NUTRITION, FLUIDS AND ACID-BASE BALANCE FOR THE SURGICAL PATIENT

Dr Sanjay De Bakshi

MS; FRCS (Eng; Edin)

With permission & thanks from the PLEXUS APP

HOW IS NUTRITION ASSESSED?

TOPIC 1

- As patient of 53 years has come in for surgery for chronic pancreatitis. He is a an alcoholic and a smoker. You have been asked to assess him for impending surgery. He is 60 kgs and he was 65 kgs 6 months ago. He is 2 meters tall.
- Is the weight loss?

1. Mild
2. Moderate
3. Severe

TOPIC 2

- As patient of 53 years has come in for surgery for chronic pancreatitis. He is a an alcoholic and a smoker. You have been asked to assess him for impending surgery. He is 60 kgs and he was 65 kgs 6 months ago. He is 2 meters tall.
- Calculate his BMI and find out whether he is

1. Underweight
2. Normal
3. Overweight
4. Obese

NUTRITIONAL ASSESSMENT THE COMMANDMENTS

- History –including that of unintentional weight loss
- BMI.
- Vitamin deficiency.
- Cardiovascular – CCF/ High-output phase.
- Thyromegaly
- Neurologic – peripheral neuropathy , diminished reflexes, tetany, handgrip strength, getting up from a chair.
- Wound healing
- Antropometric data
  - Triceps skinfold thickness.
  - Mid-arm circumference
- Biochemical measurements

NUTRITIONAL RISK INDEX (NRI)

- The Nutrition Risk Index (NRI) was developed by the Veterans Affairs Total Parenteral Nutrition Cooperative Study Group originally for cancer populations . It is derived from the serum albumin concentration and the ratio of the actual to usual body weight.
- •NRI = [1.519 × serum albumin (g/L)] + [41.7 × (present weight/usual weight)]
- •The following risk groups were used:

1. -Not at risk: NRI >100
2. -Mild risk: NRI between 97.5 and 100
3. -Moderate risk: NRI between 83.5 and 97.5
4. -Severe risk: NRI <83.5

ANTHROPOMETRIC MEASUREMENTS

- Weight loss (over 6 months):
  - Mild (<5%)
  - Moderate (5%-10%)
  - Severe (>10%)
- BMI
  - BMI= weight(kg)/height(m)^2
  - BMI= weight(lb) x 703/height(in)^2

BMI	DEFn
<18.5	Underweight
18.5 – 22.9	Normal
23.0 – 24.9	Overweight
>25	Obese
ASIA-PACIFIC COHORT	

ANTHROPOMETRIC MEASUREMENTS

- SKIN FOLD THICKNESS (can be used to determine body density & percentage of body fat). SITES
  1. Triceps – midway between the acromion and the olecranon with arm held by the side.
  2. Chest/Pectoral – Diagonal fold between anterior axillary line and nipple Men ½ way Women $^1/_3$ way.
  3. Subscapular – Diagonal fold (45°) 1-2 cms below the inferior angle.
  4. Abdomen – Vertical fold 2cm to the right of the umbilicus.
  5. Suprailiac – Diagonal fold, natural angle of iliac crest, just superior in anterior axillary line.
  6. Thigh – Vertical fold on the anterior midline of thigh, midway between proximal border of the patella and inguinal crease

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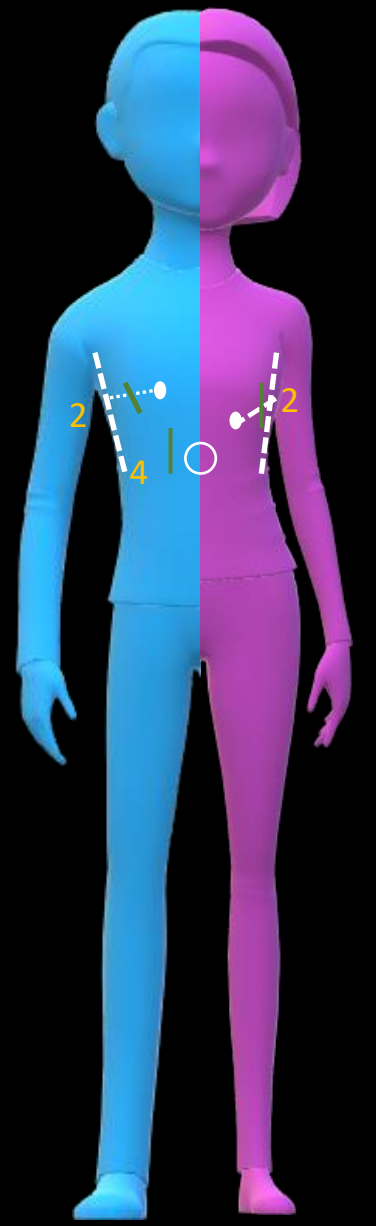

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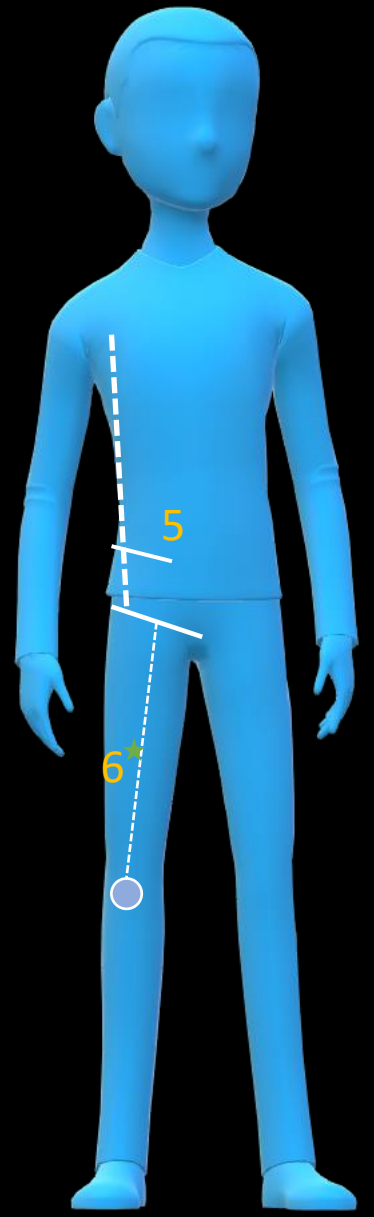


BODY DENSITY & PERCENTAGE OF BODY FAT

- Formulas for both men and women for body density
  - triceps, suprailiac & thigh for women &
  - chest, abdominal, thigh for men
- Body density used to measure percentage of body fat.

ASSESSMENT OF MUSCLE WASTING

CIRCUMFERENTIAL MEASUREMENTS

- Circumference measurement is performed with a measuring tape positioned perpendicular to the length of the arm, at the marked location.
- The arm is flexed 90º at the elbow.

OTHER AREAS

CLINICAL

1. TEMPLE.
2. CLAVICLE
3. SHOULDER
4. SCAPULA/RIBS
5. QUADRICEPS
6. INTEROSSEOUS - MUSCLE DORSUM OF THENAR AREA

FUNCTIONAL

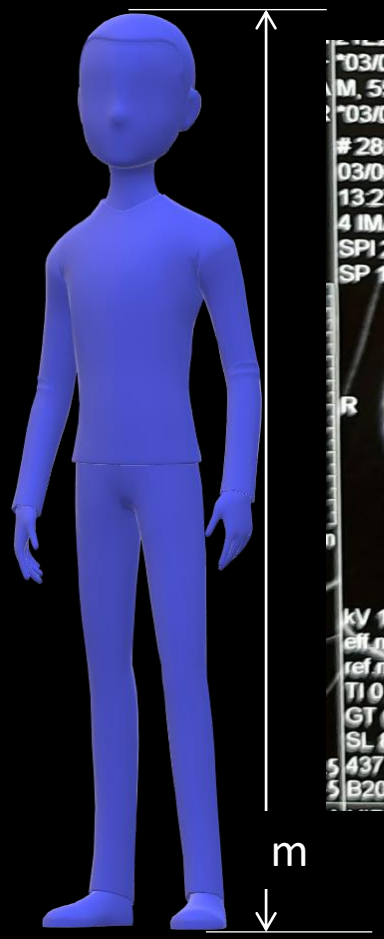
1. GRIP STRENGTH – USING A DYNAMOMETER.
2. RISING FROM CHAIR

GRADING FUNCTIONAL TESTS OXFORD SCALE

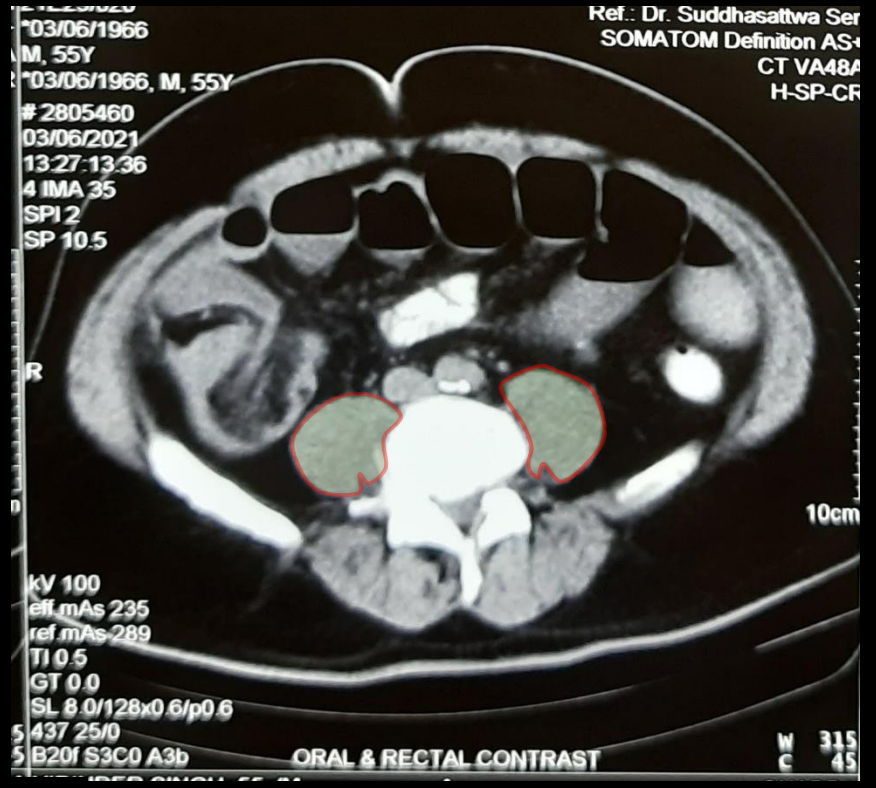
1. Flicker of movement
2. Through full range actively with gravity counterbalanced
3. Through full range actively against gravity
4. Through full range actively against some resistance
5. Through full range actively against strong resistance

USING A CT SCAN

- Cross-sectional areas of psoas muscle at the level of third lumbar vertebra were measured by computed tomography and normalized by the square of height to calculate
- Psoas muscle index
- (PMI, cm^2/m^2).



Compliments to Dr Suddhasattwa Sen
MS; MRCS; DNB (Gen & GI surg)



3.74 cm^2/m^2 for male individuals and
2.29 cm^2/m^2 for female individuals.

Masatsugu O et al. Analysis of the optimal psoas muscle mass index cut-off values, as measured by computed tomography, for the diagnosis of loss of skeletal muscle mass in Japanese people. https://doi.org/10.1111/hepr.13499

USING A CT SCAN

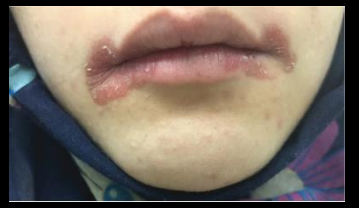
- Cross-sectional areas of psoas muscle at the level of third lumbar vertebra were measured by computed tomography and normalized by the square of height to calculate psoas muscle index (PMI, cm^2/m^2).

AGE GROUP	PMI (mm^2/m^2)	
	MALES	FEMALES
21-30	840.3	782.76
31-40	825.53	605.86
41-50	722.68	522.65
51-60	642.57	415.79
61-70	492.28	408.1

P. Lakshmi Prashanthi, Rajoo Ramachandran, Adhilakshmi, Prabhu Radhan, Venkata Sai. Standardization of PSOAS muscle index measurements using computed tomography. International Journal of Contemporary Medicine Surgery and Radiology. 2020;5(1):A169-A172.

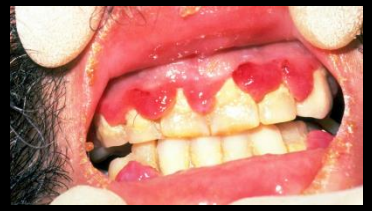
VITAMIN DEFICIENCY

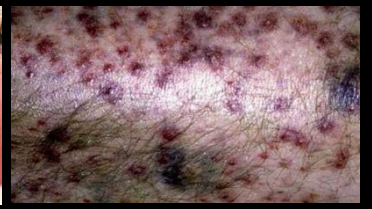
- Vitamin B ; riboflavin



https://www.rdhmag.com/

- Vitamin C (*Scurvy*)



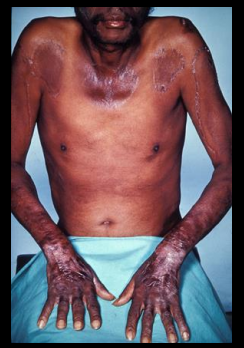


https://www.healthline.com/health/scurvy#symptoms

- Vitamin A night blindness

https://www.liberaldictionary.com/xerophthalmia/

- Vitamin B3 deficiency
  pellagra



https://en.wikipedia.org/wiki/Pellagra

BLOOD BIOCHEMISTRY

- Albumin – longest half life. • 18-20 days.
- Serum transferrin –mid-range half life. • 8 – 9 days.
- Serum pre-albumin (transthyretin) - shortest half life. • 2-3 days.
- CRP – to check why?

HOW IS NUTRITION TREATED IN THE PERIOPERATIVE PERIOD

TOPIC 3

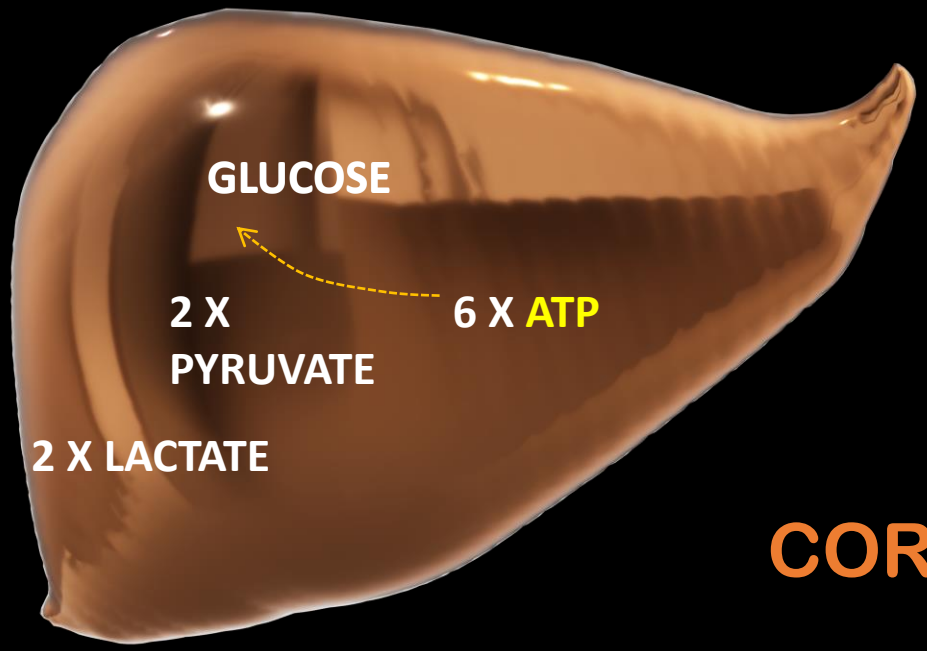
- As patient of 53 years has come in for surgery for chronic pancreatitis. He is a an alcoholic and a smoker. You have been asked to assess him for impending surgery. He is 60 kgs and he was 65 kgs 6 months ago. He is 2 meters tall.
- How much of caloric intake would you give him? How much of calories would he need?

1. 800kcal/day
2. 1000 kcals/day
3. 1500kcals/day
4. 2500kcals/day.

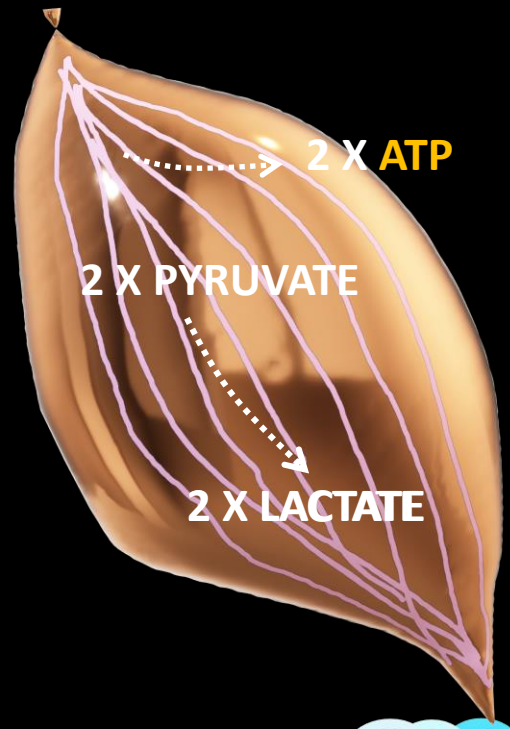
METABOLIC RESPONSE TO STARVATION

12 hours or less- all food from last meal absorbed.

- Plasma insulin levels fall and glucagon levels are elevated.
- Liver glycogen is therefore converted to glucose. (200 mgm approximately). Cerebral tissue, RBC, WBC, renal medulla can only utilize glucose for their needs)
- Muscle glycogen broken down to lactate, transported to the liver where it is converted into glucose (Cori cycle).


GLUCOSE
2 X
PYRUVATE
6 X ATP
2 X LACTATE

CORI CYCLE


2 X ATP
2 X PYRUVATE
2 X LACTATE

METABOLIC RESPONSE TO STARVATION

Prolonged fasting > 24 hours.

- Glycogen stores depleted, fresh source needed.
- Glutamine and alanine liberated from skeletal muscle (approximately 75 gm/24hours), transported to liver, where they are converted to glucose (gluconeogenesis). Can be avoided by providing exogenous glucose.

METABOLIC RESPONSE TO STARVATION

Further fasting (after 48-72 hours)

- Fat oxidation occurs to provide energy requirements.
- Fat is broken down to glycerol which is converted to glucose and fatty acids to be used as fuel by most tissues in body.
- Fatty acids converted in liver to ketones under influence of low insulin. These are utilized by the CNS as fuel.
- Conversion of T4 (inactive) to T3 (active) inhibited – causing reduced metabolism.

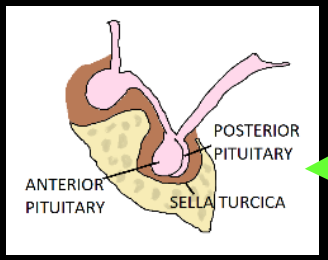
METABOLIC RESPONSE TO TRAUMA AND SEPSIS

- Both the intensity and the duration of response is directly proportional to the degree of injury.
- Genetic variability an important factor in the intensity of response.

MEDIATORS

INCREASED ADIPOCYTES
INCREASED LIPOLYSIS

ACTH; GH

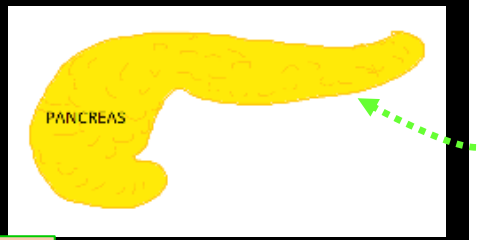


INCREASED HEPATIC LIPOLYSIS

ADRENALINE
CORTISOL

ACUTE PHASE HEPATIC PROTEIN SYNTHESIS

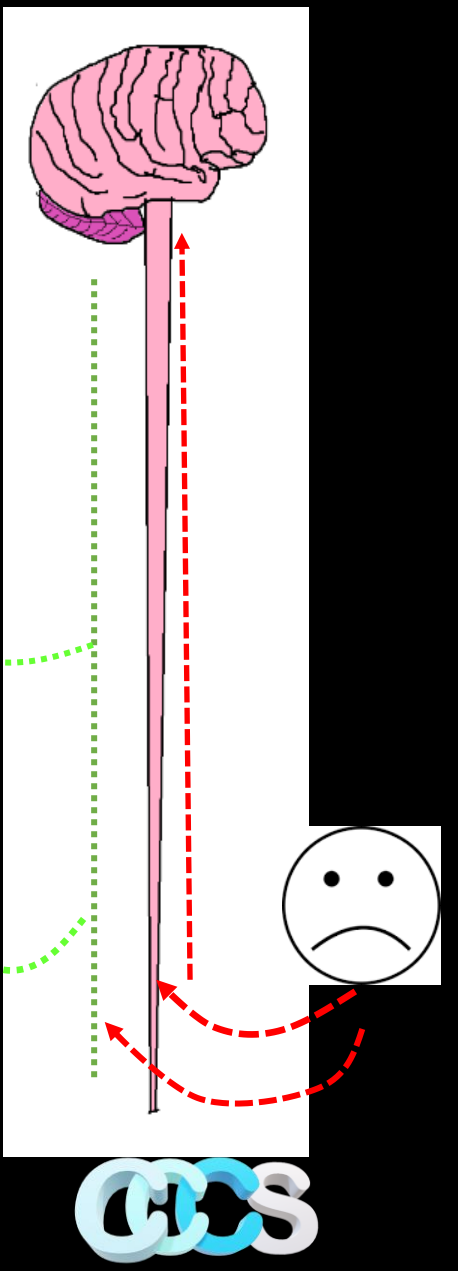
GLUCAGON
IL-1; IL-6; IL-8;
TNFα



PYREXIA
HYPERMETABOLISM

INSULIN; IGF -1;
TESTOSTERONE; T3

INNATE
IMMUNITY



MALNUTRITION UNIVERSAL SCREENING TOOL (MUST)

BMI

>20= 0

18.5-20 = 1

<18.5 = 3

WEIGHT LOSS IN LAST 6 MONTHS

<5% = 0

5-10% = 1

>10% = 2

ACUTE DISEASE EFFECT

No nutritional intake for the last 5 days.

0 =LOW - Routine clinical care

1 = MEDIUM - Observation.

2+= HIGH - Treatment

MUST

The MUST Report. Development and use of the 'malnutrition universal screening tool' (MUST) for adults. A report by the Malnutrition Advisory Group of the British Association for Parenteral and Enteral Nutrition. Report No. 152, 2003, ISBN 1 899467 70X).

Routine clinical care

- Repeat screening.
- Hospitals- weekly
- Care homes - monthly
- Community - evety year for special groups eg: - elderly

Observation

- Hospital & Care homes- documentation of dietary and fluid intake
- Community - frequent screening.

Treatment

- Enteral and Parenteral support in consultation with Dietician.

SPECIFIC REQUIREMENTS

- **ENERGY REQUIREMENTS**
- **For the stable patient it is usually 20 -30 kcal/kg/day. Usually works out to be 1300-1800 kcal/day.**

SPECIFIC REQUIREMENTS

CARBOHYDRATES

- Obligatory requirement (e.g. CNS; RBC) – 2gm/kg/day. (100 to 200 g/day)
- Maximum amount of glucose that can be oxidized – 4mg/kg/day. (Non-oxidized glucose converted to fat). Amount given during nutritional support should stay below this level.

SPECIFIC REQUIREMENTS

FAT

- Dietary fat = four long-chain fatty acids
  - Saturated – Palmitic (C16) ; Stearic (C18).
  - Unsaturated – Oleic (C18 with one double bond); Linoleic (C18 with two double bonds). – essential as they cannot be synthesized inside the body. (Soybean and Sunflower oil emulsions rich source).
  - Essential fatty acids – (100 -200 g/week).

SPECIFIC REQUIREMENTS

PROTEIN

- Stable patient without malnutrition/stress – 0.10-0.15g/kg/day.
- Hypermetabolic state – 0.20-0.25g/kg/day. (Maximum 14g/day)

SPECIFIC REQUIREMENTS

VITAMINS, TRACE ELEMENTS, AND MINERALS

- Water soluble vitamins – B & C. Requirements soften increased. Required for collagen formation and healing.
- Fat soluble vitamins A, D, E & K.- absorption compromised in obstructive jaundice and steatorrhoea.
- Sodium, potassium, phosphate, magnesium, zinc and iron are all essential and their supplements have to be met.

SPECIFIC REQUIREMENT IN CKD (non-dialysis)

- eGFR>60m - mL/min/1.73 m^2 SAME AS FOR GENERAL POPULATION
- eGFR < 60mL/min/1.73 m^2
- Daily protein input of 0.8/kg/day.
- Full of vegetables.
- BP
  - Hypertensive – Sodium < 2g/day.
  - Not hypertensive – Sodium < 2.3g/day.
- Potassium – guided by blood levels.
- Calcium < 1500 mg/day; Phosphorus - 0.8 to 1g/day.
- Calorie- 25 to 25 kcal/kg/day.
- Fat < 30% of daily intake; Saturated fats < 10%
- Dietary fibre 25 to 35g/day.

SPECIFIC REQUIREMENTS IN CLD
The Assessment.

CLD

CPT

CPT - C

CPT A or B

BMI

<18.5 kg/m^2

18.5 – 29.9 kg/m^2

>30 kg/m^2

HIGH RISK

OBESITY

ASSESS FOR SARCOPENIA

NUTRITIONAL ASSESSMENT
LIFESTYLE MODIFICATION

MUSCLE MASS
DEXA

APPROPRIATE NUTRITIONAL SUPPLEMENT

EASL Clinical Practice Guidelines on nutrition in chronic liver disease. Journal of Hepatology 2018

SPECIFIC REQUIREMENTS IN CLD

- Enteral route preferred. Naso-gastric/jejunal tube if non-bleeding varices. NO to PEG!!!!
- Parenteral route where enteral fails or portosystemic shunting present.
- COMPENSATED CLD
  - 25-35 kcal/kg body weight per day of nonprotein energy and
  - 1-1.2 g/kg body weight per day of protein or amino acids.
- COMPLICATED CLD
  - 35-40kcals/kg/day of non protein energy.
  - 1.5 g/kg/day of protein or amino acid.
- 4 to 5 meals a day including late night snack.
- Branched chain amino acids but extremely unpalatable. Enteral tube at night.
- Correct hypovitaminoses.
- Correct reduced trace mineral levels.

RE-FEEDING SYNDROME

- First discovered in far Eastern POWs when prisoners were fed after prolonged starvation it precipitated cardiac failure.

PROLONGED STARVATION

REDUCED INSULIN

INTRACELLULAR ELECTROLYTE LOSS (PO_4)

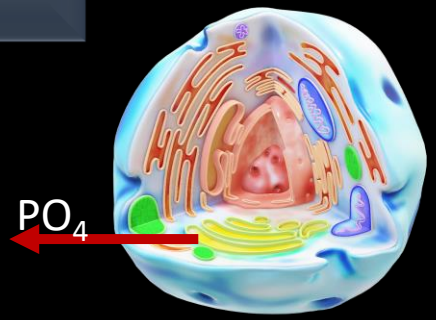
REFEEDING START OF CARBOHYDRATES

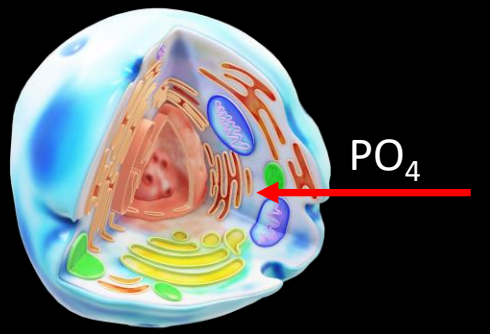
SECRETION OF INSULIN INCREASES

SECRETION OF INSULIN INCREASES

INTRACELLULAR SHIFT OF PHOSPHATES

PROFOUND HYPOPHOSPHATAEMIA





SERUM

RE-FEEDING SYNDROME

- Phosphate needed to generate Adenosine Triphosphate from Adenosine Diphosphate and Adenosine monophosphate.
- Once Serum concentrations are less than 0.50 mmol/L (N= 0.85-1.4 mmol/L)

- Produces:-
  - Rhabdomyolysis,
  - Leucocyte dysfunction,
  - Respiratory failure,
  - Cardiac failure,
  - Hypotension,
  - Arrhythmias,
  - Seizures,
  - Coma,
  - Sudden death.

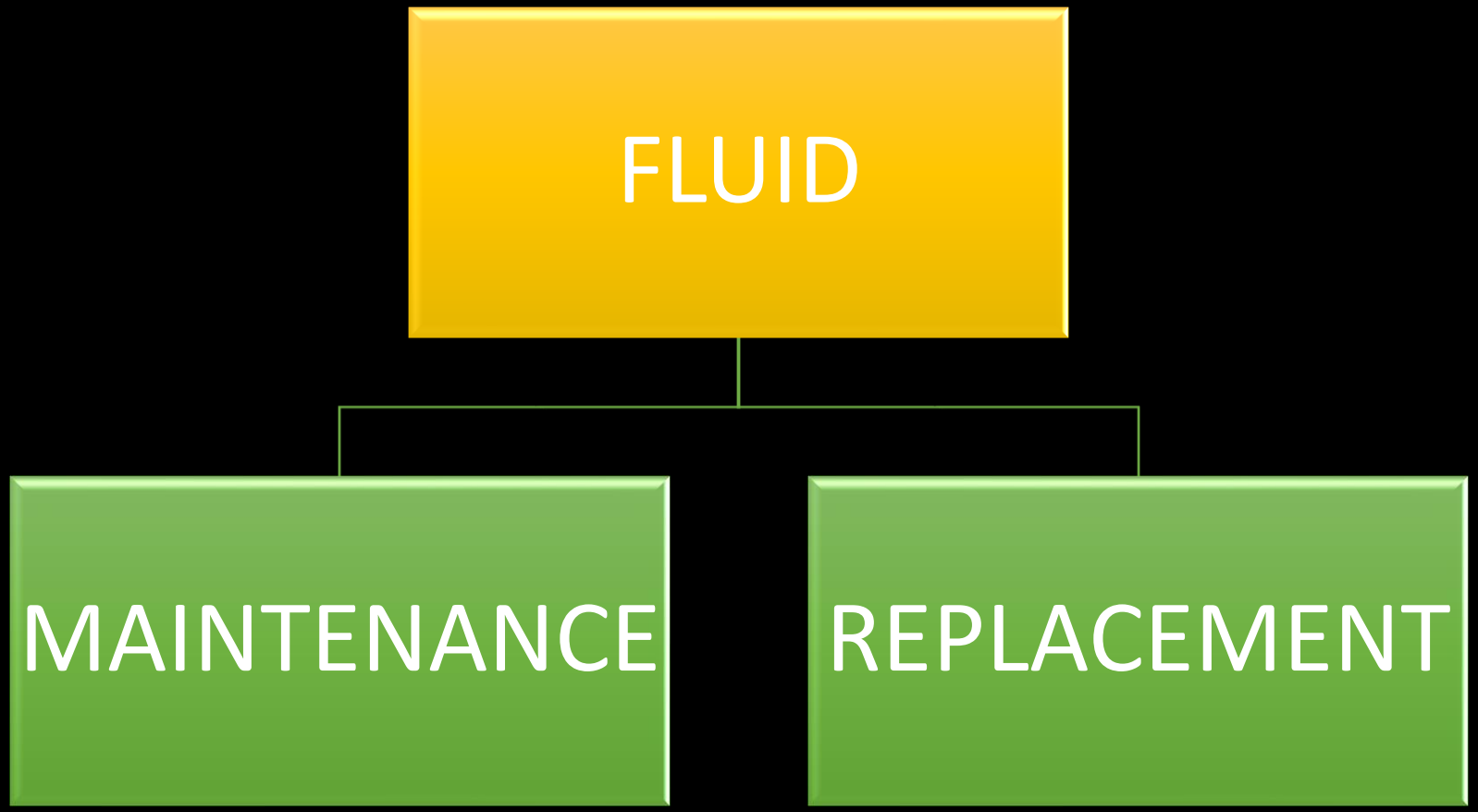
TOPIC 3

- 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.
- 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. (Closest answer)

1. 1000ml/24 hours
2. 1500ml/24 hours.
3. 2000ml/24 hours
4. 3000ml/24hours
5. 3500ml/24hours

HOW MUCH OF FLUID TO BE GIVEN?

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.

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

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

COMPOSITION OF IV FLUIDS

TYPE OF FLUID	OSMOLALITY	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	Phos phate 15		20

All in mmol/L except Osmolality inMOsm/kg

Buffers as follows:- Plasma – bicarbonates, haemoglobin

RL – lactate

Isolyte M– acetate

COMPOSITION OF FLUIDS

- 3260 ml/24 hours.
- WHAT FLUIDS?

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	133	0	55
5D	500	0		0	
ISOLYTE M	500	18		17.5	
5D	500	0		0	
NS + KCl 20ml	500 10	77 +20		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 RL or Plasma-Lyte.

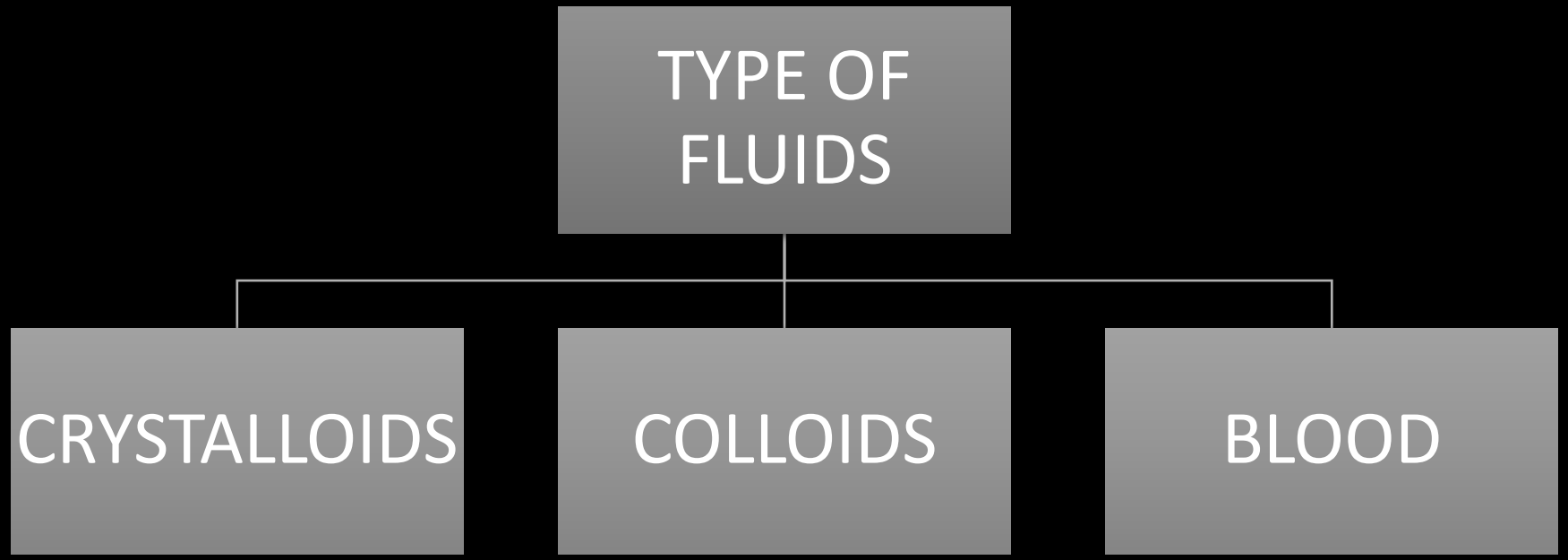
FURTHER READING

- Fluid management in
- Per-operative phase
  - ✓Pre-operative
  - ✓Intra-operative
  - ✓Post-operative
- Critical care
  - ✓Rescue
  - ✓Optimization
  - ✓Stabilisation.
  - ✓De-escalation.
- ERAS PROTOCOL

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.

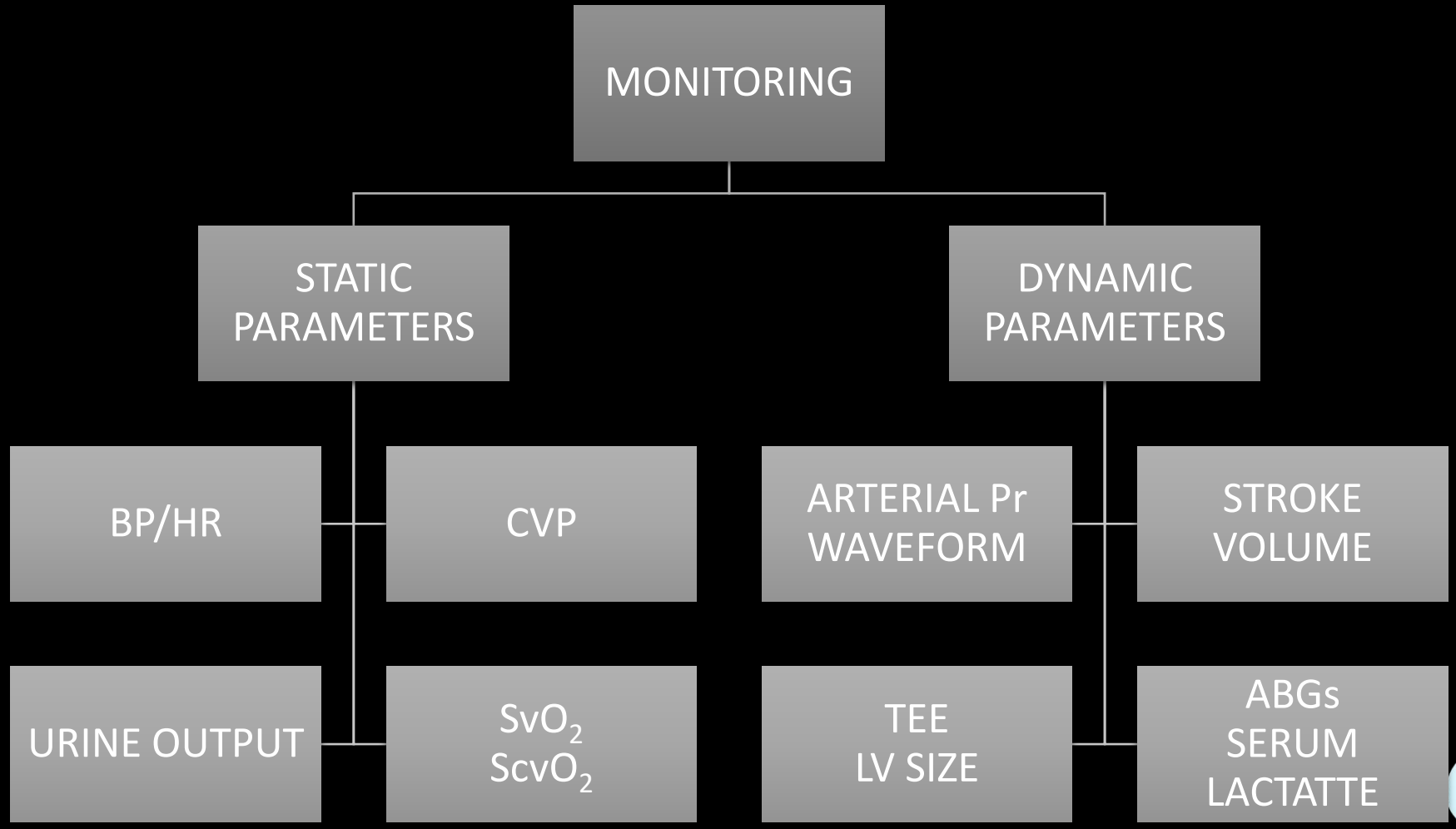
ANAESTHESIA RELATED FACTORS

- 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 to be used 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
    a) PPV
    b) Systolic pressure variations (SPV).

Goal-directed Strategy

- **INVASIVE MONITORING NEEDED.**
- **Commercially available device which provides an automatic calculation of**
  - **PPV (Pulse Pressure Variation).**
  - **SVV (Stroke Volume Variation).**
  - **SPV (Systolic Pressure Variation).**

Goal-directed Strategy

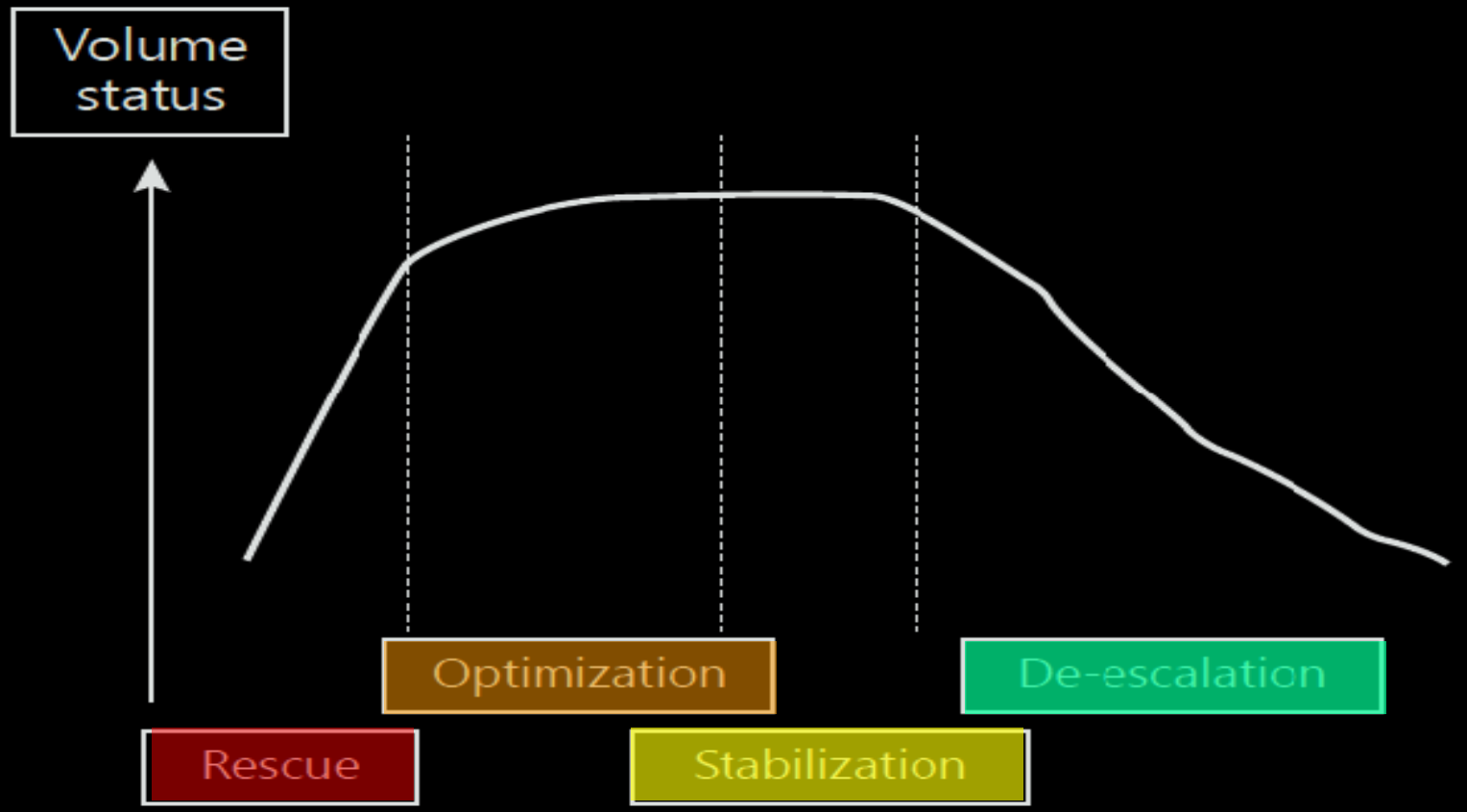
- Assessment of Fluids
- PPV or SPV
  - If respiratory variations in PPV or SPV > 10% patient presumed to be fluid responsive and fluid boluses of balance crystalloid solution 250ml) given.
  - Once change is <10% fluid is stopped.
- SV
  - SV value <10% fluid boluses are stopped.
- TEE
  - LV cavity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

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 TREATMENT

	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

Optimum monitoring	RESCUE	OPTIMISATION	STABILISATION	DESCELATION
Echo doppler				
CVP Monitoring				
ScvO2				
Cardiac output				
Fluid responsiveness				
Fluid challenge				

Acute Dialysis Quality Initiative. www.ADQI.com

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 Sydney Ringer discovered “Ringer’s” solution in 1882, when his technician replaced distilled water with London tap water containing the “correct" amount of Calcium.

Dr. Alexis Hartmann, an American Paeditrician, 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

TRANSFUSION OF BLOOD AND BLOOD PRODUCTS

- Must read.
- In most textbooks.
- More on the Android or IOS playstore
- CALCUTTA CHIRURGIAE COLLECTIVE PLEXUS APP.

TOPIC 4

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.

Infused 5% Dextrose only on Day 1.Plenty of plain water orally on Day 2. What is the cause?

1. Hyperkalaemia.
2. Undiagnosed previous epilepsy.
3. Dilutional hyponatraemia.
4. Hypoxia.
5. Pulmonary embolism.

TOPIC 4

- DILUTIONAL HYPONATRAEMIA
- 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.

- PLAN OF TREATMENT
- Withhold water.
- SLOW Infusion of 3 per cent Sodium Chloride.
- Intravenous administration of 20mgm of Furosemide.

Treatment of Hyponatraemia

In asymptomatic patients: When symptoms are absent, the focus of therapy should be on identifying and correcting the underlying cause of hyponatraemia.

- If **hypovolemic** on the basis of clinical assessment and urine sodium concentration, normal saline solution should be administered initially to correct the extracellular fluid volume deficit.
- If **hypervolemic**, salt and water restriction is key.
- If **euvolemic** and **hyponatremic**, therapy consists primarily of water restriction. When the cause of the syndrome of inappropriate ADH is unknown or not treatable, other methods can be used, including increased dietary protein and salt and use of urea, loop diuretics and, rarely, demeclocycline hydrochloride (Declomycin).

Treatment of Hyponatraemia

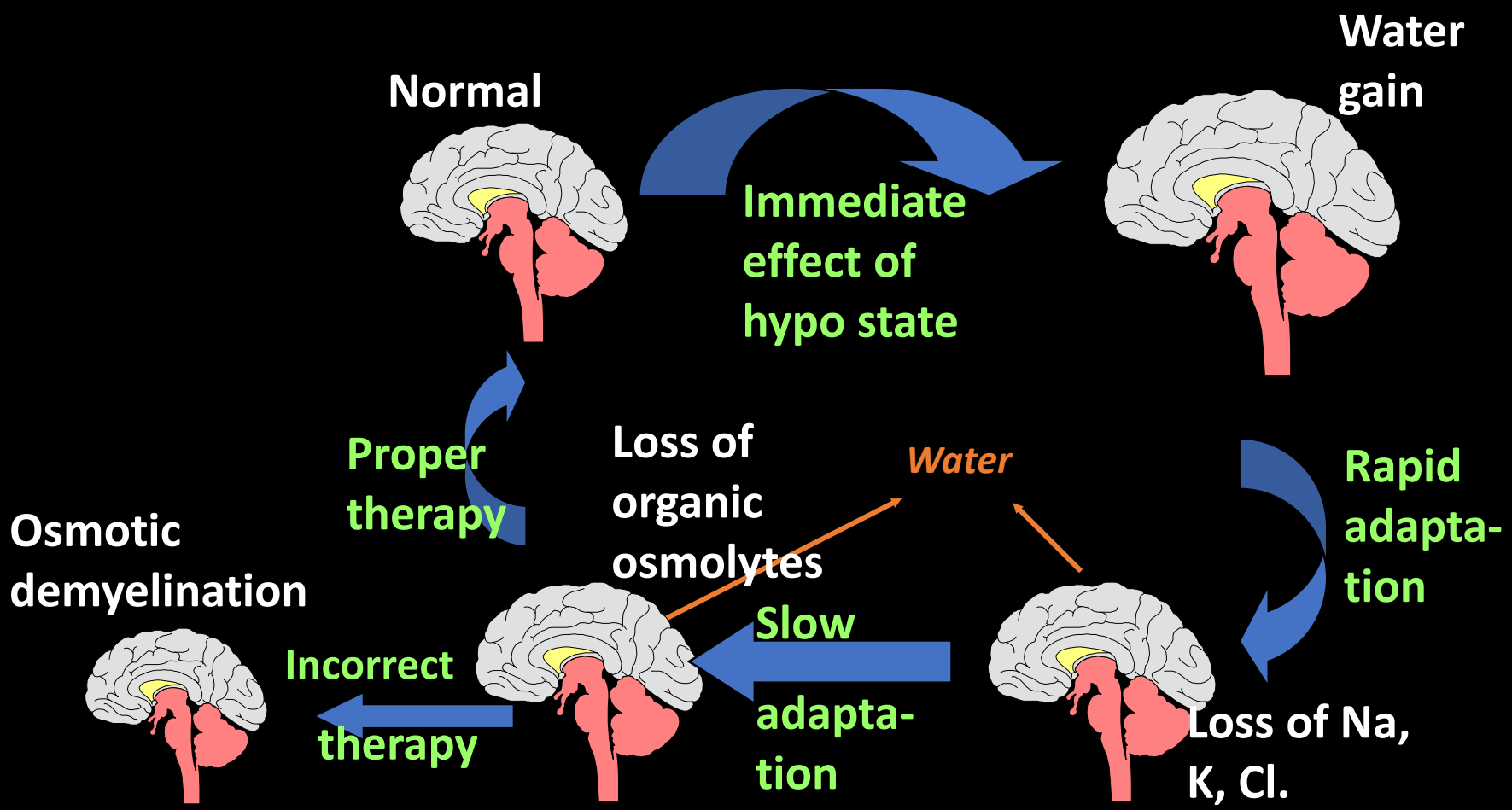
In symptomatic patients: Patients with acute symptomatic hyponatremia are candidates for aggressive treatment

- Hyponatremia can be corrected with administration of hypertonic saline solution (3%) at a rate of about **1 mL/kg per hour**. A loop diuretic may be added to enhance water excretion if urine osmolality is greater than 300 mOsm/kg.
- The serum sodium concentration should be raised no more than **25 mEq/L in the first 48 hours**, at a rate of no more than **2 mEq/L per hour**, and the target goal should be **120 to 125 mEq/L**.

With use of this combination therapy, sodium lost in the urine is replaced with an equal amount of sodium in a smaller volume.

Treatment with hypertonic saline solution is advocated only for patients with severe hyponatremia who have profound neurologic symptoms.

Effect of hyponatraemia and its correction



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.

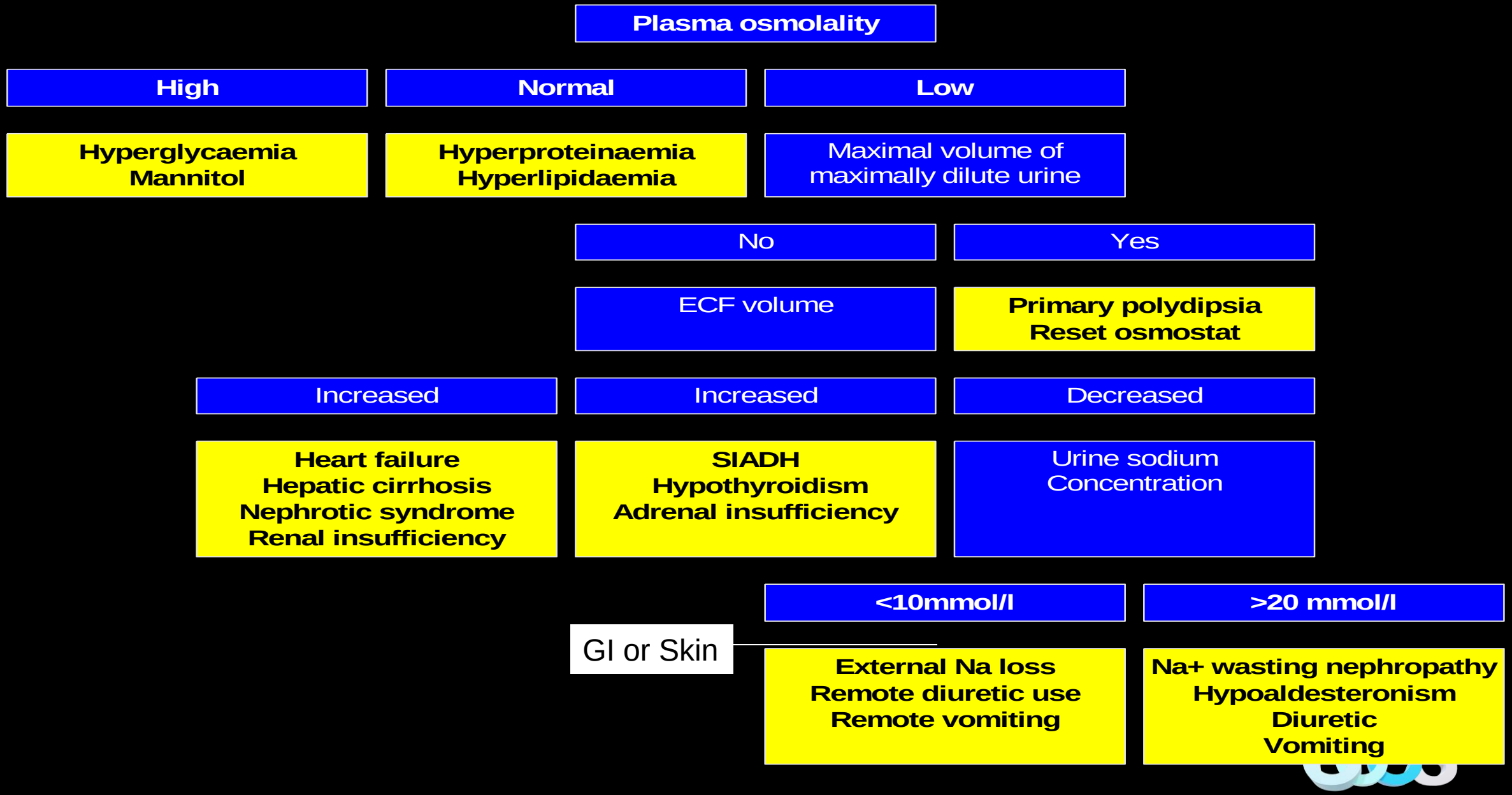
Total body water (TBW) decreases; total body sodium (Na^+) decreases to a greater extent. The extracellular fluid (ECF) volume is decreased.

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

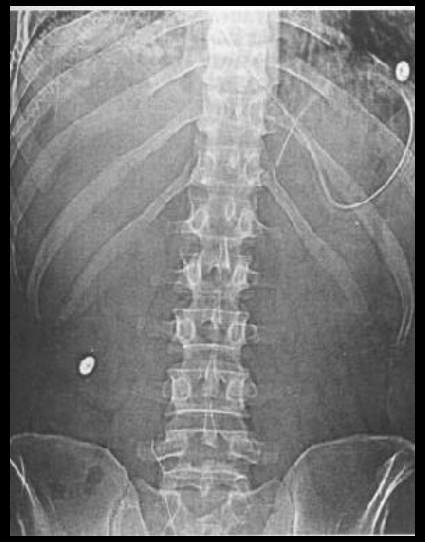


CASE CAPSULE

?

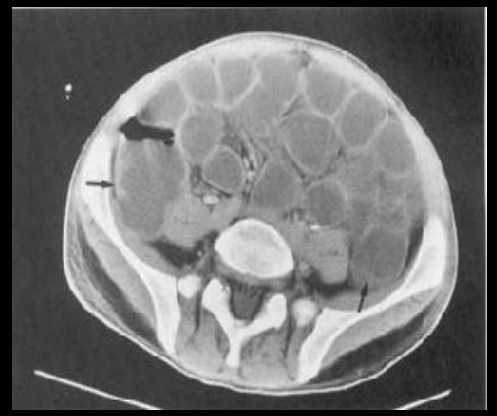
- An elderly lady of 63yrs. Undergoes a difficult resection- anastomosis for a gangrenous segment of small intestine, which was incarcerated under a post-operative band.
- Her abdomen is distended, she is obtunded, and her bowel sounds are absent.
- The tongue is red and swollen, skin turgor is diminished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

CASE CAPSULE



Straight X-ray

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



CT Scan

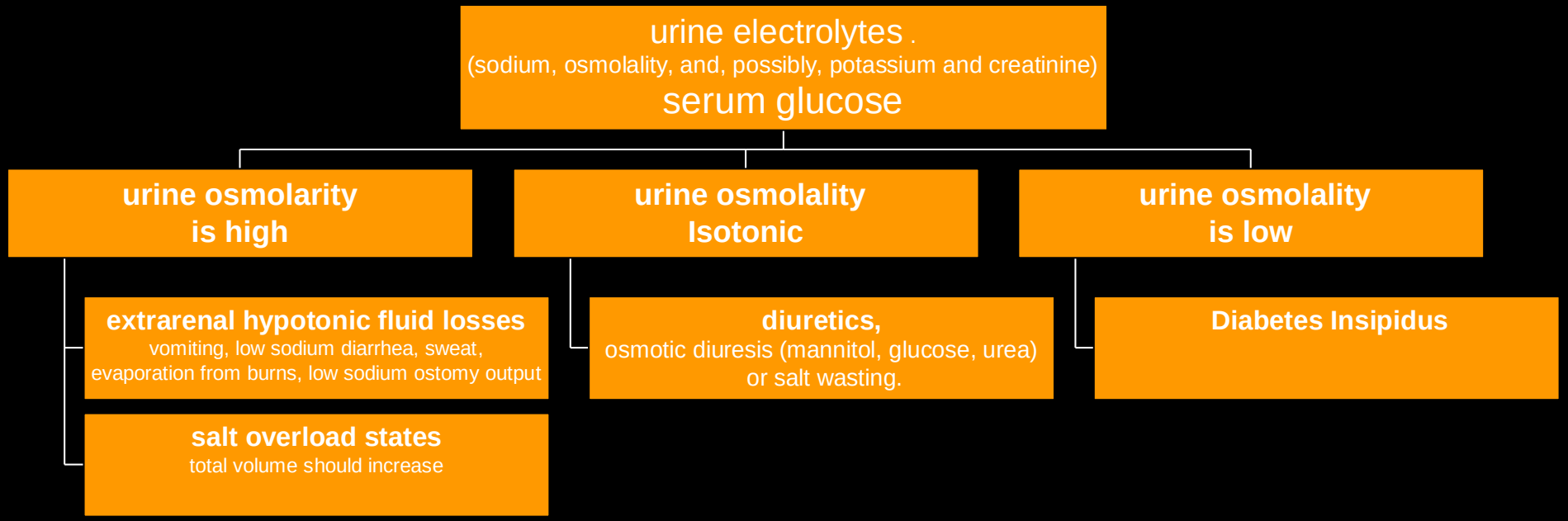
HYPERNATRAEMIA DUE TO SODIUM-POOR FLUID LOSS

TYPES OF HYPERNATRAEMIA

HYPERNATRAEMIA

Hypovolemic hypernatremia	Hypervolemic hypernatremia	Euvolemic hypernatremia
water deficit > sodium deficit	sodium gains > water gains	sodium gains = water gains
Na H_2O	Na H_2O	Na H_2O

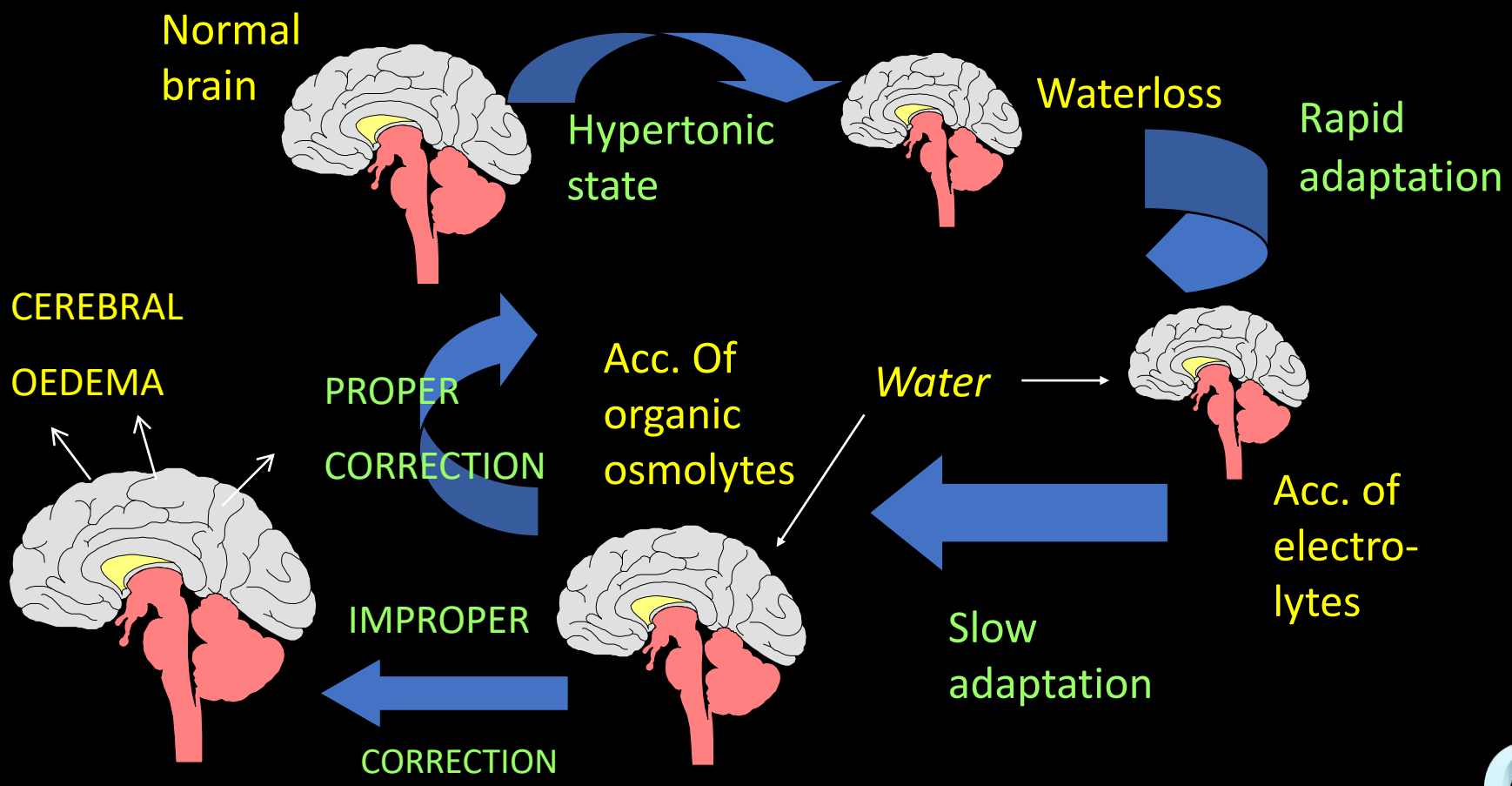
HYPERNATRAEMIA



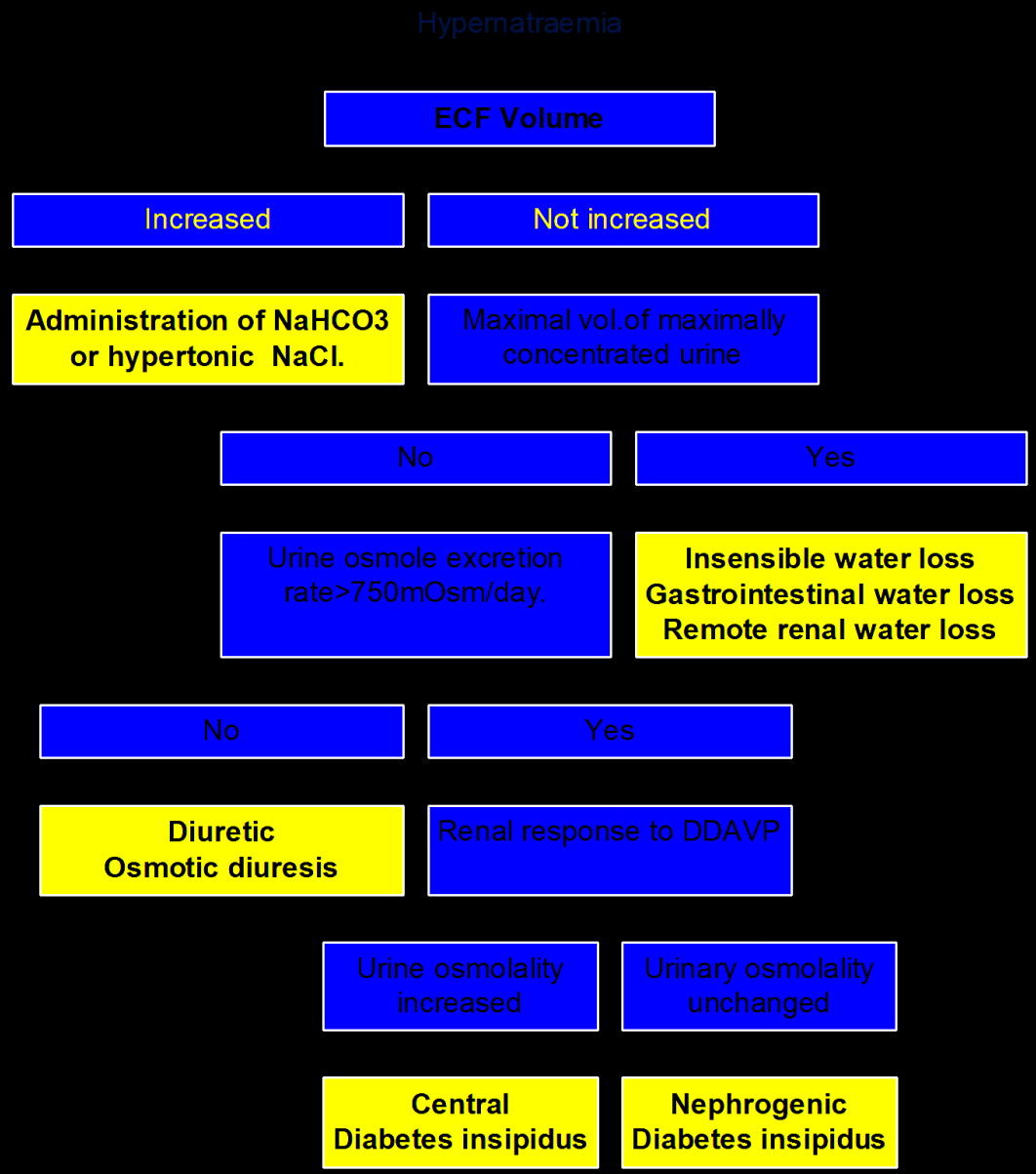
THUMB RULE:-

- 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):

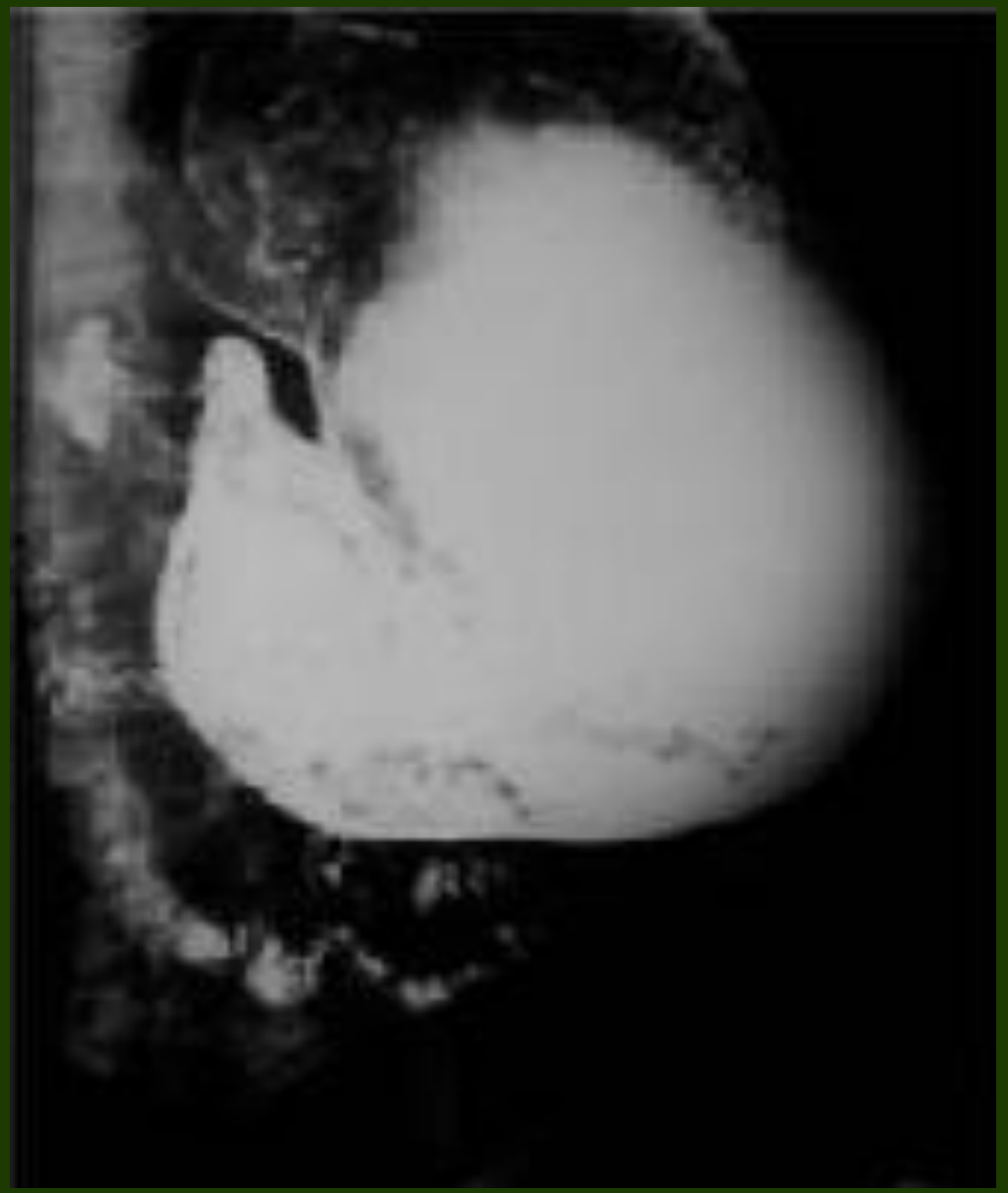
  Water deficit = total body water (serum sodium concentration ÷ 140 - 1)

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.

TOPIC 5

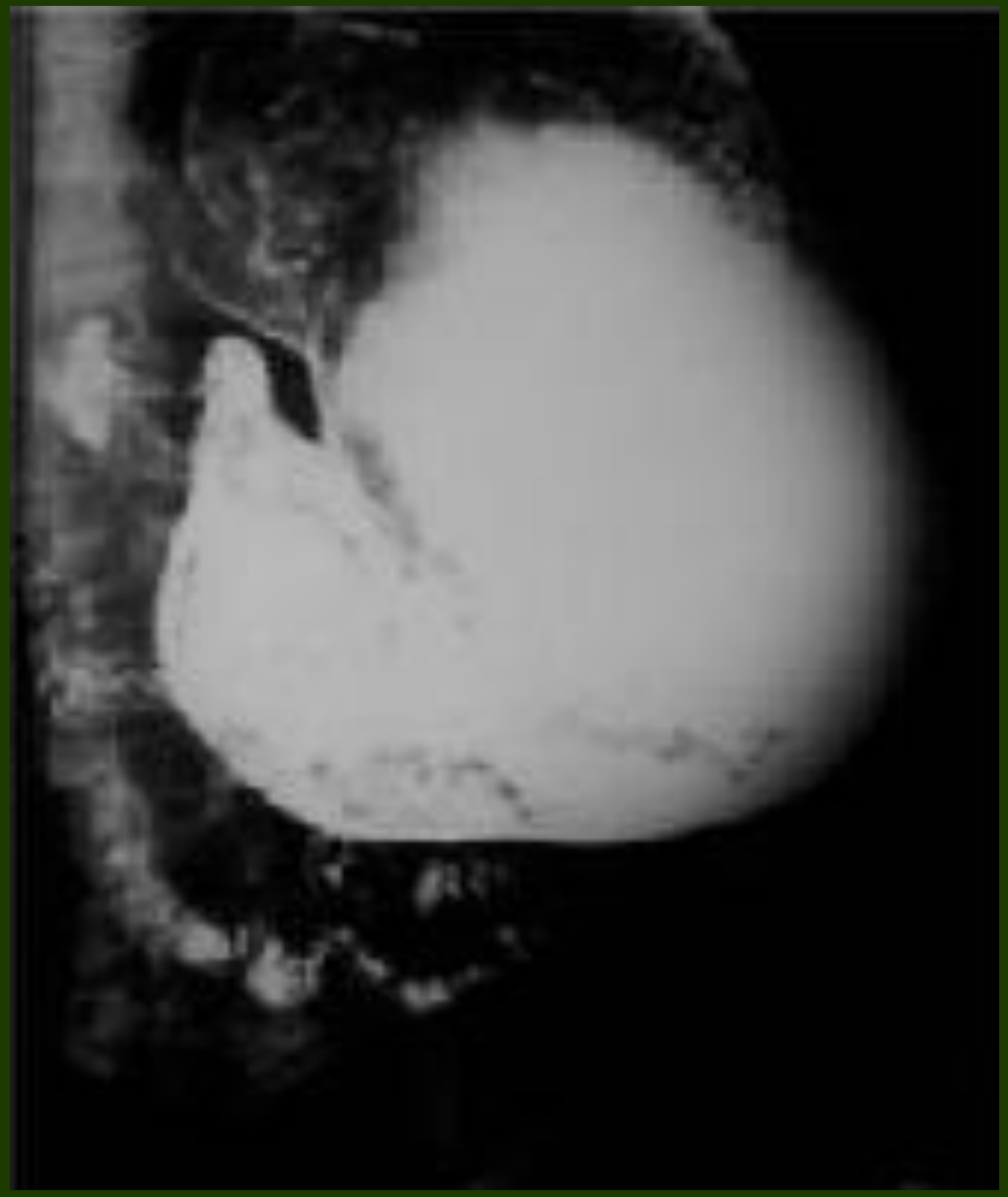
- A 62 year old man, presents with painless,profuse projectile vomiting containing old food material.
- No history of any previous surgery, he complains of a long standing, mild
- Epigastric pain.
- On Examination; the patient had a BP of 100/60;Pulse110/min. Eyes shrunken,
- Decreased skin turgor, slow 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-



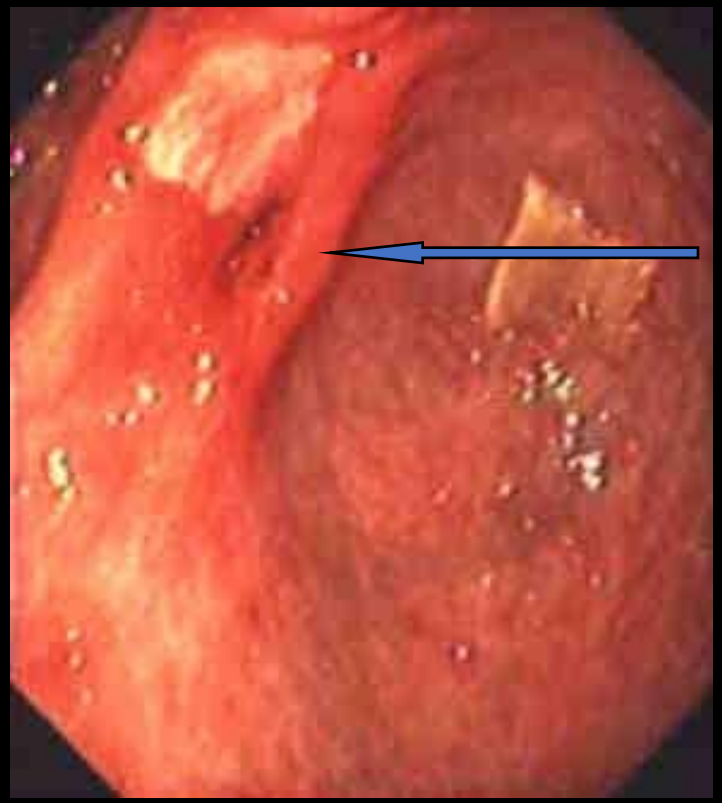
TOPIC 5

- Serum abnormalities you would anticipate for this condition are all except–

1. Hyponatraemia.
2. Hyperchloraemia.
3. Hypokalaemia.
4. Metabolic acidosis.
5. Hypoalbuminaemia.



TOPIC 5



Upper GI Endoscopy Revealed the following finding.

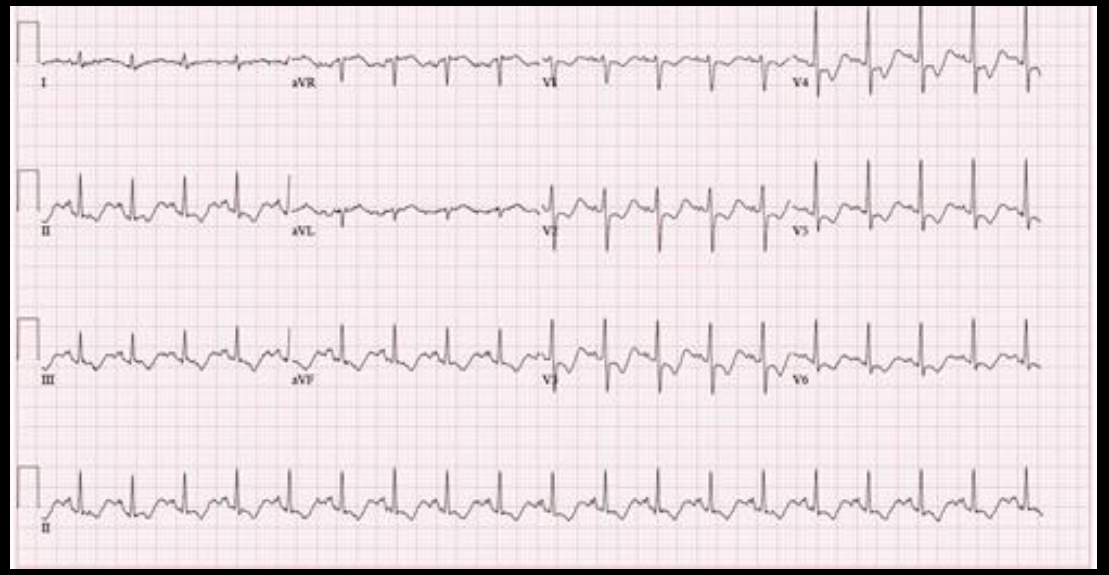
BLOOD BIOCHEMISTRY

- 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

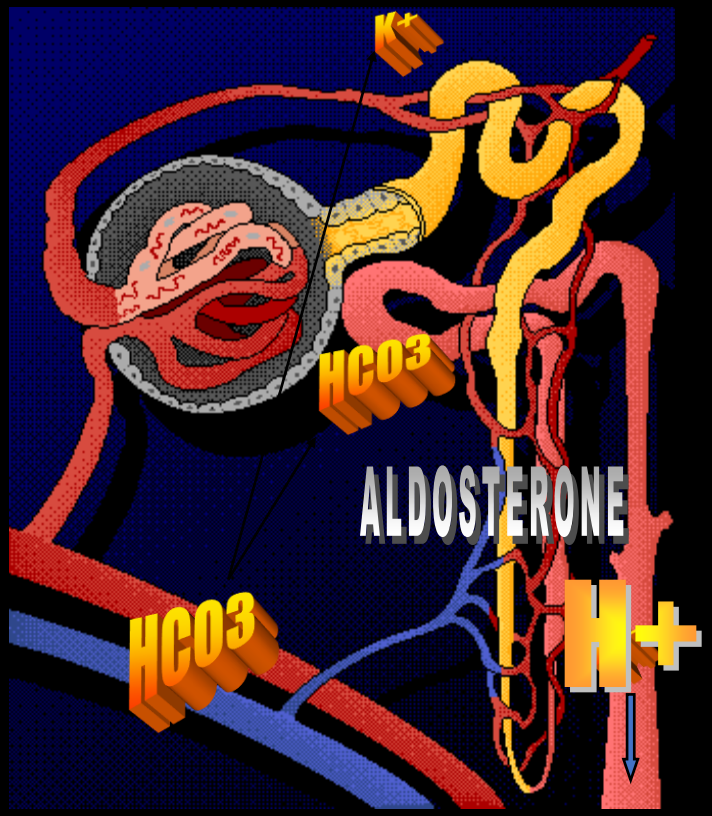
- GOO with-
  - ➢Hyponatraemia.
  - ➢Hypokalaemia.
  - ➢Hypochloraemia.
  - ➢Hypoalbuminaemia.
  - ➢Metabolic Alkalosis.

  - ➢ECG changes



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.

Pathophysiology 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

oRenal tubular acidosis

oHyperaldosteronism

oMagnesium depletion

oLeukemia (mechanism uncertain)

oVomiting or nasogastric suctioning

oDiarrhea

oEnemas or laxative use

oIleal loop

HYPOKALAEMIA

- Renal losses
- Medication effects
- GI losses
- Transcellular shift

oDiuretics (most common cause)

oBeta-adrenergic agonists

oSteroids

oTheophylline

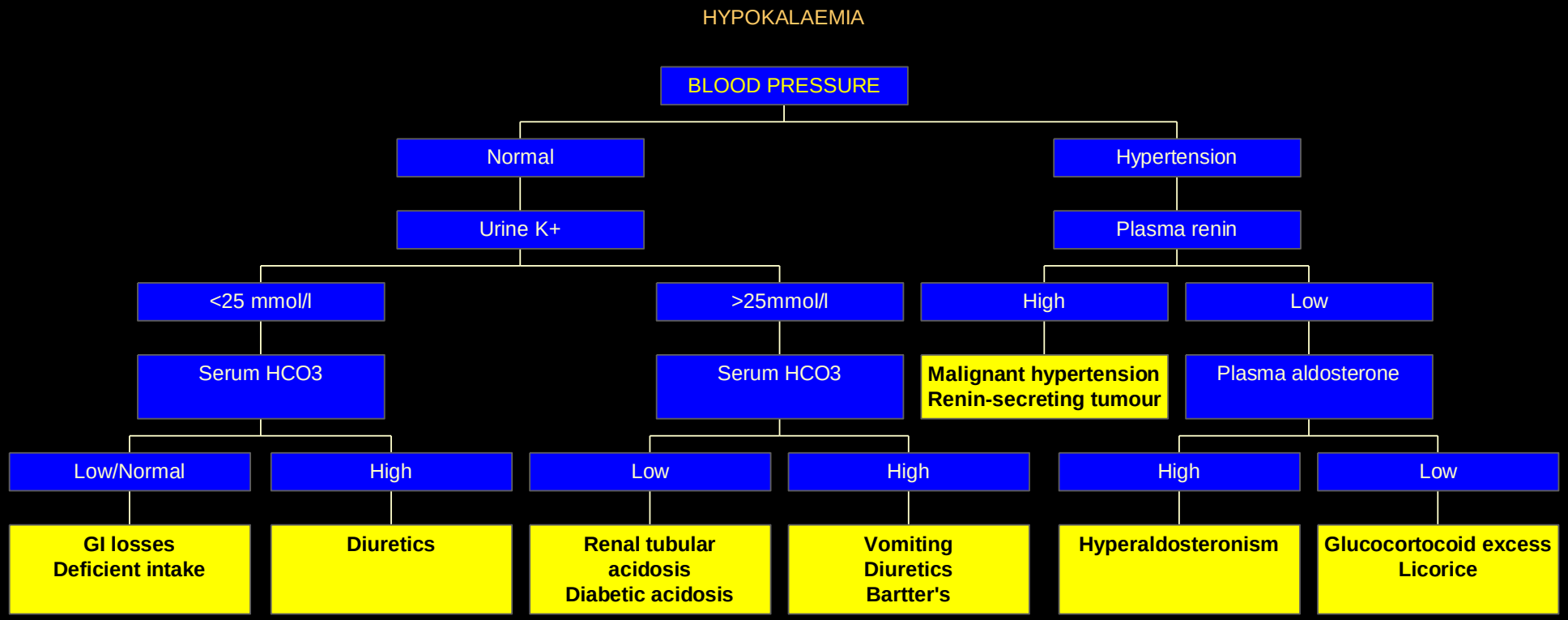
oAminoglycosides

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%)

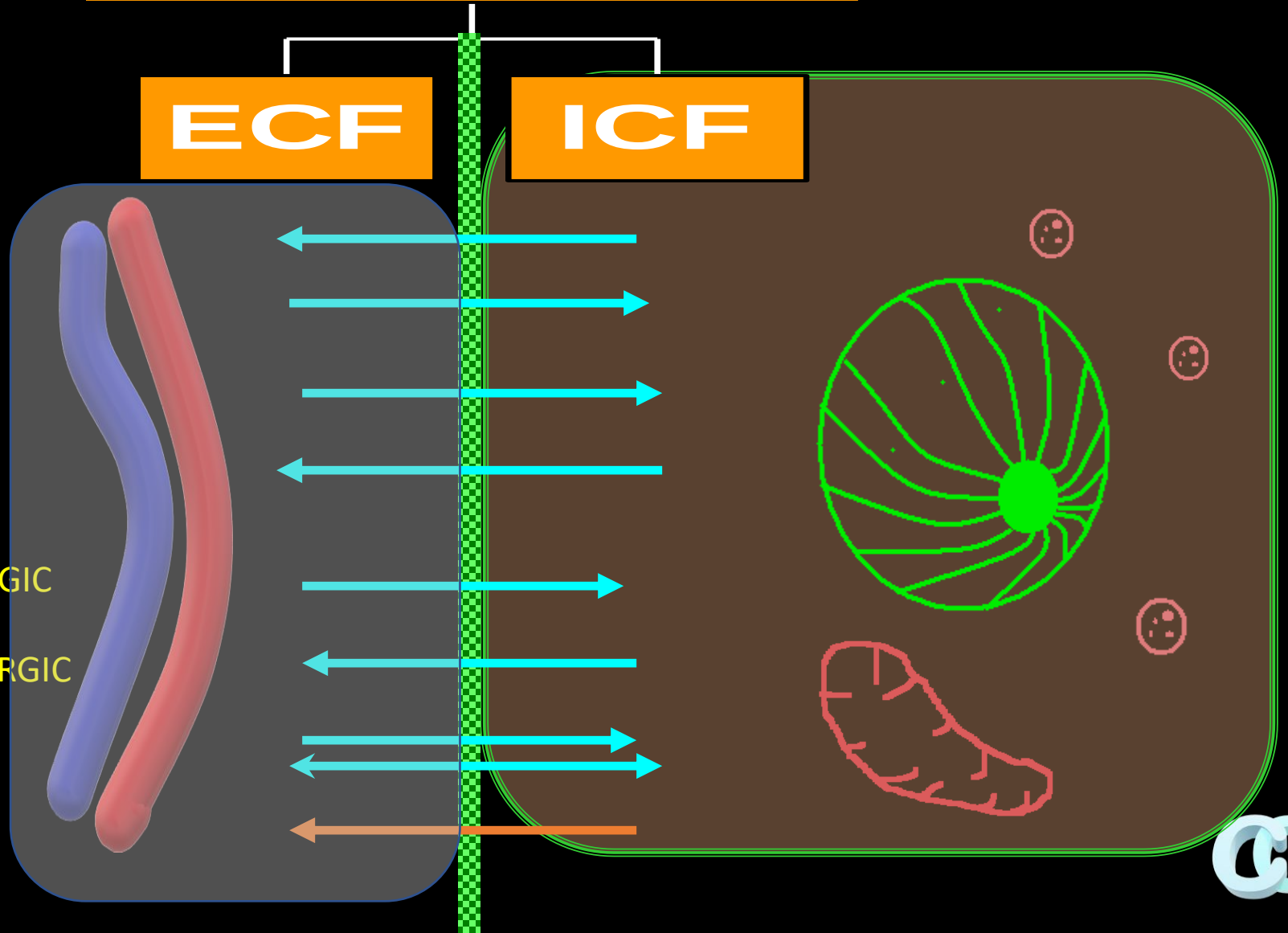
LOSS

URINE (90-95mEq/D)

STOOL (5-10mEq/D)

SWEAT (<5mEq/D)

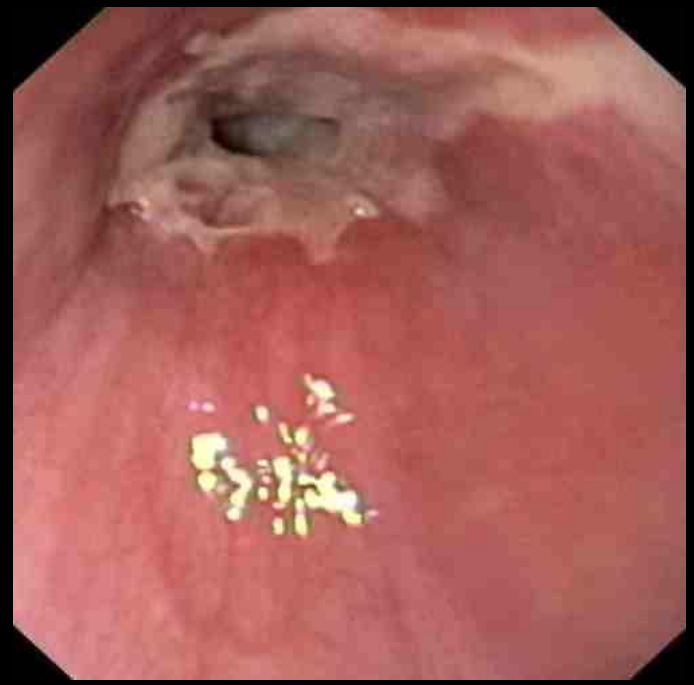
POTASSIUM BALANCE



Treatment of Hypokalaemia

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

- use oral K^+ supplements, at least 80mmol/24 hours
- normal maximum daily oral dose is 100mmol/l
- K^+ must be closely monitored and supplements stopped when $K^+ > 4.0$ mmol/l



- NEVER AS ORAL POTASSIUM TABLETS
- may cause nausea, vomiting and GI ulceration

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

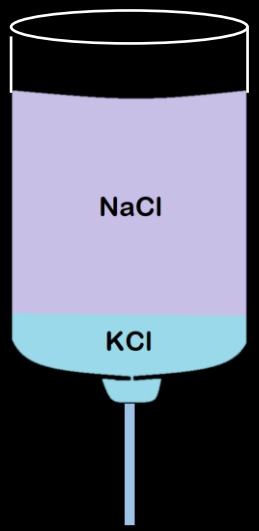
Maximum Concentration

- 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!

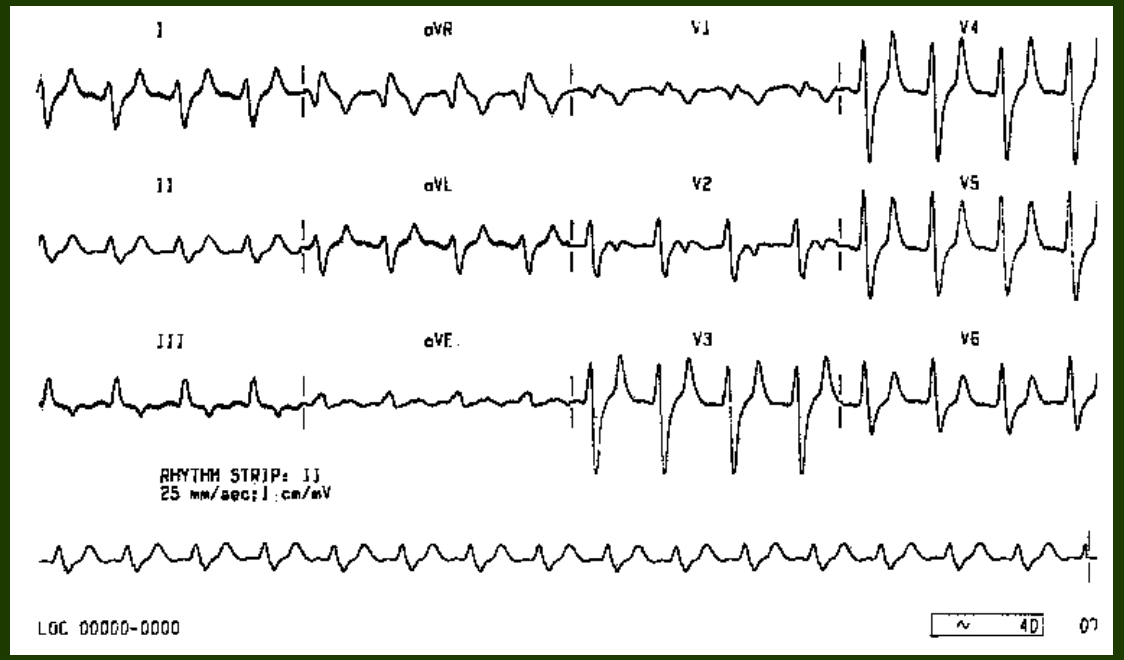
Strong Potassium Chloride Solution:

- 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)



TOPIC 6

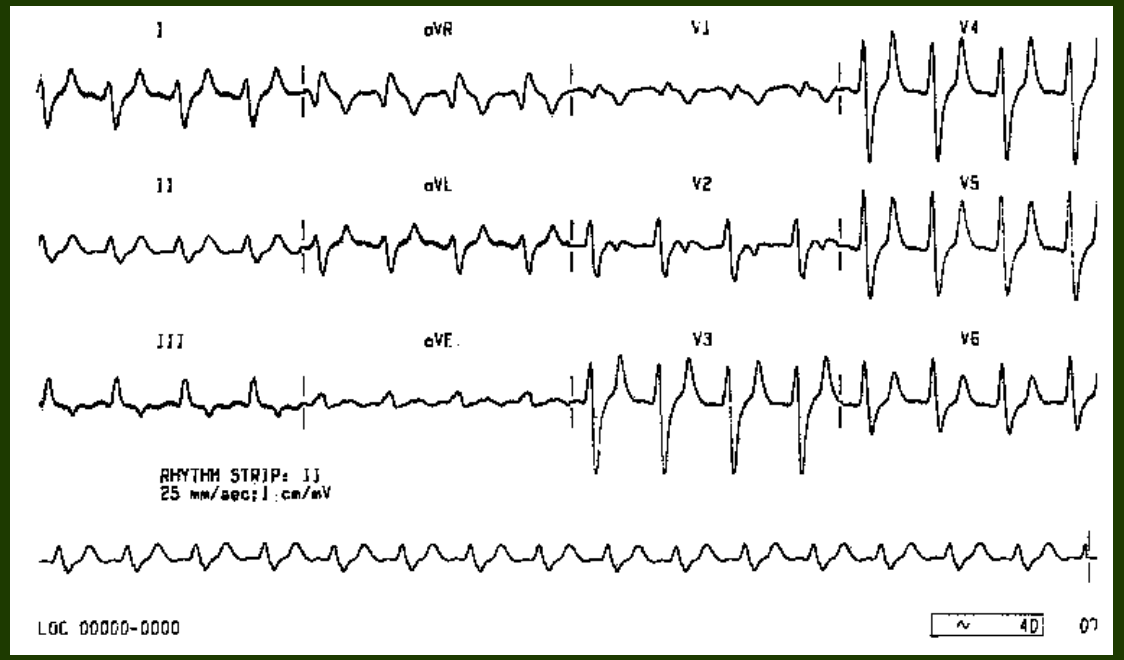
- An 62 yrs old farmer, sustained a crush injury to both his legs due to a tractor injury
- He was admitted at a peripheral hospital for 3 days before being transferred to the referral center.
- On examination he was found to have a thready irregular pulse, hypotension, was oliguric.
- The crush injury 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 relevant history was that of long standing small joint arthritis for which the patient had been on NSAIDS.
- He has brought along an ECG with him:-



TOPIC 6

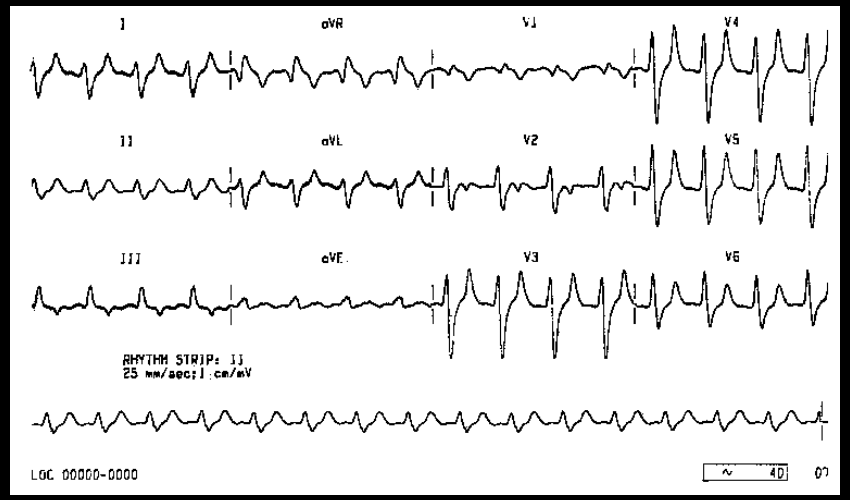
- He has brought along an ECG with him:-
- The most likely critical diagnosis which need immediate treatment is-

1. Sepsis.
2. Compartment syndrome.
3. Haemorrhage.
4. Hyperkalaemia.



TOPIC 6

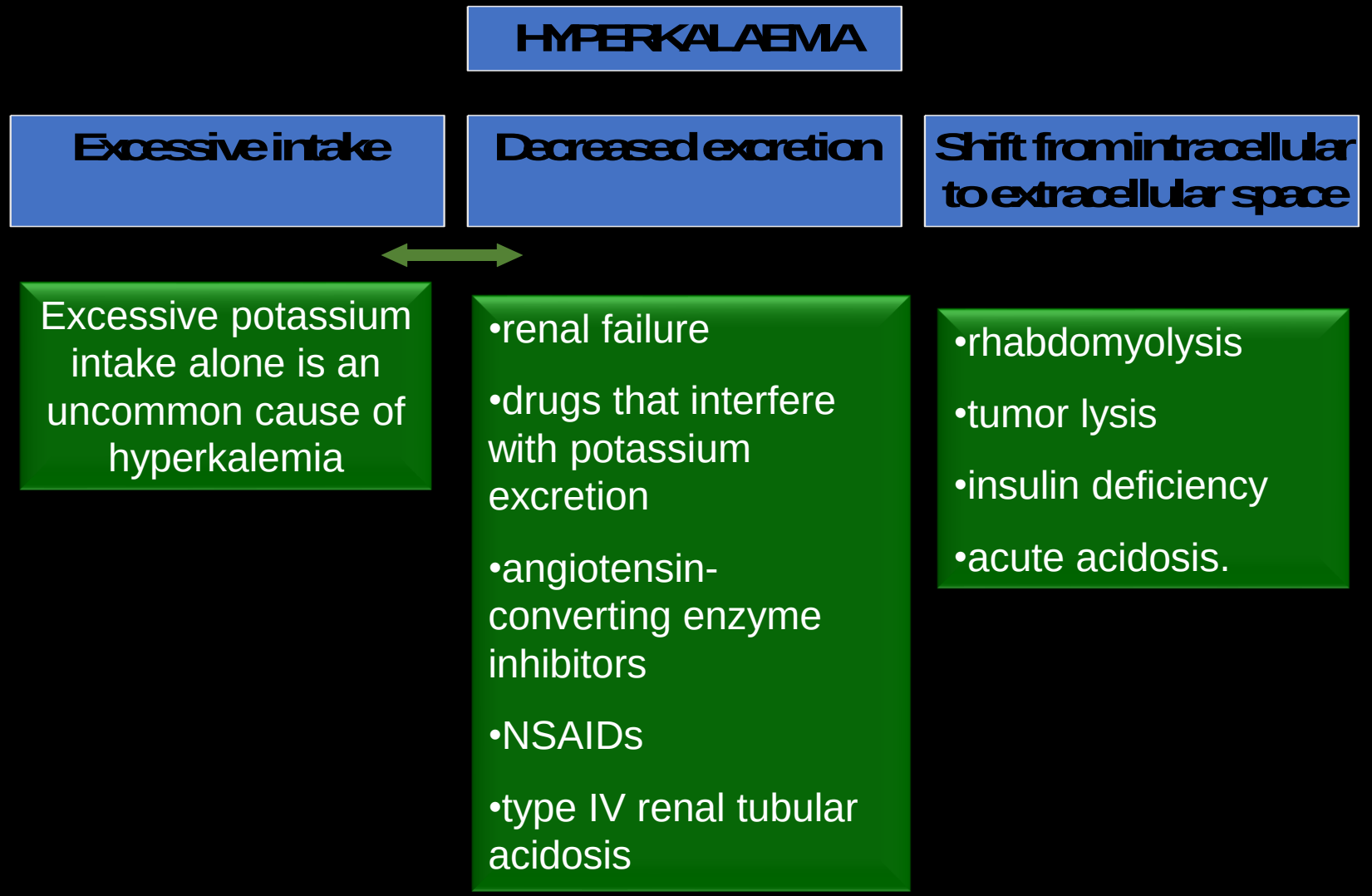
ECG



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

Peaked T waves
P wave widening/flattening, PR prolongation
Bradyarrhythmias: sinus bradycardia, high-grade AV block with slow junctional and ventricular escape rhythms, slow AF
Conduction blocks (bundle branch block, fascicular blocks)
QRS widening with bizarre QRS morphology.
WORSENING HYPERKALAEMIA
Development of sine wave appearance (pre-terminal rhythm)
Ventricular fibrillation
PEA with bizarre, wide complex rhythm
Asystole

CAUSES OF HYPERKALAEMIA



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

TOPIC 7

- 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?

1. Metabolic acidosis.
2. Metabolic alkalosis.
3. Respiratory acidosis.
4. Respiratory alkalosis.

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

TOPIC 8

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

1. Low.
2. Normal.
3. High.

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

TOPIC 9

- A 44 year old man was admitted with a 2 day history of acute gastroenteritis leading to severe diarrhoea.
- Is the compensation adequate?

1. YES.
2. NO.

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

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,
- HCO3 = 16
- Na= 134
- Cl= 108

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.
- 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 ↓

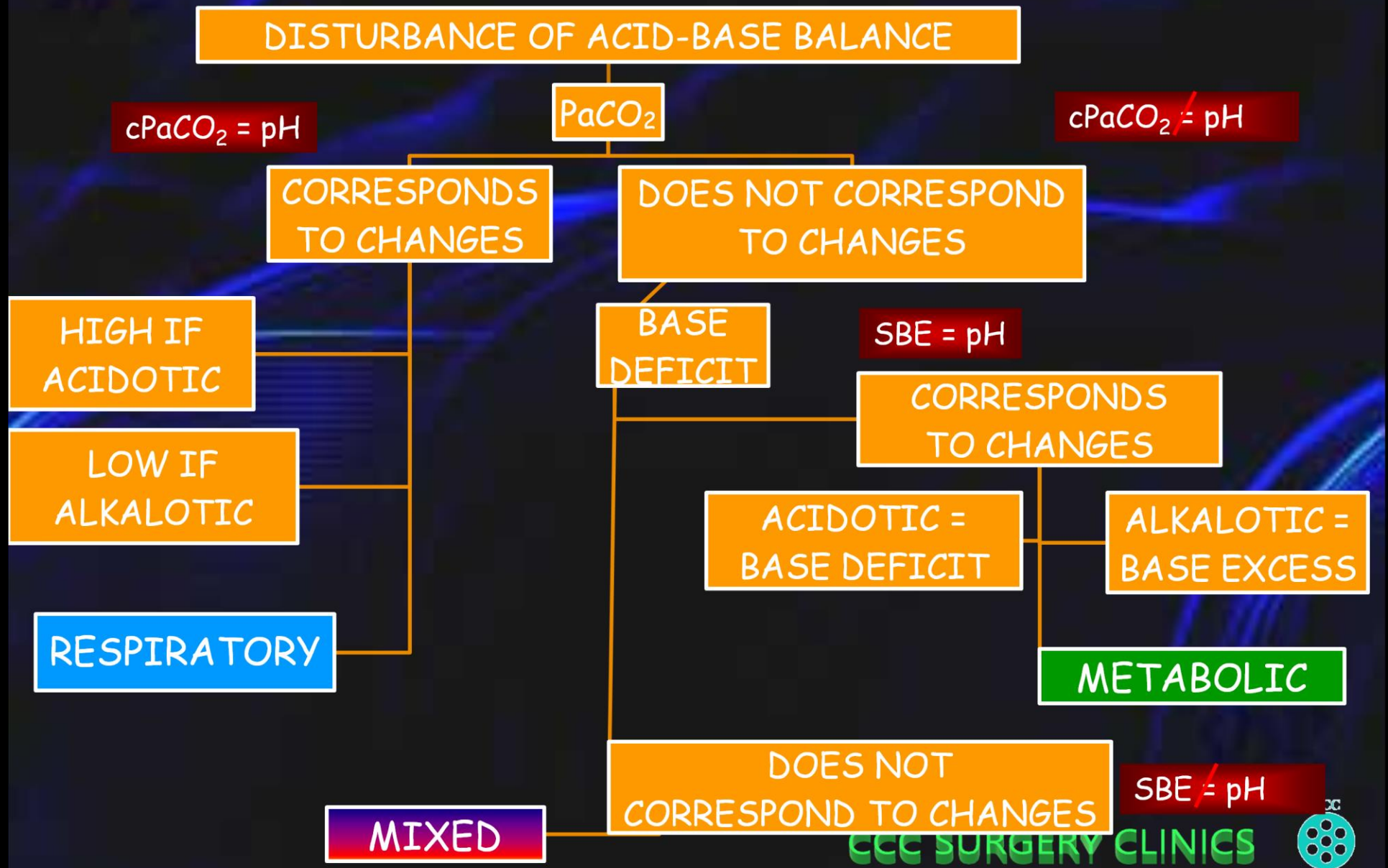
INTERPRETATION OF BLOOD GASES

'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 = $(Na^+ + K^+) - (HCO3^- + Cl^-) = 12\text{-}16_{mEq/l}$

Anion Gap = $(Na^+) - (HCO3^- + Cl^-) = 8\text{-}12_{mEq/l}$

Low Anion Gap<6mEq/L
Hypoalbuminaemia
Monoclonal protein

ANION-GAP CHANGES

Anion Gap = $(Na^+ + K^+) - (HCO3^- + Cl^-) = 12\text{-}16_{mEq/l}$

Anion Gap = $(Na^+) - (HCO3^- + Cl^-) = 8\text{-}12_{mEq/l}$

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 = $(Na^+ + K^+) - (HCO3^- + Cl^-) = 12\text{-}16_{mEq/l}$

Anion Gap = $(Na^+) - (HCO3^- + Cl^-) = 8\text{-}12_{mEq/l}$

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.

ASSESSMENT OF ACIDOSIS

DELTA RATIO?

- △ANION GAP = anion gap- 12 (the calculated anion gap minus the normal anion gap)
- △HCO_3 GAP = 24 – $HC0_3$ (to assess the body's ability to change HCO_3- in response to a metabolic acid. In cases with a pure anion gap metabolic acidosis, the rise in anion gap from 12 should equal a fall in HCO_3- from 24)

DELTA RATIO

$$\text{DELTA RATIO} = \frac{\text{ANION GAP} - 12}{24 - HCO_3^-}$$

DELTA RATIO

WHEN TO USE

- Can check delta ratio in the presence of a high anion gap metabolic acidosis (HAGMA) to determine if it is a ‘pure’ HAGMA or
- if there is coexistant normal anion gap metabolic acidosis (NAGMA) or metabolic alkalosis.

DELTA RATIO

< 0.4

Hyperchloraemic normal anion gap metabolic acidosis (NAGMA)

the reason here is that the acid involved is effectively hydrochloric acid (HCl) and the rise in plasma [chloride] is accounted for in the calculation of anion gap (ie chloride is a ‘measured anion’).

The result is that the ‘rise in anion gap’ (the numerator in the delta ratio calculation) does not occur but the ‘decrease in bicarbonate’ (the denominator) does rise in numerical value.

The net of both these changes then is to cause a marked drop in delta ratio (commonly to < 0.4)

DELTA RATIO

0.4– 0.8

Consider combined HAGMA + NAGMA, BUT note that the ratio is often < 1 in acidosis associated with renal failure)

DELTA RATIO

1 – 2

Usual for uncomplicated HAGMA.

lactic acidosis: average value 1.6

DKA more likely to have a ratio closer to 1 due to urine ketone loss (esp. if patient not dehydrated)

DELTA RATIO

> 2

A high delta ratio can occur in the situation where the patient had quite an elevated bicarbonate value at the onset of the metabolic acidosis.

Such an elevated level could be due to a pre-existing metabolic alkalosis, or to compensation for a pre-existing respiratory acidosis (ie compensated chronic respiratory acidosis).

DELTA RATIO SCENARIOS

DELTA RATIO	ASSESSMENT GUIDELINES
<0.4	Hyperchloraemic normal anion gap acidosis
<1	High anion-gap & normal anion-gap acidosis
1 - 2	Pure anion-gap acidosis Lactic acidosis; average value 1.6 DKA more likely to have a ratio closer to 1 due to urinary ketone loss
>2	High Anion-gap acidosis and concurrent metabolic acidosis or Pre-existing compensated respiratory acidosis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