COMMUNICATIONS:

2. WATER IN DIAGNOSIS AND TREATMENT

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From the time of immemorial, water had been used externally to reduce the temperature in fever and/or waking up the patient from sudden unconsciousness, and internally as a carrier of various drugs, the discovery of the specific water-channel protein aquaporin 1 (AQP1), and the subsequent identification in mammals of nine other members of the aquaporin family, have suggested that, in many cases water movement across membranes is facilitated by water channels.[1,2] A few members of the family can transport ions. Some, like AQP1 are expressed in various tissues, whereas others, like AQP2, seem to be confined to a single site. Most aquaporin seem to be constitutively expressed, but the expression of some can be regulated, e.g. in the kidney high concentrations of vasopressin increase concentrations of AQP2 and AQP3. Recently we have shown that water diuresis renography is useful in obstructive uropathy,[3] and side-effects of frusemide and mannitol can be avoided.

Jens Jordan and colleagues reported that oral water has substantial pressor effects[4] and observed a volume-dependent pressor response in patients with autonomic failure with 240 ml of water, the rise was about 15 mm Hg less than with 480ml,[5] they speculated that gastric distension may have been the stimulus that activated sympathetic reflexes, as previously described in normal people whose stomach had been distended with a barostat and whose sympathetic nerve activity had been directly measured with micro neurography.[6] In 1978, Kossoff and colleagues showed that the ultrasonic examination of upper abdomen is facilitated through the liquid-filled stomach[7] and recently we showed that water is a good echo-contrast in gastric emptying studies.[8] However, the precise mechanisms causing the pressor response to oral water, especially in patients with sympathetic denervation still remain to be determined.[9] To avoid urinary retention secondary to impacted pelvic mass, one should limit fluid intake before sleep.[10] King et al. studied two unrelated women with a deficiency of aquaporin-1and found that they had impaired urinary concentrating ability, suggesting that aquaporin-1 has a physiologic role in renal function. They performed renal and bladder ultrasonography and measured the glomerular filtration rate (GFR)) with Tc-99m DTPA (technetium-99 metastable diethylenetriamine penta acetic acid) by nuclear medicine techniques.[11] The pathophysiology associated with the aquaporin family of water-channel proteins includes mutations in some patients with nephrogenic diabetes insipidus (AQP2) and cataracts (AQP0) and abnormal transport of aquaporin-5 in patients with Sjogren's syndrome. Aquaporin-1 is essential for maximal urinary concentrating ability.

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