Urine Osmolarity and the Excretion of Prostaglandin E2 in Man

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1,000 1
500- 1
100 200 PGE2(pg/ml)

Fig. 1. Correlation between urine osmolarity (A), urine flow rate (B) and urinary concentrations of PGE2 during 11 h of fluid deprivation in 2 healthy subjects under basal conditions (●, △) and following pretreatment with indomethacin (IND; •, A).

We have studied urine flow rates (ml/min), urine osmolarity (mosm/1), and the urinary concentration (pg/ml) and excretion rates (ng/2 h) of prostaglandin E2 (PGE2) in 2 healthy subjects after an oral water load (1,000 ml) and during subsequent fluid deprivation for 11 h. Both with and without inhibition of endogenous prostaglandin synthesis with indomethacin (150 mg/day for 3 days) a rise in the concentration of urinary PGE2 was seen during anti-diuresis in parallel with urinary osmolarity (without indomethacin: r = 0.85, p < 0.001; during indomethacin: r = 0.91, p < 0.001; fig. 1A) and in reciprocity with urine flow rate (without indomethacin: r = -0.73, p < 0.01; during indomethacin: r = -0.68, p < 0.01; fig. 1B). No correlation was seen between urine flow rate and the excretion of PGE2 during antidiuresis.
High urine flow rates (> 4 ml/min) increase prostaglandin excretion in a nonspecific manner in the presence of unchanged prostaglandin concentrations [Kirschenbaum and Serros, 1980]. In addition, the excretion of PGE2 seems to be unchanged during antidiuresis in the presence of a rise in PGE2 concentrations. The latter may be induced either by endogenous ADH or simply by prolonged contact time in a diffusion-regulated system [Kirschenbaum and Serros, 1980]. Thus studies dealing with determinations of urinary PGE2 in man need to define the respective state of hydration and to report both the concentrations and the excretion rates of PGE2, since omission of either parameter may lead to misinterpretation.

References