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# The effects of childbirth on the pelvic-floor

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## 1 Introduction

The outcome of vaginal birth can be devastating for mother and child. In the developing world there is a significant maternal morbidity and mortality rate [4]. This is especially true when labor is obstructed. In Ethiopia the maternal mortality rate is estimated to be about 2000 per 100000 births (2 %). The associated gynecological morbidity includes fistula (vesico-vaginal and rectal), amenorrhea and vaginal scarring. Various orthopedic injuries are observed, like: "…bone resorption, marginal fractures, spurs, and bony obliteration of the symphysis as well as symphyseal separation and … gait disorders." [4]. In 20 percent there is an associated neurologic injury. In addition, injuries to the peroneal nerve or to the sacral plexus of nerves results in pelvic neurologic damage [4, 10]. In developed nations such tragic outcome of delivery is very rare due to the great achievements gained in modern practice of obstetrics during the past 100 years. More commonly, women experience occult injuries to the pelvic floor, that can result in problems like anal and urinary incontinence, and pelvic organ prolapse. Though those consequences of childbirth are not life-threatening, those conditions are still by far underestimated and tabooed. Very often for the affected women the strains are debilitating. There can be a considerable impact on life quality. This is especially true for anal incontinence which is more common than previously recognized. During the recent past, the understanding for the etiology and pathology of anal incontinence grew considerably. It is now clear that birth trauma is the main etiological factor [59]. The growing knowledge of the pelvic floor consequences of childbirth offers the chance of developing prevention- and therapy-strategies. On the other hand it lead to sometimes emotionalized debates about subjects like cesarean on demand or use of episiotomy. A study commonly cited in this context is the one conducted by Al-Mufti: it showed that 1/3 of all female gynecologists and obstetricians would choose an elective cesarean section to protect their pelvic floors [2]. This article reviews the pelvic floor sequelae of childbirth. The main focus will be anal incontinence for reasons of relevance to the present discussion in literature, but urinary incontinence and pelvic organ prolapse will be considered as well.

## 2 Anal incontinence

### 2.1 Anatomy and physiology

Fecal continence is accomplished by a complex interaction of different anatomical and physiological components. A dysfunction in one of those components – independently or in combination with other components – can lead to anal incontinence. Anal incontinence means at least one of the following: involuntary loss of gas, mucus, liquid or solid stool, dysfunction of discrimination between liquid or flatus (fine control of continence) or urgency.

The rectum, the anus and the pelvic floor muscles are richly provided with sensory receptors. This is important for the fine control of continence (differentiation of liquid and flatus) and for the recognition of rectal distension.

The internal (IAS), external (EAS) and puborectalis muscle are the muscles that mainly participate in maintaining continence. The IAS develops spontaneous tone due to its own myogenic activity. It provides 50 to 85 percent of resting tone. The EAS and the anal cushions are responsible for the remainder (15 to 50 percent) [13, 15, 21, 32]. The IAS lies between the anal mucosa and the EAS and extends more than a centimetre above the cranial margin of the EAS with a total length of about 3 cm [12].

The EAS and the puborectalis muscles are voluntarily innervated. They also develop a spontaneous tone, directly proportional to rectal distension [8].

The puborectalis muscle is part of the levator ani muscle. It inserts two-sided at the pubic bones passes the vagina and rectum bilaterally and merges behind the rectum building a u-shaped sling around the rectum thus obstructing the anal canal. In addition the puborectalis causes the so called anorectal angle between the distal rectum and the anal canal (about 90°), which effectively supports the continence mechanisms.

The EAS covers the IAS like a cuff, inserting ventrally at the perineal body and posteriorly at the anococcygeal ligaments. Functionally the EAS is able to obstruct the anal canal forming a sagittally directed opening.

### 2.2 Incidence and etiology and associated risk factors

The etiology of anal incontinence is complex. There are obstetric and non-obstetric reasons. Following childbirth it can be broadly classified as nerve injury or traumatic sphincter injury. A combination of both is very common in manifest anal incontinence [27]. The insight in risk factors is very important for the development of prophylactic strategies. For that reason the etiology of anal incontinence and associated risk factors are shortly discussed.

#### 2.2.1 Nerve injury

For many years obstetrical nerve injury following childbirth was thought to be the main cause of anal incontinence [52, 5]. The anatomical situation of the pudendus nerve at the outer border of the levator ani muscle made this idea very plausible. In addition, visible sphincter trauma (3rd degree tear) usually is not recognized in more than 3 percent. It has been hypothesized that distortion of the pudendal nerve during vaginal birth causes denervation injury of the pelvic floor muscles (indirect muscle trauma). Measurement of the pudendal nerve terminal motor latency (PNTML) allows diagnosis of an obstetrical injury to the pudendus nerve. Basically, this test measures the time interval between transvaginal or transanal electrical stimulation of the pudendus nerve and the contraction of the EAS. In a prospective study with 128 women which were assessed during pregnancy and after delivery 16 % showed a prolonged PNTML indicating some degree of nerve lesion [55]. This prolongation, however, persisted in only 1/3 of the women 6 months after delivery. Prolongation of PNTML was associated with heavier child weights and with prolonged 2nd stage of labor. Similar results were published by Allen and Snooks [1, 51]. In addition forceps delivery turned out to be a risk factor for nerve injury. Denervation of the pelvic floor muscles can persist for years or even progress [53]. Most of the time, however, these injuries are reversible. At present it is estimated, that only about 10 percent of idiopathic anal incontinence is due solely to nerve injury without coexisting structural defects [8].

#### 2.2.2 Sphincter damage

Tears of the EAS or IAS are therefor at least partially responsible for about 90 percent of complaints of symptoms of anal incontinence. Dependent on the location of the muscle lesion, different forms of anal incontinence can develop. A lesion of the IAS commonly leads to passive involuntary loss of faeces, not recognized by the patient due to sensory dysfunction. A lesion of the EAS more likely leads to urge-incontinence [20].

3rd degree tears of the perineum with visible sphincter damage (disruption of the EAS or IAS) after delivery is diagnosed in about 0.5 to 3 percent of all vaginal deliveries [3, 70]. Liberal use of median episiotomy, however, increases the frequency of 3rd degree tears considerably (at least

15 %) [31]. The incidence of anal incontinence following a 3rd degree tear is published by Crawford and colleagues: 9 to 12 months after delivery of the first child 23 percent complained of symptoms of anal incontinence (17 percent incontinence for flatus, 3 percent for liquid of solid feces) [9].

Macroscopically non-visible (occult) sphincter trauma, however, is much more common after vaginal delivery, than previously recognized due to the low frequency of 3rd degree tears. Sultan and colleagues examined the sphincter morphology with the use of anal endosonography [56]. This technique provides a clear image of the IAS and EAS and various structural abnormalities. It turned out, that 35 percent of primigravidae who delivered vaginally had de novo sonographic defects of the sphincter muscles, which persisted at 6 months after delivery. Women with sphincter defects had lower resting anal pressures than those with no defect. All incontinent women had structurally lesions of the sphincter muscles. Only 4 percent of multiparous women developed new sonographic defects, indicating, that the main damage was set during the first vaginal delivery. All but one lesions were located in the IAS. A sonographically demonstrable defect clearly was associated with symptoms of anal incontinence. However, only 1/3 of all women with occult sphincter damage complained of such problems. 8 of 10 women after forceps delivery showed sonografic defects, whereas no woman after vacuum delivery showed a sphincter lesion. Posterolateral episiotomy turned out to be an additional risk factor for occult sphincter trauma. Results published by Groutz and colleagues support the correlation between use of episiotomy and the development of new anal incontinence [23]. Occult sphincter trauma could even be demonstrated with the perineum remained intact. Similar results were published by Frudinger and colleagues [16, 17]. Occult sphincter trauma was detectable with intact perineum and after episiotomy without any further tearing.

Sultan determined the prevalence of occult anal sphincter trauma 5 years after randomization to forceps and vacuum delivery [54]. 44 women participated in this study. 82 percent of forceps and 48 percent of vacuum deliveries had occult sphincter defects. In addition, a significant fall in maximum squeeze anal pressure in the forceps group compared to the vacuum group could be demonstrated. Twice as many in the forceps group suffered from anal incontinence, though statistical significance was not reached. The authors concluded, that vacuum delivery is likely to be less traumatic to the sphincter apparatus than forceps extraction.

In a recent study published by Zetterström the frequency of occult sphincter trauma after vaginal delivery was 20 percent [71]. The lesions all were located in the EAS sphincter. This is in contrast to the study of Sultan mentioned earlier. An explanation for the difference was not given. The association of sphincter lesion and symptoms of anal incontinence, however, was not that pronounced. With regard to cesarean section, both investigators observed neither an occult sphincter lesion nor anal incontinence. Similar results were published by Crawford [9].

Varma and colleagues demonstrated occult sphincter trauma after uncomplicated vaginal delivery in only 8.7 percent (n = 159) [62]. Forceps delivery turned out to be the only risk factor: 83 percent showed occult lesions after forceps extraction. No correlation was found between other routinely measured delivery variables like head circumference, baby weight, maternal body mass index, epidurals, episiotomy, length of each stage of labor and duration of active pushing. The authors concluded that instrumented deliveries warrant routine postnatal anorectal assessment.

Zetterström and colleagues investigated in a prospective observational study the incidence and degree of anal incontinence after vaginal delivery among primiparous women and tried to define associated risk factors [72]. 349 women were assessed with questionnaires. At 5 months postpartum, 27 percent of the women had symptoms of anal incontinence (2 % fecal incontinence, 25 % incontinence for flatus). At nine months postpartum the situation was similar (1 % fecal incontinence, 26 % incontinence for flatus). The multivariate analysis revealed the following risk factors for anal incontinence at 5 months postpartum: higher maternal age, duration of second stage of labor (> 1 h) instrumental vaginal delivery (forceps and vacuum) and a clinically diagnosed sphincter tear at delivery (3rd degree tear).

### 2.3 Prevention of anal incontinence

Last but not least, prevention of anal incontinence also is necessary from an economic point of view. Mellgren and colleagues tried to determine the long-term costs associated with anal incontinence related to obstetric injuries [35]. They estimated the average cost per patient to be $17.166. Though these US-American data cannot easily be transferred to the conditions elsewhere, it demonstrates the economic relevance.

Due to the risk factors mentioned so far in this article, it should be possible to give recommendations for the prevention of anal incontinence: In the case of an instrumental delivery the vacuum extraction should be preferred to forceps delivery. Restrictive use of episiotomy is very likely to reduce the risk of sphincter damage. This is true for the median episiotomy (reduction of 3rd degree tears) as well for the posterolateral episiotomy (occult sphincter trauma). Avoidance of long duration of the second stage of labor is desirable. However, one should take into account that a cesarean section performed in late labor prevents direct muscle trauma, but does not prevent lesion of the pudendal nerve [19]. That is why damage to the anal sphincter mechanism (nerve injury) can occur even in the absence of attempted vaginal delivery. Finally, in the case of foetal macrosomia a primary cesarean section should be considered for prevention of pudendal lesion.

With regard to the mode of delivery in the case of persistent anal incontinence after a first vaginal delivery, Fynes and colleagues published important data [18]. They examined the effects of first and second vaginal deliveries on anal physiology and continence in 59 previously nulliparous women. Pre- and postnatal bowel-function was assessed by a special questionnaire; in addition, anal manometry and measurement of the PNTML was performed. It could be demonstrated, that primiparous women with persistent symptoms of anal incontinence experience deterioration after a second vaginal delivery. In addition, women with transient anal incontinence or with occult anal sphincter injury after their first vaginal delivery are at risk of persisting anal incontinence after a second vaginal delivery. The authors therefore postulate early anal-sphincter repair and elective cesarean delivery of subsequent pregnancies to be the appropriate approach for women with symptomatic, large anal-sphincter defects. Women with occult anal injury should be counseled to enable them to make an informed choice about the mode of their subsequent deliveries.

## 3 Urinary incontinence

### 3.1 Prevalence

Prevalence rates of de novo urinary incontinence in the puerperium vary from 0.7 to 38 percent [14, 37, 65]. Studies investigating the persistence of postpartum incontinence show controversial results: Viktrup and colleagues report a 24 % persistence rate 5 years after delivery, whereas Mac Arthur postulates a rate of about 75 % [33]. The prevalence of stress urinary incontinence after cesarean delivery is significantly lower compared to vaginal delivery [36, 68]. For that reason, the epidemiology of stress urinary incontinence most likely is associated with vaginal delivery. The pathophysiology, however, still is poorly understood.

### 3.2 Pathophysiology

The continence mechanism is considered to rely basically on the integrity of four intercalated components: the intrinsic and extrinsic sphincter mechanisms, a bladder neck that is well anchored to the pubic bone (by the pubourethral ligaments) and lateral pelvic wall, and an intact innervation to these structures [58]. Damage to one of these structures during vaginal delivery possibly results in urinary incontinence. For that reason, several investigators addressed the question to what is the influence of childbirth on the different structures participating in the continence mechanism.

Van Geelen and colleagues assessed the changes to urethral pressure profile in pregnancy and after delivery in (previously) nulliparous women [61]. They observed a considerable decrease of both the urethral pressure and length parameters in all women after vaginal but not after abdominal delivery 8 weeks postpartum. There was no significant association to the duration of second stage of labor, use of episiotomy or infant birth weight. The authors concluded that pregnancies followed by a first vaginal delivery commonly result in de-

creased urethral closure pressure and reduced urethral length contributing to postpartum stress incontinence. Shortening of the functional urethral length after vaginal delivery was confirmed by Meyer and colleagues [36]. However, they found no change in any other indices of urethral function like decrease of urethral pressure or pressure transmission.

Peschers and colleagues assessed postpartum changes in urethral movement during the Valsalva maneuver and pelvic floor muscle contraction sonographically [39]. They found the bladder neck at rest to be significantly lower after vaginal delivery than after elective cesarean delivery or in nulligravid controls. This observation possibly explains the decreased transmission of pressure to the urethra after vaginal delivery [40]. Bladder neck mobility had increased in primigravidas 6 to 10 weeks after vaginal delivery [39]. Greater antenatal bladder neck mobility turned out to be a risk factor for postpartum stress incontinence [28].

Based on neurophysiological and histomorphological studies there is evidence, that the innervation of the pelvic floor muscles is damaged in postpartum stress incontinence. Prolongation of the pudendal nerve terminal motor latency (PNTML) after vaginal delivery was shown repeatedly [52, 55, 60]. Neurophysiolgical studies using single-fibre EMG or concentric-needle EMG showed, that vaginal birth often is followed by partial denervation and subsequent reinnervation of the pelvic floor musculature [1, 50, 53]. Women with urinary stress incontinence seem to have a significant increase in denervation of the pelvic floor compared with asymptomatic women [50]. Postpartum partial denervation and reinnervation was confirmed by histomorphological studies [22].

In general, however, it is usually not possible to assign a single cause to be responsible for the development of incontinence in a particular woman.

### 3.3 Risk factors and prevention

As with anorecatal incontinence, prevention of urinary incontinence is desirable also from an economic point of view. In 1995 costs totaled $26.3 billion in the USA [67]. The knowledge of risk factors for urinary incontinence would make it possible to develop prophylactic strategies. Unfortunately the situation for urinary incontinence is not as conclusive as for anal incontinence. The precise role of pregnancy and delivery in the development of persistent stress incontinence remains unclear. The discussion in literature therefore is controversial.

Some studies found the length of second stage of labor to be associated with the development of urinary incontinence [14, 25, 66]. The same accounts for head circumference and for birth weight [66]. This is in contrast to other investigations, that did not demonstrate an association with head circumference or with birth weight [14, 30, 42].

Use of median or mediolateral episiotomy obviously does not prevent the development of urinary incontinence [29, 42, 48]. On the contrary: Viktrup and colleagues found mediolateral episiotomy to be associated with a higher incidence of postpartum urinary incontinence.

With regard to epidural anaesthesia during labor the results are controversial, too. Whereas Dimpfl and colleagues found epidurals to be protective against urinary incontinence, this was not confirmed by other investigators: on the contrary, postpartum stress incontinence appeared to be more frequent in women who had received epidural anesthesia [14, 25, 64].

Johanson and colleagues undertook a five year follow-up of women delivered by forceps or vacuum extractor in a randomized controlled study. Urinary incontinence of various severity was very common in this population (44 %; n = 225), but there was no significant differences between the instruments [26]. In addition, in a recent study operative vaginal delivery was not found to be associated with increased risk of stress urinary incontinence [24]. Additional possible risk factors for the development of stress urinary incontinence are grand multiparity (> 5 deliveries) and obesity (body mass index > 30 $kg/m^2$) [41, 68]. Chaliha, however, found no association to the antenatal body mass index [7]. In a prospective study they tried to assess whether physical markers of collagen weakness (striae, hernia, varicose veins, and joint mobility) and the body mass index can predict postpartum urinary incontinence.

Unfortunately in this study (n = 549 nulliparas) this was not possible.

In consideration of the mentioned contradictory statements concerning the risk factors for development of postpartum or persistent urinary incontinence, there are no present definitive recommendations for reducing the risk of stress incontinence after vaginal delivery.

## 4 Pelvic relaxation and organ prolapse

### 4.1 Epidemiology and physiology

Pelvic relaxation − resulting in uterine prolapse, cystocele, rectocele or enterocele − is a very common gynecological problem, frequently causing great strain and distress for the affected women. It is among the most common indications for major gynecological surgery (e. g. hysterectomies) [63]. In a British study, the annual incidence of hospital admission with prolapse before age 60 was 2 per 1000 person-years [34]. The prevalence of any degree of genital prolapse in a general population of women 20 to 59 years of age was estimated to be 30 percent [47]. However, only 2 percent of all women had a prolapse that reached the introitus. In an American study, the lifetime risk of undergoing a single operation for pelvic organ prolapse or urinary incontinence by age 80 peaked 11 percent [38].

During the recent past our knowledge of how the pelvic organs are kept in place has increased considerably. Basically, prevention of pelvic organ prolapse depends on two dynamically interacting functional systems [11]: First, the endopelvic fascia, by which the uterus and vagina are attached to the pelvic sidewalls. Second, the levator ani muscles, which close the pelvic floor (the so-called urogenital hiatus of the levator ani) and support the pelvic organs. It is important to keep in mind, that well functioning pelvic floor muscles are crucial for prevention of prolapse.

### 4.2 Etiology and risk factors

Relaxation or damage to the pelvic floor muscles result in opening of the pelvic floor. Thus, the pelvic organs are no longer supported by the muscles and must be held in place solely by the connective tissue of the endopelvic fascia surrounding the pelvic organs. Connective tissue, however, never is able to withstand chronic stretching and will finally become damaged, thus failing to hold the vagina in place [11].

For that reason vaginal birth − resulting in damage to the pelvic floor muscles − is thought to be the main reason for development of prolapse. In fact, increasing parity showed the strongest association to the development of pelvic organ prolapse resulting in an eleven fold increase in risk in multiparity (> three deliveries) compared with nullipaous women [34]. The etiology of the damage to the pelvic floor muscles has been discussed in connection with the etiology of anorectal incontinence, which is basically the same (neural and/or muscular damage). Thus the risk factors for developing neural or muscular trauma should be similar like for developing pelvic organ prolapse.

Pelvic organ prolapse should be assessed by standardized vaginal examination described by the International Continence Society (ICS) [6]. Pelvic floor muscle testing includes inspection (movement of the perineum during pelvic floor muscle contraction or straining), palpation (localization of atrophic or asymmetric segments), electromyography (visualization of individual or grouped motor unit action potentials) and pressure recording (measurements of pelvic floor muscle control and strength) [6].

Descent of the perineum six to eight weeks postpartum during rest or during straining was observed by several investigators [49, 52, 55]. Simultaneously there was a considerable decrease in strength of contraction assessed with vaginal cones [43], intravaginal squeeze pressure measurements [36, 44] and with a digital muscle strength score [45].

There is evidence that episiotomy does not prevent pelvic relaxation [43, 69, 70]. On the contrary: Röckner found in a well designed study in patients with mediolateral episiotomy a considerable impairment in pelvic floor muscle strength compared with those with spontaneous or no laceration [43].

Samuelsson and Colleagues studied possibly related factors for development of genital prolapse in a general population [47]. 487 women participated in this study. As previously mentioned, 30.8

percent of all women had at least some degree of prolapse as diagnosed by vaginal examination. In the multivariate analysis the following factors turned out to be significantly and independently associated with prolapse: age, parity and pelvic floor muscle strength and − among parous women − additionally the maximum birth weight.

With regard of the significance of pelvic muscle strength for prevention of prolapse Sampselle and colleagues published interesting data of a prospective and randomized trial which showed that pelvic muscle exercise during pregnancy improves postpartum pelvic muscle strength and reduces postpartum symptoms of urinary incontinence [46]. For that reason, pelvic floor muscles exercise during pregnancy is strongly recommended for all pregnant women.

## 5 Conclusion

Vaginal delivery basically is associated with the risk of pelvic floor damage. Elective cesarean section seems to be the only highly effective strategy to prevent pelvic relaxation (anorectal and urinary incontinence and pelvic organ prolapse), because after cesarean section pelvic floor damage is rare. Cesarean section, however, has its own risks (increased mortality and morbidity). For that reason Sultan and Stanton recommend elective cesarean section only when a woman is at increased risk for pelvic floor damage (e. g. after an incontinence operation) [57]. Nevertheless, elective cesarean section may be the best mode of delivery for a particular woman even without recognizable increased risks for pelvic floor damage − if this is desired by the woman. It is not necessarily unreasonable to permit women the option of an elective cesarean birth to preclude potential injury to their pelvic floor. One prerequisite, however, is detailed counseling about the pros and cons of vaginal versus abdominal delivery enabling the woman to make an informed choice about her mode of delivery.

**Abstract**

Basically, vaginal delivery is associated with the risk of pelvic floor damage. The pelvic floor sequelae of childbirth includes anal incontinence, urinary incontinence and pelvic organ prolapse. Pathophysiology, incidence and risk factors for the development of the respective problems are reviewed. Where possible, recommendations for reducing the risk of pelvic floor damage are given.

**Keywords:** Anal incontinence, cesarean section, childbirth, mode of delivery, pelvic floor, pelvic relaxation, prolapse, urinary incontinence, vaginal delivery.

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