Question 107
Which of the following is more appropriate for a 17 year old female suffering from IDDM
pH: 7.05
PO2 : 108
PCO2 : 12
HCO3 : 5
Base Excess : -30
Choices
A) Metabolic Acidosis
B) Respiratory Acidosis
C) Metabolic Alkalosis
D) None of the above
Answer
A) Metabolic Acidosis
Reference:
Harrison
Discussion
Arterial Blood Gas (ABG), an investigation which plays an important role in therapeutic decision making requires proper interpretation. A proper understanding of various components that are analysed is vital. As a detailed discussion of acid base disturbances is out of scope of this article; few basic facts are discussed in the following paragraph.

Drawing blood for ABG:
- A plastic / glass syringe is used.
- 0.1ml of Heparin is used for 1ml of blood drawn, as an anticoagulant. (Heparin is withdrawn into the syringe and pushed back, thus allowing heparin to just coat the syringe)
- The safest place to draw blood for ABG is radial artery at the wrist. Femoral artery also can be used.
- The syringe should be sealed immediately with cap (or needle tip inserted to a cork) to avoid air bubbles.
- Blood drawn should be analysed within 10 mts. Otherwise it should be cooled to 4°C with ice slush when a delay of up to one hour is acceptable. (Usually the syringe is sent in a flask with ice).
- Routine practice of temperature correction for blood gas measurements is not required.

Analysis:
The parameters analysed are
- PO2
- PCO2
- HCO3

PO2
Accepted Arterial O2 tension at room are as below.

<table>
<thead>
<tr>
<th>Criteria</th>
<th>mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults &amp; Children Normal</td>
<td>97</td>
</tr>
<tr>
<td>New born</td>
<td>&gt;80</td>
</tr>
<tr>
<td></td>
<td>40-70</td>
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</tbody>
</table>
Few Questions from the Chapter - Acid Base Balance of the book
FirsTest Series - Biochemistry
Author - Dr.M.Amali Bruno Publisher Kalam Books.

<table>
<thead>
<tr>
<th>Old individuals (Age in yrs)</th>
<th>60</th>
<th>&gt; 80</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>70</td>
<td>&gt; 70</td>
</tr>
<tr>
<td></td>
<td>80</td>
<td>&gt; 60</td>
</tr>
<tr>
<td></td>
<td>90</td>
<td>&gt; 50</td>
</tr>
</tbody>
</table>

The relationship between PaO₂ and SaO₂ are

<table>
<thead>
<tr>
<th></th>
<th>PaO₂</th>
<th>O₂ Saturation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypoxemia</td>
<td>&lt; 80</td>
<td>&lt; 95</td>
</tr>
<tr>
<td>Mild</td>
<td>60-79</td>
<td>90-94</td>
</tr>
<tr>
<td>Moderate</td>
<td>40-59</td>
<td>75-89</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt; 40</td>
<td>&lt; 75</td>
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</tbody>
</table>

pH:
The first step in an ABG interpretation is to look whether there is acidemic or alkalemic

<p>| | |</p>
<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>Normal</td>
<td>7:4 (7.35 to 7.45)</td>
</tr>
<tr>
<td>Acidemia</td>
<td>&lt; 7.35</td>
</tr>
<tr>
<td>Alkalemic</td>
<td>&gt; 7.45</td>
</tr>
</tbody>
</table>

 Acidemic, Alkalemic refer to the pH change only
 Acidosis, Alkalosis refer to the entire clinical & Biochemical picture.

HCO₃⁻
The second step is to look into HCO₃. The primary change in HCO₃ is called as Metabolic'

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>Normal</td>
<td>24 mmol/L (22-26)</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>&lt; 22 mEq/L</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>&gt; 26 mEq/L</td>
</tr>
</tbody>
</table>

PCO₂
The third step in an ABG interpretation is to look into the PCO₂. The primary change is PCO₂ is refered to as Respiratory'

<p>| | |</p>
<table>
<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>40 mmHg (35-45)</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>&gt; 45mm Hg</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>&lt; 35mm Hg</td>
</tr>
</tbody>
</table>

Wherever there is a change in PH, compensation occurs. Respiratory Compensation occurs for a metabolic acid-base disturbance and vice versa and in such cases PCO₂ & HCO₃ move in the same direction
- If HCO₃ decreases (Metabolic acidosis) then PCO₂ also decreases (Respiratory compensation)
- If PCO₂ increases (Respiratory acidosis), HCO₃ also increases (Metabolic compensation)
Few Questions from the Chapter - Acid Base Balance of the book FirsTest Series - Biochemistry
Author - Dr. M. Amali Bruno Publisher Kalam Books.

The normal expected rates of compensation are as follows: Please note that the unit of HCO₃⁻ is mEq/L (milliequivalent per litre) and the unit of PaCO₂ is mm Hg (millimeter of Mercury)

<table>
<thead>
<tr>
<th>Primary Disorder</th>
<th>Defect</th>
<th>Effect on pH</th>
<th>Compensatory Response</th>
<th>Expected Response</th>
<th>Limit of Compensation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Metabolic Acidosis</strong></td>
<td>HCO₃⁻ Decreased</td>
<td>Gain in H⁺ or loss of HCO₃⁻</td>
<td>Decrease Respiratory Alkalosis PaCO₂ Decreased</td>
<td>ΔPaCO₂ = ΔHCO₃⁻ x 1.2 (1 to 1.5)</td>
<td>PCO₂ = 12mmHg</td>
</tr>
<tr>
<td><strong>Metabolic Alkalosis</strong></td>
<td>HCO₃⁻ Increased</td>
<td>Gain in HCO₃⁻ or loss of H⁺</td>
<td>Increase Respiratory Acidosis PaCO₂ Increased</td>
<td>ΔPaCO₂ = ΔHCO₃⁻ x 0.7 (0.25 to 1.0)</td>
<td>PCO₂ = 55mmHg</td>
</tr>
<tr>
<td><strong>Respiratory Acidosis</strong></td>
<td>PaCO₂ Increased</td>
<td>CO₂ Retention</td>
<td>Decrease Metabolic Alkalosis HCO₃⁻ Increased</td>
<td>Acute: ΔHCO₃⁻ = ΔPaCO₂ x 1.0 (0.7 to 1.3)</td>
<td>HCO₃⁻ = 32 mmol/L</td>
</tr>
<tr>
<td><strong>Respiratory Alkalosis</strong></td>
<td>PaCO₂ Decreased</td>
<td>CO₂ Washout</td>
<td>Increase Metabolic Acidosis HCO₃⁻ Decreased</td>
<td>Acute: ΔHCO₃⁻ = ΔPaCO₂ x 0.2 (0.1 to 0.3)</td>
<td>HCO₃⁻ = 12-20 mmol/L</td>
</tr>
<tr>
<td><strong>Chronic:</strong></td>
<td></td>
<td></td>
<td></td>
<td>Chronic: ΔHCO₃⁻ = ΔPaCO₂ x 0.4 (0.3 to 0.5)</td>
<td>HCO₃⁻ = 12-15 mmol/L</td>
</tr>
</tbody>
</table>

If the compensation does not match, then a mixed acid base disorder is to be suspected.

Base Excess / Deficit is another parameter seen is any ABG report. The concept of base excess / deficit is found on the premise that the degree of deviation from the normal total buffer base availability can be calculated independent of compensatory CO₂ changes. A negative base excess is referred to as deficit. An abnormal pH with a base excess within 5mmol./L denotes a relatively normal and balanced metabolic acid base status. An abnormal pH with a base excess outside 10mmol/L signifies significant metabolic acid base disturbance.

A few examples of acid base disturbances are interpreted below.

1. 17 yrs old F, IDDM
   
   pH: 7.05  PCO₂: 12  PO₂: 108  
   HCO₃ : 5  BE: -30

   **Solution:**
   
   pH: 7.05 - Acidemic.
   
   HCO₃ : 5 Primary metabolic disturbance.
   
   HCO₃ deficit = 24 - 5 = 19
   
   Expected PCO₂ = 40-(19X1.2) = 40-22.8 = 17.2
   
   Actual PCO₂ >12

   **Interpretation:**
   
   Metabolic acidosis with respiratory alkalosis.
2. Collapsed person, intubated, given NaHco3
   pH: 7.51, PCO2: 35, PO2: 62
   HCO3: 27, BE: +5

   Solution:
   pH: 7.51 - Alkalemia
   HCO3: 27 - Primary metabolic problem.
   HCO3 excess = 27 - 24 = 3
   Excepted PCO2 = 40 + (3 * 0.7) = 42.1
   Actual PCO2 = 35
   Interpretation: Metabolic alkalosis with respiratory acidosis

3. 24 yrs F Broken ankle.
   pH: 7.55, PCO2: 27, PO2: 105,
   HCO3: 23, BE: 0

   Solution:
   pH: 7.55 - Alkalemia.
   PCO2: 27 Primary respiratory disturbance
   PCO2 Deficit = 40 - 27 = 13
   Expected HCO3 = 24 - (13 * 0.2) = 21.4
   Actual HCO3 = 23
   Interpretation: Respiratory alkalosis.

4. 45yrs Female.
   pH: 7.3, PCO2: 46, PO2: 55,
   HCO3: 24, BE: 0

   Solution:
   pH: 7.3 Acidemia
   PCO2 = 46 Primary respiratory problem
   PCO2 Excess = 46 - 40 = 6
   Expected HCO3 = 24 + (6 * 0.1) = 24.6
   Actual HCO3 = 24
   Interpretation: Acute Respiratory acidosis

5. 50 yrs Male
   pH: 7.43, PCO2: 50, PO2: 50,
   HCO3: 28, BE: +4

   Solution:
   pH: 7.43 Normal range
   PCO2: 50 Excess: 10
   Expected HCO3 : 24 + 3.5 = 27.5
Acid Base Balance of the book
FirstTest Series - Biochemistry
Author - Dr.M.Amali Bruno Publisher Kalam Books.
Book Available Online at www.first-test-series.com and
www.targetpg.com

Actual HCO₃ : 28
Interpretation :
Chronic respiratory acidosis.

6. 24 yrs Female, Unknown pill ingestion.
   pH:7.1,   PCO₂: 55,   PO₂:42,
   HCO₃: 17,   BE:-11

Solution:
pH:7.1 - Acidemia
HCO₃:17 Primary metabolic disturbance
HCO₃ deficit: 40-17=23
Expected PCO₂ : 40 - (23X1.2)=40 - 27.6 = 12.4
Actual PCO₂ : 55
Interpretation:
Metabolic and respiratory acidosis.

Author’s Note: The authors would like to thank Dr.V.Rqamasubramanian, MD DM (Nephro), Reader in Nephrology, Tirunelveli Medical College for his patient explanation of the fundamentals and model clinical situations

Question
108. Normal anion gap metabolic acidosis is caused by: (AIPG 2003)
1.Cholera.
2.Starvation
3.Ethylene glycol poisoning.
4.Lactic acidosis.

Answer
1.Cholera.

Reference
Harrison 15th Edition Chapter 50 and Page 285
About Anion Gap : Fundamental Points
Blood has Anions and Blood has Cations. It is a simple fact that the net charges of Anions should be equal to Net charges of Cations. The Anions are Chloride, Bicarbonate, Phosphate, Sulphate, Albumin, lactate, uremic anions, salicylates, ethylene glycol, formate etc. Sodium is the predominant cation and other cations are Potassium, Calcium, Magnesium, Globulin etc.

So

Total Charges of Anion = Total Charge of Cation
Cl⁻ + HCO₃⁻ + Other Anions which are not measured = Na⁺ + Other Cations which are not measured

Upon Re arranging
Other Anions which are not measured = \( \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-) \)

So

\[ \text{Anion Gap} = \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-) \]

Now this value (Sum of Unmeasured Anions – Sum of Unmeasured Cations) is called as Anion Gap which is equal to the difference between measured cations and measured anions.

So

**Anion Gap is**
- Difference between Measured Cations and Measured Anions
- or
- Difference between Unmeasured Anions and Unmeasured Cations

**Why is this Anion Gap so important Clinically.**
Say you are treating a case of Diabetic Ketoacidosis. You want to know the level of acids in the blood. For that the easiest way is to measure the concerned acid. But that facility is not available in all centres. So you measure Serum Sodium, Potassium and Bicarbonate. When you find that there is a lot of unmeasured anions, you assume that that is because of the ketoacids. All evaluations of acid-base disorders should include a simple calculation of the anion gap (AG);
- It represents those unmeasured anions in plasma (normally 10 to 12 mmol/L)
- It is calculated as follows: AG = \( \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-) \).
- The unmeasured anions include anionic proteins, phosphate, sulfate, and organic anions.

Before proceeding further, please note that few books include Potassium in the calculation of Anion Gap and few books take that as an unmeasured cation.

Now Anion Gap will Increase when (Such conditions, when, associated with Metabolic Acidosis, are called as High Anion Gap Metabolic Acidosis or HAGMA)
- Unmeasured Anions are increased
  1. Lactic acidosis
     - Anaerobic Metabolism Predominating - Shock / Cardio Pulmonary Arrest
     - Severe Anaemia,
     - Poisoning with CO or Cyanide,
  2. Ketoacidosis,
     - Diabetes
     - Alcoholics
     - Starvation
  3. Ingested toxins
     - Ethylene Glycol
     - Salicylates
     - Methanol
4. Acute and chronic renal failure.
5. In addition, the AG may increase with an increase in anionic albumin, either because of increased albumin concentration or alkalosis, which alters albumin charge.

Unmeasured Cations are decreased
1. Hypokalemia (when Potassium is not included in the calculation of Anion Gap)
2. Hypocalcemia
3. Hypomagnesemia

Now Anion Gap will Decrease when (Such conditions, when, associated with Metabolic Acidosis, are called as **Low Anion Gap Metabolic Acidosis or LAGMA**)

Unmeasured Cations are increased
1. Multiple Myeloma (remember that Globulin is positively charged)
2. Hypercalcemia
3. Lithium Toxicity

Unmeasured Anions are decreased
1. Low Albumin - A reduction in the major plasma anion albumin concentration (nephrotic syndrome)
2. A decrease in the effective anionic charge on albumin by acidosis; or
3. Hyperviscosity and severe hyperlipidemia, which can lead to an underestimation of sodium and chloride concentrations.

There will be no Change in Anion Gap when (Such conditions, when, associated with Metabolic Acidosis, are called as **Normal Anion Gap Metabolic Acidosis or NAGMA** eg Diarrhoea, Fistula, Ureterosigmoidostomy, Renal Tubular Acidosis, Ingestion of Ammonium Chloride, Mineralocorticoid deficiency)

- Measured Anions are increased
- Measured Anions are decreased
- Measured Cations are increased
- Measured Cations are decreased

How to find Solve MCQs regarding this topic – Ask the following questions
1. What ion increases or decreases in this condition
2. It is an measured ion (Sodium, Chloride or Bicarbonate) or an unmeasured ion

Then follow the following Flow Chart

- Increase or Decrease of a Measured Ion | Normal Anion Gap Metabolic Acidosis (NAGMA)
- Increase of an Unmeasured Anion (for example lactate) | That is High Anion Gap Metabolic Acidosis
- Increase of an Unmeasured Cation (or example lithium) | That is Low Anion Gap Metabolic Acidosis
- Decrease of an Unmeasured Anion (for example albumin) | That is Low Anion Gap Metabolic Acidosis
- Decrease of an Unmeasured Cation (or example Calcium) | That is High Anion Gap Metabolic Acidosis

The osmole gap
The osmole gap may be helpful in diagnosing a suspected ingestion of a toxic substance. An elevated osmole gap (>20 mOsm/L) with a metabolic acidosis can suggest the presence of osmotically active agents such as methanol, ethylene glycol, or ethanol.

**Osmole Gap = Measured Serum Osmolality – Estimated Serum Osmolality**

Estimated Serum Osmolality = \(2(Na^+) + \frac{[Glucose]}{18} + \frac{[BUN]}{2.8}\)

Normal serum osmolality is 280-295 mOsm/L

**Question**

109. A 50 Kg. man with severe metabolic acidosis has the following parameters: pH 7.05, pCO2 12mm Hg., pO2 108mm Hg. HCO3 5 meq/L. base excess -30 mEq/L. The approximate quantity of sodium bicarbonate that he should receive in half hour is: (AIPG 2003)

1. 250mEq.
2. 350mEq.
3. 500mEq.
4. 750mEq.

**Answer**

1. 250mEq.

**Reference**

Harrison 15th Edition Chapter

**Discussion**

Estimated replacement dose of sodium bicarbonate = (Base deficit in mEq/L) x (Body weight in kg) x 0.25 (some books give the formula as Base Deficit x Body Weight x 0.3)

This is Used in metabolic acidosis with severe sodium bicarbonate deficit. In cardiac arrest give 1/2 the deficit immediately, otherwise replace with 1/2 the deficit over 8-12 hrs.

Calculating the amount of bicarbonate replacement necessary must take into account the effect of nonbicarbonate buffers on exogenously administered bicarbonate. Multiply the desired increase in plasma bicarbonate concentration by the apparent volume of distribution and weight. The bicarbonate deficit can be calculated as follows:

**Bicarbonate Replacement**

- Not usually recommended unless pH < 7.2 or HCO3- deficit is greater than 5 meq/L.
- 8.4% NaHCO3 = 1 meq/ml
- 1 gm of baking soda = 12 mEq of NaHCO3
- Formula for Bicarbonate Replacement
  - 0.3 x (BW in Kg) x (HCO3- deficit) = mEq of NaHCO3 needed
  - 0.3 assumes 30% of BW is extracellular fluid.
  - If the figure of 0.3 is used, bicarbonate replacement can be given fairly rapidly (30 minutes to 1 hour). If figure of .5 is used bicarbonate replacement should be given over 12-24 hour period and half of it should be given in the first half hour
If you have a mixed respiratory/metabolic acidosis, it is important to address the respiratory acidosis first, before treating the metabolic acidosis.

**Explanation**

In our case:

Replacement = 0.5 x Body Weight x (Desired HCO₃ – Measured HCO₃)

Replacement in ½ hour = 0.25 x Body Weight x (Desired HCO₃ – Measured HCO₃)

= 0.25 x 50 x (25-5) = 250 mEq

**Comments**

- The base excess is defined as the Base that must be added to Restore a normal pH. The base excess is defined as the amount of H⁺ ions that would be required to return the pH of the blood to 7.35 if the pCO₂ were adjusted to normal.
- It is a calculated figure which provides an estimate of the metabolic component of the acid-base balance.
- Base Excess = (Actual pH - Predicted pH) * 67 and the predicted pH is calculated based on PaCO₂ (see Blood Gas)
- Normal Range: -2 to +2 meq/L
- Because the base excess is a calculated (not a measured) value, it may be inaccurate and misleading. Despite these problems, it is important to understand the concept.
- When interpreting blood gas results, the following heuristic is useful:
  - a base excess > +3 = metabolic alkalosis
  - a base excess < -3 = metabolic acidosis
- Simple Logic Interpretation
  - Positive (Base Excess) Metabolic Alkalosis
  - Negative (Base Deficit) Metabolic Acidosis
- Application in Neonatology: In Severe Acidosis (Base Excess < -10) (When Base Excess is negative, it is base deficit) Calculate Total Body Bicarbonate deficit Deficit = (Base Deficit) x (Weight in kg) x 0.3 and Administer 25% of bicarbonate deficit (~1 meq/kg)
- Note that in our case the Base Excess is –30 and this confirms metabolic Acidosis

**Question**

110. Causes of metabolic alkalosis include all the following except. (AIPG 2003)
1. Mineralocorticoid deficiency.
2. Bartter's syndrome.
3. Thiazide diuretic therapy.
4. Recurrent vomiting.

**Answer**

1. Mineralocorticoid deficiency.

**Reference**
Discussion
Metabolic alkalosis occurs as a result of net gain of Bicarbonate \( [\text{HCO}_3^-] \) or loss of nonvolatile acid (usually HCl by vomiting) from the extracellular fluid. Since it is unusual for alkali to be added to the body, the disorder involves a generative stage, in which the loss of acid usually causes alkalosis, and a maintenance stage, in which the kidneys fail to compensate by excreting \( \text{HCO}_3^- \) because of volume contraction, a low GFR, or depletion of Cl\(^-\) or K\(^+\).

Under normal circumstances, the kidneys have an impressive capacity to excrete \( \text{HCO}_3^- \). Continuation of metabolic alkalosis represents a failure of the kidneys to eliminate \( \text{HCO}_3^- \) in the usual manner. For \( \text{HCO}_3^- \) to be added to the extracellular fluid, it must be administered exogenously or synthesized endogenously, in part or entirely by the kidneys. The kidneys will retain, rather than excrete, the excess alkali and maintain the alkalosis if volume deficiency, chloride deficiency, and K\(^+\) deficiency exist in combination with a reduced GFR, which augments distal tubule H\(^+\) secretion; or hypokalemia exists because of autonomous hyperaldosteronism. In the first example, alkalosis is corrected by administration of NaCl and KCl, while in the latter it is necessary to repair the alkalosis by pharmacologic or surgical intervention, not with saline administration.

Explanation
1. In patients with Decreased Mineralocorticoid Production or Action, disorders of aldosterone biosynthesis or action are associated with high renin levels, salt wasting, and hyperkalemia. The aldosterone levels may be low or elevated. In patients with a deficiency in aldosterone biosynthesis, the transformation of corticosterone into aldosterone is impaired, owing to a mutation in the aldosterone synthase (CYP11B2) gene. These patients have low to absent aldosterone secretion, elevated plasma renin levels, and elevated levels of the intermediates of aldosterone biosynthesis (corticosterone and 18-hydroxycorticosterone). Pseudohypoaldosteronism type I (PHA-I) is an autosomal recessive disorder that is seen in the neonatal period and is characterized by salt wasting, hypotension, hyperkalemia, and high renin and aldosterone levels. In contrast to the gain-of-function mutations in the epithelial sodium channel (ENaC) in Liddle's syndrome, mutations in PHA-I result in loss of ENaC function.

2. Hypokalemia, metabolic alkalosis, and normal to low blood pressure are the clinical findings characteristic of Bartter's syndrome. In antenatal and classic Bartter's syndrome, impaired Cl\(^-\) reabsorption in the thick ascending limb of the loop of Henle is the underlying defect. Inadequate Cl\(^-\) reabsorption causes volume depletion and activates the renin-angiotensin system. Distal delivery of NaCl and water are high in the presence of high aldosterone, promoting secretion of K\(^+\) and H\(^+\) ions. Prostaglandin overproduction is mediated by volume depletion, hypokalemia, and high angiotensin II and kallikrein levels. Increased prostaglandin production contributes to the severity of disease by inducing resistance to the pressor effects of angiotensin II and reducing reabsorption in the thick ascending limb of the loop of Henle. Mutations in the bumetanide-sensitive Na:K:2Cl channel, the apical ATP-regulated K\(^+\) channel, and the basolateral Cl\(^-\) channel have been described in classic and antenatal Bartter's.

3. Thiazide diuretics increase urinary excretion of sodium and water by inhibiting sodium reabsorption in the early distal tubules. They increase the rate of delivery of tubular fluid and electrolytes to the distal sites of hydrogen and potassium ion secretion, while plasma volume contraction increases aldosterone production. The increased delivery and increase in aldosterone levels promote sodium reabsorption at the distal tubules, thus increasing the loss of potassium and hydrogen ions. And this leads to Alkalosis.

4. Recurrent vomiting leads to loss of Acid and that leads to Alkalosis.

Comments
Metabolic acidosis leads to hyperkalemia as a result of cellular shifts in which \( H^+ \) is exchanged for \( K^+ \) or \( Na^+ \). For each decrease in blood pH of 0.10, the plasma \( K^+ \) should rise by 0.6 mmol/L. This relationship is not invariable. Diabetic ketoacidosis, lactic acidosis, diarrhea, and renal tubular acidosis (RTA) are often associated with potassium depletion because of urinary \( K^+ \) wasting.

**Question.**

111. All of the following statements are correct about potassium balance, except: (AIPG 2003)

1. Most of potassium is intracellular.
2. Three quarter of the total body potassium is found in skeletal muscle.
3. Intracellular potassium is released into extra-cellular space in response to severe injury.
4. Acidosis leads to movement of potassium from extracellular to intracellular fluid compartment.

**Answer**

4. Acidosis leads to movement of potassium from extracellular to intracellular fluid compartment.

**Reference**

Ganong 20th Edition Page 30
Harrison 15th Edition Page 278

**Discussion**

- Potassium is a major intracellular cation and is one of the determinants of Resting membrane potential as well as the process of Depolarisation and Repolarisation
- Total body stores account to 150 gms and the skeletal muscle stores is 110 gms
- 98% intracellular and 2% extracellular
- Intracellular concentration maintained by Na+K+ATPase Pump
- In acidosis, the “pump” mechanism is altered and the intracellular Potassium “wanders out of its home into the ECF”
- During Tissue injury, due to the loss of cell membrane integrity, hyperkalemia occurs
- Hypokalemia (Increased Potassium INSIDE cells)
  - Insulin
  - Aldosterone
  - Alkalosis
  - Beta adrenergic drugs (Salbutamal)
- Hyperkalemia (Decreased Potassium OUTSIDE cells)
  - Diabetes
  - Addisons
  - Acidosis
  - Beta Blockers – Propanolol
  - Cell Injury
  - Exercises

**Explanation**

1. Most of potassium is intracellular.
2. Three quarter of the total body potassium is found in skeletal muscle.
3. Intracellular potassium is released into extracellular space in response to severe injury. Remember that Rhabdomyolysis leads to Hyperkalemia.
4. Acidosis leads to movement of potassium from intracellular to extracellular fluid compartment. During a metabolic acidosis, excess hydrogen ions move toward the intracellular compartment and potassium moves out of the cell into the extracellular space (serum). For every decrease in the serum pH by 0.1, a concomitant increase in the serum potassium level by 0.5 mEq occurs. As a result, hyperkalemic arrhythmias (peaked T waves and QRS widening) and ventricular fibrillation may occur. Other acute metabolic effects of acidemia include insulin resistance, increased protein degradation, and reduced adenosine triphosphate (ATP) synthesis. During acidemia, the oxyhemoglobin dissociation curve shifts to the right; oxygen has a lower affinity for hemoglobin, but hemoglobin releases oxygen more readily. Also, nonspecific gastrointestinal complaints, such as abdominal pain, nausea, or vomiting, may be present.