

**AZRA NAHEED MEDICAL COLLEGE LAHORE**  
**MBBS 2019-20**  
**2<sup>nd</sup> Year MBBS**  
**(Physiology)**  
**RENAL END-MODULAR EXAMINATION**

**Total Time: 35 minutes**

**Total Marks: 30**

**DATE: 15-04-2020**

**Q1. Amina came to doctor & complained of having vomiting, diarrhea, she also reported of very little urine output since 8 hours. On examination B:P is 80/40.**

- A) Write Formula, used to calculate the GFR in healthy adult? (2)**  
**B) How sympathetic stimulation influences the GFR? (1.5)**  
**C) What changes do you expect in GFR & renal plasma flow in this case? (1.5)**

Reference: Guyton and Hall 13<sup>th</sup> edition page 337

<b>Q.1</b>	<b>Answer keys</b>	<b>Marks</b>
<b>A</b>	<b><math>GFR = K_f \times \text{Net filtration pressure}</math></b>	<b>2</b>
<b>B</b>	Sympathetic stimulation- decreased renal blood flow- decreased net filtration pressure- decreased GFR	<b>1.5</b>
<b>C</b>	In this scenario, there is decreased ECF volume- Renal plasma flow- decreased hydrostatic pressure- decreased net FP – decreased GRF	<b>1.5</b>

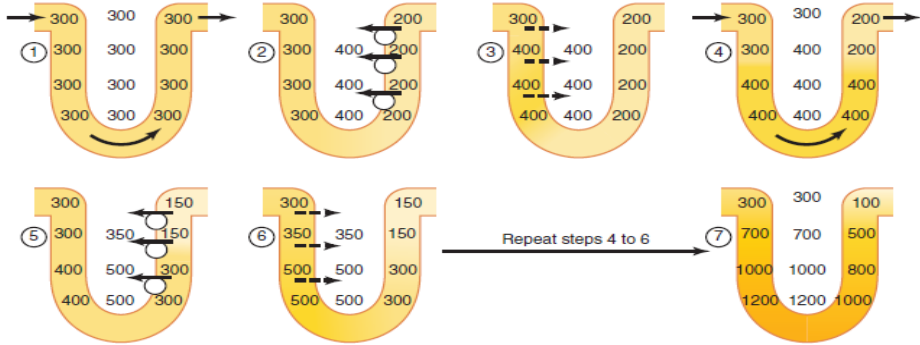
- Q2.A) Define Transport maximum, value of transport maximum for Glucose? (2)**  
**B) Why does glucose start appearing in urine when exceeds 200mg/dl? (1)**  
**C) What is difference between term (Transport Maximum) TM & renal threshold? (2)**

Reference: Guyton and Hall 13<sup>th</sup> edition Ch 28 page 351

<b>Q.2</b>	<b>Answer keys</b>	<b>Marks</b>
<b>A</b>	<p>For most substances that are actively reabsorbed or secreted, there is a limit to the rate at which the solute can be transported called the <i>transport maximum</i>. In the adult human, the transport maximum for glucose averages about 375 mg/min, whereas the filtered load of glucose is only about 125 mg/min (<math>GFR \times \text{plasma glucose} = 125 \text{ ml/min} \times 1 \text{ mg/ml}</math>). With large increases in GFR and/or plasma glucose concentration that increase the filtered load of glucose above 375 mg/min, the excess glucose filtered is not reabsorbed and passes into the urine.</p>	<b>2</b>
<b>B</b>	<p>When the filtered load exceeds the capability of the tubules to reabsorb glucose, urinary excretion of glucose does occur. when the plasma concentration of glucose rises above about 200 mg/100 ml, increasing the filtered load to about 250 mg/min, a small amount of glucose begins to appear in the urine. This point is termed the <i>threshold</i> for glucose. <i>Note that this appearance of glucose in the urine (at the threshold) occurs before the transport maximum is reached.</i></p>	<b>1</b>
<b>C</b>	<p>One reason for the difference between threshold and transport maximum is that not all nephrons have the same transport maximum for glucose, and some of the nephrons therefore begin to excrete glucose before others have reached their transport maximum. <i>The overall transport maximum for the kidneys, which is normally about 375 mg/min, is reached when all nephrons have reached their maximal capacity to reabsorb glucose.</i></p>	<b>2</b>

- Q3. A) Amjad is in desert & is dehydrated what different mechanisms will be initiated by the kidney to compensate for this decreased ECF volume? (2)**  
**B) Describe in detail the counter current multiplier mechanism. (3)**

Reference: Guyton and Hall 13<sup>th</sup> edition page 375

Q.3	Answer keys	Marks
A	1. Countercurrent multiplier mechanism 2. Urea cycle 3. Role of ADH 4. Countercurrent exchanger mechanism	2
B	 <p><b>Step 1:</b> assume that the loop of Henle is filled with fluid with a concentration of 300 mOsm/L, the same as that leaving the proximal tubule</p> <p><b>Step 2:</b> the active ion pump of the <i>thick ascending limb</i> on the loop of Henle reduces the concentration inside the tubule and raises the interstitial concentration; this pump establishes a 200-mOsm/L concentration gradient between the tubular fluid and the interstitial fluid (step 2).</p> <p><b>Step 3:</b> is that the tubular fluid in the <i>descending limb of the loop of Henle</i> and the interstitial fluid quickly reach osmotic equilibrium because of osmosis of water out of the descending limb. The interstitial osmolarity is maintained at 400 mOsm/L because of continued transport of ions out of the thick ascending loop of Henle. Thus, by itself, the active transport of sodium chloride out of the thick ascending limb is capable of establishing only a 200-mOsm/L concentration gradient, which is much less than that achieved by the countercurrent multiplier system.</p> <p><b>Step 4:</b> is additional flow of fluid into the loop of Henle from the proximal tubule, which causes the hyperosmotic fluid previously formed in the descending limb to flow into the ascending limb.</p> <p><b>Step 5:</b> Once this fluid is in the ascending limb, additional ions are pumped into the interstitium, with water remaining in the tubular fluid, until a 200-mOsm/L osmotic gradient is established, with the interstitial fluid osmolarity rising to 500 mOsm/L</p> <p><b>Step 6:</b> once again, the fluid in the descending limb reaches equilibrium with the hyperosmotic medullary interstitial fluid and as the hyperosmotic tubular fluid from the descending limb of the loop of Henle flows into the ascending limb, still more solute is continuously pumped out of the tubules and deposited into the medullary interstitium.</p>	3

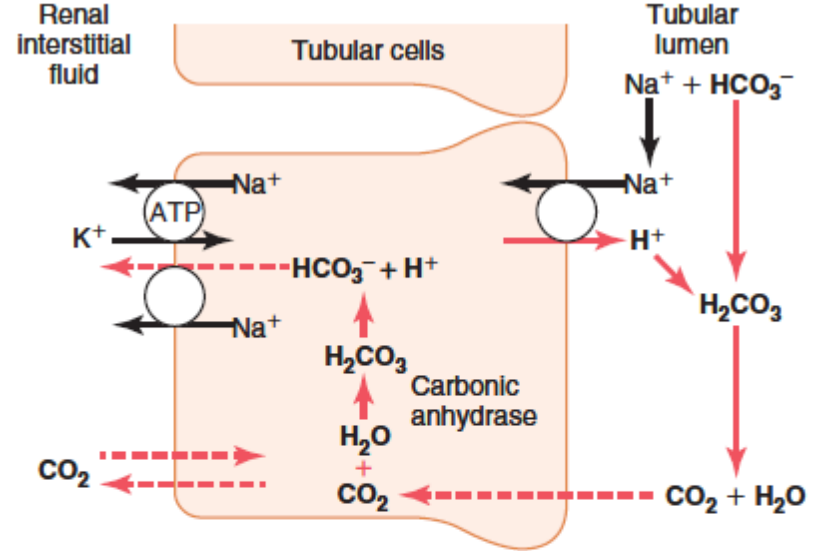
**Q4. A) Define Tubular Secretion? What is the mechanism of secretion of H<sup>+</sup> ions in proximal & late distal & collecting tubules?**

(2)

**B) Describe the role of Proximal convoluted tubules in acidosis.**

(3)

Reference: Guyton and Hall 13<sup>th</sup> edition page 416

Q.4	Answer keys	Marks
A	<p><b>Tubular secretion is defined as the direct transport of substances from ECF to tubular lumen</b></p> <p>In proximal tubule- Secondary active transport In late distal tubule- Primary active transport</p>	2
B	 <p>The diagram illustrates the cellular mechanisms in a tubular cell. On the left, renal interstitial fluid is shown. On the right, the tubular lumen is shown. Inside the cell, carbonic anhydrase catalyzes the reaction of CO<sub>2</sub> and H<sub>2</sub>O to form H<sub>2</sub>CO<sub>3</sub>, which dissociates into HCO<sub>3</sub><sup>-</sup> and H<sup>+</sup>. HCO<sub>3</sub><sup>-</sup> is reabsorbed into the interstitial fluid via a Na<sup>+</sup>-HCO<sub>3</sub><sup>-</sup> cotransporter. H<sup>+</sup> is secreted into the lumen via a Na<sup>+</sup>-H<sup>+</sup> cotransporter. In the lumen, H<sup>+</sup> combines with HCO<sub>3</sub><sup>-</sup> to form H<sub>2</sub>CO<sub>3</sub>, which dissociates into CO<sub>2</sub> and H<sub>2</sub>O. CO<sub>2</sub> is reabsorbed into the interstitial fluid. ATP is used for the active transport of Na<sup>+</sup> and K<sup>+</sup> on the interstitial side.</p> <p><b>Figure 31-5.</b> Cellular mechanisms for (1) active secretion of H<sup>+</sup> into the renal tubule; (2) tubular reabsorption of HCO<sub>3</sub><sup>-</sup> by combination with H<sup>+</sup> to form carbonic acid, which dissociates to form carbon dioxide and water; and (3) sodium ion reabsorption in exchange for H<sup>+</sup> secreted. This pattern of H<sup>+</sup> secretion occurs in the proximal tubule, the thick ascending segment of the loop of Henle, and the early distal tubule.</p>	3

- Q5. A) if you hold your breath. How would the ratio of HCO<sub>3</sub>/H<sub>2</sub>CO<sub>3</sub> ratio changes. (2)**  
**B) Describe which factors regulate the K<sup>+</sup> secretion? (3)**

Reference: Guyton and Hall 13<sup>th</sup> edition page 390

Q.5	Answer keys	Marks																
A	<b>Hypoventilation</b> – increased PCO <sub>2</sub> – HCO <sub>3</sub> /H <sub>2</sub> CO <sub>3</sub> ratio decreased, pH decreased leading to respiratory acidosis	2																
B	<table border="1"> <thead> <tr> <th data-bbox="305 541 699 646">Factors That Shift K<sup>+</sup> Into Cells (Decrease Extracellular [K<sup>+</sup>])</th> <th data-bbox="699 541 1089 646">Factors That Shift K<sup>+</sup> Out of Cells (Increase Extracellular [K<sup>+</sup>])</th> </tr> </thead> <tbody> <tr> <td data-bbox="305 646 699 730">Insulin</td> <td data-bbox="699 646 1089 730">Insulin deficiency (diabetes mellitus)</td> </tr> <tr> <td data-bbox="305 730 699 814">Aldosterone</td> <td data-bbox="699 730 1089 814">Aldosterone deficiency (Addison's disease)</td> </tr> <tr> <td data-bbox="305 814 699 856">β-adrenergic stimulation</td> <td data-bbox="699 814 1089 856">β-adrenergic blockade</td> </tr> <tr> <td data-bbox="305 856 699 898">Alkalosis</td> <td data-bbox="699 856 1089 898">Acidosis</td> </tr> <tr> <td></td> <td data-bbox="699 898 1089 940">Cell lysis</td> </tr> <tr> <td></td> <td data-bbox="699 940 1089 982">Strenuous exercise</td> </tr> <tr> <td></td> <td data-bbox="699 982 1089 1066">Increased extracellular fluid osmolarity</td> </tr> </tbody> </table>	Factors That Shift K <sup>+</sup> Into Cells (Decrease Extracellular [K <sup>+</sup> ])	Factors That Shift K <sup>+</sup> Out of Cells (Increase Extracellular [K <sup>+</sup> ])	Insulin	Insulin deficiency (diabetes mellitus)	Aldosterone	Aldosterone deficiency (Addison's disease)	β-adrenergic stimulation	β-adrenergic blockade	Alkalosis	Acidosis		Cell lysis		Strenuous exercise		Increased extracellular fluid osmolarity	3
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**Q6. Salma who is known case of D.M, came to hospital & told to doctor that she is having breathlessness. Laboratory tests showed**

**Na<sup>++</sup>=140mEq/L, CL=105mEq/L, HCO<sub>3</sub><sup>-</sup>= 6mEq/L**

- A) Diagnose on the basis of Anion Gap. (1.5)**  
**B) What is anion Gap, Calculate & give its normal value? (1.5)**  
**C) How the body will compensate in this case? (2)**

Reference: Guyton and Hall 13<sup>th</sup> edition page 426

<b>Q.6</b>	<b>Answer keys</b>	<b>Marks</b>
<b>A</b>	<b>Metabolic acidosis (29mEq/L)</b>	<b>1.5</b>
<b>B</b>	The “anion gap” (which is only a diagnostic concept) is the difference between unmeasured anions and unmeasured cations  <div style="background-color: #e0e0e0; padding: 5px; margin: 5px 0;"> <math display="block">\text{Plasma anion gap} = [\text{Na}^+] - [\text{HCO}_3^-] - [\text{Cl}^-]</math> <math display="block">= 144 - 24 - 108 = 12 \text{ mEq/L}</math> </div>	<b>1.5</b>
<b>C</b>	<b>Primary compensation:</b> Increased ventilation & decreased PCO <sub>2</sub> <b>Renal compensation:</b> by adding new HCO <sub>3</sub> to ECF	<b>2</b>

**MCQ s Key Renal End-Modular**

1	E	11	E
2	A	12	B
3	E	13	D
4	C	14	D
5	D	15	E
6	A	16	B
7	B	17	C
8	B	18	C
9	A	19	B
10	C	20	B