Long-term risks of bladder augmentation in pediatric patients

J. Christopher Austin

Department of Urology, University of Iowa, Iowa City, USA

Correspondence to J. Christopher Austin, MD, FAAP, Department of Urology, University of Iowa, 200 Hawkins Dr., 3 RCP, Iowa City, IA 52242-1089, USA
Tel: +1 319 356 0743; fax: +1 319 356 3900;
e-mail: chris-austin@uiowa.edu

Current Opinion in Urology 2008, 18:408–412

Purpose of review
Bladder augmentation is still a commonly performed reconstructive procedure for pediatric patients with severe bladder dysfunction. Recent developments in the long-term risks associated with this procedure are reviewed.

Recent findings
There are metabolic changes in these patients after incorporation of bowel into the urinary tract. Linear growth and bone mineral density are more affected by the primary disorder rather than bladder augmentation. There is a high rate of reoperation in patients after bladder augmentation for perforation, bladder stones, and bowel obstruction. Bladder cancer has been reported in neurogenic bladder patients after bladder augmentation but also in these patients without augmentation.

Summary
Bladder augmentation is associated with a number of potential long-term risks, including a high risk of needing further surgery and development of serious complications such as bowel obstruction or bladder perforation. Bladder stones continue to be common in patients after bladder augmentation. Multiple cases of bladder cancer have been reported recently in young adults with a history of bladder augmentation in childhood and reinforce the need for lifelong follow up for these patients. Future studies will hopefully define the benefits and role of cancer surveillance for these patients.

Keywords
bladder augmentation, bladder cancer, bladder exstrophy, bladder stones, spina bifida

Curr Opin Urol 18:408–412

0963-0643

Introduction

Bladder augmentation reliably increases the bladder capacity, improves bladder compliance, and often cures incontinence due to neurogenic bladder dysfunction. It is frequently performed on pediatric patients with spina bifida and bladder exstrophy. Most surgeons will exhaust all conservative methods for the treatment of bladder dysfunction before recommending bladder augmentation. The biggest concerns are the potential postoperative complications and long-term risks that come with bladder augmentation. Bowel segments commonly used include ileum and sigmoid colon, with stomach being used infrequently. Notable long-term risks include the formation of bladder stones, chronic bacteruria, mucusuria, metabolic changes from exposure of the bowel to urine, bladder perforation, bowel complications, and the potential risk of bladder or bowel patch malignancy. In this article these long-term complications will be reviewed.

Metabolic complications

The incorporation of intestine into the urinary tract results in predictable metabolic changes due to the absorptive properties of bowel. When exposed to urine, colon or ileum results in the absorption of ammonium and chloride ions which can cause hyperchloremic metabolic acidosis [1]. The extent and clinical significance of this absorption depends on the patient's renal function and the amount of intestine used for bladder reconstruction. Most bladder augmentation procedures use significantly less small or large bowel than the amounts used for neobladder formation after cystectomy, and thus rates of metabolic acidosis in patients undergoing bladder augmentation are very low with normal renal function [2,3].

The primary clinical concern with chronic metabolic acidosis is the possibility of skeletal growth impairment and osteopenia [4]. There has been an ongoing debate as to whether or not bladder augmentation leads to impaired skeletal growth and bone demineralization. Publications have both supported and refuted these effects on patients [2,3,5,6,7•,8–11]. Differences in study design and length of follow up have contributed to these differences. Osteopenia has typically been measured using dual energy X-ray absorptiometry scanning. Decreased bone density in patients following bladder augmentation has been reported in up to 75% of patients with neurogenic bladder dysfunction and in up to 32% of patients without neurogenic

Table 1 Summary of publications examining the effect of bladder augmentation on growth and bone mineral density

Author	*n*[a]	Patients	Outcome	Control[b]	Conclusions
Taskinen *et al.* [7•]	54	C	BMD	No	Patients at risk for osteopenia but not due to augmentation
Boylu *et al.* [6]	15	C	BMD	No	Decreased BMD due to underlying disease, not bladder augmentation
Canturk *et al.* [5]	6	E	% Ht/BMD	Yes	Decreased BMD and height not different in patients with bladder augmentation
Hafez *et al.* [3]	25	E	% Ht/BMD	No	32% with decreased BMD; normal height
Mingin *et al.* [2]	33	C	% Ht/BMD	Yes	No difference in height and BMD versus controls
Gerhartz *et al.* [8]	123	E	% Ht	Yes	No significant effect on growth with extended follow up
Feng *et al.* [9]	18	E	% Ht	Yes	Height % less in augmentation patients
Gros *et al.* [10]	17	E	% Ht	Yes	% height decreased after surgery in 82%

C, combined series of patients with neurogenic bladder and exstrophy; BMD, bone mineral density; E, series of exstrophy patients and nonneurogenic bladder patients; % Ht, height measurement percentiles based on age from growth charts.

[a] Number of patients with bladder augmentation in study.

[b] Studies indicated as 'controlled' used age and disease-matched control groups or preoperative values from individual patients rather than normal historical values from healthy patients.

bladder [3,6]. Table 1 summarizes more recent published studies on growth and bone mineral density in children undergoing bladder augmentation. Many studies are uncontrolled and compared their results with historically published age and sex-matched normal values. Ambulatory and activity status, as well as renal function have known significant effects on bone mineral density which must be controlled for [5,6,7•]. When all these factors are considered, it does not appear that pediatric patients suffer from decreased linear growth or significant bone demineralization due to bladder augmentation [2,6,7•,8].

Gastrocystoplasty has been used in patients with compelling reasons to avoid the use of small or large intestine. These include patients with short gut syndrome or prior history of radiation therapy to the pelvis [11]. The stomach has a significantly different physiology when incorporated into the urinary tract, as its function is secretion of acid and enzymes for digestion rather than absorption. Gastric augmentation can therefore eliminate the absorptive problems with intestine including metabolic acidosis [12]. The secretion of acid can irritate the bladder and urethra, leading to hematuria and dysuria, which is present in varying degrees of severity in up to one-third of patients and most problematic if the patients have normal bladder and urethral sensation [13–15]. Most cases of hematuria/dysuria syndrome are minor and can be controlled pharmacologically with H2 histamine blockers or proton pump inhibitors. The secretion of acid and chloride can predispose patients to develop metabolic alkalosis. In times of illness and dehydration these effects can lead to severe hypochloremic, hypokalemic metabolic alkalosis [13]. Given the risks of metabolic complications, serum electrolytes should be monitored routinely during follow up after bladder augmentation.

Need for reoperation

Bladder augmentation is a major surgical procedure with multiple short and long-term potential complications. As the majority of pediatric patients who undergo bladder augmentation have either spina bifida or bladder exstrophy, the chances are that they have undergone multiple prior surgical procedures. Parents will want to know what the chances are that their child will need further surgery for bladder augmentation related problems. A recent large review showed that the reoperation rate for bladder augmentation related procedures was 34% [16]. This emphasizes the importance of preoperative counseling to families and patients that bladder augmentation comes with a significant risk of reoperation.

Bladder perforation

Most pediatric urologists consider bladder perforation to be one of the most serious complications related to bladder augmentation. It is a potentially life-threatening complication which needs to be suspected in any patient with a prior history of bladder augmentation presenting with abdominal pain or other symptoms of an acute abdomen. The incidence of this dreaded complication varies from series to series but its occurrence is unfortunately not a rare event. Bladder perforation rates up to 13% have been reported [17]. A large series of 500 consecutive bladder augmentations from Indiana University reported an 8.6% rate of perforation [18]. One of the patients died as a result of this complication. The risk was highest in those with sigmoid colon used as the augmentation segment (27% versus 5% for ileum and stomach). These findings are in contrast to those of DeFoor *et al.* [19] who reported a higher rate with ileum (4% versus none for gastric or sigmoid augmentation). Most patients with bladder augmentations have spina bifida and a ventriculoperitoneal shunt. Perforation causes not only peritonitis but also potential shunt infection and malfunction [20]. It is recommended that shunts be temporarily externalized if perforation occurs. Most authors recommend immediate exploration and repair of perforations, although select patients have reportedly been managed nonoperatively with success [17–21]. Causes of perforation remain the

subject of speculation but include traumatic catheterization, noncompliance with catheterization, chronic or acute overdistention, infection, and ischemia. Risk factors for perforation include bladder neck surgery, presumably due to the increased outlet resistance allowing for increased storage pressure [22•]. Having a continent catheterizable channel may be protective, possibly by facilitating catheterization to improve patient compliance with catheterizations [18]. Patient compliance with intermittent catheterization is difficult to objectively measure. It is important to emphasize the need for life-long intermittent catheterization and compliance with this regimen, as the consequences of perforation of the augmented bladder can be disastrous.

Bowel obstruction

Late mechanical bowel obstruction is a relatively uncommon event for patients after bladder augmentation. The incidence of 2–6% has been reported with long-term follow up after enterocystoplasty. The Indiana group [16] recently reported a 3.2% rate of bowel obstruction requiring surgical treatment. They noted that bowel obstruction was six times more common in patients with gastric augmentations, with 10% requiring surgical treatment of a bowel obstruction versus ileum (2%) and sigmoid colon (3%). It was hypothesized that this increase was related to the long vascular pedicle traversing the abdomen. DeFoor *et al.* [15] reported a need for surgical treatment of bowel obstruction following gastrocystoplasty in 5% at a mean follow-up of nearly 10 years. There is no clear advantage of the use of one type of bowel segment over the other with regards to bowel obstruction.

Bladder stones

The formation of bladder stones is one of the most common long-term problems encountered with bladder augmentation patients. Its occurrence is not limited to patients with augmentation [23]. The risk is present with any bowel segment used but is the lowest after gastrocystoplasty [16,24,25]. This is likely due to the decreased rates of bacteruria and mucus production following gastrocystoplasty compared with other bowel segments. While nearly 100% of patients have chronic bacteruria after bladder augmentation with bowel, it is only present in one-third of patients following gastrocystoplasty [15]. When stones are diagnosed they should be treated. Routine surveillance for bladder stones with yearly radiographs of the abdomen (KUB) or ultrasound of the bladder is recommended [23]. Most stones are composed of struvite, but may also form on foreign bodies [24]. There has been an ongoing debate as to the best way to prevent and manage these stones. Potential surgical treatments include cystoscopic, percutaneous, or open removal. The choice of treatment should be individualized to the patient based on the size and number of stones, and the potential for safe and effective endoscopic treatment. The time to recurrence for bladder stones has been shown to be similar for open versus endoscopic removal and recovery is shorter for patients treated endoscopically [26]. A higher incidence of bladder stones has been reported in patients who catheterize through a continent catheterizable channel versus urethral catheterization [16,23]. It is hypothesized that urethral catheterization will facilitate more complete bladder emptying and removal of accumulated mucus and debris more efficiently. Hensle *et al.* [27] showed in one longitudinal series that irrigation regimens using saline to remove mucus decreased the incidence of bladder stones from 43 to 7%. Despite treatment, the rate of recurrence, which has been reported to be as high as 44%, emphasizes the need for continued surveillance postoperatively [26]. The role of metabolic factors is being explored as a contributing factor for bladder stone formation. Hamid *et al.* [28••] reported 24 h urine results in 15 patients with stones and bladder augmentation compared with controls with bladder augmentation but no history of stones. The patients with stones had significantly lower urine volumes, increased calcium excretion, and lower citrate levels. There have been no studies demonstrating the effect of altering these factors on bladder stone recurrence, but this is an intriguing finding not often considered in patients with bladder stones.

Bladder cancer

Of all potential complications the most worrisome is the potential that bladder augmentation increases a patient's risk for bladder cancer. Bladder cancers in patients who have previously undergone augmentation cystoplasty were first reported in adults who had undergone augmentation for conditions such as genitourinary tuberculosis [29]. The first reports of pediatric patients with bladder augmentations developing cancer in adulthood were reported more recently. Soergel *et al.* [30] reported three patients with spina bifida who died from metastatic transitional cell cancer in young adulthood. Subsequently, other studies have reported bladder cancer in patients who have had bladder augmentation with all types of bowel segments. Gastric carcinoma has been reported in multiple patients following gastric augmentation and sets this bowel segment apart, as malignancies of the small bowel and colon have not yet been reported in patients undergoing bladder augmentation as children [31,32,33•,34••]. Malignancies of the bowel patch have been rarely reported in adults predominately when colon was used for augmentation [35]. These reports have created concern about the long-term follow up for these patients and the need for bladder cancer screening.

The incidence of bladder cancer in patients with bladder augmentation with bowel has been estimated to be

between 1.2 and 3.8% [30]. The risk with gastrocystoplasty may be higher, with rates of 1–10% in individual series [33•]. All reports and estimates are based on retrospective reviews. Smeulders and Woodhouse [36] have estimated that adult patients with bladder exstrophy have an estimated risk of bladder cancer of 4% at age 40, which is a 27-fold increase from the general population. Most series were limited only to patients with bladder augmentation. No study has conclusively proven that bladder augmentation is an independent risk factor for development of bladder cancer. In a series of patients with neurogenic bladder dysfunction due to spina bifida treated for bladder cancer at the University of Iowa [37••], eight patients were reported but only one patient had prior bladder augmentation. Combining all prior published cases of patients with spina bifida and bladder cancer with those eight showed a median age at diagnosis of 35 years and only 37% had undergone bladder augmentation. The demographics and tumor histologic types for these patients are shown in Tables 2 and 3. The most striking difference in the patients with bladder cancer associated with spina bifida (whether with bladder augmentation or not) was the stage of the tumor and survival. While nearly two-thirds of adults presenting with bladder cancer have superficial, low-stage tumors, nearly all patients with spina bifida had advanced stage, invasive bladder cancer, with nearly all having locally invasive disease or lymph node metastases. Additionally, nearly half have other rare histologic tumor types, such as adenocarcinoma or squamous cell carcinoma. The mean survival for these collective patients was 17 months.

This high-stage, aggressive disease with poor survival is why these patients are being strongly considered for bladder cancer screening programs. Various protocols have been recommended, including cystoscopy. There are no published trials of results of screening protocols for these patients. The only comparable studies come from patients with spinal cord injury. These patients have been known to have an increased risk of bladder cancer which peaks at 20–30 years after their injury [38]. There have been multiple studies evaluating the value of screening cystoscopy in these patients and none have shown a definitive advantage [39–41], though one study showed a trend toward lower stage tumors in patients that underwent screening [41]. More important than screening programs is patient and clinician education about this problem. Most patients still present with gross hematuria but may ignore it, or they have been treated with courses of antibiotics for chronic bacteruria rather than evaluated for alternative causes for the hematuria, such as a bladder tumor. The youngest patient reported was 19 years old and the median age is 35 [34••,37••]. This is much younger than adults with bladder cancer typically present. In addition, patients will lack the traditional risk factors for bladder cancer, such as smoking. It is important to consider bladder cancer in young adults with lifelong bladder dysfunction with complaints of hematuria, more frequent urinary tract infection, difficulty catheterizing, or changes in continence and other symptoms.

Table 2 Demographics of 19 published patients with spina bifida and bladder cancer

Median age	37 years (range 23–73)
Bladder augmentation	37%
Median interval from augmentation to cancer	14 years (range 8–21)
Median survival	6 months (range 1–55)
Patients with advanced stage at diagnosis	93%

Table 3 Tumor histology for 19 published patients with spina bifida and bladder cancer

Tumor histology	% patients
Transitional cell carcinoma	58
Squamous cell carcinoma	21
Adenocarcinoma	16
Gastric signet ring carcinoma[a]	5

[a] Formed in the stomach portion of the augmentation.

Most recent studies reporting bladder cancer in adults with pediatric bladder dysfunction or bladder augmentation have recommended a screening protocol with yearly or biennial cystoscopy, cytologies, and ultrasound of the kidney and bladder [30,33•,34••,37••]. At our institution, we recommend yearly cystoscopy, cytologies, and renal and bladder ultrasound for patients with neurogenic bladder dysfunction on clean intermittent catheterization at age 20 or 10 years after bladder augmentation. It is unknown if this evaluation will be frequent enough to improve the stage at diagnosis and survival, but our experience so far justifies following these patients closely. We are also educating our patients to return sooner for evaluation for gross hematuria or other new symptoms related to their bladders. Over the next decade studies will hopefully clarify the role and benefits of screening these patients for bladder cancer, and perhaps offer some insight as to why these tumors form and behave differently from most adults with bladder cancer.

Conclusion

Bladder augmentation is associated with a number of potential long-term risks, including a high risk of further surgery and development of serious complications such as bowel obstruction or bladder perforation. Bladder stones continue to be common in patients after bladder augmentation. Multiple cases of bladder cancer have been reported recently in young adults with a history of bladder augmentation in childhood and reinforce the need for lifelong follow up for these patients. Future studies will hopefully define the benefits and role of cancer surveillance for these patients.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 442–443).

1 Koch M, McDougal W, Thompson C. Mechanisms of solute transport following urinary diversion through intestinal segments: an experimental study with rats. J Urol 1991; 146:1390–1397.

2 Mingin G, Nguyen H, Mathias R, *et al.* Growth and metabolic consequences of bladder augmentation in children with myelomenigicele and bladder exstrophy. Pediatrics 2002; 110:1193–1198.

3 Hafez A, McLorie G, Gliday D, *et al.* Long-term evaluation of metabolic profile and bone mineral density after ileocystoplasty in children. J Urol 2003; 170: 1639–1641.

4 Minervini R, Felipetto R, Cecchi M, *et al.* Densiometric and biochemical alterations in bony tissues induced by ureterosigmoidostomy. Urol Int 1995; 54:217–219.

5 Canturk F, Tander B, Tamder B, *et al.* Bladder exstrophy: effect on bone age, bone mineral density, growth, and metabolism. Bone 2005; 36:69–73.

6 Boylu U, Horsanili K, Tanriverdi O, *et al.* Evaluation of bone mineral density after ileocystoplasty in children with and without myelomeningocele. Pediatr Surg Int 2006; 22:375–379.

7 • Taskinen S, Fagerholm R, Makitie O. Skeletal health after intestinal bladder augmentation: findings in 54 patients. BJU Int 2007; 100:906–910.

A recent study of 54 patients after bladder augmentation demonstrating the significant differences in the bone mineral density of neurogenic versus ambulatory patients.

8 Gerhartz E, Preece M, Duffy P, *et al.* Enterocystoplasty in childhood: a second look at the effect on growth. BJU Int 2003; 91:79–83.

9 Feng A, Karr S, Elder J. Influence of enterocystoplasty on linear growth in children with exstrophy. J Urol 2002; 167:2552–2555.

10 Gros D, Dodson J, Lopatin U, *et al.* Decreased linear growth associated with intestinal bladder augmentation in children with bladder exstrophy. J Urol 2000; 164:917–920.

11 Kurzrock E, Baskin L, Kogan B. Gastrocystoplasty: is there a consensus? World J Urol 1998; 16:242–250.

12 Gosalbez R, Woodard J, Broeker B, Warshaw B. Metabolic complications of the use of stomach for urinary reconstruction. J Urol 1993; 150:710–712.

13 Kurzrock E, Baskin L, Kogan B. Gastrocystoplasty: long-term followup. J Urol 1998; 160:2182–2186.

14 Plaire C, Snodgrass W, Grady R, Mitchell M. Long-term followup of the hematuria-dysuria syndrome. J Urol 2000; 164:921–923.

15 DeFoor W, Minevich E, Reeves D, *et al.* Gastrocystoplasty: long-term follow-up. J Urol 2003; 170:1647–1650.

16 Metcalf P, Cain M, Kaefer M, *et al.* What is the need for additional bladder surgery after bladder augmentation in childhood? J Urol 2006; 176:1801–1805.

17 Sherkarriz B, Upadhyay J, Demirbilek S, *et al.* Surgical complications of bladder augmentation: comparison between various enterocystoplasties in 133 patients. Urology 2000; 55:123–128.

18 Metcalfe P, Casale A, Kaefer M, *et al.* Spontaneous bladder perforations: a report of 500 augmentations in children and analysis of risk. J Urol 2006; 175:1466–1471.

19 DeFoor W, Tackett L, Minevich E, *et al.* Risk factors for spontaneous bladder perforation after augmentation cystoplasty. Urology 2003; 62: 737–741.

20 Barker G, Lackgren G, Stenberg A, Arnell K. Distal shunt obstruction in children with myelomeningocele after bladder perforation. J Urol 2006; 176:1726–1728.

21 Adams W, Shortliffe L. Management of delayed bladder augmentation perforation. Nat Clin Pract Urol 2006; 3:341–344.

22 • Blok B, Al Zahrani A, Capolicchio J, *et al.* Postaugmentation bladder perforation during urodynamic investigation. Neurourol Urodyn 2007; 26:540–542.

Case report of spontaneous rupture during a urodynamic study when a patient's bladder was being filled to a supranormal bladder capacity at the point where the fill pressures were increasing, cautioning against continuing filling during studies past the point at which the bladder compliance decreases.

23 Barroso W, Jednak R, Fleming P, *et al.* Bladder calculi in children who perform clean intermittent catheterization. BJU Int 2000; 85:879–884.

24 Kaefer M, Hendren W, Bauer S, *et al.* Reservoir calculi: a comparison of reservoirs constructed from stomach and other enteric segments. J Urol 1998; 160:2187–2190.

25 Defoor W, Minevich E, Reddy P, *et al.* Bladder calculi after augmentation cystoplasty: risk factors and prevention strategies. J Urol 2004; 172:1964–1966.

26 Roberts W, Gearhart J, Mathews R. Time to recurrent stone formation in patients with bladder or continent reservoir reconstruction: fragmentation versus intact extraction. J Urol 2004; 172:1706–1709.

27 Hensle T, Bingham J, Lam J, Shabsigh A. Preventing reservoir calculi after augmentation cystoplasty and continent urinary diversion: the influence of an irrigation protocol. BJU Int 2004; 93:585–587.

28 •• Hamid R, Robertson W, Woodhouse C. Comparison of biochemistry and diet in patients with enterocystoplasty who do and do not form stones. BJU Int 2008; 18 February [Epub ahead of print].

A study of the 24 h urine results of patients who do and do not form stones after enterocystoplasty. The patients who formed stones had decreased urine volumes, citrate levels, and increased calcium excretions. These findings suggest that metabolic and dietary factors may play a role in bladder stone formation in patients after enterocystoplasty.

29 Filmer R, Spencer J. Malignancies in bladder augmentations and intestinal conduits. J Urol 1990; 143:671–678.

30 Soergel T, Cain M, Misseri R, *et al.* Transitional cell carcinoma following augmentation cystoplasty for the neuropathic bladder. J Urol 2004; 172: 1649–1651.

31 Baydar D, Allan R, Castellan M, *et al.* Anaplastic signet ring cell carcinoma arising in gastrocystoplasty. Urology 2005; 65:1226.

32 Esquena Fernandez S, Abascal J, Tremps E, Morote J. Gastric cancer in augmentation gastrocystoplasty. Urol Int 2005; 74:368–370.

33 • Castellan M, Gosalbez R, Perez-Brayfield M, *et al.* Tumor in bladder reservoir after gastrocystoplasty. J Urol 2007; 178:1771–1774.

A case series of four patients with bladder cancer following gastrocystoplasty. Three patients had gastric carcinoma of the stomach patch. This series suggests that gastrocystoplasty may result in high tumor development of the bowel patch.

34 •• Vemulakonda V, Lendvay T, Shnorhavorian M, *et al.* Metastatic adenocarcinoma after augmentation gastrocystoplasty. J Urol 2008; 179:1094–1097.

This series of two patients with gastric adenocarcinoma following gastrocystoplasty again suggests that this bowel segment may have an increased predisposition to malignancy. The authors report that only two of 72 patients were affected but recommend a screening protocol including annual imaging, cystoscopy and biopsy in patients 5 years following gastrocystoplasty.

35 Austen M, Kalble T. Secondary malignancies in different forms of urinary diversion using isolated gut. J Urol 2004; 172:831–838.

36 Smeulders N, Woodhouse C. Neoplasia in adult exstrophy patients. BJU Int 2001; 88:623–628.

37 •• Austin JC, Elliot S, Cooper CS. Patients with spina bifida and bladder cancer: atypical presentation, advanced stage, and poor survival. J Urol 2007; 178: 798–801.

This series of eight adult patients with spina bifida and bladders shows that bladder cancer can occur in patients in the absence of bladder augmentation. The cancers are atypical in presentation and usually advanced at presentation. Survival was poor. Review of prior published cases reveal that only 37% have undergone bladder augmentation and median age of patients is 35 years.

38 Pannek J. Transitional cell carcinoma in patients with spinal cord injury: a high risk malignancy? Urology 2002; 59:240–244.

39 Hamid R, Bycroft J, Arya M, Shah P. Screening cystoscopy and biopsy in patients with neuropathic bladder and chronic suprapubic indwelling catheters: it is valid? J Urol 2003; 170:425–427.

40 Yang CC, Clowers DE. Screening cystoscopy in chronically catheterized spinal cord injury patients. Spinal Cord 1999; 37:204–207.

41 Navon J, Soliman H, Khonsari F, Alhering T. Screening cystoscopy and survival of spinal cord injured patients with squamous cell cancer of the bladder. J Urol 1997; 157:2109–2111.