

4. Treatment options for Diabetes insipidus? MOA, mechanism of action? How do vasopressin alter V1 and V2 receptors?

Treatment options for Diabetes Insipidus is Vasopressin. The most important action of vasopressin is its antidiuretic action on the collecting ducts of the kidney. Vasopressin binds to V2 receptors on the cell surface of tubular cells, initiating an intracellular cascade which results in the generation of the water channel, aquaporin-2. Vasopressin acts on V1, V2, V3, and oxytocin-type receptors (OTR).

V1 receptors are found on vascular smooth muscle of the systemic, splanchnic, renal, and coronary circulations. They are also located on myometrium and platelets. These G-protein-coupled receptors activate phospholipase C via Gq G-protein, which ultimately leads to an increase in intracellular calcium. The major effect is to induce vasoconstriction, the magnitude of which is dependent on the vascular bed. In the pulmonary circulation, vasodilation is produced via nitric oxide release.

V2 receptors are predominantly located in the distal tubule and collecting ducts of the kidney. These G-protein-coupled receptors stimulate Gs G-protein to activate adenylyl cyclase, increasing cAMP, causing the mobilization of aquaporin channels. These channels insert into the apical membrane of the distal tubules and collecting duct cells. V2 receptors are essential for plasma volume and osmolality control. Their presence on endothelial cells induces the release of Von Willebrand Factor (vWF) and Factor VIII:coagulant (FVIII:bc). vWF protects FVIII from breakdown in plasma and is important in binding platelets to the site of bleeding.

5. A patient underwent total thyroidectomy for a benign thyroid mass 1 month PTA. Her lab tests showed elevated TSH and low FT4. What medication would you give? What is its MOA? What are the available preparations and proper instruction for its intake? What would you monitor after starting the medication to check for the adequacy of its dose?

Medications that you can give are the Levothyroxine (LT4). T4 and T3 bind to thyroid receptor proteins in the cell nucleus and cause metabolic effects through the control of DNA transcription and protein synthesis. For the preparation of choice for replacement & suppression therapy because of its stability, uniform content, low cost, long half-life (7 days), and conversion to produce both T3 & T4, and Liotrix - synthetic preparation of T4 and T3 in a 4:1 weight-based ratio. These hormones enhance oxygen consumption by most tissues of the body and increase the basal metabolic rate and the metabolism of carbohydrates, lipids and proteins. A 4:1 combination of synthetic T4 and T3, also expensive with the same disadvantages as liothyronine. You should still check the T3 and T4 levels of the patient while giving the medication.

# Answers To Endocrine Case Study

**Paul W. Ladenson**

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**Child and Adolescent Psychiatry** G. Pirooz Sholevar,1977      *Endocrinology & Metabolism, March 2001 - February 2002* Paul W. Ladenson,2000-10      *Neurology Continuing Education Review* James H. Halsey,Paul D. McDowell,1977      *Heart and Vascular Systems Basic Sciences* Robert A. Woodbury,Clinton B. Nash,Frank D. Sticht,1970      **Plastic and Reconstructive Surgery** Lars M. Vistnes,Donald R. Laub,Richard F. Ott,1978      **Case Studies in Critical Care Nursing** Sheila Drake

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