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# Guyton and ganong physiology

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This chapter is vaguely relevant to Section E (i) of the CICM Primary Syllabus 2017, which expects the exam candidate to "explain mechanisms of transport of substances across cell membranes, including an understanding of the Gibbs-Donnan effect." The Gibbs-Donnan effect is certainly not a mechanism of transport across cell membranes; rather, transport across cell membranes is the mechanism of the Gibbs-Donnan effect; but such objections are unnecessarily academic. Question 14 from the second document of 2017 dedicated 40% of brands to the Gibbs-Donnan effect. Apparently, a large number of test candidates confused it with the electrochemical gradients that produce and maintain the resting membrane potential, which examiners saw as a minor disaster. To prevent future confusion, the Gibbs-Donnan effect can be summarized as follows: The Gibbs-Donnan effect describes the uneven distribution of permeating charged ions on both sides of a semi-permeable membrane that occurs in the presence of impermeable charged ions. At Gibbs-Donnan equilibrium, on each side of the membrane, each solution will be electrically neutral. The product of diffusible ions on one side of the membrane will be equal to the product of diffusible ions on the other side of the membrane. The electrochemical gradients produced by the uneven distribution of charged ions produce a potential transmembrane difference that can be calculated using the Nernst equation. The presence of impermeable ions on one side of the membrane creates an osmotic diffusion gradient that attracts water into that compartment. The mechanisms that maintain the potential of the resting membrane and the mechanisms of the Gibbs-Donnan effect are different phenomena: Donnan equilibrium is a completely passive process: active transporters are not involved in maintaining this equilibrium. A female equilibrium is an equilibrium, i.e. the ion concentrations on both sides of the barrier are static. If the Donnan equilibrium were to become fully established, the increase in intracellular ions would cause the cells to swell due to the osmotic influx of water. In a Donnan equilibrium, the resting membrane potential would be only about -20 mV. This potential would exist even if the membrane permeability for all ions were the same. The resting membrane potential, on the other hand, requires different permeability for potassium and sodium, and is actively maintained by the constant Na<sup>+</sup>/K<sup>+</sup> ATPase activity. Because biological membranes (especially of excitable tissues) are never in equilibrium, the Goldman-Hodgkin-Katz equation is usually a better choice to explain their electrochemical behavior. The most in-depth and definitive resource for this topic should be Nicholas Sperelakis' Cell Physiology Source Book, where Chapter 15 (p.243 of the 3rd edition) discusses Gibbs-Donnan in minuscule detail. It's probably also a good reference for a discussion on why the Gibbs-Donnan Gibbs-Donnan is not the main mechanism responsible for the potential of rest membrane. Guyton & Hall mentions the feminine effect in relation to shifts of capillary fluid around page 196 of the 13th edition, and the treatment of this phenomenon is more dissatisfactory, the revision of ganong "of medical physiology" does a slightly better job (P.6 of the 23rd edition.) three paragraphs which is probably good enough for the work of the government. If one is temperamentally unsuitable to piracy, you can pay for these textbooks and find these references within them. Alternatively, Nguyen & Kurtz (2006) has a free online article discussing the concept in great detail, with overmuch algebra and a focus on Gibbs-Donnan's balance between interstitial and intravascular fluid. The definition and history of Gibbs-Donnan (or just Donnan) the effect could expect it to be better defined by Frederick George Donnan himself (for example in a posthumous reprint of his 1911) sheet but unfortunately Donnan himself had never familiar with the needs of primary candidates and therefore did not make any effort to shorten his principle in a memorable soundbite. Instead, the sheet is an excellent long-form explanation, well written of the effect, probably better than anything, later published in glossy textbooks. If one needs a short definition, it can be reconstructed from the first paragraph of the encyclopedia of membranes (Drolioli & Day, 2015): "The Donnan effect is the phenomenon of the predictable and unparallelled distribution of ions loaded with permeated on both sides of a semipermeable membrane, in the presence of waterproof ions loaded" is the feminine effect, or is the effect of Gibbs-Donnan? Donnan never called its effect "the feminine effect," but from 1911 onwards it became known as such, and at this stage there was zero Gibbs in the public mentions of this concept. J.W. Gibbs was predominantly a physicist and a mathematician who contributed (massively) to chemistry a few decades before Donnan arrived, the relationship between the feminine effect and the published works of Gibbs was dissociated in 1923 by G. Adair, who found a Gibbsian equation since 1906 which was essentially identical to the equation of Donnan. There is a doubt that Donnan was significantly influenced by Gibbs, to the extent of giving addresses in his honor and describing it as a genius man, combining deep insight with the highest logical reasoning powers ("Donnan, 1925.) later publications of Donnan (eg. Donnan, 1924) are well furnished with appropriate attributes, some chloride ions are diffused into the intracellular compartment. By necessity, they are accompanied by some potassium ions, so as to preserve electron neutrality. The chloride ions are also rejected by the negatively charged protein in the intracellular compartment, so that most of the chloride remains on the extracellular side of the membrane. So, Electron neutrality is preserved, equilibrium of the total concentration of diffusible ions, such that the product of the extracellular diffusible ions concentrations is equal to the product of the intracellular diffusible ions concentrations: [K<sup>+</sup>]<sub>ext</sub> [Cl<sup>-</sup>]<sub>ext</sub> = [K<sup>+</sup>]<sub>int</sub> [Cl<sup>-</sup>]<sub>int</sub> Without falling into a pair of quadratic equations, suffice it to say that if we started with 100 mmol / l, a, on both sides, once the proteins are added, it ends with about 33 mmol / l of chloride on the side, as well as 133 mmol / l of potassium; The extra ionic molecules came from the extracellular fluid, and therefore that compartment becomes relatively ion-poor, with about 66.6 mmol / l of each species. Now, of course, because there is an electric gradient as well as a chemical diffusion gradient that acts on the ions, there will be a slightly unparallelled distribution of charge through the membrane, leading to a potential difference. This is a family concept discussed large lengths in the chapter on resting membrane potential. Basters say that for each ion the balance between the concentration gradient and the electric gradient is described by the Nernst equation, and the total potential difference through the membrane that derives from the combined effect of all ion movements can be described by the Goldman-Hodgkin-Katz equation, taking into account the fact that for every ion the permeability of the membrane will be different. In short, the Gibbs-Donnan effect sets a transmembrane potential difference because the distribution of ions loaded through the membrane is irregular. This potential difference is apparently quite small. Sperelakis (2011) gives a value of -20 mV, even if it is not clear from where that number comes from. So we are now at the Gibbs-Donnan balance: the products of widespread ion concentrations must be the same on both sides, and on each side of the membrane the electric neutrality is preserved. However, the presence of non-diffusible proteins makes the total concentration of intracellular molecules much higher than the concentration of extracellular molecules: intracellular concentration = [k<sup>+</sup>] int + [cl<sup>-</sup>] int + [p<sup>-</sup>] intercellular concentration = [k<sup>+</sup>] ext + [Cl<sup>-</sup>] Ext In fact, in this experiment of thought (wildly physiologically inaccurate) the difference in osmolality is quite stark (there are about 134 MOSM / L difference.) With this type of osmotic gradient, water would overlap through the membrane, causing the cell to inflate horribly and explode. Obviously, this does not happen in vivo. Na<sup>+</sup> / K<sup>+</sup> ATPase plays an important role in the prevention of cellular osmotic explosion pumping three sodium ions out of the cell in exchange for two potassium. The terrible permeability of the cell membrane sodium means that sodium generally maintains the extracellular compartment, keeping osmolyte. As a result, a second Donnan effect (this time with non-widespread ions that are extracellular sodium) is established through the membrane, which maintains an osmotic anti-gradient for water movement. Thus, there is a "Double effect Donnan" in action in every cell phone membrane. For examination purposes, the CMM trainee would be advised to avoid terms such as OsmoExplosion: The formal statement would be that "Sodium pumps powered by ATP decrease intracellular osmolality actively transporting sodium intracellular fluid, thus maintaining the homeostasis of the cell volume through a second Donnan effect". Donnan DiÆ Na<sup>+</sup>/K<sup>+</sup> + ATPase in maintaining a stable cell volume has been well established by a number of early authors who disable the pump using various methods and then observed while the cells are reduced and broken. For example, Russo et al (1977) used hypothermia to stop all cellular metabolic activity and thus abolish ion pumping. The rat liver slices were incubated at 1 Å C for 90 minutes and then examined under an electron microscope and compared to normopermic controls. With ion pumps disabled, the cells have increased in size markedly. Their water content has increased by about 60% and their sodium content more than quadrupled. The Gibbs-Donnan effects in addition to the cellular scale in addition to influencing the confusing ATP-Pump-Pump-Infected cell environment, the Gibbs-Donnan effect also influences other macroscopic environments, and through a detailed discussion of these issues falls outside the scope of this chapter, it would be stubborn to ignore these applications of the concept altogether. In short, wherever a membrane separates the compartments and isolates a non-dispersible substance within one of them, we can find some application of the Gibbs-Donnan effect. In Australia, Kerry Brandis' VivaÆ Physiology is usually the first detailed introduction to this concept he meets after leaving Med school, and the example discussed below has been elaborated from his excellent notes on the subject. If one demands something more substantial from the published literature and is not willing to pay for the book by Brandis, Nguyen & Kurtz (2006) - he produced an excellent revision of the subject, settling with a dense grove of mathematical derivations. To retain some vestiges of focus of the examination, these have been omitted from the discussion below. In short, we are again presented with two compartments, this time interstitial and intravascular. Let's understand them with physiologically plausible electrolyte concentrations. All the ions are turning. There are no forces moving them around. Now it allows you to add some Å as before. Now, there's an electrostatic force pushing chloride out of the intravascular compartment. As a result, more chloride is collected in the interstitial fluid. The same force is to attract sodium into the intravascular compartment. This competes with the concentration gradient. In order to make the concept easier to understand, the author used graphic design at kindergarten level, representing electrochemical gradients with coloured slopes. You can almost imagine small ions slipping through them. The attractiveness of the anion protein for sodium competes with the concentration gradient sucking it into the interstitial compartment. At a certain concentration, a sort of equilibrium is reached. Of course, actually. It's not a real balance. There is still a concentration of uneven particles on both sides of the membrane. It is reached a balance between the gradient of concentration and electrostatic gradient, electrostatic. It's still water to consider. Water is osmotically attracted in the vascular compartment. The movement of the water therefore diluted the concentration of ions, and there would be a change in their concentration gradients. So there is no stable state. There is movement of some ions out of intravascular space, but to the Gibbs-Donnan balance there are even more particles in vascular compartment, exercising oncotic pressure. The oncotic force that sucks the water in the capillaries is opposed by the capillary hydrostatic pressure, which is applied by the pumping action of the heart. If this pressure becomes too large (for example if the heart fails and the widespread venous pressure increases) the capillary hydrostatic pressure exceeds the oncotic pressure of the plasma and forces the water out of the vascular compartment. The edema derives. The distribution of ions in interstitial and intravascular sectors can be expressed in terms of a coefficient that describes the distribution of the IOM in the interstitial fluid as a proportion of its concentration in the plasma. This is generally indicated as the Gibbs-Donnan factor. The value of this factor for monovalent cations is 0.95 (ie the concentration of sodium in the interstitial liquid is 0.95 is concentration in plasma). For monovalent anions, its 1.05. Cots gradients like calcium are partially linked to proteins, and the Gibbs-Donnan effect applies only to ionized forms. For them, the factor is 0.90 (and conversely 1.10 for gradient anions).

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