Post partum pelvic floor changes

I. Fonti,
R. Giordano,
A. Cacciatore,
M. Romano,
B. La Rosa

° Department of Obstetrics and Gynecology of “S. Bambino” Hospital. University of Catania, Italy.
§ Department of Obstetrics and Gynecology, Policlinico-Vittorio Emanuele. University of Catania, Italy.

Introduction
Pelvic-perineal dysfunctions, are the most common diseases in women after pregnancy. Urinary incontinence and genital prolapse, often associated, are the most important consequences of childbirth and are determined by specific alterations in the structure of neurological and musculo-fascial pelvic support. Causation is difficult to prove because symptoms occur remote from delivery. Furthermore, it is unclear whether changes are secondary to the method of childbirth or to the pregnancy itself. This controversy fuels the debate about whether or not women should be offered the choice of elective caesarean delivery to avoid the development of subsequent pelvic floor dysfunction. During pregnancy, the progressive increase in volume of the uterus subject perineal structures to a major overload. During delivery, the parties present and pass through the urogenital hiatus leading to growing pressure on the tissues causing the stretching of the pelvic floor with possible muscle damage, connective tissue and/or nerves.

In this article we aim to describe genitourinary postpartum changes with particular attention to the impact of pregnancy or childbirth on these changes.

Genital tract trauma and pelvic floor muscle injury
Pregnancy is associated with a decrease in perineal muscle strength and endurance compared with the previous state. The degree to which women improved or did not improve perineal muscle function after birth was related to perineal trauma at delivery. After controlling for parity, maternal age, birthweight, smoking status, and antepartum scores, the order of best to worst performance was cesarean birth, intact perineum, first-degree perineal injury, second- or third-degree perineal injury, and episiotomy. Although all other perineal outcome groups increased muscle function by 6 months postpartum, women with an episiotomy had a mean net loss of perineal muscle performance after birth. These observations do not support the use of episiotomy for the purpose of preserving perineal muscle function.

Pregnancy and post-partum urinary incontinence
Pregnancy and post-partum urinary incontinence are important forms of maternal morbidity. The hormonal and physical effects of pregnancy and childbirth are the major reasons of urinary incontinence. During pregnancy, mechanical and hormonal factors cause changes in renal physiology, most commonly resulting in frequency of voiding and stress incontinence. Other symptoms during pregnancy include urinary urgency, urge incontinence, incomplete emptying, and slow stream. The increase in stress incontinence during pregnancy is argued to be the result of damage to the fascias, ligaments, pelvic floor muscles and nerves supporting and controlling the bladder neck and urethra. Vaginal delivery is linked to a high rate of incontinence in the postpartum period, and women who may have been continent during the pregnancy could find themselves with stress incontinence after the birth. In fact, the reported incidence of de novo incontinence (starting again) after a first vaginal birth is 21% with spontaneous birth and 36% with forceps delivery. Many clinical studies have attempted to discover the particular obstetric event that causes the incontinence. The obvious suspects include large babies and “difficult deliveries” marked by lengthy pushing phases with or without instrumentation. No clear single event has been found to be responsible, suggesting that postpartum urinary incontinence arises from a multifactorial physiological insult. Not all incontinence is troublesome to patients. Most studies evaluating the incidence and impact of postpartum urinary incontinence compare women with any urinary incontinence with women with no incontinence, and do not include descriptions of the severity of incontinence. This omission underlines the importance of using reliable methods of obtaining information regarding functional outcomes. The use of validated and reliable questionnaires to evaluate both symptom severity and quality of life is essential for future evaluation of postpartum pelvic floor changes.

Denervation injury
Pelvic floor neuropathy is a common repercussion of childbirth, less often recognized than vaginal and per-
Pelvic organ prolapsed is the herniation of pelvic organs through the vaginal opening. By far, the greatest incidences of prolapsing are after childbirth. Pregnancy and vaginal delivery are the risk factors cited most commonly. In fact carrying the weight of a baby as it drops lower into the pelvic region; laboring to bring the baby through the birth canal, and pushing the baby into the world. All of these combine to weaken the pelvic floor and create conditions for a prolapsed uterus. Moreover, harder labours and big babies also contribute to pelvic floor weakening. Delivering a baby bigger than 8 ½ pounds can carry a risk of pelvic prolapse. Childbirth is not the only contributing factor to pelvic prolapsed. Risk factors for development of pelvic organs include aging, menopause, smoking, chronic diseases. Symptoms of prolapse may not show for years after an event such as childbirth, but when they appear are severe. Urinary incontinence associated with a cystocele can be constant and embarrassing. A rectocele can cause constipation, inability to completely void the bowels, or an inability to hold gas or bowel movements and sexual dysfunction.

Sexual function
Changes in sexual function are common in postpartum women. Many women express concern that pregnancy-induced changes in their bodies will affect their postpartum sexual function. These fears are heightened when the woman experiences significant trauma during childbirth. As a result of neuropathic changes, the sling-like components of levator ani may fail to reflexively contract and elevate sphincter pressure during a cough or sneeze. Likewise, the resting tone of the shell-like levator plate and perineal body may diminish. However, many studies have reported lower IRS scores than women who did not experience pelvic floor trauma and it can cause considerable damage to both the muscle tissue that nerve supply most of the anatomic structures maintaining pelvic support and continence.

References
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