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MUSCLE CELL JUNCTIONS OF THE DETRUSOR IN PATIENTS WITH STRESS URINARY INCONTINENCE (SUI), BLADDER OUTLET OBSTRUCTION (BOO) AND HYPERREFLEXIC NEUROGENIC BLADDER DYSFUNCTION (NBD)

Aims of Study

From molecular biology studies different cell junctions are known, including adhesion junctions and gap junctions. These junctions can be identified by different proteins at the cell membrane. In papers [Elbadawi et al. J. Urol, 1993] dealing with ultrastructural detrusor changes in various bladder dysfunctions Elbadawi proposed changes in cell junctions in the detrusor of patients with detrusor instability and with bladder outlet obstruction.
The aim of this study was to evaluate which cell junctions exist in the human detrusor and if the presence of these cell junctions correlates with a known bladder dysfunction.

Methods

Detrusor biopsies were taken from 45 patients with videourodynamically proven urinary stress incontinence (n=7), BOO (n=6) and NBD (n=32). All biopsies were immunhistochemically stained for Pan-Cadherine, α-Catenine, β-Catenine, γ-Catenine (representing proteins of cell-cell adhesion junctions), β1-Integrine (protein of cell-matrix adhesion junctions) and for Connexin 43 (protein of gap junctions). The results were evaluated quantitatively by 2 examiners without prior knowledge of clinical and urodynamic data.

Results

Pan-Cadherine, α-Catenine, β-Catenine and γ-Catenine could not be identified in any of our biopsies. All biopsies were stained for β1-Integrine without any difference between the various groups of bladder dysfunction. A limited amount of Connexin 43 was identified in the SUI group, but it was lacking in the BOO and NBD group.

Conclusions

Muscle cell – muscle cell adhesions could not be identified in the human detrusor using antibodies for Pan-Cadherine, α-Catenine, β-Catenine, γ-Catenine, but we were able to show a dominance of muscle cell - matrix adhesions. Contrary to postulated concepts we were unable to find a reduction in adhesion junctions and an increase in gap junctions in biopsies of patients with NBD. We therefore conclude that detrusor cells may only be coupled mechanically (adhesion junctions) or if electrically coupled no Connexin 43 proteins are involved.