Voiding dysfunction after vaginal prolapse surgery: etiology, prevention and treatment
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Chapter 1

Introduction and outline of the thesis
Chapter 1

Clinical problem of incomplete voiding

Pelvic organ prolapse (POP) is a common disease for which a significant number of patients will require and demand surgical correction in order to correct anatomical abnormalities and to restore normal pelvic floor function. After this type of surgery sometimes a total inability to void develops (urinary retention) and more frequently the problem of incomplete bladder emptying occurs resulting in an abnormal postvoid residual volume (PVR). With a prevalence of 1 to 22% the occurrence of this incomplete voiding is one of the most frequent complications directly related to vaginal prolapse surgery. When incomplete voiding is left untreated, the bladder can be stretched beyond its physical limits which can decrease achievable detrusor pressure and change elasticity. This can lead to impairment of bladder function in the long term.

To prevent such sequelae, bladder drainage through catheterisation is applied. However, often the initiation and continuation of catheterisation in hospitalised patients is poorly indicated. Reported reasons for prolongation of catheterisation are negligence (i.e. forgetting the presence of a catheter) and convenience for nursing staff. Also a large practice variation exists in type and duration of catheterisation. Presumably, a lack of evidence can be held accountable for this practice variation. To improve awareness and recognition, to develop optimal preventive measures and to optimise treatment more evidence is needed that evaluates common practice and to optimise this practice.

Prevention and treatment: Standard prolonged catheterisation and catheterisation when indicated

For decades gynaecologists routinely applied bladder catheterisation for several days after vaginal prolapse surgery. This postoperative standard insertion of a catheter may initially be prompted by the presence of a vaginal gauze, by inhibition of bladder sensibility and contractility through the action of anaesthetics and by impaired mobility of the patient post-operatively. It can also be considered necessary to prolong catheterisation as postoperative pain, urethral elevation and peri-urethral oedema and/or innervation trauma may hamper optimal emptying of the bladder.

Although such prolonged catheterisation appears relatively safe and straightforward, it proved to be unnecessary after vaginal hysterectomy. Furthermore, catheterisation inevitably increases the risk of urinary tract infections and results in longer hospital stay, more discomfort and higher disappointment for patients about the treatment they received. As the majority of patients does not encounter the problem of incomplete voiding the standard postoperative insertion of catheters for longer durations implies that a majority of patients receives suboptimal treatment. Therefore it could be rational to restrict standard catheterisation to the period of impaired...
mobility and the possible negative influence of anesthetics on bladder function has not worn out yet. After this period of standard catheterisation a proportion of patients will require additional treatment because of the occurrence of abnormal post void residual volume. When this complication has been diagnosed, again a great variation seems to exist between departments regarding the choice for type of catheterisation and the continuation of catheterisation. Most likely this results from the absence of studies and guidelines regarding this matter.

**Etiology**

Some risk factors have been identified for incomplete voiding after vaginal prolapse surgery. Identification of such risk factors is relevant as it could be used to adjust preventive strategies and treatment regimens. Contrary to vaginal prolapse surgery, more risk factors are known for development of incomplete voiding after incontinence surgery. Some of the identified mechanisms for the development of abnormal post void residual volume after this type of surgery may also account for prolapse surgery. We will explore the aetiology of incomplete voiding after vaginal prolapse surgery by identifying more risk factors for incomplete voiding after vaginal prolapse surgery.

**Detrusor underactivity**

Detrusor underactivity has been defined by the international continence society as a contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or a failure to achieve complete bladder emptying within a normal time span.\(^2\) It can be influenced by the following factors.

**Age**

In clinical studies of patients undergoing incontinence surgery a negative effect of age on voiding postoperatively was demonstrated.\(^{10, 21-25}\) In other studies age was related to detrusor underactivity and a reduced sensation to bladder filling.\(^{26, 27}\) On a morphological level these phenomena can be explained by a reduction in axonal content\(^{27}\) and changes in neurotransmission.\(^2\)

**Influence of anesthetics**

Anaesthetic agents used in general anaesthesia can reduce bladder function by suppression of the pontine micturition center (animal studies).\(^{29, 30}\) Intrathecally administered local anesthetics block neurons of the sacral spinal cord (S2–S4). The action is by blocking the transmission of the afferent and efferent information of nervous fibers from and to the bladder.\(^{31}\) The block as a rule wears off within 7-8 hours. To prolong the duration of sensory block with spinal anesthesia
intrathecal opioids can be administered. These agents inhibit bladder contractility and can increase bladder capacity. The duration of this effect on the bladder is dependent on the type of opioid used and the administered dose but will last maximally 24 hours. \textsuperscript{32, 33} For the purpose of analgesia in vaginal prolapse surgery, which are in general relatively short lasting operations, intrathecal opioids are seldomly administered. In the clinical studies in this thesis analgesia was mostly reached using a spinal block without addition of opioids. Therefore, on the role of anesthetics in postoperative incomplete voiding will not be focused in this thesis.

Surgical damage to innervation of the bladder
It has been well established that innervation trauma of the bladder can be a consequence of major pelvic and colorectal surgery. \textsuperscript{34, 35, 36} Although it is likely that dissection and suturing part of the bladder wall in the case of anterior repairs can also result in innervation trauma, it has not been proved. Indirect evidence comes from one study in which a higher stage preoperative anterior wall prolapse was found to be a predictor for the occurrence of abnormal post void residual volumes postoperatively. \textsuperscript{17} This can be explained by a more extensive dissection with higher stage prolapse surgery and innervation damage of the bladder as a consequence. \textsuperscript{17}

Anxiety
We hypothesise that anxiety can be of influence in the development of incomplete voiding after vaginal prolapse surgery. Anxiety is likely to obstruct bladder outflow through an alfa adrenergic stimulation of the bladder outlet \textsuperscript{8, 26, 37} and also by pelvic floor contraction. \textsuperscript{38} The idea that, pre- and postoperatively, anxiety levels rise while patients are faced with hospitalisation, loss of autonomy and (an unusual) request for efficient micturition is supported in literature. \textsuperscript{39-41} The negative effect of posing such conditions to patients was illustrated by the observation in another study that a request for micturition in a hospitalised environment led to an absolute inability to void in 8 of 18 (44\%) otherwise healthy non surgical subjects. \textsuperscript{41}

Pain
Pain is an inevitable consequence of surgery. It is known that it can negatively influence bladder function through central inhibition and is likely to cause bladder outflow obstruction through a disturbed relaxation of the pelvic floor. This has been mentioned as a possible cause of voiding impairment postoperatively. \textsuperscript{8,19, 26, 37} Support for this hypothesis comes from studies in patients undergoing proctological and posterior compartment surgery. Although there is no direct anatomical relationship
between the surgical site and the bladder in such patients, they are however still at risk of developing incomplete voiding.\textsuperscript{15,17}

**Obstruction**
The assumption that postoperative edema, haematoma formation and/or urethral elevation can contribute to a bladder outlet obstruction (BOO) leading to voiding disorder is widespread.\textsuperscript{6,42-44} However, no evidence exists to support this hypothesis. There are no urodynamic studies combining flow parameters with detrusor pressure available to establish the cause of incomplete voiding following vaginal prolapse surgery. A few studies have investigated mixed populations of incontinence and prolapse surgery. In these studies vaginal prolapse surgery shows an increase of incomplete voiding in the first few days which wears off in the longer run while incontinence procedures tend to result in incomplete voiding for longer periods. Therefore, the observed voiding disorder seems to be dominated by direct postoperative effects of the surgery\textsuperscript{24,45} and for longer term voiding disorders incontinence surgery plays a more important role. An explanation could be a more obstructive nature of stress-incontinence surgery.\textsuperscript{16,23,46} This is supported by the finding that the degree to which the bladder neck is elevated and the type of bladder neck suspension are both predictive for incomplete voiding after incontinence surgery.\textsuperscript{16,23}

At the time the studies in this thesis were designed it was common practice to routinely prolong indwelling catheterisation for several days after vaginal prolapse surgery.\textsuperscript{12,13} Further, these catheterisation protocols varied to a great extent which implied suboptimal treatment for many patients. Further, no evidence existed concerning the optimal technique and duration of treatment of incomplete voiding after surgery. Nor was there any insight in patient preferences for treating incomplete voiding and finally, the etiology of incomplete voiding after vaginal prolapse surgery was unknown. This all is important as it could give direction to new preventive strategies.

**Objectives of this thesis**
The following objectives were identified:
1. To determine the preferred practice and practice variation in Dutch hospitals concerning bladder care management after vaginal prolapse surgery.
2. To optimise postoperative bladder care. The first aim is to optimise the duration of the commonly applied standard bladder catheterisation after vaginal prolapse surgery. The second aim is to optimise the actual treatment of an established abnormal PVR and further insight will be provided in the preferences of patients.
3. To understand the underlying mechanisms of abnormal PVR.
References


