Pelvic Neuroanatomy and Technique of Abdominoperineal Resection of the Rectum for Cancer

We read with interest the recent article by Khatri et al.[1] The authors emphasize the need to conceptualize the dissection on the basis of a thorough understanding of the regional anatomy that confers improved safety. We fully agree that meticulous surgical technique is mandatory to radically remove the cancer while preserving important anatomic structures such as the membranous urethra, prostate, and presacral veins, but we are disappointed that Khatri et al[1] omitted the details of pelvic neuroanatomy and did not discuss autonomic nerve preservation, which is important to proper urinary and sexual function, during abdominoperineal resection of the rectum.[2]

Recent research results[2,3] support the feasibility of preserving pelvic neural structures but also stress their variable location, which can create difficulties in ensuring nerve preservation. In men, the inferior hypogastric plexus is located between the urinary bladder and rectum in 73% and pararectally in 27%.[3] In women, 57% of the plexuses are in the uterosacral ligament, 30% are located parametrially, 11% are between the urinary bladder and the uterus, and 2% lie pararectally.[3] Incomplete knowledge of these facts can lead to surgical misadventures. An anatomic region at high risk for injury to pelvic autonomic nerves is the retrorectal space, where the superior hypogastric plexus and the pelvic and sacral splanchnic nerves lie. Dissection along the middle rectal artery increases the risk of injury to the nerves because of the nerves' mediolateral orientation. The anterior aspect of the inferior hypogastric plexus is endangered during interventions near the ureter, uterine or inferior vesical arteries, seminal glands, and Denonvilliers fascia.[3] We appreciate the detailed anatomic description outlined by Khatri et al[1] of the arteries, muscles, and fasciae encountered during abdominoperineal resection of the rectum, but the pelvic neuroanatomy is equally important.

The distinguishing clinical characteristics of rectal cancer are a high incidence of local recurrence and autonomic nerve dysfunction after surgery.[2,3] Detailed knowledge of the anatomy of pelvic autonomic nerves enables identification and preservation of these nerves when combined with total mesorectal excision.[2] The value of the anatomic description provided by Khatri et al[1] would be increased by adding details about pelvic neuroanatomy, an important consideration in the contemporary management of rectal cancer.[2,3]

Julian E. Losanoff, MD
Edward R. Sauter, MD, PhD
Columbia, Mo

Corresponding author: Edward R. Sauter, MD, PhD, Department of Surgery, M580 Health Sciences Center, University of Missouri-Columbia School of Medicine, 1 Hospital Dr, Columbia, MO 65212 (e-mail: SauterE@health.missouri.edu).

1. Khatri VP, Rodriguez-Bigas MA, Petrelli NJ. Perineal dissection of synchronous abdominoperineal resection of the rectum: an anatomical description. *Arch Surg.* 2003;138:553-559.
2. Havenga K, Maas CP, DeRuiter MC, Welvaart K, Trimbos JB. Avoiding long-term disturbance to bladder and sexual function in pelvic surgery, particularly with rectal cancer. *Semin Surg Oncol.* 2000;18:235-243.
3. Baader B, Herrmann M. Topography of the pelvic autonomic nervous system and its potential impact on surgical intervention in the pelvis. *Clin Anat.* 2003; 16:119-130.

In reply

We thank Drs Losanoff and Sauter for their comments regarding our recent article. We are aware of the importance of pelvic neuroanatomy during the abdominal portion of rectal mobilization and routinely perform meticulous pelvic nerve-sparing total mesorectal excision for the surgical treatment of rectal cancer. However, the purpose of our article was to elucidate and emphasize the equally important perineal portion of the abdominoperineal dissection, which seems to be lacking in surgical training programs. We appreciate the response by Drs Losanoff and Sauter and would emphasize to general surgery training program directors that both the perineal and pelvic anatomy should be included as part of the training in rectal cancer surgical treatment.

Vijay P. Khatri, MD
Sacramento, Calif
Miguel Rodriguez-Bigas, MD
Houston, Tex
Nicholas J. Petrelli, MD
Newark, Del

Corresponding author: Vijay P. Khatri, MD, Division of Surgical Oncology, University of California, Davis Cancer Center, 4501 X St, Sacramento, CA 95817 (e-mail: vijay.khatri@ucdmc.ucdavis.edu).

Supranormal Trauma Resuscitation and Abdominal Compartment Syndrome

I congratulate Dr Balogh and associates for their excellent article "Supranormal Trauma Resuscitation Causes More Cases of Abdominal Compartment Syndrome."[1] Their findings reinforce several important aspects of intra-abdominal hypertension (IAH) and abdominal compartment syndrome (ACS) that we

published about 5 years ago.[2] The authors did not refer to our article, but the observations in the 2 studies are remarkably similar.

The authors emphasize the differences between IAH and ACS and make the observation that untreated IAH may lead to full-blown ACS. In addition, they mention a bladder pressure of 25 mm Hg as the critical pressure beyond which the undesirable sequelae of ACS appear to set in—points similar to those we made in a group of critically injured patients with penetrating abdominal trauma who developed primary IAH. As in the present article, our patients who had higher levels of bladder pressure also had evidence of gastric mucosal acidosis as measured with gastric mucosal pH, an observation also made by other authors.[3]

The authors' proposed cause for secondary ACS is similar to what we hypothesized for primary ACS resulting from catastrophic abdominal injuries and coagulopathy. Of particular interest is our observation, in concordance with findings in the present study, that in patients who have had sequential hits of hemorrhagic shock and subsequent IAH, the respiratory and splanchnic effects of IAH may be more severe than with either insult alone. The increased frequency of IAH associated with increased multiple organ failure and mortality was observed in our clinical study[2] and in the laboratory.[4-7] In our article,[2] we also suggested that these serious complications should be anticipated in patients who are critically injured and/or undergoing damage-control procedures. An attempt must be made to prevent IAH and ACS by leaving the abdomen open, as has been suggested by other authors.[8] Such an approach may actually decrease multiple organ failure and mortality.

This is an important study that establishes supranormal resuscitation as a cause of secondary ACS, advances the concept that IAH and ACS are gradations of the same disease, and reaffirms the splanchnic basis of multiple organ failure associated with these complications. Because the splanchnic bed seems to be adversely affected by both inadequate and overzealous resuscitation, what methods do the authors propose to prevent ACS caused by either of these 2 mechanisms?

Rao R. Ivatury, MD
Richmond, Va

1. Balogh Z, McKinley BA, Cocanour CS, et al. Supranormal trauma resuscitation causes more cases of abdominal compartment syndrome. *Arch Surg.* 2003; 138:637-643.
2. Ivatury RR, Porter JM, Simon RJ, et al. Intra-abdomnal hypertension after life-threatening abdominal trauma: prophylaxis, incidence, and clinical relevance to gastric mucosal pH and abdominal compartment syndrome. *J Trauma.* 1998;44:1016-1021.
3. Sugrue M, Jones F, Lee A, et al. Intraabdominal pressure and gastric mucosal pH: is there an association? *World J Surg.* 1996;20:988-991.
4. Simon RJ, Friedlander MH, Ivatury RR, et al. Hemorrhage lowers the threshold for intra-abdominal hypertension-induced pulmonary dysfuction. *J Trauma.* 1997;42:398-403.
5. Friedlander M, Simon RJ, Ivatury RR, et al. Effect of hemorrhage on superior mesenteric artery flow during increased intra-abdominal pressures. *J Trauma.* 1998;45:433-439.
6. Oda J, Ivatury RR, Blocher CR, et al. Amplified cytokine response after sequential insults of ischemia-reperfusion and abdominal compartment syndrome. *J Trauma.* 2002;52:625-632.
7. Rezende-Neto JB, Moore EE, Melo de Andrade MV, et al. Systemic inflammatory response secondary to abdominal compartment syndrome: stage for multiple organ failure. *J Trauma.* 2002;53:1121-1128.
8. Mayberry JC, Mullins RJ, Crass RA, et al. Prevention of abdominal compartment syndrome by absorbable mesh prosthesis closure. *Arch Surg.* 1997;132: 957-961.

In reply

We are honored that Dr Ivatury has commented on our work.[1] His seminal observations related to ACS are well known to our group, and we referenced 2 of his articles. We believe that ACS is in part an iatrogenic complication of current management of traumatic shock (ie, damage-control surgery combined with goal-oriented resuscitation in the intensive care unit). With ongoing analysis of severely injured patients who require aggressive resuscitation, our concepts of ACS pathogenesis have evolved.[1-7]

We first noted the iatrogenic nature of overzealous perihepatic packing. As a result, we minimize intraperitoneal packing and practice early pack removal. We next began to use presumptive Bogota bag (an opened, empty, sterile intravenous fluid bag) closure after damage-control laparotomy but soon recognized that this does not eliminate the problem. We therefore use routine urinary bladder pressure (UBP) monitoring.

However, UBP has limitations. Patients who develop ACS typically require emergency operative or interventional radiological procedures and arrive in the intensive care unit on average 6 hours after admission with established IAH (mean UBP of 19 mm Hg). Conventional wisdom has been IAH falsely elevates commonly measured indexes of preload (eg, central venous pressure and pulmonary capillary wedge pressure), and, therefore, patients with low cardiac output should be volume loaded. We have observed that volume loading sets up a futile cycle of crystalloid preloading. Initial volume loading may increase cardiac output, but it also increases intra-abdominal pressure. Increased intra-abdominal pressure compresses the inferior vena cava, which adversely affects cardiac preload and promotes intestinal edema. With continued preloading, UBP increases into the low 20s. At this point, the abdomen is on the steep portion of its compliance curve. A small increase in abdominal content results in a large increase in intra-abdominal pressure, which is often detected too late with intermittent UBP measurements to avoid full-blown ACS.

In regard to the question posed by Dr Ivatury, we believe that fundamental changes need to occur in management before patients reach the intensive care unit. From our models, we can accurately predict the clinical trajectory of ACS within 3 to 6 hours. These patients arrive in obvious shock, but their sources of bleeding are not always readily apparent. This is especially true in patients with blunt trauma who have pelvic and extremity fractures. Early volume loading during the ABCs (airway, breathing, circulation) of advanced trauma life support is valuable in defining hemodynamic stability, but continuing aggressive volume loading during uncontrolled bleeding sets the stage for ACS.

Failure to promptly recognize the severity of ongoing blood loss is a recurring problem in the emergency department. Early base deficit determination and central venous pressure monitoring are valuable adjuncts. Blood and fresh frozen plasma should be used liberally, and indiscriminate crystalloid infusions should be minimized. Hemorrhage con-