MODULAR COURSE

MARCH 2021

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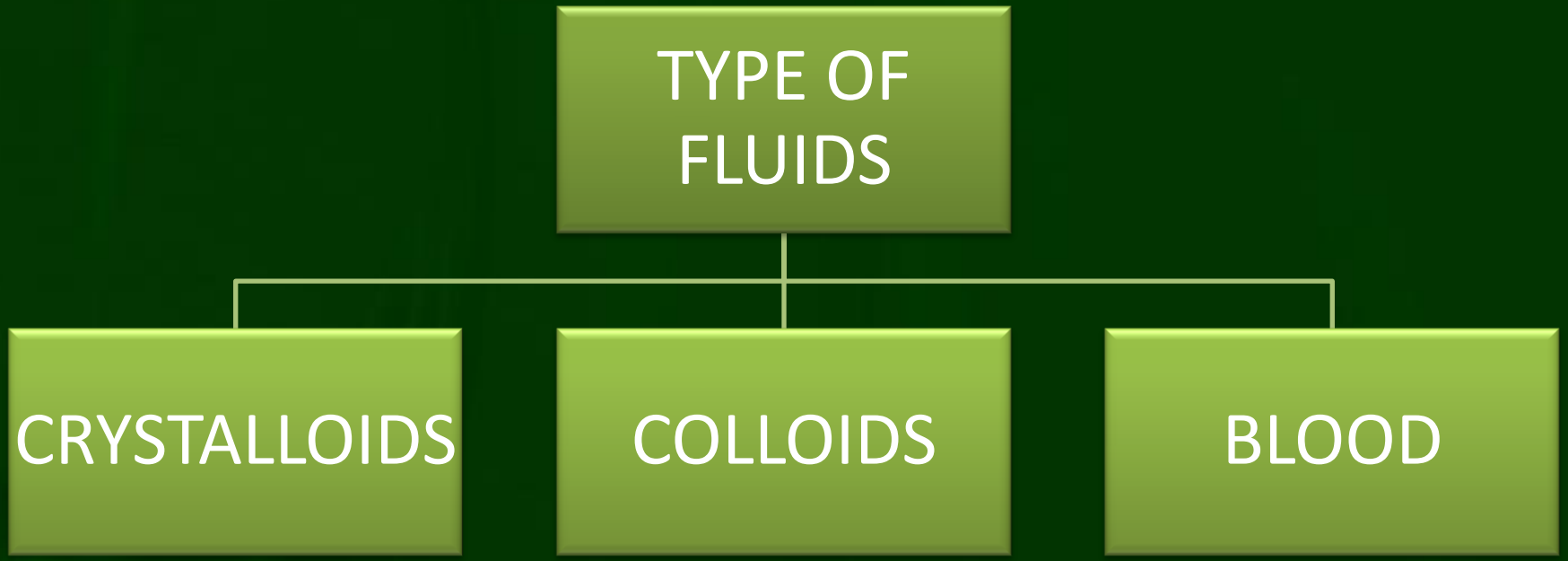
MS; FRCS (Eng; Edin {ad eundem})

FLUID MANAGEMENT

PER-OPERATIVE	CRITICAL CARE
• PRE-OP • INTRA-OP • POST-OP	• RESCUE • OPTIMIZATION • STABILIZATION • DE-ESCALATION

TYPE OF FLUIDS



PRE-OP MANAGEMENT ERAS PROTOCOL

- Identification and optimization of comorbid conditions
- Prehabilitation, if necessary
- Patient and family education and discharge planning
- Avoidance of prolonged preoperative fasting
- Pain management planning (procedure-specific multimodal opioid-sparing pain prophylactic agents administered at least two hours before surgery)
  - Oral acetaminophen 1 g
  - Oral cyclooxygenase (COX)-2 specific inhibitor
  - Oral gabapentin in selected patients undergoing procedures with a high risk for persistent postoperative pain
- For selected procedures, thromboembolism prophylaxis with subcutaneous heparin 5000 units administered 30 to 60 minutes before surgery

INTRA-OP MANAGEMENT ERAS PROTOCOL

- Use of a minimally invasive surgical approach, when feasible
- Antibiotic prophylaxis administered 30 to 60 minutes before the surgical incision
- Use of short-acting anesthetic agents (inhalation and/or IV agents) during induction and maintenance of general anesthesia
- Avoidance of fluid overload
- Lung protective mechanical ventilation
- Maintenance of normothermia
- Glycemic control
- Multimodal antiemetic prophylaxis
- Procedure-specific multimodal opioid-sparing pain prophylaxis

POST-OP MANAGEMENT ERAS PROTOCOL

- Rescue therapy for PONV.
  - IV dexamethasone 8 to 10 mg administered after induction of anesthesia, as well as
  - a 5-hydroxytryptamine type 3 (5-HT3) antagonist such as IV ondansetron 4 mg administered at the end of surgical procedure.
  - an additional antiemetic agent such as preoperative transdermal scopolamine or intraoperative IV haloperidol 0.5 to 1 mg administered shortly after induction of anesthesia for high risk patients.
- Procedure-specific multimodal opioid-sparing pain management.
- Resumption of oral feeding as soon as feasible.
- Early postoperative mobilization and physical therapy.

INTRA-OPERATIVE FLUID MANAGEMENT

- Vital to achieve optimal outcomes after surgery. Causes of fluid loss and sequestration may be:-
- Pre-operative causes.
- Anaesthesia related.
- Surgery related.

PRE-OPERATIVE CAUSES

- Preoperative fasting adds little to dehydration but ERAS protocols have clearly defined the benefit of avoiding prolonged fasting and dehydration.
- Mechanical bowel prep.
- Bowel obstruction/ Pancreatitis – third space fluid sequestration.
- Ongoing bleeding.

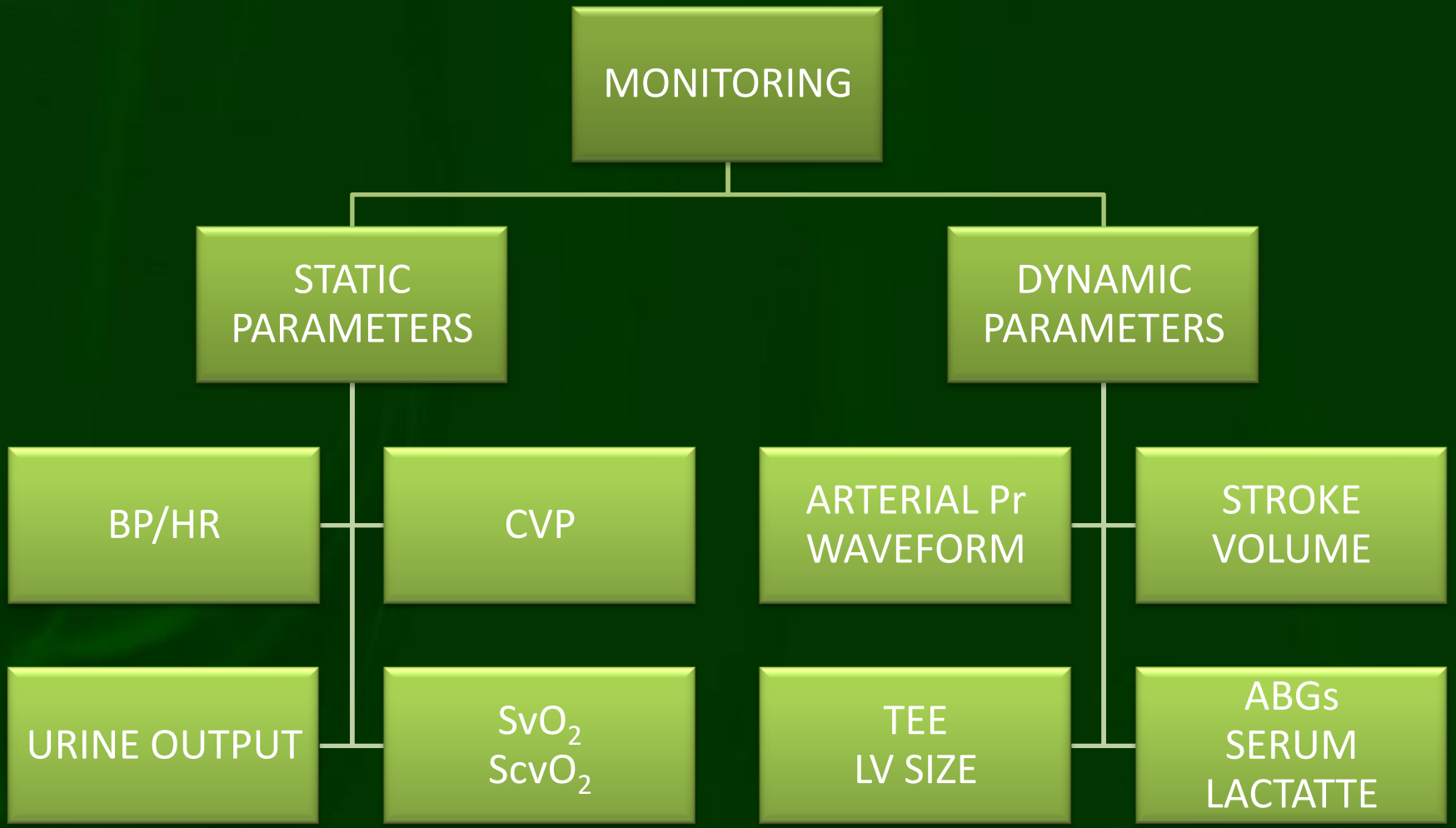
ANAESTHESIA RELATED FACTORS

- Most anaesthetic and adjuvant drugs cause dose-dependent vasodilatation and myocardial depression that may lead to hypotension.
- Sympathetic blockade increases venous capacity and dilatation of arteriolar resistance vessels – leading to peripheral pooling – may require vasopressors.
- Persistent hypotension despite corrective procedures may require vasopressors.

SURGERY RELATED FACTORS

- Haemorrhage.
- Coagulopathy (haemodilution/hypothermia).
- Abdominal tamponade
  - Laparoscopy
  - Compression of main veins.
- Prolonged operation time – loss by evaporation, bowel oedema and sequestration of fluids. Less in MIS.
- Delayed progression to oral fluids.

MONITORING FLUID REPLACEMENT DURING SURGERY



SUGGESTED PROTOCOL

- Minimally invasive surgery – 1-2 L of Balanced Electrolyte Solution during surgery (30 mins to 2 hours)
- Major Invasive Surgery

1. Restrictive zero-balance strategy (invasive surgery where blood loss< 500ml).
2. Goal-directed Strategy (Invasive Surgery where blood loss > 500ml).

Restrictive zero-balance strategy

- Per-op patient receives balanced crystalloids -1 to 3 mL/kg per hour.
- Blood loss replaced with crystalloids:blood at the rate 1.5:1.0 (Colloid:Blood at the rate 1:1).
- NO preloading of crystalloids prior to neuraxial block.
- Deep anaesthesia avoided, if it can’t vasopressors like phenyephrine or ephedrine for hypotension.
- Exceed fluid schedule ONLY in the presence of hypovolaemia.
- HOWEVER, URINE OUTPUT MUST BE KEPT IN MIND (Higher incidence of AKI).

Goal-directed Strategy

- INVASIVE MONITORING NEEDED.
- Intra-arterial waveform tracings –automated measurement of

1. Pulse pressure variations (PPV).
2. Stroke volume variations (SVV)
3. Visually estimated or manually calculated –
   I. PPV
   II. Systolic pressure variations (SPV).

Goal-directed Strategy

- INVASIVE MONITORING NEEDED.
- Commercially available device which provides an automatic calculation of
  - PPV.
  - SVV.
  - SPV.

Goal-directed Strategy

- Assessment of Fluids
- PPV or SPV
  - If respiratory variations in PPV or SPV > 10% patient presumed to be fluid responsive and fluid boluses of balance crystalloid solution 250ml) given.
  - Once change is <10% fluid is stopped.
- SV
  - SV value <10% fluid boluses are stopped.
- TEE
  - LV cavity size estimated, fluid stopped when size is normal.

POST-OPERATIVE FLUID MANAGEMENT

CASE CAPSULE

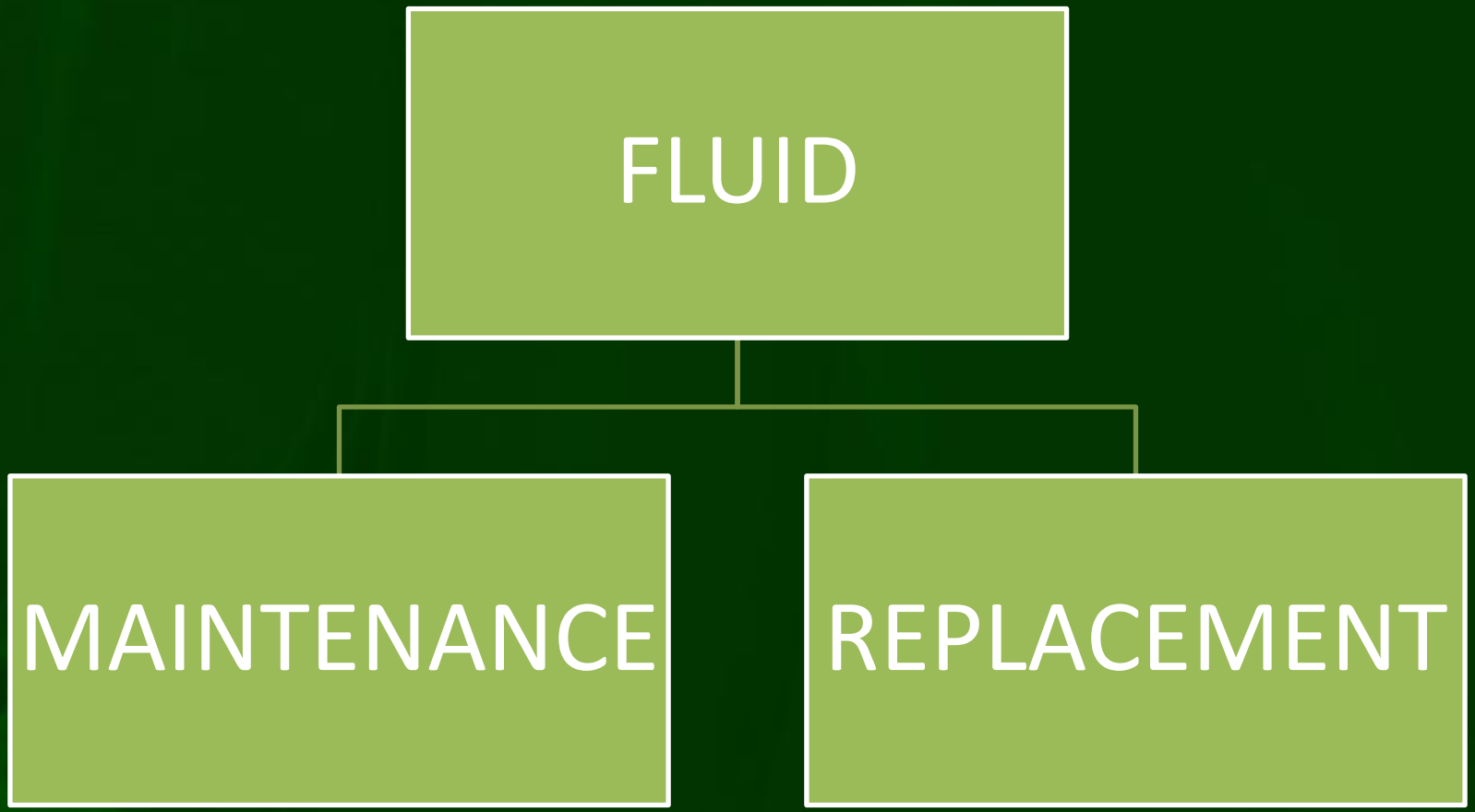
- A 37 year old male patient, weight 63 kgs, no co-morbidities, undergoes a lower radical gastrectomy for a carcinoma of the stomach 3 days ago.
- His Pulse is 84/min, BP 110/70 mmHg, Resp. 28/min, minimal drainage of 30ml of serosanguinous fluid, urine output 1270ml /last 24 hours, I/O balance 1050ml+ve, RT suction 200 ml bilious fluid.
- His Hb is 10.1gm/dL, PCV 30; Urea 43; Creat 1.5; Na^+ 132 mEq/L; K^+ 3.4 mEq/L.

CASE CAPSULE

- The patient is getting the following IV medications
- IV; Augmentin 1.2 gm TDS; Paracetamol 1gm TDS
- Calculate the amount and type of fluid you wish to give him over the next 24 hours.

ANSWERS READY?

Calculating fluid replacement



What is the rule for calculating maintenance fluids for 24 hours?

- 100/50/20 rule
  - 100mL/kg for the first 10 kg
  - 50mL/kg for the next 10 kg
  - 20mL/kg for every kg over 20

4/2/1 rule
4mls/kg/hr first 10 Kg
= 96ml/kg
2mls/kg/hr 2nd 10Kg
= 48ml/kg
1ml/kg/hr last Kgs
= 24ml/kg

Replacement of losses

- Pre-operative or pre-admission
  - Ongoing losses
  - Nasogastric aspirate
  - Vomit, diarrhoea
  - Stoma, drains, fistula etc
- Pre-operative assessment of electrolytes a must.

Composition of Different Intestinal Juices

Secretions	Na	K	Cl	HCO_3	Volume
Saliva	2-10	20-30	8-18	30	0.5-2L
Gastric	9-116	0-30	8-154	0-15	0.1-4L
Duodenum	140	5	80		0.1-2L
Ileum	80-150	2-8	45-137	30	0.1-0.9L
Colon	60	30	40		
Pancreas	115-185	3-7	55-95	115	0.1-0.8L
Bile	130-160	3-12	90-180	35	0.05-0.8L
Stool	35	70	20		
Diarrhoea	30-140	30-70	73	20-80	
Mixed G A	120	10	100		

Insensible losses

- Faeces approximately 100 ml/ day
- Lungs approximately 400 ml/ day
- Skin approximately 600 ml/ day

Daily requirement of Electrolytes

- Sodium: 1-2 mEq/kg/d
- Potassium: 0.5-1 mEq/kg/d
- Calcium: 800 - 1200 mg/d
- Magnesium: 300 - 400 mg/d
- Phosphorus: 800 - 1200 mg/**d**

Daily requirements of major electrolytes
{Easy reckoner -Rule of 1,2 & 3s}

- Potassium 1 mEq /kg/day
- Chloride 1 mEq /kg/ day
- Sodium 1-2 mEq /kg/ day
- Calcium 1 g/ day
- Magnesium 300 mEq day

COMPOSITION OF IV FLUIDS

TYPE OF FLUID	OSMO LALITY	TONICITY	Na^+	Cl^{--}	K^+	Mg^{2+}	Ca^{2+}	Buffer
Plasma	**288**	Reference	**140**	**103**	**4.5**	**1.25**	**2.5**	**24**
0.9% Normal saline	**308**	Isotonic	**154**	**154**	**0**	**0**	**0**	**0**
Ringer Lactate	**279**	Hypotonic	**130**	**111**	**4.0**	**0**	**2.7** 55.89 mg/L	**29**
Plasmalyte	**295**	Isotonic	**140**	**98**	**5.0**	**1.5**	**0**	**50** +
Sterofundin	**309**	Isotonic	**140**	**127**	**4.0**	**1.0**	**2.5** 51 mg/L	**29**
5% Glucose	**278**	Hypotonic	**0**	**0**	**0**	**0**	**0**	**0**

All in mmol/L except Osmolality inMOsm/kg

Buffers as follows:- Plasma – bicarbonates, haemoglobin

RL – lactate

Plasmalyte, Sterofundin – acetate

Plasmalyte - gluconate

Hoorn EJ. Intravenous fluids: balancing solutions. Journal of Nephrology. 2017;30(4):485-492.

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Ringer Lactate	**279**	Hypotonic	**130**	**111**	**4.0**	**0**	**2.7**	**29**
Isolyte M	**390**	Hypertonic	**36**	**49**	**35**	**Phosphate 15**		**20**

All in mmol/L except Osmolality inMOsm/kg

Buffers as follows:- Plasma – bicarbonates, haemoglobin

RL – lactate

Isolyte M– acetate

CASE CAPSULE

- Total fluid
- 1000ml + 500ml =1500ml; 20 x 43kgs = 860;
- 1500ml + 860ml = 2360ml (Insensible loss + requirement for metabolism)
- Sensible loss; Urine 1270ml + RT suction 200ml; Drain 30 ml = Total 1500ml
- Total fluid 3860ml/day (Minus Amount of IV medications)
- So minus 600ml = 3260 ml/Day
- Total Na+ = 63-126mEq
- Total K+ = 63mEq
- Total Cl- = 63mEq

CASE CAPSULE

TYPE of FLUID	FLUID ml	Na^+	Total Na^+	K^+	Total K^+

CASE CAPSULE

TYPE of FLUID	FLUID ml	Na^+ mEq/l	Total Na^+ mEq/l	K mEq/l $^+$	Total K^+ mEq/l
5D	500	0	113	0	55
5D	500	0		0	
ISOLYTE M	500	18		17.5	
5D	500	0		0	
NS + KCl 20ml	500 10	77		20	
ISOLYTE M	500	18		17.5	

PROBLEM OF Cl^-

- Hyperchloremic metabolic acidosis – Large volume 0.9% sodium chloride resuscitation generates a hyperchloremic acidosis and renal vasoconstriction, both of which contribute to unpredictable water retention and electrolyte derangement .
- In many cases, acidosis can be avoided with the use of a solution containing less chloride than 0.9% sodium chloride, such as LR or Plasma-Lyte.

CASE CAPSULE

TYPE of FLUID	FLUID ml	Na^+ mEq/l	Total Na^+ mEq/l	K^+ mEq/l	Total K^+ mEq/l	Cl^- mEq/l	Total Cl^- mEq/l
5D	500	0	83	0	59.5	0	120
5D	500	0		0		0	
RL + KCl 20ml	500	65		2 20		55.5 +20	
5D	500	0		0		0	
5D + KCl 20ml	500 10	0		0 20		0 + 20	
ISOLYTE M	500	18		17.5		24.5	

WHAT FLUID?

TYPE OF FLUIDS COMPARED

- COCHRANE ANALYSIS
- 69 studies (65 RCTs, 4 quasi-RCTs) with 30,020 participants.
- COLLOIDS STUDIED
  - Twenty-eight studied starch solutions (28),
  - Twenty dextrans (20),
  - Seven gelatins (7), and
  - Twenty two albumin or fresh frozen plasma (FFP) (22);
  - each type of colloid was compared to
- CRYSTALLOIDS.

- Colloids versus crystalloids probably makes little or no difference to mortality.
- Starches probably slightly increase the need for blood transfusion and Renal Replacement Therapy {RRT} (moderate-certainty evidence), and
- Albumin or FFP may make little or no difference to the need for renal replacement therapy (low-certainty evidence).

Lewis SR et al. Colloids versus crystalloids for fluid resuscitation in critically ill people. Cochrane Database of Systematic Reviews 2018, Issue 8.

TYPES OF CRYSTALLOIDS
A Bit of History

Dr. Hartog Jacob Hamburger, a Dutch physiologist developed the so-called "Normal Saline" in 1900

Dr. Alexis Hartmann, an American Paediatrician, modified Ringer's solution by adding the buffer lactate in 1932

TYPES OF CRYSTALLOIDS COMPARED

- Excess exogenous chloride administration has been shown to induce
  - Renal artery vasoconstriction,
  - AKI,
  - Hyperchloremic metabolic acidosis,
  - Gastrointestinal dysfunction and
  - Secretion of inflammatory cytokines
- A commonly cited concern about the use of balanced salt solutions is the risk for hyperkalemia
- However, comparative evidence has largely invalidated this suspicion and indicated -
- that the metabolic acidosis which ensues after large-volume 0.9% NaCl administration may instead trigger extracellular potassium shifts and consequent hyperkalemia

Fluid Management for Critically Ill Patients: A Review of the Current State of Fluid Therapy in the Intensive Care Unit Erin Frazee; Kianoush Kashani. Kidney Dis 2016;2:64–71

TRAUMA & SHOCK

EARLY DAYS

- Sir David Paton Cuthbertson CBE, FRSE served on the Royal Scot Fusiliers (Musketeers) during the First World War.
- Re-joined his truncated education and passed out as a doctor from the University of Glasgow.

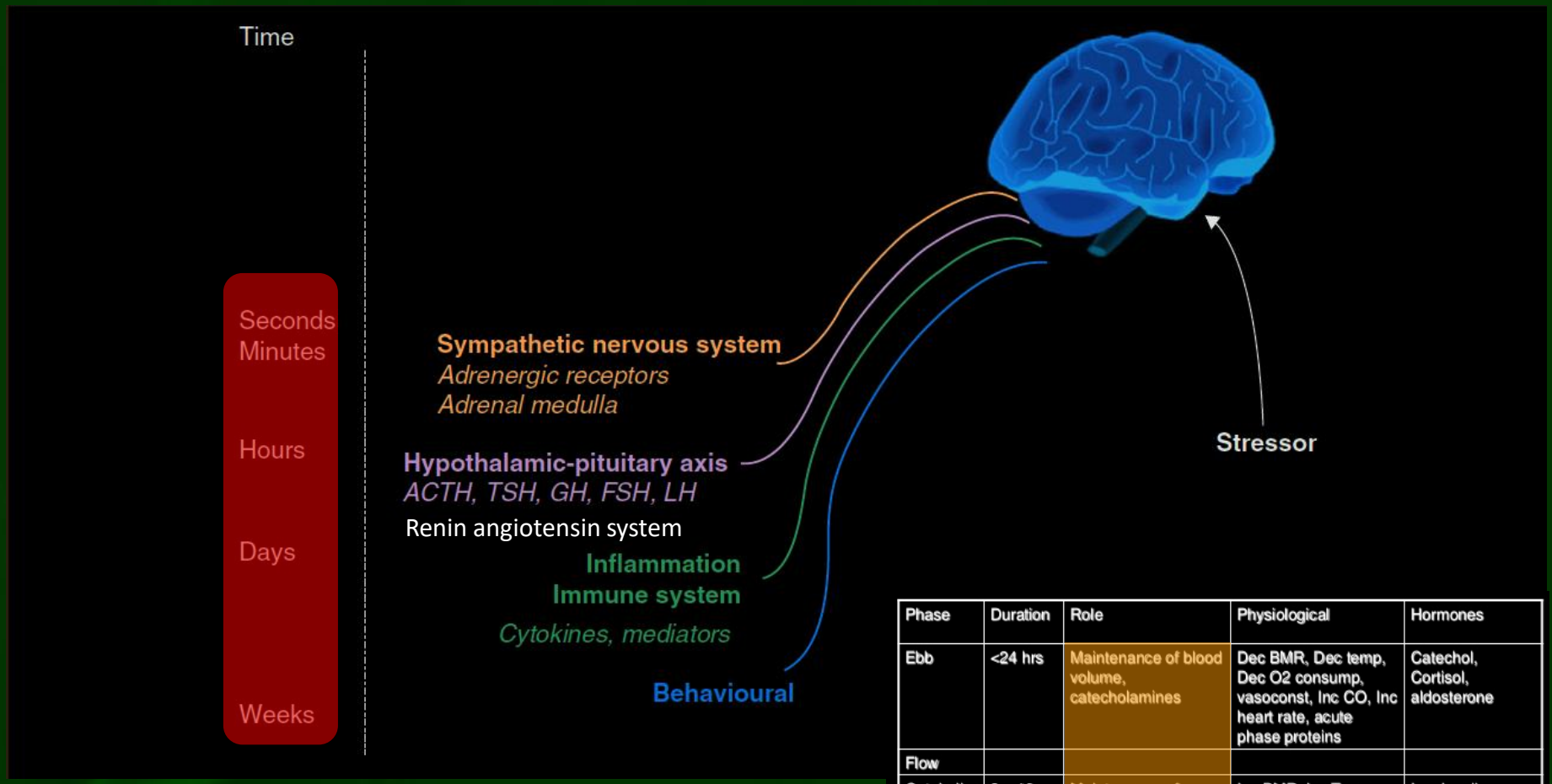
DIFFERENT PHASES following Trauma

Ebb and Flow

Phase	Duration	Role	Physiological	Hormones
Ebb	<24 hrs	Maintenance of blood volume, catecholamines	Dec BMR, Dec temp, Dec O2 consump, vasoconst, Inc CO, Inc heart rate, acute phase proteins	Catechol, Cortisol, aldosterone
Flow				
Catabolic	3 – 10 days	Maintenance of energy	Inc BMR, inc Temp, inc O2 consump, -ve N2 balance	Inc. Insulin, Glucagon, Cortisol, Catechol but insulin resistance
Anabolic (MOORE)	10 – 60 days	Replacement of lost tissue	+ve Nitrogen balance	Growth hormone, IGF

https://www.slideshare.net/sudarsanagarwal/metabolic-response-to-injury

TIME TO RESPONSE



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Ebb	<24 hrs	Maintenance of blood volume, catecholamines	Dec BMR, Dec temp, Dec O2 consump, vasoconst, Inc CO, Inc heart rate, acute phase proteins	Catechol, Cortisol, aldosterone
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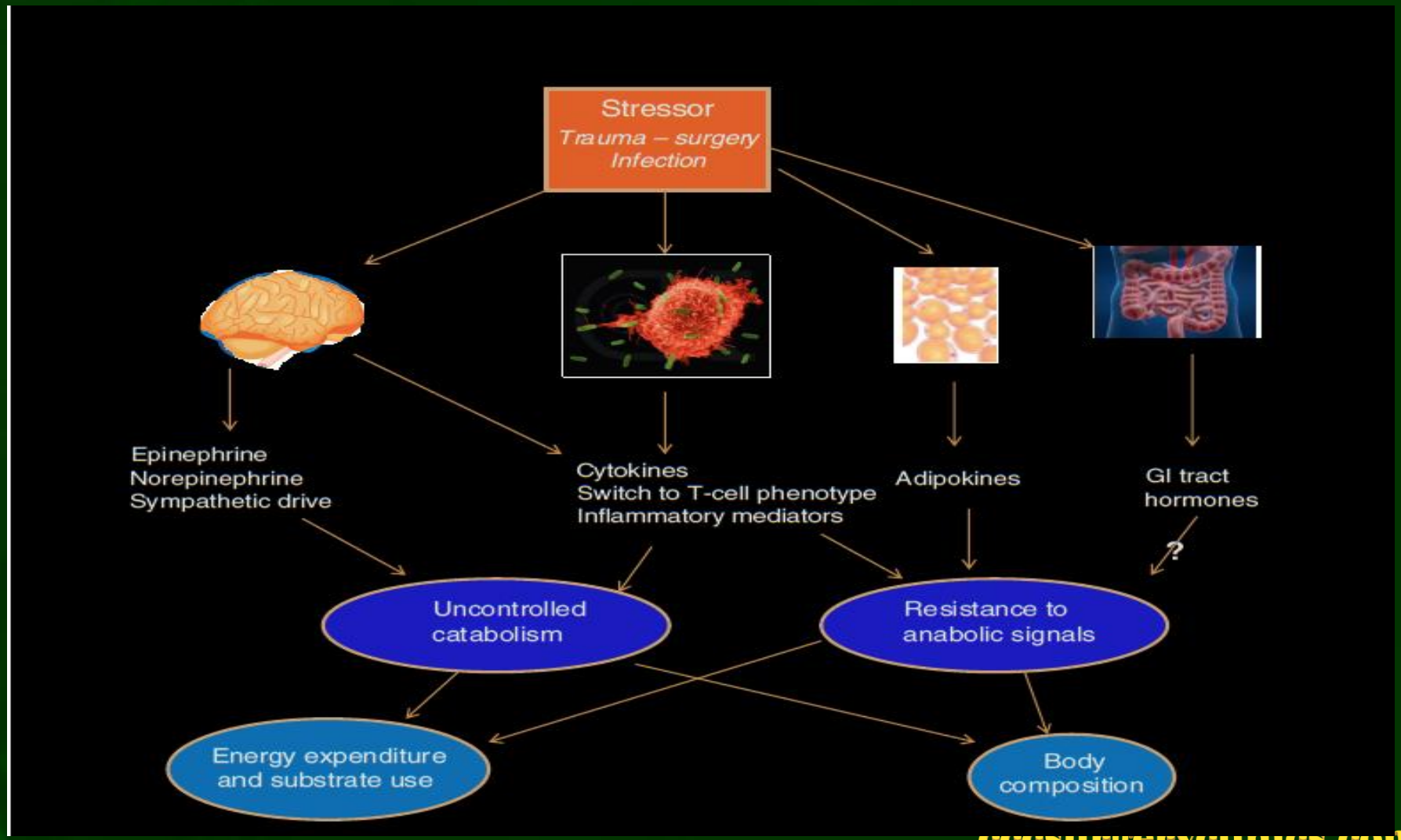
PATHOPHYSIOLOGY OF STRESS

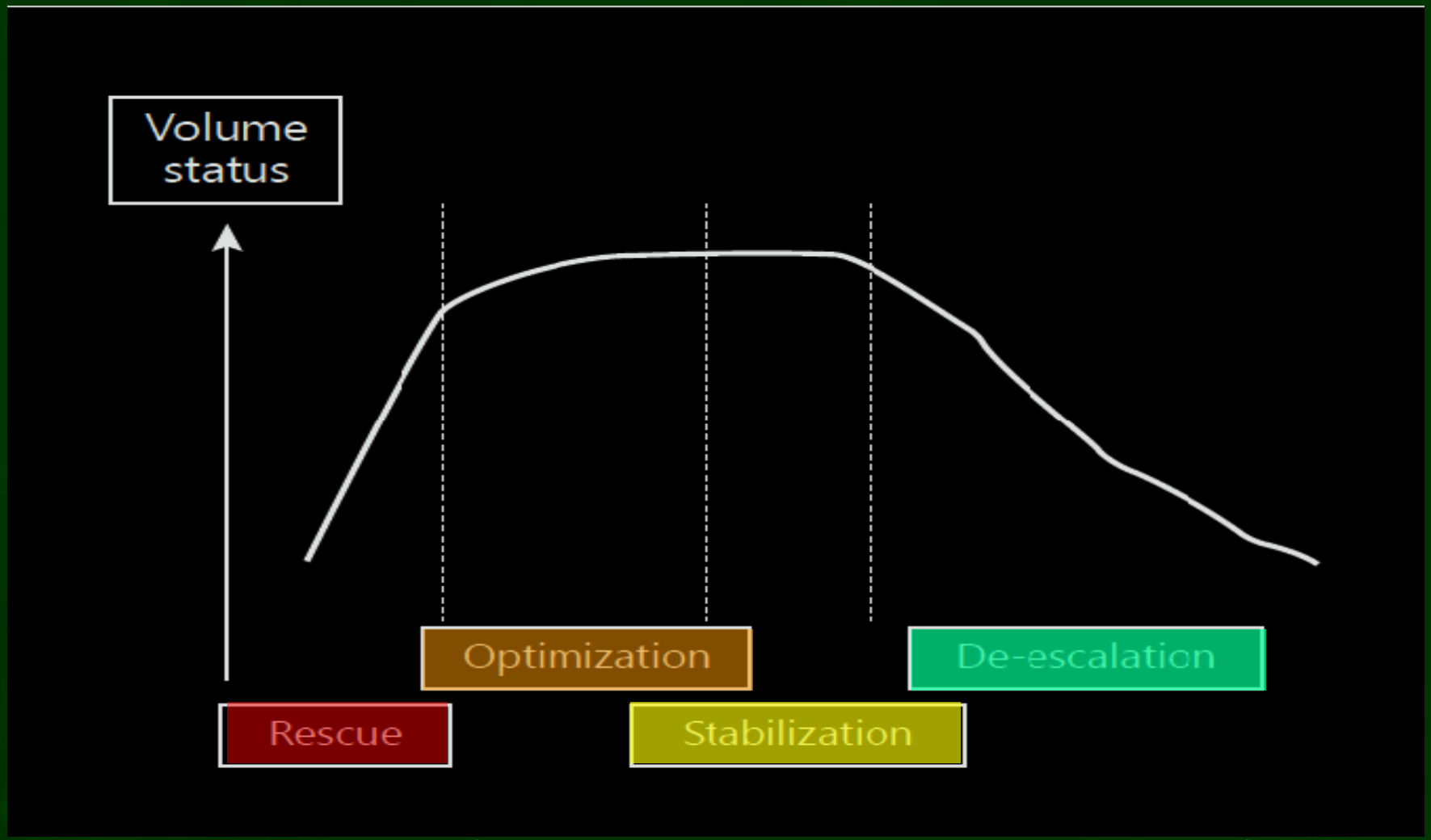


FLUID RESUSCITATION

Phase	Duration	Role	Physiological	Hormones
Ebb	<24 hrs	Maintenance of blood volume, catecholamines	Dec BMR, Dec temp, Dec O2 consump, vasoconst, Inc CO, Inc heart rate, acute phase proteins	Catechol, Cortisol, aldosterone
Flow				
Catabolic	3 – 10 days	Maintenance of energy	Inc BMR, inc Temp, inc O2 consump, -ve N2 balance	Inc. Insulin, Glucagon, Cortisol, Catechol but insulin resistance
Anabolic (MOORE)	10 – 60 days	Replacement of lost tissue	+ve Nitrogen balance	Growth hormone, IGF

- FLUID REPLACMENT
- Amount.
- Type.
  - Crystalloids
  - Colloids
  - Blood

FLUID VOLUME RESUSCITATION



Michard F, Teboul JL: Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. Chest 2002; 121: 2000–2008.

FLUID VOLUME RESUSCITATION

	RESCUE	OPTIMIZATION	STABILIZATION	DE-ESCELATION
Principles	Life-saving	Organ rescue	Organ support	De-escalation
Goals	Correct shock	Optimise & maintain tissue perfusion	Aim =zero or negative fluid balance	Mobilize accumulated fluid
Time	Minutes	Hours	Days	Often weeks
Phenotype	Severe shock	Unstable	Stable	Recovering
Fluid therapy	Rapid boluses	Titrate fluid infusion conservative use of fluid challenges	Minimal fluid maintenance. IV only if oral inadequate	Oral intake
Typical clinical scenario	Septic shock Major burns	Intra-operative GDT Burns DKA	NPO post-operative patients Pancreatitis on "drip-and-suck"	Patients on full enteral feeding in recovery phase of critical illness

Four phases of intravenous fluid therapy: a conceptual model; Hoste E A et al. British Journal of Anaesthesia 113 (5): 740–7 (2014)

PHASES OF HAEMODYNAMIC TREATEMENT

	RESCUE	OPTIMIZATION	STABILIZATION	DE-ESCELATION
Principles	Life-saving	Organ rescue	Organ support	De-escalation
Goals	Correct shock	Optimise & maintain tissue perfusion	Aim =zero or negative fluid balance	Mobilize accumulated fluid
Time	Minutes	Hours	Days	Often weeks
Haemodynamic targets	Autoregulatory thresholds of perfusion pressure	Macro/microcirculatory blood flow parameters	Weaning of vasopressors with stable haemodynamic conditions	Return to premorbidchronic values of pressure and flow
Treatment options	Rapid boluses + vasopressors	Rapid boluses + vasopressors +Inotropes	Minimal fluid maintenance. IV only if oral inadequate +decrease support	Diuretics or other means of fluid removal

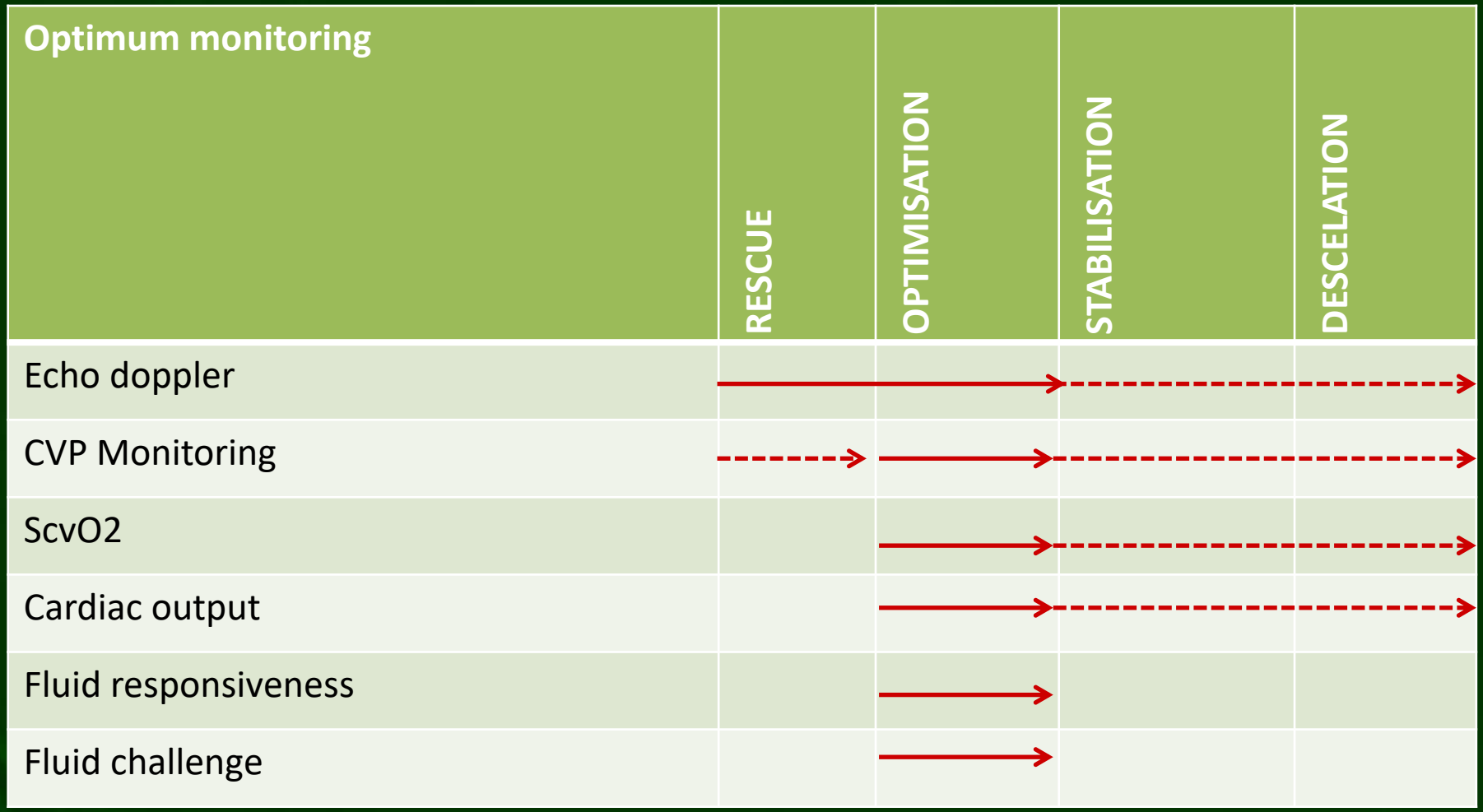
Fluid Therapy: Double-Edged Sword during Critical Care? Jan Benes et al. BioMed Research International Volume 2015

MONITORING FLUID REPLACEMENT

Minimum monitoring requirement	RESCUE	OPTIMISATION	STABILISATION	DESCELATION
Blood pressure	→	→	→	→
Heart Rate	→	→	→	→
Lactate	→	→	→	→
ABG	→	→	→	→
Pulse Volume /Capillary refill	→	→	→	→
Altered mental status	→			
Urine output		→	→	→
Fluid balance		→	→	→

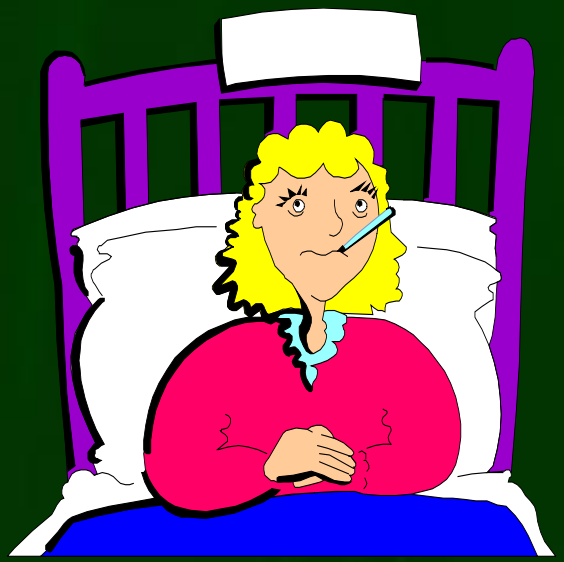
Acute Dialysis Quality Initiative. www.ADQI.com

MONITORING FLUID REPLACEMENT



Acute Dialysis Quality Initiative. www.ADQI.com

CASE CAPSULE



A healthy young lady of 32yrs. Undergoes an appendicectomy. She is fine till the third post-operative day, when she has three grand mal seizures. She recieves 20mgm of diazepam and 250mgm of Phenytoin IV and undergoes laryngeal intubation with mechanical ventilation.

CAUSE:-

Infused 5% Dextrose only on Day 1.

Plenty of plain water orally on Day 2.

?

CASE CAPSULE

Plan of treatment?

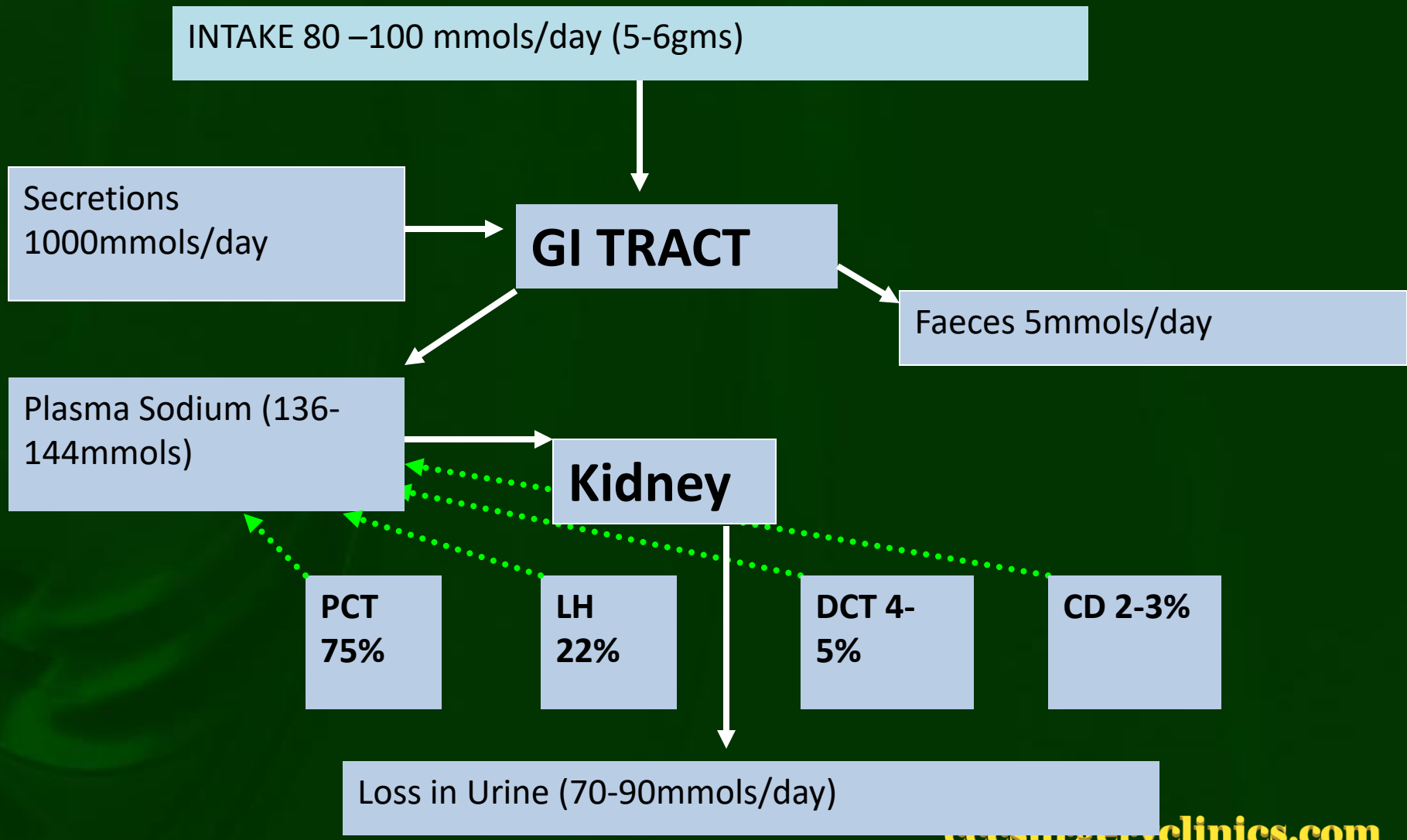
Withhold water.

SLOW Infusion of 3 per cent Sodium Chloride.

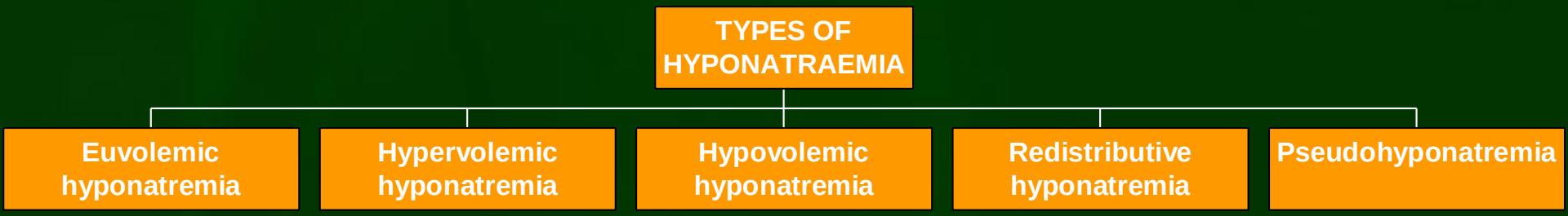
Intravenous administration of 20mgm of Furosemide.

- Her body weight is 46kgs.
- Sodium concentration- 112mmol/litre
- Potassium concentration- 4.1 mmol/litre
- Serum osmolality- 228mOsm/kg of water.
- Urine osmolality- 510mOsm/kg of water.

NORMAL SODIUM BALANCE



TYPES OF HYPONATRAEMIA



TBW increases while total sodium remains normal. The ECF volume is increased minimally to moderately, but edema is not present

Total body sodium increases, and TBW increases to a greater extent. The ECF is increased markedly, and edema is present.

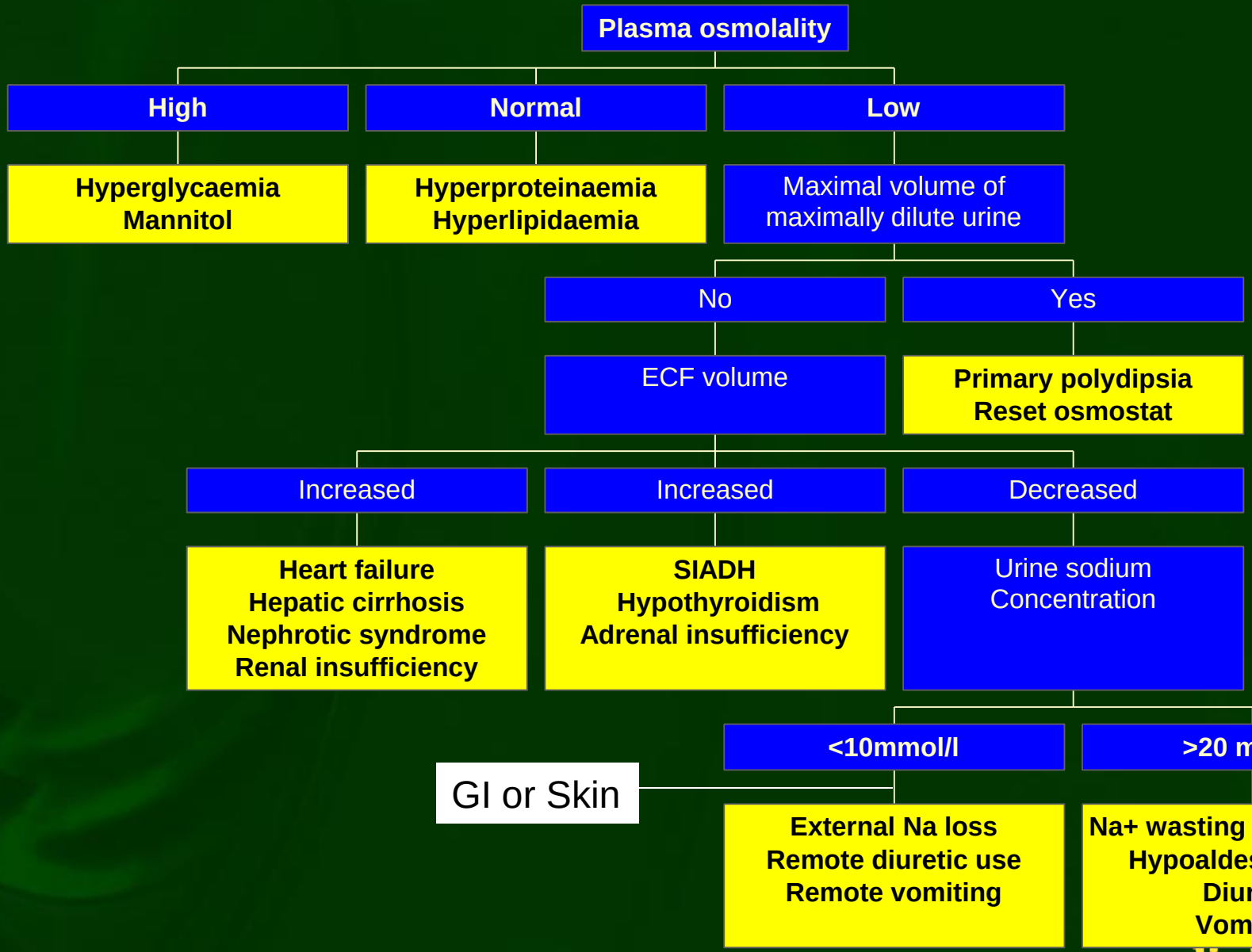
Total body water (TBW) decreases; total body sodium (Na^+) decreases to a greater extent. The extracellular fluid (ECF) volume is decreased.

Water shifts from the intracellular to the extracellular compartment, with a resultant dilution of sodium. The TBW and total body sodium are unchanged. This condition occurs with hyperglycemia

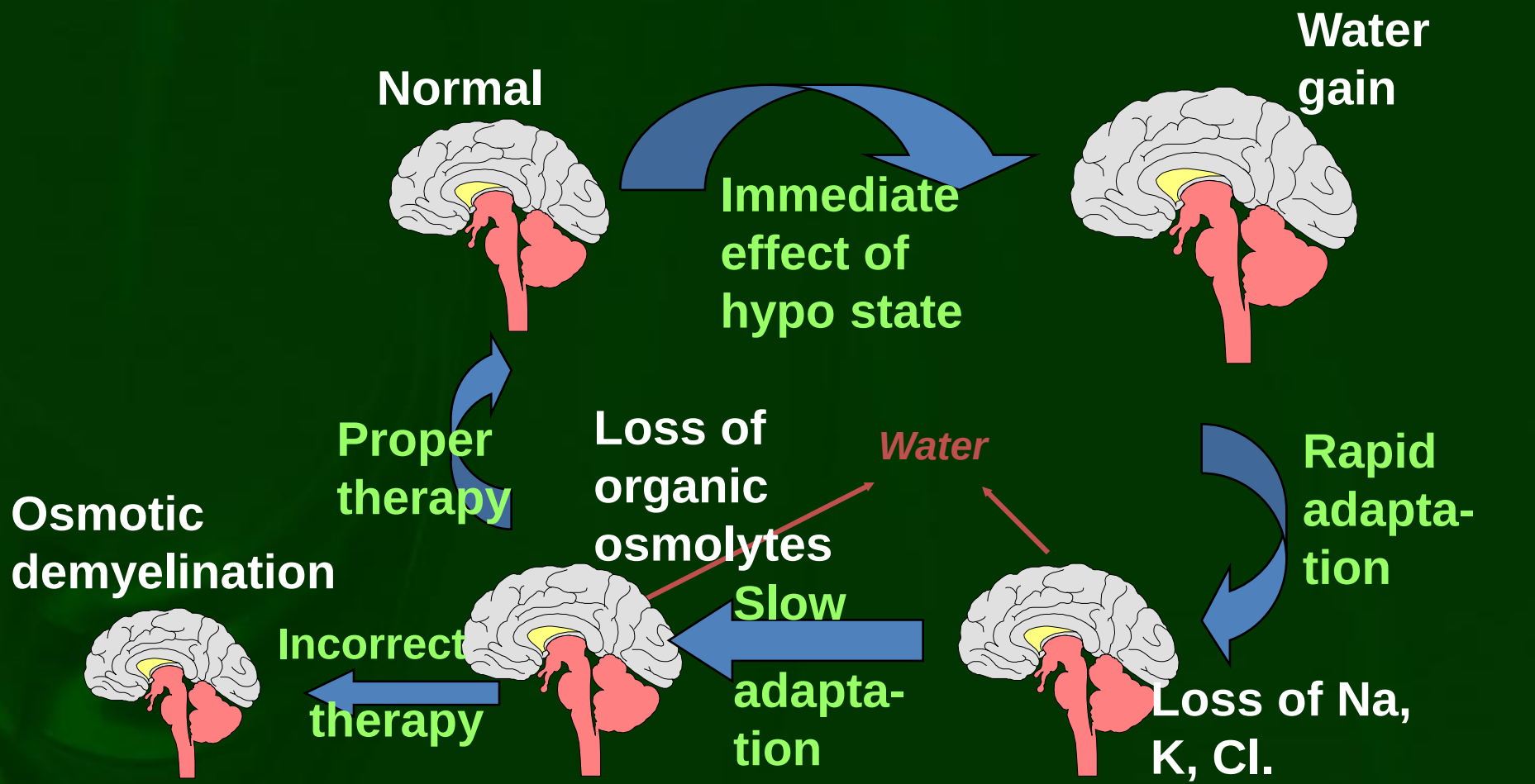
The aqueous phase is diluted by excessive proteins or lipids. The TBW and total body sodium are unchanged. This condition is seen with hypertriglyceridemia and multiple myeloma.

Na H2O Na H2O Na H2O

HYPONATRAEMIA



Effect of hyponatraemia and its correction

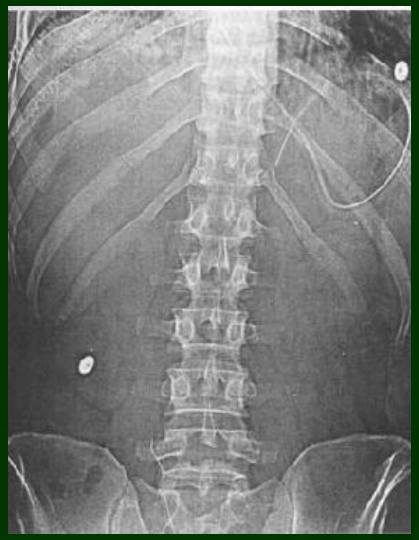


CASE CAPSULE

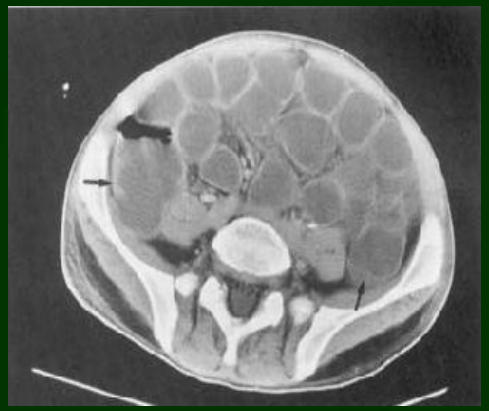
?

- An elderly lady of 63yrs. Undergoes a difficult resection- anastomosis for a gangrenous segment of small intestine, which was incarcerated under a post-operative band.
- Her abdomen is distended, she is obtunded, and her bowel sounds are absent.
- The tongue is red and swollen, skin turgor is diminished and she is not totally coherent.
- She has mild orthostatic hypotension

CASE CAPSULE



Straight X-ray

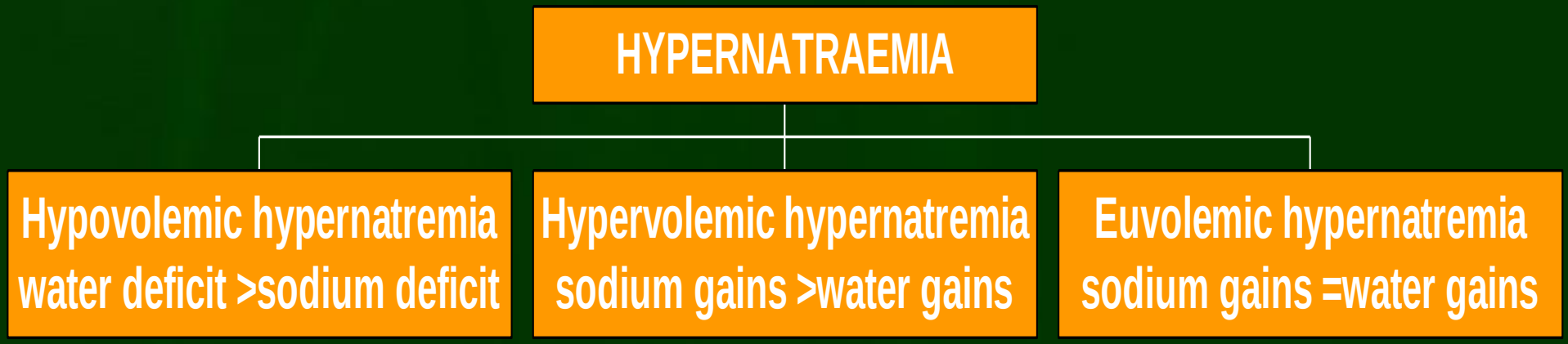


CT Scan

- Serum sodium-168mmol/liter
- Serum Potassium-4.0mmol/liter
- Body weight is 60kg.

HYPERNATRAEMIA DUE TO SODIUM-POOR FLUID LOSS

TYPES OF HYPERNATRAEMIA

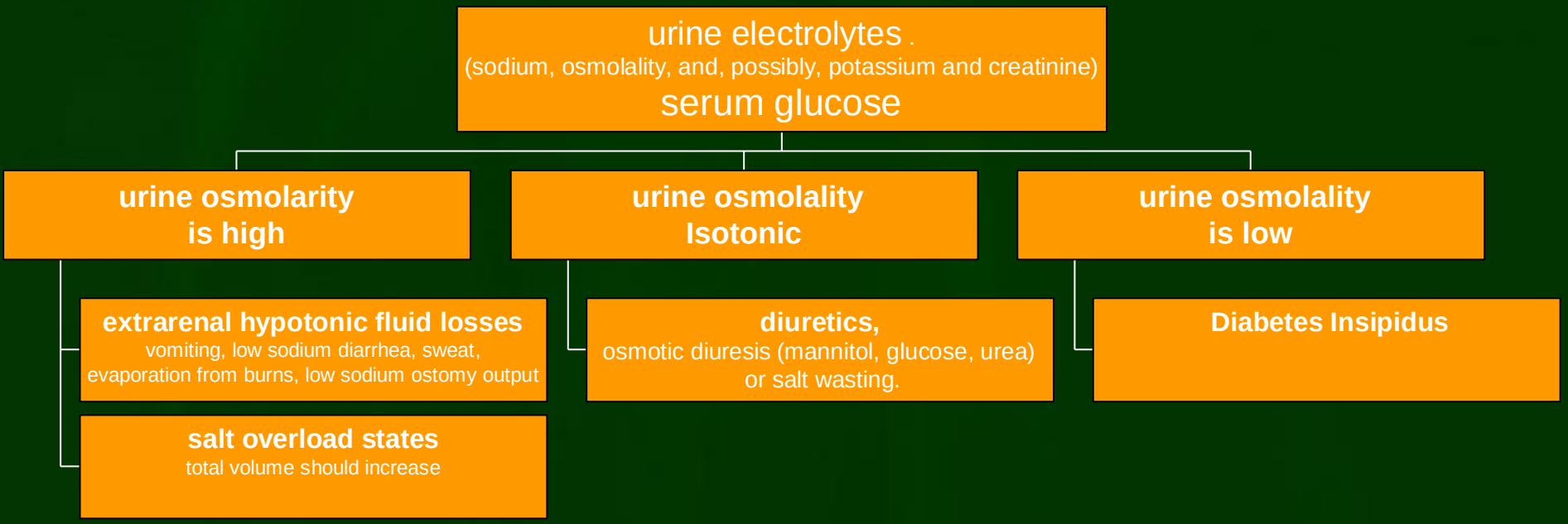


Na H2O

Na H2O

Na H2O

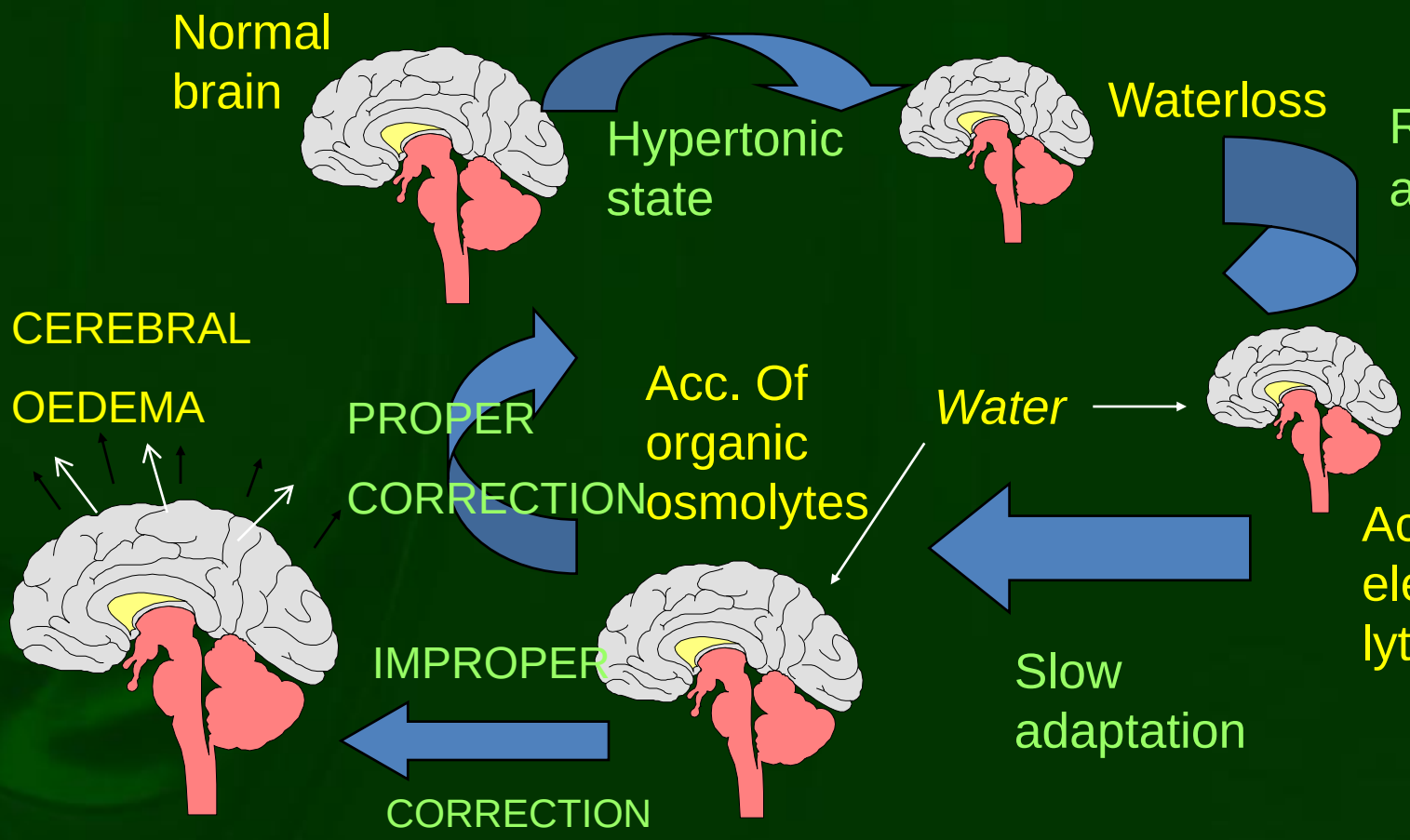
HYPERNATRAEMIA



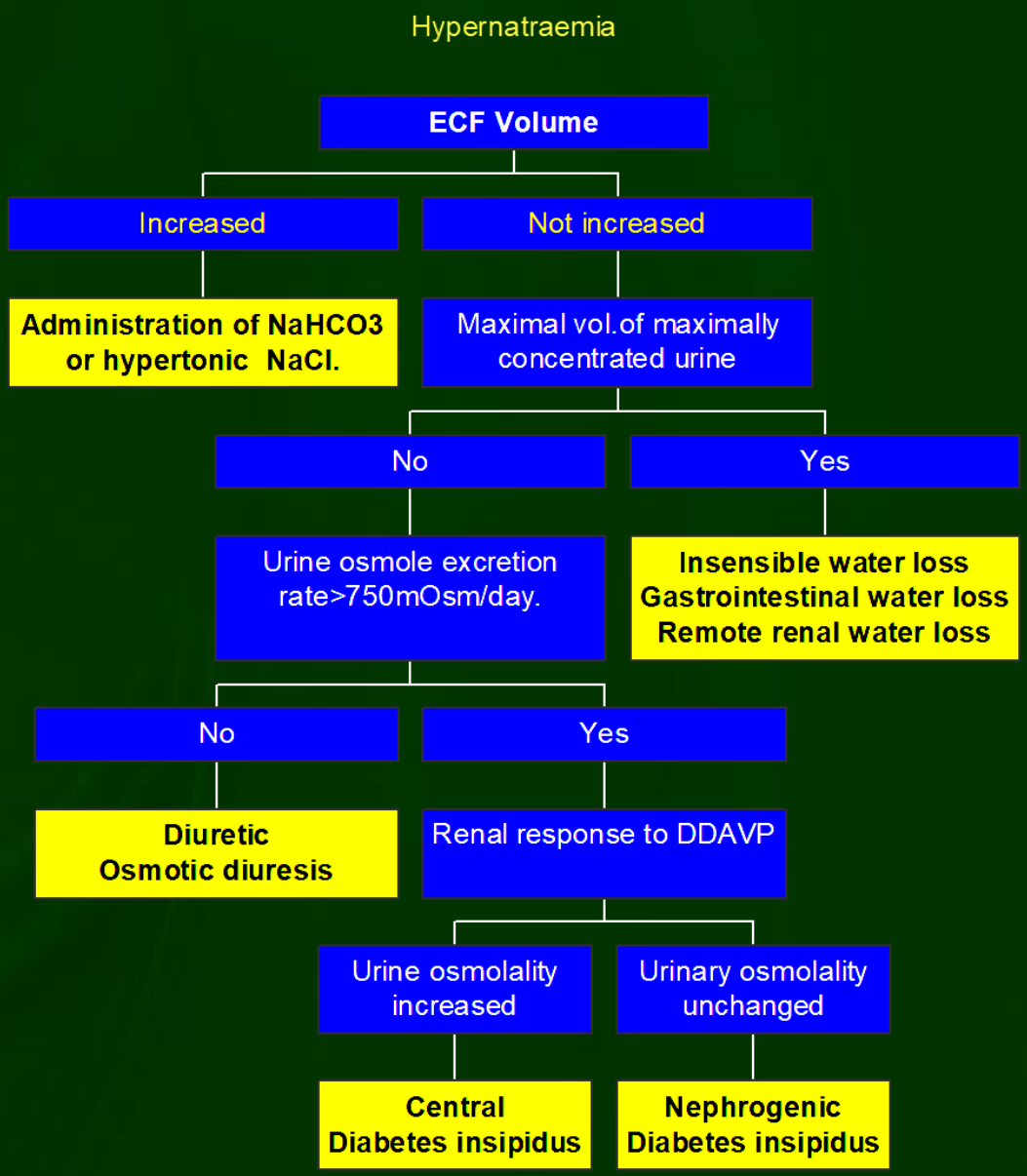
THUMB RULE:-

- Serum sodium levels of more than 190 mEq/L usually indicate long-term salt ingestion.
- Serum sodium levels of more than 170 mEq/L usually indicate DI.
- Serum sodium levels of more than 150-170 mEq/L usually indicate dehydration.

Effect of Hypernatraemia and its correction



HYPERNATRAEMIA



Treatment of Hypernatraemia

Treatment

Same general principles as that of hyponatremia . Rapid correction should be avoided because of the brain's adaptive response to hypernatremia and the potential risk of cerebral edema.

- The current recommendation is to lower the serum sodium concentration by about 0.5 mEq/L per hour and to replace no more than half the water deficit in the first 24 hours.
- The following formula can be used to calculate the water deficit (total body water, in kilograms, is 60% of lean body mass in men and 50% in women):

Water deficit = total body water (serum sodium concentration ÷ 140 - 1)

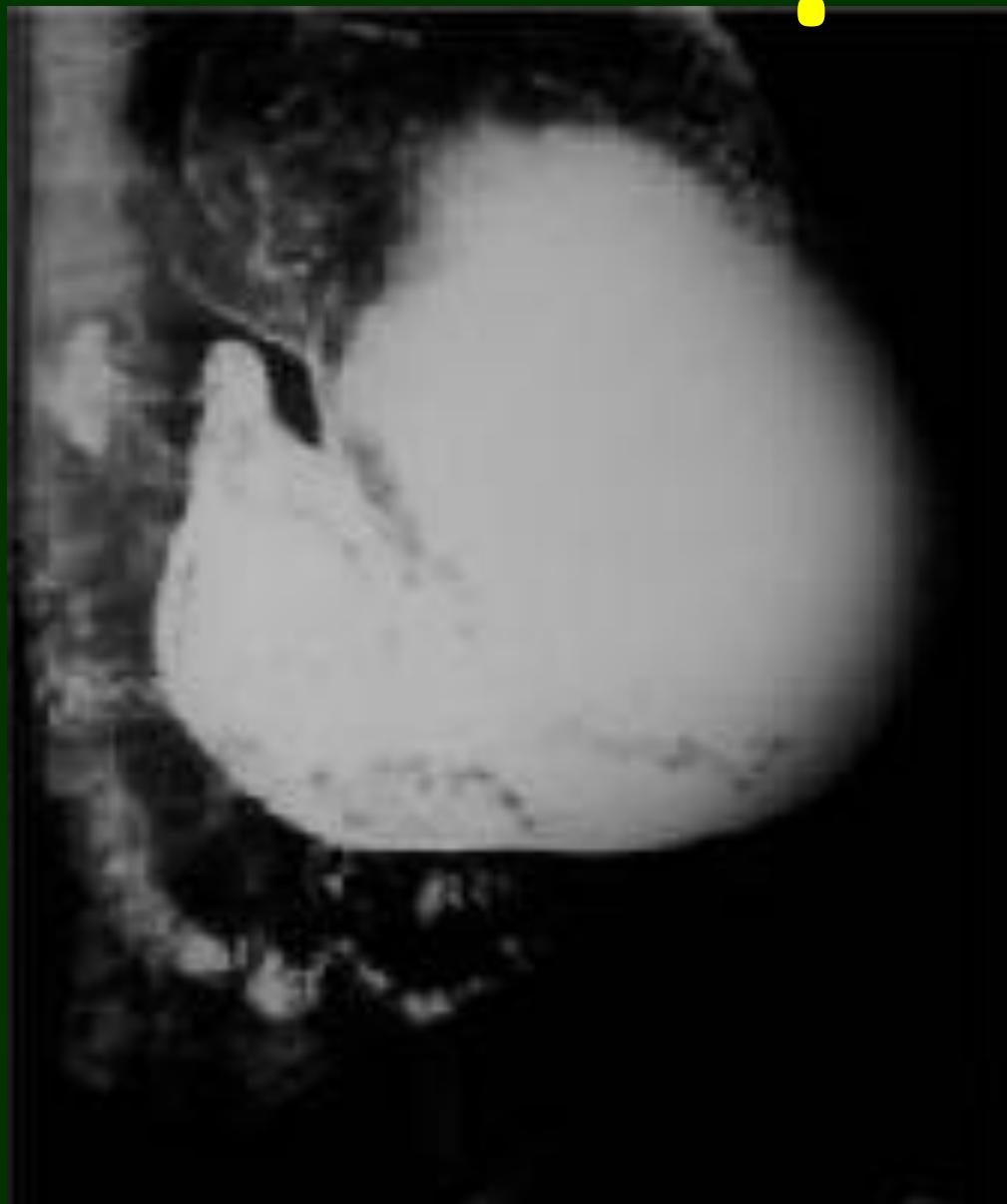
Treatment of Hypernatraemia

- In hypovolemic hypernatremia, normal saline solution is indicated initially to correct the intravascular volume deficit. When that is accomplished, more hypotonic fluids (eg, D5 half normal saline[75%] or D5 third normal [50%]) can be used.
- In hypervolemic hypernatremia, removing the source of salt excess, administering diuretics, and replacing water are important to successful therapy.
- In euvolemic hypernatremia usually require water replacement alone--either free water orally or an infusion of 5% dextrose in water.

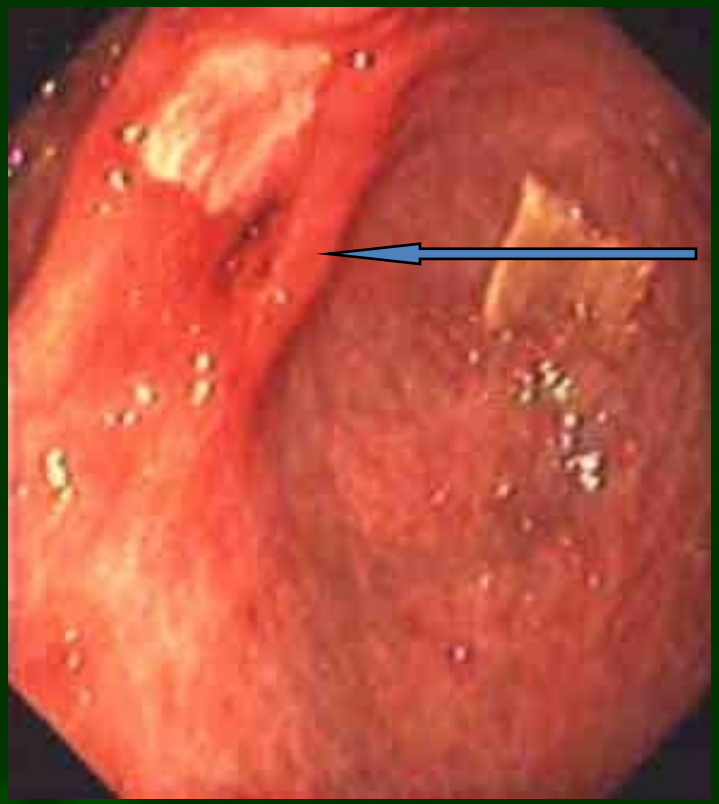
CASE CAPSULE

?

- A 62 year old man, presents with painless,profuse projectile vomiting containing old food material
- No history of any previous surgery, he complains of a long standing, mild
- Epigastric pain.
- On Examination; the patient had a BP of 100/60;Pulse110/min.Eyes shrunken,
- Decreased skin turgor, slow to questions, decreased tendon reflexes, a scaphoid abdomen with a visible peristalsis moving from left to right, no free fluid or lump in the abdomen.
- Barium meal X-ray showed the following



CASE CAPSULE



Upper GI Endoscopy Revealed the following finding.

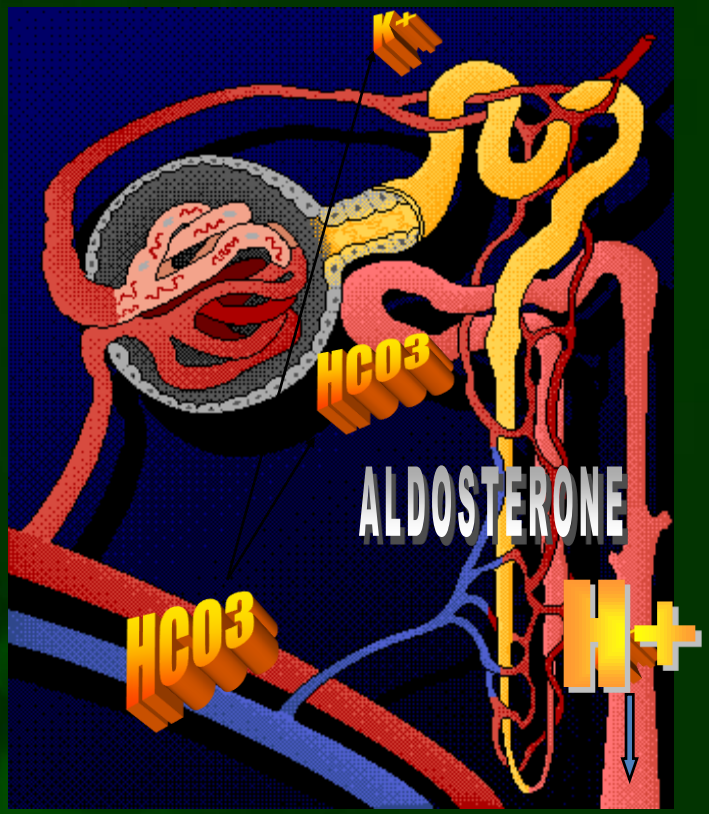
BLOOD BIOCHEMISTRY

- Na- 120mEq/l
- K - 2.8mEq/l
- Cl- 80mEq/l
- HCO_3- 38mEq/L
- Creatinine- 1.3υgm/l
- Urea- 62mgm/l
- Albumin- 2.9gm/l

SYNOPSIS

- GOO with-
- Hyponatraemia
- Hypokalaemia
- Hypochloraemia
- Hypoalbuminaemia
- Metabolic Alkalosis

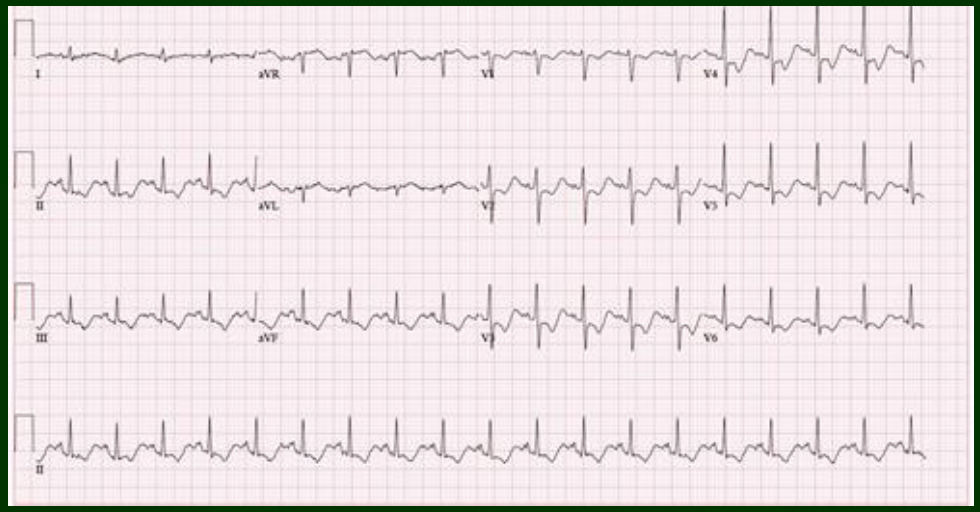
Principle of Hypokalaemia



- *Direct potassium losses contribute only minimally to actual loss.*
- Loss of gastric acid leads to metabolic alkalosis which increases tubular cell potassium concentration.
- Elevated plasma bicarbonate leads to increased bicarb to distal nephron, leading to an augmentation of potassium loss.
- Secondary aldosteronism augments potassium excretion
- Hypokalaemia-induces the excretion of H+ ions in place of K+ ions- PARADOXIC ACIDURIA

Case study -Blood results

- Haemoglobin-8.2mgm%
- TLC- normal
- Urea- 62mgm/dl
- Creatinine-1.3*u*gm/dl
- Sodium- 120mmol/L
- Potassium-2.8mmol/L
- Bicarbonate-38mmol/L



ECG changes include flattening and inversion of T waves in mild hypokalemia, followed by Q-T interval prolongation, visible U wave and mild ST depression when hypokalaemia is more severe.

oRenal tubular acidosis

oHyperaldosteronism

oMagnesium depletion

oLeukemia (mechanism uncertain)

oVomiting or nasogastric suctioning

oDiarrhea

oEnemas or laxative use

oIleal loop

HYPOKALAEMIA

· Renal losses

· GI losses

· Medication effects

· Transcellular shift

oDiuretics (most common cause)

oBeta-adrenergic agonists

oSteroids

oTheophylline

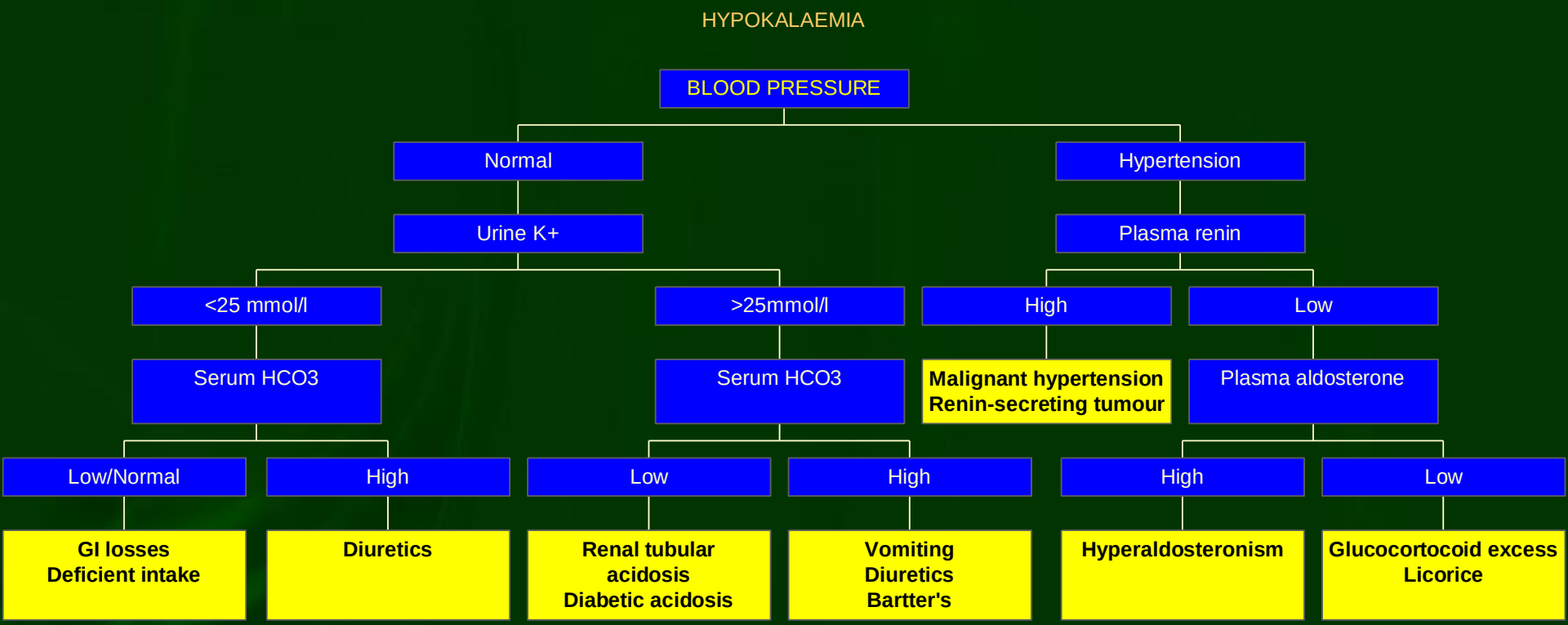
oAminoglycosides

oInsulin

oAlkalosis

Malnutrition or decreased dietary intake, parenteral nutrition

HYPOKALAEMIA



POTASSIUM BALANCE

ECF

10% 350mEq

3.5-5mEq/L

Bone 300mEq (8.6%)

Urine 90-95mEq/L(1%)

Interstitial fluid 35mEq/L(0.4%)

ICF

90% 3150mEq

140-150mEq/L

Muscle 2650 mEq/L(76%)

Liver 250mEq/L(7%)

RBC 250mEq/L(7%)

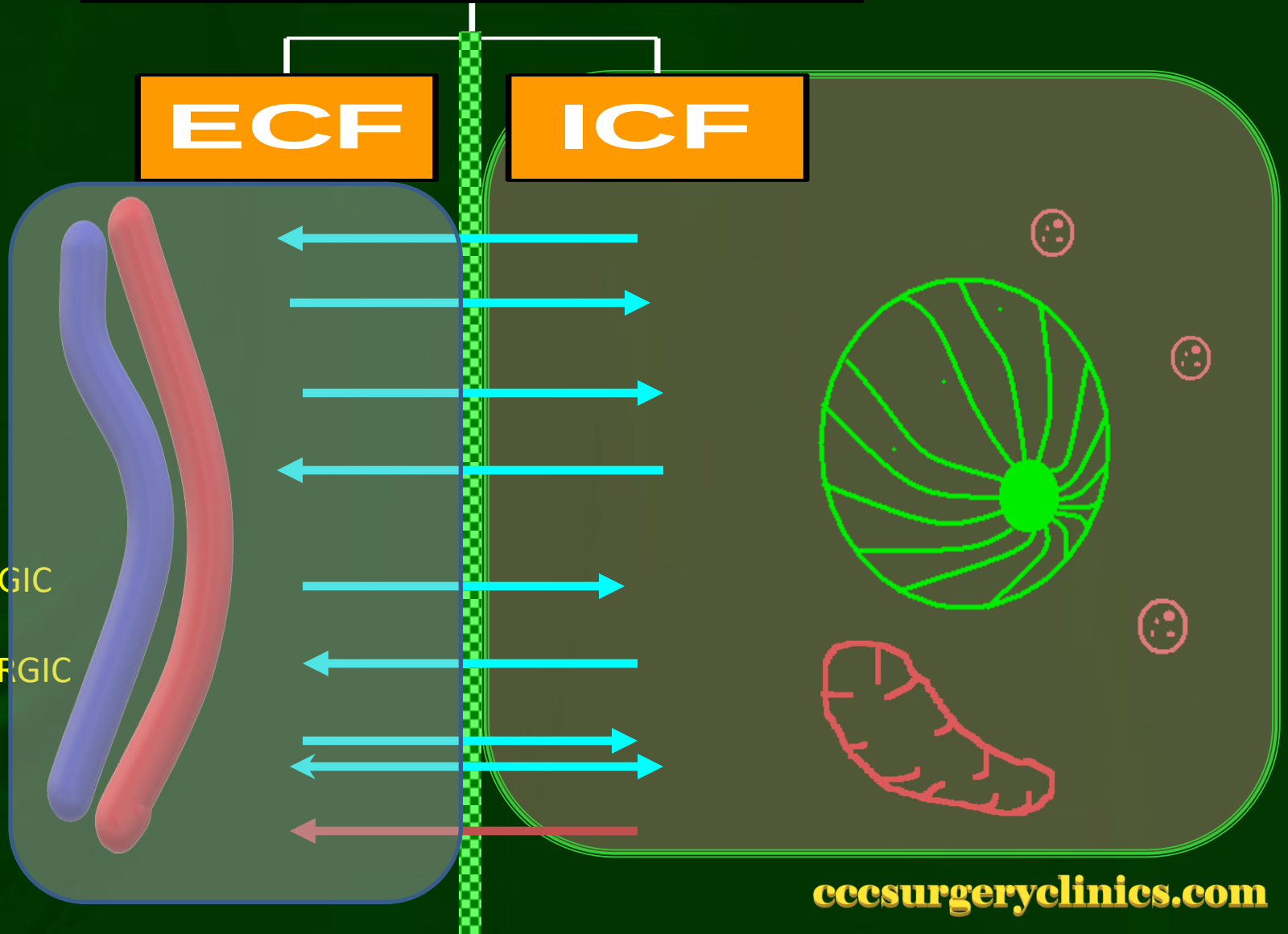
LOSS

URINE (90-95mEq/D)

STOOL (5-10mEq/D)

SWEAT (<5mEq/D)

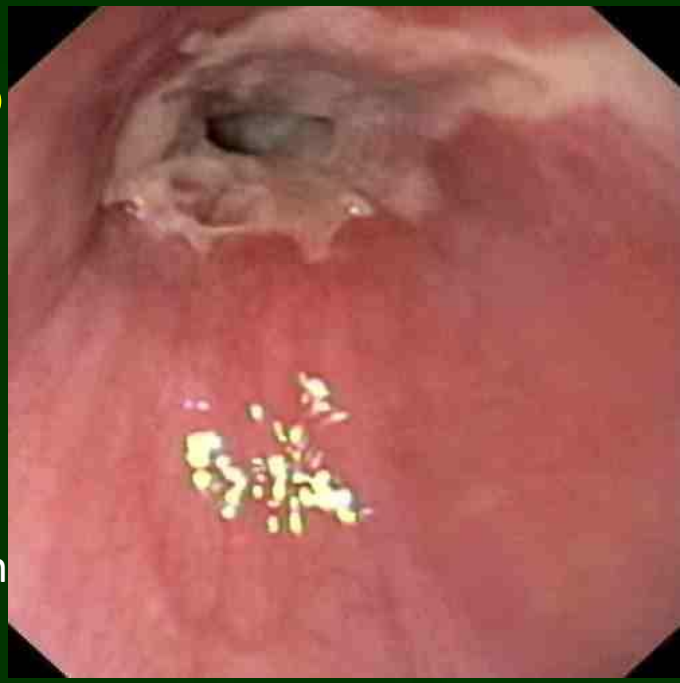
POTASSIUM BALANCE



Treatment of Hypokalaemia

If K^+ is between 2.5 - 3.5 mmol/L & no symptoms of hypokalaemia

- use oral K^+ supplements, at least 80mmo hours
- normal maximum daily oral dose is 100mmol/l



- may cause nausea, vomiting and GI ulceration
- K^+ must be closely monitored and supplements stopped wh 4.0 mmol/l

Treatment of Hypokalaemia

If K^+ is < 2.5 mmol/l and a clinical decision is made to treat with IV Potassium

- Use IV potassium either centrally or peripherally.
- Ready-made potassium containing infusion bags should be prescribed and administered, unless there is a specific indication to do otherwise
- A syringe pump may be used for central line administration.
- All patients treated with IV potassium should have at least once daily measurement of serum potassium until levels are shown to be satisfactory

Treatment of Hypokalaemia

Peripheral Line IV Administration

Rate of Administration

- 10mmol/hour
  Maximum 20mmol/hour with ECG monitoring

Maximum Concentration

- 40mmol/l
  Phlebitis may occur at concentrations > 30mmol/l

Central Line IV administration

Rate of Administration

- 10mmol/hour
  Maximum 20mmol/hour with ECG monitoring

Maximum Concentration

- Can give undiluted KCL 2mmol/ml at a rate of 10-20mmol/hr via a syringe driver with appropriate ECG monitoring

Caution!! when you add KCl to NS!

Strong Potassium Chloride Solution:

- restricted to ICU,CICU,CCU
- 10ml (20mmol) must be diluted to at least 500ml for peripheral administration
- dilute with sodium chloride 0.9%
- MIX WELL (otherwise, potassium chloride being heavier than the usual diluents will sink to the bottom if not mixed sufficiently and be given in effect undiluted as a bolus; this can be fatal)

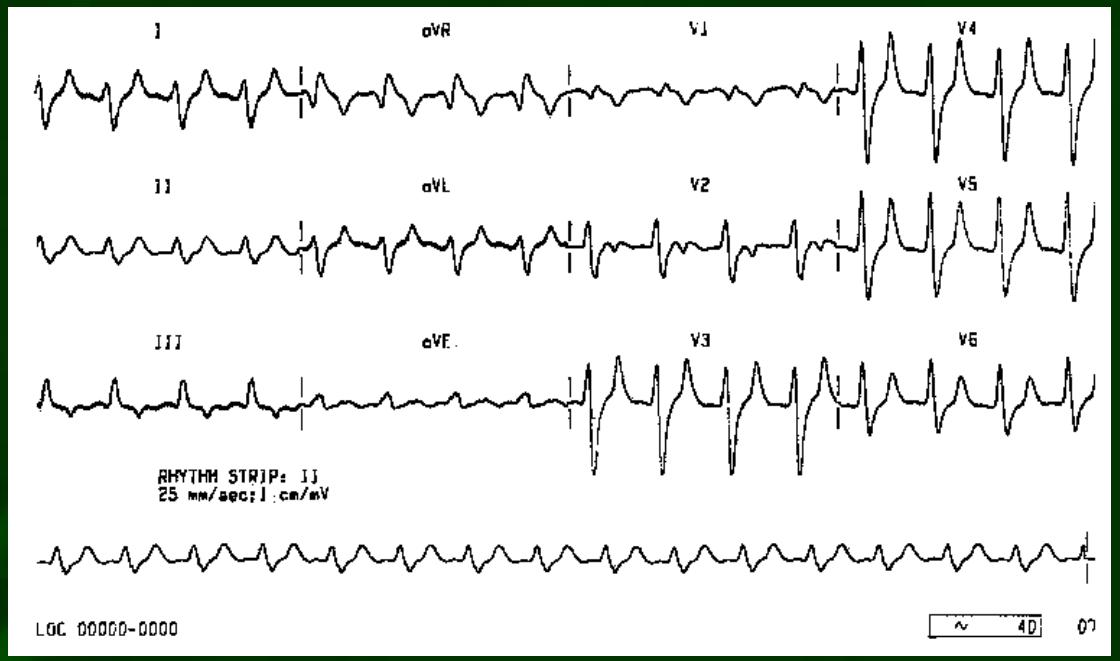
CASE CAPSULE

?

- An 62 yrs old farmer, sustained a crush injury to both his legs due to a tractor injury
- He was admitted at a peripheral hospital for 3 days before being transferred to the referral center.
- On examination he was found to have a thready irregular pulse, hypotension, was oliguric.
- The crush injury to both his legs were severe, with absent peripheral pulses with a compound injury to the right leg and a compartment syndrome of the left leg.
- In the past, the only relevent history was that of long standing small joint arthritis for which the patient had been on NSAIDS.

CASE CAPSULE

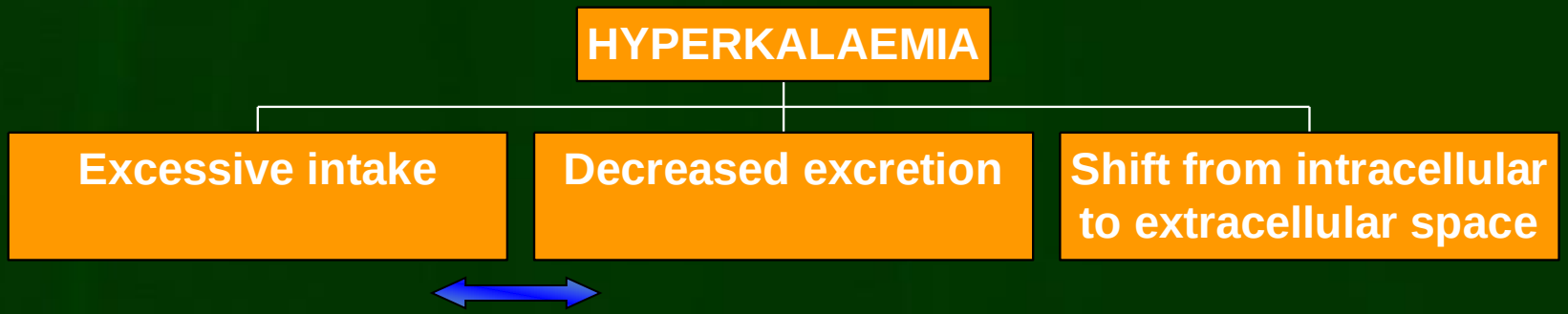
ECG



Urea-112mgm/
Creatinine-2.2*u*gm
Na-138mmol/l
K-7.4mmol/l
Hb-7.6mgm/dl
TLC-15600; N 86
X-Ray-bilateral
comm.# tib/fib

CAUSE: HYPERKALAEMIA(muscle damage+NSAIDS)

CAUSES OF HYPERKALAEMIA

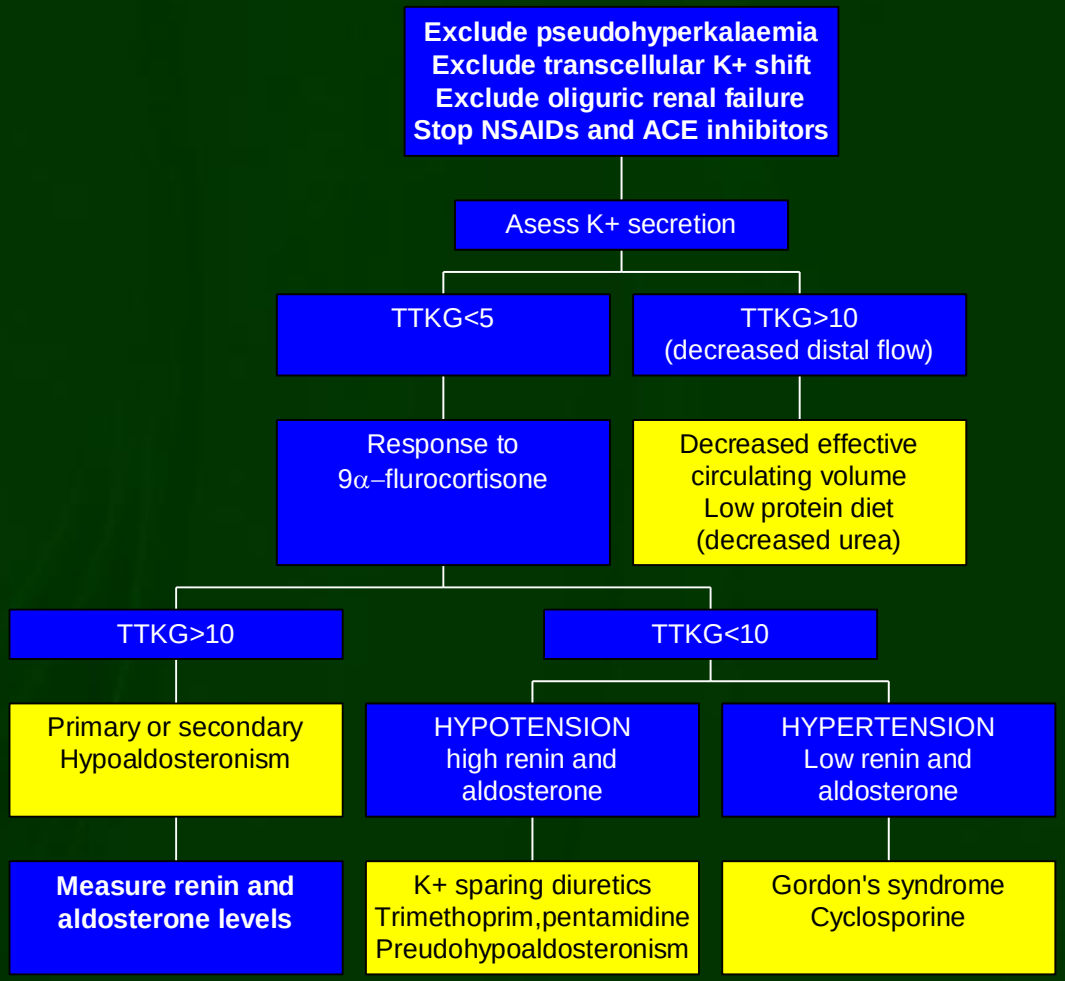


Excessive potassium intake alone is an uncommon cause of hyperkalemia

- renal failure
- drugs that interfere with potassium excretion
- angiotensin-convening enzyme inhibitor
- NSAIDs
- type IV renal tubular acidosis

- rhabdomyolysis
- tumor lysis
- insulin deficiency
- acute acidosis.

HYPERKALAEMIA



Treatment of Hyperkalaemia

Emergency treatment of hyperkalemia is targeted towards one of three objectives:

- Antagonizing calcium, eg Calcium Chloride administration
- Causing potassium to shift into cells, eg with administration of sodium bicarbonate, insulin + glucose, or nebulized albuterol
- Removing potassium from the body, eg with diuresis with a non-potassium-sparing diuretic, administration of cation exchange resin, or dialysis

Treatment of Hyperkalaemia

Therapy	Dose	Onset of Effect	Duration of Effect
Calcium chloride	5-10 ml IV of 10% solution (500-1000mg	1-3 minutes	30-60 minutes
Sodium bicarbonate	1 mEq/kg IV bolus	5-10 minutes	1-2 hours
Insulin plus glucose (use 1 unit of insulin/2.5 g glucose)	Regular insulin 10 U IV plus 50 ml D_{50} (25 g glucose) IV bolus	30 minutes	4-6 hours
Nebulized albuterol	10-20 mg nebulized over 15 minutes	15 minutes	15-90 minutes
Furosemide	40-80 mg IV bolus	With onset of diuresis	Until diuretic effect ends
Kayexalate	15-50 g PO or PR, plus sorbitol	1-2 hours	4-6 hours
Peritoneal dialysis or hemodialysis	Per institution	Immediate	Until dialysis completed

CASE CAPSULE

- A 44 year old man was admitted with a 2 day history of acute gastroenteritis leading to severe diarrhoea.
- What is the acid base disorder?
- What is the Anion Gap?
- Is the compensation adequate?

- pH = 7.31,
- PCO_2 = 33 mmHg,
- pO_2 = 93 mmHg,
- HCO_3 = 16
- Na= 134
- Cl= 108

CASE CAPSULE

- A 44 year old man was admitted with a 2 day history of acute gastroenteritis leading to severe diarrhoea.
- What is the acid base disorder?
- What is the Anion Gap?
- Is the compensation adequate?

ANSWERS READY?

CASE CAPSULE

- Overall change is acid.
- pCO2 is low so cannot be a respiratory acidosis.
- HCO3 is low so contributing to the acidosis.
- Metabolic acidosis.
- Components pulling in opposite directions
- Anion gap is Na- (Cl + HCO3) = 134- (108 + 16) = 10 = N.

- pH = 7.31,
- PCO2 = 33 mmHg,
- pO2 = 93 mmHg,
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CASE CAPSULE

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- 134- (108 + 16) = 10 = N.
- Is the compensation adequate?

- pH = 7.31,
- PCO2 = 33 mmHg,
- pO2 = 93 mmHg,
- HCO3 = 16
- Na= 134
- Cl= 108

- WINTER'S FORMULA
- PCO2 = 1.5 x [HCO3-} +10.
- = 34

- Metabolic normal anion gap acidosis with adequate compensation.

NORMAL pH

↑ pCO_2

Respiratory

↓ pCO_2

Respiratory

Acidosis

7.35 – 7.45

Alkalosis

Metabolic

Metabolic

↑H^+ / ↓HCO_3^-

↓H^+ / ↑HCO_3^-

↑ OTHER ACIDS ↓

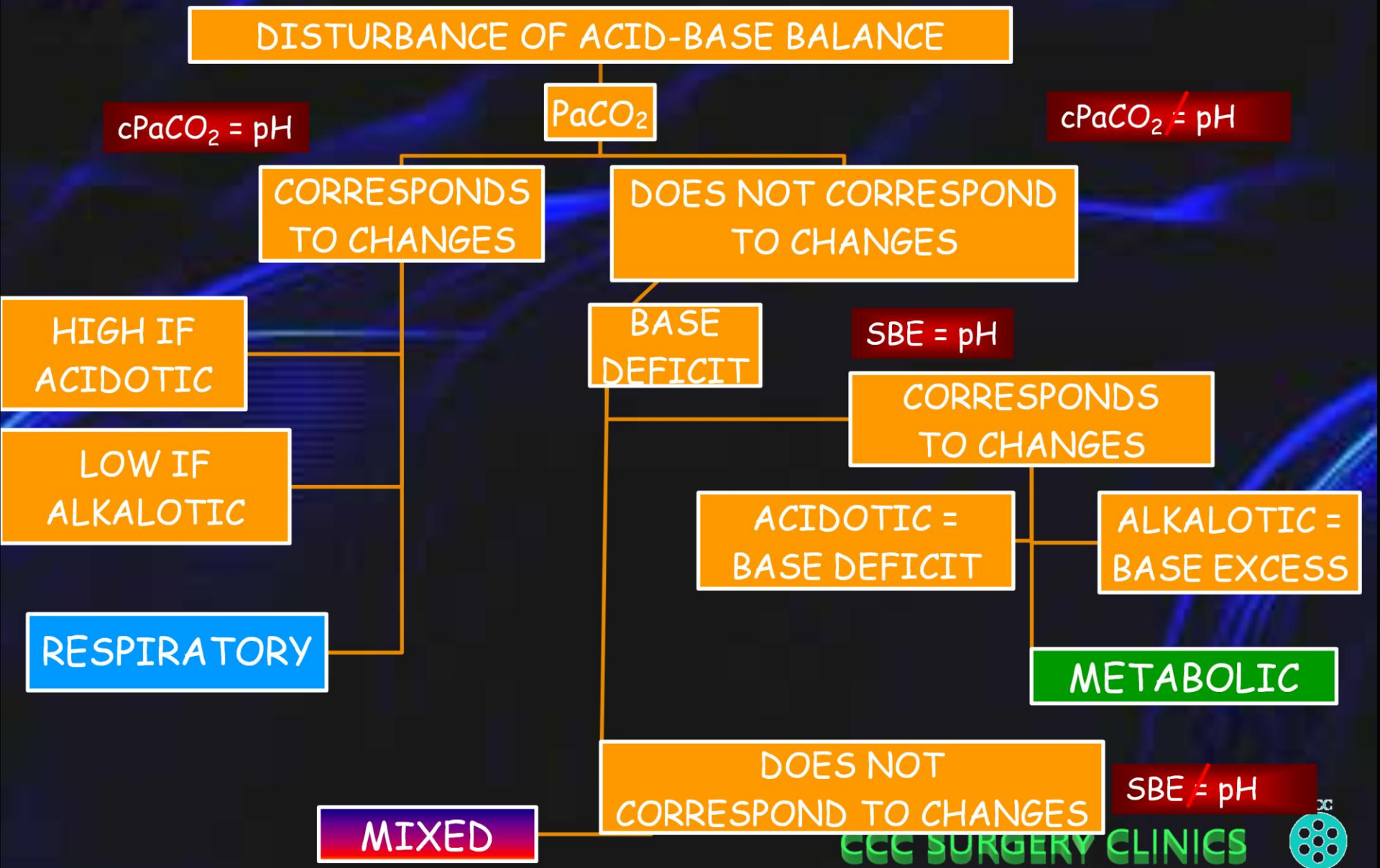
INTERPRETATION OF BLOOD GASES

'NORMAL' BLOOD GASES	
pH	7.35 – 7.45
PaO_2	13kPa
$PaCO_2$	5.3kPa
HCO_3	22 – 25mmol/l
Base deficit or excess	-2 to +2 mmol/l

INTERPRETATION OF BLOOD GASES

'NORMAL' BLOOD GASES	
pH	7.35 – 7.45
PaO_2	90-100mmHg
$PaCO_2$	35-45mmHg
HCO_3	22 – 25mmol/l
Base deficit or excess	-2 to +2 mmol/l

INTERPRETING BLOOD GASES



COMPENSATORY MECHANISMS

PHYSIOLOGIC

- Weak acids and its base salts.
- Weak base and its acid salt eg
  - Bicarbonate-carbonic acid.
  - Intracellular protein. (Hb)
  - Phosphates in bone.

PULMONARY

Changes in ventilation.

- Acidosis– increased ventilation, CO_2 blown off.
- Alkalosis- lower ventilation, CO_2 retained.

RENAL & BONE

- Acidosis – kidneys excrete excess H^+, retain $HCO3^-$.
- Alkalosis kidneys excrete $HCO3^-$, retain H^+.
- Bone –bicarb & phosphates.

TWO ISSUES

1. COMPENSATION
2. ASSESSMENT OF THE "INDEPENDENTS" THE OTHER ACIDS.

COMPENSATION

IN COMPENSATION

Standard Base Excess (SBE)

- Standard base excess is dose of acid or alkali to return the ECF to normal pH (7.40) under standard conditions (at 37^0C at a PCO_2 of 40 mm Hg).
- BASE DEFICIT = metabolic acidosis (amount of BASE reqd.)
- BASE EXCESS = metabolic alkalosis (amount of ACID reqd.)

RELATION BETWEEN BASE EXCESS AND pCO2

- Whenever the pH is normal, i.e., pH = 7.4. then the PCO_2 and the SBE are equal and opposite.
- In such circumstances,
  - if the ↑PCO_2 is described as a *representing acidosis* then logically
  - the ↑SBE must be the exact opposite, *representing alkalosis*.
- Fortunately, the slope for pCO_2/BE when ph = 7.4 gives us this ratio:
  - Five (5) mmHg **change** in the PCO2 is equivalent to a Three (3) units of **change** in the SBE.
  - Thus, *(**change** in)* pCO_2: *(**change** in)* SBE = 5:3
  - chpCO_2/chSBE=5/3

RATIOS

- $\frac{\text{Change in } pCO_2}{\text{Change in SBE}} = \frac{chpCO_2}{chSBE} = \frac{5}{3}$

- If HCO_3 is only given?

CALCULATING HCO3 from pCO_2

ACTUAL NOT CHANGE

- WINTER'S FORMULA
- When ACIDOSIS is present:-
- $pCO_2 = \{1.5 \times HCO_3\} + 10$

- FORMULA (Not-SUMMER'S ☺)
- When ALKALOSIS is present:-
- $pCO_2 = 0.7\{HCO_3\} + 21mmHg +/- 5$

ASSESSMENT OF OTHER ACIDS IF PRESENT THE INDEPENDENT H^+ :

ANION-GAP CHANGES

Anion Gap = $(Na^+ + K^+) - (HCO3^- + Cl^-)$ = 12-16mEq/l

Low Anion Gap<6mEq/L
Hypoalbuminaemia
Monoclonal protein

ANION-GAP CHANGES

Anion Gap = $(Na^+ + K^+) - (HCO3^- + Cl^-)$ = 12-16mEq/l

Normal Gap= 12-16mEq/L	
A	Acid load
C	Chronic Renal Failure
C	Carbonic Anhydrase inhibitors
R	Renal Tubular Acidosis
U	Ureteroenterostomy
E	Expansion/Extra-Alimentation
D	Diarrhoea

ANION-GAP CHANGES

Anion Gap = $(Na^+ + K^+) - (HCO3^- + Cl^-)$ = 12-16mEq/l

High Anion Gap >16 mEq/L	
U	Uraemia; Sulphate, Phosphate, Urate.
M	Massive rhabdomyolysis; H^+, Organic anions.
I	Ingestions; Methanol/Ethanol poisoning, Salicylates
L	Lactic Acidosis; L-lactate, D-lactate.
D	DKA; beta-hydroxybutyrate, acetoacetate.

OSCE II

OSCE TIPS for DNB

- You will have to answer on separate sheets, WHICH ARE COLLECTED AFTER EACH OSCE.
- Write down whatever data is given, sometimes the second/third slide may not have it.
- Don’t carry your baggage forwards. Don’t let it influence your next OSCE sessions.
- Remember 8 of 200 marks is 4%.

- **HAVE A PLAN -ANSWER IN POINT FORMS.**
- **YOU HAVE ONLY 5 mins!**
- **THIS IS AN OSCE NOT AN ESSAY TYPE QUESTION.**
- **DO NOT LET 4% SPOIL 96%!!**

ALL THE VERY BEST!

For accessing the full presentation (pdf version), go to:-
www.drsanjaydebakshi.org.