Chapter 6

Birth Trauma and Incontinence

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6.1 Morphological Changes of the Continence Controlling System of Urethra and Anus Caused by Pregnancy and Delivery

Morphofunctional causes for pregnancy- and delivery-related functional reduction of the urinary stress-continence controlling system and of the anal continence system can result from changes in the pudendal nerve conduction, a reduced contraction force of the pelvic floor muscles, a changed mobility of the bladder neck as a result of collagen tissue weakness, a changed resting tonus of the urethra or anus, continuity defects of the sphincter ani externus and/or internus or myotrophic changes of the sphincter ani internus muscle.

Retrospective histological examinations of cadavers have shown changes of morphology of the pelvic floor and urethra in correlation to parity, at which constitutional or age-related changes come in and cannot be distinguished. For the first time, magnetic resonance imaging makes it possible to examine the course of physiological and pathological changes in morphology of the urinary stress-continence system and the anal continence controlling system in the context of pregnancy and delivery. Therefore the following concentrates on results of the magnetic resonance imaging.

6.1.1 Urethra

No correlation could be found between parity and urethral diameter in urogynecologically healthy women with stress urinary incontinence (Tunn et al. 1998b). Six months after spontaneous delivery no significant changes in the anatomical length of the urethra, independent of parity, could be found (Hayat et al. 1996). There were no examinations to be found on the delivery-related changes of the zonal anatomy of the urethra.

6.1.2 Levator ani Muscle

Sequel examinations of the signal intensity of the levator ani muscle postpartum in correlation with the obturatorius internus (first day postpartum, 1, 2 and 6 weeks postpartum) showed an increased signal intensity in the levator ani on the first day postpartum, a signal intensity expressing its chemical compound (Tunn et al. 1999). Some explanations for the increase in signal intensity are known: an elevated proportion of water or fat in muscle tissue, an increase in extracellular fluid or a glycogenolysis-related accumulation of tissue lactate (Fleckenstein et al. 1992, 1993; Schedel et al. 1995). By correlating histological examinations and those done by magnetic resonance imaging on striated muscle tissue, a heightened signal intensity could be shown as a result of a widened extracellular space and incomplete muscle fiber regeneration (Gejo et al. 2000), meaning reversible tissue changes. Signal intensity in primiparae diminished 6 weeks after delivery to values comparable to those of the obturatorius internus muscle through reconvalescence of the levator ani. In multiparae the starting point of signal intensity was reached again after 6 months only (Tunn et al. 1999). Comparable findings have been seen on striated muscle tissue after denervation and reinnervation (Uetani et al. 1993). In several observations, persistence of heightened signal intensity and total loss of the levator ani seemed possibly to be expressing the loss of striated muscle fibers after laceration, infarction or denervation, respectively, as has been seen in skeletal muscles by MRI (de Smet 1993; Khoury et al. 1997).