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Updated Content for All India Exam Preparation

ALL INDIA PG

20 Authors

for All India PG Entrance Examination

Volume 1

Anatomy, Physiology, Biochemistry, Pathology, Pharmacology, Microbiology and Forensic Medicine

- Compiled by Subject specialists to meet the latest NBE pattern of All India entrance and DNB entrance
- Content is good to read for any PG entrance exams
- Highly authentic questions and answers
- Last minute reading points, completely consolidated content, tabular columns, figures and flowcharts
- Expected image-based questions—subject-wise
- Last 10 years original AIPG questions and NEET Pattern questions

Editors
Antan Uresh Kumar T
Rajamahendran R

Foreword
J RadhaKrishnan

A must-buy book for All India, AIIMS, PGI, JIPMER, DNB, FMGE & State entrance exams

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6. Microbiology **749**

7. Forensic Medicine **913**

3

BIOCHEMISTRY

- General Biochemistry
- Carbohydrate Chemistry & Metabolism
- Lipid Chemistry & Metabolism
- Structure and Functions of Proteins and Enzymes
- Metabolism of Amino Acids
- Molecular Biology
- Molecular Diagnostics

SUBCELLULAR ORGANELLES AND CELL MEMBRANE

Ref: Vasudevan 7/e p10-23

Bacterial & Mammalian cells

	Prokaryotic cell	Eukaryotic cell
Size	Small	Large
Cell wall	Rigid	Lipid bilayer
Nucleus	Not defined	Well defined
Organelles	Nil	Many–mitochondria, lysosomes

PLASMA MEMBRANE

- The plasma membrane separates the cell from the external environment. It has highly selective permeability properties so that the entry and exit of compounds are regulated.
- Made up of lipids—Phospholipids are amphipathic in nature. Also contain cholesterol.
- Proteins—Peripheral and integral proteins
- Small amount of carbohydrates as Glycoproteins and Glycolipids.

Marker Enzymes

Subcellular organelle	Marker enzyme
Nucleus	
Mitochondria	ATP synthase–inner membrane
Lysosomes	Cathepsin
Golgi complex	Galactosyl transferase
Microsomes	Glucose 6 phosphatase
Cytoplasm	Lactate dehydrogenase

Metabolic Functions of Organelles

Nucleus	DNA replication, transcription
Endoplasmic reticulum	Synthesis of Protein, Glycoproteins, cholesterol (partial), drug metabolism.
Golgi body	Maturation of synthesized proteins
Lysosomes	Degradation of protein, carbohydrate, lipids & nucleotides
Mitochondria	ETC, generation of ATP, TCA cycle, beta oxidation of FA, urea heme,pyrimidine synthesis, gluconeogenesis, (all partly)
Cytosol	Protein synthesis, glycolysis, glycogen metabolism, HMP shunt, transaminations FA synthesis, purine synthesis, urea heme, pyrimidine synthesis(all partly)

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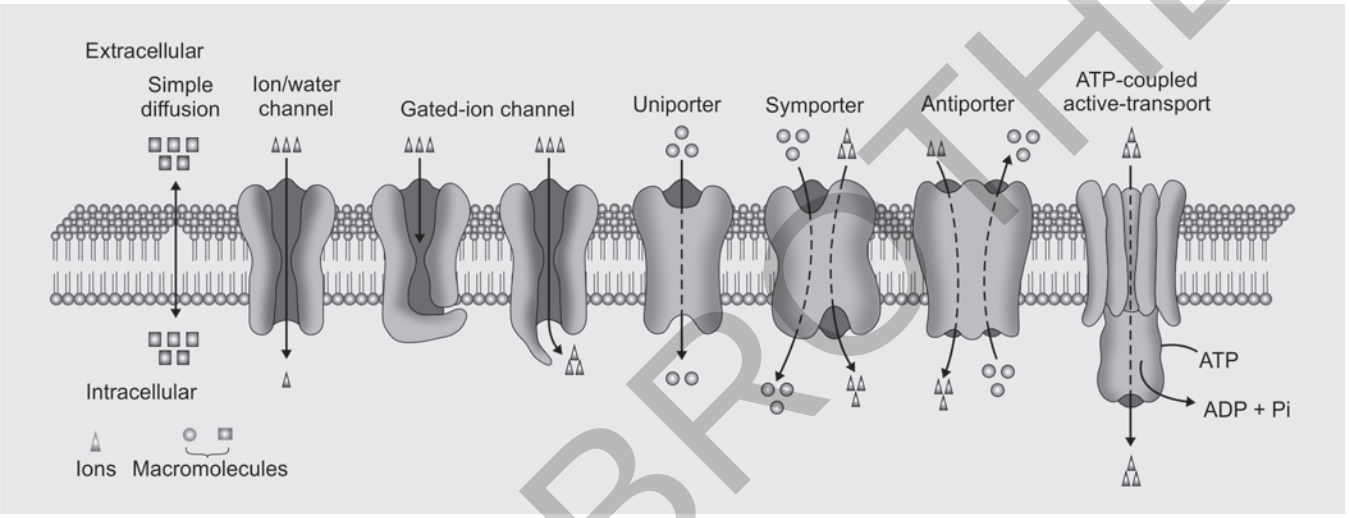
Types of Transport Mechanisms

	Carrier	Against gradient	Energy required	Examples
Simple diffusion	No	No	Nil	Water
Facilitated diffusion	Yes	No	Nil	Glucose to RBCs
Primary active	Yes	Yes	Direct	Sodium pump
Secondary active	Yes	Yes	Indirect	Glucose to intestine
Ion channels	Yes	No	No	Sodium channel

Transport Systems



Ion Channels

- Specialized transmembrane proteins that allow the selective entry of various ions.
- Involved in impulse propagation, synaptic transmission and neurotransmitter secretion

Leaky channels	Voltage gated channels	Ligand gated channels
• Always open • E.g. All cells possess leaky K+ channels • There will be a net influx of K+ ions through the channels unless K+ is at equilibrium	• The gates open/close in response to changes in membrane potential • E.g. Voltage gated Na+ channels • They are closed during resting state but membrane depolarization causes them to quickly open and then to close	• The channel has a receptor to a specific ligand or chemical. • Interaction of the ligand with the receptor regulates the opening & Closing of the channel. • E.g. Acetyl Choline mediated Na+ channels.

- **Amelogenin:** protein present in enamel of teeth, functions as a calcium channel.
- **Ionotropic receptors** are neurotransmitter receptors that form ion channel pores in the post synaptic membrane. They are either excitatory (e.g., (Na+) channels) or inhibitory (e.g., Cl-channels) and are rapid in action, **operating in milliseconds**
- **Ionophores** are molecul**es th**at act as membrane shuttles for various ions. E.g. Valinomycin for K+

WATER & ELECTROLYTES

Ref: Vasudevan 7/e p407 - 419/Harper 30/e p13

Water forms hydrogen-bonded clusters with itself and with other proton donors or acceptors.

Hydrogen bonds account for the surface tension, viscosity, liquid state at room temperature, and solvent power of water.

- Water is the most abundant constituent of the human body
- In an average 70-kg man the water content varies from 60 – 70 % of the total body weight
- Total body water = 70 kg × 0.60 = 42 L.

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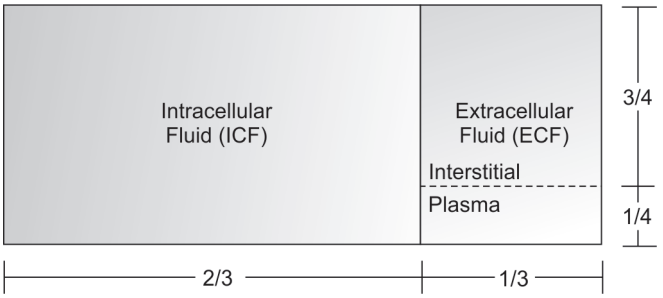
Body Water Compartments



Osmolality

Osmolality is the osmotic pressure exerted by the number of moles per kg of solvent.

The osmolality of plasma varies from 285 to 295 Mosm/kg.

It is maintained by the kidney.

Na+ is the major determinant of serum osmolality.

Measurement

- Osmometer: Direct measurement
- Indirect: Plasma osmolality mOsm/kg

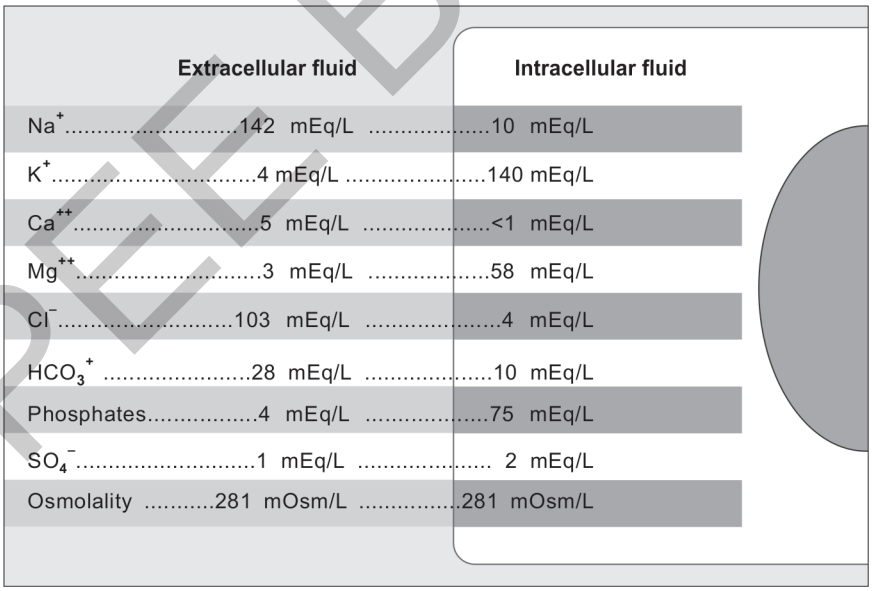
$$2\,(\text{Serum Na}) + \frac{\text{Glucose (mg)}}{18} + \frac{\text{Uria (mg)}}{2.8}$$

Electrolytes

- Substances that can dissociate in solution are called electrolytes

  NaCl = Na+ + Cl-
- Water molecules completely surrounds these dissociated ions and prevent union of positively charged particles with negatively charged ones
- Positive ions are called Cations
- Negative ions are called Anions
- The major intracellular cation is K, with an average concentration of 140 mEq/L. The extracellular K concentration is 3.5 to 5 mEq/L.
- The major extracellular cation is Na, with an average concentration of 140 mEq/L and an intracellular Na concentration of 12 mEq/L.

Electrolyte Concentration



- Removal of Na from the cell is by ACTIVE TRANSPORT —Sodium Pump that involves expenditure of energy in the form of ATP
- The enzyme involved is Na – K ATPase

Regulation of sodium and water balance: Kidneys play a major role

1. Aldosterone
2. ADH
3. Renin angiotensin system

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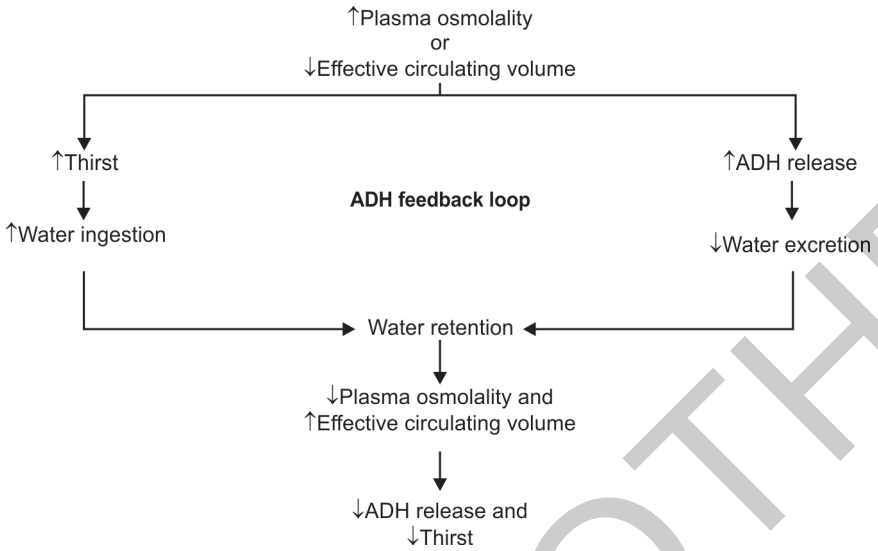
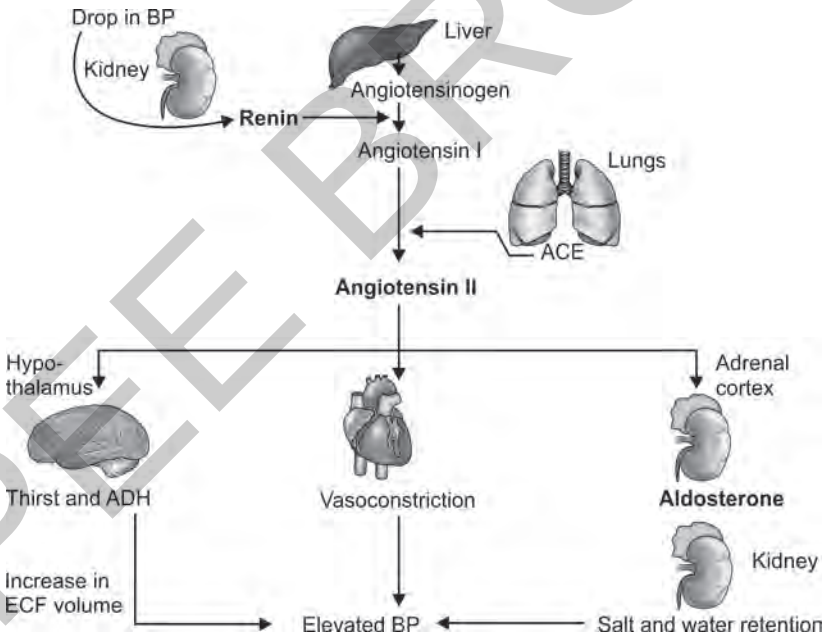
ADH By Thirst Center



Renin and Aldosterone



Control of Sodium and Water

Factor	Stimuli of action	Effect
Extracellular osmolality	Thirst & ADH	Water intake; reabsorption from kidney
Hypovolemia	Thirst & ADH; stimulates aldosterone	• Water retention • Sodium retention
ECF Expansion	Inhibits aldosterone	Sodium reabsorption
Hypoosmolality	Inhibits secretion of ADH	Water reabsorption

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Disturbances in Fluid Volume

The abnormalities in fluid and electrolyte balance is expressed in terms of TONICITY

1. Hypertonic: increased osmolality
2. Hypotonic: decreased osmolality

Abnormality	Biochemical features	Osmolality
Expansion of ECF		
Isotonic	Na & water retention	Normal
Hypotonic	Relative water excess	Decreased
Hypertonic	Relative Na excess	Increased
Contraction of ECF		
Isotonic	Loss of Na & water	Normal
Hypotonic	Relative Na loss	Decreased
Hypertonic	Relative water loss	Increased

Electrolytes

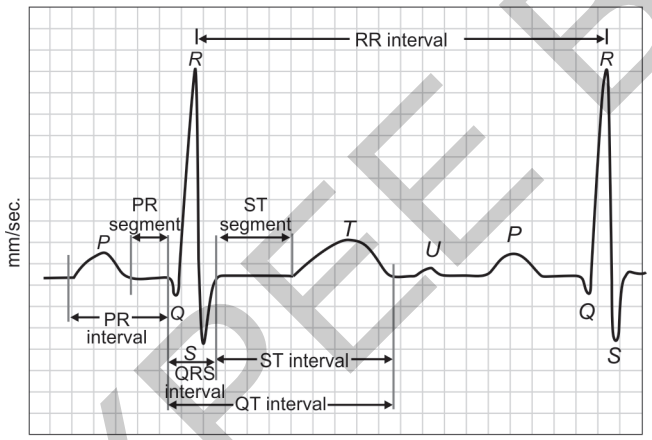
Sodium – 136 – 145 mmol/l
Potassium – 3.5 – 5.0 mmol/l
Chloride – 98 – 107 mmol/l
Bicarbonate – 24 – 28 mmol/l

Potassium

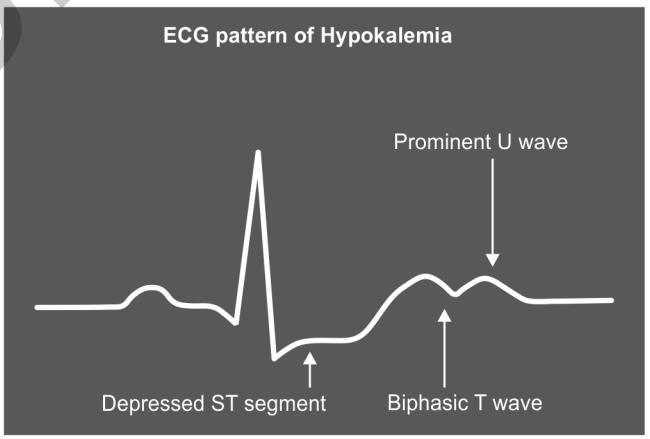
- Maintains intracellular osmotic pressure
- Required for the regulation of acid base and water balance in the cells
- Required for transmission of nerve impulse
- ECF K influences activity of cardiac muscle
- **RDA:** 3 – 4 g/day
- Sources: Banana, orange, pineapple, potato, beans, chicken, liver, tender coconut water is richest source
- Hormone–Aldosterone increases K excretion
- Insulin increases Na KATPase activity that leads to shift of K into the cells
- Deficiency of insulin leads to shift of K from ICF to ECF
- Tissue destruction and break down results in the release of intracellular

Hypokalemia: Signs/ symptoms

- Irritability
- Muscular weakness of lower extremities
- Tachycardia
- Cardiomegaly
- Cardiac arrest
- **ECG changes:** *flattening or inversion of T waves, prominent 'U' wave, depression of ST – segment.*



mm/mV 1 square = 0.04 sec/0.1mV



Causes of Hypokalemia

Increased renal excretion	Redistribution or shift of K+	Loss through GIT	Drugs	Excess IV saline infusion
Cushing syndrome	Alkalosis	Diarrhea	Insulin	
RTA	Insulin therapy	Vomiting	Salbutamol	
Renal artery stenosis	Hypokalemic periodic paralysis	Low K+ diet	Osmotic diuretics	
Hypomagnesemia	Thyrotoxic periodic paralysis	Malabsorption	Thiazides, Acetazolamide	
Hyperaldosteronism		Pyloric obstruction	Corticosteriods	

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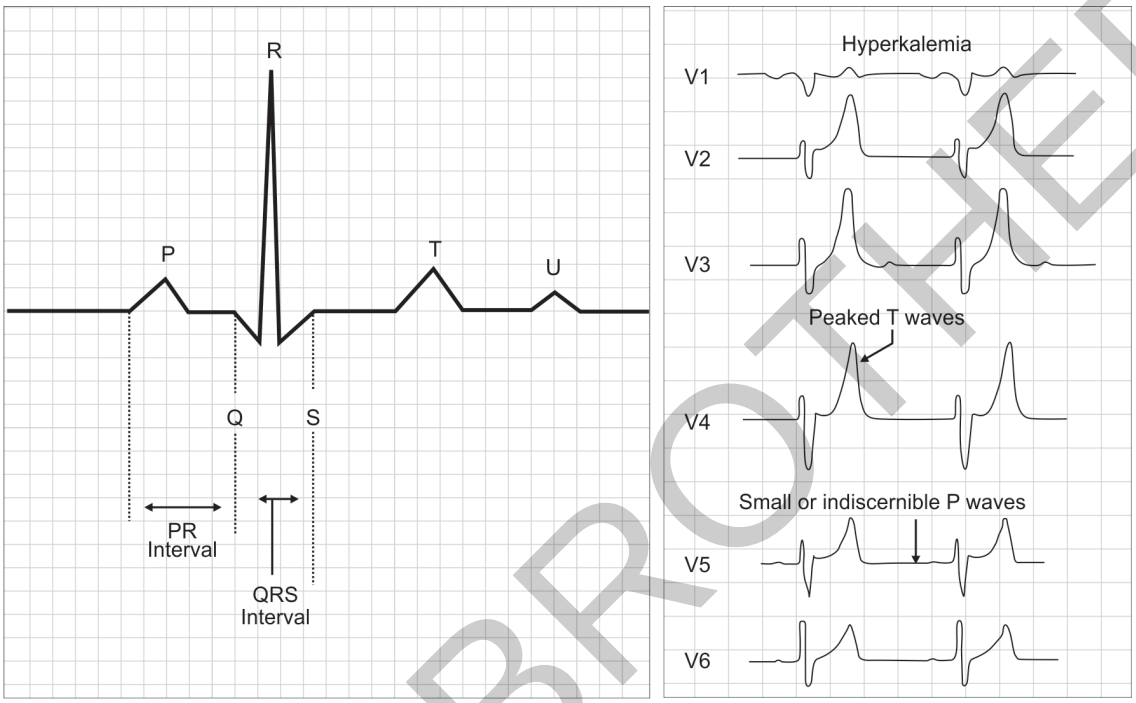
Hyperkalemia: Signs/symptoms

- CNS depression
- Mental confusion
- Numbness
- Bradycardia
- Cardiac arrest
- **ECG changes:** *Tall T waves, widening of QRS complex*



The electrocardiogram

Causes of HyperkalemiaW

Decreased renal excretion of K^+	Entry of K^+ into extracellular space	Redistribution of K^+ into extracellular space	Pseudohyperkalemia	Drugs
UTI Obstruction	Hemolysis	Metabolic acidosis	Hemolysis (improper blood collection)	Spiranolactone
Renal failure	Tissue necrosis, burns	Insulin deficiency (DM)	Thrombocytosis	ACE inhibitors
Addison's	Tumor lysis	Tissue hypoxia	Leucocytosis	Beta blockers
Volume depletion (CCF)	Crush injury, rhabdomyolysis			Digoxin
	Excess K+ supplementation			Cyclosporine

Redistribution of Serum K

Increase K^+ entry into cells leading to Hypokalemia	Impaired K+ entry into cells – Hyperkalemia
Insulin	Glucagon
Betablockers	Acidosis
Alkalosis	Increased osmolality & catabolism

Sodium:

- Chief cation of ECF
- Regulates the ECF fluid volume
- Total body content is 4000meq
- 50% in bones
- 40% in ECF
- 10% in ECF
- The glomerular filtrate formed contains 800gms/day

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- Out of this 99% is reabsorbed from the proximal convulated tubules along with water
- Aldosterone increases sodium reabsorption
- **RDA:** 5–10 gms/day
- **Sources:** common salt, bread, whole grains, leafy vegetables, nuts, eggs, milk.

Hypernatremia

1. Cushings syndrome
2. Prolonged cortisone therapy
3. Pregnancy
4. Dehydration
5. Exchange transfusion
6. Primary Hyperaldosteronism
7. Excess salt intake
8. Drugs: Ampicillin, Tetracycline, Anabolic steroids, OC pills, Loop diuretics, Osmotic diuretics

Hyponatremia

1. Vomiting
2. Diarrhea
3. Burns
4. Addison's disease
5. Renal tubular acidosis
6. CRF, Nephrotic syndrome
7. CCF
8. Hyperglycemia, Ketoacidosis
9. SIADH
10. Pseudo or dilutional Hyponatremia – Myeloma, Mannitol
11. Drugs: ACE inhibitors, Lithium, NSAIDs, Vasopressin, Oxytocin, Chlorpropamide

Diagnostic Criteria of SIADH

1. Hyponatremia (<135 mmol/L)
2. Decreased Osmolality (<270 mOsm/kg)
3. Urine sodium > 20 mmol/L
4. urine osmolality > 100 mOsm/kg

Causes of SIADH

1. Infections
2. Malignancy
3. Trauma—abdominal surgery, head injury
4. CNS dioredrs—meningitis, encephalitis, cerebral hemorrhage, brain abscess
5. Drugs like–thiazides diuretics, carbamezepine, opiates.

ACID BASE BALANCE

Ref: Vasudevan 7/e p390-405

Hydrogen ions (H+) are present in all body compartments.

Maintenance of appropriate concentration of hydrogen ion (H+) is critical to normal cellular function.

Terms	Definition
pH	Negative logarithm of hydrogen ion concentration
Acids	Proton donors
Bases	Proton acceptors
pK value	pH at which the acids are half ionized
Alkali reserve	Bicarbonate available to neutralize acids
Buffers	They are solutions that can resist change in pH when acid or alkali is added.

Factors affecting PH of Buffer:

1. **The value of pK:** The lower the value of pK, the lower is the pH of the solution.
2. The ratio of salt to acid concentrations: pH will not change, as long as the ratio of the concentrations remains the same.

Normal pH:

1. Blood pH = 7.38 – 7.42
2. Normal urine has a pH around 6; lower than that of extracellular fluid (pH=7.4). This is called *acidification of urine*.

The pH of the urine may vary from as low as 4.5 to as high as 9.8, depending on the amount of acid excreted.

Regulation of pH

First line defense	Blood buffers
Second line defense	Respiratory regulation
Third line defense	Renal regulation

Blood Buffers

1. Bicarbonate buffer: Normal bicarbonate level of plasma is 24 mmol/L. The normal pCO2 of arterial blood is 40 mm of Hg.
   Alkali reserve: Bicarbonate represents the alkali reserve and it has to be sufficiently high to meet the acid load.
2. Protein buffer: Hb is the major protein in buffering system
3. Phosphate buffer: Intracellular buffer. Na2HPO4/ NaH2PO4

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Renal regulation

1. Excretion of H^+
2. Reabsorption of bicarbonate (recovery of bicarbonate)
3. Excretion of titratable acid (net acid excretion)
4. Excretion of $NH4^+$ (ammonium ions)

Relationship of pH with K+ Ion Balance

- Increase in H+ in extracellular fluid (ECF), there is exchange of H+ with K+ from within the cells. Net effect is an apparent increase in ECF potassium level (Hyperkalemia).
- Acute acidosis—Hyperkalemia and
- Acute alkalosis—Hypokalemia.
- In renal tubular acidosis, due to failure to excrete hydrogen ions, potassium is lost in urine and Hypokalemia results.

Classification of Acid-Base Disturbances

Acidosis (fall in pH)

a. Respiratory acidosis: Primary excess of carbonic acid.
b. Metabolic acidosis: Primary deficit of bicarbonate

Alkalosis (rise in pH)

a. Respiratory alkalosis: Primary deficit of carbonic acid.
b. Metabolic alkalosis: Primary excess of bicarbonate

Normal serum electrolytes and arterial blood gas values

pH	7.35 – 7.45
Bicarbonate	22 – 26 mmol/L
Chloride	96 – 106 mmol/L
Potassium	3.5 – 5 mmol/L
Sodium	136 – 145 mmol/L
PO2	85 – 100 mmHg
PCO2	35 – 45 mmHg

pCO2	>45 mmHg	Respiratory acidosis
pCO2	<35 mmHg	Respiratory alkalosis
HCO3	>33 mmol/L	Metabolic alkalosis
HCO3	<22 mmo/L	Metabolic acidosis

Acid base disturbances	pH	Primary change	Secondary change
Metabolic acidosis	Decreased	Bicarbonate deficient	Decrease in PaCO2
Metabolic alkalosis	Increased	Bicarbonate excess	Increase in PaCO2

Contd...

Contd...

Acid base disturbances	pH	Primary change	Secondary change
Respiratory acidosis	Decreased	Excess carbonic acid	Increase in bicarbonate
Respiratory alkalosis	Increased	Deficient carbonic acid	Decrease in bicarbonate

Causes of Acid-Base Disturbances

Respiratory acidosis	Respiratory alkalosis	Metabolic acidosis	Metabolic alkalosis
Pneumonia	High altitude	Diabetic ketoacidosis	Severe vomiting
Bronchitis, asthma	Hyperventilation	Lactic acidosis	Cushing syndrome
COPD	Hysteria	Renal failure	Milk alkali syndrome
Narcotics , sedatives	Febrile illness	Renal tubular acidosis	Diuretic therapy
Respiratory muscle paralysis	Septicemia	Diarrhea	
Trauma to CNS	Meningitis	Addison's disease	
Ascites	CCF		
Peritonitis			
Sleep apnea syndrome			

Anion Gap

- To maintain the electrical neutrality the sum of the cations and anions in ECF is always equal
- Sodium and potassium – 95% of cations
- Chloride & HCO3 – 86% of anions
- Hence there is always difference between the measured cations and anions
- The unmeasured anions—ANION GAP
  Anion gap: (Na+ + K+) - (HCO3– + Cl–).
  Normal = 12 mmol/L.

1. High anion gap metabolic acidosis (HAGMA)
2. Normal anion gap metabolic acidosis (NAGMA)
3. Decreased anion gap metabolic acidosis (DAGMA)

HAGMA Causes (LA – MUD – PE – S)

- Lactic acidosis
- Alcohol ketoacidosis
- Methanol toxicity

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- Uremia
- Diabetic Ketoacidosis
- Parcetamol toxicity
- Ethylene poisoning
- Salicylate poisioning

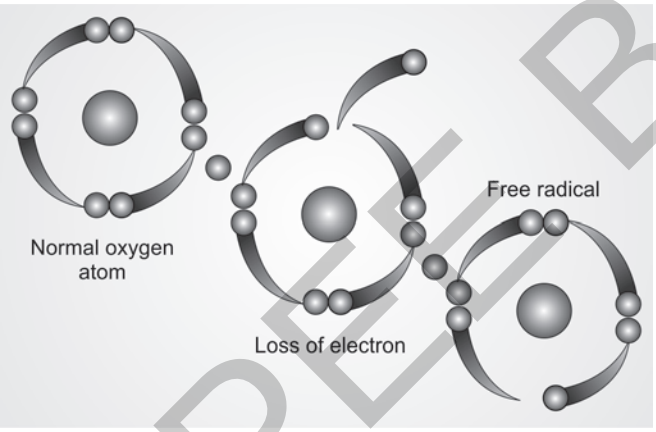
NAGMA Causes

- GIT fluid loss
  - Diarrhea
  - Pancreatitis
  - Intestinal fistula
- RTA – Renal Tubular acidosis – defective acidification of urine
- Acetazolamide therapy

Low Anion Gap Metabolic Acidosis

- Hypoalbuminemia
- Multiple myeloma (paraproteinemia)
- Bromide intoxication
- Hypercalcemia

Free Radicals and Antioxidants



A free radical is a molecule or molecular fragment that contains one or more unpaired electrons in its outer orbit.

Represented by R•

Has tendency to acquire electron from other substance

Reactive Oxygen Species (ROS)

- They are products of partial reduction of oxygen highly reactive and cause damage to living cells
- Highly reactive
- Very short half life **(10^{-9}-10^{-12} sec)**
- Generate new free radical by chain reaction
- Causes damage to cells & tissues

They are:

1. Super oxide
2. Hydroperoxyl radical
3. Hydrogen peroxide
4. Hydroxyl radical
5. Lipid peroxide radical
6. Singlet oxygen
7. Nitric oxide
8. Peroxy nitrite

Sources of ROS

1. During normal oxidation of food due to leak in the ETC
2. Production of H2O2 or super oxide by enzymes like Xanthine oxidase
3. Peroxisomal generation
4. During the synthesis of prostaglandins
5. Oxidation of heme to bile pigments
6. Peroxidation by lipooxygenase in platlets & leucocytes
7. Synthesis of nitric oxide
8. Auto oxidation due to metals.

Fenton Chemistry

$$Fe^{3+} + O_2^- \rightarrow Fe^{3+} + O_2$$

$$Fe^{2+} + H_2O_2 \rightarrow Fe^{3+} + OH^{\cdot} + OH^-$$

Haber-Weiss Reaction

$$O_2^- + H_2O_2 \rightarrow O_2 + OH^{\cdot} + OH^-$$

LDL Peroxidation

$$OH^{\cdot} + LDL \rightarrow H_2O + LDL - ox$$

9. Respiratory burst
10. Drug metabolism—parcetomol, halothane
11. Ionizing radiation—X–rays
12. Photolysis of oxygen by light
13. Cigarette smoking
14. Alcohol

Respiratory Burst

NADPH oxidase in the inflammatory cells (neutrophils, eosinophils, monocytes and macrophages) produces superoxide anion by a process of respiratory burst during phagocytosis.

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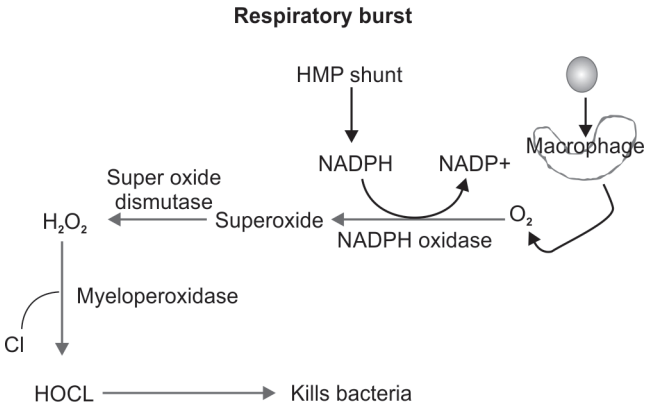

Antioxidants

- *A biological anti oxidant may be defined as a substance (present in low concentrations when compared to oxidant substrate) that significantly delays or inhibits oxidation of a substrate.*

Classification

Enzymatic antioxidants:

1. Superoxide dismutase
2. Catalase
3. Glutathione peroxidase
4. Glutathione reductase

Nutrient antioxidants:

1. Carotenoids
2. Vitamin E
3. Ascorbic acid
4. Selenium
5. Caffeine

Metabolic antioxidants:

1. Glutathione
2. Ceruloplasmin
3. Albumin
4. Bilirubin
5. Transferrin
6. Ferritin
7. Uric acid

Damage & Clinical Significance of ROS

1. Chronic inflammation—rheumatoid arthritis, chr. ulcerative colitis,etc
2. Acute inflammation
3. ARDS, broncho pulmonary dysplasia
4. Retrolentel fibroplasias—due to release of Thromboxane
5. Cataract
6. Reperfusion injury – due to increased activity of Xanthine oxidase and decreased availability of scavenging enzymes – Peroxidase
7. Atherosclerosis & MI
8. Carcinogenesis: DNA damage leads somatic mutation and cancer
9. Radiotherapy in the treatment of cancer produces ROS that triggers cell death
10. Aging process: Parkinsonism, Alzhimers disease

Psoralens: a plant product used in the treatment of psoriasis is by the effect of singlet O2 that is released from the product

XENOBIOTICS

Compound that is foreign to the body. Most of them are metabolized in the liver in 2 phases

Phase-I	Phase-II (Conjugation)
Xenobiotics are converted to more polar hydrophilic derivate. **Hydroxylation** is the major reaction catalyzed by mono oxygenases or cytochrome P450s. Cyt.P-450is the most versatile bio-catalyst known. Other reactions include deamination, desulfuration, epoxidation, peroxygenation, reduction, hydrolysis, dehalogenation.	Conjugated with glucuronic Acid, sulfate or glutathione, and excreted in urine or bile. 5-types: a. Glucuronidation-most common conjugation reaction. UDP-glucuronic acid is the glucuronyl donor. b. Sulfation- sulfate donor is PAPS (adenosine 3'-phosphate 5'- phosphosulfate). c. Conjugation with glutathione-γ glutamyl cysteinyl glycine d. Acetylation: Acetyl CoA is acetyl donor. Eg:-Isoniazide is subjected to it. e. Methylation: S-Adenyl Methionine (SAM) is the methyl donor.

- In certain cases, phase 1 metabolic reactions convert xenobiotic from inactive to biologically active compounds. In these instances, the original xenobiotics are referred to as "prodrugs" or "procarcinogens".

Cytochrome P-450

- All are hemoproteins, involved in metabolism of xenobiotics and many endogenous compounds e.g., Steroids, eicosanoids.
- Also called as "Mixed function Oxidases". NADPH (not NADH) is involved in the reaction mechanism.
- Microsomal P450 systems use NADPH- cytochrome P450 reductase for transfer of electrons. Cytochrome b5 is also used in some cases.

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Antan Uresh Kumar T completed his schooling at Villupuram, Tamil Nadu. He completed UG at Kilpauk Medical College, PG at Stanley Medical College (Guided by Prof DR Gunasekaran MS and Prof Dr K Ravishankar, Prof Dr Anto, Prof Dr Kannan, Prof Dr Vasuki) and MCh Urology at Institute of Urology (Guided by Prof V Kamaraj and Prof K Thiyagarajan, Prof V Selvaraj and Prof C Ilamparuthi) Madras Medical College. He worked as Assistant Professor at Government Villupuram Medical College. Currently, he is working as Assistant Professor at Institute of Urology, Madras Medical College under Prof P Govindarajan. He is renowned surgeon and practising Urologist and Transplant Surgeon. He is a team member of Madras Kidney Foundation—Excellence for transplant. He is very skillfull in Urology practise and wonderful Laparoscopic surgeon too.



Rajamahendran R is a renowned author/faculty in South India since 2007. He completed his MBBS and MS General Surgery from Kilpauk Medical College, Chennai. He joined MCh Surgical Gastroenterology in Madras Medical College, Chennai by acquiring rank 1 in State entrance examination. He also acquired Gold Medal in Esophagus Medical Exam during MCH SGE Course. He is the Founder/Faculty/Director of Koncpt PG Medical Coaching Center in Tamil Nadu, that started in the year 2009 and is running all over the state. He is leading Gastrointestinal and Laparoscopic Surgeon in Villupuram district and the Founder/Director of RRM Gastrosurgical and Research Center, Villupuram. He is presently working as Surgical Gastroenterologist in Government Medical College, Villupuram. He is very famous for all his publications.

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