α -Adrenolytic Treatment of the Congenital Neuropathic Bladder

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Key Words

Neurogenic bladder dysfunction
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Residual urine
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16 patients (11 children and 5 adults) with complications due to congenital neuropathic bladder were treated by phenoxybenzamine blocking the α -adrenergic receptors. Causes of the bladder disorder were myelo-meningocele, sacral teratoma, and unknown neurological deficiency. The patients received a longterm oral treatment of phenoxybenzamine in a dose of 0.3–0.5 mg/kg body weight/day, the longest therapeutic period lasting up to 1 year. Adverse side effects were seen occasionally in the form of minor orthostatic trouble during the first weeks and a permanent loss of ejaculation in men.

All patients were examined before and under treatment by IVP, voiding cystourethrogram, and cystometry with the following results: (1) pathologically elevated urethral and intra-vesical pressure levels present in most cases were normalized or at least significantly decreased; (2) residual urine in 11 patients was abolished completely in 6 and diminished in 4 cases; (3) reflux in 8 patients disappeared in 4 and was functionally improved in 3 cases; dilatation of the upper urinary tract stases was relieved in 4 of 7 patients and improved in one; (4) continence was restored in 3 of 8 trainable patients out of a total of 13 and was improved in 3 others, and (5) radiologically we observed a relaxation of the trigone and the bladder wall when contracted. Phenoxabenzamine therapy was all together successfull in 15 out of the 16 cases. A constantly beneficial effect was obtained in the presence of a lower motor neuron lesion whereas an improvement in upper lesions was encountered only in cases with elevated intra-vesical pressure. A complete failure was noted in a patient with automatic bladder in which upper urinary tract complications remained unchanged and the well-trained reflex voiding was also disturbed.