Stress Urinary Incontinence in Women

II. Abnormalities of Glycogenolysis in Tissues Related to the Lower Urinary Tract

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(Accepted April 5, 1996)

The aim of the study was the investigation of the biochemical condition of elements likely to directly participate in active dosing of the urethral lumen. We estimated glycogenolysis in urinary bladder, perivesical connective tissue and levator ani muscle (LAM) samples obtained intraoperatively from 80 stress incontinent women. Glycogen content as well as activities of active and total glycogen phosphorlyase and acid exo-1,4-alpha-glucosidase were measured. Material from the urinary bladder and perivesical connective tissue was insignificantly altered, and glycogen contents in the bladder (2.03±1.38 g/100 g wet tissue) were considered to be normal. In the LAM glycogenolysis was much more activated than in other tissues (p<0.001 by Fischer's exact test). Of LAM specimens 78% (22/28) revealed imbalanced biochemistry of glycogen with activation of hydrolytic decomposition. We conclude that stress urinary incontinence in women is frequently associated with metabolic alterations in the perirethral striated fibres. This study indirectly supports our recent hypothesis on the pathogenesis of the disease in terms of muscle fibre type transitions.

Introduction

Current concepts on urinary continence mechanism in women remain under debate, however, they uniformly underline the existence of two distinct components involved in this process: a passive - connected mainly with intrinsic elements of the urethral wall, and an active - associated with contractile structures related to the lower urinary tract [1, 2].

There is reason to believe that dysfunction of the active component may particularly contribute to the pathogenesis of genuine stress urinary incontinence (GSI). Passive uninhibited leakage of urine due to rises of intraabdominal pressure cannot be sufficiently prevented by additional occlusive forces which should then be generated.

To date few studies on biochemical changes in GSI have been concentrated on collagen in the area of continence or in organs remote from it. The aim of this investigation was to focus on the biochemical condition of elements likely to directly participate in active closing of the urethral lumen. In addition, the study was planned to test our recent hypothesis on muscle fibre type transitions of the pelvic floor as a possible background of GSI [3]. Examination of glycogen decomposition dynamics was the method of choice for evaluation of intracellular metabolic requirements [4].

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Subjects and methods

Our study, approved in advance by the Commission on Human Studies at the School of Medicine, Bialystok, involved 90 women with GSI who gave their informed written voluntary consent. Controls were not examined since available data allowed to evaluate the biochemical condition of analyzed tissues in healthy subjects (see Discussion).

To be included in the study the patient had to give a typical history and present clinical symptoms of stress incontinence, a Marshall test had to be positive, and maximal urethral closure pressure had to be 40 cm of water or less [5]. The observed leakage conformed to the second degree of severity according to Ingelman-Sundberg [6]. For differential diagnosis in some women cystometric, cystoscopic, and polycystographic examinations were performed.

GSI surgical correction was the Marshall–Marchetti urethrovesical suspension modified by Burch, Durfee or Łotocki [7] and/or posterior colporrhaphy. Samples for determinations were obtained from the outer muscular layer below the vesical dome, the loose connective tissue of the Retzius space, and the medial margin of the levator ani muscle (LAM). These tissues were examined because the outer layer of the anterior vesical wall possesses fibres which penetrate the vesical neck and urethra [8], the retropubic connective tissue transmits intraabdominal pressure to the proximal urethra [9], and puborectal fascicles of the LAM exert external compression on the urethra [10]. Obtaining samples caused no significant tissue loss. When appropriate, single haemostatic sutures were applied.

Biochemical determinations were conducted immediately after sampling or later. In such cases the specimens were frozen (−20 °C), which had no influence on glycogen or enzymatic activities [11]. Samples obtained from 8 women at the beginning of the study were used to verify the methods, and 2 patients with concomitant detrusor instability were excluded. Thus, we present the findings from 80 patients aged 31–74 years (mean 50.9±8.9), with parity: 0–7 times (mean 2.9±1.4). Their GSI lasted from 3 to 240 months (mean 47.8±42.1). As no quantitative correlation between target receptors and circulating female sex hormones had been found [12, 13], we introduced an estimation of individual hormonal efficiency due to menopause. Three women previously underwent posterior colporrhaphy.

In each tissue we determined glycogen content, as well as activities of active and total glycogen phosphorylase (GP) [EC 2.4.1.1.] and acid exo-1,4-alpha-glucosidase (commonly called glucoamylase) [EC 3.2.1.3.]. When specimens weighed less than 150 mg, enzymatic measurements of glycogenolysis were favoured over glycogen evaluation.

Glycogen determination

It was carried out according to Lo et al. [14]. Glycogen from a sample of minimum 35 mg was isolated by basic digestion. Further, the essay utilized the finding that in the presence of concentrated H₂SO₄ glycogen, hydrolysed to