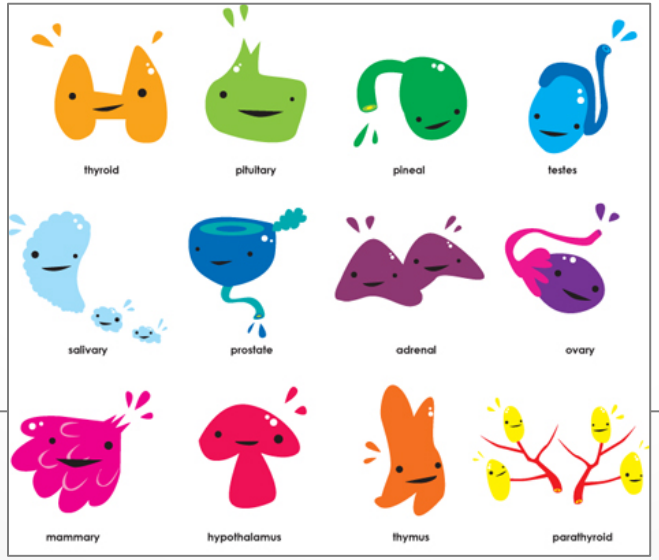
Endocrine Emergencies 101 for Nurses

with a paediatric twist

Kate Davies
Senior Lecturer in Children's Nursing
London South Bank University
London, UK

Introduction

- Non diabetic endocrine emergencies rare
- Consider clinical situations that require immediate attention and the need for specialist referral
- Mix of clinical scenarios and what we see in paediatrics



Case study 1

- 3/52 baby
  - 2nd opinion
  - ? Future management
- Born at term
- Weight 4.53kg
- Non-consanguineous parents
- No family history

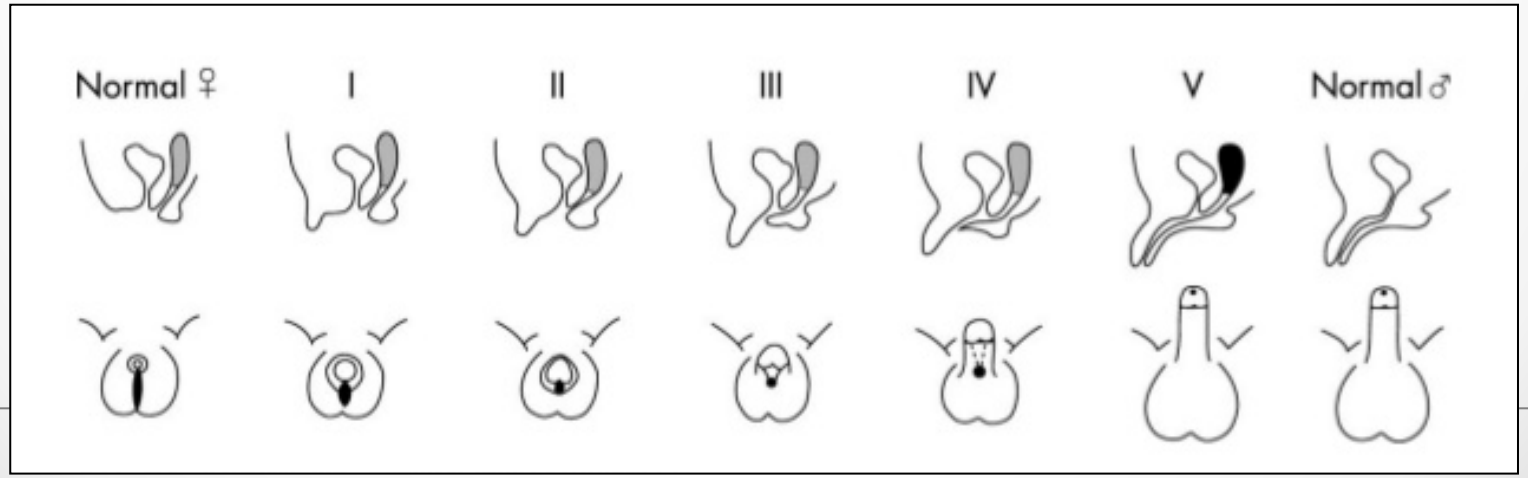
Case study 1

- Ante-natal USS
  - Boy
- Baby born
  - Male SOR assigned
  - Given a male name
- Post natal check
  - No testes in scrotum
- Urgent USS..
- DIAGNOSIS?

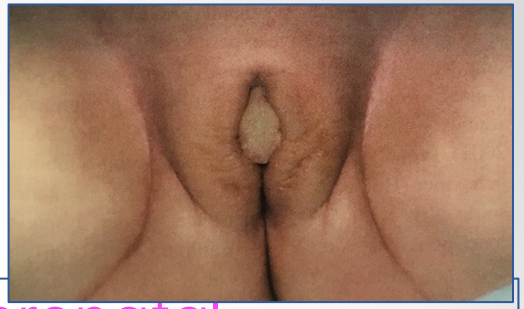
- Normal bladder and kidneys
- No testes or ovaries
- Chromosomes
  - **46XX**
- Bloods
  - ↑ 17OHP
    - 101.9nmol/L (NR: 0-5nmol/L)
- Local Paediatrician
  - 21OHD CAH
  - Hydrocortisone and Fludrocortisone

Referral to tertiary centre

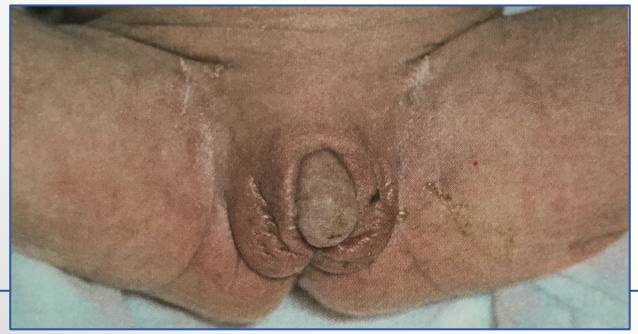
- Parents very anxious
  - Genital reconstructive surgery
- Referral into the DSD MDT
- On examination
  - Prader V in Prader scoring system



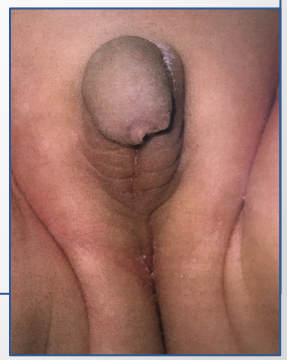
46XX CAH



- Baby will have been exposed to excess male hormone in-utero
- The genitalia will look like a boy's:
  - Labia will fuse to look like a scrotum
  - Clitoris enlarges and looks like a penis
- Can sometimes be so severe, sex assignment is difficult
  - Need karyotype
  - Will still have normal internal structures
  - Surgery may be needed to correct outer appearance
    - CONTROVERSIAL



- Exposure to prenatal androgens and Prader 3 virilisation at birth
- Same baby at age 8 weeks at the time of genital reconstruction, showing some regression of virilisation after starting steroid treatment
- Another baby girl with a more severe form of 21OHD, leading to more severe virilisation (Prader IV)



Further investigations

- Repeat Pelvic USS
  - No testes
  - Ovaries seen
- Review of management, including intensive support and input from CNS

Change from hydrocortisone suspension to tablets

Medication review

- Stay on same dose of Fludrocortisone
- Hydrocortisone suspension tds
  - 2/2/3mg
- Changed to hydrocortisone 10mg tablets
  - 1.25mg qds
  - Guidance given on crushing and mixing with water breast milk
  - Dosage titrated against BSA calculations
    - Side effects of underdosing → androgenisation
    - Side effects of overdosing → Cushings
    - Regular bloods
- Intensive emergency management training
  - x 3 emergency hydcrocortisone packs prescribed and administered

Instructions for Hospital Doctor

Dear Doctor,

If this patient is brought to hospital as an emergency the following management is advised:

1) Insert an IV cannula
2) Take blood for U&Es, glucose, and perform any other appropriate tests (e.g. urine culture)
3) Check capillary blood glucose level
4) Give 100 mg hydrocortisone intravenously as bolus (unnecessary if patient has already been given IM hydrocortisone)
5) Commence IV infusion of 0.45% sodium chloride and 5% glucose at maintenance rate (extra if patient is dehydrated). Add potassium depending on electrolyte
6) Commence hydrocortisone infusion (50 mg hydrocortisone in 50ml 0.9% sodium chloride via syringe pump)
7) Monitor for at least twelve hours before discharge

IMPORTANT! If blood glucose is < 2.5 mmol/l, give bolus of 2 ml/kg of 10% glucose

If patient is drowsy, hypotensive and peripherally shut down with poor capillary return give 20ml/kg of 0.9% sodium chloride stat.

If in any doubt about this patient's management, please contact the urgent advice numbers

Useful Contact Numbers:

GOSH Switchboard
Tel: 020 7405 9200

For Urgent Advice:
Tel: 020 7405 9200 and ask to be put through to the endocrine registrar on call

University College Hospital Switchboard
Tel: 0845 155 5000

For Urgent Advice:
Tel: 0845 155 5000 and ask to be put through to the endocrine registrar on call.

Great Ormond Street Hospital for Children NHS Trust and University College London Hospitals NHS Foundation Trust

CORTISOL DEFICENCY

THE OWNER OF THIS CARD IS ON CORTISOL REPLACEMENT THERAPY

Name
Address
Affix photo here
Tel
Mobile:
Date of Birth ___/___/___
Hospital No
Consultant
Hospital
Address
Tel Fax
General Practitioner
Address
Tel Fax

Great Ormond Street Hospital for Children NHS Trust and University College London Hospitals NHS Foundation Trust

3. Draw up 2mls of cooled, boiled water into a 2ml syringe
4. Mix the crushed ¼ of a tablet with the 2mls of cooled boiled water
5. Then draw up 1ml of the mixture to give 1.25mg
6. Give by mouth as shown by ward nurses

Great Ormond Street Hospital for Children NHS Trust

Great Ormond Street Hospital for Children NHS Trust

Great Ormond Street
London WC1N 3JH

Tel: 020 7405 9200

Gastroenterology, Endocrinology, Metabolic & Adolescent Medicine (GEMA)
Direct Line: 0207-813-8214

cortisone (oral)
cortisone (oral)
supplements 5mmol/ml 30% solution:
ospital Doctor

nts cortisol deficiency, if this patient is brought to hospital as an emergency, gement is advised:

wsy and unresponsive give IM hydrocortisone in the following doses (0-1yr – 25mgs; 1-5 yrs – 50mgs; > 5yrs – 100mgs) if patient has not already ocortisone administered by ambulance crew or parents.

for U&Es, glucose and osmolality

ose is < 2.5 mmol, give bolus of 2mg/kg 10% dextrose

- If patient is drowsy, hypotensive and peripherally shut down, give 20ml/kg of nor insert an IV cannula and then continue with usual dextrose saline infusion
- Continue with bolus IV hydrocortisone at 2mg/kg every 4 hours until patient is to oral fluids and then swap to double usual oral Hydrocortisone doses until patient recovered and back to normal self (usually 2-3 days on double usual hydrocortis doses).
- Important: Please admit for a minimum of 12 hours

If there is ar
Street Hosp
a Great
on Call)

for Children NHS Foundation Trust: Information for Families

renal hyperplasia (CAH)

reat Ormond Street Hospital
dical condition congenital
what to expect when your
sment and treatment.

Congenital adrenal hyperplasia is

Great Ormond Street Hospital for Children NHS Trust

Great Ormond Street
London WC1N 3JH

Tel: 020 7405 9200

Gastroenterology, Endocrinology, Metabolic & Adolescent Medicine (GEMA)
Direct Line: 0207-813-8214

Date:
Reference:

Dr
Paediatric Consultant

Dear Dr

RE:

……is a …. year old ……under the care of …… at Great Ormond Street Hospital. He is a boy/girl with ……, he/she was referred with ……and we have since found he also has cortisol deficiency.

He/She has been commenced on Hydrocortisone at a dose of 2.5mg mane, 2.5mg at lunchtime, and 2.5mg nocte. ……'s mum has had education in his/her management during times of illness and has been trained in giving IM hydrocortisone should the need arise.

I would be extremely grateful if you could arrange for …… to have fast track access at the …… should he/she require emergency IM hydrocortisone. Please let us know on the number below.

Please do not hesitate to contact me should you require more information on 0207 813 8214.

Many thanks,

Yours sincerely

……
Clinical Nurse Specialist

Great Ormond Street Hospital for Childre

Cortisol deficiency steroid replacement the

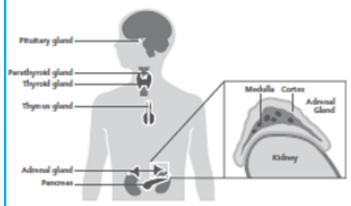
This leaflet explains about cortisol deficiency and how it is treated. It also contains information about how to deal with illnesses, accidents and other stressful events in children on cortisol replacement.

Where are the adrenal glands and what do they do?

The adrenal glands rest on the tops of the kidneys. They are part of the endocrine system, which organises the release of hormones within the body. Hormones are chemical messengers that switch on and off processes within the body.

The adrenal glands consist of two parts:

- the medulla (inner section) which makes the hormone 'adrenaline' which is part of the 'fight or flight' response a person has when stressed.
- the cortex (outer section) which releases several hormones.



●●●○○ O2-UK 22:02 100%

Great Ormond Street Hospital for Children NHS Foundation Trust

My Cortisol

London Ambulance Service NHS Trust

Patient Specific Protocol
PSP Paediatric Steroid Dependent Crisis

PSP

This protocol has been specifically prepared for STEROID DEPENDENT CRISIS patients and details the treatment to be given in specified circumstances.

Patient's Name: **Date of Birth:**

NHS Number:

Address:

School:-

Local hospital:

Reason for protocol: Administration of IM hydrocortisone in possible adrenal crisis

Specific Treatment / Instructions: Patient may have an adrenal crisis if IM hydrocortisone not administered in an emergency situation

In the event that this child is involved in an accident or develops diarrhoea or vomiting and presents with any symptoms of a steroid dependent crisis whilst at **Home or at School** they are to be administered IM hydrocortisone as detailed over leaf.

Note:- The IM hydrocortisone (Efcortesol) is kept both by the parents and by the school in an emergency pack.

Please transport this child to the above local hospital if possible, otherwise to the nearest paediatric A&E unit.

All other aspects of clinical care remain unchanged.

For further advice if necessary please contact the Endocrine Registrar on call via switchboard at Great Ormond Street Hospital on 020 7405 9200

1. Efcortesol 1ml ampoule (Hydrocortisone 100mg/ml - as sodium phosphate)

Dose: Age 0-1 years 25 mg IM
Age 1-5 years 50mg IM
Age 5+ years 100mg IM

2. Please also administer Glucogel (Hypostop) 25 gram tube, required dose in an emergency - up to 1/3 tube if not already previously administered by carers.

Following administration of the hydrocortisone remove to hospital with full monitoring and oxygen therapy as required.

All other aspects of clinical care remain unchanged.

If required contact EOC and ask for the Clinical Support Desk

PTO for further general info on Steroid Dependent Crisis

How to give an emergency injection of Efcortesol®

Information for families

Great Ormond Street Hospital for Children NHS Trust
University College London Hospitals NHS Trust

What is a DSD?

- Congenital conditions in which development of chromosomal, gonadal or anatomic sex is atypical
- True genital ambiguity
  - 1 in 5000 / 1 in 4500 births
- Genital anomalies
  - 1 in 300 births

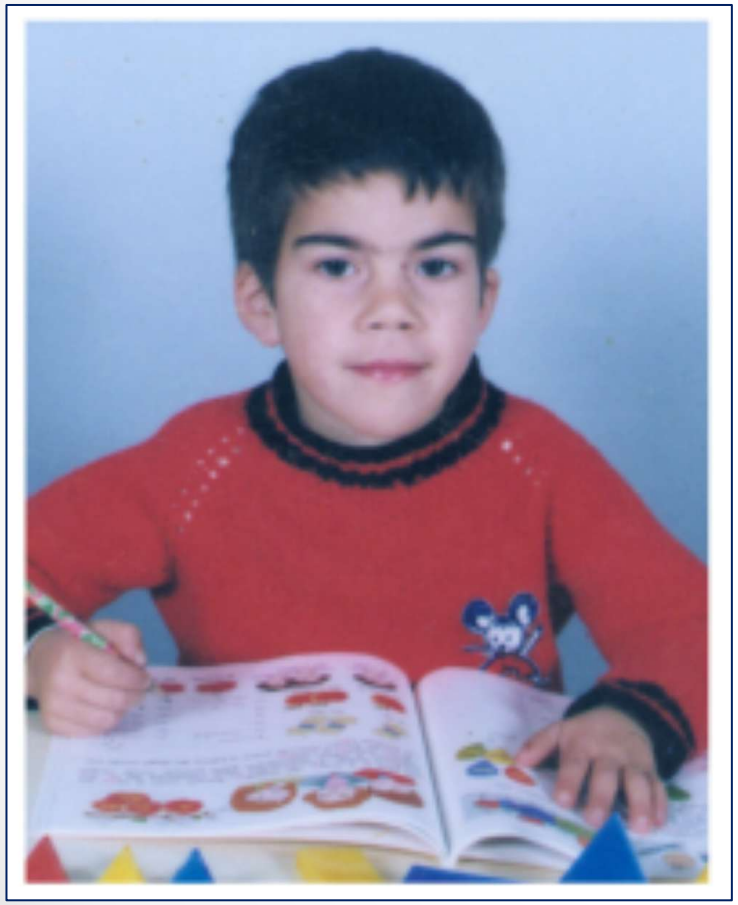
Classification of DSD

- **46,XY DSD (under virilised genetic male)**
  - Disorders of testicular development
    - Ovotesticular DSD
  - Disorders of androgen synthesis / action
    - CAIS
  - Others
    - Hypospadias
- **46,XX DSD (over virilised genetic female)**
  - Disorders of ovarian development
    - Ovotesticular DSD
  - Androgen excess
    - CAH

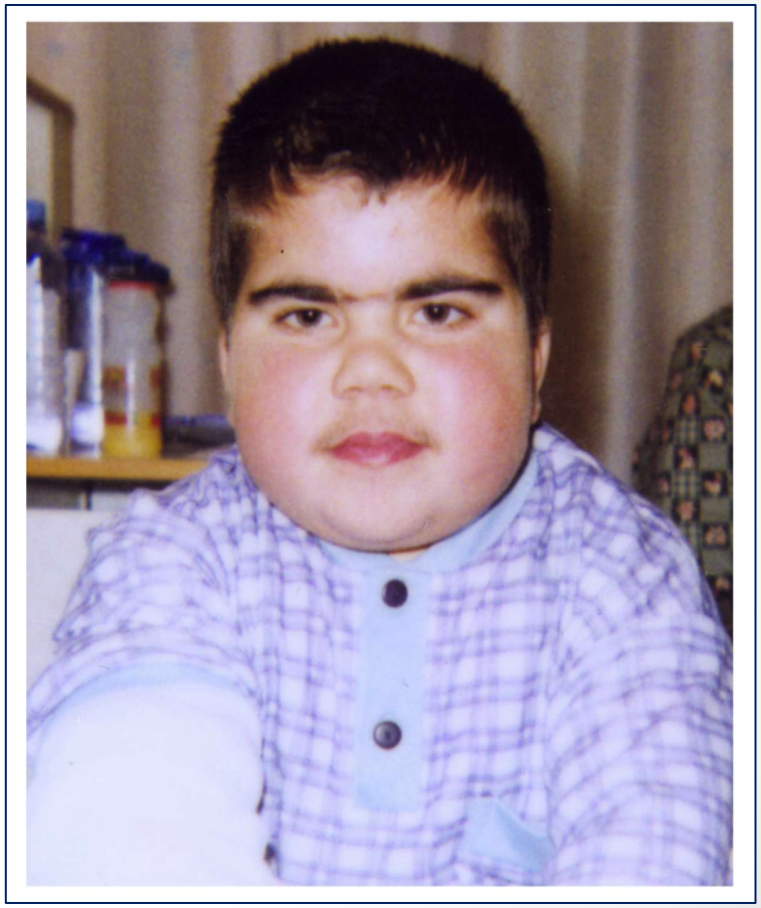
- **Sex chromosome DSD (variable)**
  - Turner syndrome
  - Klinefelter syndrome
  - Mixed gonadal dysgenesis

Case study 2

January 2001



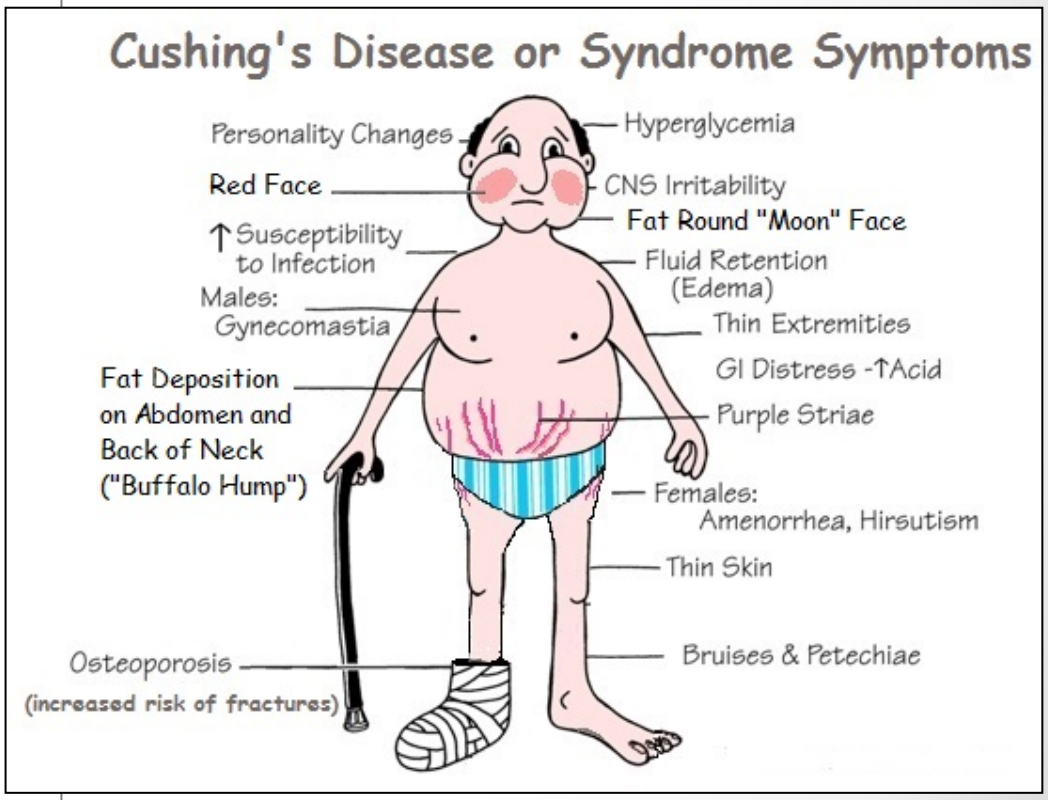
February 2002



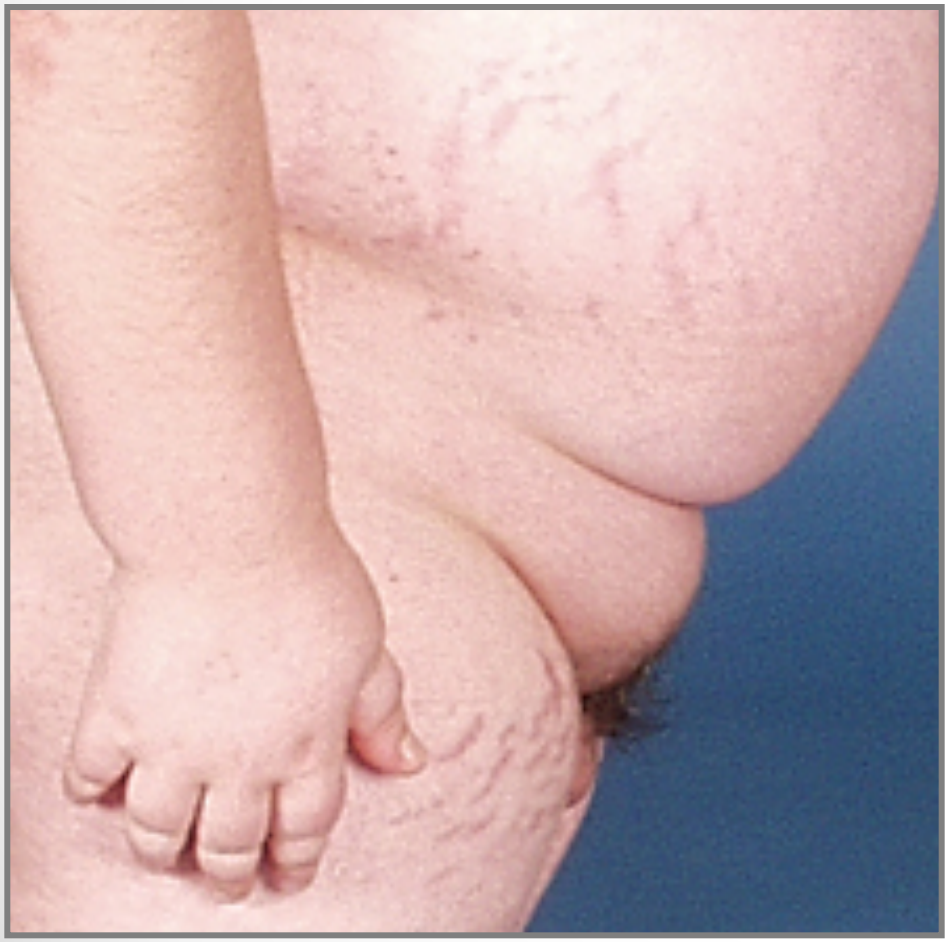
Case study 2

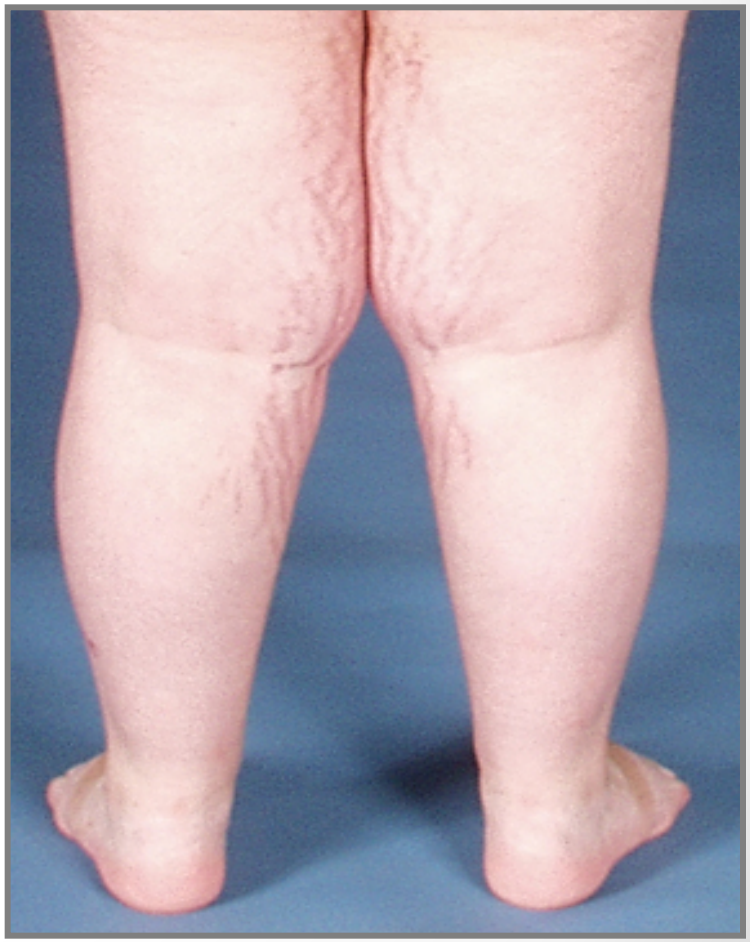
- Facial appearance
  - 'Moon face'
- Weight gain
  - Truncal obesity
  - Buffalo hump
- Skin
  - Thin and fragile
  - Stretch marks
- Muscle weakness
- Mood disturbance
- Menstrual disturbance
- Hypertension
- Related excess androgen production

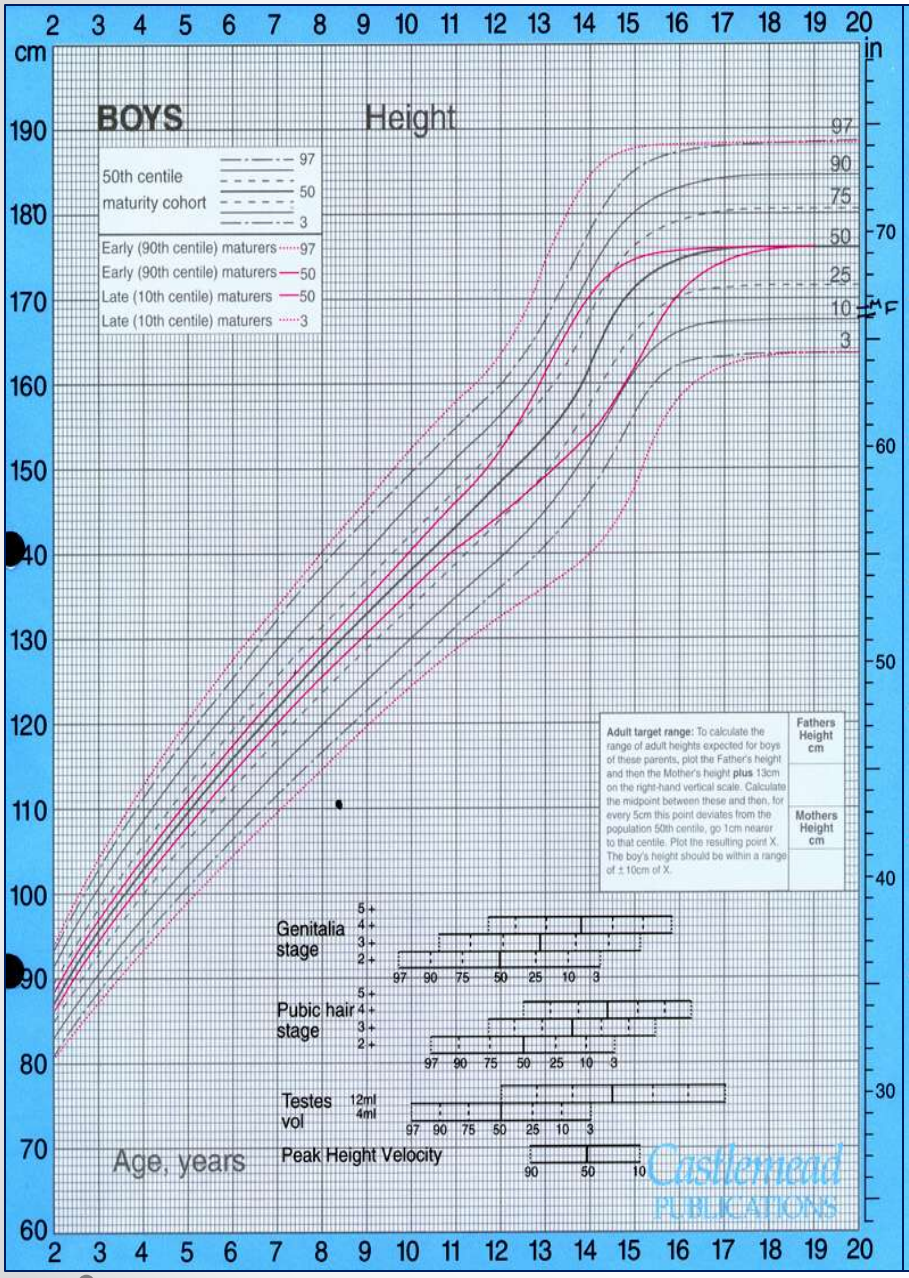
- **Growth failure in children**
- **Abnormal virilisation**

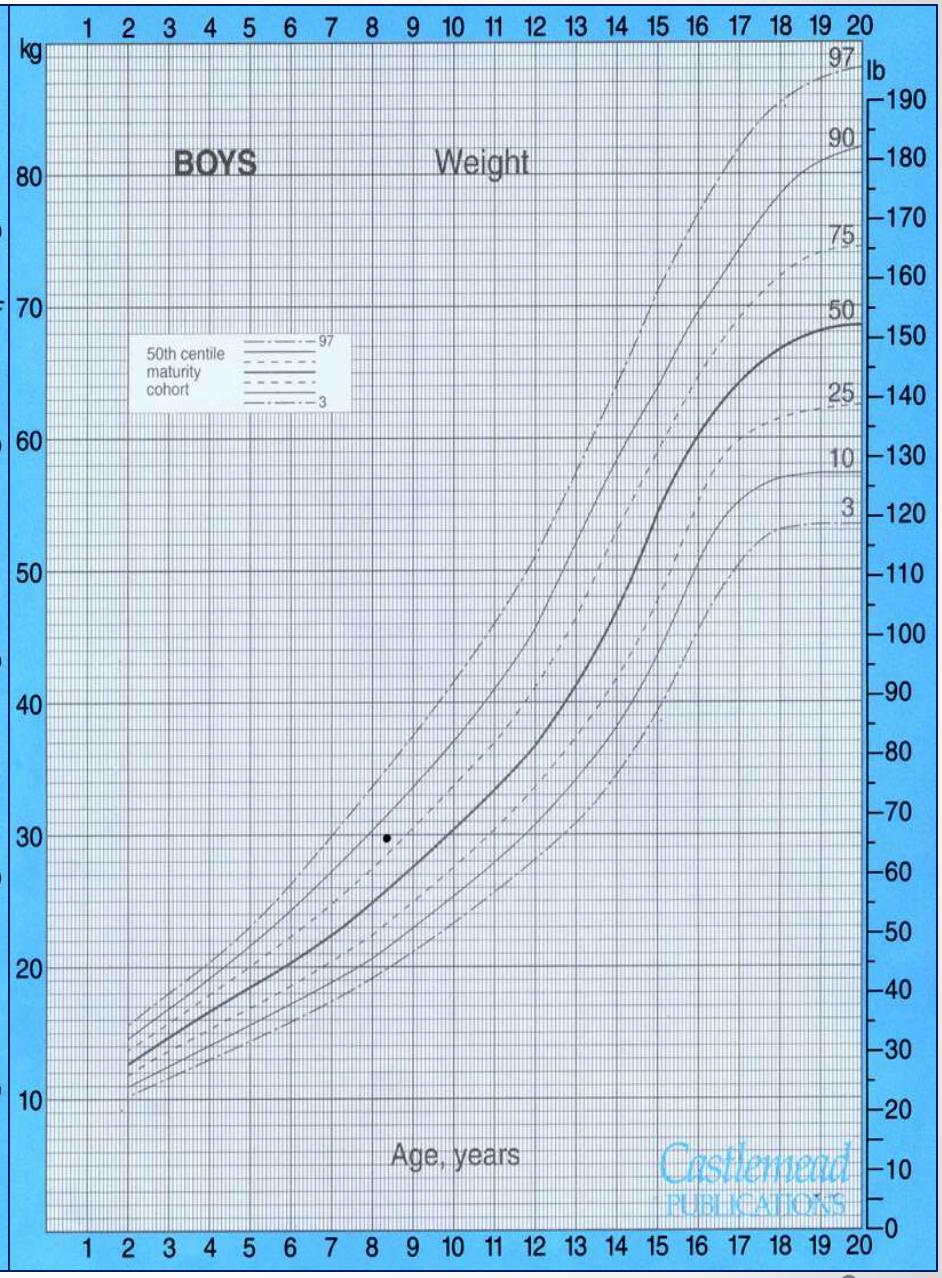


Abnormal virilisation in a 6.2 yr old prepubertal boy with Cushing's disease






BOYS
Height
cm
in
50th centile
maturity cohort
97
50
3
Early (90th centile) maturers 97
Early (90th centile) maturers 50
Late (10th centile) maturers 50
Late (10th centile) maturers 3
97
90
75
50
25
10
3
Adult target range: To calculate the range of adult heights expected for boys of these parents, plot the Father's height and then the Mother's height plus 13cm on the right-hand vertical scale. Calculate the midpoint between these and then, for every 5cm this point deviates from the population 50th centile, go 1cm nearer to that centile. Plot the resulting point X. The boy's height should be within a range of ± 10cm of X.
Fathers Height cm
Mothers Height cm
Genitalia stage
Pubic hair stage
Testes vol
12ml
4ml
Peak Height Velocity
Age, years
Castlemead PUBLICATIONS

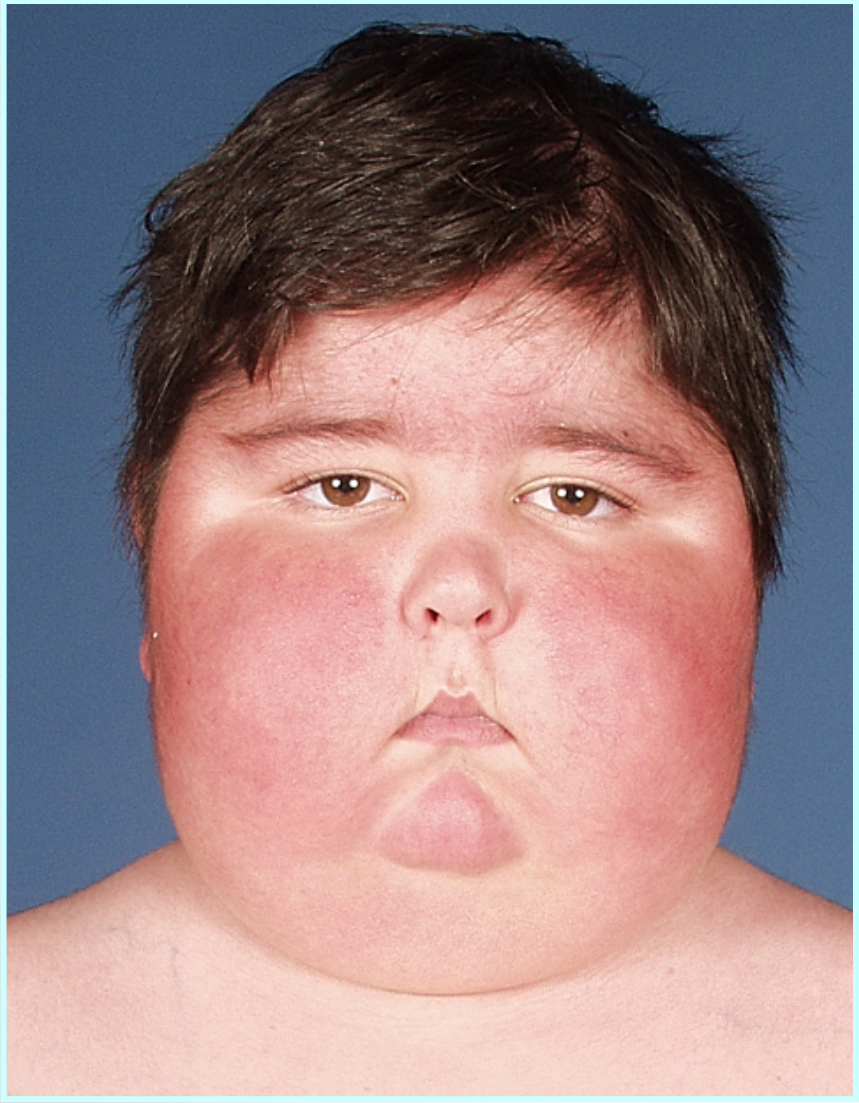

BOYS
Weight
kg
lb
50th centile
maturity
cohort
97
3
97
90
75
50
25
10
3
Age, years
Castlemead PUBLICATIONS

Rapid progression of severe paediatric Cushing's disease in an 6.2 yr old boy

Age 4 yrs

Age 5 yrs

Age 6 years



- 2 year history of rapid weight gain
- Purple striae
- Depressive state
- Growth failure
- Hypertension 135/55
- Virilised
  - G3 P2 Prepubertal testes
- Raised liver enzymes
- Abdominal U/S
  - Enlarged fat laden liver
  - Enlarged adrenals

Endocrine investigations?

- Urinary free cortisol
  - **1098nmol/24h (NR 40 – 340)**
- Midnight cortisol
  - **>1650nmol/l (NR <50)**
- Low dose dexamethasone suppression test
  - **Failure to suppress cortisol (>1650nmol/l)**
- Adrenal androgens
  - **Raised:**
    - **Androstenedione 36.3 nmol/l (NR <1.0)**
    - **DHEA-S 1.4 μmol/l (NR <0.5)**
    - **Testosterone 8.1 nmol/l (NR <0.8)**

Next steps?

Pituitary surgery?

Adrenalectomy?

Pituitary surgery plan

- Control the hypercortisolaemia
- Stabilise the child's general condition
- Treatment was initiated with **ketoconazole** 200 mg 8-hourly, but discontinued after 6 weeks due to deteriorating liver function
- **Metyrapone** 250 mg 8-hourly was substituted but was not tolerated even when given by naso-gastric tube, because of persistent nausea and vomiting

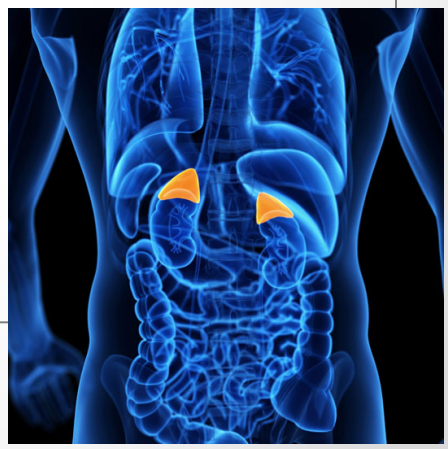
The serum cortisol remained elevated at > 1,250 nmol/l

What happened next?

- The child's general condition deteriorated rapidly with hyponatraemia secondary to persistent vomiting, inadequate nutritional support and incipient respiratory failure

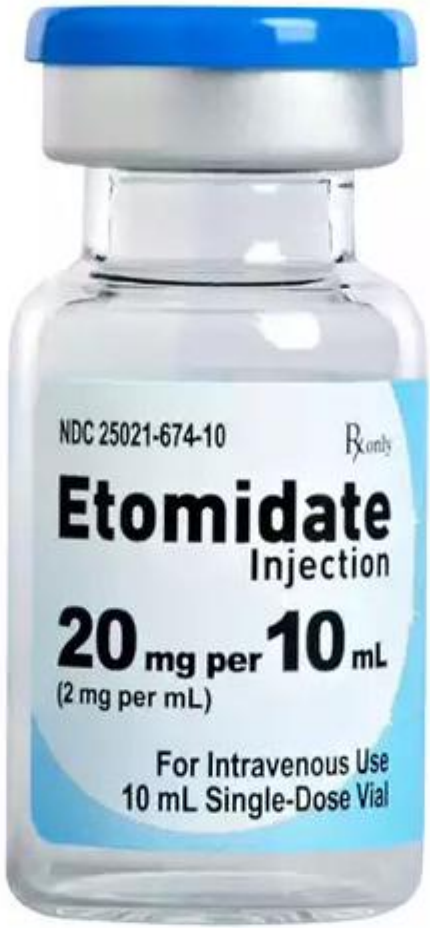
- The decision was taken to transfer the child to a PICU and to stabilise his condition in preparation for bilateral adrenalectomy

  - **Life saving procedure!**



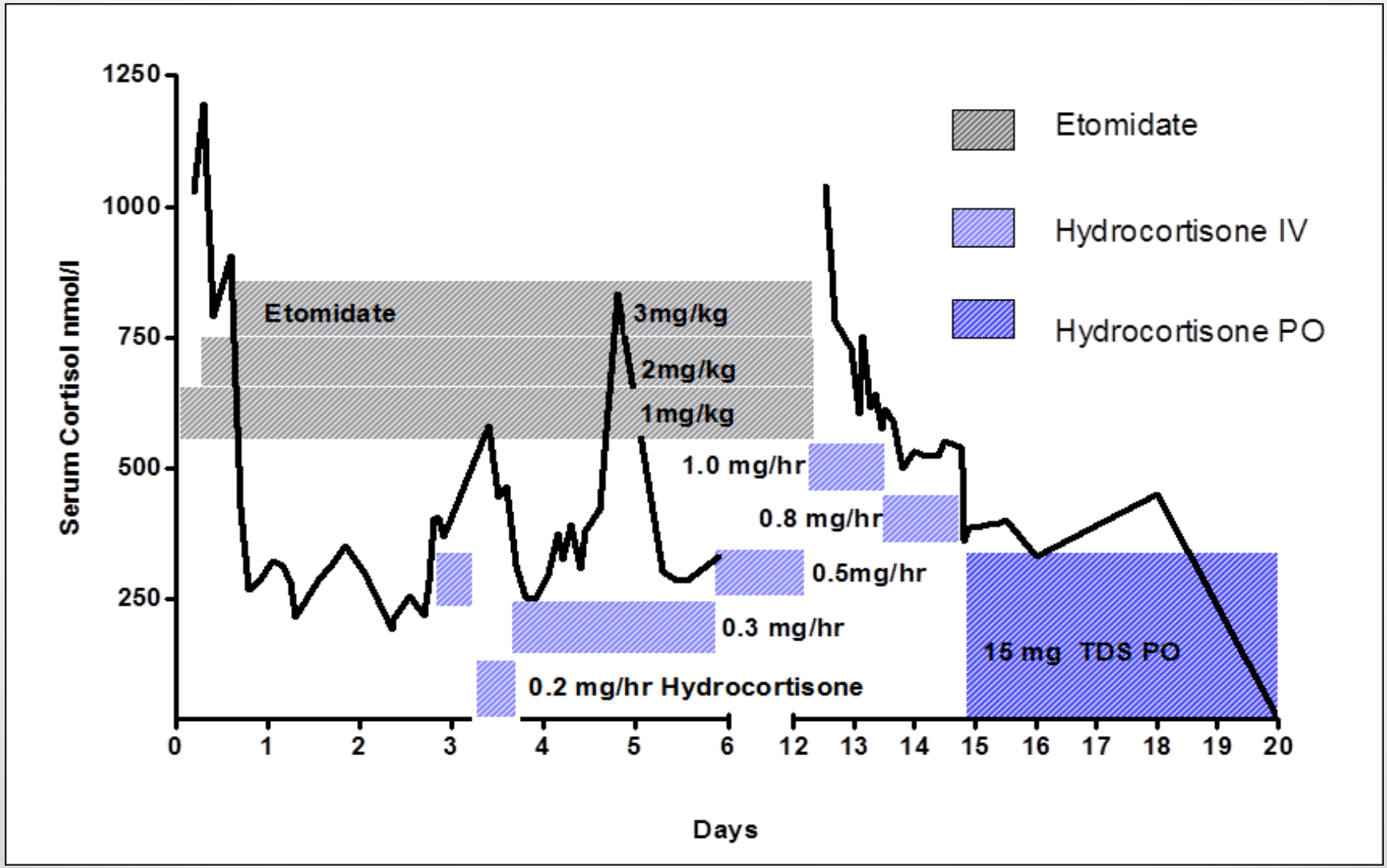
How could his hypercortisolaemia be controlled prior to surgery?

Severe Cushings - Etomidate



- Etomidate
  - IV anaesthetic agent
  - Suppresses corticosteroid synthesis
    - Adrenal suppression

Control of hypercortisolaemia with adrenolytic therapy - IV Etomidate



Finally.. Progress!

- Post-operatively, the patient made good progress
- Serum cortisol pre-hydrocortisone was <20 nmol/l, indicating removal of all adrenal tissue, and he recovered steadily on replacement therapy of hydrocortisone 5 mg three times daily and fludrocortisone 50g twice daily

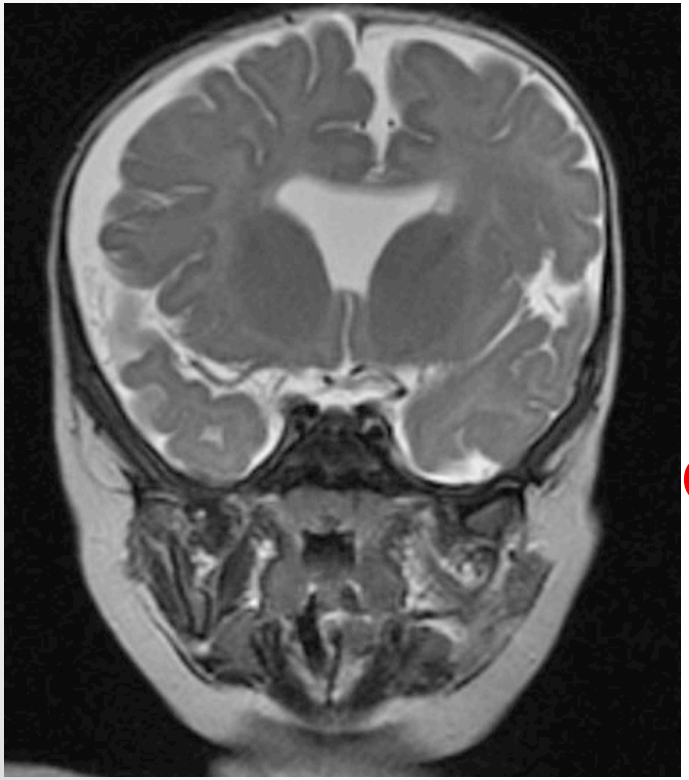
Case study 3 – 13 week old baby girl

- Admitted for poor feeding
  - Born at 41 weeks
  - Weighed 3.4kg
  - Mother was 29 years of age
  - Hypoglycaemic at 24 hours
  - Phototherapy for jaundice
  - 6 weeks
    - Not fixing and following
  - 10 weeks
    - Small optic discs
    - Absent electroretinogram response to light

- On examination
  - Pale
  - Still
  - Weight 5.36 kg (-1.05 SD)
  - Length 60.5cm (0.21 SD)
  - Hypotonic
  - Roving nystagmus

- **Likely diagnosis?**
- **What further assessment is needed?**

Septo Optic Dysplasia

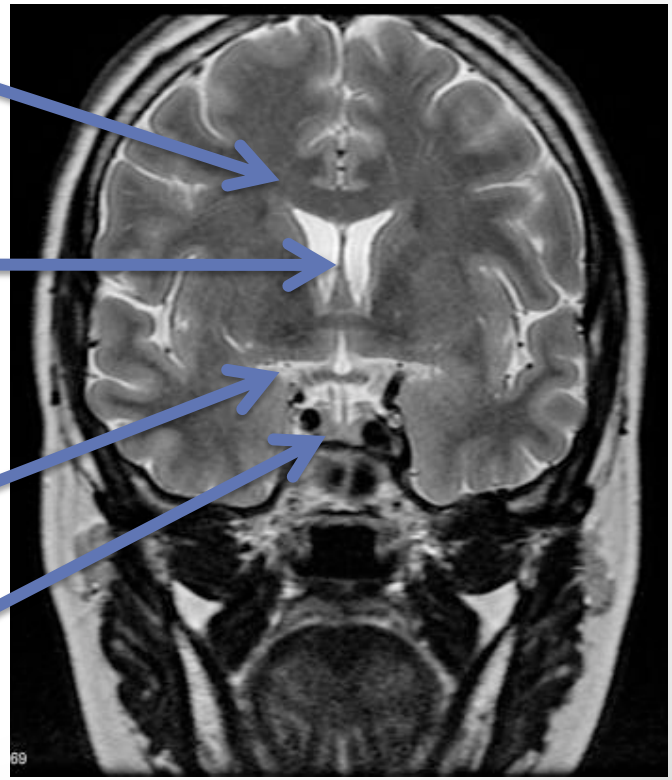


Corpus callosum

Septum pellucidum

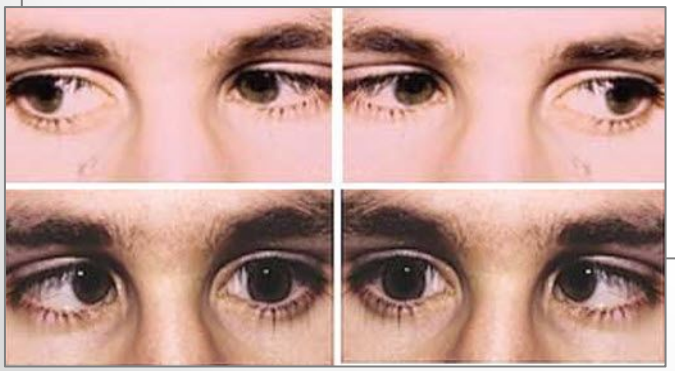
Optic nerves

Pituitary

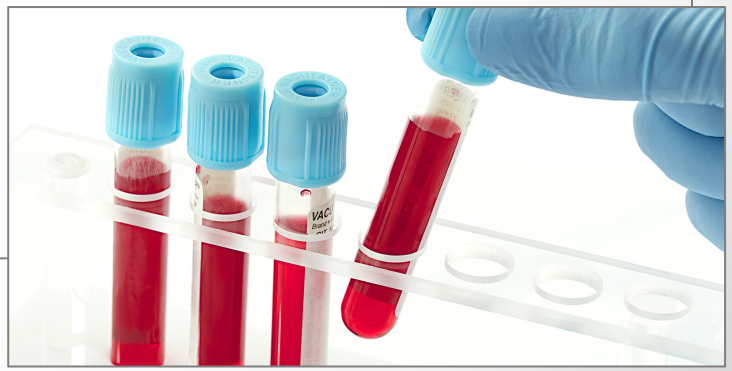


Why?

- Nystagmus
- Failure to fix and follow
- Neonatal hypoglycaemia
- Jaundice
  - Optic Nerve Hypoplasia
  - Hypopituitarism



- Cranial ultrasound
- MRI
  - Absence of septum pellucidum
- Bloods
  - T4
  - TSH
  - Random cortisol
  - Electrolytes



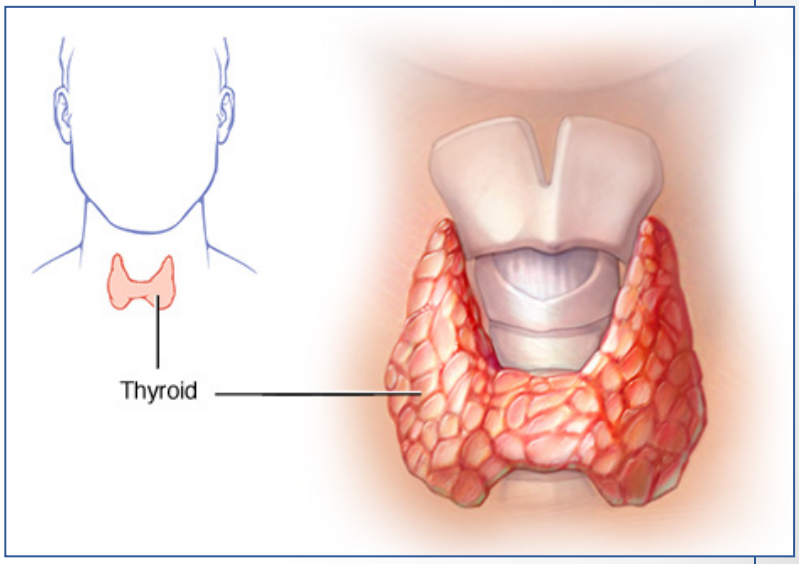
What would the results show?

- Serum sodium
  - 163mmol/L (NR 135 - 145
- Urine osmolality
  - 132mOsm/kg
    - NR 500 – 800 mOsm/kg water
      - Large values = concentrated urine
      - Lower values = dilute urine
- Random cortisol
  - 220nmol/L (NR >500)
- Peak cortisol on standard synacthen test (ACTH 250mcg)
  - 1269nmol/L

- Free T4
  - 13.9pmol/L (NR 10-20)
- TSH on stimulation test
  - 0 mins 3.8
  - 30 mins 26
  - 60 mins 33 mU/L (NR 0.5 – 5)
- Peak GH on stimulation
  - Low normal 8µg/L (NR >7)

TRH test

- TRH 7 mcg/kg slow IV injection over 3 mins
- Interpretation
  - In normal people, a rise in TSH at 20 mins with a fall at 60 is seen
  - In hypothalamic hypothyroidism
    - TSH increases at 20m, and continues to rise at 60
  - In hypothyroidism 2° to hypopituitarism
    - No change in TSH level



What do the results mean?

What treatment should the baby have?

Further diagnosis and treatment

- Baby has diabetes insipidus
- Hypothalamic hypothyroidism
- No cortisol deficiency

WHY?

- DDAVP
  - 25mcg daily initially
- Thyroxine
  - FT4 falls to 8.9pmol/L at age 5 months

What happens next with the baby?

Baby unwell again

- Admitted acutely unwell to the ER
  - Poor perfusion
  - Mottled and cold peripheries
  - Capillary glucose 1.3mmol/L (NR 4-7)
  - Sodium
    - 167mmol/L
  - Lab glucose
    - 2.2mol/L
  - Treatment?
    - IV fluids
    - DDAVP dose adjusted
    - DI difficult to control
      - Sodium fluctuating between130 and 156 mmol/L

What is the explanation for the poor progress?

ACTH deficient

- DI cannot be properly controlled with cortisol deficiency
  - Latter required for water excretion
- Peak cortisol on synacthen was exaggerated as low dose was not used
- IM injection after time 0
  - **< 6/12 : 62.5mcg**
  - 6/12 – 2yrs : 125mcg
  - > 2yrs: 250 mcg

- Random cortisols during illness were never >500
- Baby started on hydrocortisone
  - When on full replacement
    - DI stabilised
- Height
  - Remained on 10th centile
  - Age 5 years
    - Fell to 3rd centile
    - GH therapy commenced

Comments on Case Study 3

- Difficulty in diagnosing adrenal insufficiency
- Important role of cortisol in water balance
- Evolving pattern of GHD in SOD
  - Normal levels often found in infancy

Case study 4

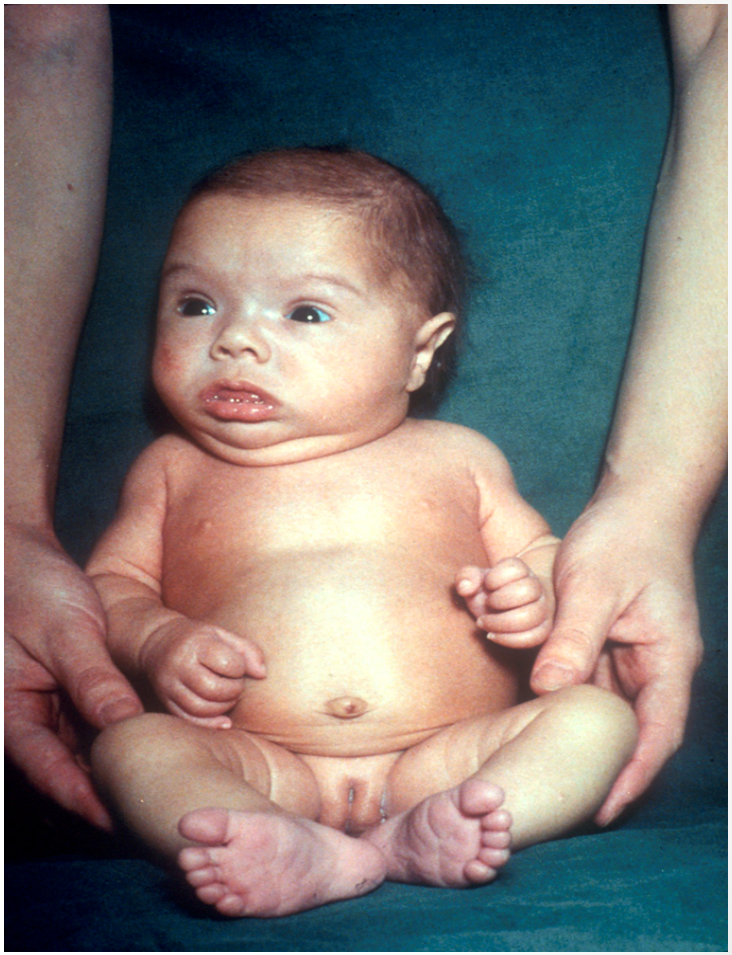
- Baby aged 10 days referred to the paediatric endocrine clinic
- Fit and well
  - Beginning to feed poorly
  - Slightly jaundiced

What could be the indication for referral?

Congenital hypothyroidism

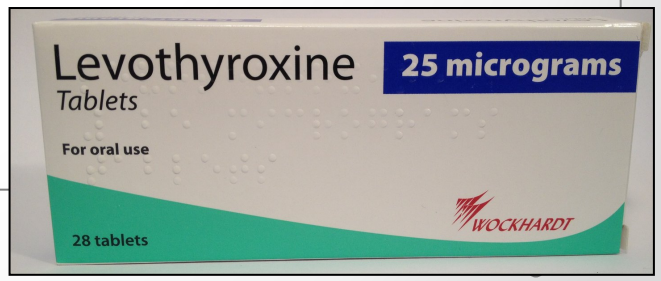
Congenital hypothyroidism

- All babies screened at 5 days of life
  - Guthrie Test - TSH
    - Sleepiness
    - Poor feeding
    - Constipation
    - Goitre
    - Oedema
    - Jaundice
- Treatment:
  - Thyroxine tablets
    - 100 mcg / m2 / day

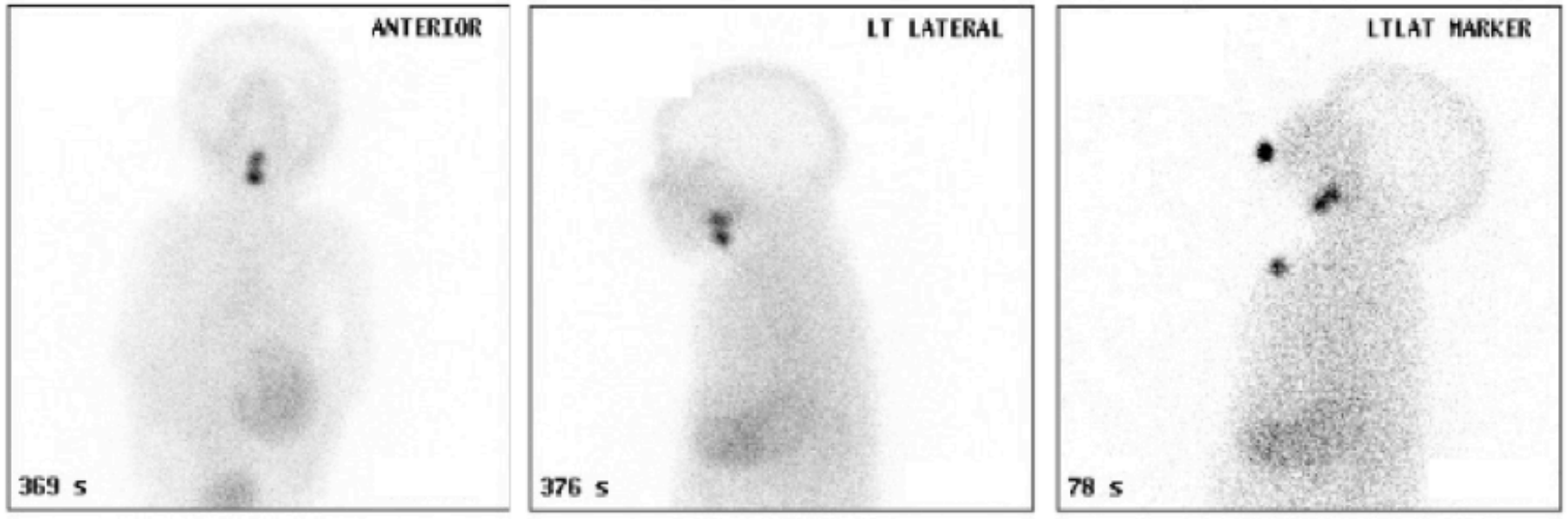


Screening

- Guthrie card at 5-8 days – detects TSH
- Notification if positive TSH >20mU/L
- Start treatment within 24 hours
- If borderline TSH 6-19.9mU/L - repeat
- Thyroid isotope scan usually performed
  - Assess size and site of gland



Technetium scan



Flow sheet false positive

Blood spot 26mU/l

Date	TSH	Free T4	Free T3	Thyroxine dose (mcg)	Comments
13/8/2015	7.43 (<6)	20.7 (12.5-24.6)		-	Rpt 2/52
27/8/2015	5.2	19.2		-	Discharge

Flow sheet double borderline

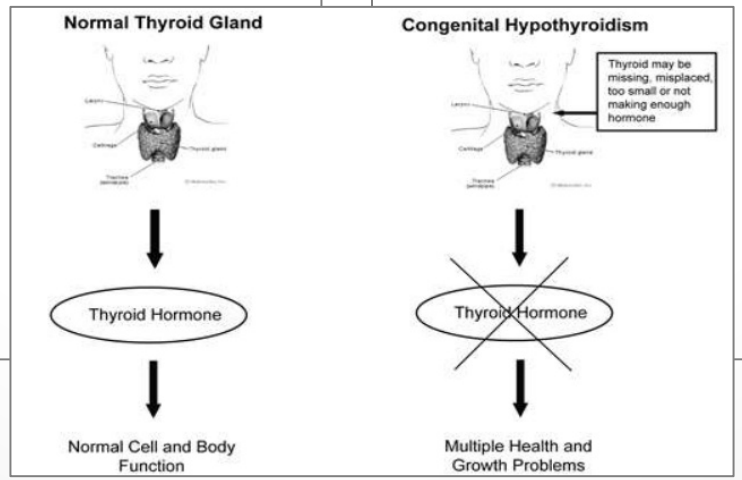
1st blood spot 9.8mU/l; 2nd spot 10mU/l

Date	TSH	Free T4	Thyroxine dose (mcg)	Comments
30/5/2015	26.2 (<6)	15.3 (12.5-24.6)	-	Started 25mcg OD
12/6/2015	6.1	18.7 (9-19.6)	25 mcg OD	Continue
31/7/2015	19.2	14.0	25 mcg OD	Increase to 35mcg OD
1/10/2015	0.6	23.6	35mcg OD	Reduced to 30mcg OD
2/2/2016	3.4	15.3	30mcg OD	Continue
1/4/2016	1.2	19.2	30mcg OD	Continue
2/6/2016	2.2	16.7	30mcg OD	Switched to tablets 37.5/25 mcg OD

Congenital hypothyroidism

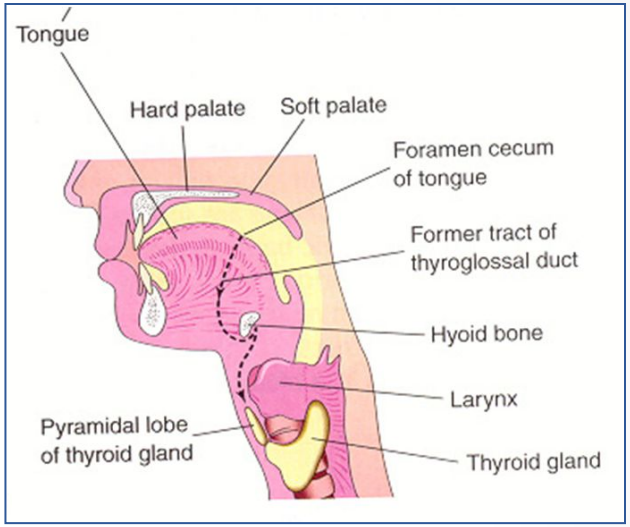
- Defects in T4 synthesis
- TRH and TSH deficiency
- TSH receptor defect
- Maternal disease
  - Drugs
- Trisomy 21

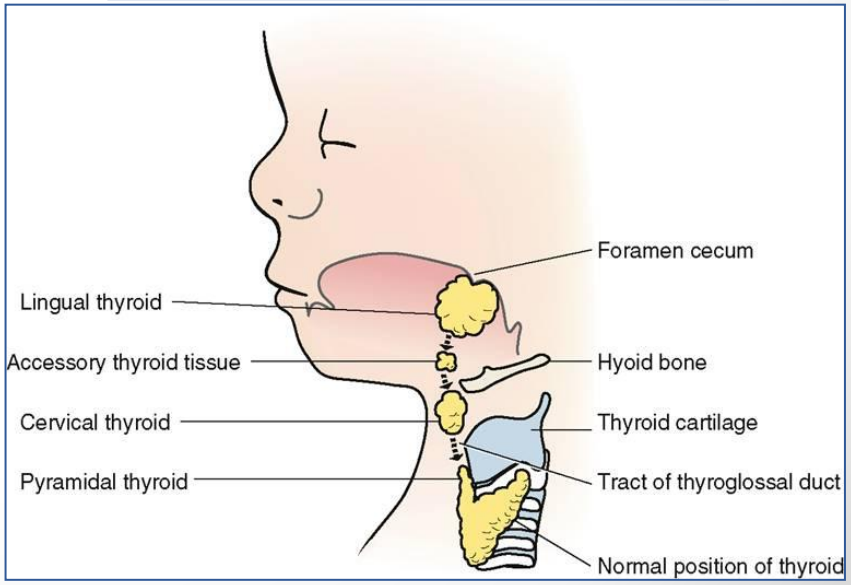
- Thyroid dysgenesis
  - Missing
  - Ectopic
  - Underdeveloped



Thyroid gland development – 4- 7 weeks

- Thyroglossal duct degenerates between 7-10 weeks
- Thyroid reaches its end location anterior to the trachea by week 7
- If parts of the duct remain the person may also have a pyramidal lobe
  - 50% of the population
- Ectopic thyroid tissue left behind during migration
  - Common but asymptomatic
  - Parts of the duct may persist
    - Midline, movable cyst in the child





Treatment and follow up

Treatment

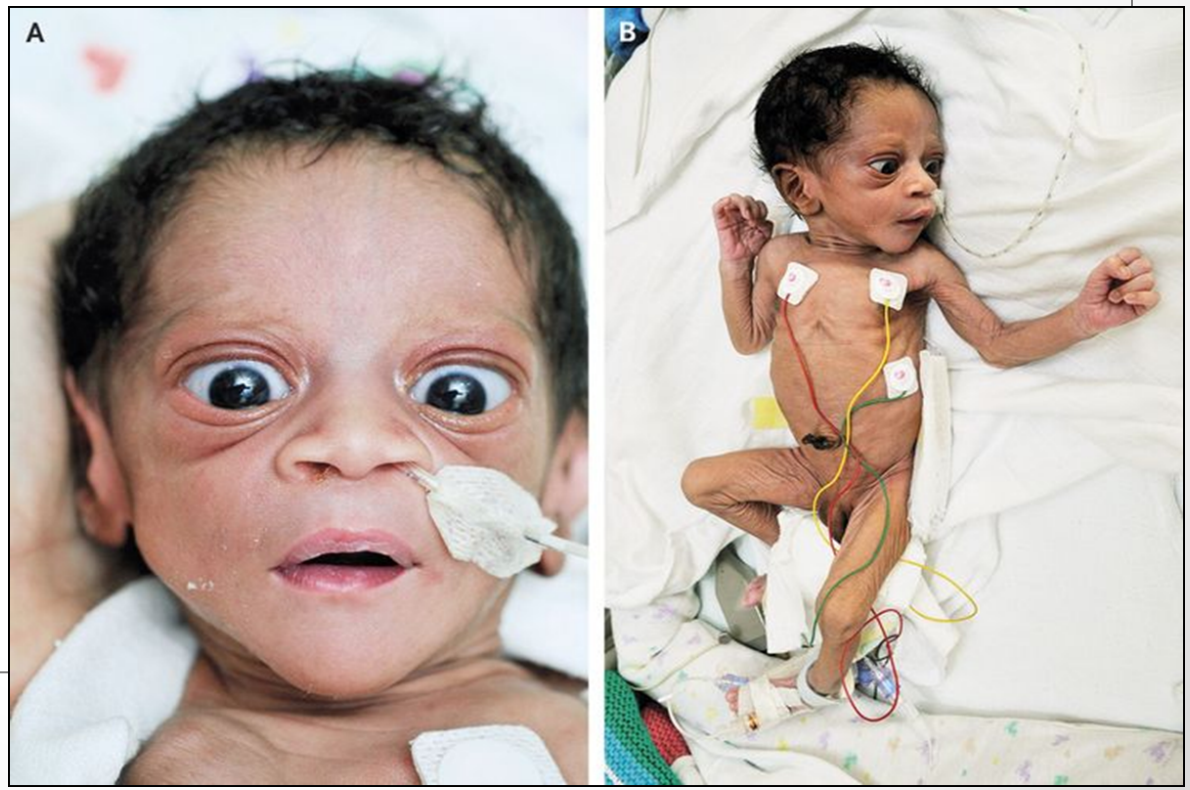
- Thyroxine
  - 25-50 mcg daily (10mcg/kg/d)
  - Crushed tablet or solution
  - NOT suspension
    - Limited stability
- Neonates have higher normal range fT4
- Maintain in upper half of normal range

Follow up

- Development, hearing and growth
- Often subtle speech delay
- Detect those with transient neonatal hypothyroidism
- Decision on withdrawal at 3 years

Case study 5

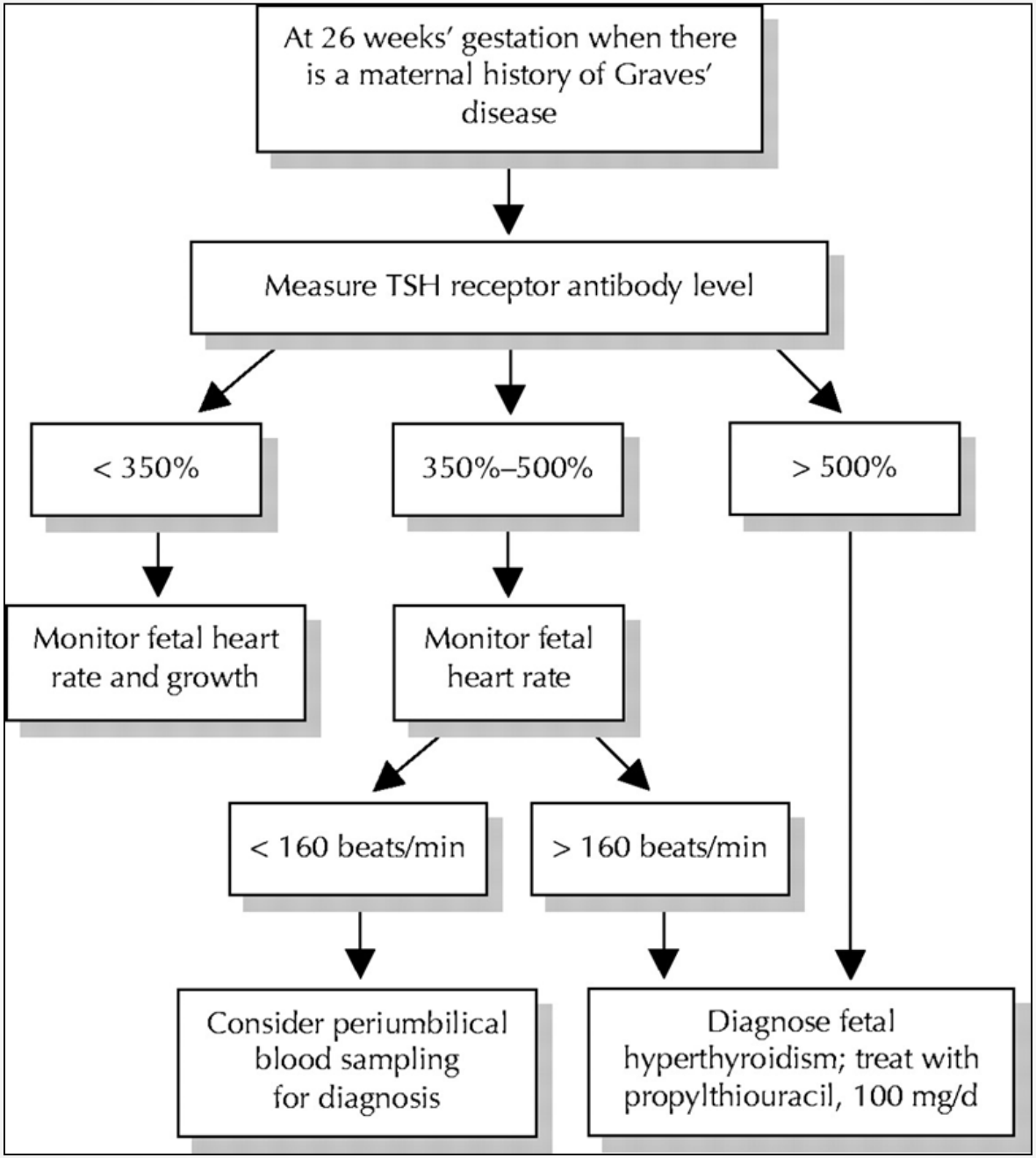
- Presentation of newborn baby
  - Tachycardic
  - Irritability
  - Restlessness
  - Hypertensive
  - Exophthalmos
  - Periorbital oedema
  - Goitre



Neonatal thyrotoxicosis

- Caused by trans-placental transfer of maternal TSH receptor antibodies
  - Stimulates the foetal and neonatal thyroid gland
- May occur in infants with mothers of some degree of hyperthyroidism

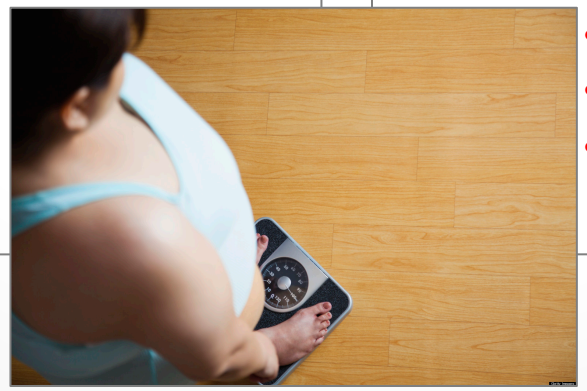
- Usually simply biochemical
- Signs and symptoms
  - Goitre
  - Tachycardia
  - Arrthymias
  - Hypertension
  - Cardiac failure
  - ↑ appetite
  - Weight loss
  - Diarrhoea
  - Irritability
  - Exophthalmus


At 26 weeks' gestation when there is a maternal history of Graves' disease
Measure TSH receptor antibody level
< 350%
350%–500%
> 500%
Monitor fetal heart rate and growth
Monitor fetal heart rate
< 160 beats/min
> 160 beats/min
Consider periumbilical blood sampling for diagnosis
Diagnose fetal hyperthyroidism; treat with propylthiouracil, 100 mg/d

Case study 7

- 14 year old Asian girl
  - 6 week history
    - Polyuria
    - Polydipsia
    - Weight loss
- Grandfather developed diabetes in his 50s and takes tablets

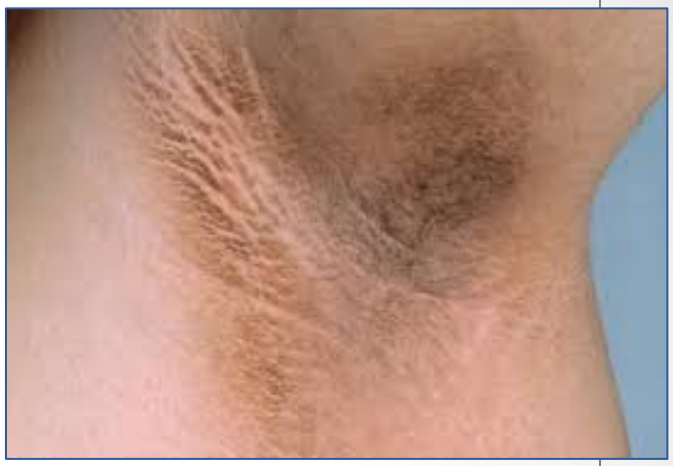
- On examination
  - Overweight
  - BMI – 98 / 99%ile
  - Pink stretch marks
  - Acanthosis nigricans
  - Blood glucose
    - 26mmol/L (468mg/dL)
    - Not acidotic
  - Urine
    - 3+ glucose
    - Moderate ketones



- **Diagnosis?**
- **Investigations?**
- **Treatment?**

Type 2 diabetes

- High risk ethnic group
- Family history
- Acanthosis
- BMI – obese
- Ketonuria
  - Unusual, but does occur in a 3rd of cases



- Because of the weight loss and ketonuria
  - Difficult to diagnose between Type 1 and Type 2 diabetes

Investigations to confirm?

- Islet cell and GAD antibodies
  - GAD test
    - Blood test to measure whether the body is producing a type of antibody which destroys its own GAD cells
      - Negative in Type 2
      - Positive in Type 1
- C-Peptide
  - Reflects amount of natural insulin that the child is producing
    - Normal or increased in Type 2
    - Low in Type 1

Treatment

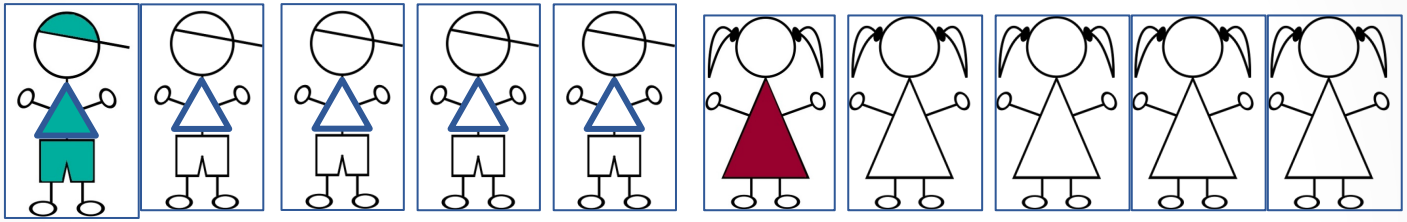
- Results of bloods may take some weeks…
  - High blood glucose
  - Ketosis
    - Basal bolus regime of insulin
    - Dietary treatment and good exercise regime very important
  - When ketosis has resolved
  - Blood glucose lowered
    - Metformin gradually introduced
      - Gradually increase
      - Decrease then stop insulin

National Child Measurement Programme

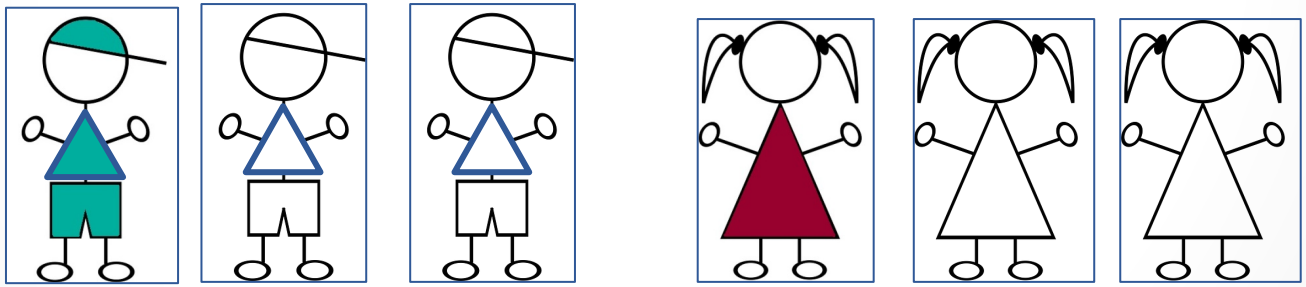
- Measures and records the height and weight of over one million children each year in the UK
  - Reception age (4-5 years)
  - End of primary school age (10-11 years)
- Principally to assess weight and obesity levels in schools
- Parents can opt out

Prevalence of excess weight among children in the UK – 2014 / 2015

One in five children in Reception is overweight or obese (boys 22.6%, girls 21.2%)

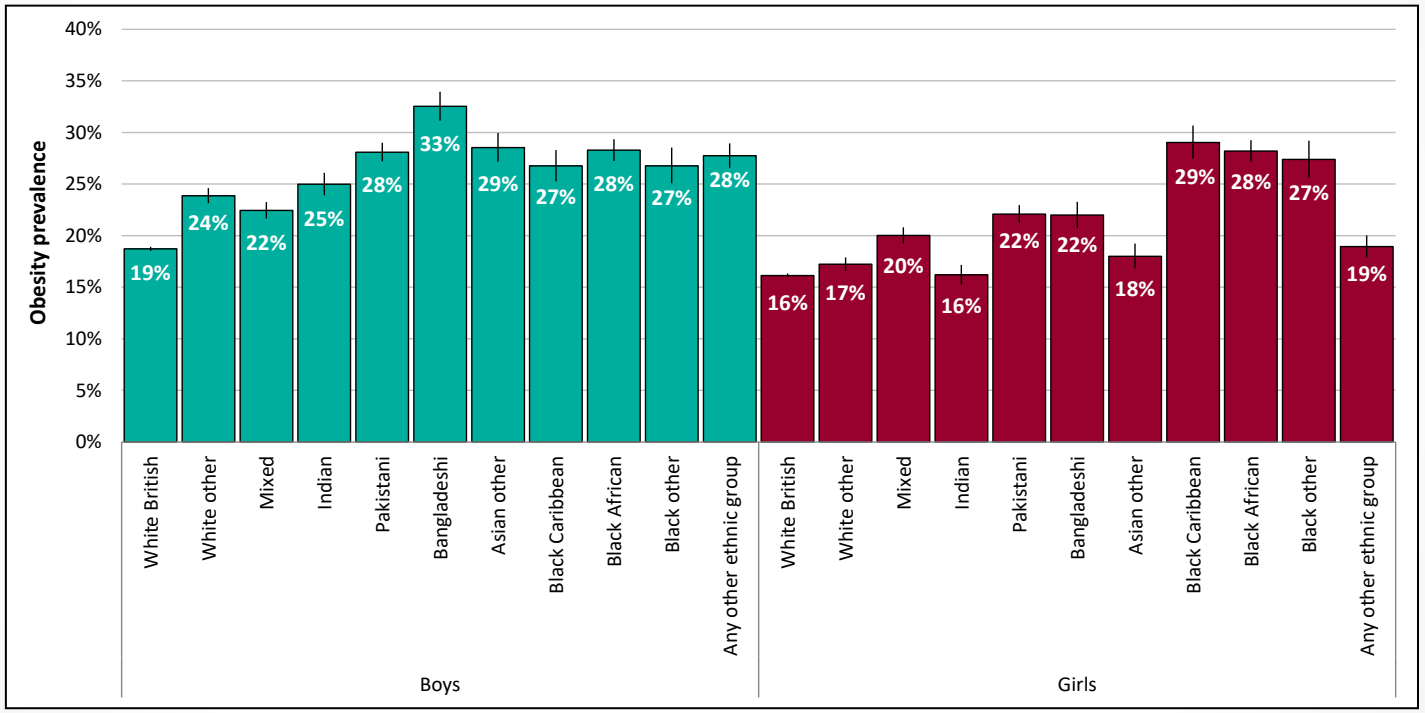


One in three children in Year 6 is overweight or obese (boys 34.9%, girls 31.5%)



Obesity prevalence by ethnic group: Year 6

NCMP 2014/15



Child obesity: BMI ≥ 95th centile of the UK90 growth reference

Causes of increasing childhood obesity

- Genes
  - Monogenic
  - Polygenic
  - Co-morbidity genes
- Environment
  - Quality of food supply
  - Food convenience
  - Food industry
  - Price
  - Activity

Food industry

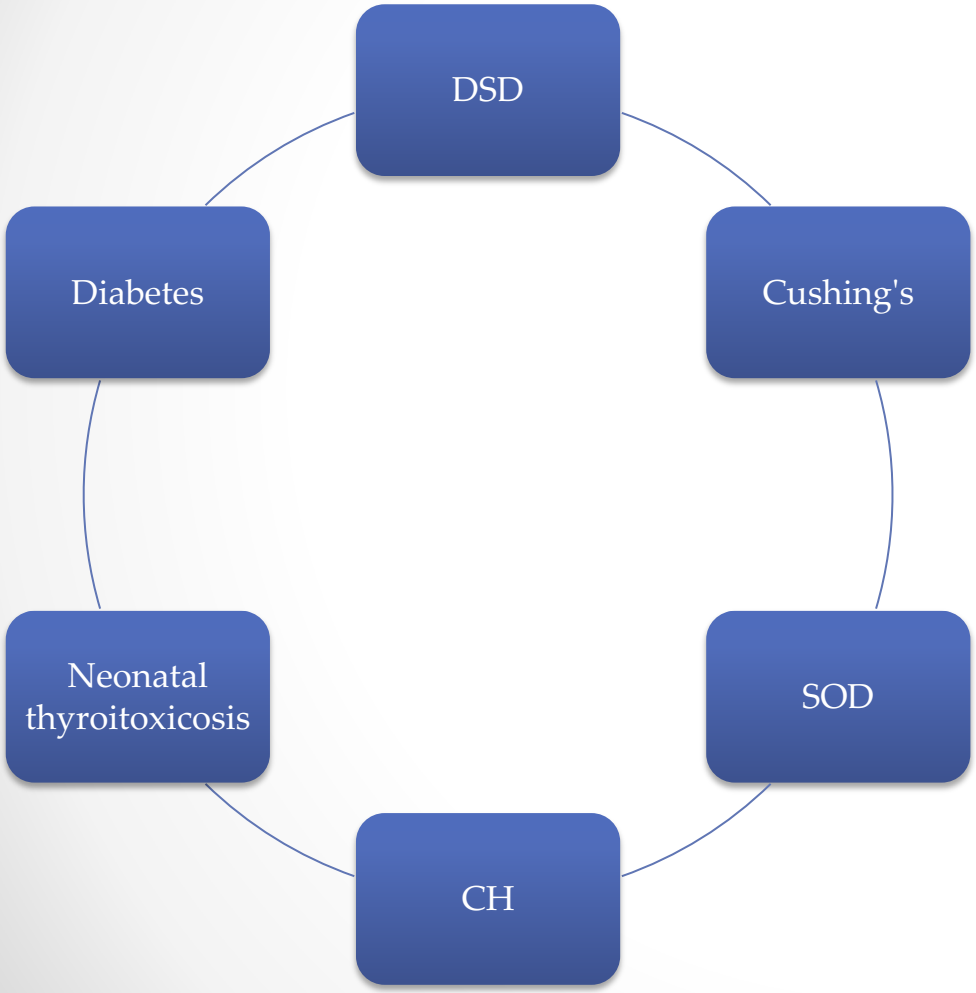
- £600 million spent last year marketing high sugar products to children
- Advertising – traditional and new
  - Advergames
    - Promoting material to websites
  - Characters
  - Social media
- Sponsorship of events, programmes and infrastructure

Management of Type 2 Diabetes

- Education
- Behavioural changes
- Dietary management
- Glycaemic monitoring
- Pharmacological therapy
  - Decrease insulin resistance
  - Increase insulin secretion
  - Slow post absorptio

- Biguanides
  - Metformin
  - Acts on insulin receptors in liver, muscle and fat tissue
  - Long term use – associated with 1 – 2% reduction in HbA1c
- Insulin
  - Small doses can be effective
- Sulphonylureas
  - Increase insulin secretion
    - Useful when there is residual beta cell function

Paediatric Emergencies



- Some more urgent than others
  - Urgent treatment
  - Urgent referral

Differences between adults and paediatrics?

References

- Davies, K. (2016) *Disorders of Sex Development – Ambiguous Genitalia* Journal of Pediatric Nursing 31(4)pp463 - 466
- Greening, J. E., Brain, C. E., Perry, L. A., Mushtaq, I., Sales Marques, J., Grossman, A. B. & Savage, M. O. (2005) *Efficient short-term control of hypercortisolaemia by low-dose etomidate in severe paediatric Cushing's disease* Hormone Research 64 (3) pp140-143
- Ogilvy-Stuart, A. L. (2002) *Neonatal thyroid disorders* Arch Dis Child Fetal Neonatal Ed 87: F165 – 171
- Raine, J. E., Donaldson, M. D. C., Gregory, J. W. and Van Vliet, G. (2013) *Practical Endocrinology and Diabetes in Children* Wiley-Blackwell