

Physiological Impact of Carbon Dioxide Pneumoperitoneum on Cardiovascular Function and Respiratory Mechanics in Advanced Laparoscopic Surgery

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ABSTRACT

Carbon dioxide pneumoperitoneum is an essential component of advanced laparoscopic surgery, but the increase in intra-abdominal pressure required to create the surgical workspace produces important hemodynamic and respiratory alterations. This study analyzed the principal physiological effects of pneumoperitoneum on cardiovascular performance and respiratory mechanics, considering the influence of insufflation pressure, patient positioning, obesity, duration of surgery, mechanical ventilation, and cardiopulmonary reserve. An analytical review of relevant physiological, anesthesiological, and surgical evidence was performed, integrating studies focused on venous return, preload, afterload, systemic and pulmonary vascular resistance, arterial pressure, stroke volume, cardiac output, respiratory compliance, airway pressures, diaphragmatic displacement, gas exchange, and carbon dioxide elimination. The findings showed that pneumoperitoneum can decrease cardiac output while simultaneously increasing mean arterial pressure and systemic vascular resistance, particularly when higher insufflation pressures are used. Respiratory effects were characterized by cephalad diaphragmatic displacement, reduced respiratory system compliance, increased peak and plateau airway pressures, and impaired regional ventilation. These changes were intensified by steep Trendelenburg positioning, prolonged procedures, and obesity. The evidence also demonstrated that arterial pressure alone may not accurately reflect circulatory performance and that elevated airway pressures should be interpreted in conjunction with compliance and thoracoabdominal mechanics. Overall, pneumoperitoneum represents a dynamic cardiopulmonary condition rather than an isolated surgical maneuver. Its physiological impact depends on the interaction between intra-abdominal pressure, ventilation, positioning, body habitus, and baseline cardiopulmonary function. Individualized insufflation pressures, respiratory settings, PEEP, hemodynamic monitoring, and perioperative management may reduce physiological stress and improve safety during complex laparoscopic and robotic-assisted procedures.

KEYWORDS

pneumoperitoneum, laparoscopic surgery, hemodynamics, respiratory mechanics, intra-abdominal pressure, cardiac output, systemic vascular resistance, pulmonary compliance, airway pressure, Trendelenburg position, carbon dioxide, robotic surgery, mechanical ventilation, PEEP, perioperative physiology

INTRODUCTION

Laparoscopic surgery has transformed modern surgical practice by reducing the magnitude of abdominal wall trauma, postoperative pain, hospital stay, wound-related complications, and recovery time when compared with conventional open procedures. Its progressive expansion into increasingly complex operations—including advanced gastrointestinal, bariatric, colorectal, gynecologic, urologic, hepatobiliary, and robotic-assisted procedures—has also increased the physiological demands imposed on patients during surgery. Unlike open surgery, most laparoscopic procedures require the creation and maintenance of a pneumoperitoneum, usually with carbon dioxide (CO₂), to obtain adequate visualization and working space within the abdominal cavity. Although this intervention is technically essential, the resulting elevation of intra-abdominal pressure produces substantial and sometimes clinically relevant alterations in cardiovascular and respiratory physiology [1]–[3].

The physiological consequences of pneumoperitoneum result from the interaction between mechanical compression of intra-abdominal structures, changes in intrathoracic pressure, neurohumoral activation, patient positioning, CO₂ absorption, anesthetic effects, baseline cardiopulmonary reserve, and the duration and magnitude of insufflation pressure [1], [2]. These mechanisms become especially relevant during advanced laparoscopic procedures, in which operative times may be prolonged and extreme positions such as steep Trendelenburg or reverse Trendelenburg are frequently required. Consequently, the physiological response to pneumoperitoneum cannot be interpreted exclusively as a simple effect of increased intra-abdominal pressure; rather, it represents a dynamic interaction among cardiovascular loading conditions, pulmonary mechanics, gas exchange, vascular resistance, and compensatory autonomic responses [3], [9], [17].

From a hemodynamic perspective, the elevation of intra-abdominal pressure generated by CO₂ insufflation modifies venous return, cardiac preload, systemic vascular resistance, arterial pressure, and cardiac output. During the initial phases of insufflation, compression of the abdominal venous reservoir may transiently displace blood toward the thoracic compartment. However, as intra-abdominal pressure increases, compression of the inferior vena cava and other major venous structures may progressively impair venous return, particularly in hypovolemic patients or when higher insufflation pressures are used [1], [3]. Simultaneously, pneumoperitoneum increases systemic vascular resistance and ventricular afterload through both mechanical and neurohumoral mechanisms. These changes can increase mean arterial pressure despite a reduction in stroke volume or cardiac output, illustrating the complexity of cardiovascular adaptation during laparoscopy [4], [6].

The cardiovascular response is further influenced by activation of the sympathetic nervous system and release of vasopressin and other vasoactive mediators. Joris *et al.* demonstrated significant hemodynamic and endocrine changes associated with laparoscopy, supporting the concept that pneumoperitoneum induces not only mechanical cardiovascular alterations but also a systemic neurohumoral response capable of increasing vascular tone [4]. This phenomenon is clinically important because increased systemic vascular resistance can substantially elevate left ventricular afterload, while increased pulmonary vascular resistance may also influence right ventricular performance. In patients with limited cardiac reserve, pulmonary hypertension, ventricular dysfunction, ischemic heart disease, or significant valvular abnormalities, these variations may become poorly tolerated even when conventional arterial blood pressure appears preserved.

Direct intraoperative assessment of cardiovascular function has confirmed that pneumoperitoneum can reduce cardiac filling and modify ventricular performance. Transesophageal echocardiographic investigations have demonstrated changes in cardiac dimensions and hemodynamic function during laparoscopic surgery, highlighting the effects of insufflation on preload and cardiac output [5]. Moreover, the pressure selected for pneumoperitoneum appears to be a major determinant of the magnitude of these alterations. Dexter *et al.* observed clinically relevant differences between high- and low-pressure capnoperitoneum, reinforcing the principle that lower insufflation pressures may reduce cardiovascular disturbance when adequate surgical exposure can still be maintained [6].

Patient position adds another level of complexity. Head-up positioning, commonly used during upper abdominal and hepatobiliary surgery, can reduce venous return through gravitational displacement of blood toward the lower extremities. When combined with pneumoperitoneum and the vasodilatory effects of general anesthesia, this reduction in preload can contribute to decreased stroke volume and cardiac output [7]. Conversely, steep Trendelenburg, frequently required in advanced pelvic and robotic surgery, promotes cephalad blood redistribution but simultaneously increases intrathoracic pressure and central venous pressure. Therefore, measurements such as central venous pressure may not accurately represent true ventricular preload under these circumstances because they are influenced by increased pleural and abdominal pressures.

These observations have led to increasing interest in individualized hemodynamic management during laparoscopic surgery. Larsen *et al.* demonstrated that pneumoperitoneum can substantially influence cardiac function and systemic hemodynamics during laparoscopic cholecystectomy [8]. In advanced surgery, the clinical significance of these effects may be amplified by prolonged operative duration, repeated changes in position, greater insufflation requirements, obesity, advanced age, and underlying cardiovascular disease. For these reasons, conventional static variables alone may be insufficient to interpret circulatory status, and physiological understanding remains essential when assessing arterial pressure, cardiac output, preload responsiveness, and the need for fluid administration or vasoactive support.

Respiratory physiology is similarly affected. The increase in intra-abdominal pressure displaces the diaphragm in a cephalad direction, reduces functional residual capacity, alters chest wall mechanics, and decreases respiratory system compliance [2], [11], [13]. These changes increase airway pressures for a given tidal volume and favor dependent lung compression, particularly in the dorsal and basal regions. The resulting reduction in aerated lung volume promotes atelectasis, deterioration of ventilation-perfusion matching, and increased risk of intraoperative hypoxemia. These consequences are particularly important during prolonged laparoscopic or robotic procedures in which both pneumoperitoneum and restrictive positioning are maintained for extended periods.

Several physiological investigations have documented the decline in respiratory compliance associated with pneumoperitoneum. Oikkonen and Tallgren identified measurable reductions in respiratory compliance during laparoscopy using continuous respiratory monitoring [12], while Obeid *et al.* demonstrated a direct relationship between increased intra-abdominal pressure and impaired pulmonary compliance [13]. Bardoczky *et al.* similarly reported significant ventilatory changes during pneumoperitoneum monitored by continuous spirometry [14]. Together, these findings support a consistent mechanical model in which abdominal insufflation increases diaphragmatic loading, reduces thoracic compliance, and increases the pressure required to ventilate the lungs.

More detailed studies of respiratory mechanics have further demonstrated that pneumoperitoneum modifies both global and regional lung behavior. Loring *et al.* showed that surgical pneumoperitoneum substantially affects respiratory mechanics in humans, reflecting alterations in lung and chest wall interactions rather than merely an increase in airway pressure [15]. Such changes are relevant because airway pressure does not necessarily correspond directly to transpulmonary pressure. During pneumoperitoneum, part of the elevated airway pressure may be required to overcome increased chest wall and abdominal elastance rather than representing excessive alveolar distending pressure. This distinction becomes important when selecting ventilatory parameters designed to minimize ventilator-associated lung stress.

CO₂ insufflation adds a metabolic component to these mechanical respiratory effects. Carbon dioxide diffuses rapidly across the peritoneal surface into the systemic circulation and must subsequently be eliminated through pulmonary ventilation. As a result, arterial partial pressure of CO₂ and end-tidal CO₂ tend to increase after insufflation. Controlled ventilation therefore generally requires adjustment of minute ventilation to maintain adequate CO₂ elimination. If ventilation is insufficient, hypercapnia may produce respiratory acidosis, sympathetic stimulation, tachycardia, increased systemic vascular resistance, and changes in pulmonary vascular tone [1], [2]. Consequently, the respiratory and cardiovascular effects of pneumoperitoneum are physiologically interconnected rather than independent phenomena.

The importance of these alterations is accentuated in patients with obesity. Obesity itself reduces functional residual capacity, increases chest wall elastance, promotes dependent atelectasis, and can impair ventilation-perfusion relationships even before pneumoperitoneum is established. Nguyen and Wolfe emphasized that the physiological impact of pneumoperitoneum in patients with morbid obesity involves significant cardiovascular, respiratory, and metabolic changes [9]. In patients undergoing laparoscopic gastric bypass, pneumoperitoneum has also been associated with deterioration in intraoperative pulmonary mechanics and gas exchange [10]. Sprung *et al.* further demonstrated that obesity, pneumoperitoneum, and body position can interact to adversely affect respiratory mechanics and oxygenation [11]. These findings are particularly relevant because advanced laparoscopic surgery is increasingly performed in obese and metabolically complex populations.

Modern robotic-assisted surgery has introduced additional physiological challenges. Robotic pelvic procedures commonly combine CO₂ pneumoperitoneum with steep Trendelenburg positioning, creating marked cephalad displacement of abdominal contents. This configuration can substantially reduce pulmonary compliance, increase airway pressures, alter regional ventilation, and promote dependent atelectasis [16]–[19]. Kim *et al.* demonstrated changes in diaphragmatic excursion and lung compliance during major laparoscopic pelvic surgery, illustrating the progressive mechanical consequences of prolonged intra-abdominal insufflation [16]. Similarly, Brandão *et al.* identified changes in global and regional respiratory mechanics during robotic-assisted laparoscopic surgery [17].

The interaction between pneumoperitoneum, patient position, and positive end-expiratory pressure has therefore become an important area of perioperative research. Shono *et al.* evaluated the distribution of ventilation during pneumoperitoneum combined with steep Trendelenburg positioning and demonstrated that PEEP can meaningfully influence regional ventilation [18]. Nevertheless, higher PEEP levels may simultaneously affect venous return and hemodynamic function. Therefore, respiratory optimization cannot be considered independently of cardiovascular physiology. The optimal intraoperative strategy requires balancing alveolar recruitment and oxygenation against the potential circulatory consequences of increased intrathoracic pressure.

Body habitus is another relevant determinant of respiratory response. Tharp *et al.* found that both patient characteristics and dynamic surgical conditions independently affect pulmonary mechanics during robotic-assisted laparoscopic

surgery [19]. This observation supports the concept that standardized ventilatory settings may not be appropriate for every patient. Individual factors such as obesity, baseline respiratory disease, abdominal compliance, surgical position, insufflation pressure, and duration of pneumoperitoneum may substantially modify the physiological response.

Contemporary approaches to mechanical ventilation during advanced laparoscopic and robotic surgery consequently emphasize lung-protective ventilation, individualized PEEP, appropriate tidal volume selection based on predicted rather than actual body weight, recruitment strategies when indicated, and careful monitoring of airway and driving pressures. Chiumello *et al.* highlighted the need for physiologically guided ventilatory strategies during robotic urological and gynecological surgery, particularly because of the combination of pneumoperitoneum and steep Trendelenburg [20]. However, ventilation strategies intended to improve pulmonary mechanics must be integrated with hemodynamic assessment because excessive PEEP or aggressive recruitment maneuvers can increase intrathoracic pressure and further reduce venous return.

Despite decades of research on pneumoperitoneum, the topic remains highly relevant because laparoscopic surgery has expanded far beyond the relatively short and uncomplicated procedures that characterized its early development. Current practice includes prolonged oncological resections, complex pelvic procedures, bariatric surgery, multi-quadrant abdominal surgery, advanced hepatobiliary operations, and robot-assisted interventions. These procedures expose patients to longer periods of elevated intra-abdominal pressure and frequently involve extreme positioning. Moreover, contemporary surgical candidates often present with obesity, advanced age, cardiovascular disease, pulmonary disease, metabolic abnormalities, or multiple comorbidities that can reduce tolerance to acute physiological disturbances.

A further reason for continued investigation is that the physiological effects of pneumoperitoneum depend on several potentially modifiable variables. Insufflation pressure, patient positioning, intravascular volume status, anesthetic technique, ventilatory strategy, PEEP, CO₂ elimination, and vasoactive management can all influence the cardiovascular and respiratory response. Understanding the relative contribution of these variables is essential for designing perioperative strategies that maintain adequate surgical conditions while minimizing physiological disturbance.

In this context, the central research question of the present study is: **How does CO₂ pneumoperitoneum modify cardiovascular and respiratory physiology during advanced laparoscopic surgery, and which perioperative factors determine the magnitude and clinical relevance of these changes?** Secondary questions address the effects of intra-abdominal pressure on venous return, ventricular loading conditions, systemic vascular resistance, cardiac output, diaphragmatic displacement, respiratory system compliance, airway pressure, gas exchange, CO₂ homeostasis, and regional ventilation. Particular attention is also given to the interaction between pneumoperitoneum and obesity, prolonged Trendelenburg positioning, insufflation pressure, and mechanical ventilation.

The study is based on the hypothesis that pneumoperitoneum produces predictable but dynamically interconnected hemodynamic and respiratory alterations whose magnitude increases with higher intra-abdominal pressure, prolonged exposure, unfavorable positioning, reduced cardiopulmonary reserve, and elevated body mass. A complementary hypothesis is that appropriate adjustment of insufflation pressure, ventilatory parameters, PEEP, patient positioning, and hemodynamic management may reduce the physiological burden associated with advanced laparoscopic procedures without necessarily compromising surgical exposure.

To address these questions, the present work adopts an analytical review approach integrating physiological, anesthesiological, and surgical evidence. Studies addressing cardiovascular function, pulmonary mechanics, gas exchange, intra-abdominal pressure, patient positioning, obesity, robotic surgery, and perioperative ventilation were considered in order to construct a comprehensive physiological interpretation of pneumoperitoneum. Foundational investigations describing the mechanisms of cardiovascular and respiratory alteration were integrated with more recent studies evaluating contemporary laparoscopic and robotic surgical conditions [1]–[3], [15]–[20]. This design allows classical physiological mechanisms to be examined in relation to current surgical practice.

The methodological approach is therefore directly aligned with the research questions. Evidence related to hemodynamic changes is analyzed through variables including arterial pressure, systemic vascular resistance, venous

return, stroke volume, and cardiac output, whereas respiratory consequences are assessed through compliance, airway pressure, diaphragmatic displacement, ventilation distribution, oxygenation, and CO₂ elimination. Additional modifiers—including intra-abdominal pressure, body position, obesity, duration of surgery, and ventilatory strategy—are incorporated to explain variability among patients and procedures.

DEVELOPMENT

Carbon dioxide pneumoperitoneum is the fundamental condition that enables most laparoscopic procedures by creating sufficient intra-abdominal working space. However, increasing intra-abdominal pressure produces physiological effects that extend beyond the surgical field. The magnitude of these effects depends primarily on insufflation pressure, duration of exposure, patient positioning, cardiovascular reserve, pulmonary mechanics, body habitus, and the interaction with general anesthesia and positive-pressure ventilation [1]–[3]. In advanced laparoscopic surgery, these factors frequently coexist and may significantly modify perioperative homeostasis.

From a **hemodynamic perspective**, pneumoperitoneum alters venous return, cardiac preload, ventricular afterload, vascular resistance, and cardiac output. At moderate pressures, abdominal compression can initially mobilize venous blood toward the thoracic compartment; however, progressive elevation of intra-abdominal pressure compresses the inferior vena cava and abdominal venous circulation, potentially reducing effective venous return [1], [3]. At the same time, systemic vascular resistance generally increases due to both mechanical vascular compression and sympathetic and neuroendocrine activation. This response may increase mean arterial pressure despite a simultaneous reduction in stroke volume or cardiac output [4], [5].

Insufflation pressure is therefore a major determinant of cardiovascular response. Studies comparing lower and higher pneumoperitoneum pressures have shown that elevated pressures tend to produce more pronounced changes in vascular resistance and cardiac performance [6]. These effects are particularly relevant in patients with hypovolemia, ventricular dysfunction, ischemic heart disease, pulmonary hypertension, or reduced physiological reserve. Patient position further modifies these responses. Reverse Trendelenburg may decrease venous return through gravitational pooling, whereas steep Trendelenburg may increase central venous pressures but does not necessarily improve effective ventricular filling because intrathoracic pressure is simultaneously elevated [7], [8].

The **respiratory effects** of pneumoperitoneum are largely related to cephalad displacement of the diaphragm caused by increased abdominal pressure. This mechanical shift reduces functional residual capacity, decreases respiratory system compliance, and increases airway pressures during controlled ventilation [11]–[15]. The reduction in aerated lung volume favors compression of dependent pulmonary regions and atelectasis, which may worsen ventilation-perfusion matching and oxygenation. These alterations are especially important during prolonged procedures in which pneumoperitoneum is maintained for several hours.

Carbon dioxide absorption adds another physiological challenge. CO₂ rapidly crosses the peritoneal surface and enters systemic circulation, increasing the amount of carbon dioxide that must be eliminated by the lungs. Consequently, minute ventilation commonly needs to be increased during laparoscopy to maintain acceptable arterial CO₂ levels. When compensation is insufficient, hypercapnia and respiratory acidosis may develop, potentially increasing sympathetic activity, heart rate, systemic vascular resistance, and pulmonary vascular tone [1], [2]. Therefore, respiratory and hemodynamic effects should be interpreted as interconnected responses rather than isolated phenomena.

Advanced laparoscopic and robotic surgery may intensify these alterations because pneumoperitoneum is often combined with steep Trendelenburg positioning. This combination further displaces the diaphragm, reduces lung compliance, raises airway pressures, and changes regional ventilation distribution [16]–[19]. PEEP and recruitment strategies can improve lung aeration and reduce collapse in dependent regions, but excessive intrathoracic pressure may impair venous return and cardiovascular performance. For this reason, ventilatory optimization must be individualized rather than based solely on predetermined airway-pressure targets [18], [20].

Obesity represents an additional factor of clinical relevance. Obese patients often have reduced functional residual capacity and respiratory compliance even before abdominal insufflation begins. When obesity, pneumoperitoneum, anesthesia, and surgical positioning are combined, respiratory impairment becomes more pronounced and may be accompanied by deterioration in oxygenation and gas exchange [9]–[11]. These findings support the need for individualized intraoperative management that integrates insufflation pressure, ventilatory strategy, positioning, hemodynamic monitoring, and the patient's underlying cardiopulmonary condition.

GENERAL OBJECTIVE AND SPECIFIC OBJECTIVES

General Objective

To **analyze** the hemodynamic and respiratory physiological effects produced by carbon dioxide pneumoperitoneum during advanced laparoscopic surgery, integrating the influence of intra-abdominal pressure, patient positioning, ventilatory management, and individual clinical characteristics in order to identify factors that may contribute to safer perioperative management.

Specific Objectives

Cognitive Domain

1. To **identify** the principal cardiovascular and respiratory physiological mechanisms associated with CO₂ pneumoperitoneum during laparoscopic surgery.
2. To **explain** the relationship between increased intra-abdominal pressure and changes in venous return, cardiac preload, systemic vascular resistance, ventricular afterload, stroke volume, and cardiac output.
3. To **analyze** the effects of pneumoperitoneum on diaphragmatic displacement, functional residual capacity, respiratory system compliance, airway pressures, ventilation-perfusion relationships, and gas exchange.
4. To **compare** the physiological consequences of different insufflation pressures and surgical positions, particularly Trendelenburg and reverse Trendelenburg.
5. To **evaluate** the influence of obesity, cardiopulmonary reserve, duration of surgery, mechanical ventilation, and CO₂ absorption on the magnitude of intraoperative physiological alterations.

Psychomotor Domain

6. To **apply** physiological principles to the interpretation of intraoperative hemodynamic and respiratory monitoring parameters during advanced laparoscopic procedures.
7. To **differentiate** clinically relevant changes in airway pressure, compliance, end-tidal CO₂, arterial pressure, cardiac output, and oxygenation that may occur during pneumoperitoneum.
8. To **integrate** information obtained from cardiovascular and respiratory monitoring to support appropriate adjustments in ventilation, PEEP, insufflation pressure, positioning, fluid administration, and vasoactive therapy.

Affective Domain

9. To **value** the importance of continuous physiological assessment and individualized perioperative management in patients undergoing advanced laparoscopic surgery.
10. To **promote** a patient-safety-oriented approach that recognizes the potential hemodynamic and respiratory risks associated with pneumoperitoneum.
11. To **support** multidisciplinary decision-making among surgeons, anesthesiologists, and perioperative teams to minimize preventable physiological disturbances and optimize intraoperative care.

OBJECT OF STUDY

The object of study is the **physiological response to carbon dioxide pneumoperitoneum during advanced laparoscopic surgery**, with particular emphasis on the hemodynamic and respiratory alterations generated by increased intra-abdominal pressure.

The phenomenon under investigation includes changes in **venous return, cardiac preload, systemic vascular resistance, ventricular afterload, stroke volume, cardiac output, arterial pressure, diaphragmatic position, respiratory compliance, airway pressures, pulmonary ventilation, gas exchange, and CO₂ elimination** [1]–[3], [11]–[15].

The population of interest comprises **adult patients undergoing advanced laparoscopic or robotic-assisted surgical procedures** in which CO₂ pneumoperitoneum is required. Particular attention is given to patients exposed to prolonged insufflation, Trendelenburg or reverse Trendelenburg positioning, obesity, and reduced cardiovascular or respiratory reserve, as these conditions may intensify the physiological effects of pneumoperitoneum [9]–[11], [16]–[20].

The system under analysis is therefore the **cardiopulmonary interaction produced by the combined effects of pneumoperitoneum, mechanical ventilation, anesthesia, patient positioning, and individual clinical characteristics**. This approach allows the study to evaluate pneumoperitoneum not as an isolated surgical maneuver, but as a dynamic physiological condition capable of modifying multiple interconnected variables throughout the intraoperative period.

The main variables considered are intra-abdominal pressure, insufflation duration, surgical position, respiratory system compliance, peak and plateau airway pressures, oxygenation, end-tidal CO₂, systemic vascular resistance, arterial pressure, venous return, and cardiac output. Their analysis provides the basis for understanding the physiological impact of pneumoperitoneum and identifying factors associated with greater cardiopulmonary stress during advanced laparoscopic surgery.

METHODOLOGY

This study was conducted using the **Scientific Method**, structured as an analytical review focused on the physiological effects of carbon dioxide pneumoperitoneum during advanced laparoscopic surgery. This methodological approach was selected because it allows the identification of a clinical problem, formulation of research questions and hypotheses, systematic collection of evidence, comparative analysis of findings, and construction of evidence-based conclusions.

The research process was organized around the following central question: **How does CO₂ pneumoperitoneum affect hemodynamic and respiratory physiology during advanced laparoscopic surgery, and which factors modify the magnitude of these effects?**

A structured literature search was performed using scientific publications addressing pneumoperitoneum, laparoscopic surgery, robotic surgery, cardiovascular physiology, pulmonary mechanics, intra-abdominal pressure, Trendelenburg positioning, obesity, mechanical ventilation, and perioperative monitoring. Relevant studies included physiological investigations, clinical studies, randomized trials, observational studies, and narrative or analytical reviews directly related to the research topic [1]–[20].

The main search concepts included combinations of the terms *pneumoperitoneum, laparoscopic surgery, hemodynamic effects, cardiac output, venous return, systemic vascular resistance, respiratory mechanics, lung compliance, airway pressure, carbon dioxide, Trendelenburg, robotic surgery, and mechanical ventilation*.

Studies were included when they examined adult patients undergoing laparoscopic or robotic-assisted surgery and reported relevant cardiovascular or respiratory physiological variables. Publications addressing the effects of intra-abdominal pressure, surgical positioning, obesity, ventilation strategies, or insufflation pressure were also considered. Studies without a direct relationship to CO₂ pneumoperitoneum or without clinically interpretable physiological outcomes were excluded.

The information obtained from the selected literature was organized into two principal analytical domains. The **hemodynamic domain** included venous return, preload, afterload, systemic vascular resistance, arterial pressure, stroke volume, and cardiac output. The **respiratory domain** included diaphragmatic displacement, respiratory system compliance, airway pressures, functional residual capacity, ventilation-perfusion relationships, oxygenation, and CO₂ elimination.

Additional variables such as insufflation pressure, duration of pneumoperitoneum, patient positioning, obesity, baseline cardiopulmonary reserve, PEEP, and mechanical ventilation were analyzed as modifying factors because they may alter the intensity of the physiological response [6], [9], [11], [18]–[20].

Finally, findings from individual studies were compared to identify consistent physiological patterns, differences between clinical conditions, and potential interactions between cardiovascular and respiratory responses. This methodological structure allows the study to be replicated by applying the same research question, inclusion criteria, physiological variables, evidence classification, and comparative analytical process.

PHASES OF DEVELOPMENT

Phase 1. Identification of the Research Problem

The first phase consisted of identifying the physiological alterations produced by CO₂ pneumoperitoneum during advanced laparoscopic surgery. Particular attention was given to changes in cardiovascular performance, respiratory mechanics, intra-abdominal pressure, surgical positioning, and ventilatory conditions.

Phase 2. Formulation of the Research Question and Hypothesis

The central research question was established to determine how pneumoperitoneum modifies hemodynamic and respiratory physiology and which perioperative factors increase or reduce these effects. Based on previous physiological evidence, the hypothesis proposed that higher intra-abdominal pressures, prolonged insufflation, obesity, extreme positioning, and reduced cardiopulmonary reserve are associated with greater physiological disturbance.

Phase 3. Collection of Scientific Evidence

Relevant scientific literature was identified using terms related to *pneumoperitoneum*, *laparoscopic surgery*, *hemodynamics*, *cardiac output*, *respiratory mechanics*, *lung compliance*, *airway pressure*, *Trendelenburg*, *obesity*, and *mechanical ventilation*. Studies directly evaluating cardiovascular or respiratory effects of CO₂ insufflation were prioritized [1]–[20].

Phase 4. Selection and Classification of Information

The selected evidence was classified into two main analytical domains. The first included hemodynamic variables such as venous return, preload, afterload, systemic vascular resistance, arterial pressure, stroke volume, and cardiac output. The second included respiratory variables such as lung compliance, airway pressures, diaphragmatic displacement, gas exchange, and CO₂ elimination.

Phase 5. Comparative Analysis

The findings of the selected studies were compared according to insufflation pressure, patient positioning, duration of pneumoperitoneum, obesity, and ventilatory strategy. This phase allowed the identification of recurring physiological patterns and differences associated with specific surgical conditions [4], [6], [14], [16], [19].

Phase 6. Quantitative Representation of Results

The principal findings were organized into graphical representations to facilitate comparison. The figures summarized changes in hemodynamic variables, the influence of low- and high-pressure pneumoperitoneum, alterations in respiratory mechanics, the combined effect of pneumoperitoneum and Trendelenburg positioning, and the influence of body habitus on transpulmonary driving pressure.

Phase 7. Interpretation of Findings

The quantitative results were interpreted according to established cardiovascular and respiratory physiological mechanisms. Particular emphasis was placed on the relationship between increased intra-abdominal pressure, vascular resistance, cardiac output, diaphragmatic displacement, pulmonary compliance, and airway pressure.

Phase 8. Discussion and Integration

The results were contrasted with previous evidence to explain agreements, variability among studies, and relevant physiological interactions. The analysis integrated cardiovascular and respiratory responses rather than evaluating them as isolated phenomena.

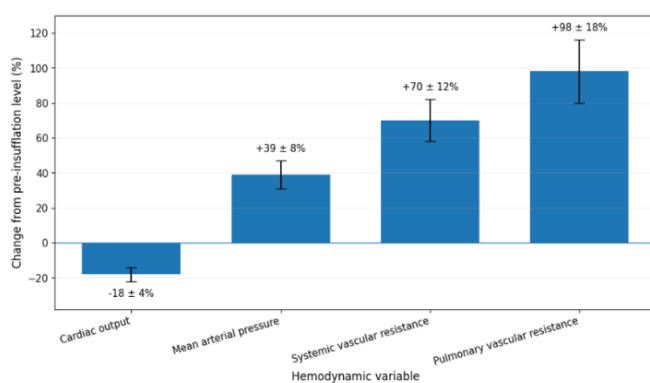
Phase 9. Formulation of Conclusions

The final phase consisted of synthesizing the evidence and determining that the physiological effects of pneumoperitoneum depend on multiple interacting factors. The conclusions emphasized the importance of individualized insufflation pressure, ventilatory management, hemodynamic monitoring, surgical positioning, and patient-specific cardiopulmonary characteristics during advanced laparoscopic surgery.

RESULTS AND DISCUSSION

Figure 1.

Hemodynamic response to CO₂ pneumoperitoneum during laparoscopic surgery



Data: Joris *et al.*, *Journal of the American College of Cardiology* (1998), reference [4].

Figure 1 demonstrates the magnitude and direction of the principal hemodynamic changes observed after the establishment of CO₂ pneumoperitoneum. The quantitative values represented in the graph correspond to the physiological changes reported by Joris *et al.* [4], in whom peritoneal insufflation produced a **reduction in cardiac output of 18 ± 4%**, accompanied by increases of **39 ± 8% in mean arterial pressure (MAP)**, **70 ± 12% in systemic vascular resistance (SVR)**, and **98 ± 18% in pulmonary vascular resistance (PVR)**. These results show that the cardiovascular response to pneumoperitoneum is characterized not simply by changes in arterial pressure, but by simultaneous modifications in cardiac performance and vascular load [4].

The reduction in cardiac output is particularly relevant because it occurs despite the considerable elevation in MAP. This apparent dissociation indicates that preservation or elevation of arterial pressure during pneumoperitoneum should not automatically be interpreted as preservation of systemic blood flow. Increased intra-abdominal pressure can compress the inferior vena cava and the abdominal venous circulation, modifying effective venous return and ventricular filling. At the same time, compression of arterial and microvascular territories increases vascular impedance and contributes to an elevation in ventricular afterload [1], [3]. Consequently, the heart may eject blood against a substantially higher resistance while receiving a reduced or mechanically altered preload.

The **18% reduction in cardiac output** represented in the figure is consistent with the general physiological behavior described in previous investigations of laparoscopic surgery. The magnitude of the response, however, is not uniform among patients. Differences in intravascular volume, anesthetic depth, insufflation pressure, body position, cardiovascular reserve, and duration of pneumoperitoneum can substantially influence the final cardiac response [3], [6], [7]. Hirvonen *et al.* [7], for example, reported that cardiac index could decrease during sustained pneumoperitoneum and emphasized the additional influence of head-up positioning on venous return. This variability explains why similar intra-abdominal pressures may produce relatively modest effects in one patient but clinically relevant decreases in cardiac performance in another.

The increase in **MAP of approximately 39%** provides evidence of a strong pressor response during the early period following insufflation [4]. This elevation results from the interaction between mechanical vascular compression and neurohumoral activation. Increased intra-abdominal pressure contributes directly to greater vascular resistance, while peritoneal distension and CO₂-related physiological responses activate sympathetic mechanisms. Joris *et al.* additionally demonstrated marked increases in vasopressin and catecholamine concentrations during pneumoperitoneum, supporting the participation of neuroendocrine mechanisms in maintaining or increasing arterial pressure [4].

The most pronounced systemic change shown in Figure 1 is the **70% increase in systemic vascular resistance**. This finding indicates a considerable rise in the resistance against which the left ventricle must eject blood. The elevation in SVR increases left ventricular afterload and can contribute to the reduction in stroke volume and cardiac output, particularly in patients whose ventricular contractile reserve is limited [1], [4]. The simultaneous occurrence of elevated MAP and decreased cardiac output is therefore physiologically coherent: arterial pressure is maintained predominantly by increased vascular resistance rather than by increased forward flow.

This pattern also illustrates why MAP alone represents an incomplete indicator of cardiovascular stability during advanced laparoscopy. A patient may maintain an apparently adequate or even elevated MAP while cardiac output and systemic oxygen delivery are reduced. In the study represented in the figure, changes in oxygen delivery followed the changes in cardiac output, reinforcing the importance of interpreting pressure-based measurements together with variables that reflect flow and tissue perfusion [4]. This distinction becomes increasingly important during prolonged or complex laparoscopic procedures.

The **98% increase in pulmonary vascular resistance** was the largest proportional change observed [4]. Increased PVR may result from a combination of elevated intrathoracic pressure, alterations in lung volumes, mechanical ventilation, hypercapnia, pulmonary vascular compression, and changes in pulmonary vascular tone. Although these alterations are generally tolerated by healthy individuals, a marked increase in right ventricular afterload may be more important in patients with pulmonary hypertension, right ventricular dysfunction, chronic pulmonary disease, or limited cardiopulmonary reserve [1], [2], [4].

The data also demonstrate that the hemodynamic response to pneumoperitoneum is strongly pressure dependent. Higher insufflation pressures produce greater mechanical compression of abdominal vessels and may enhance the reduction in venous return and cardiac performance. Dexter *et al.* [6] demonstrated differences between high- and low-pressure capnoperitoneum, with greater cardiovascular disturbance at higher pressures. This supports the physiological relationship between intra-abdominal pressure and the severity of hemodynamic alterations observed during laparoscopy.

Patient positioning can further modify these changes. Head-up or reverse Trendelenburg positioning promotes gravitational redistribution of circulating blood and may reduce effective preload. Hirvonen *et al.* [7] identified decreases in cardiac index, stroke index, and cardiac filling pressures with head-up positioning, showing that the final intraoperative hemodynamic state reflects the combined effect of anesthesia, pneumoperitoneum, and body position rather than insufflation pressure alone.

Figure 2.

Hemodynamic effects of low- versus high-pressure CO₂ pneumoperitoneum

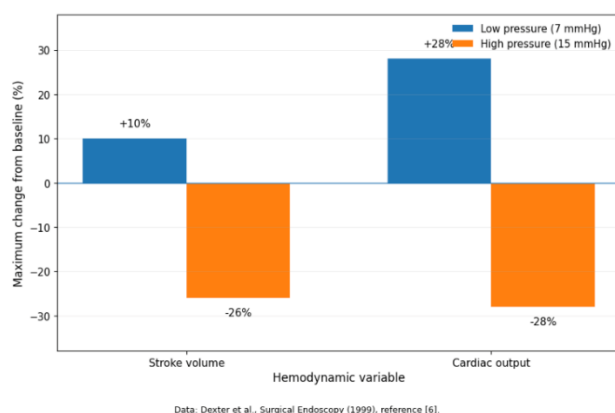


Figure 2 shows the differences in stroke volume and cardiac output according to the intra-abdominal pressure used to establish CO₂ pneumoperitoneum. The graph is based on the randomized clinical study by Dexter *et al.* [6], which compared a **low-pressure pneumoperitoneum of 7 mmHg** with a **high-pressure pneumoperitoneum of 15 mmHg** during laparoscopic cholecystectomy. The findings demonstrate a clearly different cardiovascular response between the two insufflation strategies: low pressure was associated with a peak **10% increase in stroke volume and 28% increase in cardiac output**, whereas high pressure produced maximum reductions of approximately **26% in stroke volume and 28% in cardiac output** [6].

The direction of these changes is physiologically important. At 7 mmHg, the moderate increase in intra-abdominal pressure may mobilize blood from the abdominal venous reservoir toward the central circulation without producing sufficient compression of the inferior vena cava to markedly impair venous return. Under these conditions, ventricular filling can be maintained and stroke volume may transiently increase. The observed 28% increase in cardiac output in the low-pressure group therefore suggests that moderate abdominal insufflation can be hemodynamically tolerated when venous return and ventricular loading conditions remain favorable [3], [6].

In contrast, increasing intra-abdominal pressure to 15 mmHg changes this balance. At higher pressures, compression of the inferior vena cava and abdominal venous system becomes more pronounced, increasing resistance to venous return and limiting right ventricular filling. This reduction in effective preload contributes to the **26% decrease in stroke volume** observed in the high-pressure group [6]. Because cardiac output is determined by the product of heart rate and stroke volume, a substantial reduction in stroke volume can translate into a marked reduction in systemic blood flow even when compensatory tachycardia occurs.

The **28% reduction in cardiac output at 15 mmHg** is particularly relevant because it demonstrates that the physiological impact of pneumoperitoneum is strongly pressure dependent. Dexter *et al.* specifically reported that

insufflation to 15 mmHg diminished venous return and reduced cardiac output, whereas low-pressure pneumoperitoneum minimized these adverse hemodynamic effects [6]. This finding supports the concept that the pressure necessary to obtain surgical exposure should not be considered physiologically neutral.

Another important aspect is the simultaneous increase in arterial pressure reported during high-pressure insufflation. In the high-pressure group, heart rate and mean arterial blood pressure increased despite the reductions in stroke volume and cardiac output [6]. This apparent paradox results primarily from an increase in systemic vascular resistance. Therefore, arterial pressure may remain stable or rise even when forward blood flow is reduced. This pattern reinforces the finding described in Figure 1, where pneumoperitoneum was associated with increased vascular resistance and arterial pressure despite decreased cardiac output [4].

The difference between low- and high-pressure pneumoperitoneum also reflects changes in ventricular afterload. Higher intra-abdominal pressure produces mechanical compression of the abdominal arterial circulation and activates sympathetic and neurohumoral mechanisms. Increased systemic vascular resistance increases the pressure against which the left ventricle must eject blood. Thus, high-pressure pneumoperitoneum can simultaneously reduce preload and increase afterload, creating a hemodynamic situation unfavorable for maintenance of stroke volume [1], [3], [4].

These effects may become more significant in patients with limited cardiovascular reserve. A young and otherwise healthy patient may compensate for a moderate reduction in stroke volume through increases in heart rate, vascular tone, and contractility. In contrast, patients with left ventricular dysfunction, coronary artery disease, pulmonary hypertension, right ventricular impairment, advanced age, or relative hypovolemia may have a reduced capacity to compensate for the combined effects of decreased venous return and increased vascular resistance [1], [3], [6].

The findings represented in Figure 2 also illustrate why the use of the lowest effective insufflation pressure has received considerable attention in laparoscopic surgery. A subsequent body of randomized evidence has compared low-pressure approaches—commonly between approximately 6 and 10 mmHg—with conventional pressures ranging approximately from 12 to 15 mmHg. These investigations consistently recognize intra-abdominal pressure as an important modifiable component of the physiological burden associated with pneumoperitoneum.

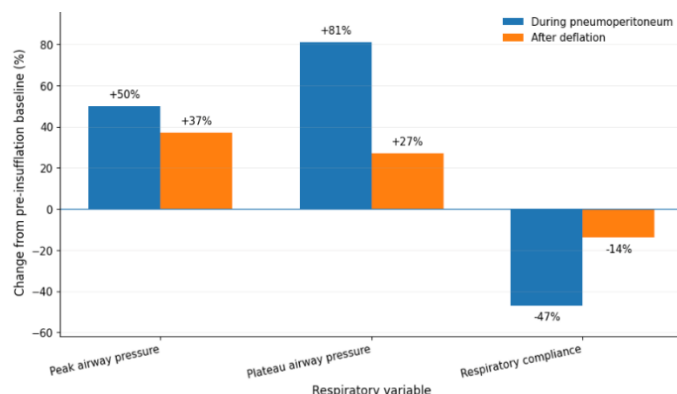
Nevertheless, the relationship between pressure and cardiovascular response should not be interpreted as completely linear or identical in every patient. The effect of pneumoperitoneum depends on baseline volume status, abdominal compliance, anesthetic depth, ventilation, surgical position, body habitus, and pre-existing cardiopulmonary function. For example, reverse Trendelenburg positioning can further decrease venous return by promoting peripheral blood pooling, while steep Trendelenburg may increase measured central venous pressures without necessarily producing an equivalent improvement in effective cardiac preload [7], [8].

The data also emphasize the distinction between **pressure variables and flow variables**. During high-pressure pneumoperitoneum, an increase in MAP does not necessarily indicate improved circulation. In the study represented here, systemic pressure increased while cardiac output fell [6]. Therefore, relying exclusively on blood pressure could underestimate the reduction in cardiac performance produced by increased intra-abdominal pressure. In complex laparoscopic procedures, a more complete interpretation should consider heart rate, stroke volume, cardiac output, perfusion variables, and the temporal relationship with changes in insufflation pressure.

From the perspective of advanced laparoscopic surgery, this distinction becomes even more relevant because pneumoperitoneum may be maintained for prolonged periods. A transient fall in cardiac output during short insufflation may be well tolerated, whereas sustained reductions during prolonged oncologic, bariatric, colorectal, urologic, or robotic procedures may create a more persistent hemodynamic burden. The effect can be amplified by anesthesia, mechanical ventilation, PEEP, extreme positioning, blood loss, and changes in intravascular volume [3], [7], [20].

Figure 3.

Changes in respiratory mechanics induced by CO₂ pneumoperitoneum



Data: Bardoczky et al., Anaesthesia (1993), reference [14].

Figure 3 demonstrates the marked changes in respiratory mechanics that occur during CO₂ pneumoperitoneum and the degree to which these alterations persist immediately after abdominal deflation. The quantitative values are derived from Bardoczky *et al.* [14], who continuously evaluated airway pressures and respiratory system compliance in patients undergoing laparoscopy with an intra-abdominal pressure of approximately 1.6 kPa. During pneumoperitoneum, **peak airway pressure increased by 50%, plateau airway pressure increased by 81%, and respiratory system compliance decreased by 47%** compared with pre-insufflation conditions.

The **50% increase in peak airway pressure** reflects the greater total pressure required to deliver a constant tidal volume once the abdomen is insufflated. Pneumoperitoneum increases intra-abdominal pressure and displaces the diaphragm cephalad, reducing the available thoracic volume and modifying the mechanical relationship between the lungs and chest wall. Consequently, the respiratory system becomes less distensible, requiring higher inspiratory pressures to achieve the same ventilatory target [2], [13], [14].

The increase in **plateau airway pressure was even greater, reaching 81% above the pre-insufflation level** [14]. This variable is particularly relevant because plateau pressure more closely reflects the elastic load imposed on the respiratory system after inspiratory flow has ceased. Its substantial elevation therefore indicates that pneumoperitoneum produces a major increase in respiratory system elastance rather than simply an increase in airway resistance.

This finding can be explained by the cephalad displacement of the diaphragm and compression of dependent lung regions. As intra-abdominal pressure rises, the diaphragm moves upward, reducing functional residual capacity and decreasing the volume of aerated lung available for ventilation. Basal and posterior pulmonary regions are especially susceptible to compression, facilitating airway closure and atelectatic changes. The remaining ventilated lung therefore receives a greater proportion of the tidal volume, while the pressure required to expand the respiratory system increases [11], [13]–[15].

The **47% reduction in respiratory system compliance** represents the most important mechanical deterioration demonstrated in the figure [14]. Compliance describes the change in volume achieved for a given change in pressure; therefore, a reduction approaching one half of the baseline value indicates a substantial increase in the mechanical load against which the ventilator must operate. Bardoczky *et al.* observed this deterioration while minute ventilation remained constant, demonstrating that the change was directly related to the mechanical consequences of increased intra-abdominal pressure rather than to modifications in the ventilatory pattern.

Other studies have documented the same direction of change. Oikkonen and Tallgren [12] evaluated 32 patients undergoing laparoscopic procedures and found that respiratory compliance decreased abruptly by approximately **20% immediately after CO₂ insufflation**, with further deterioration during continued pneumoperitoneum in some surgical positions. This provides additional evidence that decreased respiratory compliance is a reproducible consequence of abdominal pressurization rather than a finding restricted to a single surgical population.

The difference in magnitude between studies—approximately 20% in the investigation by Oikkonen and Tallgren [12] versus 47% in Bardoczky *et al.* [14]—also demonstrates that the respiratory response to pneumoperitoneum is not uniform. Variability may depend on intra-abdominal pressure, patient position, baseline pulmonary mechanics, anesthetic technique, surgical characteristics, body habitus, and the timing of measurements. Thus, the mechanical effect should be interpreted as a dynamic response rather than as a fixed percentage reduction applicable to every patient.

A particularly important observation in Figure 3 is that respiratory mechanics did not return immediately to their original values following deflation. After release of pneumoperitoneum, peak airway pressure remained approximately

37% above baseline, plateau pressure remained **27% above baseline**, and respiratory compliance recovered only to **86% of its initial value**, corresponding to a residual reduction of approximately 14% [14].

This incomplete immediate recovery suggests that the mechanical consequences of pneumoperitoneum can persist beyond the period of active insufflation. Possible contributing factors include residual dependent atelectasis, incomplete diaphragmatic repositioning, changes in chest wall mechanics, and persistent reduction of functional lung volume. The finding is particularly relevant in prolonged advanced laparoscopic procedures because repeated or sustained exposure to elevated abdominal pressure may produce mechanical changes that are not instantly reversed when pneumoperitoneum is discontinued.

The persistent elevation of peak and plateau pressures after deflation also indicates that normalization of abdominal pressure does not necessarily result in immediate normalization of pulmonary mechanics. Therefore, intraoperative respiratory assessment should consider the temporal evolution of the patient rather than assuming that lung mechanics will immediately return to baseline at the end of insufflation [12], [14].

The decrease in compliance also has direct consequences for mechanical ventilation. If tidal volume is maintained unchanged while compliance decreases, higher airway pressures will be required. Conversely, if clinicians attempt to avoid excessive pressure without modifying other ventilatory parameters, delivered tidal volume and alveolar ventilation may decrease. This interaction explains why pneumoperitoneum often requires adjustment of ventilatory settings during laparoscopic surgery [14], [20].

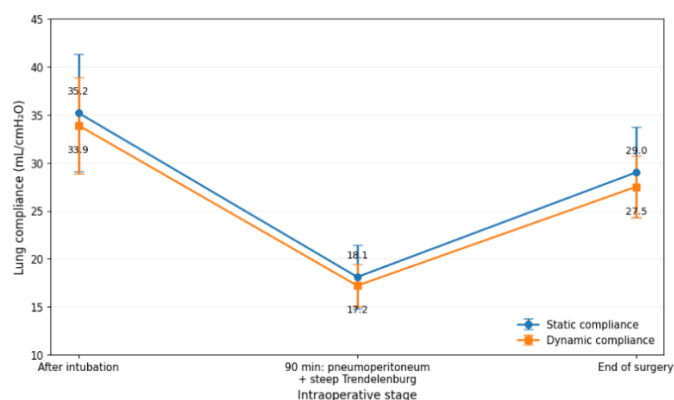
Increased airway and plateau pressures must also be interpreted in relation to chest wall mechanics. During pneumoperitoneum, a considerable portion of the increased airway pressure may be required to overcome the elevated elastic load imposed by the abdomen and chest wall rather than reflecting an equivalent increase in transpulmonary pressure. Studies examining the mechanical effects of surgical pneumoperitoneum have demonstrated that abdominal insufflation substantially alters the interaction between the lung and chest wall [15]. This distinction is important when assessing the potential risk of pulmonary overdistension solely from airway pressure values.

The mechanical changes represented in Figure 3 can also influence gas exchange. Reduced functional lung volume and increased dependent atelectasis may worsen ventilation-perfusion matching. At the same time, absorbed CO₂ increases the ventilatory requirement. The anesthetic team therefore faces two simultaneous processes: **more ventilation may be required to eliminate absorbed CO₂, while the respiratory system becomes mechanically more difficult to ventilate** [1], [2], [14]. This interaction is one of the principal respiratory challenges of advanced laparoscopic surgery.

These effects may become more pronounced in patients with obesity. Reduced baseline functional residual capacity and increased chest wall load can already compromise respiratory mechanics before pneumoperitoneum begins. Once intra-abdominal pressure is increased, additional cephalad diaphragmatic displacement can result in a greater deterioration in compliance and higher airway pressures [9]–[11]. The combination with steep Trendelenburg may further intensify this mechanical impairment.

Figure 4.

Effect of prolonged pneumoperitoneum combined with steep Trendelenburg positioning on pulmonary compliance



Data: Kim et al., PLOS ONE (2018), reference [16]. Values are mean $\pm$ SD.

Figure 4 demonstrates the evolution of static and dynamic pulmonary compliance during major laparoscopic pelvic surgery requiring prolonged CO₂ pneumoperitoneum and steep Trendelenburg positioning. The quantitative data correspond to the prospective observational study by Kim *et al.* [16], which evaluated patients undergoing pelviscopic radical hysterectomy and compared respiratory mechanics after tracheal intubation, after 90 minutes of pneumoperitoneum combined with Trendelenburg positioning, and at the end of surgery. Both static and dynamic compliance changed significantly throughout the procedure ($P < 0.001$) [16].

After intubation, mean **static pulmonary compliance was 35.2 ± 6.1 mL/cmH₂O**, while dynamic compliance was **33.9 ± 5.0 mL/cmH₂O**. After 90 minutes of pneumoperitoneum with steep Trendelenburg, these values decreased to **18.1 ± 3.3 mL/cmH₂O** and **17.2 ± 2.2 mL/cmH₂O**, respectively [16]. This represents an approximate **48.6% reduction in static compliance and 49.3% reduction in dynamic compliance**, indicating that nearly half of the initial distensibility of the respiratory system was lost during the combined surgical condition.

This marked decrease reflects the mechanical consequences of increased intra-abdominal pressure together with cephalad gravitational displacement of the abdominal contents. CO₂ insufflation increases pressure beneath the diaphragm, while Trendelenburg positioning further shifts the abdominal viscera toward the thoracic cavity. The resulting cephalad displacement of the diaphragm reduces available thoracic volume and increases the elastic load imposed on the respiratory system [16], [18]. The lungs therefore require considerably greater pressure to generate the same tidal volume.

The magnitude of this mechanical deterioration is also demonstrated by the change in **peak inspiratory pressure**. In the same study, PIP increased from **12.8 ± 2.0 cmH₂O after intubation to 25.6 ± 2.8 cmH₂O after 90 minutes of pneumoperitoneum and Trendelenburg positioning**, representing approximately a twofold increase [16]. Thus, the decrease in compliance was accompanied by a substantial increase in the pressure required to ventilate the patient.

These results are physiologically consistent because compliance is inversely related to the pressure required to produce a given change in lung volume. When abdominal insufflation and positioning reduce respiratory system compliance, maintenance of the same tidal volume necessarily requires increased airway pressure. Consequently, a simultaneous decrease in compliance and elevation of peak or plateau pressure should be expected during prolonged laparoscopic pelvic and robotic procedures [16]–[18].

The mechanical alterations are strongly related to diaphragmatic behavior. Kim *et al.* found that diaphragmatic excursion progressively decreased during the procedure and reached its greatest reduction after 90 minutes of Trendelenburg positioning with pneumoperitoneum [16]. The restricted movement of the diaphragm provides a direct anatomical explanation for the reduction in pulmonary compliance represented in Figure 4. The combination of abdominal pressure and gravitational displacement limits diaphragmatic excursion and increases compression of dependent pulmonary regions.

Compression is especially important in the dorsal and basal portions of the lungs. As the diaphragm moves cephalad, dependent alveolar units become increasingly susceptible to reduced aeration and atelectasis. In robotic-assisted surgery, electrical impedance tomography studies have confirmed redistribution of ventilation away from dependent dorsal lung regions during pneumoperitoneum combined with Trendelenburg positioning [18]. Shono *et al.* demonstrated that this condition substantially affects regional ventilation and respiratory mechanics during robot-assisted laparoscopic prostatectomy.

Gas exchange also changed during the period of greatest mechanical impairment. In the study by Kim *et al.*, PaO₂ decreased from **240.2 ± 52.4 mmHg to 204.6 ± 40.2 mmHg**, while PaCO₂ increased from **35.8 ± 4.1 to 40.7 ± 4.7 mmHg** during pneumoperitoneum and Trendelenburg positioning. End-tidal CO₂ simultaneously increased from **30.6 ± 2.7 to 33.1 ± 4.2 mmHg** [16]. These alterations demonstrate that the mechanical consequences shown in the figure occur together with measurable changes in oxygenation and CO₂ homeostasis.

The increase in carbon dioxide levels results from both pulmonary mechanical changes and systemic absorption of insufflated CO₂. When lung compliance decreases, effective alveolar ventilation can become more difficult to maintain without increasing respiratory rate or modifying other ventilatory parameters. At the same time, continuous absorption of CO₂ through the peritoneal surface increases the amount that must be eliminated through the lungs. These simultaneous processes explain the progressive rise in PaCO₂ and EtCO₂ observed during prolonged pneumoperitoneum [1], [2], [16].

At the end of surgery, both static and dynamic compliance showed substantial recovery but did not return completely to their initial values. Static compliance increased from 18.1 to **29.0 ± 4.7 mL/cmH₂O**, while dynamic compliance increased from 17.2 to **27.5 ± 3.2 mL/cmH₂O** [16]. Nevertheless, these final values remained approximately **18% below the measurements obtained after intubation**, indicating incomplete immediate normalization of pulmonary mechanics.

The persistence of altered compliance after termination of the main surgical condition suggests that removal of pneumoperitoneum and restoration of position do not instantly reverse all respiratory consequences. Residual

atelectatic areas, altered diaphragmatic mechanics, reduced functional lung volume, and prolonged mechanical compression may contribute to this incomplete recovery. Kim *et al.* similarly found that diaphragmatic excursion had not completely returned to its initial condition at the end of surgery [16].

The findings have particular relevance to **advanced robotic-assisted procedures**, where pneumoperitoneum and Trendelenburg positioning may be maintained for several hours. Brandão *et al.* [17] demonstrated important changes in global and regional respiratory mechanics during robotic-assisted laparoscopic surgery, while Tharp *et al.* [19] showed that dynamic surgical conditions and body habitus independently impair pulmonary mechanics. Therefore, the approximately 50% reduction in compliance represented in Figure 4 illustrates a physiological phenomenon that may become even more important in patients with obesity or reduced baseline pulmonary reserve.

Ventilatory strategy can modify the magnitude of this deterioration. Shono *et al.* [18] compared PEEP levels of 5 and 15 cmH₂O during pneumoperitoneum combined with Trendelenburg positioning. Higher PEEP increased ventilation of the most dorsal lung region from $5.5 \pm 3.9\%$ to $9.9 \pm 4.7\%$ and reduced driving pressure from 14.9 ± 2.5 to 11.5 ± 2.8 cmH₂O, demonstrating that changes in ventilatory management can alter the mechanical and regional consequences of the surgical condition [18].

However, respiratory optimization must remain integrated with cardiovascular physiology. Higher PEEP increases intrathoracic pressure and may potentially reduce venous return, particularly when it is combined with elevated intra-abdominal pressure. Thus, improvements in dorsal ventilation and compliance must be balanced against their possible hemodynamic effects [18], [20]. This interaction is particularly relevant in advanced laparoscopic surgery, where respiratory and cardiovascular changes occur simultaneously.

Figure 5.

Independent effects of body habitus, pneumoperitoneum, and Trendelenburg positioning on transpulmonary driving pressure

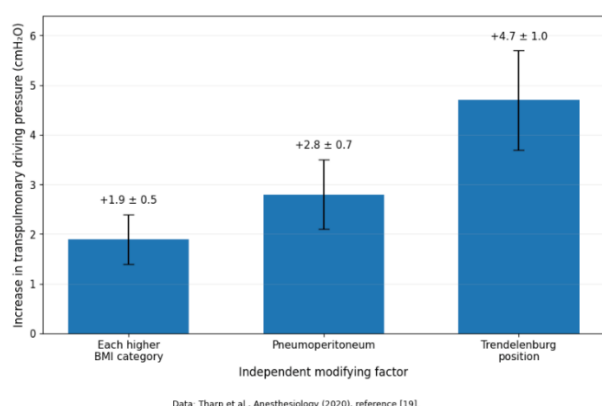


Figure 5 integrates three major determinants of respiratory mechanical impairment during robotic-assisted laparoscopic surgery: **body habitus, pneumoperitoneum, and Trendelenburg positioning**. The quantitative data are derived from the study by Tharp *et al.* [19], which evaluated respiratory mechanics in **91 patients with body mass index values ranging from 18.3 to 60.6 kg/m²** at successive stages of robotic laparoscopic surgery. The analysis demonstrated that each of these factors independently increased transpulmonary driving pressure, indicating progressively greater mechanical stress imposed on the lungs during intraoperative ventilation [19].

Increasing BMI category was independently associated with an average 1.9 ± 0.5 cmH₂O increase in transpulmonary driving pressure [19]. This finding demonstrates that body habitus modifies pulmonary mechanics even before the additional mechanical effects of pneumoperitoneum and surgical positioning are considered. Obesity is associated with increased abdominal mass, reduced functional residual capacity, elevated pleural pressure, reduced chest wall compliance, and a greater tendency toward dependent airway closure and alveolar collapse. Consequently, patients with higher BMI generally require greater pressure to maintain comparable ventilation during anesthesia.

The influence of obesity was also evident in end-expiratory transpulmonary pressure. Patients with **BMI ≥ 40 kg/m² had a mean end-expiratory transpulmonary pressure of -7.5 ± 6.3 cmH₂O**, compared with only -1.3 ± 3.4 cmH₂O in patients with BMI < 25 kg/m² [19]. A more negative end-expiratory transpulmonary pressure indicates a greater tendency toward compression and collapse of dependent pulmonary units at end expiration. Therefore, increasing BMI

does not merely increase airway pressure; it fundamentally alters the balance between alveolar pressure and surrounding pleural pressure.

The second factor represented in Figure 5 is **pneumoperitoneum**, which independently increased transpulmonary driving pressure by $2.8 \pm 0.7 \text{ cmH}_2\text{O}$ compared with baseline conditions [19]. The magnitude of this effect was greater than that associated with a single increase in BMI category, demonstrating the substantial mechanical influence of abdominal insufflation itself.

This increase results primarily from elevation of intra-abdominal pressure and cephalad displacement of the diaphragm. As abdominal volume and pressure increase, the diaphragm is pushed toward the thoracic cavity, reducing functional lung volume and increasing pleural pressure. Dependent pulmonary regions become compressed, respiratory system elastance increases, and greater pressure is required to generate the same tidal volume [11], [15], [19].

Pneumoperitoneum additionally reduced end-expiratory transpulmonary pressure by approximately $3.4 \pm 1.3 \text{ cmH}_2\text{O}$ **relative to baseline** [19]. Thus, the increase in driving pressure occurs simultaneously with greater mechanical conditions favoring end-expiratory lung collapse. This combination explains why pneumoperitoneum is commonly associated with decreased respiratory compliance, increased airway pressure, dependent atelectasis, and redistribution of ventilation.

The third and most pronounced factor represented in the graph is **Trendelenburg positioning**, which produced an additional $4.7 \pm 1.0 \text{ cmH}_2\text{O}$ **increase in transpulmonary driving pressure** [19]. Among the individual modifying conditions represented in Figure 5, Trendelenburg therefore produced the largest measured increase. The magnitude of this effect reflects the additional gravitational displacement of abdominal contents toward the diaphragm when the patient is placed in a head-down position.

Trendelenburg positioning also reduced end-expiratory transpulmonary pressure by approximately $4.5 \pm 1.5 \text{ cmH}_2\text{O}$ **relative to baseline** [19]. This finding indicates that the position not only increases the pressure necessary to ventilate the lungs but also enhances the tendency toward end-expiratory compression. When Trendelenburg is combined with an already established pneumoperitoneum, the respiratory system must therefore operate under two simultaneous sources of diaphragmatic loading.

The results are consistent with those reported by Brandão *et al.* [17], who demonstrated progressive deterioration in global and regional respiratory mechanics when pneumoperitoneum was followed by steep Trendelenburg during robotic-assisted procedures. In that investigation, the proportion of respiratory driving pressure transmitted to the lungs decreased from approximately $63 \pm 15\%$ **at baseline to $49 \pm 14\%$ during Trendelenburg**, reflecting a particularly important increase in chest wall elastance [17].

This observation is physiologically relevant when interpreting airway pressures. During pneumoperitoneum and Trendelenburg, elevated airway pressure does not necessarily indicate that the entire pressure increase is being transmitted directly across the lung. A considerable proportion may be required to overcome the increased mechanical load of the chest wall and abdomen. Nevertheless, the increase in **transpulmonary driving pressure** demonstrated by Tharp *et al.* confirms that the lungs themselves also experience increased mechanical loading [19].

Figure 5 also emphasizes that these factors should not be considered independently in actual advanced laparoscopic surgery. A patient with obesity undergoing robotic pelvic surgery may simultaneously experience the baseline mechanical burden associated with increased BMI, the additional effect of CO₂ pneumoperitoneum, and the further deterioration produced by Trendelenburg positioning. Their coexistence explains why pulmonary mechanics may deteriorate profoundly during complex robotic procedures even in the absence of pre-existing intrinsic pulmonary disease.

Evidence from other robotic procedures supports this interaction. In prospective observations of robotic-assisted surgery, pneumoperitoneum has been associated with a decrease in respiratory compliance from approximately 42.5 **to $26.7 \text{ mL/cmH}_2\text{O}$** , while plateau pressure increased from approximately 16.1 **to 23.6** during abdominal insufflation. Obesity was also significantly correlated with lower compliance and larger disturbances in carbon dioxide homeostasis. Similarly, studies of robotic-assisted laparoscopic prostatectomy have demonstrated that increasing BMI predicts higher peak inspiratory and driving pressures and lower lung compliance during pneumoperitoneum and steep Trendelenburg. Patients with BMI above approximately 31 kg/m^2 were more likely to reach peak inspiratory pressures of $35 \text{ cmH}_2\text{O}$ **or greater**, further illustrating the interaction between obesity and surgical conditions.

The results represented in Figure 5 also have implications for PEEP requirements. Tharp *et al.* calculated that the PEEP necessary to maintain a positive end-expiratory transpulmonary pressure was greater than the PEEP actually applied in **79% of patients at baseline, 88% during pneumoperitoneum, and 95% during Trendelenburg** [19]. Estimated optimal PEEP values demonstrated considerable interindividual variability, ranging from 0 **to $36.6 \text{ cmH}_2\text{O}$** , depending on body habitus and surgical stage.

This wide range is an important quantitative finding because it demonstrates that a single fixed PEEP value cannot reproduce the same mechanical condition in every patient. Two individuals receiving identical ventilator settings may

have markedly different transpulmonary pressures because of differences in BMI, abdominal pressure, chest wall mechanics, and position. Therefore, the respiratory response observed during laparoscopic surgery exhibits substantial interindividual variability [18], [19].

Shono *et al.* [18] similarly demonstrated that ventilatory conditions can modify regional ventilation during pneumoperitoneum combined with steep Trendelenburg. Higher PEEP improved dorsal ventilation and reduced driving pressure under these conditions, supporting the existence of potentially recruitable dependent lung regions during robotic surgery [18].

However, the values represented in Figure 5 should not be interpreted as a simple arithmetic formula in which all three increments can automatically be added to predict an individual patient's final driving pressure. The study evaluated independent and dynamic effects across surgical conditions, and individual responses showed substantial variability [19]. Instead, the graph illustrates the **relative magnitude and direction of each modifying factor**, demonstrating that increasing BMI, pneumoperitoneum, and Trendelenburg all independently shift pulmonary mechanics toward greater respiratory loading.

The figure therefore complements the preceding respiratory results. Figure 3 demonstrated that pneumoperitoneum increases airway pressures while markedly reducing compliance, and Figure 4 showed an approximately 50% reduction in compliance when pneumoperitoneum was prolonged in steep Trendelenburg. Figure 5 identifies the variables responsible for part of this heterogeneity and quantitatively demonstrates that body habitus and dynamic surgical conditions independently contribute to deterioration in lung mechanics.

DISCUSSION

The findings of this study confirm that CO₂ pneumoperitoneum produces a complex and clinically relevant interaction between cardiovascular and respiratory physiology during advanced laparoscopic surgery. The results demonstrate that the physiological burden is not determined exclusively by intra-abdominal pressure, but by the combined effects of vascular compression, altered venous return, changes in ventricular loading conditions, diaphragmatic displacement, decreased respiratory compliance, CO₂ absorption, mechanical ventilation, body habitus, and surgical positioning [1]–[3].

From the hemodynamic perspective, the results showed a characteristic pattern consisting of **reduced cardiac output together with increased mean arterial pressure and systemic vascular resistance**. This finding is particularly important because it demonstrates that an apparently preserved or elevated arterial pressure does not necessarily indicate adequate systemic blood flow. Joris *et al.* reported a reduction in cardiac output associated with substantial increases in systemic and pulmonary vascular resistance during pneumoperitoneum [4]. Similar observations were described by Cunningham *et al.* and Larsen *et al.*, who identified alterations in cardiac filling and ventricular performance during laparoscopic procedures [5], [8].

The reduction in cardiac output can be explained by the simultaneous effects of decreased effective venous return and increased ventricular afterload. Elevated intra-abdominal pressure compresses the vena cava and abdominal venous circulation, potentially limiting preload, while sympathetic and neurohumoral activation increases systemic vascular resistance [1], [4]. Therefore, the cardiovascular system may maintain arterial pressure primarily through vasoconstriction rather than through preservation of cardiac output. This distinction is clinically relevant in patients with limited cardiac reserve, in whom increased afterload may further impair ventricular performance.

The results also demonstrated a clear influence of **insufflation pressure**. Low-pressure pneumoperitoneum was associated with more favorable changes in stroke volume and cardiac output, whereas higher pressure produced reductions in both variables [6]. These findings support the concept that the physiological effects of pneumoperitoneum are pressure dependent. The clinical implication is not that all laparoscopic procedures should necessarily be performed at minimal pressure, but that the pressure selected should represent the lowest level capable of maintaining adequate surgical exposure. Excessive insufflation pressure may add cardiovascular stress without providing proportional physiological benefit.

This observation is especially relevant in advanced laparoscopic procedures, where pneumoperitoneum may be maintained for prolonged periods. Short-term cardiovascular compensation observed in healthy patients may not adequately represent the physiological challenge faced by patients undergoing prolonged oncologic, bariatric, colorectal, urologic, or robotic surgery. In these situations, the effects of pneumoperitoneum can interact with blood loss, fluid shifts, anesthetic-induced vasodilation, positive-pressure ventilation, and extreme positioning [3], [7].

Patient positioning represents another determinant of hemodynamic variability. Reverse Trendelenburg may reduce venous return by promoting gravitational displacement of blood toward the lower extremities, whereas steep Trendelenburg increases central venous pressure but also increases intrathoracic pressure [7]. Consequently, static filling pressures may become difficult to interpret because an elevated central venous pressure does not necessarily

represent increased effective ventricular preload. This reinforces the importance of evaluating hemodynamic variables in their physiological context rather than interpreting isolated measurements.

The respiratory findings were equally consistent. Pneumoperitoneum produced a marked increase in peak and plateau airway pressures and a substantial reduction in respiratory system compliance [12]–[15]. Bardoczky *et al.* demonstrated that compliance could decrease by nearly one half during pneumoperitoneum, while airway pressures increased markedly [14]. The present analysis therefore supports the concept that increased intra-abdominal pressure significantly modifies thoracoabdominal mechanics.

The principal mechanism is cephalad displacement of the diaphragm. As abdominal pressure increases, the diaphragm moves toward the thoracic cavity, reducing functional lung volume and increasing the elastic load of the respiratory system [13], [15]. Dependent pulmonary regions become more susceptible to compression and atelectasis, which contributes to decreased compliance and altered ventilation-perfusion matching. These effects become particularly important during prolonged surgical procedures because mechanical impairment may persist even after deflation.

The results also showed that respiratory mechanics do not necessarily return immediately to baseline after the pneumoperitoneum is released. Residual reductions in compliance and persistent elevations in airway pressures have been documented after abdominal deflation [14]. This suggests that pneumoperitoneum can leave temporary residual changes related to atelectasis, altered diaphragmatic position, and incomplete recovery of functional lung volume. Therefore, respiratory assessment should continue during emergence and early postoperative recovery rather than assuming complete normalization after CO₂ evacuation.

The interaction between pneumoperitoneum and **steep Trendelenburg positioning** produced one of the most pronounced respiratory changes identified in this study. Kim *et al.* reported that static and dynamic compliance decreased by approximately half after prolonged pneumoperitoneum combined with Trendelenburg positioning [16]. These results are consistent with studies of robotic-assisted surgery showing significant deterioration in regional and global respiratory mechanics under similar conditions [17]–[19].

The importance of Trendelenburg lies in its additive mechanical effect. Pneumoperitoneum already displaces the diaphragm cephalad, and the head-down position further shifts abdominal contents toward the thorax. The resulting reduction in available thoracic space increases pleural pressure, decreases dependent lung aeration, and raises airway pressures. This explains why robotic pelvic surgery represents one of the most demanding scenarios for intraoperative respiratory management.

Obesity further magnifies this physiological burden. Patients with elevated BMI commonly present with reduced functional residual capacity, increased chest wall elastance, and a greater tendency toward airway closure even before pneumoperitoneum is established [9]–[11]. Tharp *et al.* demonstrated that increasing BMI, pneumoperitoneum, and Trendelenburg independently increased transpulmonary driving pressure [19]. These findings indicate that obesity is not merely a demographic characteristic but an important modifier of intraoperative respiratory physiology.

The interaction among obesity, pneumoperitoneum, and positioning has important implications for ventilatory management. Standardized ventilator settings may not provide equivalent mechanical conditions in patients with different body habitus or surgical positions. A fixed PEEP level, for example, may be insufficient in one patient and excessive in another. This variability supports the use of individualized ventilatory strategies based on respiratory mechanics, oxygenation, airway pressures, and the patient's physiological response [18]–[20].

The role of PEEP is particularly relevant. Higher PEEP may improve dependent lung aeration and reduce regional collapse during pneumoperitoneum and Trendelenburg [18]. However, excessive PEEP increases intrathoracic pressure and may reduce venous return, especially when intra-abdominal pressure is already elevated. Consequently, respiratory optimization must be integrated with hemodynamic evaluation. Improving compliance or oxygenation at the expense of clinically significant cardiovascular impairment would represent an incomplete physiological strategy. CO₂ absorption represents another component linking respiratory and cardiovascular responses. The peritoneal absorption of carbon dioxide increases arterial and end-tidal CO₂, requiring increased minute ventilation to maintain acceptable acid-base status [1], [2]. If CO₂ elimination becomes insufficient, hypercapnia may stimulate sympathetic activity and increase systemic and pulmonary vascular resistance. Therefore, the metabolic consequences of CO₂ insufflation may further reinforce the cardiovascular changes produced by mechanical compression.

The findings collectively demonstrate that pneumoperitoneum should be considered a **dynamic cardiopulmonary condition rather than an isolated surgical technique**. Cardiovascular and respiratory changes occur simultaneously and frequently influence each other. Increased airway pressure can increase intrathoracic pressure and modify venous return; reduced cardiac output can affect systemic oxygen transport; atelectasis can alter gas exchange; and hypercapnia can modify vascular tone. For this reason, isolated interpretation of a single variable may lead to an incomplete assessment of the patient's physiological state.

The clinical heterogeneity observed across studies is also relevant. The magnitude of changes in cardiac output, vascular resistance, compliance, airway pressure, and gas exchange varied among investigations [4], [6], [12], [14],

[16], [19]. This variability likely reflects differences in insufflation pressure, duration, patient population, baseline cardiopulmonary status, anesthetic technique, surgical position, and ventilatory strategy. Rather than representing contradictory findings, this heterogeneity reinforces the importance of individualized assessment.

One limitation of the available evidence is that several foundational physiological studies were conducted during earlier stages of laparoscopic development and frequently involved relatively short procedures in selected populations. Contemporary advanced laparoscopic and robotic procedures may expose patients to longer pneumoperitoneum times, more extreme positioning, and more complex comorbidity profiles. Nevertheless, the physiological mechanisms identified in these earlier studies remain consistent with more recent investigations using modern respiratory monitoring and robotic surgical conditions [15]–[20].

Another limitation is the diversity of outcome measures used across studies. Some investigations focused on cardiac output or vascular resistance, whereas others evaluated airway pressures, respiratory compliance, transpulmonary pressure, regional ventilation, or gas exchange. This methodological heterogeneity limits direct numerical comparison between all studies. However, the consistency in the direction of the principal findings strengthens the overall interpretation: pneumoperitoneum increases vascular and respiratory mechanical load, while higher pressures, obesity, and extreme positioning tend to intensify these effects.

In summary, the evidence supports a model in which the physiological impact of advanced laparoscopic surgery results from the cumulative interaction of **intra-abdominal pressure, patient positioning, ventilatory strategy, CO₂ absorption, and individual cardiopulmonary characteristics**. The most relevant findings were reduced cardiac output under higher-pressure conditions, increased systemic and pulmonary vascular resistance, marked reductions in respiratory compliance, elevated airway pressures, and additional impairment associated with obesity and steep Trendelenburg [4], [6], [14], [16], [19].

These findings support the use of an individualized perioperative approach based on the lowest effective insufflation pressure, continuous interpretation of hemodynamic and respiratory variables, appropriate ventilatory adjustment, careful use of PEEP, and consideration of patient-specific risk factors. The physiological consequences of pneumoperitoneum are generally reversible, but their magnitude may become clinically significant when multiple adverse modifiers coexist. Understanding these interactions is therefore essential for optimizing safety during increasingly complex laparoscopic and robotic surgical procedures.

CONCLUSION

Carbon dioxide pneumoperitoneum produces significant and predictable hemodynamic and respiratory alterations during advanced laparoscopic surgery. The available evidence demonstrates that increased intra-abdominal pressure modifies venous return, cardiac preload, systemic and pulmonary vascular resistance, ventricular afterload, stroke volume, and cardiac output, while simultaneously reducing respiratory compliance, increasing airway pressures, displacing the diaphragm cephalad, and altering gas exchange [1]–[4], [12]–[15].

The magnitude of these effects depends on several interacting factors. Higher insufflation pressures are associated with greater hemodynamic impairment, whereas prolonged pneumoperitoneum, steep Trendelenburg positioning, obesity, and reduced cardiopulmonary reserve intensify respiratory mechanical deterioration [6], [9]–[11], [16]–[19]. In particular, the combination of pneumoperitoneum and Trendelenburg can reduce pulmonary compliance by approximately half and substantially increase the pressure required to maintain ventilation [16].

The findings also show that arterial pressure alone cannot adequately represent cardiovascular stability during pneumoperitoneum, since systemic vascular resistance may increase while cardiac output decreases [4], [6]. Similarly, increased airway pressure should be interpreted together with respiratory compliance, chest wall mechanics, positioning, and transpulmonary pressure rather than as an isolated indicator of pulmonary stress [15], [17], [19].

The physiological effects of pneumoperitoneum should therefore be understood as part of a dynamic cardiopulmonary interaction in which abdominal pressure, mechanical ventilation, patient position, CO₂ absorption, and individual characteristics act simultaneously. This interaction explains the substantial variability observed among patients and supports the need for individualized intraoperative assessment.

From a perioperative perspective, the evidence favors the use of the **lowest effective insufflation pressure compatible with adequate surgical exposure**, together with continuous hemodynamic and respiratory monitoring, appropriate adjustment of minute ventilation, individualized PEEP, careful fluid and vasoactive management, and timely modifications in positioning when clinically required [6], [18], [20].

In conclusion, pneumoperitoneum is not merely a technical component of laparoscopic surgery but a major physiological intervention capable of modifying cardiovascular performance and respiratory mechanics. A detailed understanding of these effects is essential for anticipating intraoperative deterioration, interpreting monitoring variables correctly, and optimizing anesthetic and surgical management. As laparoscopic and robotic procedures become increasingly complex and prolonged, individualized strategies based on cardiopulmonary physiology will remain fundamental for improving perioperative safety and maintaining adequate organ perfusion and ventilation.

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