

Review

Bladder function after ambulatory surgery

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Abstract

Micturition is a complex process under both involuntary and voluntary control. A variety of pathological conditions, as well as certain surgical and anesthetic procedures cause urinary retention, which may have long lasting consequences. Patients undergoing ambulatory surgery have traditionally been required to void prior to discharge; however, this practice is increasingly being questioned. Ultrasound scanning of the bladder is an accurate method of measuring urine volume in postoperative patients. It may be useful as a non-invasive method of monitoring bladder volume, thus avoiding unnecessary bladder catheterization whilst at the same time preventing prolonged overdistension. We present an algorithm for managing ambulatory patients in both low and high-risk groups for postoperative urinary retention.

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1. Introduction

Until relatively recently, voiding was considered one of the prerequisites for discharge from an ambulatory surgery center [1]. Current evidence suggests this criterion is no longer valid, at least not for all patients [2]. In this review, our goal is to discuss bladder function following ambulatory surgery under the following headings: anatomy and physiology of voiding; the potential complications of urinary retention and overdistension of the bladder; the risk factors for retention; incidence and diagnosis of urinary retention. We shall also review strategies for safe management of bladder function after ambulatory surgery, and the use of ultrasound measurement of bladder volume.

2. Anatomy and physiology of micturition

2.1. Anatomy of the lower urinary track

The anatomy and nerve supply of the lower urinary tract in males is shown in Fig. 1. The detrusor muscle is composed of smooth muscle fibers; as the bladder fills, stretch receptors in the bladder wall transmit sensory signals via pelvic splanchnic nerves to synapse in the sacral cord, with projections to the micturition center in the brain. The efferent limb of the reflex includes:

- (a) Preganglionic parasympathetic neurones originating from S 2–4 traveling in pelvic splanchnic nerves to peripheral ganglion cells in the wall of the bladder. Activation of these fibers initiates contraction of the detrusor muscle.
- (b) Sympathetic efferents emanating from T10 to L2 traveling through superior and inferior hypogastric plexuses to innervate the internal urethral sphincter. These fibers are inhibited during voiding resulting in opening of the bladder neck, decrease in urethral pressure and increase in detrusor tone. These fibers are active during continence.
- (c) Somatic efferents arising from S 2–4, traveling in the pudendal nerves to the striated muscle of the external urethral sphincter. Inhibition of these nerves results in external sphincter relaxation during voiding.

2.2. The micturition reflex

Voiding is a reflex action (the micturition reflex) that requires simultaneous contraction of the detrusor muscle of the bladder, and relaxation of the internal and external urethral sphincters. Micturition is complex, being controlled at both a spinal level and by higher centers in the brain [3,4]. The stimulus to trigger this reflex is stretching of the bladder as urine volume increases. The bladder volume at which there is a strong desire to void is termed the “cystometric capacity”. This volume is similar in men and women and varies between 400 and 600 ml [5,6]. Maximal rates of urine flow

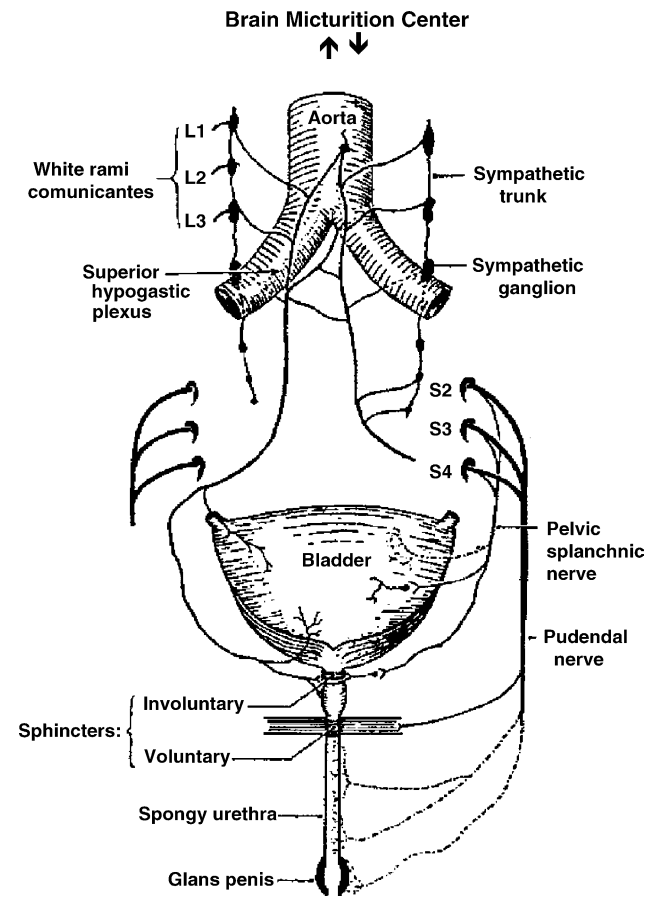


Fig. 1. Diagram of the micturition reflex neural pathways. Reproduced from Moore KL. Clinically Oriented Anatomy. 3rd ed. Baltimore: Williams and Wilkins; 1992, with permission.

are measured when detrusor pressure reaches between 43 and 50 cm of water [5].

2.3. Higher center control of micturition

The entire micturition reflex arc is subject to modulation or control by centers in the brain located in the dorsolateral pons (the pontine micturition center), the diencephalon and the cerebral cortex. Both voluntary and involuntary control of micturition is influenced by these centers in the brain. Further modulation of the micturition reflex can occur within the spinal portion of this pathway. There are a number of receptors in the micturition pathway capable of responding to dopamine, serotonin, norepinephrine, GABA, excitatory and inhibitory amino acids, opioids, acetylcholine and neuropeptides [4]. The precise role of these receptors in the normal voiding mechanism is unclear.

3. Urinary retention and overdistension of the bladder

3.1. Etiology and effects of urinary retention

The causes of urinary retention or failure to void are numerous and described in Table 1. Continued distention of the bladder as occurs with urinary retention has a number of consequences. In some instances, bladder sphincters fail and overflow incontinence ensues. In rare instances, perforation of the bladder may result from persistent ischemia triggered by over distention of the bladder or other insults [7,8]. With chronic obstruction, there may be overdistension of the ureters (hydronephrosis), and ultimately, urosepsis due to stasis [9].

3.2. Animal models of bladder overdistension

A number of animal models have been developed to investigate the pathophysiological consequences of bladder overdistension. Bladder overdistension for 3–10 h or longer is followed by decreased parasympathetic activity, structural changes in parasympathetic efferent nerve endings in the wall of the bladder, decreased cholinergic nerve density and patchy areas of hypoinnervation [10–13]. These changes are believed to be ischemic in origin. Bladder distention and contraction against a closed bladder neck have been shown to cause ischemia and hypoxia of the bladder wall followed first by endothelial cell damage, submucosal hemorrhages and submucosal edema; and then by progressive neurologic injury [14–17]. Other studies have reported that in a majority of animals, a temporary period of dysfunction is followed by full return to normal function in approximately 1–2 weeks [15–17]. The injury caused by stretching appears to be variable in severity, persistence and frequency of occurrence; and thus dependent on the duration of overdistension and pressure attained within the bladder cavity.

3.3. Effects of bladder overdistension in humans

The effects of urinary retention in humans have not been extensively studied because it is not possible for ethical reasons, to investigate the effects of bladder overdistension other than in anecdotal fashion. Thus, Mayo et al. [18] described a series of four patients with overstretched bladders that occurred during labor and childbirth. They reported frequency, stress incontinence, and the patients were only able to void by straining. One patient, who was in urinary retention for 48 h, with a residual volume of 2500 ml, had an atonic bladder for 3 weeks, but recovered normal voiding

Table 1
Causes of urinary retention

Failure or depression of bladder contraction	Neuraxial local anesthetic [5] Neuraxial or systemic opioids [41–43] Anticholinergic agents Ischemia of parasympathetic nerve endings in the wall of the bladder caused by bladder over distention [19]
Failure of sphincters to relax	Increased sympathetic activity caused by pain, emotion or bladder over distention [50]
Mechanical obstruction to urine out flow	Enlarged prostate gland Child birth Rectal pathology Radiation therapy Instrumentation
Lack of coordination between bladder contraction and sphincter relaxation	Spinal cord injury or dysfunction [4,51]
Failure of sensory input to reach spinal cord or higher brain centers	Neuropathies including diabetes Spinal cord injury or dysfunction Spinal and epidural anesthesia [5]

function after 2 months. Three others continued to void by straining; two of these received operative repairs, and one eventually required an ilioconduit. In three of these women, bladder biopsies revealed collagen deposition in the intercellular space as described in animals subjected to overdistension [19]. Although overdistension certainly occurred in all these women, other forms of bladder trauma during childbirth could have accentuated or accounted for some of the ensuing bladder dysfunction.

3.4. Repeated episodes of retention

In a report by Tammela et al. [20], it was noted that patients who developed one episode of retention after surgery, were more likely to develop a second episode than patients without an antecedent episode. The authors hypothesized that the first episode may have caused a stretching injury to the bladder that subsequently predisposed to a second episode of retention, re-retention. This relationship was confirmed in two subsequent studies [21,22]. In the latter studies, bladder distension was prevented by overnight catheterization on the night of surgery. Subsequent episodes of overdistension (> 700 ml) and retention were less common when patients had been protected by overnight catheter drainage, as compared to drainage on an as needed basis. At the very least, an initial episode of over distention may serve as a marker identifying patients prone to develop retention.

In a previous study [2], we observed no change in bladder function in 24 patients in whom postoperative bladder volumes exceeded 600 ml for 1–2 h, as compared to patients with lesser maximum volumes in the first 5 days after surgery.

Overall, the existing data suggest that sustained overdistension of the bladder (>3–4 h) is undesirable because it may be associated with temporary alteration of bladder function for days or weeks, and in some instances, may even lead to permanent damage and altered function.

Permanent injury may be manifest by one or more of the following conditions; weak stream, inability to completely empty the bladder leading to frequency and nocturia, the need for multiple, daily bladder catheterizations, or a permanent indwelling catheter.

4. Risk factors for urinary retention

There are numerous factors cited in the literature as predisposing to urinary retention [22–29]; these are outlined in Table 2.

In practical terms, the factors that are most often associated with urinary retention after outpatient surgery include:

- (a) Spinal/epidural anesthesia.
- (b) Groin hernia repair or rectal surgery.
- (c) Urologic surgery.
- (d) Previous history of urinary retention.
- (e) Underlying neurologic dysfunction.
- (f) Excessive fluid administration.

Table 2
Risk factors for urinary retention [22–29]

Surgical procedures and factors	Gynecological Anorectal Urological Inguinal hernia repair Recumbency
Urethral obstruction	Instrumentation Radiation therapy Prostatic enlargement Childbirth
Medical conditions	Preceding history of urinary retention Neurological dysfunction including diabetes Psychological factors
Anesthetic factors	Excessive fluid administration Opioids Anticholinergics Neuraxial blockade

4.1. Surgical procedures

In two previous studies, the incidence of retention in patients undergoing non-pelvic surgery, in the absence of the above risk factors, was very low —0.5% [2,23]. Similarly, the incidence of retention in patients undergoing outpatient gynecologic surgery (transvaginal surgery, or pelvic laparoscopy) was very low (0% of 40 patients). After inguinal hernia repair, the incidence has varied from 14 to 35% [2,30]. In our studies [2,23], where bladder volume was measured by ultrasound, we observed an incidence of 5%, with no incidence of recurrence (re-retention) after a single in–out catheterization in patients undergoing herniorrhaphy. After rectal surgery, reported rates have again varied from 1 to 52% [2,23,28,29] depending on methods of measurement and fluid management. We observed an incidence of urinary retention of 20% when bladder volumes were monitored by ultrasound, with a 25% incidence of re-retention following a single in–out catheterization.

In one study of recumbent patients confined to bed after foot surgery, we observed an 18% incidence of retention both in patients who had analgesia provided by sciatic nerve block, or by systemic opioids. Retention appeared to be related primarily to recumbency, particularly in patients with a history of retention (unpublished observations).

4.2. Pediatric patients

In pediatric patients, urinary retention has been less well studied. Although neuraxial (caudal) anesthesia was previously thought not to affect the ability to void in infants and children, a recent report by Koomen et al. [30] describes two children who developed urinary retention after caudal anesthesia with 0.25% bupivacaine. The diagnosis was made by ultrasound scanning of the bladder and confirmed by bladder drainage. The authors suggested ultrasound would be a useful non-invasive tool for evaluating bladder function in selected

pediatric patients after surgery (caudal anesthesia, hypospadias repair, etc.), and recently ultrasound scanners have been developed specifically for assessing bladder volume in pediatric patients.

4.3. Neuraxial anesthesia

4.3.1. Spinal local anesthesia

The incidence of retention after spinal anesthesia in adults varies considerably, again depending on the method of detection, and the type and dose of local anesthetic. A number of studies confirm that urinary retention is associated with long acting spinal anesthetics such as bupivacaine [5,31,32] and tetracaine [33]. In an elegant study, Kamphuis [5] demonstrated that the micturition reflex took on average 460 min to recover after spinal anesthesia with 10 mg of bupivacaine, compared to 235 min after 100 mg of lidocaine. More recently Breebaat et al. [31] demonstrate the ability to void after spinal anesthesia with 60 mg of lidocaine returned after 245 ± 65 min, 40 min faster than after 10 mg of levobupivacaine or 15 mg of Ropivacaine. Kopacz and colleagues have recently published a series of studies using 2-chloroprocaine as a short-acting local anesthetic for spinal anesthetics. When compared to both bupivacaine [34] and procaine [35] in volunteers, bladder function returned to normal more quickly after 2-chloroprocaine than the other two local anesthetics. Furthermore in an observational study of 122 patients who received 2-chloroprocaine spinal anesthesia [36], only five patients had problems voiding postoperatively and of these, four had undergone transurethral surgery and the other underwent a perirectal procedure.

4.3.2. Epidural local anesthesia

Mulroy et al. [32] demonstrated that epidural anesthesia with 2-chloroprocaine resulted in more rapid recovery of bladder function than lidocaine. The frequency of catheterization in women undergoing caudal epidural anesthesia for childbirth has also been demonstrated to be increased with longer acting local anesthetics [37].

When the route of neuraxial administration is compared, several studies have demonstrated more rapid resolution of the urinary effects of epidural local anesthetics compared to those of spinal local anesthesia [32,38].

4.4. Opioids and urinary retention

4.4.1. Neuraxial opioids

Urinary retention is well reported after epidural administration of morphine, the incidence varies from 15 to 90%. In some studies the effect is dose related [39,40] although Rawal et al. [41] demonstrated immediate detrusor relaxation and urinary retention lasting between 14 and 16 h in 15 male volunteers who received varying doses of epidural morphine. This study also demonstrated that complete reversal of urinary retention due to epidural morphine could be achieved with 0.8 mg of intravenous naloxone. The effects of intrathe-

cal sufentanil and morphine on bladder function were studied in human male volunteers [42]. A dose-dependant suppression of detrusor contractility and decreased sensation of urge was reported. Recovery from these effects was faster after sufentanil compared to morphine, and was dose dependent.

When fentanyl was added to local anesthetics procaine, lidocaine and bupivacaine for spinal anesthesia, the urinary effects were found to be prolonged [32].

4.4.2. Systemic opioids

Rawal [41] demonstrate that doses of intramuscular or intravenous morphine comparable to epidural opioid doses do not cause urinary retention. Other studies however have shown urodynamic effects due to systemically administered opioids. Malinovsky et al. [43] made cystomanometric measurements in postoperative patients who were given a variety of different opioids. He found that intravenous fentanyl, buprenorphine, morphine and nalbuphine all altered the central control of bladder activity and caused delayed full bladder sensation. Of the opioids studied however, only fentanyl and buprenorphine inhibited detrusor contraction.

4.4.3. Mechanisms of opioid induced urinary retention

The mechanisms of urinary retention following opioid administration, both systemically and neuraxially are multiple and not fully understood [41,43]. The neuraxial effects may occur centrally at the level of the primary micturition center in the pons where opioid receptors are present. Alternatively, the rapid onset of detrusor relaxation also suggests an inhibitory effect of epidural opioids on sacral parasympathetic outflow. It has also been suggested that opioid receptors are present in the bladder in a similar way to the ones demonstrated in the vas deferens and ileum of animals, and that urinary retention is a result of direct action of opioids on bladder opioid receptors [44].

5. The importance of residual volume and voiding by straining

In a study of voiding in patients undergoing spinal anesthesia in our institution [45], bladder volume was monitored in all patients before and after voiding, and all patients were required to void or were catheterized before discharge. Of particular interest and concern was the observation that although some patients reported having voided, the postvoid residual urine volume was still very high (400–700 ml) signifying that the micturition reflex had not really recovered. Patients however, were able to force urine flow by tightening their abdominal muscles (voiding by straining), particularly when they were aware that voiding was a necessary prerequisite to being discharged to home. In such instances, measurements of voided volume (obtained by having patients void into a urine collection receptacle) were very low, and consistent with ultrasound measurements of bladder volume before and after voiding. Patients were unable to sense overdis-

tension when it existed (painless retention); neither were nurses able to correctly estimate bladder volume using traditional methods of palpation, and knowledge of the patient's fluid status, duration of surgery, etc. Similar findings, but slightly less remarkable, were observed in patients undergoing a variety of surgeries under general or local anesthesia (non-neuraxial blocks) [23]. There was an inverse correlation between voided volume and the residual volume. Thus, it was likely that patients who had voided at least 300–400 ml had low residual volume (<200–300 ml), whereas patients who voided less than 300–400 ml tended to have high residual volumes (>300–400 ml). Using a portable ultrasound to scan the bladder provided the only reliable means of determining bladder volume before or after voiding [23]. With spinal anesthesia in particular, the residual volume was often exceedingly high (greater than the normal bladder capacity of 600 ml) even though patients reported having “voided”. Discharge of such patients may expose them to the potentially harmful side effects of prolonged overdistension. The likelihood of overdistension appears to be minimized by using short duration drugs for spinal anesthesia [32].

6. Risks of bladder catheterization

The hazards of unnecessary urinary catheterization include; urethral injury (creation of a false passage, stricture formation, prostatitis, hemorrhage), bladder injury and infection [46]. In a recent review of catheter-associated urinary tract infections (CAUTI), Tambyah [47] notes that the most important risk factors for CAUTI are prolonged catheterization, female sex and catheterization outside the sterile environment of the operating room.

Finally, most patients find catheterization, even in-out catheterization both uncomfortable and embarrassing and would prefer to avoid it if possible.

7. Ultrasound measurements of bladder volume

7.1. Validation of ultrasound measurement of bladder volume

A number of studies have demonstrated that ultrasound scanning is superior to other methods of predicting bladder volume, such as palpation, duration of surgery and estimation from the volume fluid administered intraoperatively [2,23,45,48]. In one study [23], fluids administered intraoperatively or duration of surgery had weak but significant correlations with bladder volume at the end of surgery (correlation coefficients of 0.26 and 0.32, respectively). The bladder scan measurements of bladder volume on the other hand, correlated strongly ($r = 0.9$, $p < 0.0001$) with catheterized urine volumes. In the same study patients and nurses were asked to estimate the of urine volume just before voiding. These “guesses” were compared with volume of urine measured by



Fig. 2. BladderScan™ BVI 3000 portable bladder ultrasound device.

ultrasound and by actual volume voided. Patients and nurses were unable to accurately estimate urine volume in 56 and 46% of cases, respectively [23] (Figs. 2–4).

7.2. Portable ultrasound devices

Portable ultrasound devices are available that permit non-invasive measurement of bladder volume, and in many instances avoid unnecessary bladder catheterization. They may also be used to measure postvoid residual bladder volume to confirm that voiding has been complete.

Nurses can learn to use the ultrasound device after five minutes of bedside instruction [45]. Ultrasound scanners are available in some institutions for use in the postanesthesia care unit (PACU), the emergency room and on the ward to monitor bladder volume. The portable ultrasound scanner is accurate $\pm 20\%$ at bladder volumes of <700 ml, and $\pm 25\%$ at



Fig. 3. BladderScan™ BVI 3000. The scanner is gently but firmly moved over the lower abdomen. The scanner may be used for both males and females by selecting the appropriate button during the device set up.



Fig. 4. BladderScan™ BVI 3000. Close up of the screen. The volume bladder is shown on the left and the icon on the right demonstrates a good quality scan because the white area extends across the crosshairs of the dark circle.

volumes >700 ml [49]. It is sufficiently sensitive and accurate enough to detect clinically significant overdistension.

8. Safe management of bladder function after outpatient surgery

There are several studies suggesting that voiding before discharge is unnecessary in outpatients without risk factors for an increased incidence of retention [10,23,32].

8.1. Low-risk of urinary retention

Low-risk patients can be defined as having the following characteristics:

- General anesthesia, peripheral nerve block or monitored anesthesia care (MAC).
- Non-pelvic, non-urologic surgery.
- Most outpatient gynecologic surgeries (transvaginal, or pelvic laparoscopy who undergo intraoperative bladder drainage).
- Most patients having spinal or epidural anesthesia with short-acting local anesthetics such as lidocaine, procaine or 2-chloroprocaine.

In one study of 242 low-risk patients [2], voiding was not required in patients fit for discharge. There were no subsequent episodes of retention in the 12% who left without voiding or in the remaining 88%. In patients with no other risk factors for urinary retention, who were deliberately administered significant quantities of IV fluids intraoperatively (10 ml/kg), or given anticholinergic drugs or moderate doses of opioids, there were also no incidences of urinary retention. The likelihood of retention approximated zero (one patient requested bladder drainage immediately on arriving in the recovery room with a bladder volume of 600 ml). In another study,

only one of 229 low-risk patients requested catheterization because of discomfort at a bladder volume of 420 ml before discharge [23]. Mulroy, as previously described, reported a 1.5% incidence of retention (three out of 201 ambulatory patients) with short-acting neuraxial blocks [32]. However, it should be noted that the bladder volume was monitored by ultrasound in their patients, and patients were only discharged without having voided if the bladder volume was <400 ml at the time of discharge.

8.2. High-risk of urinary retention

High-risk patients can be defined as having:

- Pelvic surgery (hernia, rectal, penile, urologic).
- A positive history of retention or spinal cord disease.
- Spinal or epidural anesthesia with agents of long duration such as bupivacaine, tetracaine and ropivacaine.
- The use of neuraxial opioids combined with local anesthetics.

Their risk of retention varies from approximately 3–20% [24–26]. Factors that may increase the likelihood of retention, particularly in high-risk patients; include mandatory recumbency, anticholinergics, neuraxial or systemic opioids, high volumes of intravenous or oral fluids.

The following recommendations are made for managing bladder function after ambulatory surgery, based on the information provided above:

- Request that all patients empty their bladder before surgery.
- Use short-acting local anesthetic agents such as lidocaine, procaine or 2-chloroprocaine for neuraxial anesthesia.
- Avoid neuraxially administered opioids and large doses of systemic long acting opioids.
- Identify patients who are NOT at significant or increased risk of retention, and allow such “low-risk” patients to be discharged without concern for voiding.
- Identify patients who are at increased risk of retention, and require that such patients either:
 - Void spontaneously and have a residual volume of <300 ml measured by ultrasound or a voided volume of >300 ml if ultrasound is not available.
 - Undergo in–out catheterization to empty the bladder completely if unable to void within an hour of otherwise being fit for discharge if ultrasound is not available or if the bladder volume exceeds 500–600 ml for 1 h as measured by ultrasound.
- Tell all patients (both high and low-risk) to return to a hospital if unable to void in 8–10 h (See Table 3).

It is important that patients and staff appreciate that overdistension for >4 h should be avoided. In Table 3 “critical times” have been calculated based on various predicted bladder volumes at the time of discharge. The critical time is the number of hours to achieve a bladder volume exceeding normal bladder capacity of 600 ml for 4 h calculated for two

Table 3
Predicted times to achieve critical bladder volume of 600 ml for >4 h

Starting residual volume in the bladder (ml)	Critical time in hours to achieve bladder volume of >600 ml for 4 h	
	Urine formation at 50 ml/h	Urine formation at 100 ml/h
0	16	10
100	14	9
200	12	8
300	10	7
400	8	6
500	6	5
600	4	4

rates of urine formation (50 ml/h and 100 ml/h), and with varying residual bladder volumes at the time of discharge. The critical time is conjectured as the limit of “safe time” for a patient to be unable to void after leaving the hospital. It is based on the results of animal studies and may be somewhat conservative.

9. Conclusion

It is important to question the old barriers to efficient recovery and discharge after ambulatory surgery. Thus, the need to void before discharge may obviously be unnecessary in a majority of situations. However, it is important that our interest and attentiveness to potential problems relating to bladder function not be discarded entirely. Rather, the emphasis must change towards educating patients and nursing staff about safer practices, treatment algorithms and modern non-invasive modalities of assessing bladder function.

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