DOI:https://doi.org/10.53350/pjmhs20231710218

ORIGINAL ARTICLE

Influence of Intraoperative Hemodynamic Stability on Wound Dehiscence in Patients Receiving General Anesthesia for Major Abdominal Surgery

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ABSTRACT

Background: Wound dehiscence, the separation of a surgical incision at the fascial level, remains a serious complication following major abdominal operations. Inadequate tissue perfusion due to intraoperative hemodynamic instability may impair collagen synthesis and increase the risk of fascial separation, yet prospective data in this setting are limited.

Objective: To determine whether intraoperative deviations in mean arterial pressure (MAP) and heart rate (HR) beyond 20 % of baseline values are associated with higher rates of wound dehiscence in patients undergoing elective major abdominal surgery under general anesthesia.

Methods: This prospective observational study enrolled 100 patients aged 18–65 years undergoing elective major abdominal surgery at Dr. Ruth K.M. Pfau Civil Hospital Karachi and Bolan Medical Complex Hospital Quetta from January 2022 to January 2023. Hemodynamic stability was defined as maintenance of MAP and HR within ± 20 % of baseline throughout the procedure. Episodes exceeding these thresholds for more than ten consecutive minutes despite initial corrective measures (fluid bolus, anesthetic adjustment, vasoactive support) were classified as "intraoperative hemodynamic instability." Patients were monitored for 30 days postoperatively for wound dehiscence, defined as fascial separation requiring manual or surgical intervention. Data were analyzed using SPSS version 25, with chi-square tests for categorical comparisons and multivariate logistic regression to identify independent predictors of dehiscence ($p < 0.05$ significant).

Results: Sixty-five patients (65 %) maintained intraoperative hemodynamic stability, while 35 (35 %) experienced instability. Overall wound dehiscence occurred in 10/100 (10 %) patients: 2/65 (3.1 %) in the stable group versus 8/35 (22.9 %) in the unstable group ($p < 0.001$). Multivariate analysis identified intraoperative hemodynamic instability as an independent predictor of dehiscence (odds ratio = 4.5; 95 % CI: 1.6–12.6; $p = 0.003$), after adjustment for age, BMI, diabetes, and duration of surgery.

Conclusion: Intraoperative hemodynamic instability defined as sustained deviations in MAP and HR beyond 20 % of baseline significantly increases the risk of postoperative wound dehiscence in major abdominal surgery. Vigilant hemodynamic management may reduce this complication.

Keywords: Wound dehiscence; intraoperative hemodynamic instability; major abdominal surgery; general anesthesia; fascial separation.

INTRODUCTION

Wound dehiscence, defined as the partial or complete separation of a surgical incision at the fascial level, represents a serious postoperative complication in major abdominal surgery. Its consequences extend beyond mere discomfort, frequently resulting in prolonged hospitalization, increased need for reoperation, and elevated risk of sepsis and mortality[1]. Reported incidence rates vary from approximately 2 % to over 10 %, with higher figures observed in patients who present with risk factors such as malnutrition, obesity, diabetes mellitus, or corticosteroid use. The multifactorial nature of wound dehiscence underscores the importance of examining both patient-related and perioperative variables that may compromise wound integrity[2].

Among perioperative factors, intraoperative hemodynamic stability occupies a pivotal position because tissue perfusion and oxygen delivery are fundamental to each phase of wound healing. During the initial inflammatory stage, adequate blood flow ensures delivery of neutrophils and macrophages to the wound site, facilitating bacterial clearance and debris removal[3]. Subsequently, fibroblast proliferation and collagen deposition during the proliferative phase depend on uninterrupted oxygen and nutrient supply; even brief episodes of hypotension may induce local hypoxia, impairing collagen synthesis and delaying wound tensile strength development[4]. In contrast, excessive tachycardia can elevate myocardial oxygen demand and trigger relative ischemia, further compromising microcirculatory blood flow in the abdominal wall. Experimental models have demonstrated that transient intraoperative hypotension reduces capillary perfusion at the incision margins and weakens fascial tensile strength, creating a substrate for mechanical failure[5].

Major abdominal operations such as colectomy, gastrectomy, hepatectomy, and pancreatectomy often involve prolonged operative times, considerable fluid shifts, and substantial anesthetic requirements. Surgical manipulation itself can provoke sympathetic responses that cause surges in heart rate and blood pressure, whereas inhalational anesthetics may induce vasodilation and myocardial depression, resulting in hypotensive episodes if not promptly managed[6]. Although continuous arterial pressure monitoring and corrective measures (e.g., fluid boluses, vasoactive agents) are standard practice, maintaining intraoperative mean arterial pressure and heart rate within narrow desired thresholds remains challenging. Retrospective analyses have suggested an association between prolonged intraoperative hypotension and increased rates of surgical site infections, but the specific relationship between hemodynamic fluctuations and wound dehiscence has not been systematically evaluated in a prospective manner[7, 8].

The present study was aimed to address this knowledge gap by investigating whether intraoperative deviations in mean arterial pressure and heart rate defined as excursions beyond 20 % of baseline values lasting more than ten consecutive minutes correlate with the incidence of postoperative wound dehiscence in patients undergoing elective major abdominal surgery under general anesthesia. By identifying the extent to which hemodynamic instability compromises wound healing, perioperative care teams may refine anesthetic management strategies such as goal-directed fluid therapy and proactive vasoactive support to preserve tissue perfusion, reduce the

Received on 23-04-2023
Accepted on 26-08-2023

likelihood of fascial separation, and ultimately improve surgical outcomes[9].

MATERIALS AND METHODS

A prospective observational study was undertaken at two tertiary care centers: Dr. Ruth K.M. Pfau Civil Hospital Karachi and Bolan Medical Complex Hospital Quetta. The investigation spanned from January 1, 2022, to January 31, 2023. Ethical approval was secured from the institutional review boards of both hospitals, and all participants provided written informed consent prior to inclusion.

Patients aged 18–65 years who were scheduled for elective major abdominal surgery under standardized general anesthesia protocols were screened for eligibility. Major abdominal surgery was defined as any operation requiring a midline laparotomy or equivalent extensive incision with an anticipated duration exceeding two hours (for example, colectomy, gastrectomy, hepatectomy, or pancreatectomy). A total of 100 consecutive patients meeting these criteria were enrolled: 55 from Dr. Ruth K.M. Pfau Civil Hospital Karachi and 45 from Bolan Medical Complex Hospital Quetta.

Inclusion criteria comprised American Society of Anesthesiologists (ASA) physical status class I–III and body mass index (BMI) ≤ 40 kg/m². Exclusion criteria were emergency surgery, ASA class IV or V, connective tissue disorders (such as Ehlers–Danlos syndrome), chronic immunosuppression (including HIV infection or long-term corticosteroid therapy), prior abdominal radiation, coagulation abnormalities (international normalized ratio > 1.5 or platelet count < 100 × 10⁹/L), and severe preexisting organ dysfunction (for instance, decompensated heart failure or end-stage renal disease). During the preoperative assessment, demographic details (age, sex, BMI), comorbidities (hypertension, diabetes mellitus), and baseline laboratory values (hemoglobin, serum albumin) were recorded.

Anesthetic management was standardized across both centers. Premedication consisted of midazolam 0.05 mg/kg administered intravenously. Induction was achieved with propofol (2 mg/kg IV) and fentanyl (2 µg/kg IV), followed by rocuronium (0.6 mg/kg IV) to facilitate tracheal intubation. Maintenance anesthesia employed sevoflurane (1–2 vol %) in an oxygen–air mixture (FiO_2 = 0.5), supplemented by intermittent fentanyl boluses as needed. Volume-controlled ventilation targeted tidal volumes of 6–8 mL/kg and end-tidal carbon dioxide of 35–40 mm Hg. Immediately following induction, an arterial line was placed in the radial artery for continuous mean arterial pressure (MAP) monitoring; heart rate (HR) was recorded via continuous electrocardiography. Baseline MAP and HR were documented after induction and before the first skin incision.

Intraoperative fluid management followed a goal-directed strategy when possible, using dynamic indices of volume responsiveness; if these indices were unavailable, a fixed-rate crystalloid infusion of 6 mL/kg/hour was administered. Packed red blood cell transfusion was indicated for hemoglobin levels below 8 g/dL or for persistent hemodynamic instability despite adequate volume resuscitation. Vasoactive agents phenylephrine or ephedrine to treat hypotension, and esmolol to manage tachycardia were administered at the discretion of the attending anesthesiologist. Hemodynamic stability was defined as maintenance of MAP and HR within ± 20 % of each patient's baseline values throughout the surgical procedure. Any occurrence in which MAP or HR deviated beyond these limits for more than ten consecutive minutes, despite initial corrective measures (e.g., fluid bolus or adjustment of anesthetic depth), was categorized as "intraoperative hemodynamic instability."

All surgical procedures were performed by attending surgeons with at least five years of experience in major abdominal operations. Prophylactic antibiotics (cefuroxime 1.5 g IV and metronidazole 500 mg IV) were administered within 60 minutes before skin incision. Fascia closure was standardized: continuous suturing with polydioxanone 1-0, subcutaneous closure using interrupted polyglactin 910 2-0, and skin approximation with sterile staples. Subcutaneous drains were placed according to the surgeon's clinical judgment. Postoperative wound dressings were changed on the second postoperative day, and wounds were inspected daily by surgical teams until discharge.

Following surgery, patients were monitored for 30 days. Wound assessments occurred daily during hospitalization and at scheduled outpatient visits on postoperative days 7, 14, and 30. Wound dehiscence was defined as separation of the fascial layer requiring either manual reapproximation or surgical intervention, confirmed clinically by the attending surgeon. Secondary outcomes included superficial and deep surgical site infections (SSI), classified according to Centers for Disease Control and Prevention (CDC) criteria, length of hospital stay, and 30-day readmission rates.

All data were entered into a secure database and analyzed using SPSS version 25 (IBM Corp., Armonk, NY, USA). Continuous variables were evaluated for normality using the Shapiro–Wilk test and are reported as mean ± standard deviation or median (interquartile range) as appropriate; comparisons between groups utilized Student's t-test or Mann–Whitney U test. Categorical variables are presented as frequencies and percentages, with group comparisons made via chi-square or Fisher's exact test. Variables yielding a p-value < 0.10 in univariate analysis were included in a multivariate logistic regression model to identify independent predictors of wound dehiscence. Statistical significance was set at $p < 0.05$.

RESULTS

A total of 100 patients were enrolled, with 65 (65.0 %) maintaining intraoperative hemodynamic stability (stable group) and 35 (35.0 %) experiencing significant hemodynamic fluctuations (unstable group). Baseline demographic and perioperative characteristics were comparable between the two cohorts (Table 1). The mean age in the stable group was 48 ± 10 years versus 50 ± 11 years in the unstable group (p = 0.27). Gender distribution did not differ significantly (male: 36/65 [55.4 %] stable vs. 20/35 [57.1 %] unstable; p = 0.85). Body mass index (BMI) averaged 27.2 ± 3.4 kg/m² in the stable group versus 28.1 ± 3.8 kg/m² in the unstable group (p = 0.19). The prevalence of diabetes mellitus was 15/65 (23.1 %) in the stable cohort and 10/35 (28.6 %) in the unstable cohort (p = 0.53), while hypertension was observed in 18/65 (27.7 %) versus 12/35 (34.3 %) patients, respectively (p = 0.48). Distribution of ASA classification was similar: ASA II comprised 46/65 (70.8 %) stable and 23/35 (65.7 %) unstable (p = 0.60). Mean duration of surgery was 210 ± 45 minutes in the stable group and 225 ± 50 minutes in the unstable group (p = 0.12). Estimated intraoperative blood loss had a median of 300 mL (IQR: 200–450) for stable patients compared to 350 mL (IQR: 250–500) for unstable patients (p = 0.11). Packed red blood cell transfusion was required in 7/65 (10.8 %) stable patients versus 6/35 (17.1 %) unstable patients (p = 0.35).

Table 1: Demographic and Perioperative Characteristics

Characteristic	Stable (n = 65)	Unstable (n = 35)	p-Value
Age (years), mean ± SD	48 ± 10	50 ± 11	0.27
Gender, n (%)			0.85
Male	36 (55.4)	20 (57.1)	
Female	29 (44.6)	15 (42.9)	
BMI (kg/m²), mean ± SD	27.2 ± 3.4	28.1 ± 3.8	0.19
Diabetes Mellitus, n (%)	15 (23.1)	10 (28.6)	0.53
Hypertension, n (%)	18 (27.7)	12 (34.3)	0.48
ASA Classification, n (%)			0.60
II (mild systemic disease)	46 (70.8)	23 (65.7)	
III (severe systemic disease)	19 (29.2)	12 (34.3)	
Duration of Surgery (minutes), mean ± SD	210 ± 45	225 ± 50	0.12
Estimated Blood Loss (mL), median (IQR)	300 (200–450)	350 (250–500)	0.11
Intraoperative Blood Transfusion, n (%)	7 (10.8)	6 (17.1)	0.35

Intraoperative hemodynamic data for the unstable cohort indicated a mean of 3.2 ± 1.1 episodes during which mean arterial pressure (MAP) or heart rate (HR) deviated beyond ± 20 % of baseline values for more than ten consecutive minutes. The lowest recorded MAP in the unstable group was 58 ± 7 mm Hg compared to a baseline of 78 ± 9 mm Hg ($p < 0.001$); the highest recorded HR during instability episodes was 110 ± 12 beats/min versus baseline 78 ± 10 beats/min ($p < 0.001$). Vasoactive agents were administered to 29/35 (82.9 %) patients in the unstable group, whereas only 10/65 (15.4 %) of the stable group required vasoactive support for transient fluctuations ($p < 0.001$).

Within the 30-day postoperative period, wound dehiscence occurred in 10/100 (10.0 %) patients. Among those, 2/65 (3.1 %) belonged to the stable cohort, and 8/35 (22.9 %) belonged to the unstable cohort ($p < 0.001$). Superficial surgical site infections (SSI) were identified in 7/65 (10.8 %) stable patients and 9/35 (25.7 %) unstable patients ($p = 0.03$). Deep SSI occurred in 2/65 (3.1 %) stable patients and 4/35 (11.4 %) unstable patients ($p = 0.07$). The mean length of hospital stay was significantly shorter for stable patients (7 ± 2 days) compared to unstable patients (10 ± 3 days; $p < 0.001$). Thirty-day readmission rates did not differ significantly (stable: 3/65 [4.6 %] vs. unstable: 5/35 [14.3 %]; $p = 0.08$) (Table 2).

Table 2: Incidence of Postoperative Outcomes

Outcome	Stable (n = 65), n (%)	Unstable (n = 35), n (%)	p-Value
Wound Dehiscence	2 (3.1)	8 (22.9)	< 0.001
Superficial SSI	7 (10.8)	9 (25.7)	0.03
Deep SSI	2 (3.1)	4 (11.4)	0.07
Length of Stay (days), mean ± SD	7 ± 2	10 ± 3	< 0.001
30-Day Readmission, n (%)	3 (4.6)	5 (14.3)	0.08

Univariate analysis identified intraoperative hemodynamic instability, superficial SSI, deep SSI, and prolonged hospital stay (≥ 10 days) as candidate predictors of wound dehiscence ($p < 0.10$). These variables were entered into a multivariate logistic regression model (Table 3). After adjusting for age, BMI, diabetes mellitus, and duration of surgery, intraoperative hemodynamic instability remained an independent predictor of wound dehiscence (odds ratio $^2 = 4.5$; 95 % confidence interval [CI]: 1.6–12.6; $p = 0.003$). Deep SSI also demonstrated an independent association with dehiscence (OR = 3.2; 95 % CI: 1.0–10.4; $p = 0.04$). Neither diabetes mellitus nor BMI was independently associated with wound dehiscence in the adjusted model.

Table 3: Multivariate Logistic Regression of Risk Factors for Wound Dehiscence

Variable	OR	95 % CI	p-Value
Hemodynamic Instability (Yes vs. No)	4.5	1.6–12.6	0.003
Deep SSI (Yes vs. No)	3.2	1.0–10.4	0.04
Diabetes Mellitus (Yes vs. No)	1.8	0.6–5.7	0.29
BMI (per kg/m² increase)	1.1	0.9–1.3	0.21
Duration of Surgery (> 240 min vs. ≤ 240 min)	1.3	0.5–3.6	0.54

Finally the patients with intraoperative hemodynamic instability exhibited a significantly higher incidence of wound dehiscence (22.9 % vs. 3.1 %; $p < 0.001$), longer hospital stays (10 ± 3 days vs. 7 ± 2 days; $p < 0.001$), and increased rates of superficial SSI (25.7 % vs. 10.8 %; $p = 0.03$) compared to those who maintained stable hemodynamics. In multivariate analysis, intraoperative hemodynamic instability and deep SSI emerged as independent predictors of wound dehiscence.

DISCUSSION

The present study demonstrates a clear association between intraoperative hemodynamic instability and an increased risk of postoperative wound dehiscence in patients undergoing elective major abdominal surgery under general anesthesia[10]. Patients who experienced sustained deviations of mean arterial pressure (MAP) and heart rate (HR) beyond 20 % of baseline values for more than ten consecutive minutes exhibited a significantly higher incidence of wound dehiscence (22.9 %) compared to those who maintained stable intraoperative hemodynamics (3.1 %). This fourfold increase underscores the critical importance of tight hemodynamic control during prolonged abdominal procedures. Hemodynamic instability likely compromises wound healing through two principal mechanisms: reduced tissue perfusion and a heightened inflammatory milieu[11].

First, adequate tissue perfusion is fundamental at every stage of wound healing. Early phases demand sufficient oxygen and nutrient delivery to support neutrophil and macrophage infiltration, bacterial clearance, and the initiation of fibroblast proliferation. In our unstable cohort, episodes of hypotension reflected by a mean lowest MAP of 58 ± 7 mm Hg versus the baseline of 78 ± 9 mm Hg would have reduced capillary perfusion at the incision site, creating localized hypoxia and nutrient deprivation[12]. Animal studies have shown that even short durations of intraoperative hypotension impair collagen synthesis and weaken fascial tensile strength, thereby predisposing to mechanical failure of the suture line. In addition, sustained tachycardia (mean peak HR of 110 ± 12 beats/min) may further compromise microcirculatory blood flow by increasing myocardial oxygen demand and generating relative ischemia[13]. Taken together, the combination of intraoperative hypotension and tachycardia in the unstable cohort likely led to suboptimal wound edge perfusion, delaying collagen deposition and reducing the early tensile strength that is essential to resist intra-abdominal pressure during the immediate postoperative period[14].

Second, hemodynamic fluctuations can amplify the systemic stress response and enhance proinflammatory cytokine release. Major abdominal surgeries inherently induce a systemic inflammatory response through extensive tissue manipulation. When compounded by perfusion deficits, ischemia–reperfusion phenomena can augment oxidative stress and inflammatory mediator production[15]. This exaggerated inflammatory state may disrupt the balance between matrix metalloproteinases and tissue inhibitors, promoting premature collagen degradation and impairing the remodeling phase of wound healing. In our study, the unstable cohort exhibited a higher incidence of both superficial (25.7 % vs. 10.8 %) and deep (11.4 % vs. 3.1 %) surgical site infections. Suboptimal perfusion impedes neutrophil and lymphocyte trafficking, undermining the local immune response and facilitating bacterial colonization. Consequently, infection further compromises wound strength and contributes to dehiscence. Indeed, multivariate analysis revealed that deep surgical site infection independently increased the odds of wound dehiscence by more than threefold, emphasizing the interplay between hemodynamic instability, tissue oxygenation, and infection risk[16].

Our findings align with retrospective observations that intraoperative hypotension correlates with higher rates of surgical site complications, but our prospective design and standardized definitions of hemodynamic instability strengthen the evidence base. Unlike earlier studies that loosely defined hypotension thresholds or lacked uniform monitoring, we employed continuous invasive arterial monitoring and predefined a ± 20 % deviation from baseline as the cut-off for instability[17]. By capturing episodes lasting more than ten consecutive minutes, we identified clinically meaningful perturbations that persisted despite standard corrective measures underscoring the challenge of maintaining stable perfusion in complex abdominal cases. Moreover, by focusing specifically on fascial dehiscence rather than general wound complications, we have highlighted a discrete, high-morbidity outcome that directly correlates with intraoperative management[18].

These observations carry important implications for perioperative care. First, anesthetic teams should target individualized hemodynamic goals, avoiding rigid reliance on absolute MAP thresholds. Rather than applying a one-size-fits-all

MAP of 65–70 mm Hg, clinicians should consider each patient's baseline values and comorbidities, titrating vasoactive agents to maintain perfusion within 20 % of baseline. Second, goal-directed fluid therapy protocols utilizing dynamic indices of fluid responsiveness may reduce both hypotensive episodes and excessive fluid administration[18]. Excessive crystalloid infusion can lead to tissue edema, further impairing oxygen diffusion at the wound edge. Conversely, restrictive fluid strategies without dynamic assessment may prompt hypotension. By continuously assessing stroke volume variation or pulse pressure variation (where available), anesthesiologists can optimize intravascular volume, mitigate hemodynamic swings, and support microcirculatory flow to the surgical site[19].

Third, proactive use of short-acting vasopressors or inotropes may stabilize MAP without compromising cardiac output. In our unstable cohort, 82.9 % required vasoactive support, indicating that standardized anesthetic regimens alone were insufficient to maintain hemodynamic targets. Early administration of phenylephrine for isolated hypotension, combined with esmolol for sympathetic surges, can preserve perfusion while avoiding excessive tachycardia. Importantly, incremental titration of these agents, rather than bolus dosing, may prevent overshooting target ranges and reduce the risk of rebound fluctuations. Integration of closed-loop vasopressor delivery systems where available could further standardize blood pressure control and minimize human variability[20].

In addition to anesthetic considerations, surgical technique and timing warrant attention. Although all attending surgeons in this study had at least five years of experience with extensive abdominal procedures, procedural factors such as duration of pneumoperitoneum (in laparoscopic cases) or prolonged retraction during open exposure may predispose to visceral hypoperfusion and reflex bradycardia or tachycardia[21]. Minimizing unnecessary retraction time, coordinating with anesthesia to optimize depth of neuromuscular blockade, and ensuring efficient surgical workflow can reduce sympathetic surges that trigger hemodynamic swings. Collaboration between surgeon and anesthesiologist in anticipating critical phases such as manipulation of major vessels or bowel handling allows for preemptive adjustment of anesthetic depth and vasoactive dosing, thereby maintaining hemodynamic equilibrium[22].

Despite these insights, the study has limitations. As a prospective observational investigation, causality cannot be definitively established. Although multivariate analysis adjusted for key confounders such as age, body mass index, diabetes, and duration of surgery unmeasured variables like intra-abdominal pressure magnitude, surgeon–patient anthropometric mismatch, or individual variations in inflammatory response may still influence outcomes[23]. The sample size of 100 patients, while adequate to detect a significant association, limits the granularity of subgroup analyses (for example, comparing specific procedures or stratifying by diabetic control). Additionally, the threshold of ± 20 % deviation from baseline for more than ten minutes, though clinically reasonable, remains somewhat arbitrary; alternative definitions of instability may yield different associations. Finally, this study was conducted at two tertiary hospitals in Karachi and Quetta, which may limit generalizability to institutions with differing patient populations, resource availability, or perioperative protocols[23].

Future research should focus on randomized trials that test specific hemodynamic management algorithms aimed at minimizing wound dehiscence. For instance, a trial comparing standard anesthetic protocols against goal-directed therapy protocols with predefined MAP and HR targets tied to baseline hemodynamics could clarify the causal impact of hemodynamic optimization[24]. Likewise, studies employing microcirculatory monitoring tools such as near-infrared spectroscopy or laser Doppler flowmetry during surgery may elucidate real-time perfusion dynamics at the incision margins, bridging the gap between systemic hemodynamics and local tissue perfusion. Investigating adjunctive postoperative interventions such as negative-pressure wound therapy in patients with intraoperative instability could further reduce dehiscence risk. Additionally, exploring the molecular milieu of ischemia–reperfusion injury in the wound edge through tissue biopsies or biomarker assays might identify therapeutic targets to enhance collagen synthesis under compromised perfusion[25].

CONCLUSION

In summary, intraoperative hemodynamic instability defined as sustained deviations of mean arterial pressure and heart rate beyond 20 % of baseline values is strongly associated with an increased risk of postoperative wound dehiscence in patients receiving general anesthesia for major abdominal surgery. This association persists even after adjusting for comorbidities and surgical duration. The findings highlight the need for individualized perioperative hemodynamic management that prioritizes maintenance of blood pressure and heart rate within narrow ranges relative to each patient's baseline. By implementing goal-directed fluid therapy, proactive vasoactive support, and close coordination between surgical and anesthetic teams, clinicians can optimize tissue perfusion, mitigate the risk of fascial separation, and improve overall surgical outcomes. Future randomized studies are warranted to confirm whether targeted intraoperative hemodynamic protocols can causally reduce wound dehiscence and enhance postoperative recovery.

Funding: This study did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflicts of Interest: The authors declare no competing interests.

Ethical Approval: Ethical clearance was obtained from the appropriate Institutional Review Board before initiating the study.

Informed Consent: Written informed consent was obtained from all study participants.

Authors' Contributions: All authors contributed equally in the completion of current study.

Acknowledgements: The authors thank the participants and institutional staff for their cooperation and support throughout the research.

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This article may be cited as: Jamal SN, Noreen N, Laghari QA, Aslam RS, Ramani RK, Iqbal M: Influence of Intraoperative Hemodynamic Stability on Wound Dehiscence in Patients Receiving General Anesthesia for Major Abdominal Surgery. Pak J Med Health Sci, 2023;17(10): 218-222.