 doi:10.32114/CCI.2026.9.2.1.6

CRITICAL CARE INNOVATIONS JOURNAL

REVIEW ARTICLE

Crit. Care Innov. 2026; 9(2): 1-6

ISSN 2545-2533

Received: 11.04.2026
Accepted: 28.05.2026
First online: 29.05.2026
Published: 30.06.2026

Mechanical stress and geometry of fascial closure in critically ill patients after laparotomy

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Author Contributions (CRediT Taxonomy):
Conceptualization - **A**
Data Curation - **B**
Formal Analysis - **C**
Funding Acquisition - **D**
Investigation - **E**
Methodology - **F**
Project Administration - **G**
Resources - **H**
Software - **I**
Supervision - **J**
Validation - **K**
Visualization - **L**
Writing (Draft Preparation) - **M**
Writing (Review & Editing) – **N**
Approved the final version - **O**

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ABSTRACT

Wound dehiscence after laparotomy remains a severe and potentially life-threatening complication, particularly in critically ill patients exposed to increased intra-abdominal pressure, mechanical ventilation, and repetitive mechanical stress. Traditionally, fascial failure has been attributed primarily to impaired biological healing; however, this perspective does not fully explain the dynamic mechanical environment of the abdominal wall in the postoperative period. This review proposes a unified mechanical and geometric framework for understanding fascial failure after laparotomy. It emphasizes that successful closure depends not only on tissue quality but also on the distribution of forces and the geometry of the fascial defect. Intraoperative techniques such as progressive suturing and central force redistribution partially address this problem by modifying defect geometry and reducing peak tension. However, these strategies do not extend into the postoperative phase, where critically ill patients are exposed to repeated dynamic loading. This mismatch between tissue strength and mechanical stress may lead to progressive suture failure and wound dehiscence. Understanding fascial closure as a dynamic system influenced by both mechanical forces and geometric configuration may provide new insights into the prevention of postoperative complications and guide the development of future therapeutic strategies.

KEY WORDS: Laparotomy, wound dehiscence, fascial stress, geometry of closure, mechanical loading.

INTRODUCTION

Laparotomy remains a fundamental surgical approach in both elective and emergency settings and is associated with a substantial risk of postoperative complications, including wound dehiscence [1–5]. Despite advances in surgical technique and perioperative care, the incidence of fascial failure has not been completely eliminated. Traditionally, wound dehiscence has been interpreted primarily as a consequence of impaired biological healing. Factors such as infection, malnutrition, and systemic inflammation are well recognized contributors to poor tissue integrity [5]. However, this biological perspective alone does not fully explain the occurrence of fascial failure, particularly in patients with otherwise adequate healing potential. Despite this, these mechanical aspects are rarely addressed explicitly in routine postoperative care.

An alternative perspective emphasizes the role of mechanical stress acting on the healing abdominal wall. Following laparotomy, the fascial closure is exposed to a complex and dynamic mechanical environment. In critically ill patients, this environment is further intensified by mechanical ventilation, repeated coughing, and fluctuations in intra-abdominal pressure [6–9]. Importantly, fascial closure is not a static event but a dynamic process in which tissue strength evolves over time while being continuously challenged by mechanical forces. The outcome depends not only on the magnitude of these forces but also on their distribution and geometric configuration. Recent advances in surgical techniques suggest that modifying defect geometry and redistributing forces may be as important as reducing their magnitude [10].

LITERATURE REVIEW

Mechanical Environment in Critical Illness

The abdominal wall in critically ill patients is subjected to a highly dynamic and often extreme mechanical environment. Unlike elective surgical recovery, critically ill patients experience repeated fluctuations in intra-abdominal pressure driven by respiratory support, coughing, and systemic instability [6-9]. Mechanical ventilation plays a key role by increasing intra-thoracic pressure, which is transmitted to the abdominal cavity. This results in sustained elevation of intra-abdominal pressure and increased tension across the abdominal wall [7,8]. In everyday clinical practice, these forces are difficult to quantify, yet their impact is clearly observable.

Coughing generates transient but substantial peaks in pressure, often exceeding baseline physiological loads. Repeated coughing episodes result in cyclic loading, which may promote progressive weakening of the suture-tissue interface [7,8]. Mobilization further contributes to mechanical stress through activation of abdominal musculature and changes in body position. These forces are not static but dynamic and repetitive, creating conditions that favor fatigue failure of both biological tissues and suture material [5]. This dynamic environment suggests that fascial closure should be evaluated not under static conditions but as a structure subjected to continuous and variable loading.

Biology Versus Mechanics of Fascial Healing

Fascial healing is a complex biological process involving inflammation, collagen deposition, and remodeling. However, during the early postoperative period, the mechanical strength of the wound is primarily determined by the integrity of the suture line rather than newly formed tissue [5]. Although the exact thresholds at which mechanical stress leads to failure remain unclear, the clinical relevance of this interaction is difficult to ignore.

Mechanical stress directly influences this process. Excessive tension may impair perfusion at the wound edges, leading to ischemia and delayed healing [11,12]. Conversely, insufficient mechanical stability may prevent effective tissue integration. In critically ill patients, the balance between biological healing and mechanical stress is often disrupted. Systemic factors such as sepsis and malnutrition reduce tissue strength, while mechanical forces are simultaneously increased [6–9]. This interaction highlights that fascial healing cannot be understood without considering both biological and mechanical components.

Geometry of Fascial Closure

An often underappreciated factor in fascial closure is geometry. While force magnitude is important, the distribution of force and the spatial configuration of the defect play a critical role in determining mechanical stress [10]. In lateral abdominal wall defects, unfavorable force vectors and absence of a central anchoring structure lead to inefficient load distribution and localized stress concentration [1,2].

Modifying the geometry of the defect can significantly reduce effective tension. Reduction of the angle between fascial edges and shortening of the distance between them allows for more efficient load transfer and lower peak stress [10]. This concept supports the idea of geometry-driven closure, in which optimization of spatial configuration is central to reducing mechanical stress.

Intraoperative Control of Mechanical Stress

Modern surgical techniques aim to address mechanical stress through progressive approximation and redistribution of forces. The Laparoscopic Suitcase Technique provides a structured approach to central tension redistribution, allowing gradual modification of defect geometry and controlled fascial closure. By reducing the angle between fascial edges and redistributing forces centrally, this technique transforms closure from a high-tension maneuver into a stepwise process [10]. Similarly, retromuscular reconstruction techniques aim to achieve tension-free closure by restoring anatomical planes and distributing forces across a broader surface area [2,13]. These approaches demonstrate that intraoperative control of mechanical stress is achievable and can significantly influence surgical outcomes.

The Postoperative Loss of Control

Despite advances in intraoperative techniques, mechanical control is often lost in the postoperative period. Following surgery, the abdominal wall is exposed to uncontrolled forces generated by ventilation, coughing, and mobilization [6-9].

This represents a critical gap in current surgical practice. While intraoperative techniques optimize initial closure conditions, they do not address ongoing mechanical stress during recovery. In critically ill patients, this mismatch between tissue strength and applied stress may lead to progressive suture failure and eventual wound dehiscence [5,8]. It is therefore possible that even technically correct closure may fail if postoperative mechanical conditions are not adequately controlled.

Fascial Closure as a Dynamic System

Fascial closure should be considered a dynamic system rather than a static endpoint. In this system, tissue strength evolves over time, while mechanical forces continue to act on the closure. Failure occurs when applied stress exceeds tissue strength at any point during healing [5,8]. Geometry plays a central role in this relationship by influencing how forces are distributed. Favorable geometric configurations reduce peak stress even if total force remains unchanged. This perspective highlights the importance of continuous stress control rather than focusing solely on intraoperative closure.

External Interventions and Load Modulation

Various strategies may be used to modulate mechanical stress in the postoperative period. These include optimization of ventilatory parameters, adequate pain control, and structured mobilization [6-9,14]. External abdominal support represents one potential method of reducing excessive abdominal wall expansion and peak stress [15-19]. However, its effectiveness depends on mechanical properties and appropriate application [14-18, 20].Importantly, such interventions should be viewed as part of a broader strategy aimed at controlling mechanical stress rather than as isolated solutions.

Clinical Implications for Critical Care

Recognition of mechanical stress as a central factor in fascial failure has important implications for critical care practice. Patients at high risk of wound dehiscence may benefit from targeted strategies aimed at reducing mechanical stress, including optimization of intra-abdominal pressure, careful ventilatory management, and appropriate use of external support [21-24]. Integration of these principles into postoperative care may improve outcomes and reduce complication rates. Future research should focus on quantifying mechanical forces acting on the abdominal wall and developing methods to monitor and control these forces in real time [5,10]. Advances in biomechanical modeling, sensor technology, and personalized medical devices may enable more precise management of fascial stress.

CONCLUSIONS

Wound dehiscence after laparotomy should not be viewed solely as a failure of biological healing but as a consequence of uncontrolled mechanical stress acting on the healing fascia. Integration of mechanical forces, tissue biology, and geometric configuration provides a comprehensive framework for understanding fascial closure. Extending stress control beyond the operating room into the postoperative period may represent a key step in preventing this serious complication, particularly in critically ill patients.

SUPPLEMENTARY INFORMATION

Funding: *No fund was received related to this study.*
Institutional Review Statement: *The study was conducted according to the guidelines of the Declaration of Helsinki.*
Informed Consent Statement: *Not applicable.*
Data Availability Statement: *The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.*
Conflicts of Interest: *KM has received payments for organizing workshops and lectures for Medtronic, Olympus, TZMO, BBraun, Corza Medical and BD (Bard) and conducting educational activities. MR declare that she has no conflict of interest.*

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