Mini Review Article

Diaphragm as a Diuretic

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Abstract

Antidiuretic Hormone (ADH) is released by the posterior pituitary. It regulates facultative water reabsorption by increasing the water permeability of principal cells in the last part of the distal convoluted tubule and throughout the collecting duct. In the absence of ADH, the apical membranes of principal cells have a very low permeability to water. Within principal cells are tiny vesicles containing many copies of a water channel protein known as aquaporin-2.

Keywords:Basolateral membranes; Antidiuretic hormone; Dehydration; Natriuretic peptide

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ADH stimulates insertion of the aquaporin-2-containing vesicles into the apical membranes *via* exocytosis. As a result, the water permeability of the principal cell's apical membrane increases, and water molecules move more rapidly from the tubular fluid into cells.

Because the basolateral membranes are always permeable to water, water molecules move rapidly into the blood.[1] The kidneys can produce as little as 400-500 mL of very concentrated urine each day when ADH concentration is maximal, for instance during dehydration.

When ADH level declines, the aquaporin-2 channels are removed from the apical membrane *via* endocytosis.

The kidneys produce a large volume of dilute urine when ADH level is low. A negative feedback system involving ADH regulates facultative water reabsorption. When the osmolarity or osmotic pressure of plasma and interstitial fluid increases-that is, when water concentration decreases-by as little as 1%, osmoreceptors in the hypothalamus detect the change.[2] Their nerve impulses stimulate secretion of more ADH into the blood, and the principal cells become more permeable to water.

As facultative water reabsorption increases, plasma osmolarity decreases to normal-that is, when water concentration increases.

A second powerful stimulus for ADH secretion is a decrease in blood volume, as occurs in hemorrhaging or severe dehydration.

In the pathological absence of ADH activity, a condition known as diabetes insipidus, a person may excrete up to 20 liters of very dilute urine daily.

When the contraction of the diaphragm is maximized or contracted beyond the involuntary contraction of the diaphragm that is performed in normal or "quiet breathing, it creates a greater compression against the vena cave. The vena cava is a large vein that is very close to and runs directly into right atrium of the heart. The vena cava also receives blood from all the other veins of the middle and lower parts of the body before the venous blood returns to the heart.[3] Now, this greater compression by the diaphragm against the vena cava causes an increase in the volume of blood that returns to the heart via the right atrium. This increased volume of blood that returns to the heart will create an increase in the stretching of the heart (cardiac) muscle cells of the right atrium. This increase in the stretching of the heart muscle cells of the right atrium will stimulate them to release the hormone called Atrial Natriuretic Peptide (ANP). It is this hormone, atrial natriuretic peptide that once released from the heart cells travels to the kidneys and acts on the kidneys creating the conditions for the kidneys to expel more salt and water in the urine, hence the effects of a diuretic. When atrial natriuretic peptide (ANP) is "deployed" and acts on the kidneys quite a few things happen.[4]

The first thing that happens is the glomerular filtration rate (GFR) or basically the kidney filtration rate increases by way of maximizing the surface area of the kidney's filtration membrane by relaxation of mesangeal cells. This increases the volume of available filtrate that contains water, ions, wastes, toxins etc., to be available for reabsorption, or excretion, setting the stage for the excretion of this more available water and salt. This increased volume of available filtrate creates the possibility of a greater urine output. The proximal convoluted tubule is the first structure that the kidney filtrate passes through. The proximal convoluted tubule is responsible for reabsorption of water and many of the filtered solutes back to the bloodstream and makes the largest contribution. The proximal convoluted tubule reabsorbs about 65% of the filtered water, sodium and potassium and 100% of most filtered organic solutes such as glucose and amino acids, as well as 50% of filtered Chloride, as well as 80-90% of filtered bicarbonate, as well as 50% of

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the filtered urea and a variable amount of filtered calcium, magnesium and phosphate. In addition, the proximal convoluted tubule secretes variable amounts of hydrogen ions, ammonium ions and urea. Most solute reabsorption in the proximal convoluted tubule involves sodium.[5]

The Effect of Atrial Natriuretic Peptide on the proximal convoluted tubule

In regards to the function of the proximal convoluted tubule, where most of the reabsorption of water and sodium or salt takes place, atrial natriuretic peptide actually inhibits the reabsorption of sodium and water in the proximal convoluted tubule, the very part of the renal tubule where most of the reabsorption of water and sodium occurs, yet allows the normal reabsorption of potassium, chloride bicarbonate, urea, calcium, magnesium and phosphate leaving behind mostly water and sodium behind. ANP also inhibits water and sodium reabsorption in the collecting duct as well. The collecting duct being the last structure of the renal tubule before the filtrate is finally eliminated to become urine.[1]

Once the filtrate leaves the proximal convoluted tubule it continues to flow along to the next structure of the renal tubule. Keep in mind the filtrate still contains most of its water and sodium. The next structure is called the loop of Henle (Nephron loop). The function of the loop of Hanley remains unchanged and reabsorbs about 15% of the filtered water; 20-30% of the filtered sodium and potassium; 35% of the filtered chloride; 10-20% of filtered bicarbonate; and a variable amount of the filtered calcium and magnesium.[5] Reabsorption of water occurs in the descending limb of the loop of Henle as the thick ascending limb of the loop of Henle is virtually impermeable to water. As the remaining filtrate flows through the thick ascending loop of Henle the fluid then enters the distal convoluted tubule. The initial part of the Distal Convoluted Tubule (DCT) reabsorbs about 10-15% of filtered water, 5% of the filtered sodium and 5% of the filtered chloride. Once the fluid reaches the end of the distal convoluted tubule and collecting duct, the amount of water and solute reabsorption vary depending on the body's needs. The ability of reabsorption of water by the distal convoluted tubule and collecting duct is dependent upon availability of the hormone ADH and the ability of reabsorption of sodium is dependent on the availability of the hormone aldosterone. In the collecting duct there are cells called principal cells and they reabsorb sodium but have a very low permeability to water and the amount of water and solute reabsorption vary depending on the body's needs. In addition to inhibiting sodium and water reabsorption at both proximal convoluted tubule and the collecting duct, arial natriuretic peptide also suppresses the secretion of other hormones that effect the kidneys including aldosterone and antidiuretic hormone (vasopressin). Aldosterone stimulates the principal cells in the collecting duct to reabsorb more sodium which would contribute to more reabsorption of water and less excretion of water and atrial natriuretic peptide suppresses the secretion of this hormone to eliminate this effect. Antidiuretic hormone regulates facultative water reabsorption by increasing the water permeability of principle cells in the last part of the distal convoluted tubule and throughout the collecting duct. In the absence of ADH the principal cells have very low permeability to water. Atrial natriuretic peptide also suppresses the secretion of this hormone as well. So in combination of the suppression of the secretion of aldosterone by ANP prevents reabsorption of sodium in the collecting duct of the kidney as well as the suppression of the secretion of ADH by ANP prevents the reabsorption of water in addition to the inhibition of the filtered water reabsorption and inhibition of the sodium reabsorption at the proximal convoluted tubule contributes to the ultimate result of natriuresis and diuresis by the kidneys , which decreases blood volume and blood pressure. Maximizing the contraction of the diaphragm maximizes the oxygen input, maximizes the carbon dioxide output, maximizes the venous blood return to the heart, which in combination maximizes the efficiency of the heart, which then maximizes the surface area of the filtration capillaries in the kidneys, therefore maximizing the kidney filtration rate, therefore maximizing the water and salt excretion in the form of urine from the body. The amount of ATP used by sodium-potassium pumps in the renal tubule is about 6% of the total ATP consumption of the body at rest.[3]

References

1. Sharma P, Nagarajan V. Pleural effusion from leaky diaphragm-the hepatic hydrothorax. J Gen Intern Med. 2014;29(9):1309.
2. Bretag AH, Dawe SR, Kerr DI, Moskwa AG. Myotonia as a side effect of diuretic action. Br. J. Pharmacol. 1980;71(2):467.
3. Stevens WW, Sporn PH. Bilateral diaphragm weakness after chemotherapy for lymphoma. Am. J. Respir. 2014;189(7):12.
4. Brugge VA, Schooley DA, Orchard I. Amino acid sequence and biological activity of a calcitonin-like diuretic hormone (DH31) from Rhodnius prolixus. J. Exp. Biol. 2008;211(3):382-390.
5. Reid H. An obscure case of ruptured diaphragm. Br. Med. J. 1957;1(5023):863.