

Schrier RW, Gross P, Gheorghide M, et al. Tolvaptan, a selective oral vasopressin V_2 -receptor antagonist, for hyponatremia. *N Engl J Med.* 2006;355:2099-112.

Editor's Comment:

Management of SIADH in children is primarily accomplished by fluid restriction. In critical situations slow intravenous infusion of 3% saline may be

considered in an amount calculated to increase the serum sodium concentration to values that ameliorate symptoms (approximately 125 mEq/L) while carefully monitoring urine output.² Very rapid increase in serum sodium concentrations may lead to central pontine myelinolysis. The non-peptide antagonists of the V_2 receptor have not been examined or approved for use in children as yet, but would appear to be promising therapeutic agents that have been termed "aquaretics." In addition to the renal V_2 receptor,

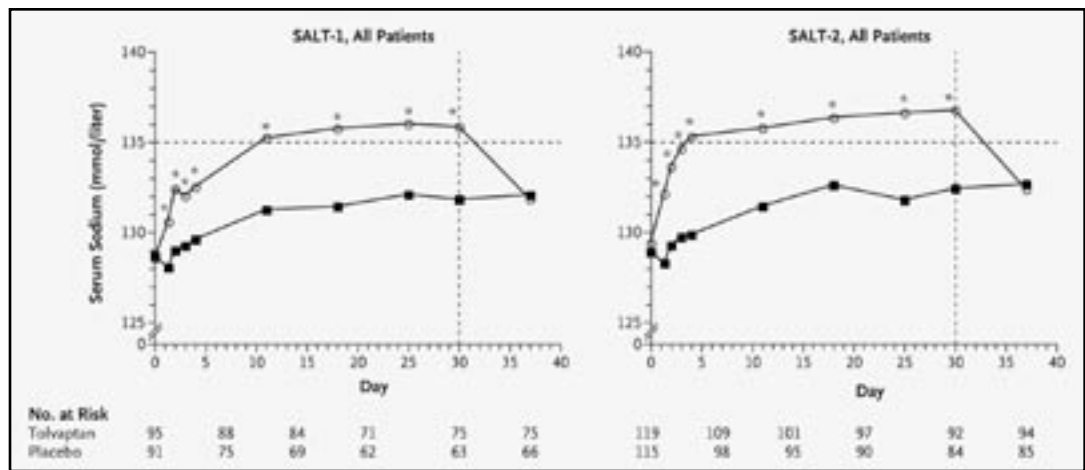


Figure 2. Mean serum sodium concentrations attained with tolvaptan (circles) and placebo (squares). Reprinted with permission from: Schrier RW, et al. *N Engl J Med.* 2006;355:2099-112. Copyright © MMS. 2006. All rights reserved.

there are V_{1a} and V_{1b} receptors that mediate the vasoconstrictive and adrenocorticotropin-releasing properties of ADH.

Allen W. Root, MD

References

1. Hays RM. *N Engl J Med.* 2006;355:2146-8.
2. Robertson GL. Clinical disorders of the posterior pituitary. In: Pescovitz PH, Eugster EA, eds. *Pediatric Endocrinology: Mechanisms, Manifestations, and Management.* Philadelphia, PA: Lippincott Williams & Wilkins;2004:90-107.

Growth Hormone Treatment in Cystic Fibrosis

This multi-center study assessed auxological, respiratory, bone health, and quality of life variables in 61 pre-pubertal children with cystic fibrosis (CF) who were randomized to receive either growth hormone (GH [0.3 mg/kg/wk]) or no GH for one year. At the end of one year, there was cross-over and those who received GH stopped therapy and those not on treatment started GH therapy. Both groups were then followed for a second year. Significant improvements in gain in height velocity, weight, lean body mass, bone mineral content, quality of life, and hospitalization rates were demonstrated in the subjects treated with GH. Improvements were also maintained following discontinuation of GH.

Hardin DS, Adams-Huet B, Brown D, et al. Growth hormone treatment improves growth and clinical status in prepubertal children with cystic fibrosis: results of a multicenter randomized controlled trial. *J Clin Endocrinol Metab.* 2006;91:4925-9.

Editor's Comment: *Hardin is one of the few pediatric endocrinologists who is successfully addressing the problems of chronic severe childhood illness on growth, puberty, bone health, and quality of life. She and her co-authors are to be congratulated on this publication, which not only reports impressive positive results of GH*

therapy in children with CF, but can also be considered a notable achievement in terms of interdisciplinary collaboration. A defect in GH action is predictable from the effect of chronic infection and inflammation in children with CF. Insulin-like growth factor (IGF)-I levels have also been shown to be correlated with BMI and disease activity score, which as stated in this paper, may relate to long-term morbidity and mortality.

It is pertinent to ask why convincing results such as these are apparently not changing clinical practice more rapidly. Poor interdisciplinary discussions and interchange must be responsible. Sub- or super-specialization within pediatrics may be considered synonymous with progress, but barriers can be constructed which make interspecialty and joint clinical management difficult. Pediatric endocrinologists are in a position to collaborate with many subspecialists, such as gastroenterologists, rheumatologists, hematologists, etc. Of course, stretched resources may make this difficult. However, as Hardin and her collaborators have demonstrated in this important study, the patient may have a great deal to gain from closer working relationships between colleagues in different pediatric disciplines. Well done!

Martin O. Savage, MD