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Author(s): M. Jóźwik and M. Jóźwik

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Institution City Country: Department of Gynecology, Institute of Obstetrics and Gynecology, Białystok Medical University, Białystok, Poland

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Title (type in CAPITAL LETTERS): STRESS URINARY INCONTINENCE IN TERMS OF MUSCLE FIBER TYPES TRANSITIONS.

Aims of Study: The mechanisms of continence of urine remain under debate. Likewise, the pathogenesis of stress urinary incontinence (SUI) in women is not fully elucidated. This paper presents the application of muscle fiber types transitions, a physiological mechanism occurring in normal skeletal muscles subjected to intense work [1], to striated muscles at the area of continence.

Methods: Literature was extensively searched in order to establish: 1) the muscle fibre type contents of the striated muscle, or rhabdosphincter, of the urethra and the periurethral portions of the levator ani muscles, and 2) the direction, or sequence, of fiber type transitions. These two muscle groups were considered since they share innervation from the pudendal nerve [2]. Thus, damage to this innervation could trigger the same changes in each muscle group. Later, the data obtained from the searches were combined to provide an alternative concept of pathogenesis of SUI.

Results: Both the rhabdosphincter [3] and periurethral portions of the levator ani [4] contain type I and type II fibers. The direction of transition in overburdened muscles is from type II to type I fibers [5]. Only fast-twitch type II fibres, and not slow-twitch type I fibers, are specialized in the quick reflex occlusion of the urethral lumen at strain. If the common SUI feature of pudendal denervation occurs, the remaining non-denervated muscles are subjected to excessive work load. This increased burden initiates transition from type II to type I fibers. Once triggered, the transition continues, creating a vicious circle. The subsequent decrease in the content of type II fibers, or their lack, manifests as SUI.

Conclusions: The present paper suggests that particular types of muscle fibres, rather than muscles as a whole, assure stress continence. Beneficial elsewhere, the physiological adaptative mechanism of muscle fiber types transitions can have detrimental effects on the urethral occlusive

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Author(s): M. Jóźwik and M. Jóźwik

mechanisms. Functional deficiency of urethral and periurethral type II muscle fibers is an important mechanism in the pathogenesis of the clinical entity of SUI.

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