D. Role of Diuretic Therapy

Many English and American authors regard a diuretic agent as the drug of choice for the treatment of chronic heart failure (Eliot et al. 1980). In Germany, diuretic therapy is initiated mainly when the prior administration of digitalis glycosides fails to produce an adequate effect. Diuretics can favorably influence one major component of heart failure: congestion. Reducing fluid in the extravascular compartments leads to a clear diminishment of congestion in various organs. The clinical condition improves substantially, dyspnea is reduced, and peripheral edema subsides.

I. Renal Function in Heart Failure

The kidney relies on an adequate arterial perfusion pressure for the excretion of sodium and water. Since renal perfusion decreases in heart failure, corresponding to the decrease in cardiac output, and since the reduction of cardiac output is associated with a fall in blood pressure, heart failure is accompanied by enhanced reabsorption of sodium and water. This results in a net increase in arterial blood volume and an elevation of venous filling pressure. The elevated filling leads to an increase in cardiac output with a normalization of renal blood flow and perfusion, thus deactivating the process of increased sodium and water retention. In severe cases of heart failure, however, the elevated filling no longer results in an improvement of cardiac pumping performance and fails to interrupt the compensatory mechanism of renal salt and water retention.

The increased sympathetic adrenergic activity and stimulation of the renin-angiotensin-aldosterone system leads to a proportionate reduction of renal perfusion as well as to renal vasoconstriction. The vasoconstrictive effect is exerted in the efferent renal arterioles distal to the glomeruli. In this way a normal glomerular filtration pressure is maintained and the hydrostatic pressure in the peritubular capillaries is reduced.

The reduction of renal blood flow is greater than the reduction in the glomerular filtration rate (GFR) (Fig. 122) (Cannon 1977). The filtration fraction, that is, the ratio of glomerular filtration rate to renal blood flow, is increased in heart failure leading to a higher protein concentration in the peritubular capillaries. Interstitial renal fluid can more readily return to the intravascular space as the hydrostatic pressure is reduced and the colloid-osmotic pressure of the peritubular system is elevated.
II. Sites of Action for Various Diuretics

The three most important diuretic agents for the treatment of heart failure are loop diuretics, benzothiadiazines and potassium-sparing diuretics such as the aldosterone antagonists. Loop diuretics (prototypes are furosemide, ethacrynic acid) act on the ascending limb of the loop of Henle whereas benzothiadiazines, the potassium-sparing diuretics, and aldosterone antagonists act on the distal tubule. Their various effects on urinary output and the excreted quantities of sodium, potassium, chloride and bicarbonate are listed in Figure 123 (Hierholzer and Kleinschmidt 1977). Table 7 includes characteristic features such as the site of action, mode of action, generic name, average daily dose, and duration of action (Schüren 1982).

III. Hemodynamic Changes after Diuretic Therapy in Acute Left Ventricular Failure

The reduction of blood volume following administration of a diuretic results in a downward trend on the ventricular function curve since the reduction of left ventricular filling pressure produces a decrease in cardiac output. Within one hour after intravenous administration of furosemide, left ventricular filling pressure is reduced by 25 to 35 percent. In the majority of cases this is associated with a decrease in cardiac output but only a minimal reduction of arterial blood pressure.