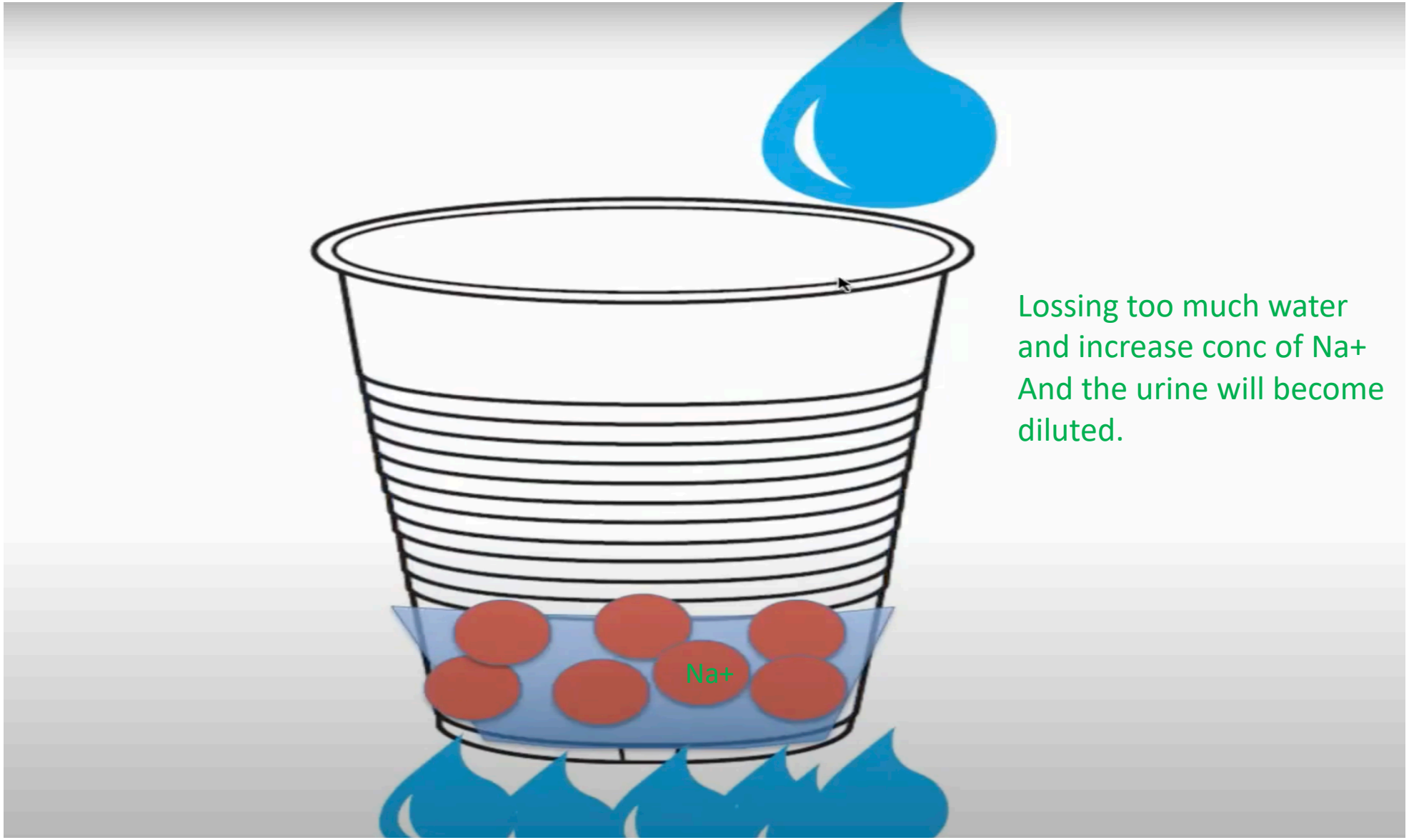
-There are receptors in hypothalamus called **osmoreceptors** that sense the osmolarity in the blood, and we have **baroreceptors** in optic chiasma sense the blood pressure (hypo/hyper/ euvolemia).

- Any change in BP or osmolarity will stimulate
the secretion of desmopressin from the pituitary gland.

- The action of decompression (ADH) works on the kidney and closes it as if you close the water pipe so the kidney (decreases urine loss).




Na+



Lossing too much water
and increase conc of Na+
And the urine will become
diluted.

Symptoms

- Polyuria
- Polydipsia
- Dehydration
- Irritability
- Growth failure
- Hyperthermia
- Weight loss

In very young children

Investigations

- ↑ Na
- ↑ serum osmolality
- ↓ Urine osmolality
- ↓ Urine Na
- ↓ Urine Specific gravity

Lossing too much water and increase conc of Na+ And the urine will become diluted.

-At end of the day during fasting normally the person stops peeing.

-During Fajr and dhohur you go to toilet, after dhohur you stop because kidney shut off (it is trying to preserve water) if you continue to lose urine,(risk of dehydration).

- **Normal response:** normal serum Na And the urine will stop.
- **Abnormal:** increase urine loss every hour (polyuriea) + increase serum Na -> DI is confrimed

Give desmopressin to differentiate between central and nephrogenic.
- **Central:** urine is concentrated
- **Nephrogenic :** no change in the urine (why)? Because the kidneys (receptors) has abnormal respond to desmopressin .

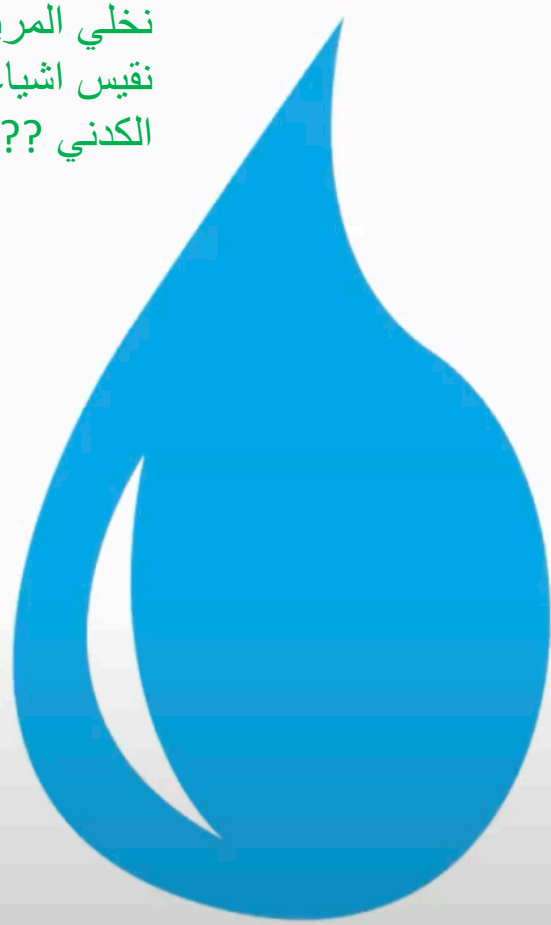
- **Psychogenic DI:** normal water deprivation test (normal serum Na And the urine will stop).

How to test for Di ?

Water deprivation test 8-10hr

نخلي المريض يصوم عن السوائل من ٨-١٠ ساعات
نقيس اشياء معينة كل ساعة عشان نشوف ردة فعل الكدني ?? shut off or continue losing urine

- Q1hr:
  - serum Na
  - serum osmolality
  - Urine osmolality
  - Urine Na
  - Urine Specific gravity
  - Weight
  - +/- desmopressin injection



Causes Centera DI

Congenital	• Agenesis of the pituitary • Septo-optic dysplasia	Common in baby Less than 1 y
Tumor	• Craniopharyngioma • Histocytosis	Common in child more than 1-2 y/o
Iatrogenic	• Surgical removal • Radiation	
Infectious	• Meningitis	

Treatment

- Desmopressin
- Free water access

How to replace desmopressin?
- **(Iv – subcutaneous**) -> at hospital
- **(Intranasal – sublingual**) -> at home

- Most of those patients can not fast (risk of dehydration and shock)

Ex:

Target Na: 140 (كم انا ابغى الصوديوم يوصل)

Actual Na: 155

Weight :20 kg

نعوض في المعادلة

Fluid deficit = +1.28 L

Fluid replacement

- Deficit + maintenance + ongoing

Water deficit	For 24 hr	Urine output

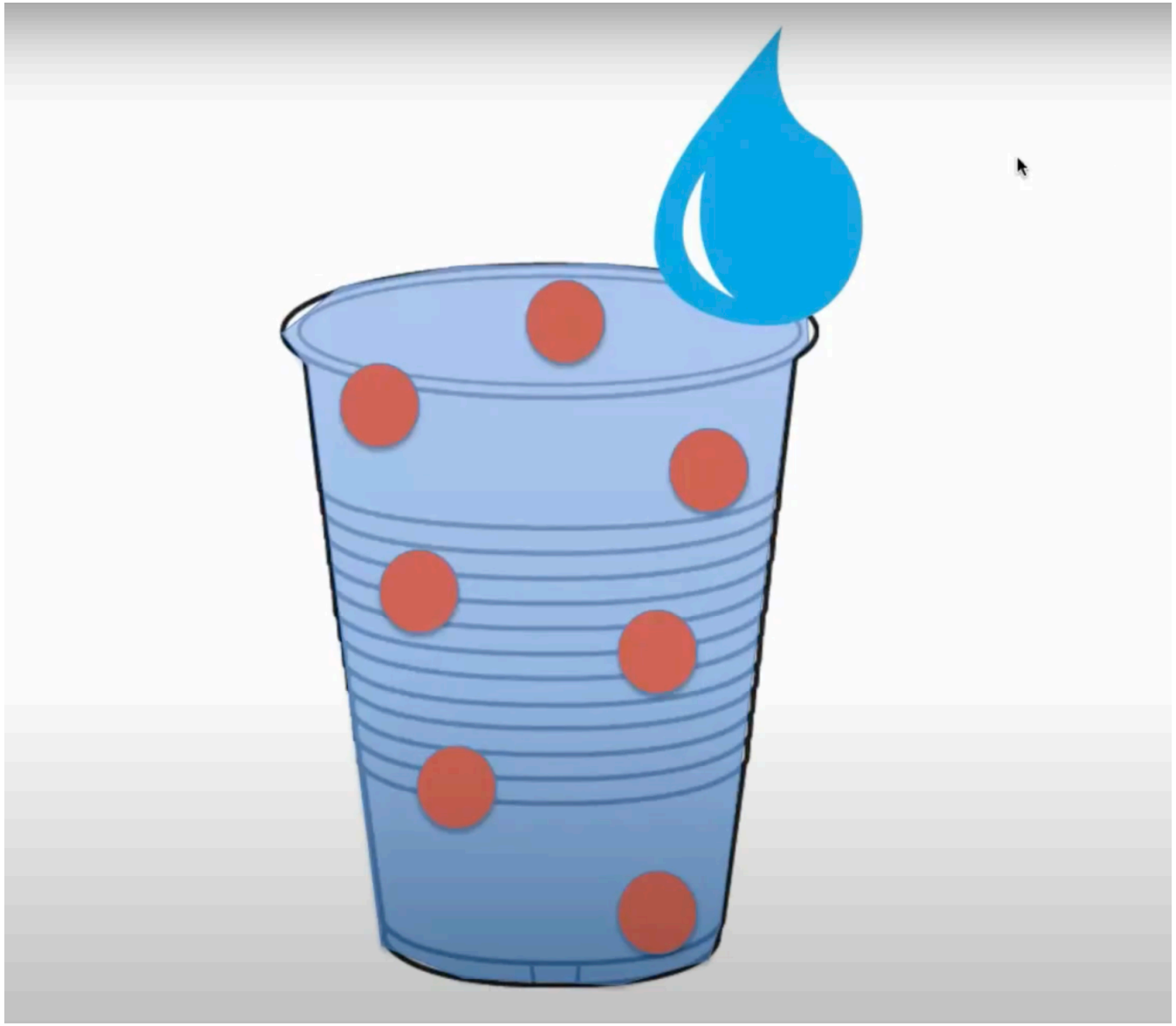
Deficit: 0.6X weight X $\frac{\text{(actual Na-target Na)}}{\text{target Na}}$

Target Na: 140

Actual Na : Na of the patient

SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE

-Pathophysiology: no water loss (opposites of DI) all of the fluids will reabsorbed again فيضان داخلي بالجسم as the excess water in body there will be low Na (diluted hyponatremia)



Urine is not excreting and we still adding water.

- Low serum Na

Symptoms

- Anorexia
- Nausea
- Muscle cramps
- Weakness
- Confusion
- Seizure Why? Electrolyte imbalance (severe hyponatremia) -> less than 120
- Coma

Causes Of SIDH

Tumor	• Small cell Lung ca
Iatrogenic	• Postoperative fluid load • Postoperative pituitary stalk injury
Infectious	• Meningitis • GBS pneumonia
Medications	• Anti seizure • Chemotherapy • Lithium

Most common in pedia

Investigations

- ↓ Na
- ↓ serum osmolality
- ↑ Urine osmolality If he pee
- ↑ Urine Na
- ↑ Urine Specific gravity

Treatment

- Free Water restriction

Ex:
Target Na: 140
Actual Na: 120
Weight :20 kg
نعوض في المعادلة
Fluid excess = -1.7 L

Fluid management

- Deficit + maintenance + ongoing

- Don’t replace fluid quikly (risk of cerebral pontine demyelination)

Fluid excess | For ~~24~~ hr | Urine ~~output~~

Deficit: 0.6X weight X $\frac{\text{(actual Na-target Na)}}{\text{target Na}}$

Answer (-) -> the amount of fluid that patient has to urinate it To bring the Na back to normal

-هنا الجواب اللي حيطلع معانا (-) (وهي الكمية اللي المفروض المريض **يطلعها** عشان يرجع لتوازن السوائل الطبيعي) (الناتج حيكون بالسالب -)
- عكس معادلة Di (الناتج + وهي الكمية اللي المفروض **نعطيها** للمريض عشان يرجع لتوازن السوائل) (الناتج حيكون بالموجب +)

Case

Diagnosis -> DI in this case

- Normal average of pee -> 5-6 times / day.

- A 3-year old boy polyuria/polydipsia since age 10 months
- He drinks about 2.5 to 3 litres of fluid per day
- He gets up twice at night to drink, mother changes dippers 12 times daily

- What is the most likely test to confirm your diagnosis?
  - Serum Glucose
  - Serum Na
  - Blood gas
  - Urine culture

Serum glucose (false answer) why? Because if he has DM he will present with DKA at younger age

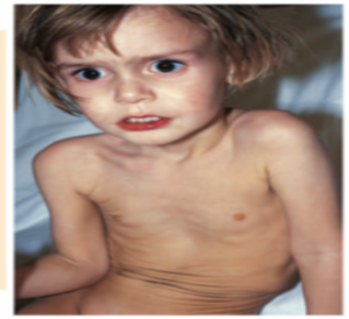
ماحيجلس سنتين متواصلة عنده DKA. اذا كان عمره شهرين ممكن وقتها نفكر في DKA .

The typical story of DM1 -> 2 week of symptoms(polyuria, polydipsia and polyphagia) present with DKA. If someone came with same symptoms but duration is month or year this is not any more DM1, think for other ddx such as DI (without polyphagia)or DM2.

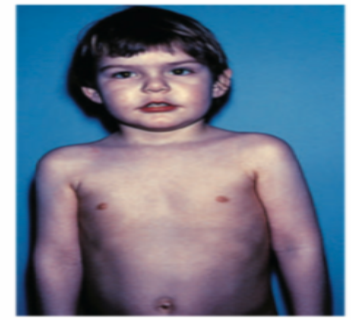
Diabetic ketoacidosis

Box 26.4 Essential early investigations in diabetic ketoacidosis

- Blood glucose (>11.1 mmol/L)
- Blood ketones (>3.0 mmol/L)
- Urea and electrolytes, creatinine (dehydration)
- Blood gas analysis (severe metabolic acidosis)
- Evidence of a precipitating cause, e.g. infection (blood and urine cultures performed)
- Cardiac monitor for T-wave changes of hypokalaemia
- Weight (compare with recent clinic weight to ascertain level of dehydration)



(b)



(c)

(a) Diabetic ketoacidosis management

Follow this regimen if: hyperglycaemia (blood glucose >11 mmol/L, acidosis (pH <7.3 and/or bicarbonate <15 mmol/L), blood ketone usually >3 mmol/L and clinical dehydration and/or vomiting, drowsy or clinically acidotic. (Follow guidelines from the British Society of Paediatric Endocrinology and Diabetes to reduce the risk from hypokalaemia, aspiration and cerebral oedema).

1. Fluids

If in shock, initial resuscitation is with 0.9% saline (10 ml/kg). Dehydration should then be corrected gradually over 48 hours (see Fig. 26.7b and c). Rapid rehydration should be avoided as it may lead to cerebral oedema. Initial rehydration fluids need to be taken into account in calculating fluid requirements. 0.9% saline with 40 mmol/L KCl is recommended for first 12 hours, adding 5% glucose when blood glucose <14 mmol/L. After 12 hours, if plasma sodium level is stable, 0.45% saline/5% glucose with 40 mmol/L KCl is recommended. Monitor:

- fluid input and output
- blood glucose (hourly), blood ketones (1-2 hourly), electrolytes, creatinine and acid–base status 2–4 hourly
- neurological state.

Consider transfer to PICU and central venous line (CVP) and urinary catheter if shocked or in coma. A nasogastric tube is passed for acute gastric dilatation if there is vomiting or depressed consciousness.

2. Insulin

Insulin infusion (0.1 units/kg per h) is started after intravenous fluids running for 1 hour. Do not give a bolus. Monitor the blood glucose hourly. Aim for gradual reduction of blood glucose . Change to a solution containing 5% glucose when the blood glucose has fallen to 14 mmol/L to avoid hypoglycaemia.

3. Potassium

Although the initial plasma potassium may be high, due to displacement from cells in exchange for hydrogen ions, it will fall following treatment with insulin and rehydration. Potassium replacement must be instituted as soon as maintenance fluids are started (unlike adults, it can be assumed that the child will have normal renal function and the greatest risk is from total body potassium depletion). Continuous cardiac monitoring and 2–4 hourly plasma potassium measurements are indicated until the plasma potassium is stable.

4. Acidosis

Although a metabolic acidosis is present, bicarbonate should be avoided unless the child is shocked. The acidosis will correct with fluid and insulin therapy.

5. Re-establish oral fluids, subcutaneous insulin and diet

Do not stop the intravenous insulin infusion until 1 hour after subcutaneous insulin has been given.

6. Identification and treatment of an underlying cause

Ketoacidosis may be precipitated by an intercurrent infection. Diabetic ketoacidosis causes neutrophilia but not a fever. Antibiotics may be indicated. If the child was known to have diabetes, consider the reason for the ketoacidosis.

Figure 26.7 **(a)** Diabetic ketoacidosis management; **(b)** boy with severe dehydration and weight loss from diabetic ketoacidosis; and **(c)** 4 months later. (Photos b and c courtesy of Jill Challener.)

Summary

Regular assessment of the child with diabetes

Assessment of diabetes:

- Any episodes of hypoglycaemia, diabetic ketoacidosis, hospital admission?
- Is there still awareness of hypoglycaemia?
- Absence from school? School supportive of diabetes care?
- Interference with normal life?
- HbA_{1c} results – less than 48 mmol/mol (6.5%)?
- Diary of blood glucose results or blood glucose read-out– are appropriate actions to results being taken?
- Insulin regimen – appropriate?
- Lipohypertrophy or lipoatrophy (Fig. 26.8 a and b) at injection sites?
- Diet – healthy diet, manipulating food intake and insulin to maintain good control?

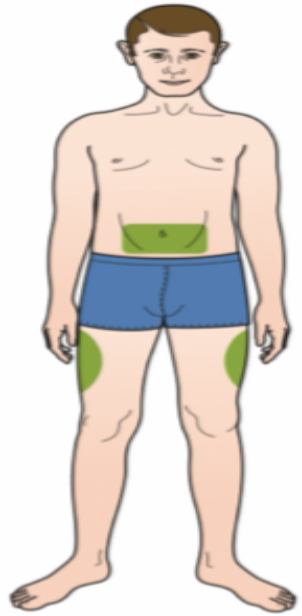
General overview (periodic):

- Normal growth and pubertal development, avoiding obesity – measure height and weight and BMI and plot on growth chart at each visit
- Blood pressure check for hypertension yearly (age-specific centiles)
- Renal disease – screening for microalbuminuria, an early sign of nephropathy, annually from 12 years
- Circulation: - check pulses and sensation
- Eyes – retinopathy or cataracts are rare in children, but should be monitored annually from 12 years, preferably with retinal photography
- Feet – maintain good care, avoid tight shoes and obtain prompt treatment of infections - annually
- Screening for coeliac and thyroid disease at diagnosis, thyroid screening annually, coeliac again if symptomatic.
- Annual reminder to have flu vaccination

Knowledge and psychosocial aspects:

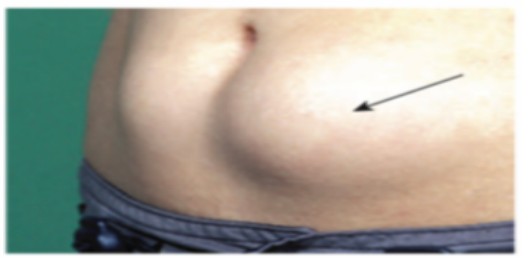
- Good understanding of diabetes, would participation/holidays with other diabetic children be beneficial? Member of Diabetes UK?
- Becoming self-reliant, but appropriate supervision at home, school, diabetic team?
- Taking exercise, sport? Diabetes not interfering with it?
- Leading as normal life as possible?
- Smoking, alcohol?
- Is 'hypo' treatment readily available? Is stepped approach known?
- What are the main issues for the patient? Are there short-term goals to allow engagement with improving control?

(a)



(b)

Injection sites – check for lipohypertrophy or lipoatrophy



(c)

Figure 26.8 **(a)** The regular assessment of the child or young person with diabetes; **(b)** injection sites; and **(c)** lipohypertrophy (arrow) of abdomen from insulin injections.

Box 26.5 Tests to perform when hypoglycaemia is present

Blood

- Confirm hypoglycaemia with laboratory blood glucose
- Growth hormone, IGF-1, cortisol, insulin, C-peptide, fatty acids, ketones (acetoacetate, 3-hydroxybutyrate), glycerol, branched-chain amino acids, acylcarnitine profile, lactate, pyruvate

First urine after hypoglycaemia

- Organic acids
- Consider saving blood and urine for toxicology, e.g. salicylate, sulphonylurea

Box 26.6 Causes of hypoglycaemia beyond the immediate neonatal period

Fasting

- *Insulin excess*
  - Excess exogenous insulin, e.g. in diabetes mellitus/insulin given surreptitiously
  - β-cell tumours/disorders – persistent hypoglycaemic hyperinsulinism of infancy, insulinoma
  - Drug-induced (sulphonylurea)
  - Autoimmune (insulin receptor antibodies)
  - Beckwith syndrome
- *Without hyperinsulinaemia*
  - Liver disease
  - Ketotic hypoglycaemia of childhood
  - Inborn errors of metabolism, e.g. glycogen storage disorders
  - Hormonal deficiency: GH↓, ACTH↓, Addison disease, congenital adrenal hyperplasia

Reactive/nonfasting

- Galactosaemia
- Leucine sensitivity
- Fructose intolerance
- Maternal diabetes
- Hormonal deficiency
- Aspirin/alcohol poisoning