Augmentation Cystoplasty: What Are the Indications?

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Introduction
Attempts to increase bladder capacity date back to the end of the 19th century. The first ileocystoplasty was performed in dogs by Tizzoni and Foggi in 1888 [1], shortly followed by cystoplasty in humans by Mikulicz in 1989 [2]. A wide variety of tissues, ranging from intestinal segments to fascial grafts and gallbladder [3], have been studied as potential materials for augmentation. Intestinal segments gained popularity in the mid-1950s and after successful introduction of intermittent catheterization, augmentation cystoplasty became widely accepted and used by the urologic community. Short- and long-term complications of intestinocystoplasties along with advances in cell biology and tissue engineer-

Anatomic/Structurally Contracted Bladder
Bladder compliance is defined by the viscoelastic properties of the bladder wall stroma and facilitated by spinal sympathetic reflexes. The viscoelasticity of the stroma is determined by the ratio of collagen and elastin. Insults to the bladder stroma such as chronic inflammation, radiation therapy, chronic bacterial, or parasitic infections can lead to the replacement of bladder stroma by collagen and, therefore, a decrease in bladder compliance. This decrease in bladder compliance caused by collagen deposition in the bladder wall is often not responsive to pharmacologic manipulation, behavioral modification, or nerve stimulation. Commonly, augmentation cystoplasty is required to achieve normal storage function.
**Tuberculous contracted bladder**

In the past, tuberculous cystitis was the number one indication for bladder augmentation. Development of potent antituberculous drugs has decreased prevalence of severe cystitis requiring augmentation. The genitourinary tract is the most common site, after the lungs, for tuberculous infection. The infection almost always affects the kidneys during the primary exposure to infection but does not present clinically. The spread to the kidneys from the lungs is usually hematogenous. Bladder tuberculous infection is almost always secondary to renal involvement. Initially, cystitis occurs, eventually causing bladder mucosal ulceration and thickening of the bladder wall. End-stage disease causes scarring and bladder fibrosis, resulting in diminished capacity of the urinary bladder. Bladder wall calcification is uncommon. Bladder tuberculosis may also be complicated by fistulae or sinus tract formation, although these complications are rare. Patients with tuberculous bladder need to be evaluated by an infectious disease specialist before surgery and appropriately cleared for augmentation surgery. The appropriate course of antituberculous drugs should also be completed preoperatively.

**Radiation cystitis**

Radiation therapy is an important management tool for the treatment of malignancies of the pelvis such as prostate, colon, rectum, uterus, or ovary. Despite significant improvement in administration of the radiation therapy, there is still a significant potential for the development of injury to the urinary bladder. Approximately 15%–20% of patients undergoing pelvic radiation will develop chronic bladder complications, sometimes presenting years after therapy [5]. A small proportion will require surgical intervention to alleviate symptoms related to scarred small capacity bladder. Radiation cystitis patients constitute less than 5% of bladder augmentation patients. The low number of augmentations performed after radiation therapy is likely a reflection of advanced age and comorbidities in this older patient population compared with patients such as those with spinal cord injury who tend to be younger, healthier, and not dealing with a potential life-threatening cancer. Before proceeding with surgical treatment, general health and oncologic prognosis of the patients after pelvic radiation must be considered. Overall, more than 60% of patients with radiation cystitis undergoing bladder augmentation are satisfied with their degree of continence and their voiding pattern after enterocystoplasty [6•].

**Schistosomiasis**

Schistosomiasis is one of the most common parasitic infestations in the world and is caused by the *Schistosoma* genus of fluke. The form of schistosomiasis affecting the urinary tract involves *Schistosoma haematobium*. The disease is endemic in the Middle East, India, Africa, Central America, and South America; yet, it is rare in the United States. Calcification in the wall of the bladder or distal ureters can be identified on plain radiographs. With regard to the bladder, indistinctness or hazy changes are caused by submucosal edema and pseudotubercules. The body forms an intense granulomatous reaction to the ova, and fibrosis ensues. This fibrosis traps the ova in the tunica propria of the bladder wall where the ova die and become calcified. The bladder wall becomes fibrotic, but can be distensible and may maintain a normal capacity. Similar to tuberculosis, in addition to detailed clinical urologic evaluation of the patient’s bladder, the decision to proceed with bladder augmentation has to be supported by the infectious disease specialists.

**Neurogenic Bladder**

As mentioned earlier, storage/filling of the bladder depends on bladder compliance, absence of detrusor contraction, and competency of the bladder outlet. In the setting of neurologic disease or injury, sympathetic facilitation of bladder compliance is altered and increased afferent input leads to decreased compliance without viscoelastic changes to bladder wall.

Isolated decreased bladder compliance without overt active detrusor is commonly seen in lesions below the sacral cord or with injury to the peripheral reflex arc. Diminished compliance in combination with an intact or dyssynergic outlet leads to a high pressure, small capacity bladder with potential for subsequent upper urinary tract damage. Decreased compliance with an incompetent outlet can lead to urinary incontinence and symptoms of urinary frequency/urgency in the sensate bladder.

Uninhibited bladder contractions with dyssynergia of the striated sphincter or both striated (DESD) and smooth sphincters are commonly seen in lesions above the sacral spinal cord (Fig. 1). In these patients every bladder contraction generates a force against the dyssynergic sphincter and therefore propagates pressure upwards. This often leads to prominent and severe upper tract injury if not addressed appropriately in a timely manner. Patients with DESD are often initially managed with therapies aimed at either the outlet or the bladder. To minimize dysynergia, patients can be offered transurethral incision of the external sphincter through a variety of methods such as laser, cold knife, and hot knife sphincterotomy. This can be combined with reflex voiding in patients using an external collection device, such as a condom catheter. Efforts to decrease bladder pressures can be medically attempted with the use of anticholinergics; this form of management is often combined with clean intermittent catheterization (CIC). If adequately low storage pressures cannot be maintained or patients continue to have urinary incontinence between catheterizations, surgical intervention may be required.

Although there are some general correlations between the level of injury and bladder behavior, several studies have