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EDITORIAL

# Intraperitoneal wound in abdominal surgery

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## Abstract

The intraperitoneal wound is often forgotten after transperitoneal surgery. This review is a on the peritoneum and the implications of peritoneal injury after surgery. This review will focus on the intraperitoneal wound response after surgical injury.



**Key words:** Peritoneum; Vagus nerve; Abdominal surgery; Cytokine; Laparoscopy; Inflammation

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## INTRODUCTION

An abdominal operation combines a somatic abdominal wall wound with a second wound to the peritoneal cavity and viscera. Little attention has been paid the peritoneal wound that communicates directly to the brain by the vagus nerve. Traditionally most interventions have focused on producing relief from the physiological burden created by the somatic abdominal wall wound. However recent research indicates that the "forgotten" intraperitoneal wound may be clinically important especially in those who undergo extensive transperitoneal injury.

## PERITONEUM

The peritoneum is a serous membrane that lines the abdominal cavity and the intra-abdominal viscera. This dynamic cellular membrane has important functions[1]. It provides a frictionless environment for movement of abdominal organs and is a metabolically active sheet of tissue that envelops the majority of the abdominal viscera and is protected by macrophage scavengers. These scavengers play a key role in the local immune response by producing local mediators such as interleukin-6 (IL-6), tumor necrosis factor (TNF-$\alpha$) and oxygen radicals[2]. Furthermore the peritoneum is unique compared to surrounding organs in that it carries a lower level of anti-inflammatory pathways resulting to a greater adhesion forming pathways after injury compared to the regeneration that occurs in other organs[3]. The peritoneum is highly metabolically involved and active, enveloping the majority of the abdominal viscera[4].

Because the entire peritoneal cavity is linked *via* transcoelomic spread of immuno-humoral factors in the peritoneal fluid, it exhibits a coordinated response to injury which is generalised and not limited to the localised area of insult[5,6]. This is supported by the fact that there are much higher cytokine concentrations in peritoneal fluid than in plasma after gastrointestinal surgery suggesting that cytokine production occurs in a compartmentalised fashion within the abdominal cavity[7,8].

The nerve supply to the peritoneum is conveyed along the autonomic nervous system from the parasympathetic and sympathetic system. It can convey sensory fibres via the cranial nervous system, namely the sub-diaphragmatic vagus afferents. The sub-diaphragmatic vagal afferents, about 50000 in number are almost all made from low threshold unmyelinated (C) fibres. They convey background sensations such as mechanical stretch, satiety, fullness, nausea and vomiting sensations[9]. Afferent vagal inputs originating from the peritoneum and abdominal viscera have great potential to modulate and regulate

behaviour in humans[10,11]. Furthermore, 90% of the subdiaphragmatic vagus is entirely afferent in nature, indicating a critically important role in direct peritoneal to central nervous system signal transmission and modulation of inflammatory processes arising from the peritoneum[10,12,13].

Spinal afferent also supply the parietal peritoneal linning and mesentery of the gastrointestinal tract, and have cell bodies located in the dorsal root ganglion projecting to the dorsal horn of the spinal cord. They follow the paths of the sympathetic (splanchnic) and parasympathetic (pelvic) efferents to the gut wall[14]. All of the visceral afferents combined make no more than 7%-10% of all afferent inflow to the spinal cord[12,14].

## SURGICAL TRAUMA TO THE PERITONEUM

Comparative studies of cellular immunity after laparoscopic and conventional trans-peritoneal surgery have demonstrated immunologic advantages conferred by reducing the somatic abdominal wall wound by performing minimal access laparoscopy[15]. In these animal models there appears to be a biological peritoneal advantage after laparoscopy when compared to open surgery. Authors have hence argued, that the peritoneal immune response after laparoscopic surgery is better preserved[15]. This mechanism is uncertain but has been thought to arise from smaller peritoneal incisions minimizing peritoneal stress, reduced exposure of peritoneum to solubilized pathogens in air, and minimal manipulation and handling of the organs.

A large component of the intra-abdominal afferent system (40%-45% in the colon and bladder) are by fibres normally unresponsive to stimuli that become activated only in the presence of inflammation and injury[16]. These "silent nociceptors" are different in that they are mainly concerned with tissue injury rather than mechanical stimuli such as stretch. One theory is that these class of nociceptors lead to abnormal autonomic regulations by insult which produces dramatic changes in the environment that surrounds the nerve endings with potential to excite distant nociceptors not affected by the initial insult[17]. What is also concerning about intraperitoneal nociceptors is that as they are not normally active, discharge after inflammation and injury may be greater in magnitude and duration than the discharges produced by acute injurious stimuli, which potentially makes the central effects even greater than the initial insult[18]. Transcoelomic spread of pro-inflammatory cytokines may activate areas of the peritoneum distant from the site on intraperitoneal injury. Thus downstream effects may persist for a long duration even after the initial injury is near or complete resolution.

The abdominal wall wound can be reduced significantly in size, by minimally access techniques, such as laparoscopy. When one considers procedures where the incision is the cause of the predominant metabolic insult to the patient, the benefits would appear to be obvious and of a significant clinical magnitude. For example in cholecystectomy the metabolic response is thought to be from the abdominal wall wound itself[19]. Therefore it can be postulated that this should translate into a lesser magnified sickness response and hence quicker recovery. The benefits of laparoscopic cholecystectomy are evident when compared to classic open colectomy[20] but not as obvious when compared to the small or "mini" laparotomy version of the same operation[21]. Therefore there may be a threshold size effect where further benefits are not seen.

However what is interesting is that in humans the peritoneal cytokine response is similar in laparoscopic and open colonic surgery[22,23]. Also the systemic pro-inflammatory concentrations after both surgical approaches represent only a small fraction of what is generated from the peritoneum. This suggests that the two intra-abdominal approaches are locally equally traumatic to the peritoneal cavity[22,24]. Thus it seems plausible that laparoscopic surgery does not confer an additional clinical advantage if we concentrate on peritoneal wound disruption. The intraperitoneal disruption is still the same no matter how access to the cavity is gained. A recent review on this topic in an optimized recovery setting has clinically confirmed that similar clinical outcomes can be reached between modalities[25]. Hence what seems to be rather important in abdominal surgery is whether the peritoneum as an entity is entered, dissected, and manipulated. This is demonstrated in clinical studies of aorta aneurysm repair, with trans-peritoneal aneurysmal repair resulting in significantly higher inflammatory response corresponding to slower clinical recovery compared to the extra-peritoneal approach where this is possible[26].

Operating on many organs such as colonic resection necessitates intraperitoneal injury and hence extra-peritoneal approach is not an option. In Part Ⅱ of this series we will focus on possible new methods to manipulate the intraperitoneal wound in order to reach improved clinical endpoints.

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