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Abstract Reproduction Form B-1

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Title (type in CAPITAL LETTERS): IS THE CURRENT CONCEPT OF THE OF HISTOMORPHOLOGICAL COMPOSITION OF THE FEMALE PELVIC FLOOR AND ITS CHANGES DUE TO VAGINAL DELIVERY CORRECT ? WE ARE IN DOUBT

In the literature of the past 15 years, it is widely accepted that pregnancy and vaginal delivery are the main causes of damage to the pelvic floor. This damage is currently understood as an important factor in the development of urinary stress incontinence and prolapse immediately or later in life [1,2].
The published data concerning the causes of pelvic floor damage are contradictory. The original hypothesis argued damage of neurogenic origin [1]. Later publications [3,4] proved no evidence for denervation/ reinnervation in biopsy specimen but speculated evidence of myogenic damage.
In summary, basic knowledge on the physiological composition of the female pelvic floor and the impact of vaginal delivery on the pelvic floor is still lacking.

Aims of the study

1. To define the normal composition of the female pelvic floor at different anatomical locations.
2. To describe the influence of vaginal delivery on the histomorphological composition of the female pelvic floor.
3. To investigate the influence of vaginal delivery and hormonal effects by comparing male and female biopsies.

Methods

In a cross sectional study, 49 unfixed and fresh female cadavers (22 nulliparous, 27 parous) and 10 male cadavers were biopsied at the Institute for Forensic Medicine from 11/96 to 2/99. All subjects were under 50 years of age (women with at least one intact ovary). After removal of the pelvic viscera, the levator ani muscle was biopsied to get representative samples at three standardised locations on each side: ventrally from the pubococcygeus muscle, in the middle from the iliococcygeus muscle and dorsally from the coccygeus muscle.
In addition to standard staining (HE, van Gieson), the biopsies were stained with **NCAM** (neuronal cell adhesive molecule) to detect denervated fibres and with **ACE** (Acetylcholinesterase) to show neuronal endplates. For fibre type identification, actomyosin ATPase was used.

Results

All biopsies of the female and the male cadavers showed some endomysial connective tissue enhancement i.e. fibrosis. As signs for damage of myogenic origin, variation in fibre diameter was found in 32/49 of the females, in 4/10 males. Centrally located nuclei could be observed in 29/49 of the females and in 5/10 in the males. All changes were pronounced in the ventral part of the levator ani muscle. NCAM- positive fibres could only be found in the closest surrounding of fibrosis. ACE staining was positive in 22%. All stained synapses were intact.
There was no statistical difference between nulliparous and parous women and men.
There was no evidence of neurogenic muscle damage such as grouped fibre atrophy, small angulated fibres or type grouping in any of the biopsies.

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Conclusions

In this study, no histomorphologic evidence of neurogenic damage to the levator ani muscle could be demonstrated in nulliparous and parous women and in men. This is in accordance with other newer studies [3,4]. There are discrete hints of changes that might be of myogenic origin.

ENCAM positive fibres, which are specific for denervation, were only found in the closest surrounding of fibrosis and never in the muscles itself. All synapses which were stained with **ACE** were intact, proving undamaged neuronal endplates.

There were no significant differences in the composition of the levator ani muscle between nulliparous, parous women and men. A possible explanation for our findings is that the general morphological definition of cell damage in striated skeletal muscles cannot be applied to the muscles of the pelvic floor. This is in accordance with Gilpin [5].

In conclusion, we would like to point out that the neurogenic changes found in electrophysiological studies of women after vaginal birth do not result in neurogenic damage to the levator ani muscle.

This allows the conclusion that the current concepts of the neurogenic cause of urinary stress incontinence, of the normal histomorphological composition of the female pelvic floor and its changes due to vaginal delivery need to be reevaluated.

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