Evidence Based Surgical Hypothesis

Metabolic response to abdominal surgery: The 2-wound model

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An abdominal operation combines a somatic abdominal wall wound with a second autonomic wound to the peritoneal cavity and viscera and little attention has been paid the autonomic/peritoneal wound that communicates directly to the brain by the vagus nerve. Moreover, vagal input originating from the peritoneum modulates and regulates postoperative recovery. Consequently, blockade of the afferent neural and inflammatory input from this autonomic/peritoneal wound will reduce postoperative neurohormonal stress and enhance patient recovery from an abdominal operation. (Surgery 2011;149:301-4.)

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The lesion which produces a painful scar is in the brain, not at the site of the wound.
—George Crile, Surgeon and Co-founder of the Cleveland Clinic, 1864–1943

HIP ARTHROPLASTY AND ABDOMINAL SURGERY FOR BENIGN CONDITIONS are both classified as “major” surgery with large systemic neuroendocrine responses. However, that is where the similarities end. In contrast with limb surgery, which disrupts the somatic nervous system alone, abdominal operations such as colectomy are accompanied by the formation of 2 different wounds, a somatic and an autonomic wound. These wounds combined are the source of loss of organ function, autonomic dysregulation, catabolism, dependency, and high vulnerability to internal and external risks. The experience for the patient is dominated by loss of control, inability to eat, disturbance of the circadian rhythm, lack of sleep, and a labile psychological state. Even after successful discharge from hospital, the patient continues to feel tired, despondent, and lacking energy to carry out usual daily tasks for up to 3 months. This debilitating postoperative fatigue results in failure to meet social and economic obligations.

Although the central nervous system (CNS) serves as the key coordinator of the maintenance and amplification of the postinjury metabolic responses exactly what drives the CNS remains somewhat poorly understood.1 This paper hypothesizes that, in abdominal surgery, there are 2 wounds that drive the CNS to coordinate a complex array of metabolic responses.

After an abdominal operation, 2 wounds are created (Fig 1). First, a wound is made in the abdominal wall to access the abdominal viscera; this is referred to from here on as the somatic wound. It is proposed that understanding the other wounds created: The peritoneal and visceral wounds, collectively making up the autonomic wound, are of vital importance where the disruption to these is great, such as after colectomy. The somatic wound reaches the CNS by the spinal cord, but the bulk of the information sent to the CNS from the autonomic wound utilizes the vagus nerve.

THE SOMATIC WOUND

The anterolateral abdominal wall is innervated by the thoracolumbar nerves from the anterior rami of various roots, projecting from the posterior column of the spinal cord. The effect of the somatic wound, whatever the size, may be “clinically controlled” by neural-axial blockade such as epidural anesthesia. This provides excellent analgesia and decreases catabolism after colectomy.2 As a result, the somatic wound has historically been uppermost in the minds of surgeons and anesthetists. This focus on the somatic wound has found expression in the ever

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developing field of minimal access surgery, which may eventually lead to virtually incisionless abdominal operations.

When one considers procedures where the abdominal wall incision is the cause of the predominant metabolic insult to the patient (e.g., cholecystectomy), the benefits of a minimal access laparoscopic approach are obvious compared with classical open cholecystectomy. However, when the intraperitoneal wound is large, the patient is given a substantial autonomic challenge, no matter the size of the abdominal wall wound. Hence, the advantages of a small somatic wound do not seem to be as dramatic where large visceral disruption is required to achieve operative outcomes.

Laparoscopic colonic operations, where the somatic wound is minimized, surprisingly have only marginal clinical benefits over open colonic procedures. In nonoptimized and nonblinded settings, this benefit may be detected with a modest reduction of hospital stay duration. However, in an optimized double blinded perioperative care setting (where evidence-based clinical pathways are utilized to provide standard perioperative care), recovery after colectomy is not detectably different when laparoscopy is compared with open operative interventions. It is therefore postulated that the somatic wound impact may not be as important, in the greater scheme, on the postcolectomy clinical state than previously thought. Thus, other factors may help to explain the prolonged convalescence after major transperitoneal operations.

**THE AUTONOMIC WOUND**

The peritoneum is a highly metabolically active sheet of tissue that envelops the majority of the abdominal viscera. This living sheet conveys sensory fibers via the subdiaphragmatic vagus nerve, which bypasses spinal cord blockade. The vagus nerve is the largest visceral sensory nerve in the body with approximately 50,000afferent fibers, most of which innervate the peritoneum. Between 80% and 90% of the subdiaphragmatic vagus is afferent in nature, indicating an important role in direct peritoneal to CNS signal transmission. Thus, vagal inputs originating from the peritoneum have great potential to modulate and regulate physiology and behavior in humans.

In addition to the peritoneum, viscera have nociceptors that are distinct and unique entities that must also be considered. Abdominal viscera are innervated by the spinal cord and vagus projections with different contributions from each. A large...
component of this afferent system (40–45% in the colon and bladder) is by normally unresponsive “silent nociceptors” that become activated only in the presence of inflammation and injury and have substantial capacity for downstream amplification. It is relevant that discharge from these nociceptors is greater in magnitude and duration after the inflammatory process has set in than during the acute insult. This produces dramatic changes in the environment that surrounds the nerve endings with the potential to excite distant nociceptors not affected by the initial insult. Thus, downstream effects may persist long after the initial injury has resolved.

Systemic proinflammatory marker concentrations (traditionally utilized to assess the inflammatory effect of major abdominal operations) represent only a small fraction of that generated from the peritoneum. Although comparative animal studies of cellular immunity after laparoscopic and conventional transperitoneal surgery have demonstrated some immunologic advantages after laparoscopy, this has not been observed in humans. The peritoneal cytokine levels are similar for laparoscopic and open colonic procedures. This suggests that, in humans, the 2 approaches are equally traumatic locally in their effect on the peritoneum and viscera and that the somatic wound is relatively minor compared with the autonomic wound in this context. This may help to explain why a recent clinical review has confirmed that laparoscopic procedures in the optimized recovery setting do not confer significant additional clinical advantages in major abdominal operations.

What seems to be important in abdominal surgery is the extent to which the peritoneum is itself entered, dissected, and manipulated. This is demonstrated by transperitoneal aneurysmal repair resulting in a significantly higher inflammatory response and slower clinical recovery compared with the extraperitoneal approach. Therefore, it is probable that the autonomic wound is a significant contributor to the overall metabolic response and clinical picture after major transperitoneal abdominal operations.

Gastrointestinal afferent pathways constitute only 7–10% of all inflow to the spinal cord, with the vagus nerve carrying by far the majority of the peritoneal and visceral nociceptor message to the CNS. This is supported by animal studies, which have demonstrated that vagal afferents respond to nociception from mechanical and chemical stimulation and that these signals lead to brain stem activation. Vagotomy blunts this response, but spinal cord transection does not.

The challenge that the autonomic wound presents to the surgeon and anesthetist is substantial. Vagotomy in humans in this context is unethical; however, it is possible to perform a transient...
chemical afferentectomy at the site of peritoneal injury with intraperitoneal local anaesthetic.\textsuperscript{12} Intra-abdominal plexus blockade may also reduce the neuroendocrine response to surgery, in addition to that provided by epidural blockade.\textsuperscript{13} Furthermore, it has been shown that it is possible to partially dampen local abdominal inflammation with glucocorticoids and that this is associated with measurable clinical benefits.\textsuperscript{14}

Figure 2 presents a schema of the pathways activated after abdominal surgery. This diagrammatically illustrates the site of action of current interventions. Substantial work has been carried out on minimizing the size of the access wound to viscera; natural orifice translumenal endoscopic surgery, which is currently in the early stages of development, has the potential to avoid creating a somatic wound altogether. Systemic anti-inflammatories, such as steroids, cause a global dampening of responses. However, transperitoneal disruption possesses a major challenge. An effective autonomic wound blockade is possible by utilizing local anesthetic directed to the intraperitoneal cavity at the site of dissection, but at present this modality is incomplete and only partially effective.

We hypothesize that the operative insult to the peritoneal cavity and viscera may be controlled with local anesthetic directed at the site of injury in the intraperitoneal cavity. When combined with other regional anesthetic techniques that target the somatic nervous system a complete but transient chemical afferentectomy to reduce the autonomic stressor and metabolic burden of intraperitoneal dissection is possible.

REFERENCES


