

Electrolyte Abnormalities in Neonates

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Electrolyte Abnormalities

- Sodium/Water Balance
- Hyponatremia/Hybernatriemia
- Hypokalemia/Hyperkalemia



Sodium and Water Homeostasis

- Sodium
 - Tight regulation
 - RAA system
 - Sympathetic NS
 - Atrial Natriuretic Peptide
 - Brain Natriuretic Peptide
 - Closely correlated ECV
 - Vascular volume
 - Serum water
- Water
 - ADH
 - Osm – 1-2% change
 - Baroreceptors
 - Osmolarity and effective perfusion

Sodium/Water Balance

- Transition from fetal physiology
 - Late term fetus
 - High F_{xNa}
 - Transition – to low F_{xNa}
 - Most species during 1st day
 - Fetal foal - before birth
- Sodium conserving mode
 - Na requirement for growth
 - Bone growth
 - ↑ body mass
 - Increase in interstitial space
 - Milk diet
 - Fresh milk is sodium poor
 - 9-15 mEq/l

Sodium/Water Balance

Sodium Overload

- Sodium containing intravenous fluids
 - 6-7 mEq Na/kg/day
 - Mare's milk – 1.8 mEq Na/kg/day
 - 3-4 X normal Na
- Sodium overloading
 - Expansion of the extracellular fluid space
 - Sodium fractional excretion will remain low
- Difficulty dealing with volume loading

Hyponatremia





Hyponatremia

Spurious hyponatremia

Dilutional hyponatremia

Depletional hyponatremia

Redistribution hyponatremia

Spurious Hyponatremia

- Normal plasma sodium concentration
- Laboratory reports a low concentration
 - Presence of interfering substances
 - Lipids or large proteins
 - Artificially dilutes sample
 - Not with modern assay techniques
 - Mistakes in sampling
 - Venipuncture site distal to a low Na drip
 - Sample is taken from a catheter
 - Infusion of a low Na solution
 - Insufficient dead space clearing

Dilutional Hyponatremia

- Lack of balance
 - Fluid intake/urine output
- Loss of integrity of the urinary system
 - Ruptured bladder
 - Ruptured/necrotic urachus
 - Fenestrated ureters
- Renal failure
 - Low GFR
 - Na wasting

Dilutional Hyponatremia

- Failed/delayed renal transition fetal to neonatal physiology
 - Low GFR
- Water overload
 - Management mistakes
 - Dilute milk replacer
 - Excessive water enemas (retained)
 - Fluid therapy errors (Na wasting renal syndromes)
 - Syndrome of inappropriate antidiuresis (SIA)
 - Appropriate antidiuresis
 - Decreased arterial volume

Dilutional Hyponatremia

- Most common form hyponatremia in neonates
- Only occurs with intake of hyponatremic fluid
 - Fresh milk
 - Hyponatremic rehydration formulas
 - Dextrose in water or half strength saline
- Not with isotonic Na containing fluids
 - Normisol-R, Lactated Ringers, Plasmalyte
 - Less marked on milk replacer than fresh milk

Hyponatremia

Syndrome of Inappropriate Antidiuresis (SIAD)

- Synonym: SIADH
 - Syndrome of Inappropriate Antidiuretic Hormone Secretion
- Hyponatremia secondary to
 - Inappropriate reabsorption of water from urine
- Diagnosis
 - High urine osmolarity
 - Hyposmolar hyponatremia - plasma
 - Normal renal function
 - Normal adrenal function
 - Euvolemia
- Can have excessive renal sodium excretion
 - Often absent in the neonate
 - Low sodium intake

Hyponatremia

Syndrome of Inappropriate Antidiuresis (SIAD)

- Clinical syndrome
 - Sudden decrease in urine output
 - High urine specific gravity
 - Weight gain
 - 10-15% of body weight overnight
 - No edema
 - Decreasing plasma sodium concentration
- ADH increased
 - No osmotic stimulus
 - No hypovolemic stimulus

Hyponatremia

Syndrome of Inappropriate Antidiuresis (SIAD)

■ SIADH

- Inappropriate vasopressin release
 - Erratic and unpredictable release vasopressin
 - Reset of the osmostat
 - Threshold for release is lowered
 - Vasopressin release not fully suppressed at low osmolarity
 - But normal at higher osmolarity
- Receptor abnormality (vasopressin release normal)
 - Hypersensitive receptors
 - Receptors continue to respond
 - After vasopressin levels decrease
 - Hypovasopressinemic antidiuresis

Hyponatremia

Syndrome of Inappropriate Antidiuresis (SIAD)

- SIAD not SIADH
 - High urine osmolarity
 - Hyposmolar hyponatremia
 - Hypovolemia
 - Appropriate vasopressin release
 - Defense of volemia

Hyponatremia

Syndrome of Inappropriate Antidiuresis (SIAD)

- Nonosmotic ADH release
 - Subclinical volume depletion
 - Pain, Stress
 - Hypotonic fluids exacerbates hyponatremia
- Abnormal adrenal function
 - Increased ACTH
 - Stimulate ADH release
 - Cortisol deficiency
 - Reduced CO and BP
 - Non-osmotic ADH release
 - Aldosterone deficiency
 - Na wasting, reductions in ECV
 - Non-osmotic ADH release
- Abnormal renal function

Depletional Hyponatremia



- Na loss > water
- Diarrhea
 - Excessive sodium loss in feces
 - Rehydration with Na poor fluids
 - Fresh/frozen milk
 - Fresh water
- Renal sodium wasting
 - Tubular disease
 - Use of diuretics
 - Endocrine disturbances
 - Cerebral Salt Wasting Syndrome
 - Rehydration with Na poor fluids
 - Fresh/frozen milk
 - Fresh water

Cerebral Salt Wasting Syndrome CSWS

- Hypothalamic-Renal Axis disease
- Characterized by
 - Natriuresis
 - Volume depletion
 - Followed by water retention
 - ADH induced
- Clinical findings
 - Hypovolemia
 - Hyponatremia
 - Inappropriately high urine osmolality
 - Not Usg by refractive index
 - Increased urine Na (> 40 mEq/l)

Cerebral Salt Wasting Syndrome

CSWS

- Pathogenesis????
 - Increased SNS outflow
 - Increased atrial and brain natriuretic peptides
- Traumatic intracranial injury
- Differentiate from SIADH
 - CSWS
 - Volume depletion
 - Increased urine sodium excretion
 - Before development of hyponatremia
 - SIADH
 - Euvolemic or hypervolemic
 - Low/normal urine Na (in neonate)

Redistribution Hyponatremia

- Low sodium concentration
 - Osmolarity normal
 - Isosmotic hyponatremia
 - Hyperosmotic hyponatremia
- Other osmotically active particles present
 - Redistribute fluid from intracellular space
 - Appropriate decrease Na concentration
 - Hyperglycemia (Na, Glu in mmol/L)
 - $Na_{corrected} = Na_{measured} + [(Glu - 5)/3.5]$
 - 1.6 mEq/l Na decrease for 5.55 mmol/L glu increase
 - Iatrogenic addition of osmoles
 - Mannitol
 - Extreme hyperproteinemia
 - Secondary to sick cell syndrome

Hyponatremia

Sick Cell Syndrome

- Critically ill patients
 - Cellular insult
 - Loss of cell wall integrity
 - Solutes leak
 - Fluid follows
 - Dilution of extracellular sodium



Hyponatremia

Clinical Assessment

- Classify hyponatremia
 - Hypervolemic
 - Euvolemic
 - Hypovolemic



Hyponatremia

Clinical Assessment

- Hypervolemic hyponatremia
 - Impaired water excretion
 - Ruptured bladder
 - Renal failure
 - Decreased GFR
 - Failure to dilute urine
 - Hypovolemia leading to fluid retention
 - Congestive heart failure
 - Hypovolemia – non-osmotic ADH
 - Poor renal perfusion
 - RAS
 - Increased sympathetic tone
 - Na retention and volume retention
 - ADH excess

Hyponatremia

Clinical Assessment

- Hypervolemic hyponatremia
 - Splanchnic/systemic vasodilatation
 - Decreased ECV
 - Non-osmotic release of ADH
 - Increased total body water + Na
 - Water > Na
 - Edema, ascites
 - Decreased arterial volume/BP
 - Stimulate ADH secretion

Hyponatremia

Clinical Assessment

- Euvolemic hyponatremia
 - Most common in hospitalized patients
 - ADH mediated water retention
 - Iatrogenic
 - Rx hypotonic fluids in patients
 - When stimulus for increased ADH secretion
 - Total body Na normal
 - No edema
 - Defines euvolemic

Hyponatremia

Clinical Assessment

- Hypovolemic hyponatremia
 - Deficit water & Na
 - Na deficit > water deficit
 - Fluid loss – renal water retention (ADH)
 - Etiology
 - Diarrhea
 - Na wasting nephropathy
 - CSWS
 - Diuretics
 - Hemorrhage
 - Appropriate ADH secretion
 - Non-osmotic release
 - Retain water because of hypovolemia
 - Further decreasing osmolarity

Hypotonic Hyponatremia

Clinical Findings

- Asymptomatic or symptomatic
- Severe
 - < 115 mEq/l – arbitrary definition
- Acute
 - $< 36-48$ hr
- Progressive brain edema
 - Weakness, depressed reflexes
 - Obtunded, abnormal sensorium
 - Hypothermia
 - Cheyne-Stokes respiration
 - Seizures, Coma
 - Brainstem herniation
 - Respiratory arrest

Hypotonic Hyponatremia

Brain Edema

- Abrupt hypo-osmolarity
 - Aquaporin-4 mediated
 - Water moves into brain cells
- Compensation
 - Decreased cerebral venous pooling
 - Decreased CSF volume
 - Pediatric – less relative CSF volume
- Cell volume regulatory ions
 - Na, K, Cl
 - Released within 5 min from cells
 - Can continue for 4 hours
 - Stretched cell membranes open ion channels
 - Hormone regulated cellular ion differences
 - Predisposes females to more damage

Hypotonic Hyponatremia Brain Edema

- Reduction of cellular osmolytes
 - Polyalcohols
 - Sorbitol
 - Inositol
 - Amino acids
 - Methylamines
 - Within 2-4 days
 - Up to 50% reduction osmolytes

Hypotonic Hyponatremia Brain Edema

- Early and late phase changes
 - Limit cellular edema
 - Limit damage
- Hypoxic ischemic encephalopathy
 - Compounds hyponatremia damage
 - Interferes with compensation
 - Cerebral edema
 - Interferes with cerebral perfusion
 - Impairs central respiratory centers

Hypotonic Hyponatremia

Chronic, severe

- Asymptomatic
 - Oligosymptomatic
 - Not treat?
- Only for days
 - Somnolent
 - Disoriented
 - Often Rx
- Causes
 - Diuretics
 - SIADH
 - Malnourishment/liver disease
 - Diarrhea

Hypotonic Hyponatremia

Chronic, severe - treated

- Risk of cerebral myelinolysis
 - Central pontine myelinolysis
 - Initial report 12 patients – weak evidence
 - Other reports extrapontine
 - May occur in 25% cases
 - Over rapid correction
 - Other risk factors
 - Hypokalemia
 - Alcoholism
 - Experimental studies – Na 118 to 140 mEq/l
 - 48 hrs – OK
 - 8 hr – 90% myelinolysis
 - Rx hypertonic saline
 - Rx ADH antagonist

Hypotonic Hyponatremia Myelinolysis - Pathogenesis

- Osmotic reloading fails
 - Rapid correction
 - Brain cell Na, K
 - Normal
 - High because of overshoot
 - Amino acid content low
- Osmotic reloading
 - Organic osmolytes take up to 5 days
 - Normal metabolic molecules
 - Parts of cellular proteins/lipids
 - External addition not speed
 - Transport depends on new gene expression
 - RNA transcription – protein production
 - Slowest where lesions occur
 - Blood brain barrier has role

Hypotonic Hyponatremia Myelinolysis - Pathogenesis

- Blood brain barrier
 - Major target of cellular edema
 - Vascular endothelial cell
 - Astrocyte foot processes
 - Cell shrink opens cell junctions
- BBB disrupted
 - Not protect
 - Plasma constituents have access

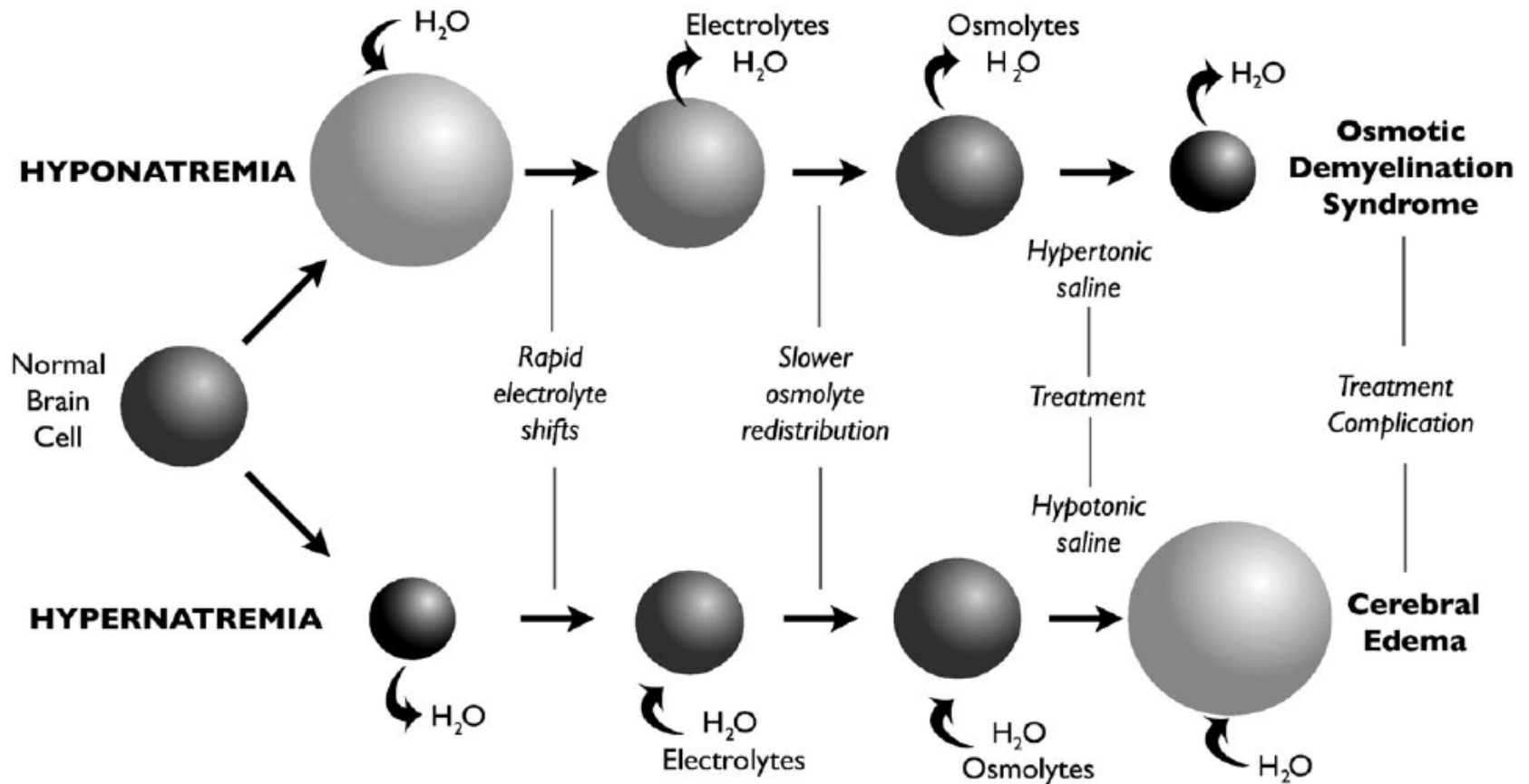
Hypotonic Hyponatremia Myelinolysis - Pathogenesis

- Factors associated
 - Overshoot hypernatremia
 - Increasing Na > 25 mEq/L 24-48 hr
 - Concurrent hypoxic event
 - Liver disease
- Rate of Na rise
 - Less important than magnitude
- Symptomatic hyponatremia
 - More important to treat
 - Than threat of myelinolysis
- Signs days after Na correction
- MRI lesions best seen 2 weeks after

Hypotonic Hyponatremia Treatment

- Recognize cause
 - Don't treat spurious, redistribution hyponatremia
- Symptomatic – euvoolemia/hypervolemia, with concentrated urine
 - Hypertonic saline
 - Furosemide – limit volume expansion
 - Stop water intake
- Symptomatic – hypovolemia
 - Isotonic fluids
- Mild symptomatic – dilute urine
 - Evaporative losses only

Hyponatremia/Hypernatremia Osmotic Shifts



Hypotonic Hyponatremia

Treatment - Correction rate

- Acute – rapid
- Chronic
 - Increase Na < 0.5 mmol/L/hr
 - Stop once serum Na = 120-126
- If seizures
 - Increase Na 1 – 1.5 mmol/L/hr for 1st 3 hr
 - Or until seizures stop
 - Give 1-2 ml/kg/hr 3% saline
 - If seizures severe up to 4-6 ml/kg/hr
 - Add furosemide
 - Stop when Na = 118 mEq/l
 - Goal 125 mEq/l

Estimate Effect of Infusate

For each liter given
Change in serum [Na] =
$$\frac{(\text{Infusate Na} + \text{Infusate K}) - \text{serum Na}}{\text{Total body water} + 1}$$

Total body water

early neonate = 0.75 X body wt

pediatric = 0.6 X body wt

adult = 0.5-0.6 X body wt

geriatric = 0.45-0.5 X body wt

Estimate Effect of Fluid-Loss

For each liter loss
Change in serum [Na] =
$$\frac{\text{serum Na} - (\text{Fluid-loss Na} + \text{Fluid -loss K})}{\text{Total body water} - 1}$$

Total body water

early neonate = 0.75 X body wt

pediatric = 0.6 X body wt

adult = 0.5-0.6 X body wt

geriatric = 0.45-0.5 X body wt

** Incorrect estimate of TBW will effect accuracy

Fluid-loss Na/K is Na/K of urine or GI fluid lost

Hypotonic Hyponatremia Treatment

- Vasopressin Antagonists
 - Better predictability
 - Better fine tuning
- Hyponatremia and ADH
 - Primarily disease of excess ADH
 - In face of continued hypotonic fluid intake
- Not use with hyponatremia because of
 - Renal disease
 - Volume contraction
- Oral products being tested

Hypotonic Hyponatremia

Vasopressin Antagonists

- Demeclocycline
 - Blocks at tubules
- Aquaretics
 - V_{1a} & V_2
 - Conivaptan
 - V_2
 - Lixivaptan
 - Satavaptan
 - Tolvaptan

Hypernatremia



Hypernatremia

- Uncommon
- Deficit of water relative to Na stores
 - Water loss > Na loss
 - GI – osmotic diarrhea
 - Insensible loss
 - Renal loss
 - Hyperglycemic hyperosmolar syndrome
 - Diabetes insipidus
 - Nephrogenic/Central
 - Medullary “washout”
 - Tubular concentration abnormality

Hypernatremia

- Deficit of water relative to Na stores
 - Lack of water intake ***
 - Must be present for hypernatremia
 - Combined
 - Free water losses
 - Renal Na conservation
 - Limited water intake
- Excess Na relative to water
 - Na intake > water
 - Sodium toxicity
 - Lack of water intake must also be present

Hypernatremia

- Clinical differentiation
 - Na toxicity
 - Low urine volume
 - High Usg
 - High FxNa
 - Free water loss – not renal
 - Low urine volume
 - High Usg
 - Low FxNa
 - Free water loss – renal
 - High urine volume
 - Low Usg
 - Low FxNa

Hypernatremia

- Hypertonic hyperosmolality - always
- Causes of hypernatremia
 - Spurious
 - Excessive free water loss
 - Pure water loss
 - Hypotonic fluid loss
 - Hyperosmotic intake
 - Iatrogenic

Spurious hypernatremia

- Sampling errors

- Blood samples from the intravenous catheter

- Not large enough presample
- Sample contamination
 - with saline



Hypernatremia

Increased free water loss



- Increased insensible loss
 - Increased respiratory rate
 - Low humidity
 - High body temperature
 - External warming
 - Radiant heat
 - Hot air heat
- Increased insensible loss with limited intake
 - Hot weather
 - Neonate unable to nurse
 - Lack opportunity
 - NE

Hypernatremia

Increased free water loss

- Water loss
 - Diabetes insipidus
 - Unusual because of neonate's diet
 - $U_{osm} < P_{osm}$
- Water > Na loss
 - Osmotic diuresis
 - Hyperglycemic hyperosmolar syndrome
 - Need interruption nursing/water access
 - Milk replacer with no water access
 - Glucosuria
 - Mannitol
 - Furosemide
 - Renal disease
 - Diarrhea
 - Excessive sweating

Hypernatremia

Hyperosmotic Intake

- High sodium maternal milk
 - Excessive sodium intake relative to free water
- Iatrogenic mishaps
 - Improperly mixed electrolyte solutions
 - Without the opportunity/ability to drink fresh water
 - Improperly mixed milk replacer
 - All powdered milk replacers are sodium rich
 - Use of hypernatremic intravenous fluids solutions
 - 5% sodium bicarbonate
 - Hypertonic saline
 - Use of saline in oxygen humidifiers
 - Hypertonic enemas (retained)

Hypernatremia

Normal defense against

- Concentrate urine
 - Osmolar release ADH
- Thirst
 - Only develops if can't get to water

Hypernatremia Treatment

- Recognize cause
 - Eliminate/manage underlying problem
- If developed acutely (hours)
 - Can be corrected over hours ($\downarrow\text{Na}$ 1 mmol/hr)
 - Usually acute sodium loading
- If developed slowly (over days)
 - Intracellular accumulation organic osmolytes
 - Correct slowly to avoid cerebral cellular edema
 - $\downarrow\text{Na} < 0.5$ mmol/hr (target $\downarrow\text{Na}$ 10 mmol/day)

Hypernatremia Treatment

- If Na > 170
 - Don't decrease < 150 for 48-72 hr
- Oral fluid therapy
 - As soon as possible
 - Na and K in milk
- Seizures during treatment
 - Common
 - From cerebral edema
 - Slow correction

Hypernatremia

Estimate Effect of Infusate

For each liter given

$$\text{Change in serum [Na]} = \frac{(\text{Infusate Na} + \text{Infusate K}) - \text{serum Na}}{\text{Total body water} + 1}$$

Total body water

early neonate = 0.75 X body wt

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Hypokalemia



Hypokalemia

- Hypokalemia common in neonates
- Anabolic increase in cell mass (growth)
 - Potassium major intracellular ion
- Renal K wasting
 - Diuresis
 - Renal pathology

Hypokalemia Stress/sepsis

- Resting muscle
 - Uses 10% of available $\text{Na}^+:\text{K}^+$ ATPase activity
- Stimulated acutely by
 - Insulin
 - Epinephrine
 - Contractile activity

Hypokalemia Stress/sepsis

- Stress/Sepsis → ↑ epinephrine
 - ↑ $\text{Na}^+:\text{K}^+$ ATPase activity
 - Significant intracellular shifts of K → hypokalemia
 - ↑ ATPase demand
 - ↑ glucose utilization/requirement
 - ↑ glucose transport into the cell resulting
 - → further shift K intracellular??

Hypokalemia Pathogenesis

- Loss of 1% total body potassium
 - Disturbs transcellular distribution
 - Results in physiological changes
- Blood levels
 - Not correlate total body stores
 - Rapid drop more likely clinical signs
- Loss 100 mEq K
 - Blood level decreases by 0.3 mEq/l
 - If there are no confounders

Hypokalemia

Signs - man

- Muscle weakness
- Paralytic ileus
- Cardiac arrhythmias
 - Atrial tachycardia
 - Atrioventricular dissociation
 - Ventricular tachycardia/fibrillation
 - Flat or inverted T-waves
 - ST-segment depression
 - U-waves

Hypokalemia

Signs - man

- $K < 2.5$
 - Rhabdomyolysis with myoglobinuria
 - Acute renal failure
- $K < 2.0$
 - Ascending paralysis
 - Impairment of respiratory function
- Not recognize in neonatal foals

Hypokalemia

- High levels of potassium in milk
 - Will support growth requirements
- Stressed/Septic neonates
 - Not tolerate oral feeding
- Neonates require significant K supplementation
 - Prolonged intravenous glucose
 - Parenteral nutrition
 - Limited or no milk feeding
- Glucocorticoid administration
 - Mineralocorticoid receptor stimulation
 - → urine loss of potassium

Hypokalemia Treatment

- Neonates
 - Milk diet – not need supplement
 - Fluids only
 - If normal plasma levels – 3 mEq/kg/d
 - If hypokalemic – 6-12 mEq/kg/d or more

Hyperkalemia



- Differential diagnosis
 - Ruptured bladder
 - Urinary tract defect
 - Sick cell syndrome
 - Iatrogenic

Hyperkalemia

- Loss of integrity lower urinary tract
 - ↑K only when on a milk diet
 - Also true for ↓Na, ↓Cl
 - Receiving parenteral nutrition
 - ↑K only occur with overzealous K administration
- Sick cell syndrome
 - Suffer global cell insult
 - Perinatal hypoxic ischemic asphyxial insults
 - ↑K = 6-8 mEq/l
- Hyperglycemia – no insulin response
- Iatrogenic in the face of renal insufficiency

Hyperkalemia Signs

- Impaired neuromuscular transmission
 - Cardiac
 - Neuromuscular paresis/paralysis
- General fatigue
- Weakness
- Paresthesia

Management of Hyperkalemia

Questions to be asked:

Is there an emergency related to hyperkalemia?

If so, what is the plan for therapy?

Is pseudohyperkalemia present?

Did this hyperkalemia develop acutely?

Toxicity more related to rate of increase than level

Toxicity associated with acid-base status

What role of

High K intake

Decreased renal K excretion

Transcellular K shift

Management of Hyperkalemia

- ECG changes
 - Peaked T wave
 - False + depending on lead placement
 - Decrease/absence P waves
 - False + depending on lead placement
 - Prolonged PR interval
 - Widened QRS
 - Sine wave QRS
 - Asystole
- ECG not sensitive indicator of hyperkalemia
 - $K > 6.0$ – 46% abnormal ECG (in man)
 - $K > 6.8$ – 55% abnormal ECG (in man)
- Not predicable progression

Management of Hyperkalemia

- Pseudohyperkalemia
 - Sample hemolysis
 - Long, tight tourniquet (man)
 - Leukocytosis ($>70,000$; serum)
 - Thrombocytosis ($>1,000,000$; serum)
 - Measure plasma vs serum
 - If serum > 0.3 higher - pseudohyperkalemia

Management of Hyperkalemia

Urgent Therapy

- Oppose direct toxic effects
 - Ca therapy
- Promote cellular uptake K
 - Insulin (glucose)
 - β_2 -agonists (albuterol)
 - NaHCO_3 - not
- Remove from body
 - Drain uroabdomen
 - Diuretics
 - Cation exchange resins

Management of Hyperkalemia

Urgent Therapy

- Calcium
 - Ca gluconate – less irritating
 - CaCl – Ca more biologically available
 - Slow IV push
 - ECG changes within 1-3 minutes
 - Lasts 30-60 minutes

Management of Hyperkalemia

Urgent Therapy

- Insulin
 - Increase Na/K pump – primarily muscle/liver
 - Increase Na/H exchanger as well – may be important
 - Onset 20 min with peak 30-60 min
 - Dose related
 - Use glucose only to prevent hypoglycemia
 - Dose – 10 units as a bolus
 - Lasts 4-6 hr
 - CRI use in neonates
 - Hypoglycemia risk – monitor

Management of Hyperkalemia

Urgent Therapy

- Adrenergic Agents
 - β_2 -receptor in the muscle and liver
 - Directly activate Na/K-ATPase
 - Increase cAMP
 - Albuterol
 - By insufflation or IV
 - K drop within 30 min, peak 90-120 min
 - Drop 0.6-1 mEq/l
 - Has no effect 40% dialysis patients (man)
 - Effect additive to insulin

Management of Hyperkalemia

Urgent Therapy

- NaHCO_3
 - Poor response
 - Requires 4 hrs for effect
 - K decreased < 0.35 mEq/l at 6 hr
- Combined therapy
 - Insulin + β_2
 - Different mechanisms
 - In theory additive 60-100%
 - But not all respond to β_2
 - Combination – less frequent hypoglycemia
 - Insulin + NaHCO_3
 - Potentiate effect?

Management of Hyperkalemia

Direct Elimination from Body

- Gastrointestinal Route
 - Sodium polystyrene sulfonate
 - Cation exchange resin
 - 1 gm removes 0.5-1 mEq/l K
 - 4-6 hr required for full effect
 - Oral, enema
 - Constipates – give with laxative
 - Minimal short term effect
 - Depends on gut perfusion

Management of Hyperkalemia

Direct Elimination from Body

- Renal
 - Na diuresis
 - Deliver Na to distal K secreting sites
 - Renal disease often attenuates effect
- Abdomen
 - Draining uroabdomen
 - Peritoneal dialysis



