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α -Adrenergic Blockade in the Diagnosis of Detrusor Instability Secondary to Infravesical Obstruction

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Key Words

Infravesical obstruction

Detrusor instability

α -Adrenergic blockade

Abstract

Detrusor instability secondary to infravesical obstruction is probably caused by overactive stretch receptors in the bladder wall. Since recent studies have shown that α -adrenergic blockade with phenoxybenzamine reduces the intravesical pressure under micturition, we undertook this investigation in order to see if phenoxybenzamine has an effect on detrusor instability caused by infravesical obstruction. We found that the detrusor instability was abolished by phenoxybenzamine, which supports the 'stretch receptor theory'. Further, phenoxybenzamine is probably a good test in the diagnosis of detrusor instability secondary to infravesical obstruction.

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Introduction

The cause of detrusor instability in patients with infravesical obstruction is not fully understood, but is probably caused by a lower threshold of the supposed stretch receptors in the bladder wall (5). We have previously found that these stretch receptors could be inhibited by lignocaine applicated intravesically (6). The detrusor instability disappeared, although only for a short period. With the use of lignocaine applicated intravesically we found that it probably is possible to find the patients with secondary overactive detrusor function. These patients will benefit from surgical treatment since the detrusor instability disappears after operation, while patients with idiopathic detrusor instability will not benefit from surgical treatment.

Since recent studies have shown that α -adrenergic blockade with phenoxybenzamine reduces the intravesical pressure under micturition [4], we undertook this investigation in order to see if phenoxybenzamine has an effect on detrusor instability secondary to infravesical obstruction.

Patients and Methods

The study was based on patients with prostatic hypertrophy and detrusor instability. Excluded were patients with known cerebral diseases, uncompensated heart disease, previous myocardial infarct, angina pectoris, peripheral vascular diseases, and hypotension. Altogether, 10 men (median age 70 years; range 61–81) took part in the study. The patients were examined with uroflow and cystometry 3 months, 1 month, and just before operation, and 3 months after operation. Cystometry was carried out with CO₂ as the filling medium at a continuous rate of 200 ml/min through a transurethral 16FG Foley catheter. We defined detrusor instability as the presence of involuntary uninhibited detrusor contractions during cystometry that exceeded 15 cm

H20. After the first examination the patients started a treatment with Phenoxybenzamine®, 10 mg twice a day, until 1 month before operation.

Results

1 patient developed dizziness so that the treatment was stopped. 2 patients were so satisfied with the treatment that they did not want an operation. 2 patients refused the postsurgical investigation and 1 had still

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infravesical obstruction. Altogether, only 4 patients fulfilled the study. The detrusor instability disappeared in 3 of these patients while they were treated with phenoxy-benzamine and reappeared after the treatment was stopped. In these patients the detrusor instability vanished after surgery. In 1 of the 4 patients phenoxyben-zamine had no effect on the detrusor instability and the detrusor instability persisted after surgical treatment.

Discussion

The benefit of treatment with phenoxybenzamine in patients with prostatic hypertrophy has been described previously in several studies [1, 3, 4]. Caine et al. [3] found that 78% of 171 men with benign prostatic obstruction derived symptomatic relief. Gerstenberg et al. [4] found a lowering of urethral resistance, vesical opening pressure, maximum intravesical pressure, and detrusor contraction pressure in 9 men treated with phenoxybenzamine. Detrusor instability found in 3 of the patients before treatment disappeared in 1 and appeared at a higher bladder volume in 2 during treatment. It was not described whether the detrusor instability returned to the status before treatment when the medication was stopped. Neither was it described whether the detrusor instability disappeared after operation for prostatic hypertrophy.

In our study we found that in the patients where phenoxybenzamine abolished the detrusor instability, the detrusor instability disappeared postoperatively. The exact mechanism of phenoxybenzamine in removing the detrusor instability is not known, but by blocking the α -adrenergic receptors in the bladder neck, in the prostatic capsule, and in the prostatic adenomas [2], the urethral

resistance and, as a consequence, the intravesical pressure are reduced [4]. The reduced intravesical pressure probably causes a lesser distension of the bladder and thereby supports the theory that detrusor instability accompanying infravesical obstruction is caused by overactive stretch receptors in the bladder wall. Further, phenoxybenzamine is probably also a good test in the diagnosis of detrusor instability secondary to infravesical obstruction.

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