Chapter 4

RENAL PHYSIOLOGY

GLOMERULAR FILTRATION

This depends upon higher functional pressure in afferent arterioles. Filtration barrier is formed by endothelium with slit pores, basement membrane and epithelium with its interdigitating podocytes.

Filtration of macromolecules depends upon their size, shape and electrical charge. Electrostatic hindrance is provided by fixed negatively charged component of glomerular capillary wall. Podocytes have surface coating of sialoproteins which are negatively charged.

Normally about 20% of plasma appears as filtrate which contains all diffusible and ultrafiltrable substances present in plasma.

TUBULAR REABSORPTION

Proximal convoluted tubules reabsorb 80% of glomerular filtrate. Glucose, amino acids and proteins are almost completely reabsorbed. Potassium is completely reabsorbed in proximal convoluted tubule and secreted in distal tubule in exchange for sodium. Chloride reabsorption is passive.

Bulk of energy expenditure by kidney is related to sodium reabsorption in which sodium potassium activated ATP is involved. Isotonic reabsorption of water coincides with sodium reabsorption and depends upon raised oncotic pressure of plasma proteins in peritubular capillaries. This arises as result of haemoconcentration following filtration in glomeruli.

Bicarbonate reabsorption occurs actively and depends upon bicarbonate stimulated ATPase at brush border.

Tubules preferentially reabsorb certain essential amino acids. Renal aminoaciduria may be due to specific defect in carriers or enzyme system or may be part of generalized disorder of tubular function.

Phosphates are partially reabsorbed in proximal convoluted tubule. Remaining phosphate is filtered in distal convoluted tubule by H^+ ion secreted there and help in H^+ ion excretion.

CONCENTRATION AND DILUTION

Countercurrent Multiplier System of Urinary Concentration:

There is active transport of sodium chloride out of ascending limb of loop of Henle into hypertonic medulla. Fluid first follows down descending limb which has high water permeability but low permeability to sodium. Osmolality of contents rises from 300 mOsmol at entry to 1500 mOsmol at tip of loop. Thin ascending limb is relatively impermeable to water but reabsorbs sodium chloride. Collecting duct is permeable to urea which is then trapped at higher concentration in inner medula. It contributes to medullary hypertonicity.

Dilution of urine occurs in ascending limb of loop of Henle and early distal tubule. This is result of sodium reabsorption without concomitant water diffusion. There is no net addition of water to tubular fluid. When anti diuretic hormone levels are low nephron will be impermeable to water and hypotonic fluid passes through collecting tubule to be excreted as dilute urine. When ADH level is high then distal and collecting tubular walls become permeable to water. In distal tubule isotonicity with plasma is reached and in collecting tubule as they pass through highly hypertonic papillary interstitium water diffuses out of lumen down osmotic gradient into papilla as result tubular fluid becomes concentrated to same osmolality as papillary tip.

Causes of impaired diluting ability:

1. Decreased glomerular filtration rate:
    i. Chronic renal disease.
2. Endocrinal:
    i. Hypothyroidism.
    ii. Hypopituitarism.
    iii. Hypoadrenalism.
3. Syndrome of inappropriate anti diuretic hormone secretion:
    i. Secondary to malignancy.
4. Dilutional disturbance:
    (Sodium retaining states)
    i. Cirrhosis of liver.
    ii. Congestive heart failure.
    iii. Nephrotic syndrome.

HYDROGEN ION EXCRETION

Bicarbonates are buffers in body.

$H + HCo_3 = H_2Co_3 = Co_2 + H_2O$.

Kidney is instrumental in conserving bicarbonate ions and excreting H ions.

Bicarbonate is reabsorbed as result of H ion secretion into tubular lumen.

Resulting carbonic acid is dehydrated by carbonic anhydrase to carbon di-oxide which diffuses into tubular cell. There it is reconstituted into bicarbonate.

PROSTAGLANDINS AND RENAL BLOOD FLOW

Prostaglandins are synthesized in walls of blood vessels and also in renomedullary interstitium cells.

Prostaglandins are vasodilators whose releases in hypertension offsets action of angiotensin.

PGE2 is major product of interstitial cells of kidney. It opposes action of ADH on collecting ducts. Kallikrein and bradykinin action is through PGE2.

PGE causes vasodilatation of arterioles of inner cortex. Its infusion leads to increase in renal blood flow and osmotic diuresis accompanied by increased sodium and potassium excretion.

Prostacyclin produced by vascular endothelium also offsets action of pressure hormones and prevents deposition of platelets and products of coagulation.

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