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Acute Care
Pediatric Nurse Practitioner
Review Course 2020

FEN/Endocrine

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Disclosures

I have no conflicts to disclose

Objectives

1. Discuss common fluid and electrolyte abnormalities, identify potential complications of excess or deficit and management strategies.
2. List common diagnoses managed by the AC PNP such as DKA, DI and SIADH, identify presentation, diagnostic evaluation and management of these problems.
3. Discuss the diagnosis, management and treatment of common Metabolic Diseases.
4. Accurately describe all pertinent diagnostic and laboratory findings and interpretations.

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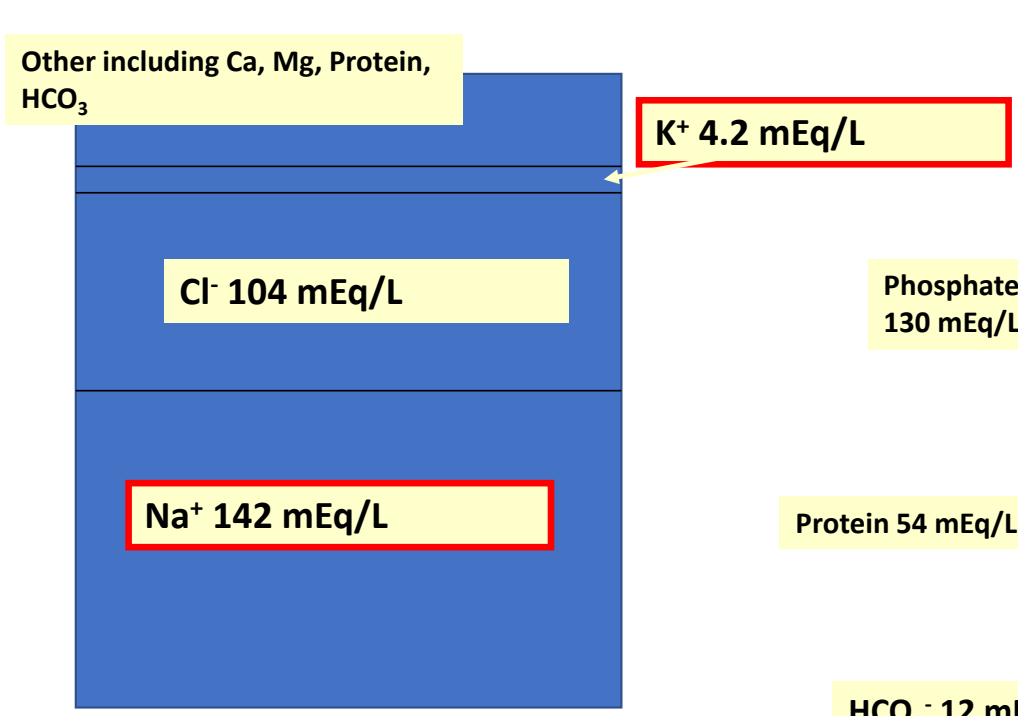
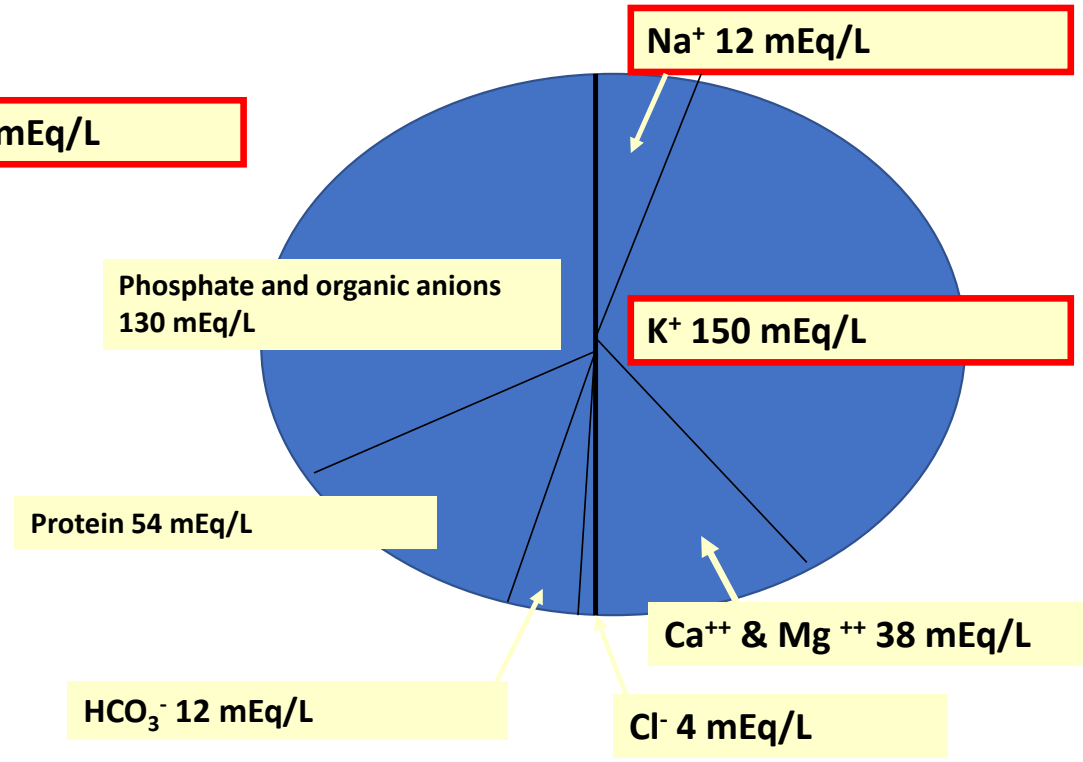
Fluid Composition in the Body

Extracellular

Intracellular





Maintenance Fluids

Daily Fluid Requirement-**Holliday-Segar Rule**

- 3–10 kg: 100 ml/kg/day
- 11–20 kg: 1000 ml +50 ml/kg each additional
- 21-70 kg 20 kg: 1500 ml +20 ml/kg for each additional kg

Ex: 27kg child

1500ml+ 7 X 20= 1500 + 140 = 1640ml/day or 68ml/hr

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Maintenance Fluids

Hourly Fluid Requirement-**4-2-1 Rule**

- 3–10 kg: **4** ml/kg/hr
- 10–20kg: 40ml/hr + **2**ml/kg/hr for each additional kg
- >21kg: 60 ml/hr+**1**ml/kg/hr for each additional kg

Ex: 27kg child

60ml/hr + 7 = 67ml/hr

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Fluid Composition

Fluid	Na ((g/L)	Cl (g/L	Glucose (g/L)
NS	154	154	0
LR	130	109	0
Hypertonic Saline	513	513	0
D51/2NS	77	77	50
Albumin	100-160	<120	0

(Modified from Kline-Tilford & Haut, 2015)

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Dehydration

Assess level of hydration: mild (5%) , moderate (10%), severe (15%)

Symptoms:

Mild-5%-Fatigue, may refuse PO, normal vitals

Moderate-10%-thirsty, restless, irritable, mild change in vitals, decreased tears, dry oral mucosa

Severe-15%-Lethargy, alteration in all vitals, cool, mottled, minimal output, deep breathing.

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Dehydration

- Types of Dehydration:
- **Isonatremic, Hypernatremic and Hyponatremic**
- **Keys to management**:

-Identify underlying problem

-Replace electrolytes slowly

-Correct fluid losses over 24 – 48 hours with fluid loss calculations based on electrolyte results and severity of dehydration.

-Maintenance and Replacement fluid infusions

-Monitor intake and output strictly, weights daily or BID

-Monitor electrolytes closely

Electrolyte Abnormalities

Hyponatremia:

Sodium levels less than 135, but severe is <125

- Causes: SIADH, Adrenal Insufficiency, hypervolemia, hypovolemia, excessive H2O intake
- Complications: **Seizures**
- Management: Correct Na at rate of 0.5meq/L/hr
- If symptomatic, NS 20cc/kg bolus, consider hypertonic saline 3-5ml/kg.
- If sodium corrected too rapidly risk of central pontine demyelination of white matter

Electrolyte Abnormalities

Hypernatremia:

Sodium levels greater than 145, severe is >160

- Causes: fluid loss, dehydration, excess sodium intake, DI
- Complications: Seizures, AMS
- Management: correct underlying cause, Correct Na at rate of 0.5meq/L/hr but no greater than 10mEq/L/day-prevent seizures

Electrolyte Abnormalities

Hyperkalemia:

- Causes: Excessive intake; decreased excretion (renal failure, beta blockers, acidosis), CAH, tumor lysis or rhabdomyolysis, RTA or hemolyzed sample.
- Signs: ECG changes - peaked T waves, depressed ST, wide QRS, absent P-wave
- Mild hyperkalemia Mgmt.: Make sure sample not hemolyzed, remove or stop all oral/IV K+ sources
- Severe: Perform EKG, get CMP, CPK, ABG, UA

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Hyperkalemia Effects on EKG

Ventricular fibrillation

Auricular Standstill

Prolonged P-R interval

- high T wave and depressed ST segment

Elevated T waves

Peaked T waves

Normal EKG

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Treatment of Severe Hyperkalemia with EKG changes

- *Stabilize the myocardium*
  - CaCl (10%) 20mg/kg IV
  - CaGluconate 60-100mg/kg/dose(max 3gms)
- Enhance movement of K+ into cells
  - Na H_2CO_3 1 to 2mEq/kg IV
  - Glucose 0.5 g/kg **PLUS** insulin 0.1 to 0.3 units/kg IV
- Remove K+ from the patient
  - Consider Kayexelate 1 g/kg PR or PR/NG-maintenance or adjunct
  - RRT = dialysis-severe
  - Diuretics/Albuterol-mild

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Electrolyte Abnormalities

Hypoglycemia:

Acute hypoglycemia – glucose less than 50 mg/dl – ketotic vs nonketotic

Causes:

Neonatal: diabetic mom, adrenocortical deficiency, inborn errors, hypopituitarism. Can be transient, immature fasting, lack of supply.

Childhood: inborn errors, growth hormone deficiency, stress, hepatic dysfunction, ingestion(beta-blockers), infection, uncontrolled diabetes

- Glucose replacement – 10% or 25% glucose – 0.5 – 1g/kg

Symptoms: jittery, irritable, seizures, vomiting, headache

*Need to send critical labs while hypoglycemic (FFA, insulin, BHOB, cortisol, acylcarnitine, lactate, pyruvate, NH4, GH, urine ketones). Endocrine consult.

Electrolyte Abnormalities

Hypocalcemia:

- **Causes**: hypoparathyroidism, Vit D deficiency, renal insufficiency, massive transfusion,rhabdomyolysis, tumor lysis and ethylene glycol ingestion.
- **Mgmt**: for hypoalbuminemia, correct Ca+ by 0.8mg/dl for each 1.0g/dl, consider ECG. Calcium replacement with calcium chloride or gluconate IV slowly and via Central line if possible.
- **Complication:** Seizures, Tetany, myocardial irritability, long QT, parasthesias

*In refractory states ensure Magnesium is normal

Metabolic Alkalosis/Acidosis

- Metabolic alkalosis
  - pH 7.52 PCO_2 40 PO_2 86 HCO_3 36
  - Chloride responsive: contraction alkalosis, diuretics, vomiting, gastric suctioning, and corticosteroid therapy
  - Chloride resistant: hyperaldosterone state, Severe K depletion
- Metabolic acidosis
  - pH 7.10, PCO_2 30, PO_2 96, HCO_3 10
  - Compensation is by hyperventilation-exhibited by low CO2
  - Bicarbonate losses occur as buffer system is imbalanced and other cations must accompany loss in the kidneys subsequently causing a loss in these electrolytes-usually potassium and sodium.

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Expected Anion Gap

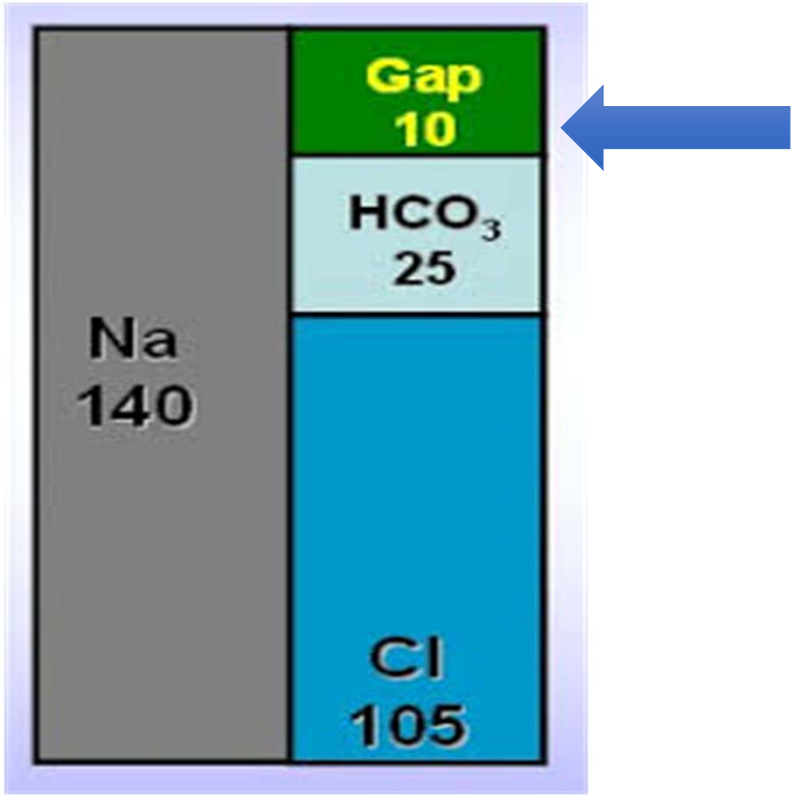
Calculation:

Cations – Anions

OR

Na - (Cl + HCO_3)

Normal range = 12± 2 mEq/L



Gap or Non-Gap Acidosis:

Example Non-Gap

Na 140, Cl 110, HCO3- 25

142-(110+25)= 7

Gap 7

Example Gap

Na 140, Cl 108, HCO3- 15

140-(108+15)= 17

Gap 17

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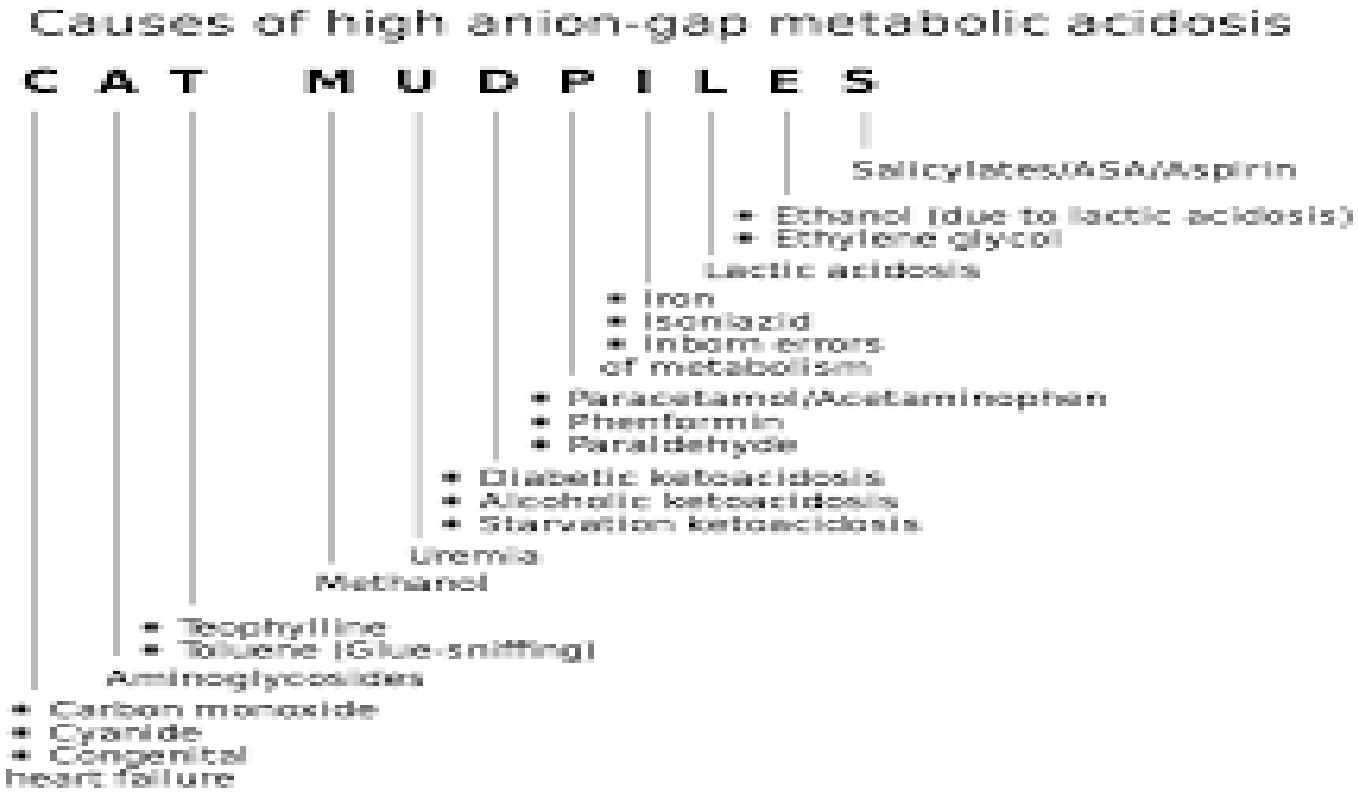
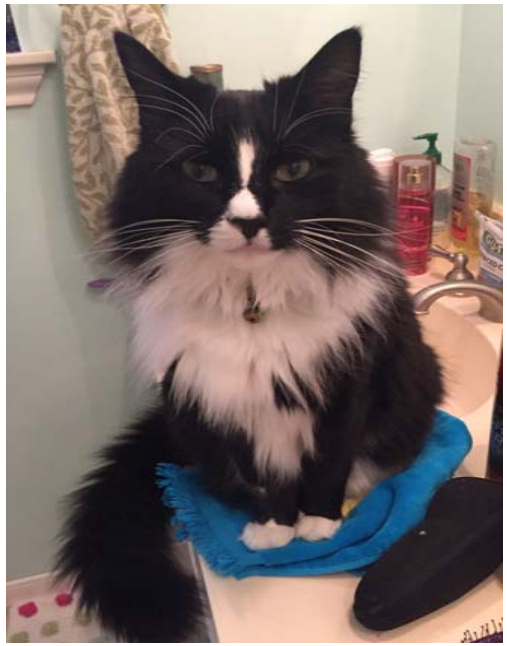
Non-Gap Acidosis

Normal Anion Gap Metabolic Acidosis
Diarrhea
Renal tubular acidosis
Adrenal insufficiency
Chronic kidney disease
Intestinal, pancreatic, biliary fistula
Hypoaldosteronism
Spironolactone, prostaglandin inhibitors, triamterene, amiloride, trimethoprim, pentamidine, cyclosporin

Gap Acidosis-CAT MUDPILES





Nutrition

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Eating Disorders-Obesity

- Eating Excess of calories.
- Defined as a BMI at or above the 95th percentile for children and teens of the same age and sex.
- **Health Risks**: Hypertension, Hypercholesteremia, impaired glucose tolerance, insulin resistance, sleep apnea, asthma, joint problems, Fatty liver disease, gallstones, and gastro-esophageal reflux, depression, behavioral problems, Low self-esteem low quality of life and suicidal or depressive behaviors.
- Labs: Cholesterol panel, fasting glucose, HGB A1C, insulin level.
- Consider psychiatric consult, nutrition or GI

Eating Disorders-Anorexia

- Restriction of calories resulting in electrolyte imbalances, intravascular volume depletion and metabolic acidosis.
- **Management**: Stabilize fluid status (tachycardia, hypotension), correct electrolytes (hypophosphatemia) and obtain psychiatric evaluation and therapy.
- **Complications:** Cardiac arrhythmias, severe hypophosphatemia and acid/base disturbance**.**

Eating Disorders-Bulemia

- Restriction of calories resulting in hunger from episodes of “binge eating”. Causes electrolyte imbalances. May have normal weight.
- **Management:** Stabilize fluid status (tachycardia, hypotension), correct electrolytes (hypophosphatemia) and obtain psychiatric evaluation and therapy.
- **Complications:** Cardiac arrhythmias, severe hypophosphatemia and acid/base disturbance, dentition problems**.**

Failure to Thrive

- Diagnosed when **<5%** on standard growth chart or when a child's weight for age crosses more than two major centile lines.
- Organic or inorganic? 90% inorganic

-inadequate caloric intake, inadequate absorption, excess metabolic demand or defective utilization.

- Genetic short stature, premature birth, constitutional
- Referrals-genetics, GI, endocrine, psych, nutrition

Enteral Nutrition

- Provision of nutrients via breast milk or formula to the GI tract.
- Preferred-start within 24-48hrs
- May improve outcomes, immune function and decrease infection and hospital LOS.

Contraindicated: Bowel obstruction. GI surgery or UGI bleed, vasopressors?

Labs: Pre-albumin, glucose, phosphorus, stool studies as needed.

Track weights, calculate daily caloric intake

Parenteral Nutrition

- Meeting nutritional needs when enteral feeding is contraindicated.
- **Solution based on**: Weight, age, organ dysfunction, disease state, metabolic needs, body composition/volume status and medications.
- Must have **reliable access.**
- **Macronutrients**: Protein, glucose and fat
- **Micronutrients**: Electrolytes, minerals and vitamins
- Daily adjustment needed based on needs
- Long-term effects:

Feeding Complications

- Refeeding
- Intolerance/Allergies
- Ileus
- Motility and emptying issues
- Obstruction-volvulus
- Constipation
- GERD

Nutrition Parameters

- Pre-albumin-Reflects last 24-48hrs, useful in acute nutritional changes. Not useful in inflammatory states, steroids or hemodialysis.
- Albumin-Less reliable, longer half-life(14-20 days). Negative acute phase protein: affected by dehydration, sepsis, trauma liver disease and albumin infusions.
- REE-Resting energy expenditure-based on age, equations to modify when sedated or paralyzed, stress or trauma states.
- Growth chart/BMI/Diet history
- Essential Fatty Acid Deficiency (EFAD)-Increase demands and limited reserves. Linoleic/alpha-linoleic are essential. Neonates occurs in days.
- Indirect calorimetry-used to ascertain substrate needs-prevents overfeeding and underfeeding. Expensive.

Nutrition and Catecholamines

- Catecholamines are released during stress, surgery, illness and this causes a counter regulatory release of hormones.

- Affects:

Decreases the affects of insulin and its release.

Glucagon and ACTH are increased which mobilizes amino acid from skeletal muscle tissue and increases cortisol levels.

This mediates release of free fatty acids.

Results: free fatty acid depletion, glycogen storage depletion, hyperglycemia, lipid intolerance and protein catabolism

Endocrine Disorders

Endocrine Disorders: SIADH, DI,CSW

SIADH Causes: CNS Injury, Diseases of hypothalamus or pituitary, hepatic disease, pulmonary disease, high dose chemotherapy	**Syndrome of inappropriate anti-diuretic hormone.** **Excess of ADH with increased permeability of renal distal tubules and collecting ducts results in increased water reabsorption and decreased urine production.**
Diabetes Insipidous (DI) Causes: CNS injury or infection, disorders of the hypothalamus, pituitary or pan-hypopituitarism, after tumor resection, primary and secondary renal defects	**Antidiuretic deficit: Inadequate levels of ADH, results in decreased water reabsorption, increased urine output, hypernatremia and dehydration** **Central: Genetic, congenital or acquired** **Nephrogenic: Congenital or acquired as a result of renal disease, metabolic conditions or medications**
Cerebral salt wasting (CSW) Causes: CNS injury or infection, endocrine disturbances, including DKA, chronic lung disease or BPD, cardiac disease	**Atrial natriuretic hormone excess, results in Na excretion into urine and diuresis with resultant Hyponatremia, euvolemia or hypovolemia**

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Lab Findings: DI, SIADH, CSW

	DI	SIADH	CSW
Serum Sodium	➢ 150	<135	< 135
Serum Osmolarity	➢ 295 mOsm/kg	<280	<280
Urine Sodium	< 30 Meq/L	>30	>80
Urine Osmolarity	<200mOsm/kg	>200	>200
Urine SG	<1.005	>1.020	>1.010
Urine output	>/=4ml/kg/hr	</= 1ml/kg/hr	2-3 ml/kg/hr

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Management

SIADH	DI	CSW
Restrict sodium, fluids, Monitoring of electrolytes and intake/output, diuretics, 9% NS	Vasopressin or DDAVP Fluid replacement Monitoring of fluids and electrolytes.	Treat underlying problem Replace sodium slowly, maintain fluid intake, monitoring of fluids and electrolytes.

Diabetes

Type I

- Pancreas no longer produces enough insulin or cells stop responding to the insulin that is produced, so that glucose levels are elevated and other metabolic problems persist.
- Presentation: **Polyuria**, **Polydipsia**, **Polyphagia**, fatigue, and weight loss- leads to dehydration.
- Complications of poorly-managed type 1 diabetes mellitus may include cardiovascular disease, diabetic neuropathy and diabetic retinopathy.

Type II

- Controlled with diet and exercise and sometimes oral hypoglycemic or combination of insulin and oral hypoglycemic. Relative insulin resistance. Numbers are increasing in pediatric population.
- Presentation: often obese child with hyperglycemia & glycosuria.
- Co-morbid conditions often present: PCOS, lipid disorders, HTN.

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Diabetic Ketoacidosis-DKA

DKA - Insulin deficiency in which a starvation state triggers a cascade of metabolic responses, including hyperglycemia and ketone body formation with lactic acidosis from decreased tissue perfusion resulting in a gap metabolic acidosis. DKA can present with initial diagnosis or throughout lifetime of patient with IDDM.

- **Presentation**: abdominal pain, vomiting, polyuria, hyperglycemia, ketonuria, lethargy, and other mental status changes.
- **PE findings:** tachycardia, signs of dehydration and Kussmal respirations.
- **Diagnostics:** Glucose, electrolytes, urinalysis, BOHB (>3mmol/L is diagnostic),venous blood gas, urine/serum ketones, CBC, liver function tests and amylase/lipase are sometimes warranted related to symptoms.

-Typical findings: hyperglycemia, low pH(<7.3), HCO3 less than 15, ketonuria, + serum and urine ketones.

DKA

- **Management:**
- Crystalloid bolus 10 ml/kg with slow replacement of fluids over 24-48hrs taking into account all fluids received. 2x maint for first 24hrs, then decrease to 1-1.5.
- Insulin drip at 0.05-0.1 units/kg/hr, add glucose when serum glucose is about 250 – 300 or if glucose levels are dropping faster than 100 dl/hr
- Replete potassium and phosphorous.
- **Labs:** glucose q1hr, chemistry, beta-hydroxybuterate, venous blood gases. Vital signs with neuro checks every 1 – 2 hours. Cardiac monitor, convert to subq insulin when ph and HCO3 are normalized.
- **Complications:** Cerebral edema-Altered mental status, confusion, seizures, headaches.

DKA-Cerebral Edema

- Critical complication seen in DKA. Only 1% of total cases
- Accounts for 60-90% of diabetes related deaths
- 10-25% of these are left with neurological deficits
- **Risk Factors:** Young age, new onset, bolus insulin dosing, bicarbonate administration, rapid fluid administration, increased BUN, low bicarb, rapid glucose correction.
- **Presentation:** Onset of headache, altered mental status/coma, hypoxia, Cushing's Triad(hypertension, bradycardia, resp depression)
- **Management:** Mannitol 0.5-1gm/kg or 3% Hypertonic saline. Fluid to manage shock but not excess-goal normal B/P, stabilize (ABCD's), increase HOB and keep midline, consider brain CT to evaluate cerebral edema.

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New Onset Diabetes

- Similar presentation, may correct quicker as some beta cell function still remains. Higher risk for complications.
- **Specific Labs**: Insulin auto-antibodies, insulin level, thyroid function tests, islet cell auto-antibodies, C-peptide levels

Adrenal Dysfunction

Adrenal Insufficiency:

-dysfunction of adrenal gland

Presentation: Hypotension, hypoglycemia, weakness, anorexia, nausea and vomiting.

-obtain CMP, glucose, ACTH, am cortisol level and aldosterone concentration.

-Mgmt: glucocorticoids, monitoring of function, endocrine consult

Types of Adrenal Insufficiency

Primary: CAH, sepsis, surgical removal, Addison disease, adrenal hemorrhage

Secondary deficiency: destroyed or inactive adrenal gland, ACTH deficiency

Tertiary insufficiency: suppression from steroids, rapid taper of steroids

Relative Insufficiency(CIRCI): critical illness/ shock

Congenital Adrenal Hyperplasia

– Most common is 21-hydroxalase deficiency.

- **Presentation**: Newborn with ambiguous genitalia, salt wasting and shock, get reduced cortisol and aldosterone production get: typical lab findings of hyperkalemia and hyponatremia with dehydration.
- **Diagnostics**: Serum electrolytes, ACTH stimulating test, Cortisol levels (timing), Newborn screen
- **Treatment:** Manage fluid and electrolytes, glucocorticoids, hydrocortisone.

Adrenal Crisis

Rapid, overwhelming process that is potentially **fatal**

-Occurs with chronic adrenal insufficiency, acute damage, abrupt withdrawal of steroids.

-**Presentation**: hypotension, fatigue, vomiting, muscle pain, anorexia, wt loss.

-**Diagnostics**: Obtain CMP, glucose, ACTH, am cortisol level and aldosterone concentration

-**Management**: glucocorticoids, monitoring of function

Hyperthyroidism

Thyroid Storm	Hyperthyroidism	Thyroiditis
Life threatening condition due to untreated hyperthyroidism; clinical findings: HTN, fever, tachycardia, sweating **Complications:** Congestive heart failure and pulmonary edema can develop rapidly and lead to death	Nervousness, irritability, emotional lability, tremor, excessive appetite, weight loss, smooth moist, skin, increased perspiration, heat intolerance. **Physical findings**: goiter, exophthalmos, tachycardia, widened pulse pressure. **Management:** PTU or methimazole, surgery or iodine.	Inflammation of the thyroid leading to hyperthyroidism **Diagnostics:** Thyroid function tests – elevated T4, decreased TSH. **Management:** varies, PTU or methimazole.

Hypothyroidism

Hypothyroidsm

Findings: growth retardation, diminished physical activity, impaired tissue perfusion, constipation, thick tongue, poor muscle tone, hoarseness, anemia, intellectual retardation. Can be associated with other disorders (trisomy 21, Diabetes).

- **Diagnostics**: Thyroid function tests – Low Free T4 and T3, elevated TSH.
- **Management**: Lifelong replacement of thyroid hormone as levothyroxine.

Sick Euthyroid

Findings: Low T3 in non-thyroidal illness during time of critical illness. Correlates with high levels of pro-inflammatory cytokines and cortisol.

- **Diagnostics**: Low T3 with normal TSH. Free T4 and reverse T3 vary but often reverse T3 goes up.
- **Management**: Not treated with thyroid hormone, will resolve once critical illness and passed.

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Hypo/Hyper Thyroidism At a Glance

Hypothyroidism	Hyperthyroidism
Hypothermia	Hyperthermia
Weight gain	Weight loss
Bradycardia, lower blood pressure	Tachycardia, hypertension
Constipation	Diarrhea
Decreased activity, listlessness	Restless, anxious
Cold intolerance	Heat Intolerance

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Metabolic Disorders

Inborn Errors of Metabolism:

- Amino Acid Disorders
- Glycogen Storage Diseases
- Lysosomal Storage Diseases
- Mitochondrial Diseases
- Organic Acidemia
- Peroxisomal Disorders
- Phenylketonuria
- Urea-cycle Diseases

- **Key Findings:** hypoglycemia, hyperammonemia, metabolic acidosis
- **Other Findings:** infant presents with seizures, lethargy or coma, recurrent unexplained illness, hepatosplenomegaly, hypotonia, signs of sepsis, developmental delay, loss of milestones, poor feeding, vomiting, eye/hair/skin abnormalities, Ketosis.

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Disorders of Protein Metabolism

Amino Acid Disorders

- Most common disorders, caused by a defect in the metabolic pathway of amino acids resulting in abnormal accumulation of amino acids in plasma, often present acutely in newborn, will result in coma, encephalopathy or death if not identified.

Urea Cycle Disorders

- Deficiency of enzyme or cofactor that transforms nitrogen to urea for excretion, presents few day after birth with poor feeding, vomiting, tachypnea and AMS after a few days of protein intake.

Organic Acid Disorders

- Enzyme deficiency or abnormal step in pathways of amino acid degradation, accumulation of abnormal organic acid metabolites and increased excretion of organic acids in the urine. Presents in newborn period with poor feeding, lethargy, acidosis and ketosis, high **ammonia** levels.

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Disorders of Fat Metabolism

Fatty Acid Oxidation Disorder:

- Pathophysiology: Single-enzyme defects either in one of the beta-oxidation steps for fatty acids or in the pathway that transfers electrons from FADH2 to the electron-transport. Previously healthy child presents with vomiting, lethargy/coma, occasionally seizures, +/- hepatomegaly triggered by an acute illness

Disorders of Glucose Metabolism

Glycogen storage disease

Carbohydrate disorders such as galactosemia

- Pathophysiology: Deficiency of galactose-1-phosphate uridyl transferase (GALT), disorders of glycogen breakdown. Present as seizures, poor growth, hypotonia, metabolic acidosis and hyperbilirubinemia.

Disorders of Organelles

Mitochondrial disorders

Lysosomal storage disorders (mucopolysacharidosis)

- Pathophysiology: Deficiency of lysosomal enzymes leading to excessive tissue storage of lipid material or incomplete degradation & storage of mucopolysaccharides
- Presentation: Developmental delay/MR, Symptoms: hepatomegaly, splenomegaly, neurologic regression, short stature, coarsening of facial features, limitation of small & large joints, cardiac disease
- Peroxisomal disorders

Question

Which of the following is a characteristic of bulimia nervosa?

A Thin appearance

B Severe hunger

C Amenorrhea

D Eating control

Answer:

Which of the following is a characteristic of bulimia nervosa?

B. Severe hunger

Question

A lethargic breastfed infant born at birthing center without a newborn screen presents to the ER with poor muscle tone, hoarse cry, normal pH and hypothermia. What does the provider suspect?

A Botulism

B Metabolic disease

C Hypernatremia

D Hypothyroidism

Answer:

A lethargic breastfed infant born at birthing center without a newborn screen presents to the ER with poor muscle tone, hoarse cry, normal pH and hypothermia. What does the provider suspect?

D. Hypothyroidism

Question

A one week old presents in profound shock with metabolic acidosis and is being fluid resuscitated. A strange odor is noted from the child, what disorder is expected?

A. Lysosomal storage defect

B. Fatty Acid Defect

C. Organic Acid Defect

D. Hypothyroidism

Answer:

A one week old presents in profound shock with metabolic acidosis and is being fluid resuscitated. A strange odor is noted from the child, what disorder is expected?

C. Organic Acid Defect

Fluid and Electrolyte Question

What electrolytes should be checked immediately on a child with new onset seizures?

A glucose, sodium and phosphorus

B sodium, calcium and phosphorus

C glucose, sodium and calcium

D calcium, sodium and magnesium

Answer:

What electrolytes should be checked immediately on a child with new onset seizures?

C. glucose, sodium and calcium

Fluid and Electrolyte Question

A 2 month old with vomiting presents with dry mucous membranes, a weak cry and cool extremities. Which course of action should be taken?

A Fluid bolus of D5NS and re-assess

B Check glucose and administer NS bolus

C Administer NS bolus and D25W 2ml/kg

D Warm, check glucose and bolus D5NS

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Answer:

A 2 month old with vomiting presents with dry mucous membranes, a weak cry and cool extremities. Which course of action should be taken?

B. Check glucose and administer NS bolus

Fluid and Electrolyte Question

A child is admitted urgently with AKI. The labs at presentation are: Na 140, Cl 110 HCO3 14. What is the anion gap?

A 14

B 16

C 13

D 17

Answer:

A child is admitted urgently with AKI. The labs at presentation are: Na 140, Cl 110 HCO3 14. What is the anion gap?

B. 16

Calories Question?

What kcal/kg/day is a 5kg infant receiving who took in 500ml of similac 27kcal/oz?

A 75

B 90

C 100

D 110

Answer:

What kcal/kg/day is a 5kg infant receiving who took in 500ml of similac 27kcal/oz?

B. 90

Question?

What is an expected electrolyte response during critical illness?

A Hypermagnesemia

B Hyperglycemia

C Hypercalcemia

D Hypokalemia

Answer:

What is an expected electrolyte response during critical illness?

B Hyperglycemia

Endocrinology Challenge Question

A 3 year old newly diagnosed diabetic presents in DKA and develops acute agitation and confusion during fluid resuscitation. What is the most likely diagnosis?

A Hemorrhagic stroke

B Cerebral edema

C Hypernatremic seizure

D Hypoglycemia

Answer:

A 3 year old newly diagnosed diabetic presents in DKA and develops acute agitation and confusion during fluid resuscitation. What is the most likely diagnosis?

B. Cerebral edema

Endocrinology Challenge Question

A 12 year old with mental challenges presents with severe diarrhea and dehydration. In the process of electrolyte evaluation and rehydration, he is noted to have excess urine output and rising sodium level.

What is the most likely diagnoses?

A. Diabetes Insipidus

B. Cerebral salt wasting

C. SIADH

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Answer:

A 12 year old with mental challenges presents with severe diarrhea and dehydration. In the process of electrolyte evaluation and rehydration, he is noted to have excess urine output and rising sodium level.

What is the most likely diagnoses?

A. Diabetes Insipidus