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What are the 3 buffer systems in the body

The human body maintains homeostasis through various physiological adaptations, one key adaptation being acid-base balance, which regulates pH levels between 7.35 and 7.45. This narrow range supports essential biological processes, such as blood oxygenation. The use of ionized intermediates in biochemical reactions becomes more difficult at neutral pH values. When the body's pH falls below 7.35, it is classified as acidemia, while a pH above 7.45 indicates alkalemia. To maintain this delicate balance, the human body employs compensatory mechanisms. Understanding these adaptations is crucial for approaching patients with conditions causing alterations in pH levels. There are four primary types of acid-based disorders: metabolic acidosis, metabolic alkalosis, respiratory acidosis, and respiratory alkalosis. In response to one condition, the body attempts to induce a counterbalancing effect through an opposite condition. For instance, if a person experiences metabolic acidemia, their body may attempt to compensate by inducing respiratory alkalosis. The term acidemia or alkalemia denotes that overall, the pH is acidic or alkalotic, respectively. While not always necessary, using this terminology can be beneficial in distinguishing between individual processes and the patient's overall pH status. A fundamental comprehension of cellular respiration is essential for grasping acid-base equilibrium in the human body. Aerobic cellular respiration is vital for human life, as humans are obligate aerobes that require oxygen to sustain life. The simplified chemical equation for aerobic cellular respiration demonstrates the conversion of glucose into carbon dioxide, generating energy. The process of glycolysis, the first stage of cellular respiration, breaks down a 6-carbon glucose molecule into 2 pyruvate molecules with minimal ATP production. The TCA cycle, which follows, generates NADH and FADH2 from their respective precursors, producing more ATP molecules. Oxygen is necessary for the TCA cycle to occur. The electron transport chain (ETC) is the final step in aerobic cellular respiration, producing most of the ATP created during this process. However, if oxygen levels are insufficient, the ETC reaction is impaired, leading to a decrease in ATP production. Aerobic cellular respiration, which includes glycolysis, results in fermentation that produces ATP from glucose molecules. Lactic acid is a byproduct of this process. During glycolysis and the TCA cycle, NAD+ gets reduced to NADH and FAD to FADH2, which drives the electron transport chain (ETC) with their gain of electrons. Ten NAD+ molecules are converted into 30 ATP-producing NADH for each glucose molecule, demonstrating why humans require oxygen for respiration. Anaerobic respiration allows some ATP production when there's insufficient oxygen but only generates two ATPs per reaction compared to the 38 produced by aerobic respiration. This is not enough to sustain life. The TCA cycle produces carbon dioxide as a byproduct that plays a crucial role in maintaining acid-base balance through buffer systems. One such system involves CO2, H2O, and their equilibrium: $\text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons \text{HCO}_3^- + \text{H}^+$. This reaction occurs without the need for an enzyme but is catalyzed by carbonic anhydrase in various tissues like red blood cells, renal tubules, and pancreatic cells. Other buffer systems include phosphate buffers, proteins, and hemoglobin. Each system in the body relies on maintaining pH balance, with the pulmonary and renal systems acting as primary regulators. The pulmonary system uses CO2 levels to adjust pH; when carbon dioxide is exhaled into the environment, it affects the pH within minutes to hours. Meanwhile, the renal system compensates by reabsorbing bicarbonate and eliminating fixed acids, either due to pathological conditions or metabolic imbalances. The kidneys play a vital role in maintaining the body's acid-base balance by excreting or reabsorbing substances that affect pH. The nephron, the functional unit of the kidney, filters blood and regulates the levels of hydrogen ions and bicarbonate. When bicarbonate is reabsorbed and/or acid is secreted into the urine, the pH becomes more alkaline. Conversely, when bicarbonate is not reabsorbed or acid is not excreted, the pH decreases. The renal system's metabolic compensation mechanism takes longer to occur than other physiological processes, typically taking days rather than minutes or hours. The body's physiological pH is essential for various life-sustaining processes, including oxygen delivery to tissues and correct protein structure. An increase in pH leads to a left shift on the oxygen dissociation curve, reducing the amount of oxygen needed to saturate hemoglobin, while a decrease in pH results in a right shift, increasing the affinity of hemoglobin for oxygen. The proper configuration of proteins is crucial for their function, and changes in pH can alter their charges, leading to denaturation. The Le Chatelier Principle states that when variables change, a system in equilibrium reacts to restore a new steady state. In the context of acid-base balance, if there is metabolic acidosis, the kidneys do not excrete enough hydrogen ions or reabsorb enough bicarbonate, prompting the respiratory system to increase minute ventilation and expel more CO2. Arterial blood gas (ABG) sampling is a diagnostic test used to assess a patient's acid-base status. By analyzing blood parameters such as pH, pCO2, pO2, HCO3-, and oxygen saturation, physicians can understand the body's acid-base balance and make informed treatment decisions. ABGs are crucial for critically ill patients as they aid in ventilator adjustments. Key ABG values include: - pH: 7.35 to 7.45 - pCO2: 35 to 45 mmHg - pO2: 75 to 100 mmHg - HCO3-: 22 to 26 mEq/L - O2 Sat: >95% The ability to swiftly analyze ABGs is vital in patient care. Assessing the pH helps determine if it's acidotic, alkalotic, or within normal limits. pCO2 levels indicate respiratory contribution; higher levels signify a lowered pH. HCO3- levels denote metabolic/kidney effects; elevated levels raise the pH. If the pH is acidotic, look for low values. Respiratory acidosis means high CO2. Metabolic compensation shows high HCO3-. Acidosis and alkalosis can be determined by which parameter is out of range. pH within normal limits but with abnormal PaCO2 or bicarb levels indicates a mixed disorder. Compensation does not always occur; clinical information becomes crucial in these cases. Electrolyte and renal function tests help clinicians determine the acid-base imbalance mechanism. The anion gap, a crucial metric in assessing metabolic acidosis, indicates the amount of conjugate base present beyond what's accounted for by hydrogen ions and bicarbonate. A patient with an elevated anion gap may have more units of conjugate base than expected due to various factors such as low albumin levels or increased presence of certain ions. This discrepancy can be a key indicator of underlying conditions contributing to the metabolic imbalance. The strong ion difference/strong ion gap approach offers a more nuanced understanding of acid-base dynamics but is considered less practical for clinical application due to its complexity and calculation demands. Mnemonics like MUDPILES or GOLDMARK can aid in recalling potential causes of high anion gap metabolic acidosis, including toxic ingestions, renal failure, and diabetic ketoacidosis. In contrast, narrow anion gap metabolic acidosis often results from the loss of bicarbonate accompanied by an increase in chloride ions. This condition may arise from severe diarrhea, certain types of kidney disease, or prolonged use of carbonic anhydrase inhibitors. The urine anion gap calculation can be a useful tool for determining the etiology. The Winter formula provides clinicians with an expected PCO2 value, essential for identifying mixed acid-base disorders. A lower than expected PCO2 may indicate respiratory alkalosis, while a higher value suggests respiratory acidosis. Respiratory acidosis is another critical aspect of acid-base balance assessment, often resulting from conditions affecting the lungs' ability to exchange gases effectively. Understanding these concepts is vital for providing accurate diagnoses and effective treatment plans in patients with metabolic or respiratory imbalances. Carbon dioxide released from cellular respiration is expelled into the environment through exhalation. In the human body, this gas combines with water to form carbonic acid via an enzymatic reaction facilitated by carbonic anhydrase. This acid then breaks down into a hydrogen ion and bicarbonate. A reduction in respiratory rate leads to decreased pH levels because there's less CO2 available for this chemical reaction to occur. Respiratory acidosis often stems from hypoventilation, which can be triggered by various factors such as COPD, opiate abuse, obesity, or brain injuries. When respiratory acidosis develops, the body should compensate by increasing bicarbonate production via the renal system. However, this doesn't always happen, and kidney problems can hinder the physiological response, posing significant risks to patients. Metabolic Alkalosis Metabolic alkalosis can be categorized into two main types: chloride-responsive and non-chloride-responsive. The latter is characterized by urine chloride levels below 20 mEq/L. Causes include vomiting, dehydration, diuretic use, and other conditions that disrupt electrolyte balance. Respiratory Alkalosis Any condition leading to excessive CO2 expulsion can cause respiratory alkalosis. This results in increased pH levels due to reduced carbonic acid production. The body compensates by decreasing bicarbonate production via the kidneys. Conditions causing respiratory alkalosis include panic attacks with hyperventilation, pulmonary embolism, pneumonia, and salicylate overdose. Understanding acid-base balance is crucial for clinicians as it's a vital aspect of human homeostasis. Diseases affecting this balance are common causes of hospital admissions, underscoring the importance of understanding its principles to provide appropriate care. Chronic metabolic acidosis and its effects on the body's buffer systems have been extensively studied [1-11]. The balance between acids and bases in the blood is crucial for proper physiological functioning, and this balance is measured using the pH scale [12]. Buffering systems, such as those found in plasma proteins, phosphate, and bicarbonate and carbonic acid buffers, help maintain a narrow pH range in the body [13-15]. These buffer systems are extremely efficient, with chemical buffers in the blood making adjustments to pH in seconds [16]. The respiratory tract can also adjust blood pH upward in minutes by exhaling CO2 from the body [17], while the renal system takes hours to days to have an effect on acid-base balance through the excretion of hydrogen ions and conservation of bicarbonate [18]. Protein buffer systems, which account for two-thirds of the buffering power of the blood and most of the buffering within cells, work predominantly inside cells and are made up of amino acids that can bind hydrogen and hydroxyl ions [19-21]. References: [1] Rev Psiquiatr Salud Ment. 2015 Jan-Mar;8(1):45-6. [2] Kidney Dis (Basel). 2017 Dec;3(4):149-159. [3] StatPearls Publishing; Treasure Island (FL): May 8, 2023. [4] Clin J Am Soc Nephrol. 2018 Apr 06;13(4):638-640. [5] Disclosure: Erin Hopkins declares no relevant financial relationships with ineligible companies. [6] Disclosure: Terrence Sanvictores declares no relevant financial relationships with ineligible companies. [7] Disclosure: Sandeep Sharma declares no relevant financial relationships with ineligible companies. [8] StatPearls [Internet]. [9] Hyperchloremic Acidosis. [10] Correlation of Urine Ammonium and Urine Osmolal Gap in Kidney Transplant Recipients. [11] Review of the Diagnostic Evaluation of Normal Anion Gap Metabolic Acidosis. In the lungs, the process of gas exchange is reversed, allowing CO2 to diffuse back into the air sacs and be exhaled. This process is a crucial part of the respiratory system. Phosphates in the blood exist in two forms: a weak acid (NaH2PO4) and a weak base (Na2HPO4). When these compounds interact with strong acids or bases, they form new compounds that help maintain the balance of acidity and alkalinity in the body. For example, when Na2HPO4 comes into contact with a strong acid like HCl, it forms NaH2PO4 and sodium chloride. The bicarbonate-carbonic acid buffer system works similarly to phosphate buffers. In this system, sodium bicarbonate (NaHCO3) is regulated by the body's sodium levels. When sodium bicarbonate interacts with a strong acid, carbonic acid (H2CO3) is formed, along with sodium chloride. Conversely, when carbonic acid comes into contact with a strong base like NaOH, it forms sodium bicarbonate and water. The bicarbonate buffer system plays a crucial role in maintaining the body's pH balance by capturing free ions and preventing significant changes in acidity or alkalinity. In normal conditions, there are 20 times more bicarbonate ions than carbonic acid molecules in the blood, making this system most efficient at buffering acidic changes. The respiratory system contributes to maintaining acid-base balance by regulating CO2 levels in the blood, which in turn affects the level of carbonic acid. Overall, the body has a complex system of buffers that work together to maintain pH homeostasis and prevent damage from excessive acidity or alkalinity. When you hold your breath, CO2 builds up in the blood, forming carbonic acid and lowering blood pH. To counteract this, increasing breathing rate or depth helps exhale more CO2, reducing blood levels of carbonic acid and adjusting pH back to normal. Conversely, excessive deep breathing can rid the blood of too much CO2, making it too alkaline, which can be corrected by rebreathing exhaled air into a paper bag. The respiratory system regulates blood pH by removing excess CO2 from the lungs' pulmonary capillaries through minor adjustments in breathing rate. During strenuous exercise, the body produces more CO2 and lactic acid, triggering an increase in respiration rate to remove excess CO2 and prevent acidosis. Chemoreceptors in peripheral blood sensors and the brain's medulla oblongata monitor CO2 levels and adjust respiratory rate accordingly. Impaired respiratory functions or reduced breathing can lead to hypercapnia (elevated CO2 levels), while hyperventilation causes hypocapnia (low CO2 levels). The renal system, on the other hand, regulates blood bicarbonate levels by controlling its excretion, with factors like diuretics, diarrhea, and Addison's disease affecting this balance. Low bicarbonate levels in blood can occur when aldosterone levels are reduced or when there's renal damage like chronic nephritis. In unmanaged diabetes mellitus, ketones binding to bicarbonate in the filtrate can prevent its conservation. The tubule cells don't absorb bicarbonate ions, so it must be supplied through a process: sodium ions reabsorbed for H+; cells produce bicarbonate that shunts to peritubular capillaries; CO2 reacts with bicarbonate forming carbonic acid, which dissociates into bicarbonate and hydrogen ions; the bicarbonate passes back into blood, while hydrogen ion is secreted into filtrate. Figure 26.17 shows this conservation process. The tubule cells are not permeable to bicarbonate, so it's conserved rather than reabsorbed. Steps 1 and 2 of bicarbonate conservation occur. The presence of sulfates, phosphates, or ammonia in the filtrate can capture hydrogen ions, making them unavailable for bicarbonate conversion. This leads to a pH imbalance and acidosis. Hydrogen ions also compete with potassium for sodium exchange in renal tubules. If there's more potassium than normal, it replaces hydrogen ions and less bicarbonate is conserved. When there's less potassium, more hydrogen ions enter the filtrate, and more bicarbonate is conserved. Chloride loss can cause an increase in bicarbonate reabsorption by the renal system. Diabetic acidosis or ketoacidosis often occurs in poorly controlled diabetes mellitus. When tissues can't get adequate glucose, they break down fatty acids for energy, producing ketone bodies that increase blood acidity. This condition can be severe and even fatal if not detected and treated properly. Early symptoms of ketoacidosis include deep, rapid breathing as the body tries to remove CO2 and compensate. The condition known as acidosis often accompanies diabetic coma, a severe complication of uncontrolled diabetes. One distinctive indicator is the sweet, acetone-like odor on an individual's breath due to the exhalation of ketones. Additionally, patients may exhibit signs such as dry, cracked skin and mouth, a flushed complexion, nausea, vomiting, stomach cramps, and discomfort. To treat diabetic coma, sugar is administered orally or intravenously. Prevention relies on regular insulin administration throughout the day. It's crucial for individuals with diabetes who rely on insulin to take their medication as prescribed; missing a dose can trigger ketoacidosis. Furthermore, research suggests that Hispanic and African-American individuals with type 2 diabetes are more likely to develop ketoacidosis compared to those from other ethnic backgrounds, although the underlying cause remains unclear.