Postoperative Urinary Retention

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KEYWORDS

• Urinary retention • Postoperative complications
• Postoperative care • Geriatrics • Voiding dysfunction

An 82-year-old male with a history of non–insulin-dependent diabetes and hypertension, both well controlled with medication, is admitted to the post anesthesia care unit (PACU) following a right inguinal herniorrhaphy. The procedure was accomplished under spinal anesthesia using 1.2 mL 0.5% bupivacaine. Blood loss was minimal and he received 1200 mL of balanced salt solution during the procedure. On admission to the PACU he is mildly sedated but arousable and reports no pain. He has some movement in his toes, but his lower extremity is still weak. Within 2 hours of his arrival, the spinal has regressed and he complains of only moderate pain. The facility requires that patients following a herniorrhaphy void before discharge, but the patient cannot urinate. One hour later he still cannot urinate and informs the nurse that he has had some prostate issues in the past.

Should you:
1) perform an in-out catheterization and discharge the patient?
2) discharge the patient with an indwelling catheter?
3) be patient and allow the patient more time to void?
4) perform an ultrasound exam of the bladder?
5) administer a small dose of naloxone?

JH Silverstein is partially funded by the National Institute on Aging Grants R01 AG029656 and R01 AG030141.
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Anesthesiology Clin 27 (2009) 465–484
doi:10.1016/j.anclin.2009.07.010 **anesthesiology.theclinics.com**
1932-2275/09/$ – see front matter

Postoperative urinary retention (PUR) can complicate any surgical procedure and is not limited to patients with preexisting urinary symptoms. Although often regarded by clinicians as a trivial or minor complication, urinary retention can be a significant source of patient anxiety and discomfort. Retention prolongs hospital stay, increases costs, and may result in significant morbidity.[1,2] In elderly patients, urinary retention can be associated with restlessness, confusion, and potential development of delirium. Urethral catheterization to treat postoperative retention conveys the risk of urinary tract infection, which increases every day a catheter remains in place.[3,4] Catheterization can result in urethral strictures, the need for additional surgery, and may be associated with a higher hospital mortality rate in vulnerable patient populations.[5] Even a single episode of bladder overdistention may lead to the deposition of collagen between the smooth muscle fibers of the detrusor, reducing their contractile function and leading to chronic impairment of bladder emptying or atony.[6] Irreversible damage to bladder nerves and postjunctional membranes has also been described.[7]

PHYSIOLOGY OF MICTURITION

The control of micturition is a complex process involving multiple afferent and efferent neural pathways, reflexes, and central and peripheral neurotransmitters. The perioperative period includes myriad insults that may interrupt this process and promote the development of urinary retention.

The bladder is under sympathetic, parasympathetic, and somatic neural control via the hypogastric, pelvic, and pudendal nerves, respectively. Lower motor neurons are under the control of upper motor neurons located in the pons, midbrain, and cerebral cortex. The storage phase of the micturition reflex is mediated by sympathetic stimulation. β_3-adrenergic receptors are found throughout the detrusor; stimulation by norepinephrine or epinephrine results in smooth muscle relaxation and permits bladder filling. Alpha-adrenergic receptors predominate in the internal urethral sphincter in both men and women, where sympathetic activation maintains contraction. Alpha receptors are also densely found throughout the bladder neck, prostate, and prostatic capsule.[8] The external urethral sphincter is a striated muscle under voluntary control. Sphincter muscle neurons located in Onuf's nucleus, in the S2-S4 segments of the spinal cord, send axons into the pudendal nerve.[9] Acetylcholine released from these axons binds to postjunctional nicotinic receptors and causes constriction of the external urethral sphincter.

When the bladder volume exceeds approximately 300 mL, impulses from stretch receptors in the bladder wall reach the sensory cortex via the pelvic splanchnic nerves; the voiding reflex can then be facilitated by centers in the pons or inhibited by midbrain centers. Central control of micturition involves multiple neurotransmitters including dopamine and serotonin. Normal bladder capacity ranges from 400 to 600 mL.[10]

The efferent limb of the micturition reflex, via parasympathetic motor nerve fibers, runs with spinal nerves from the S2 and S3 levels. These fibers terminate in ganglia within the bladder wall; short, postganglionic fibers innervate the detrusor and internal sphincter. For voiding to take place, tonic inhibition from the motor cortex must be interrupted, allowing the efferent parasympathetics to produce electrical silencing and relaxation of the external sphincter followed by detrusor contraction. Finally, the pelvic floor relaxes and the levator ani descends, allowing micturition to occur.[11]

Multiple facets of surgery, anesthesia, and the perioperative experience may interrupt the micturition reflex. Anesthesia, sedation, and analgesics interfere with the afferent limb by dulling the sensation and perception of bladder fullness, allowing painless urinary retention to develop; 61% of postoperative patients fail to experience

discomfort or a strong urge to void even as their bladder volume, as determined by ultrasound bladder scanning, exceeds 600 mL.[12] Normal functioning of the micturition reflex depends on a precise balance between sympathetic and parasympathetic tone, which is frequently disrupted during the perioperative period. Systemic sympathetic discharge and catecholamine release inhibit detrusor contraction and cause functional bladder outlet obstruction via an alpha-mediated increase in bladder outlet and proximal urethral tone. Distention of the bladder itself also leads to an increase in sympathetic motor activity.[8] Detrusor contraction can also be inhibited by a reflex involving afferent fibers of the pudendal nerve, the sacral spinal cord, and efferent pelvic parasympathetic nerves. This reflex can be triggered by peri-anal pain, dilation of the anal canal, and bladder overdistention.[13] Perineal or lower abdominal pain also inhibits the perineal relaxation that is necessary for voiding. Immobilization from intravenous lines, tubes, or drains and the need to void in the supine position can contribute to postoperative voiding dysfunction. One study found that postoperative urinary retention developed in 100% of male patients undergoing hip arthroplasty who were unable to void into a bottle while supine before surgery.[14] Some patients may also be hindered by stress, anxiety, and lack of privacy.

DIAGNOSIS OF POSTOPERATIVE URINARY RETENTION

Many studies examining the etiology of postoperative urinary retention have come to contradictory conclusions. Reconciling conflicting results is complicated by variation in how urinary retention is defined. In the past, diagnosis relied on patient discomfort or the presence of a palpable bladder.[1,13,15] The accuracy of this method is questionable, as most postoperative urinary retention is painless and physical examination unreliable. Using ultrasound monitoring, Lamonerie and colleagues[16] found that only 46% of patients with a bladder volume exceeding 500 mL appreciated that their bladders were full and felt an urge to void. The diagnosis of bladder distention by nurses has been shown to agree with ultrasound in only 54% of cases.[17] The advent of ultrasound bladder scanning, which allows rapid and accurate assessment of bladder volume, has aided in the diagnosis of postoperative urinary retention for both clinical and research purposes and is becoming available in more patient care settings.[17,18] However, variation continues to exist in the time allowed for spontaneous voiding and in the bladder volume prompting catheterization.

PERIOPERATIVE FACTORS INFLUENCING THE DEVELOPMENT OF URINARY RETENTION

Surgical Procedure

Rates of postoperative urinary retention reported in the literature vary widely. Although some variation likely reflects differing methodologies and diagnostic thresholds, trends have emerged regarding which types of surgery are likely to be complicated by urinary retention (**Table 1**).

Reported rates of urinary retention among the mixed surgical populations of large hospitals vary from 4% to 29%.[19–21] Two groups recently demonstrated that from 16% to 24% of nonambulatory patients have urinary retention upon PACU discharge, as defined by failure to void with a sonographically demonstrated bladder volume of 600 or 500 mL, respectively.[16,22] The incidence of urinary retention following outpatient surgery has been shown to be procedure-dependent. Patients undergoing hernia or anal surgery, with a combined incidence of 17%, are considered high risk for the development of urinary retention.[17] When these high-risk surgeries are excluded, urinary retention has been found to complicate only 0.0% to 0.8% of ambulatory cases.[12,17]

Table 1
Selected rates of postoperative urinary retention

Type of Surgery	Incidence of PUR	References
Low-risk ambulatory surgery	0.50%	12
Gynecological surgery	0.00%	12
High-risk ambulatory surgery	5.00%	12
Herniorrhaphy		
Local anesthesia with sedation	0.37%	59
Regional anesthesia	2.42%	59
General anesthesia	3.00%	59
Endoscopic hernia repair	4.00%–22.20%	19,24
Anorectal surgery	16.00%–20.00%	12,13,28
Mixed nonambulatory surgical		
On PACU entry	28.70%	21
On PACU discharge	16.00%–23.70%	16,22
Gynecological surgery	9.20%	34
Orthopedic		
Total knee arthroplasty	19.70%	29
Lower-limb joint replacement	18.10%	76

Patients undergoing inguinal hernia repair are particularly susceptible to postoperative urinary retention. Reported rates of retention after open inguinal or femoral herniorrhaphy vary from 5% to 26%.[12,15,23] Although some authors have found a decreased incidence with laparoscopic techniques, reports range from 4% to 22%.[24,25] Velasco and colleagues[26] reported results of a study examining postoperative complications from laparoscopic herniorrhaphy in 110 patients older than 65. The observed complication rate was 15%; however, 71% of these patients experienced acute PUR. Overall, 59% of patients were discharged on the day of surgery, and 33% were discharged on the next postoperative day.

Acute urinary retention is the most common complication after surgery for benign anorectal disease. While some series have reported incidences as low as 0.53%, the reported mean is around 15.00%.[27,28] Patients who experience an initial episode of urinary retention after anorectal surgery also appear to be prone to recurrent retention, with 50% requiring at least one additional catheterization in one study.[12]

Orthopedic patients have an elevated risk of postoperative urinary retention. Retention complicates 21% to 55% of knee arthroplasties and 11% to 48% of hip arthroplasties.[14,18,29–31] In addition to prolonging length of stay and impairing rehabilitation, urinary retention in this patient population poses a special concern, as transient bacteremia or urinary tract infection resulting from catheterization can cause deep infection in the prosthesis requiring its removal.[32] Deep joint sepsis has been reported in 0.5% to 6.2% of patients who develop urinary retention after total hip replacement.[33]

Postoperative urinary retention, diagnosed as a bladder volume in excess of preoperative bladder capacity, has been found to complicate 9.2% of elective gynecologic surgeries.[34] Although ambulatory gynecologic patients are frequently slow to void after surgery, they have been found to be at minimal risk for urinary retention, likely because of a high incidence of intraoperative catheterization resulting in decreased bladder volumes in the PACU.[17] One recent study suggests that up to 39% of women

presenting for urogynecologic assessment experience an underlying degree of significant voiding difficulty before surgery.[35]

The incidence of urinary retention after cholecystectomy reported in the literature ranges from less than 1% to 30%.[1] However, the incidence encountered in current clinical practice likely reflects the lower range of this spectrum, as the incidence has dropped with widespread adoption of laparoscopy. A meta-analysis found a retention rate of only 1.4% in patients undergoing laparoscopic cholecystectomy; the overall rate for elective cholecystectomy in modern series varies from 0.7% to 6.5% with an open approach increasing the risk of urinary retention.[1,36,37]

Prolonged postoperative urinary retention is often seen in patients undergoing complex pelvic surgery for treatment of cancers. Radical hysterectomy for treatment of cervical or endometrial cancer can lead to urinary retention, usually secondary to disruption of the sympathetic innervation from the hypogastric plexus during inferolateral dissection of the cervix. The reported rate of urinary retention requiring catheter drainage for more than 1 month after radical hysterectomy is approximately 14%.[38] Normal voiding returned within 1 month in 85.5% of patients. Another group has shown similar results with approximately 14% of women undergoing either laparoscopic or open radical hysterectomy experiencing prolonged postoperative urinary retention.[39] Another study revealed that up to 30% of women reported a feeling of incomplete bladder emptying based on self-assessment, although this was not corroborated with actual measurements of postvoid residual urine volumes.[40]

Abdominal perineal resection (APR) for the treatment of colorectal cancer is also frequently associated with prolonged urinary retention. This is most often caused by disruption of innervation from the pudendal nerve associated with the anal dissection. Reported rates of urinary retention after APR range from 15% to more than 35% and 41% for women and men respectively.[41–43] A recent prospective study of 295 women who underwent surgery for rectal cancer compared results for anterior resection and abdominal perineal resection in terms of urinary and sexual outcomes.[44] In this series, APR was associated with a significantly higher rate of urinary retention compared with anterior resection (Odds Ratio 11.7; 95% confidence interval [CI] 4.15–32.9; $P < .001$).

Anesthetic Technique

The evidence suggests that anesthetic technique affects the incidence of postoperative urinary retention. General anesthetics cause bladder atony by acting as smooth muscle relaxants and by interfering with autonomic regulation of detrusor tone. Some anesthetic agents have been shown to dramatically increase bladder capacity.[45] In vitro work with isolated human bladder strips has also demonstrated that clinical doses of halothane and thiopentone decrease bladder response to stimulation.[45] Sedative-hypnotics and volatile anesthetics inhibit the pontine micturition center and voluntary cortical control of the bladder, suppressing detrusor contraction and the micturition reflex.[46,47] Petros and colleagues[15] found that patients who underwent inguinal herniorrhaphy under general anesthesia with halothane, a potent smooth muscle relaxant, experienced a significantly higher rate of retention than cases performed with a lidocaine spinal anesthetic. The urodynamic effects of volatile anesthetics and sedative-hypnotics may be augmented by other agents commonly administered during a general anesthetic. Anticholinergic agents once commonly used for premedication and currently used in the management of the reversal of neuromuscular blockade may impair detrusor contractility and facilitate passive overfilling of the bladder by acting at cholinergic receptor sites in the smooth muscle of the bladder and urethra. However, despite the differences in their duration of action, the selection of glycopyrrolate or atropine for preventing the bradycardia associated

with reversal agents has not been found to affect the incidence of urinary retention.[48] The use of sympathomimetic agents to treat intraoperative hypotension can also promote retention, via their effects on β receptors in the bladder and α receptors in the bladder neck and proximal urethra. Indeed, a statistically significant increase in retention, to 43.8%, has been shown in patients treated with ephedrine.[21]

Intrathecal local anesthetics interrupt the micturition reflex by blocking transmission of action potentials in the sacral spinal cord. Blockade of afferent nerves results in bladder analgesia, while lack of transmission in efferent fibers causes a detrusor blockade that outlasts motor blockade by as much as several hours. Most patients will be incapable of spontaneous voiding until the sensory level has regressed to the S3 level.[49] After spinal injection of isobaric bupivacaine (20 mg), hyperbaric bupivacaine (21.5 mg), or hyperbaric tetracaine (7.5 mg), sensory block takes 7 to 8 hours to regress to the S3 level.[50] With the use of longer-acting local anesthetics, the duration of detrusor blockade allows the bladder volume to significantly exceed preoperative bladder capacity.[49] Once the bladder is sufficiently overdistended, voiding remains impaired after return of function and urinary retention develops. Many authors have substantiated the association between spinal anesthesia with long-acting local anesthetics and postoperative urinary retention.[16,17,19,21,51,52] However, recovery of bladder function after spinal injection of low-dose, shorter-acting local anesthetics occurs early enough to prevent bladder overdistention and resulting retention in most cases. Ryan and colleagues[53] demonstrated a decrease in catheterization among patients undergoing herniorrhaphy with lidocaine spinal anesthesia to 6%, compared with 30% when bupivacaine or tetracaine were used. Mulroy and collegues[54] reported that retention developed in only 2 of 201 ambulatory patients receiving short-acting epidural or spinal anesthesia. In male patients undergoing inguinal herniorrhaphy, the risk of PUR was found to be greater after spinal anesthesia than epidural anesthesia.[55]

The risk of urinary retention after neuraxial anesthesia may be modulated by other factors in addition to local anesthetic dose and duration of action. A prospective, randomized trial demonstrated that the use of epidural anesthesia did not increase the incidence of retention after hemorrhoidectomy when intraoperative intravenous fluids were limited to 200 mL ± 2 mL/kg/h of Ringer's lactate solution.[56] Lumbar spinal surgery patients experience increased rates of PUR when intrathecal local anesthetics are administered with opioids.[57,58] The addition of fentanyl to spinal anesthesia and the choice of spinal over epidural anesthesia were found to significantly increase time to discharge of ambulatory surgical patients.[54]

Local anesthesia has no negative effects on bladder function and has been found to be associated with a lower incidence of postoperative urinary retention than neuraxial or general anesthesia. A review of 72 studies found that urinary retention occurred in only 0.37% of patients undergoing hernia repair when local anesthesia with sedation was used, as opposed to an incidence of 2.42% with regional anesthesia and 3.00% with general anesthesia.[59] Very low rates of retention after herniorrhaphy under local anesthesia with sedation may reflect improved postoperative pain control resulting from local anesthetic infiltration. Urinary retention after painful groin and pelvic operations is thought to be mediated in part by reflex inhibition of detrusor contraction in response to incisional or perineal pain and edema. Perineal or lower abdominal pain also impairs relaxation of the perineal musculature, which is necessary for voiding to occur. The use of paravertebral nerve block, which also provides good postoperative analgesia, has similarly been associated with minimal risk of urinary retention after herniorrhaphy.[60]

Some studies have found an association between operative time and the development of urinary retention.[1,16,21,51] Time to void after ambulatory surgery with short-acting spinal or epidural anesthesia has been shown to correlate with total anesthetic

duration.[54] Longer operations may promote retention because of the administration of greater volumes of intravenous fluids or higher total doses of opioids and anesthetic agents; however, an association between longer procedures and urinary retention has not been confirmed in all studies.[61] Some authors have also concluded that anesthetic technique does not influence the incidence of retention.[20,34,61]

Irrespective of anesthetic modality, most patients are exposed to multiple medications throughout the perioperative period that could theoretically contribute to the development of urinary retention after surgery. By blocking the relaxing effect of beta receptors present in the bladder neck and proximal urethra, β-blockers may lead to alpha-sympathetic dominance and enhance bladder outflow resistance.[20] In neurosurgical patients, the use of β-blockers was associated with an increased risk of retention[62]; however, some studies have found that β-blockers do not influence the rate of retention.[51] Urinary retention has also been reported in association with the benzodiazepines clonazepam and diazepam and is thought to be caused by muscle relaxation.[63] The longer acting, more lipophilic benzodiazepines may be associated with higher rates of urinary retention owing to the more prolonged metabolic clearance of these agents after surgery.[63]

Intravenous Fluids

Several studies have reported that the risk of postoperative urinary retention correlates with the volume of fluids administered during the perioperative period.[22,51,61] High fluid volumes are thought to cause retention via overdistention of the bladder wall. As the bladder fills with urine, the contractility of the detrusor increases. Optimum bladder emptying occurs at an approximate volume of 300 mL, after which detrusor contractility declines.[64] Once significantly overdistended, the bladder can no longer generate sufficient contractile force to empty and retention develops.[13]

Although aggressive fluid administration has been implicated in retention occurring after a variety of procedures, its role has been most conclusively supported in patients undergoing anorectal surgery. Several early articles reported the benefits of very tight fluid restriction. Campbell[65] reported that zero anorectal patients in a series of 100 experienced urinary retention when perioperative fluids were limited to 75 mL. Bailey and Ferguson[66] found that limiting perioperative fluids to 250 mL decreased the incidence to 4% from 15% in patients receiving fluids ad libitum. Several retrospective studies have advocated more moderate fluid restriction, linking the administration of intravenous fluids in excess of 1000 mL to an increased risk of retention after anorectal surgery.[13,52] One group found that increased perioperative fluid administration increases the risk of retention in patients undergoing surgery for benign anorectal disease, but not patients having hemorrhoidectomy.[28] The American Society of Colon and Rectal Surgeons currently recommends that perioperative fluids be limited to lower the incidence of urinary retention after ambulatory anorectal surgery.[67]

Several retrospective studies in herniorrhaphy patients have found a positive correlation between the volume of perioperative intravenous fluids (IVF) administered and the incidence of postoperative urinary retention. Petros and colleagues[15] found that the risk of PUR increased after 1200 mL of intraoperative fluid. Koch and colleagues[24] found that exceeding 500 mL postoperatively conferred increased risk, whereas the volume administered intraoperatively did not affect the incidence of retention. However, in a randomized prospective trial, restricting fluids to 500 mL failed to significantly decrease the incidence of urinary retention after hernia repair.[68]

The role that intravenous fluids play in the development of urinary retention after lower risk procedures remains unclear. Many authors have failed to find a relationship between volumes of IVF administered and the incidence of retention.[20,21,53,55,69–71]

Pavlin and colleagues[12] found that the incidence of retention in low-risk ambulatory surgery patients was unaffected by randomization to the administration of high or low intraoperative fluid volumes.

Postoperative Pain and Analgesia

Postoperative pain may promote the development of urinary retention by increasing sympathetic activity, which inhibits detrusor contraction and increases outflow resistance. Perineal, lower abdominal, or pelvic pain can also directly inhibit initiation of the micturition reflex; this mechanism likely contributes to the elevated incidence of retention after hernia repair or anorectal surgery. The risk of urinary retention after hemorrhoidectomy has been found to correlate with disease severity and the amount of resection required. Distention and pain within the anal canal directly inhibit detrusor activity. More extensive dissection, resulting in increased postoperative pain and local edema, causes detrusor inhibition that promotes the development of urinary retention.[28] The risk of retention after anorectal surgery has been shown to increase with total analgesic requirement and decreases if opiates are used for pain control or if prophylactic analgesia is given with an indomethacin suppository.[13,28] The administration of local anesthetic into the operative site after hernia repair has been shown to decrease the time to voiding after surgery.[55] In neurosurgical patients, preoperative use of anti-inflammatory medications and narcotic analgesics has been found to decrease the risk of urinary retention.[62]

Opioids

Although controlling postoperative pain may reduce the contribution of increased sympathetic tone and local pain reflexes to postoperative urinary retention, opioids interrupt the micturition reflex by several mechanisms. In vitro work with isolated strips of detrusor muscle has demonstrated that encephalins act as presynaptic inhibitors of acetylcholine release from postganglionic neurons.[72] Thus, opioid analgesics reduce parasympathetic tone within the bladder, decreasing detrusor tone and permitting passive filling. They also impair perception of bladder fullness and the urge to void, decrease activity in the pelvic nerves by depressing preganglionic neurons in the sacral parasympathetic nucleus, and cause detrusor-sphincter dyssynergy secondary to failure of sphincter relaxation.[73] Many studies have found that narcotic analgesia plays a significant role in the development of postoperative urinary retention and that the risk increases with escalating doses.[1,24,29,69] Opioid-mediated depression of bladder motility is largely secondary to action at the μ-opioid receptor, and can be reversed by intravenous naloxone, which promotes detrusor contraction and sphincter relaxation. Small doses of IV naloxone (0.1 mg) have been shown to decrease bladder distention without reversing analgesia.[74]

Evidence suggests that some opioid analgesics have a greater impact on urinary function than others. One study found that meperidine use was an independent predictor of difficulty voiding after elective cholecystectomy.[36] It is also generally recommended that meperidine use be avoided in elderly patients owing to prolonged drug clearance, which can increase the risk for developing delirium. After orthopedic surgery, patients who received fentanyl for postoperative analgesia experienced significantly less urinary retention than those given morphine.[61,75]

Opioid-mediated urinary retention also appears to be influenced by route of administration. Several authors have demonstrated that risk is increased in patients using patient-controlled analgesia compared with those receiving intermittent intravenous or intramuscular opioids.[1,69] Although more effective pain control provided by patient-controlled analgesia (PCA) may lead to increased narcotic doses, some

authors have found that the additional risk conferred by PCA use cannot be attributed solely to increased total opioid dose.[76] Steadier opioid plasma concentrations achieved by PCA may enhance their impact on urinary function. Intermittent low-dose (0.1 mg IV) naloxone has been shown to increase voiding frequency, decrease bladder scan residuals, and reduce the catheterization rate in orthopedic surgery patients receiving PCA morphine without affecting pain control.[75] However, the addition of ultra–low-dose naloxone (0.006 μg/kg/h to 0.05 μg/kg/h) to PCA morphine was not found to affect rates of postoperative urinary retention.[77]

Epidural Analgesia

The highest rates of opioid-mediated urinary retention have generally been associated with epidural administration. A meta-analysis including 12,513 patients found that the use of epidural analgesia (EA) for postoperative pain control was associated with urinary retention in 23% of patients, a significant increase over the rate found in patients receiving intramuscular or PCA analgesia.[78] A meta-analysis of patients undergoing colorectal surgery found that the incidence of urinary retention increased from 1% to 10% when patients received EA instead of parenteral opioids.[79]

The effect of neuraxial opioids on voiding function may reflect peripheral, spinal, or supraspinal activity. Healthy volunteers given intrathecal morphine or sufentanil demonstrate impaired bladder contraction within 15 to 60 minutes.[80,81] Such rapid onset suggests that intrathecal opioids affect micturition primarily by inhibiting the spinal reflex responsible for detrusor contraction. A primary lumbar-spinal site of action is also supported by the increased incidence of urinary retention associated with lumbar compared with thoracic epidurals.[82] Intrathecal opioids depress preganglionic neurons in the sacral parasympathetic nucleus, decreasing pelvic nerve activity. They also activate μ and δ receptors in the dorsal horn of the spinal cord, inhibiting bladder afferents and attenuating perception of bladder sensation. Consequently, bladder capacity and compliance are increased and initiation of the micturition reflex is delayed.[83]

The extent to which epidural opioid administration negatively affects bladder motility varies with both duration of action and affinity for the μ and δ opioid receptors. Epidural morphine has been associated with rates of postoperative urinary retention as high as 10% to 15%.[84] In patients undergoing hip arthroplasty, use of epidural morphine was found to increase the incidence of bladder catheterization from 24% to 62%.[31] Healthy male volunteers given epidural morphine uniformly develop urinary retention characterized by detrusor relaxation and increased bladder capacity, which lasts an average of 14 to 16 hours.[80]

More lipophilic opioids impair bladder functioning to a lesser degree than hydrophilic morphine, as enhanced systemic uptake limits their activity at the sacral levels affecting urodynamics.[10] Recovery of normal lower urinary tract function is substantially quicker after intrathecal sufentanil than morphine.[81] A prospective, double-blind, randomized-controlled trial found that epidural anesthesia with sufentanil led to a lower incidence of micturition difficulties and bladder catheterization than morphine EA.[85] Epidural fentanyl has also been associated with a decreased incidence of urinary retention relative to morphine. One author reported that no cases of urinary retention requiring catheterization occurred in more than 3000 cases of postoperative analgesia with epidural fentanyl.[84] Animal studies have found that intrathecal fentanyl decreases mean peak urethral pressure along with bladder pressure, which may contribute to the decreased risk of bladder distention with fentanyl use.[73] A small study found that epidural tramadol, a weak μ-agonist and inhibitor of serotonin and norepinephrine uptake, increased bladder capacity and compliance without affecting voiding

function.[86] Studies offer conflicting results on the dose-dependency of urinary retention after epidural opioids.[80,81]

Nonsteroidal Anti-inflammatory Drugs and Nonopioid Analgesics

Concomitant administration of nonsteroidal anti-inflammatory drugs (NSAIDs) reduces postoperative opioid analgesic requirements and has been shown to decrease the incidence of postoperative nausea/vomiting and sedation, but not urinary retention.[87,88] The combination of propacetamol and ketoprofen was not found to decrease the incidence of retention in patients using morphine PCA after spinal surgery.[89] NSAIDS may actually facilitate urinary retention by inhibiting cyclooxygenase 2 (COX-2) and decreasing intravesical prostaglandin E_2 (PGE_2) levels. PGE_2 releases tachykinins, which stimulate detrusor smooth muscle and afferent nerve receptors, and has also been shown to relax urethral smooth muscle.[90] NSAID users have been found to experience a twofold increased risk of acute urinary retention.[63] The opioid-sparing effects of ketamine, when administered with morphine PCA after spinal surgery, were found to decrease the incidence of PUR in a small study.[91]

DEMOGRAPHIC RISK FACTORS FOR POSTOPERATIVE URINARY RETENTION

Age and Gender

Both gender and advancing age appear to modulate the risk of postoperative urinary retention. Multiple studies have concluded that older patients of both genders are at increased risk of retention.[16,19–22,28,61,69,76] The aging process includes multifactorial changes that alter voiding function and may promote the development of urinary retention after surgery.

Detrusor function deteriorates progressively with advancing age; changes may be myogenic or neurogenic in origin and include diminished detrusor contractility and pressure.[92,93] Even healthy older adults with normal urodynamic profiles exhibit evidence of impaired detrusor contractility.[92,93] In some it progresses to detrusor underactivity, characterized by contractions of decreased strength or duration that result in prolonged bladder emptying or failure to empty the bladder within a normal period.[94] Although the true incidence of overt detrusor underactivity remains unclear, the condition is particularly prevalent in frail elderly women, a population prone to PUR after orthopedic surgery. One study demonstrated that 79% of women with proximal hip fractures had bladder volumes greater than 300 mL on admission. Bladder overdistention was also present in 37% of patients before surgery and developed in 56% within the first 24 hours after surgery.[5]

Impaired bladder emptying is reported in 10.8% of women and 22.1% of men older than 60.[95] More than half of men older than 50 have lower urinary tract symptomatology.[96] Bladder outlet obstruction secondary to benign prostatic hyperplasia (BPH) is the primary source of bladder morbidity in elderly men, although some may experience comorbid detrusor decompensation, which can develop independently or in response to untreated BPH.[94] Several studies have confirmed that older males are at an increased risk for the development of PUR.[15,18,51]

Bladder sensation declines with advancing age and is manifested by an increase in the bladder volume prompting the urge to void.[92] Diabetes mellitus, one of the most common comorbid diseases in older adults, is often associated with impairment in bladder sensation, increased bladder capacity, and decreased contractility.[97] Some authors have found that patients with diabetes are prone to the development of postoperative urinary retention.[21,98] Impaired baseline bladder sensation may augment the contribution that decreased afferent activity secondary to anesthetics,

sedative-hypnotics, and analgesics makes to the development of retention. The aged may also be more susceptible to the negative urodynamic effects of analgesic and anesthetic agents because many of these drugs have a prolonged duration of action in the elderly.

The impact that gender has on PUR in younger patients remains unclear. Some authors have found no link between gender and the risk of PUR.[21,29,52] Others have reported that females are at greater risk or that women between the ages of 21 and 40 are predisposed to retention.[13,20]

Abnormal Voiding History

Whether patients with preoperative signs and symptoms of bladder outlet obstruction or other voiding dysfunction are predisposed to PUR remains unclear, but has been supported in some studies.[13,51] Tammela and colleagues[20] found that 80% of patients who developed postoperative urinary retention had abnormal voiding histories such as a history of retention after surgery or symptoms of bladder irritation or obstruction. One study of male herniorrhaphy patients found that postoperative urinary retention developed exclusively in patients with abnormal preoperative postvoiding residuals.[23] A previous history of urinary retention was found to be a predictor of PUR after knee arthroplasty.[29] One study found that whereas neither physical exam nor a detailed urological history could distinguish male hip arthroplasty patients at increased risk for retention, diminished preoperative peak urinary flow rate was a significant predictor of increased risk.[30]

However, many studies have refuted the claim that preoperative voiding dysfunction predisposes to the development of retention after surgery. Patients with preoperative signs and symptoms of bladder outlet obstruction undergoing surgery for benign anorectal disease were not found to be at increased risk of PUR.[28] Multiple studies of orthopedic surgery patients have concluded that a history of urological problems cannot predict those who will go on to develop urinary retention.[18,61,99] One recent prospective study examined 102 men undergoing either total knee or hip arthroplasty to determine if a history of voiding dysfunction measured by a standardized rating scale was predictive of PUR.[100] Although 30.4% of the men in this study developed PUR, only age older than 70 years was identified as a significant predictor variable. A case-control study of patients undergoing laparoscopic hernia repair found that those with a history of urinary retention, BPH, or prostate cancer were not at increased risk of PUR.[24]

MANAGEMENT OF POSTOPERATIVE URINARY RETENTION

Preoperative Consultation

The benefits of taking a more complete urological history as part of routine preoperative evaluation of surgical patients remains unclear, as baseline voiding dysfunction has not been consistently demonstrated to predict patients at increased risk of developing PUR. Ambulatory patients being considered for accelerated pathways permitting discharge without voiding may require screening for preoperative voiding dysfunction or a history of retention, as studies validating the safety of early discharge have excluded this patient population.[12,54]

Intraoperative Considerations

High-risk patients may benefit from efforts to minimize the risk of bladder overdistention. Patients managed without indwelling urethral catheterization should be required to void immediately before surgery. One study found that 14% of patients undergoing

common orthopedic and general surgery procedures had a sonographic bladder volume greater than 300 mL immediately before the induction of anesthesia.[64] Further research is required to determine optimum management of IV fluids; although lower fluid volumes may decrease retention rates in some patient populations, this effect must be balanced against the need to support the circulation when spinal or epidural anesthesia is used and the beneficial affect of adequate hydration on decreasing postoperative nausea and vomiting.

Diagnosis of Postoperative Urinary Retention

Postoperative urinary retention is best diagnosed noninvasively with a portable ultrasound bladder scanner, which permits rapid and accurate measurement of bladder volume.[101] This approach has been shown to be superior to diagnosis dependent on physical exam or patient symptoms and avoids unnecessary catheterization of patients with minimal bladder volumes who have failed to void secondary to underresuscitation. The ideal bladder volume at which patients should be catheterized is unclear. The benefits of earlier intervention in preventing detrusor damage and possible persistent voiding dysfunction resulting from bladder overdistention must be weighed against exposing patients to the discomfort and infectious risks associated with catheterization. As normal adult bladder capacity ranges from 400 to 600 mL; a threshold of 600 mL has been recommended.[10,17] Several studies have demonstrated that elevated bladder volumes on PACU entry increase the subsequent risk of developing urinary retention and that the incidence of bladder overdistention on PACU discharge may be high.[16,22,71] Further investigation is warranted to determine the role of more widespread use of portable ultrasound bladder monitoring in the PACU.

Duration of Bladder Catheterization

Urinary retention after ambulatory surgery is commonly managed with in-out urethral catheterization. However, the choice of indwelling or clean intermittent urethral catheterization for postoperative urinary retention in inpatients remains controversial. Animal studies have demonstrated that retention results in detrusor damage secondary to collagen infiltration and impaired contractility.[102,103] Many of these changes are reversible if the obstruction is relieved, and it has been suggested that bladder decompression facilitates normalization of detrusor contractility.[103] Consequently, some clinicians favor prolonged bladder decompression after an episode of acute urinary retention. A randomized study in patients undergoing total hip or knee replacement found that the use of indwelling catheters removed the morning after surgery decreased the risk of bladder distention, urinary retention, and the need for long-term catheterization compared with intermittent catheterization, without increasing the risk of urinary tract infection.[99] However, another randomized trial found that hip fracture patients who were intermittently catheterized on a regular schedule that prevented bladder overdistention experienced a quicker return to normal voiding than those managed with a preoperative indwelling catheter removed 48 hours after surgery.[104] Certain patients may be at increased risk for persistent voiding dysfunction and recurrent retention and therefore benefit from longer-term indwelling catheterization. Most rectal surgery patients require only 1 day of urinary drainage, but those with low-rectal cancers and metastatic lymph nodes are at increased risk of retention and best managed by transurethral catheterization for 5 days.[105] Further study is needed to clarify which patients benefit from indwelling catheterization as well as the optimal duration of bladder catheterization.

Pharmacological Therapy

Several drugs, primarily alpha-blockers and parasympathomimetics, have been investigated for their potential to prevent or treat postoperative urinary retention. The noncompetitive, long-acting alpha-blocker phenoxybenzamine facilitates micturition by decreasing urethral outflow resistance and enhancing intravesical pressure. Several studies found that prophylactic treatment with phenoxybenzamine successfully decreased the incidence of postoperative urinary retention. This effect was demonstrated in small studies of patients undergoing abdominal or vaginal hysterectomy, hemorrhoidectomy, colorectal surgery, genital prolapse repair, and inguinal herniorrhaphy.[23,106,107] Phenoxybenzamine was found to decrease rates of postoperative urinary retention among patients receiving regional anesthesia with concomitant administration of large volumes of intravenous fluids to maintain blood pressure.[8] It was also found to decrease the incidence of retention, need for catheterization, and urinary tract infection in women undergoing elective caesarian section under epidural anesthesia and receiving epidural morphine for postoperative pain control.[108] However, most studies examining prophylactic alpha blockade to prevent postoperative urinary retention were conducted at a time when routine surgeries were followed by extended hospital stays that permitted monitoring for side effects. Phenoxybenzamine is rarely used clinically now because of the potential carcinogenic risk associated with this medication. Modern alpha-blockers such as tamsulosin, alfuzosin, or long-acting doxazosin also offer the advantage of initial administration at therapeutic doses without the need for titration. One study demonstrated that administration of prazosin around the time of hernia repair significantly reduced the incidence of urinary retention compared with placebo (10.8% versus 25.0%).[109] Catheterization rates were significantly reduced as well (3.5% versus 13.8%). Although enthusiasm for preoperative administration of alpha-blockers has waned in the era of ambulatory surgery and fast-track discharge, they are still frequently used in the postoperative treatment of urinary retention, particularly in men.

Interestingly, several benzodiazepines have also been considered for the prevention or treatment of urinary retention after surgery. A randomized controlled trial found that lorazepam administered 1 hour after operation in patients undergoing ambulatory gynecological surgery had no affect on the time to void after surgery.[70] Another study found that midazolam was an ineffective treatment for postoperative urinary retention.[110]

Parasympathomimetic agents such as bethanechol and urecholine theoretically act to increase bladder detrusor contractility. However, although a pharmacologic effect can be demonstrated, their clinical utility is limited owing to poor efficacy and unfavorable side-effect profiles. Hindley and colleagues[111] showed limited clinical improvement in voiding outcomes using a combination of PGE_2 and bethanechol compared with placebo. In a series of women undergoing radical hysterectomy, Madeiro and colleagues[112] demonstrated that although administration of bethanechol appeared to improve bladder emptying, it was associated with an increase in detrusor instability and urinary urgency symptoms.

Many patients presenting for surgery, particularly the elderly, take medications that affect voiding function. Optimal perioperative management of these patients remains unclear.

Discharge Criteria for Ambulatory Surgery Patients

Traditionally, patients presenting for ambulatory surgery have been required to void before discharge; however, evidence suggests that this prerequisite may not be

necessary for all patients. Fewer than 1% of patients undergoing nonpelvic surgery under general anesthesia, peripheral nerve block, or local anesthesia with sedation will develop urinary retention, defined as the inability to void at a bladder volume of 600 mL detected by bladder ultrasound. Most of these patients will void within 3 hours of their procedure. Requiring this low-risk population to void before discharge will delay 5% to 6% of discharges.[17] Pavlin and colleagues[12] found that low-risk patients could be safely discharged with instructions to return to hospital if voiding had not occurred within 6 to 8 hours of discharge. High-risk patients, those undergoing hernia or anorectal surgery or a procedure under spinal or epidural anesthesia, were monitored with bladder ultrasound and catheterized at a bladder volume of 600 mL. Catheterized patients had an elevated risk of reretention (25%).

The risk of urinary retention in low-risk patients undergoing spinal or epidural anesthesia with short-acting agents may also be sufficiently minimal to permit discharge before voiding.[54] Mulroy and colleagues[54] evaluated an accelerated discharge pathway for ambulatory patients undergoing procaine, lidocaine, or bupivacaine (6 mg or less) spinal anesthesia or epidural anesthesia with 2-chloroprocaine or lidocaine. Patients having hernia, rectal, or urologic surgery, those older than 70, and those with a prior history of voiding difficulty were excluded. Accelerated pathway patients who failed to void spontaneously were discharged if bladder ultrasound demonstrated a bladder volume less than 400 mL. Twenty-three percent of accelerated pathway patients were successfully discharged before voiding; none suffered voiding difficulty or had to return to hospital because of retention. The accelerated discharge protocol shortened discharge time by a mean of 22 minutes.[54]

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