In patients with chronic renal failure the sodium balance can be maintained within normal limits, provided that the glomerular filtration rate (GFR) decrease is adequately compensated by increased fractional sodium excretion. Clinical observations indicate that the increase of fractional sodium excretion (FENa) is not an unlimited process and that the sodium osmotic diuresis of residual nephrons depends also on the salt intake.

A positive sodium balance (manifested by body weight gain) in patients with chronic renal failure can be caused by inadequate increase of FENa. In some other cases FENa reaches very high (maximal) values, and the positive sodium balance is caused predominantly by inadequate sodium intake.

From the therapeutical point of view it is important to distinguish these two situations with respect to the administration of high doses of furosemide or restriction of sodium intake.

The adequate value of fractional sodium excretion -(FENa)adq – can be calculated according to the following formula:

\[
\text{(FENa)adq} = \frac{0.085}{Ccr} \times \frac{\text{UNaV}}{PNa} 
\]

where \(ka\) denotes sodium intake (mmol/24 h) and \(Ccr\) denotes endogenous creatinine clearance (ml/s). The value of (FENa)adq is expressed in percent.

This formula is derived as follows: If the urinary excretion (UNaV) and GFR are related to the same time unit, and the plasma concentration (PNa) is expressed in millimoles per milliliter, then FENa is calculated according to the known formula:
Since 24 h = 86,400 s, and if UNaV is expressed in millimoles per 24 h and PNa in millimoles per liter, then the following adaptation of the formula 2 must be made:

Under conditions of a sodium metabolic steady state when the extrarenal sodium loss is small in comparison with ⅛, GFR is measured as CCT, and if for Pn⅛ a normal average value (140 mmol/l) is substituted, formula 3 can be arranged as presented by equation 1.

For instance, if in a patient with Ccr =0.1 ml/s In⅛ oscillates between 100-150 mmol/24 h, then FEn⅛ which could guarantee a stabilized state of sodium balance is: 8.3-12.5%.

Figure 1 shows the relationship between Ccr and FENa in 66 patients with chronic renal disease not receiving diuretics. If this relationship is focussed with respect to the maximal observed values of FENa, it appears that in patients with Ccr below 0.17 ml/s (10 ml/min) FENa can reach values of 25-30%.

If in a patient with positive sodium balance FENa does not reach high values, it can be assumed that the fractional basis of χ² analysis we have found that the positive effect of furosemide (expressed as Δ FENa) was significantly higher (p < 0.001) in patients with FEn⅛ below 16% than in those with higher values of FENa fig. 2).

The obtained results suggest that if in the patient with chronic renal failure a positive sodium balance develops, FENa should be investigated. If FENa does not reach high values (above 16%), the administration of furosemide will be probably helpful. In patients with sodium retention and high (or maximal) values of FENa the restriction of sodium intake is necessary.

From the practical point of view it should be remembered that for the estimation of the actual value of FENa the quantitative collection of urine is not necessary since this value can be calculated as the ratio:

\[ \text{sodium reabsorption of Na in residual nephrons is not adequately adapted. In such cases the administration of high doses of furosemide is theoretically justified. We have analyzed from this point of view the influence of furosemide (250-1,000 mg) in 28 patients with chronic renal failure with respect to the initial value of FENa. On the} \]

100.
E⅛a Per UCr PNa
(4)