Fluid and electrolyte management in neurosurgery

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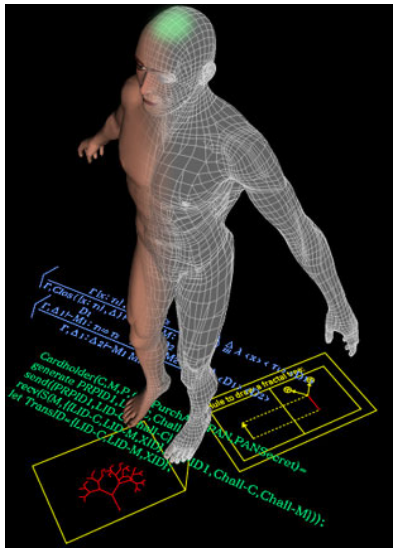
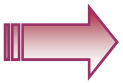
INTRODUCTION
Body Fluid Compartments:

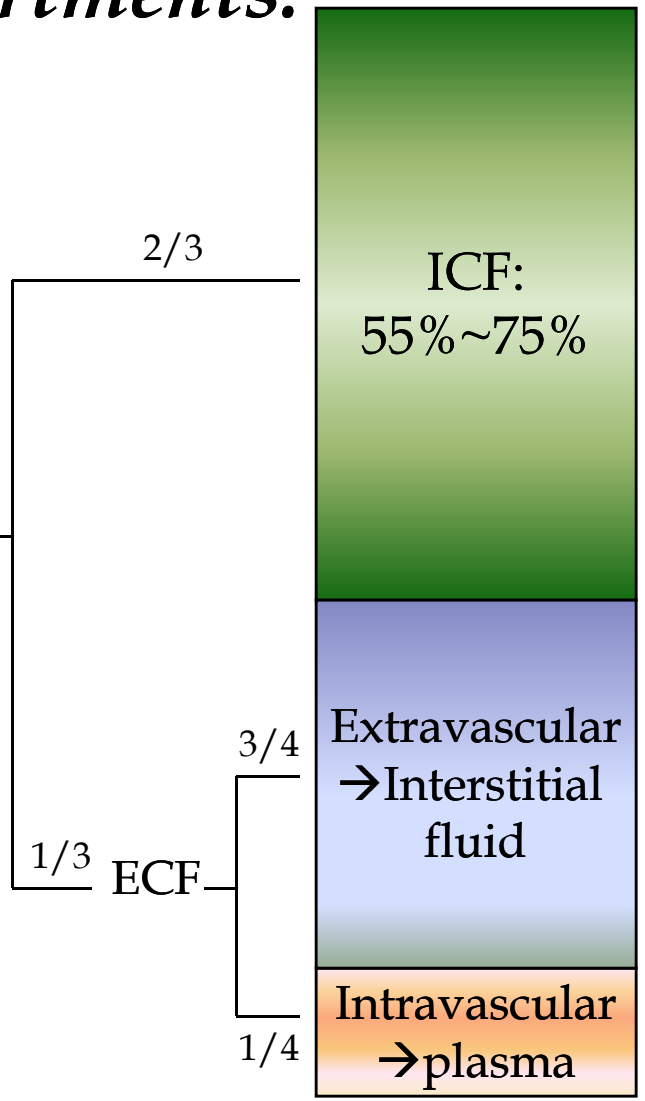


X 50~70%
lean body weight

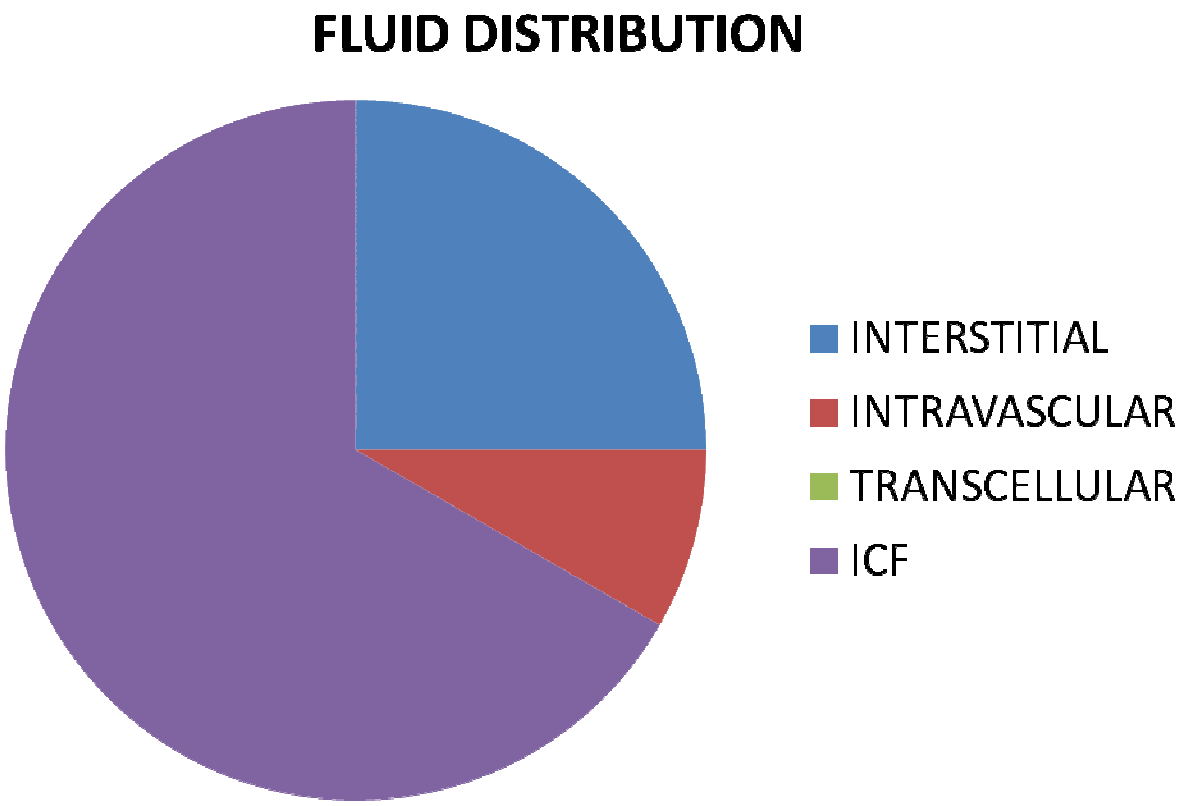


TBW

- Male (60%) > female (50%)
- Most concentrated in skeletal muscle
- TBW=0.6xBW
- ICF=0.4xBW
- ECF=0.2xBW

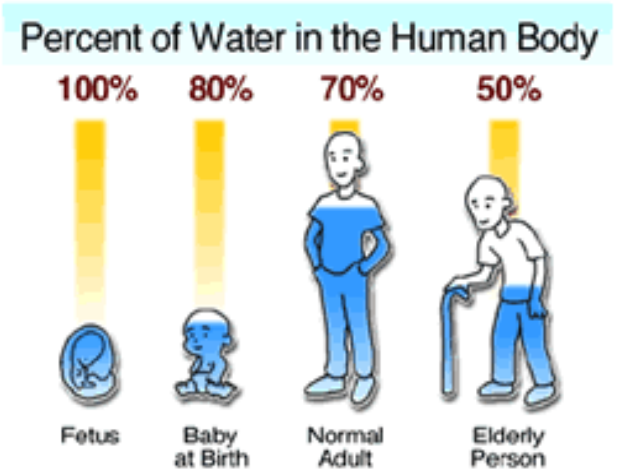


Introduction

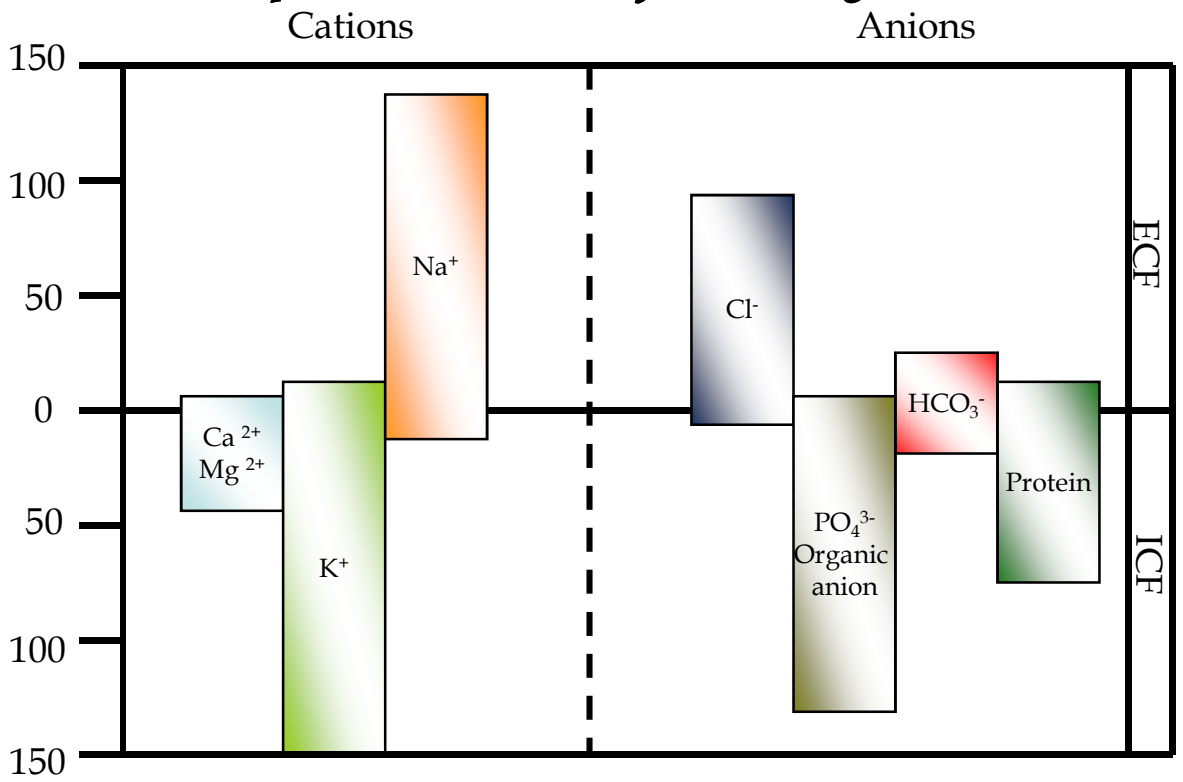


Introduction

- Total body water 94% weight in early gestation
- Decreases to 78% at term
- Reaches adult levels of 60% by 9 months



INTRODUCTION
Composition of Body Fluids:



- Osmolarity = solute/(solute+solvent)
- Osmolality = solute/solvent (290~310mOsm/L)
- Tonicity = effective osmolality
- Plasma osmolility = 2 x (Na) + (Glucose/18) + (Urea/2.8)
- Plasma tonicity = 2 x (Na) + (Glucose/18)

Objectives of IV Therapy

- Maintain daily body fluid requirements
- Restore previous body fluid losses
- Replace present body fluid losses

Average daily water balance of a healthy adult (70 kg)

Intake		output	
Beverage	1200ml	urine	-1500ml
solid food	1000ml	insens.loss	- 900ml
Oxidation	300ml	faces	-100ml

- **Fever** Pyrexia increases insensible loss by about 20% for each C rise in body temperature
- **Ventilator**
- **GI losses** Equivalent volume of normal saline added with potassium chloride use to cover the gastrointestinal fluid loss and fluid sequestrated in the bowel.

Normal volume and composition of body fluids

Source	Daily loss (ml)	$[Na^+]$ (mEq/L)	$[K^+]$ (mEq/L)	$[Cl^-]$ (mEq/L)	$[HCO_3^-]$ (mEq/L)
Saliva	~1,000	20-80	10-20	20-40	20-160
Gastric juice	1,000 – 2,000	20-100	5-10	120-160	0
Bile	~1,000	150-250	5-10	40-60	20-60
Pancreatic juice	1,000 – 2,000	120	5-10	10-60	80-120
Succus entericus	1,000 – 2,000	140	5	Variable	Variable
Colon	200-1,500	75	30	30	0
Sweat	200-1,000	20-70	5-10	40-60	0

Clinical parameters for evaluation of water balance

- CVP
- Pulse
- Peripheral Veins
- Weight
- Thirst
- Intake and Output
- Skin
- Edema
- Lab Values

Maintenance dose

For 24hrs

100ml/kg for 1-10kg

50ml/kg for 11-20

20ml/kg for 21-above

or 4ml/kg/hr for 1-10kg

2ml/kg/hr for 11-20

1ml/kg/hr for 21-above

Pediatric neurosurgical patients over 24hrs

- premature 90-100ml/kg
  at term 80-90ml/kg
  3m-1yr 70-80ml/kg
  >1yr 70ml/kg

Why quality of fluids is important?

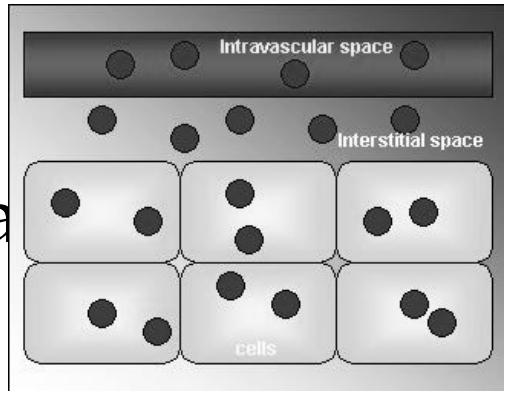
- Effect on cerebral oedema formation
- Effect on CPP
- Effect on glucose
- Effect on electrolytes

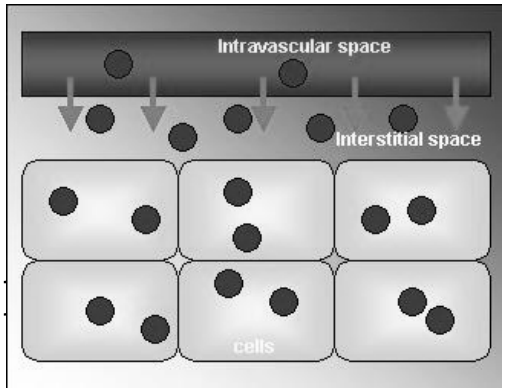
- IV fluids divided into crystalloid and colloid depending on molecular weight of solutes

crystalloids	<30,000 mmol
colloids	>30,000mmol

Crystalloids:

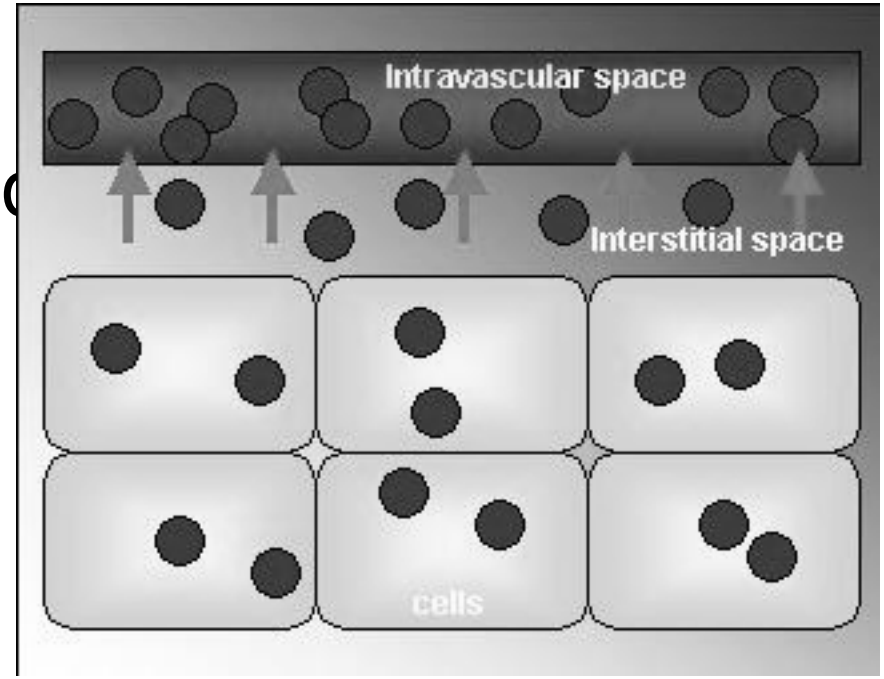
- Isotonic crystalloids
  - Lactated Ringer's, 0.9% NaCl
  - only 25% remain intravascula
- Hypertonic saline solutions
  - 3% NaCl
- Hypotonic solutions
  - D5W, 0.45% NaCl
  - less than 10% remain intra-vascularly, inadequate for flu resuscitation





Colloid Solutions:

- Contain high molecular weight substances→do not readily migrate across capillary walls
- Preparations
  - Plasma protein fracti
  - Gelatins
  - Dextrans
  - Starches



Plasma Derived Colloids

Plasma (FFP, cryoprecipitate)

- Coagulation problems only

Albumin

Plasma protein fraction/SHS

- Very expensive
- No proven benefit
- ? harmful

Colloids

	GELATINS	DEXTRANS	HES
Molecular weight	28-35 kda	40-70 kda	70-450 kda
Advantages	Improves circulation	Improves circulation	Improves circulation, endothelial function
Anaphylaxsis	High	small	minimal
VOLUME EFFECT	SHORT	MEDIUM	LONGTERM
COAGULATION	+/-	++	+
DOSE LIMITATION	NO	15ml/kg/24hr	33ml/kg/24hr

Which Fluids ?

- Depends on Nature of Loss!
- Balanced approach for resuscitation:
  2-3 crystalloid then colloid
- ? 0.9% saline or HS for head injuries
- Ringer's for other fluid resuscitation
- Colloids included for major resuscitation
- Blood as needed for Hct = 30

Crystalloid

- Extracellar expander
- Limited volume expansion
- Maintain urine output
- Reduce plasma oncotic pressure
- Variable electrolyte content
- Cheap!

Colloid

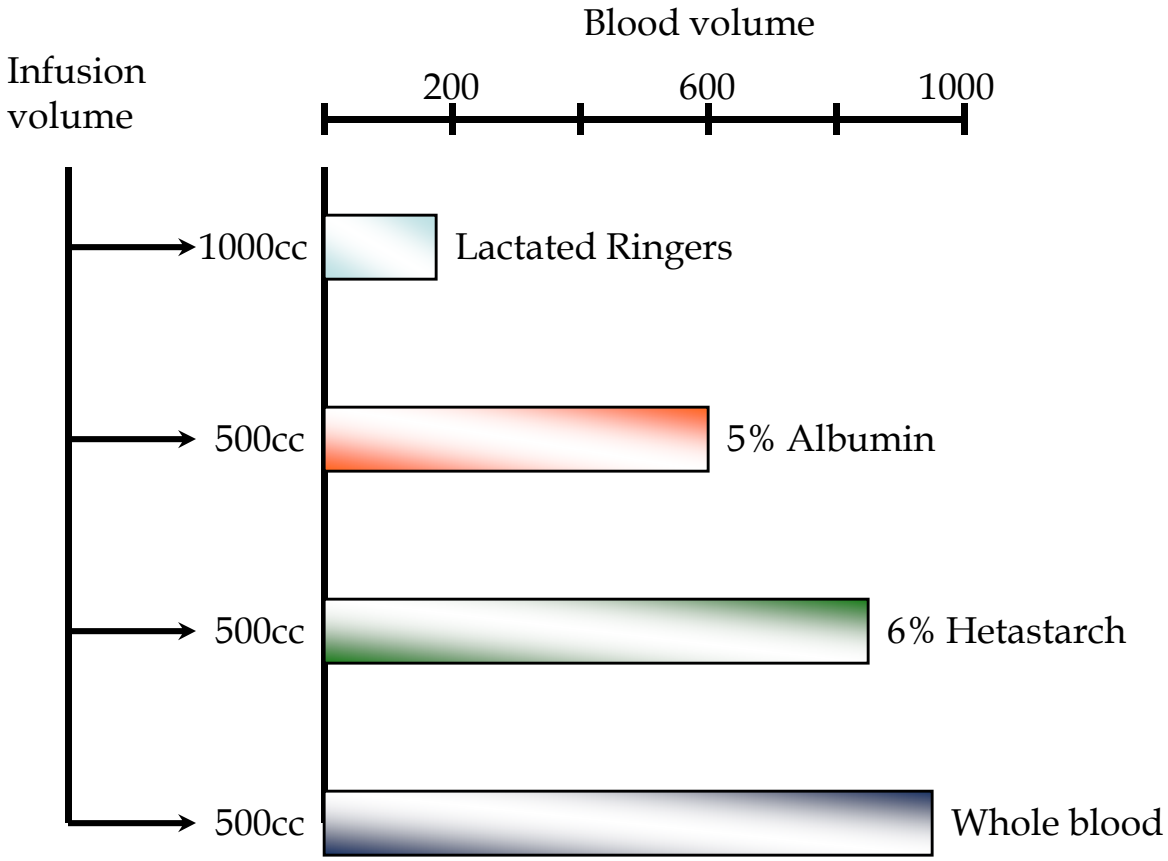
Advantages:

- Intravascular expanders
- Volume expansion
- Rapid resuscitation
- Maintain oncotic pressure
- Less tissue oedema
- Less pulmonary oedema

Disadvantages:

- Coagulation problems
- Variable electrolyte content
- Variable half-life
- Adverse reactions
- EXPENSIVE!

The Influence of Colloid & Crystalloid on Blood Volume:



HYPOOSMOLAR CRYSTALLOIDS

1. Should not be used except for treatment of D. insipidus/ chronic SDH

2. Solution of 5% dextrose is hypoosmolar

3. RL is also slightly hypoosmolar, administration of >3 litres can reduce plasma osmolality.

ISOOSMOLAR SOLUTIONS

osmolality approximately that of plasma.

Eg: NS, Plasmalyte, and Normosol R.

Hyperchloremic metabolic acidosis may result with NS

INTRAOPERATIVE FLUID INFUSION

Usually Ringer's lactate and/or Normal Saline

- Avoid dextrose fluids

Infusion rate should be to replace urine output and insensible losses ml. for ml.

Replace blood loss at 3:1 ratio crystalloids or 1:1 of colloids down to haematocrit of 25-30%

INTRAOPERATIVE FLUID INFUSION

- Permissible blood loss

$$EBV \times (H_i - H_d) / H_i$$

EBV=weight x avg blood volume

MANNITOL

Increases plasma- cerebral parenchyma osmolality gradient

dose -0.5-2.0 gm/kg

large dose enhances cerebral blood flow and free radical scavenging

HYPERTONIC SALINE

- Mechanism of action is similar to mannitol
- Strengths of HS – 3, 5 7.5%
- Increases serum Na^+ and hypokalemia causes less diuresis

Blood products

- Transfusion of blood in the form of packed cell if Hb< 8
- Dilutional clinical coagulopathy when blood loss exceeds more than one blood volume
- Fresh frozen plasma to be administered if prothrombin time> 1.5 time normal
- Dose of FFP: 10- 15 ml/ kg (to obtain 30% of plasma factor concentration)
- Prophylactic administration contraindicated
- Platelet deficiency occurs if blood loss exceeds > 1.5 times EBV (14)
- Transfusion indicated if counts less than 50,000/ cmm or if higher counts with bleeding
- Dose of platelet concentrate- one platelet concentrate per 10 kg body weight
- Use of antithrombin III controversial

Management in SAH

- Dual set of problems
  - Hypovolemia
  - Hyponatremia- CSW and SIADH
- Avoid dehydration
- Cerebral vasospasm by –
  - hypervolemia by CVP of 8- 10
  - hemodilution by PCV around 30- 35
  - hypertension
- Avoid dextrose containing solutions
- Prefer colloids for volume expansion

Electrolytes

Normal values

Na^+ - 130-149meq/l

K^+ - 3.5-5meq/l

Cl^- 95-110 meq/l

Ca- 8.1-10.4mg%

Daily recommended amount

Electrolyte	Parenteral Equivalent of RDA	Usual Intake
Sodium		**1–2 meq/kg + replacement, but can be as low as 5–40 meq/d**
Potassium		**40–100 meq/d + replacement of unusual losses**
Chloride		As needed for acid-base balance, but usually 2:1 to 1:1 with acetate
Acetate		As needed for acid-base balance
Calcium	10 meq	10–20 meq/d
Magnesium	10 meq	8–16 meq/d
Phosphorus	30 mmol	

Factors controlling sodium Reabsorption in Perioperative Period

- RAS:- Renin secretion ↠ increases formation of Angiotensin II↠↑Aldosterone ↠ Na reabsorption in Distal tubule

- Sympathetic nervous system- Increased Sympathetic activity increases sodium absorption in proximal tubule

- ADH- it has little action on sodium excretion.,it mostly maintains extracellular fluid volume

- ANP- it is released from atrial cells & causes afferent arteriolar dilation & efferent arteriolar constriction thus increasing GFR & natriuresis

HYPONATREMIA

Hypovolumic

CCF

NEPHROTIC SYNDROME

RENAL FAILURE

CIRRHOSIS

Hypervolumic

Extra renal sodium loss

diarrhea

Vomiting

Blood loss

Excessive sweating

Renal sodium loss

CSWS

Diuretics

Osmotic diuresis

Adrenal insufficiency

ketonuria

Euvolumic

- SIADH

CNS

SOL

Trauma

Hemorrhage

Stroke

Inflammatory disorders

demyelination

Drugs

Carbamazepine

Chlorpropramide

Phenothiazines

SRI

TCA

Pulmonary conditions

Infections

ALI

Neoplasia

THIAZIDE DIURETICS

HYPOTHYROIDISM

ADRENAL INSUFFICIENCY

Signs & Symptoms of Hyponatremia

Moderate-

lethargy
Nausea/vomiting
Irritability
Headache
Muscle weakness/cramps

Severe

Drowsiness
Depressed reflexes
Seizures
Coma
Death

- Cerebral edema occurs at<123meq/lit

Assesment of Pt with Hyponatremia

Clinical-

- Skin turgor & mucous membranes
- JVP
- Orthostatic variation in pulse and B.P
- daily wt.

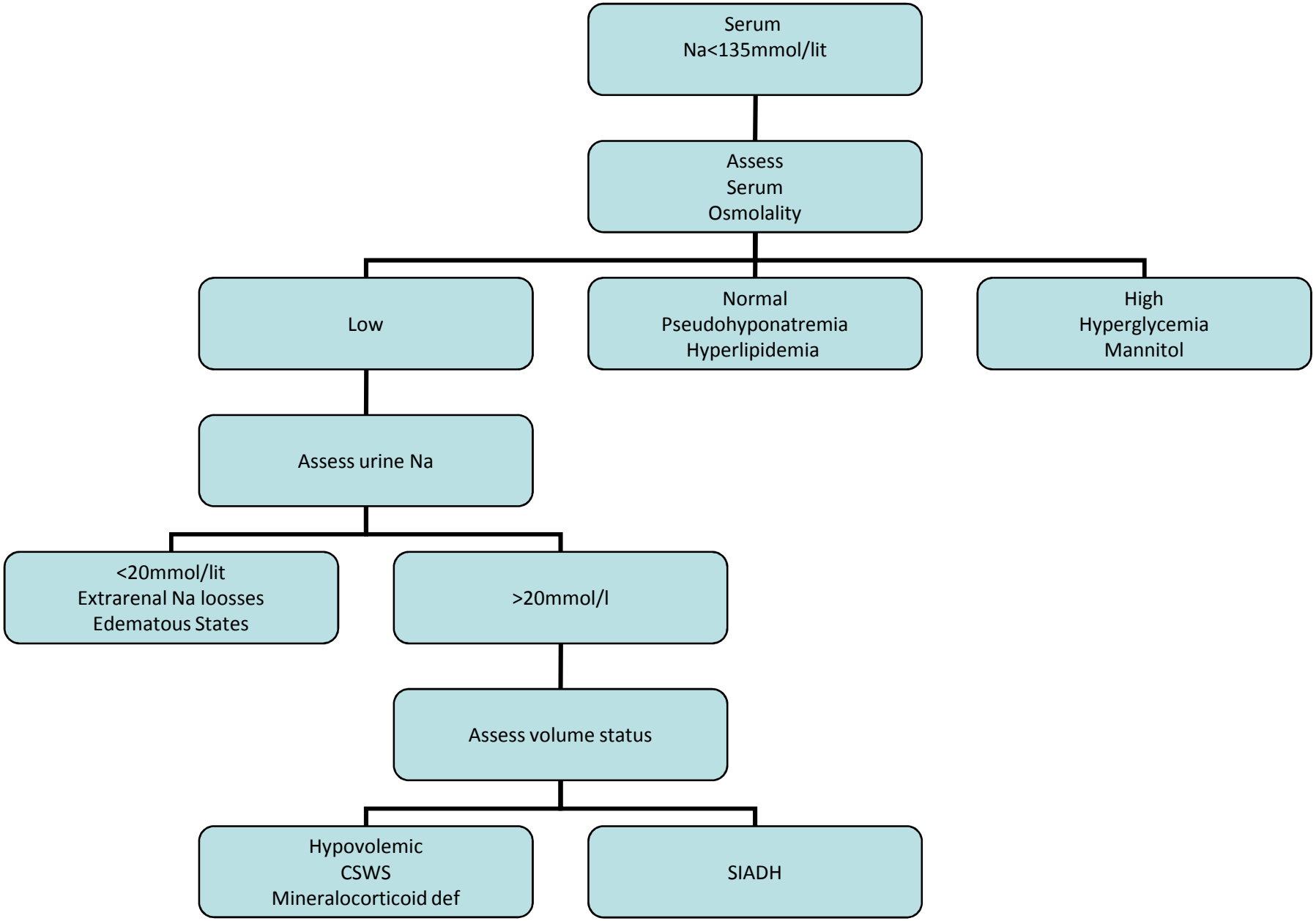
Biochemical-

- Serum Na+ & osmolarity
- Urine vol,S.G, Na+ & Osmolarity
- BUN ,Cr,K+, Uric acid, Albumin,Cortisol

HYPONATREMIA Contd…

- ECG features-
  - Mostly non specific
  - Appears when Na+< 115mmol/l
  - QRS widening,ST elevation,T inversion
  - Bradycardia, Ventricular ectopics also possible
  - At values< 110mmol/l –cardiac arrest may occur.

Algorithm for assesment of Hyponatremia



- Dose of Na^+(meq)=wt(Kg)×(140-Na^+) ×0.6

  3%NS

  Correction rate - 0.6-1mmol/l/hr till Na=125meq/l

  Half the deficit can be administered over 1st 8 hrs, rest over 1-3 days

Correct underlying disorder

Management strategies

- Hypertonic saline - symptomatic
- Fluid restriction /Normal saline - asymptomatic patients
- Salt replacement

Complication of Treatment

- Pontine Myelinolysis - quadriparesis ,ataxia, abnormal extraocular movements. can occur with rapid correction

- Renal Failure, Peripheral edema, pulm edema, heart failure

SIADH

Diagnostic Criteria of SIADH:-*summarised by Harrigan 1996*

- Serum sodium <135mmol/l
- Serum osmolality <280mmol/kg
- Urine sodium>18mmol/l
- Urine osmolality>serum osmolality
- Normal thyroid, adrenal, renal function
- Absence of peripheral edema or dehydration

Clinical features are same of hyponatremia

Specific Treatment of SIADH

- Fluid restriction- 1 l/day. (0.9% saline is usual choice)
- Furosemide
- Lithium - blocks 3,5- AMP & inhibits action of ADH on renal tubule
- Demeclocycline -900-1200 mg in divided doses, takes 3wks for maximal effect, Induces nephrogenic DI.
- Fludrocortisone -requires 1-2 wks &,retains sodium, inhibits thirst.

Cerebral salt wasting syndrome

- Renal loss of sodium due to intracranial disease, leading to hyponatremia & hypovolemia

- Causes:
  - Head injury
  - Brain tumor
  - Stroke
  - Intracerebral hemorrhage
  - Tuberculous meningitis
  - Craniosynostosis repair

Cerebral salt wasting syndrome (CSWS)

- Pathophysiology not fully understood- hypothesis
  - natriuretic response due to SNS overactivity and DA release causes urinary sodium loss
  - release - brain natriuretic peptide, C-type natriuretic peptide or an oubain like peptide, by the injured brain

- CSWS usually appears in the first week after brain injury and spontaneously resolves in 2-4 weeks

Specific Treatment of CSWS

- Fluid & Sodium Resuscitation -0.9% saline used
- acute symptomatic -hyponatremia 3% NS
- Oral fludrocortisone - 0.1mg-0.4mg to limit –ve sodium balance in pts refractory to salt & fluid therapy

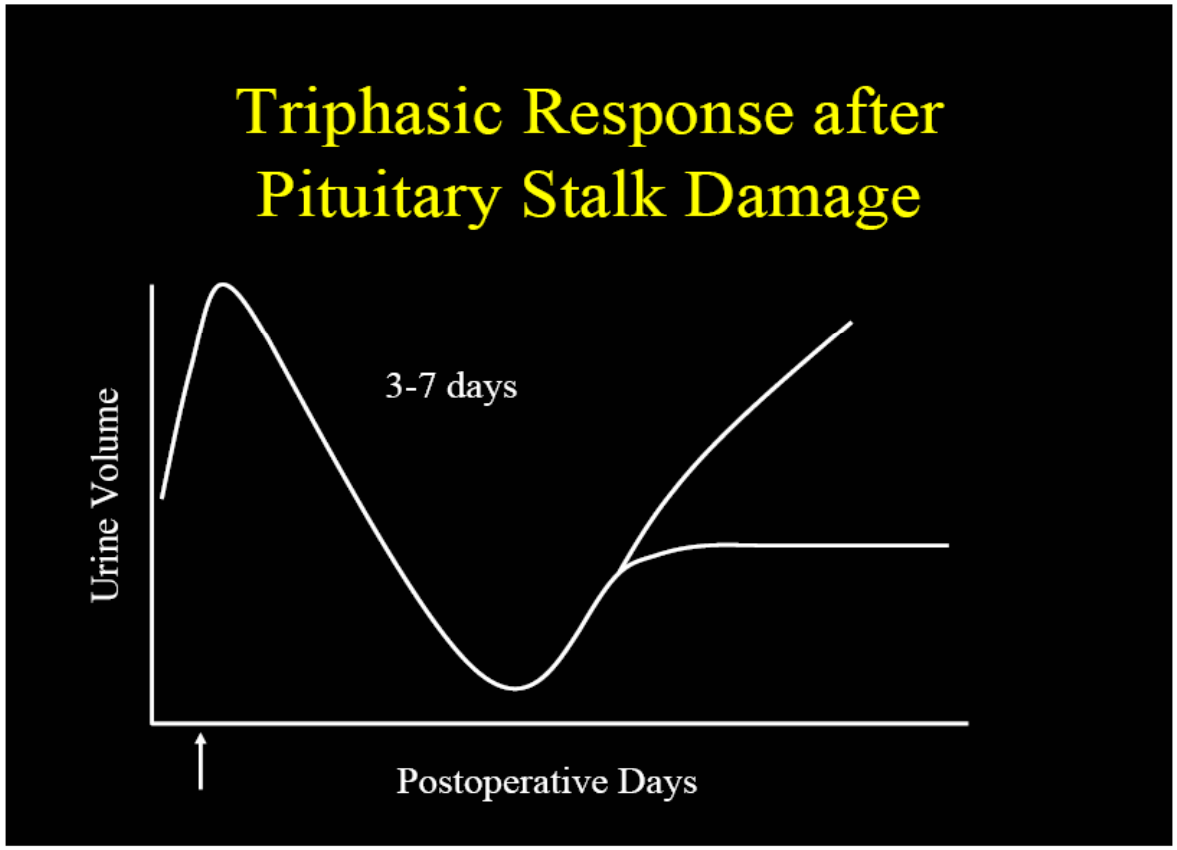
Aneurysmal SAH

- Abnormal sodium levels seen in acute period(4-10days)
- Incidence is 29%-43%
- *Sayama et al* Neurolog res2000; 22:151-55, found
  A.Com-51%-a/w hyponatremia
  MCA-18% - a/w hyponatremia
- *Moringa* et al -84% with hyponatremia had symptomatic vasospasm
- *Hasan et al Ann neurol 1990;27:106-180* didn't find difference in mortality rates
- Possible cause of hyponatremia is release of BNP

Transsphenoidal surgery for Pituitary Tumors

- DI is Common
- CSW may commonly co-exist *Albanese et al , andrews et al Neurosurg1986;18:469-471*
- CSW is thought to occur secondarily to release of BNP. After pituitary adenoma resection.
- Triple response
  - -initial 4-8 day period of DI
  - -excessive release of ADH for 14 days
  - -Permanent DI

Triple response after pituitary surgery



Surgery for Craniosynostosis

- Frequently occurs, but usually asymptomatic & transient

- Etiology not conclusive

  -could be SIADH a/w large fluid shifts

  CSWS has also been reported by

Kappy

Plastic reconstr surgery 2001;108:1501-1508

Hypernatremia

- Defined as serum Na >145mmol/l
- Clinical variables:
  1. Body weight
  2. Peripheral oedema
  3. CVP
  4. Serum sodium/ Urine spot sodium

Major causes of hypernatremia

CAUSES

- Impaired Thirst
- Solute (osmotic) diuresis
- Excessive water loss
  - Renal
  - Extrarenal
- Combined disorders

MECHANISMS

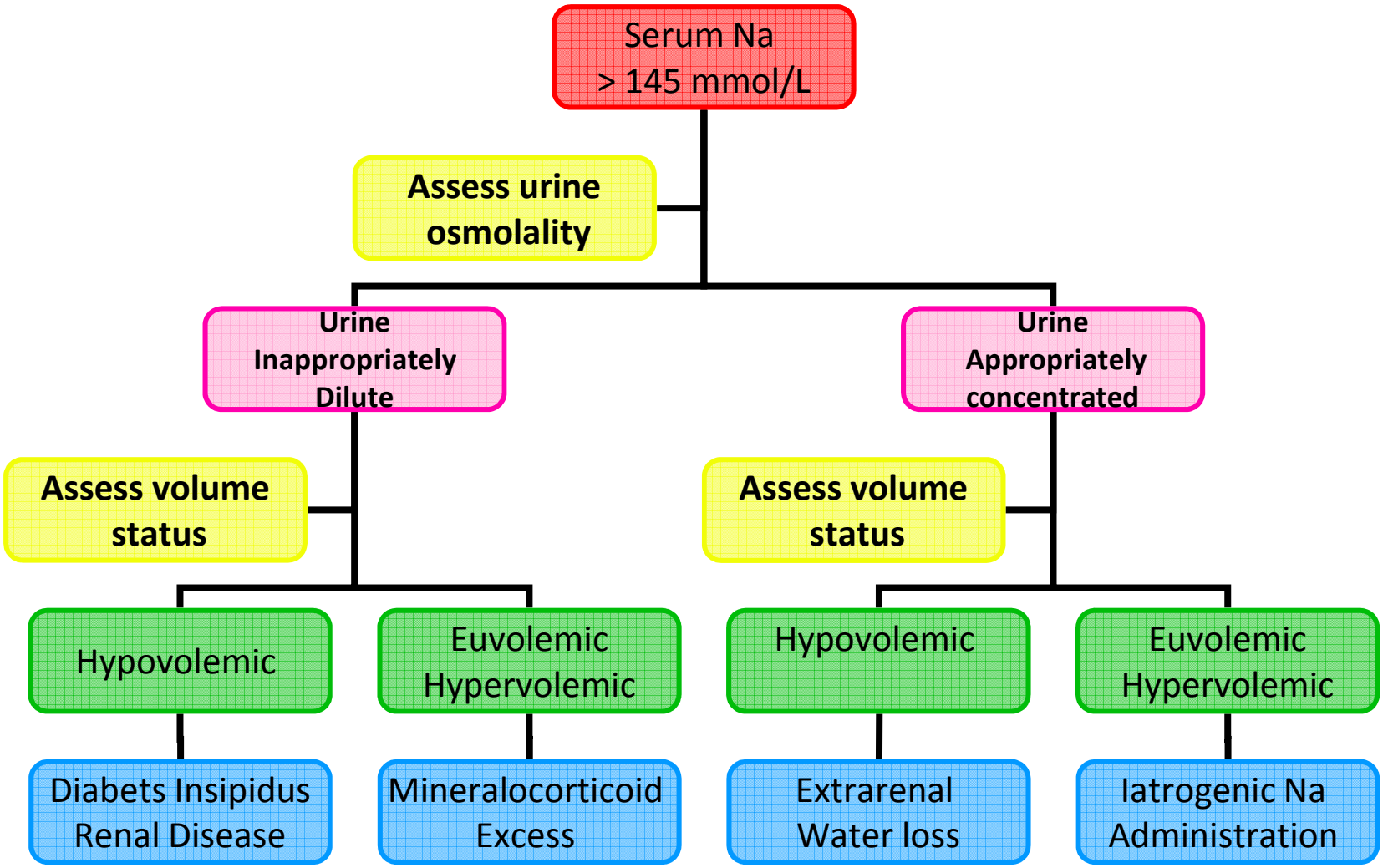
- Coma,
  Essential Hypernatremia
- Mannitol, DKA, Non ketotic hyperosmolar coma
- Pituitary DI,
  Nephrogenic DI
- Sweating
- Coma + Hypertonic nasogastric feeding

Hypernatremia

Most s/s of hypernatrmia are neurological

- Altered mental status
- Weakness
- Neuromuscular irritability
- Focal neurological deficit
- Occasional coma/seizures

Algorithim for assessment of Hypernatremia



Management of Hypernatremia

- Goals
  - Stop loss of water by T/t of cause
  - Correct water deficit
- Water deficit can be calculated as

  $(S\ Na^{+}-140/140)x\ TBW.$

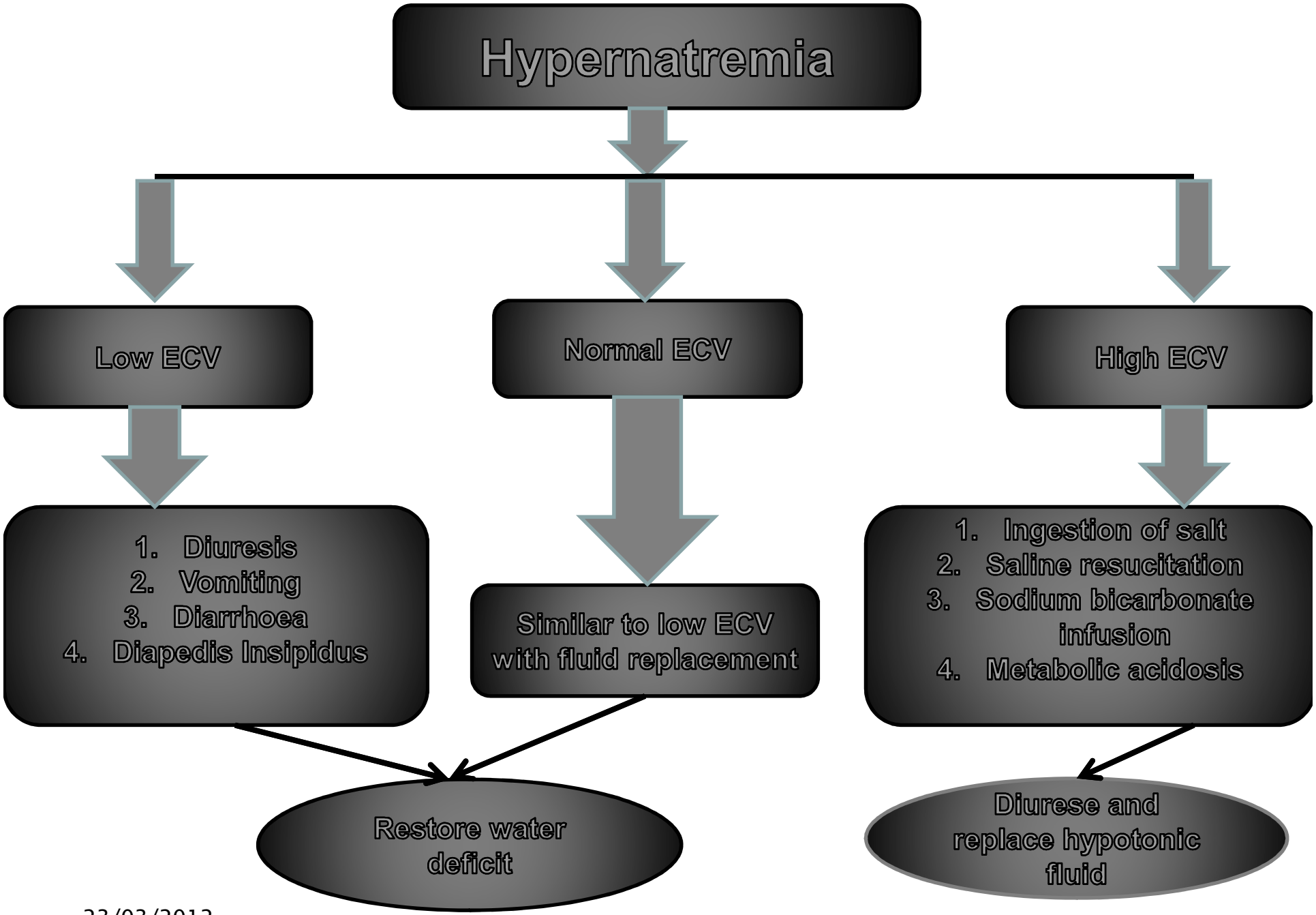
  If serum glucose is elevated, then the corrected formula is

  $S\ Na+=S\ Na^{+}+(\ S\ glu-90)/36$

  As in hyponatremia, rapid correction of hypernatremia is also dangerous

Management of Hypernatremia

- Water deficit to be corrected over 48-72 hrs
- No more than 0.5mmol/l/hr &12mmol/l/day correction should be done
- Safest method of correction is water by mouth or NG tube.
- If patient cannot take orally, 5% Dextrose can be given intravenously


Hypernatremia
Low ECV
Normal ECV
High ECV
1. Diuresis
2. Vomiting
3. Diarrhoea
4. Diapedis Insipidus
Similar to low ECV with fluid replacement
1. Ingestion of salt
2. Saline resucitation
3. Sodium bicarbonate infusion
4. Metabolic acidosis
Restore water deficit
Diurese and replace hypotonic fluid

DIABETUS INSIPIDUS

- Central DI
- Nephrogenic DI

Incidence of DI in neurosurgical unit has been reported as 3.7%. *Wong MF et al Ann Acad Med Singapore 1998*

1/3rd in SAH and TBI

1/6th in pituitary surgery and intracerebral hemorrhage

CENTRAL DI:-

- It is a failure of release of ADH from Hypothalamo-pituitary axis
- Characterised by inability to concentrate urine ,thus passage of large amount of dilute urine
- Rise in Plasma osmolality & progressive dehydration
- Particularly seen after pituitary surgery,TBI, A Com Art aneurysmal SAH, & in brain death patients

**Compromise of Hypothalamic centers or the supra optic tract above the median eminence:-permanent DI*

Damage below median eminence or removal of posterior lobe of pituitary:- transient DI

Etiology of Central DI

- Acquired
  - Head trauma
  - Post-pituitary surgery
  - Neoplasms
  - Granulomas
  - Infections
  - Inflammations
  - Chemical toxins
    - Tetrodoxins,Snake venoms
  - Vascular
  - Idiopathic
- Congenital
  - Midline craniofacial anomalies
  - Holoprosencephaly
  - Ectopia of Pituitary
- Genetic
  - Autosomal dominant
  - Autosomal Recessive
  - X-linked recessive
  - Deletion chromosome7q

ETIOLOGY OF NEPHROGENIC DI

Acquired

- Drugs
  - Lithium
  - Demeclocycline
  - Methoxyflurane
  - Amphotericin B
  - Aminoglycoside
  - Cisplatin
  - Rifampicin
  - Foscarnet
- Metabolic
  - Hypercalcemia
  - Hypokalemia
- Obstruction
- Vascular
  - SCD
  - ATN
- Granulomas
- Neoplasm
- Infiltrations
- Pregnancy
- Idiopathic

Genetic

X-linked recessive(**ADH receptor** V2).

Autosomal Recessive (Aquaporin-2 gene)

Diagnosis of postoperative diabetes insipidus

Rule out osmotic diuresis or fluid overload

Clinical signs and symptoms

Polyuria, abrupt high volumes (4 L/day–18 L/day)
(within 24–48 hours postoperatively)

Polydipsia, with craving for cold fluids

With/without hypovolemia

Diagnosis of postoperative diabetes insipidus

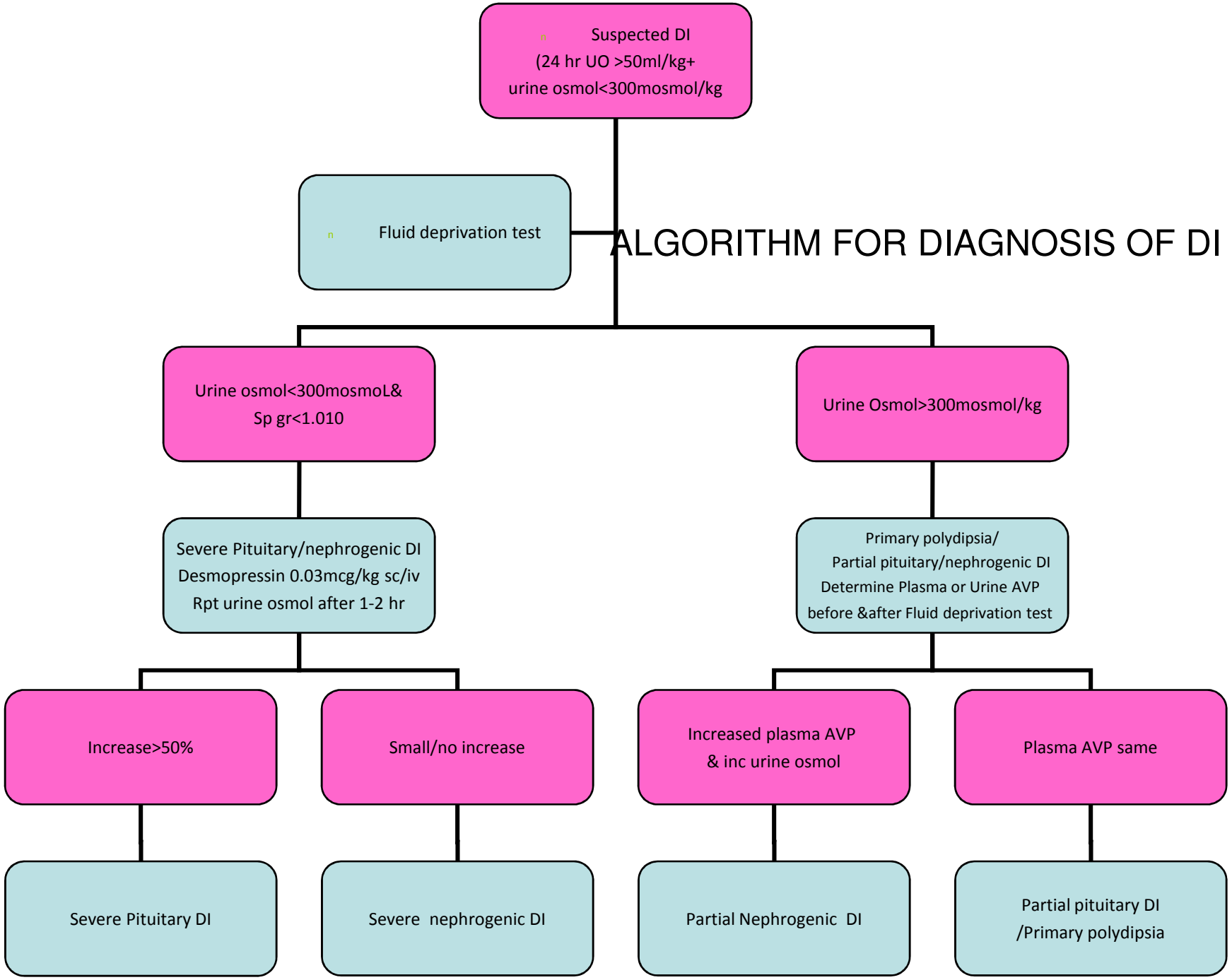
Laboratory data

Dilute urine (specific gravity <1.005 urine osmolality < 200 mOsm/kg H2O)

Normal to increased serum osmolality

Serum [Na+] greater or equal to 145 milliequivalent/L

ALGORITHM FOR DIAGNOSIS OF DI



Treatment of postoperative diabetes insipidus

Expectant monitoring

I/O CHARTING

Urine osmolality or specific gravity every 4 to 6 hours,

Serum [Na+] every 4 to 6 hours

Antidiuretic hormone therapy

Desmopressin 1 mg to 2 mg iv/sc
10mcg-20mcg nasal
Redose - urine output 200 mL to 250 mL/hr for 2 hours
with urine specific gravity < 1.005
urine osmolality < 200 mOsm/kg H2O

Maintenance

drink according to thirst

Supplement hypotonic (D5W to D51/2NSS)

Monitor for resolution of transient DI or triphasic response

Positive daily fluid > 2 L suggests SIADH

Withhold -Antidiuretic hormone therapy

Manage anterior pituitary insufficiency
stress dose -hydrocortisone 100 mg TDS

- Chlorpropramide
  - It acts by potentiating the action of AVP or activation of V_2 raceptors
  - Dose is 125-500mg OD
  - Onset slow, efficacy less,efficacy can be increased by simultaneous use of Thizides.Hypoglycemia is a significant side effect
- Clofibrate & Carbamazepine is also helpful in few patients

- Nephrogenic DI:-
  - T/t of cause & omitting the culprit drug generally cures the disease
  - Thiazide
  - Amiloride (esp in pts. On lithium)
  - Indomethacin
  - Low sodium diet

FLUID DEPRIVATION TEST

- **Indication**
- Evaluation of Diabetes Insipidus

Precautions: Requires close monitoring

- Monitor urine output
- Monitor vital signs
- Monitor weight
  - Do not allow weight loss to exceed >3-5%

CONTD..

- **Technique**
- Fluid restrict patient
  - Mild polyuria (<10 L/day)
    - Start fluid restriction 12 hours before test
  - Severe polyuria (>10 L/day)
    - Start fluid restriction 2 hours before test
- Follow Serum Osmolality to steady state
  - Serum Osmolality should approach 295 mOsm water
  - Measure Serum Osmolality hourly until endpoint:
    - Two values are within 30 mOsm of each other
    - Weight loss exceeds 3-5%
- Administer endogenous ADH
  - Vasopressin 5 units Sc
  - Intranasal DDAVP 10 ug
- Measure Serum Osmolality 1 hour after ADH administered

CONTD..

- **Interpretation**
- Water deprivation effect on urine concentrating ability
  - No response in Nephrogenic DI
  - No response in Central DI
- Exogenous ADH effect on urine concentrating ability
  - Corrects Central DI
  - Does not correct Nephrogenic DI

Principal water-electrolytes disorders

		DI	SIADH	CSWS
Etiology		Reduced secretion of ADH	Excessive release of ADH	Release of brain natriuretic factor
Urine	Output	> 30 ml/kg/h		
	specific gravity	< 1.002		
	Sodium	< 15 mEq/l	> 20 mEq/l	> 50 mEq/l
	Osmolality vs. serum osmolality	Lower	Higher	Higher
Serum	Sodium	Hypernatremia	Hyponatremia	Hyponatremia
	Osmolality	Hyperosmolality	Hypoosmolality	
Intravascular volume		Reduced	Normal or increased	Reduced

Abbreviations: ADH, antidiuretic hormone; CSWS, celebral salt-wasting syndrome; DI, Diabetes insipidus; SIADH, syndrome of inappropriate antidiuretic hormone secretion.

Hypokalemia

- Normal requirement –1meq/kg/day
- Serum potassium < 3.5 mEq/ liter
- Causes: intracellular shift

  potassium depletion

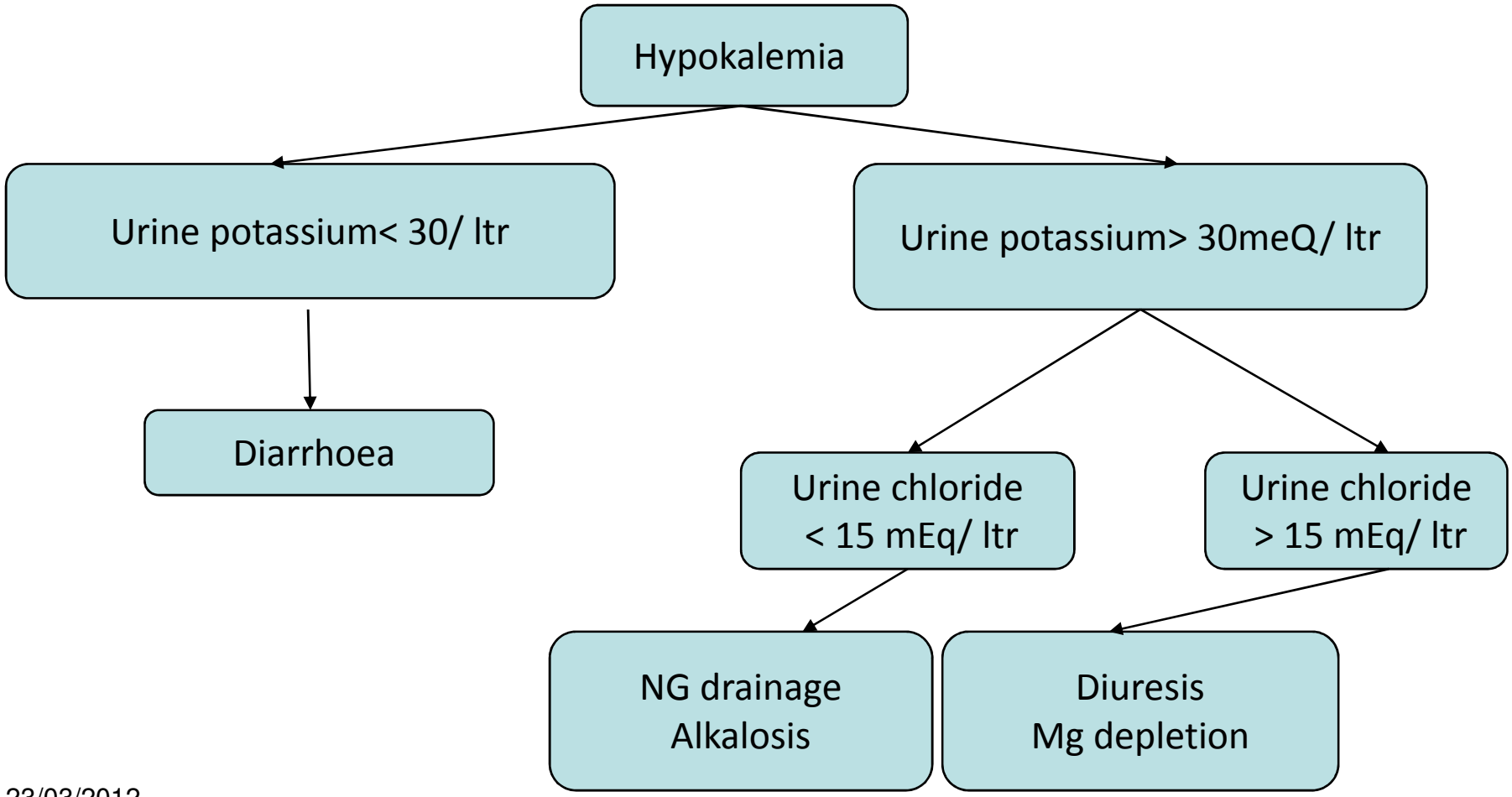
Hypokalemia

- Transcellular shift:
  - beta agonists
  - diuretics
  - alkalosis
  - hypothermia
  - insulin
- Potassium depletion:
  renal losses: diuresis/ failure
- Extra renal losses: diarrhea/ NG drainage

Clinical features hypokalemia

- Muscle weakness and mental status changes< 2.5 mEq/ liter
- ECG changes prominent U waves, flattening and inversion of T waves, prolonged QT interval
- Does not cause arrhythmias on its own but definitely is proarrythmic

Hypokalemia



Management of hypokalemia

- Correct the primary cause

  Potassium deficits in hypokalemia for 70 Kg male

Serum potassium (mEq/ L)	Potassium deficit (mEq/L)	Deficit in % body K
3.0	175	5
2.5	350	10
2.0	470	15
1.5	700	20
1.0	875	25

Management of hypokalemia

Potassium replacement

Iv replacement

Potassium chloride- concentration varies from 1 and 2 meq/ ml.

Extremely hyperosmololar and must be diluted

Infusion rate: add 20 meq to 100 ml isotonic saline

- rates of 20 mEq/ hour
- can go as high as 40 mEq/ hour
- use central line with cardiac monitoring

-If refractory check for magnesium deficiency as it promotes urinary losses

Mild cases :Oral potassium chloride – (1 TSF=20meq/l), 2tsf TDS diluted in liquids

Hyperkalemia

- Serum potassium > 5.5 mEq/ liter
- Most common cause is traumatic hemolysis during venipuncture
- 20% blood sample incidence with elevated potassium
- Source: transcellular shift (urinary K>30)

renal cause (urinary K< 30)

- Trans cellular shift:
  - Acidosis
  - Rhabdomyolysis
  - Drugs: digitalis/ beta antagonists
- Renal causes:
  - Renal failure
  - Adrenal insufficiency
  - Drugs: ACE inhibitors/ B-blockers/ cyclosporine/ digitalis/ diuretics/ heparin/ NSAIDS/ septran/ muscle relaxants
- Blood transfusions

Clinical features hyperkalemia

- Slowing of electrical conduction of the heart
- Usually starts when K level reaches 6.0 mEq/ L
- Tall tented T waves
- Decrease in P wave amplitude
- PR lengthening
- QRS prolongation with eventual asystole

Management

- Guided by serum potassium and ECG manifestation

1. Severe cases -Give 10 ml- 10% calcium gluconate over 5 minutes

    Repeat second dose if necessary

    No role for third dose

    Action lasts for 20 minutes

2. Insulin dextrose: 10 U regular insulin in 500 ml 20% dextrose to infuse over 1 hour- decreases K by an average of 1 mEq/ L

3. Loop diuretics

4. Exchange resins

5. If refractory - hemodialysis

Metabolic acidosis

High Anion Gap

- Renal failure
- toxins
- ketoacidosis

Normal anion gap

- (hyperchloremic)
- Hyperkalemia
- obstructive uropathy
- diarrhea
- renal tubular acidosis
- Some medications

Clinical features

- Headache
- Drowsiness
- Nausea/ vomiting/ diarrhea
- Kussmaul's respirations
- Fruity-smelling breathe
- Hyperkalemia
- Hypotension
- Bradycardia
- GI distention
- pH low (< 7.35)
- HCO3 low (< 22)

Management:

- Fluid resuscitation
- Correct underlying disorder
- Sodium bicarbonate only if pH< 7.20
- Method: 0.5 x body weight x base deficit

  correct half as slow infusion over few minutes

  other half to be repeated over 8 hours

  repeat ABG values

  stop when ph 7.20

Metabolic alkalosis

- Etiology:

1. Vomiting
2. Diuretics
3. Volume depletion
4. Hypokalemia
5. Organic anions like lactate
6. Chronic CO_2 retention

Metabolic alkalosis

Clinical features

- Shallow breathing
- Nausea/vomiting/diarrhea
- Confusion
- Numbness / tingling
- Hypocalcemia
- Hypokalemia
- pH high (> 7.45)
- HCO3 high (>26) cal features:

Metabolic alkalosis

- Management: saline infusion (0.2*body weight* chloride deficit)
- In resistant cases give 0.1N HCl (0.5* body weight* base excess)
- Acetazolamide

Metabolic alkalosis

- Management: saline infusion (0.2*body weight* chloride deficit)
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Respiratory acidosis and alkalosis

- Treatment of underlying cause
- Correction of oxygenation
- Sedation, reassurance and CO2 rebreathing for alkalosis
- Ventilatory support and chest physiotherapy

- THANK YOU