The responses of free water clearance to 2 hours of step increase of plasma hypertonicity induced by high salt ingestion and those free water clearances to the same hypertonic level, but with lengthened duration through vasopressin administration, have been shown to be very different. When plasma hypertonicity was induced by high salt ingestion, no change in sodium reabsorption sites was found for either the proximal tubules, where the bulk of sodium reabsorption occurs in the kidney, or in the cortical collecting tubules, where normally vasopressin causes physiologic sodium reabsorption. As the amount of salt–induced negative free water clearance is the same as that of the control to vasopressin action, it again suggests that major sodium reabsorption sites in the kidney did not respond to the passive increase in their reabsorption demands. The finding that endogenous and exogenous vasopressin can shift water between the cell and plasma without having access to intrarenal nephrons does not support the conventional osmometric theory for sampling systemic plasma by vasopressin released from the posterior pituitary. From urinary analysis, the short–term renal excretion of sodium following either high salt ingestion or vasopressin administration shows the same reabsorption benefits for hypertonicity as there is a significant decrease in the negative sodium balance due to the systemic plasma hypertonicity under the influence of sodium reabsorption at various renal reabsorption sites of the kidney. 2. Body Fluid Compartments 2.1. Intracellular Fluid (ICF) 2.2. Extracellular Fluid (ECF) 3. Impact of High Salt Intake 3.1. Sodium and Water Balance in the Body 4. Mechanisms of Water Shifting 4.1. Osmosis and Tonicity 4.2. Renal Regulation of Water Balance 5. Conclusion 5.1. Summary of Key Points