Bladder instability in patients with prostatic hypertrophy

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Summary

In 29 patients with urinary obstruction due to prostatic enlargement, medium-fill water cystometry was performed before prostatectomy and again 3 months after. A high incidence of detrusor hyperreflexia was observed pre-operatively, but findings were essentially normal 3 months after resection of the prostate. Possible mechanisms to explain these findings are developed. However, it was clear that the cystometric findings were not related to prostatic size.

Introduction

The precise cause of urinary retention associated with prostatism is not clear. The detrusor response to outflow obstruction is varied. It has been previously demonstrated that prostatic bulk is one component in the mechanism of bladder outflow obstruction (Appell *et al.*, 1980), as it may constitute a barrier to low-pressure urinary flow which is unaffected by maximal urethral myogenic relaxation. However, the clinical degree of prostatic enlargement is poorly related to the severity of outflow obstruction (Turner-Warwick *et al.*, 1973). Uroflowmetry (Shoukry *et al.*, 1975), combined urodynamic (Turner-Warwick and Whiteside, 1970), and uro-radiological (Shopfner, 1965) studies have made inroads into the understanding of obstructed micturition.

The detrusor responds to outflow obstruction in a variety of patterns and the question arises whether these differing patterns represent different reactions to the same mechanical obstruction or represent different stages in the development of the disordered vesico-urethral function. The purpose of this study was to assess detrusor function during clinical mechanical obstruction of bladder outflow by an enlargement of the prostate.

Patients and methods

Twenty-nine patients with clinical prostatic enlargement were studied at the time of proposed

transurethral prostatic resection (TURP). Eighteen of these had been admitted in urinary retention while the remaining 11 had varying degrees of prostatism. None of the patients was taking any medication and none received pre-study medication. None of the patients presented evidence of neurological disease.

Detrusor reflex function was assessed by means of room temperature medium-fill water cystometry performed in the supine position through a 12-Fr (Charrière) catheter containing two 1-mm perfusion apertures. Perfusion pressures were monitored by a standard side-arm transducer and displayed on an X-Y recorder.

All patients had repeat cystometry 3 months after TURP.

Results

Histological examination of the resected prostatic tissue revealed benign hyperplasia in all cases and all pre-operative urine cultures showed no growth of bacteria. Before TURP, cystometry revealed detrusor hyperreflexia in 12/18 patients with urinary retention and in 6/11 patients with prostatism (Table 1). Normal cystometry implied normal sensation of fullness and the presence of a detrusor reflex which could be voluntarily suppressed on command. Detrusor hyperreflexia was defined as a detrusor reflex evoked at low bladder distention which could not be voluntarily suppressed, while detrusor areflexia was defined as the absence of the detrusor reflex during bladder filling. Cystometry performed 3 months after TURP (Table 1) demonstrated no detrusor hyperreflexia in any patient who had been in urinary retention before TURP. However, 10 of the patients not in retention before TURP with detrusor hyperreflexia continued to manifest detrusor hyperreflexia postoperatively.

No relationship could be found between the cystometric findings and prostatic size.

Discussion

The results of cystometry in this group of patients

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TABLE 1 Cystometry in patients before and 3 months after transurethral prostatic resection (TURP)

Patient status (n.)	No. before	No. after	Prostatic weight (g)
(a) in urinary retention (18)			
— hyper-reflexia	12	0	11; 12; 15; 16; 18; 18; 18; 19; 22; 25; 25; 35.
— areflexia	2	2	11; 38.
— normal	4	16	10; 22; 30; 70.
(b) with prostatism (11)			
— hyper-reflexia	6	1	11; 15; 18; 18; 34; 38.
— areflexia	0	0	
— normal	5	10	12; 16; 24; 30; 30.

support the findings in prior studies utilizing water cystometry (Andersen, 1976) and gas cystometry (Andersen and Bradley, 1976) of a high incidence of detrusor hyperreflexia in patients with prostatic enlargement.

The high incidence of detrusor hyperreflexia in patients with bladder outlet obstruction due to prostatic enlargement but no obvious neurological disease may be triggered by anatomical distortion of the heavily sensory innervated posterior urethra. Andersen and Bradley (1976) likened this compression effect on sensory nerve endings to an entrapment neuropathy. If this were true, surgical reduction of the prostatic mass causing the compression would be expected to decompress the nerve endings and allow a change in the detrusor reflex function. This is precisely what was found when cystometry was performed 3 months after surgery, i.e. a return to a normal cystometric response. This is further supported by the recent study by Rao *et al.* (1979) who found that 60% of men subjected to prostatectomy had unstable bladders, but that 82% had normal studies several months after prostatectomy.

This study, however, clearly demonstrates that prostatic size alone does not relate to the cystometric findings of detrusor hyperreflexia. The interaction of smooth muscular tone of the posterior urethra under sympathetic nervous control and the prostatic mass is involved in production of outlet obstruction in this patient group, which helps to explain why patients with a lesser degree of prostatic enlargement may develop urinary retention while others with massive prostatic enlargement do not

(Appell *et al.*, 1980). The detrusor instability found in these patients appears to be related to the total outlet resistance and not to prostatic mass effect alone, and helps to explain the variety of patterns of detrusor response to outflow obstruction.

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