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Navigating Pneumoperitoneum in Severe HFrEF: A Case Report on a Physiology-Based Anesthetic Strategy

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A B S T R A C T

Laparoscopic surgery in patients with severe heart failure with reduced ejection fraction (HFrEF) presents a formidable anesthetic challenge. The cardiovascular sequelae of pneumoperitoneum can precipitate acute hemodynamic collapse in a myocardium with minimal contractile reserve. This report details the anesthetic management of a high-risk patient with an extremely low ejection fraction undergoing laparoscopic cholecystectomy, focusing on a physiology-based approach. A 63-year-old, 72 kg male with severe HFrEF (ejection fraction 24%) and NYHA class III symptoms was scheduled for laparoscopic cholecystectomy. His ASA physical status was IV. Preoperative optimization ensured he was euvolemic and on guideline-directed medical therapy. Anesthetic induction was achieved with fentanyl (1.4 mcg/kg), atracurium (0.35 mg/kg), and ketamine (1 mg/kg). Following CO_2 insufflation, the patient developed profound bradycardia (42 bpm) and hypotension (MAP 58 mmHg). This anticipated crisis was managed with atropine and a supplemental ketamine bolus (0.3 mg/kg), successfully restoring hemodynamic stability. The procedure was completed uneventfully. In conclusion, this case demonstrates that a tailored anesthetic regimen, focused on intrinsic hemodynamic support and proactive crisis management, can be a safe and effective strategy in this high-risk cohort. The successful outcome hinged not on a single agent but on a comprehensive perioperative process encompassing meticulous optimization, a deliberate choice of anesthetic modality based on patient pathophysiology, goal-directed intraoperative therapy, and a structured transition to postoperative care.

1. Introduction

The modern perioperative landscape is increasingly defined by the complex intersection of advancing surgical technology and a patient population with a significant burden of chronic disease.[1] At the forefront of this challenge lies the anesthetic management of patients with severe heart failure with reduced ejection fraction (HFrEF) who present for major surgery. HFrEF, a clinical syndrome characterized by impaired systolic function with a left ventricular ejection fraction (LVEF) of 40% or less, represents the end stage of numerous cardiovascular pathologies.[2] Its escalating prevalence, driven by an aging population and improved survival after cardiac insults, makes it a monumental and growing concern for healthcare systems globally and a daily reality for perioperative physicians. These patients are not merely "high-risk"; they exist in a state of tenuous physiological balance, possessing a severely limited cardiovascular reserve that renders them exquisitely vulnerable to the hemodynamic fluctuations inherent to surgery and anesthesia. Any iatrogenic or surgical event that increases afterload, decreases preload, or directly depresses the myocardium can disrupt this fragile

equilibrium, leading to catastrophic outcomes such as acute decompensated heart failure, malignant arrhythmias, and cardiogenic shock.[3]

The pathophysiology of HFrEF is a study in maladaptive compensation. The initial cardiac insult, whether from ischemic heart disease, chronic hypertension, or cardiomyopathy, triggers a relentless cascade of neurohormonal activation.[4] The sympathetic nervous system (SNS) and the renin-angiotensin-aldosterone system (RAAS) are chronically over-activated in a misguided attempt to maintain cardiac output and systemic perfusion.[5] This leads to a vicious cycle of progressive cardiac dysfunction. Chronic sympathetic stimulation increases heart rate and contractility, which dangerously elevates myocardial oxygen demand on an already compromised substrate, while also being directly cardiotoxic and pro-arrhythmic. The RAAS promotes sodium and water retention, leading to volume overload, and stimulates the release of profibrotic mediators that drive adverse cardiac remodeling.[6] The ventricle dilates, becomes more spherical, and its walls, laden with fibrotic tissue, become less compliant. This results in a heart that is not only a weak pump (systolic dysfunction) but also a stiff and inefficient chamber (diastolic dysfunction). The relationship between the ventricle and the arterial system—termed ventricular-arterial coupling—becomes profoundly inefficient. The failing ventricle cannot effectively transfer energy to the arterial circulation, meaning a disproportionate amount of cardiac work is wasted. This patient is therefore precariously dependent on an adequate preload to stretch the myocytes to a favorable point on a flattened Frank-Starling curve, yet is simultaneously pathologically intolerant of any increase in afterload, against which it cannot effectively eject.

Into this high-stakes physiological arena enters the unique paradox of modern minimally invasive surgery. Laparoscopy has revolutionized the surgical field, offering profound benefits in reducing postoperative pain, decreasing pulmonary complications, and accelerating recovery.[7] However, the physiological cost of entry—the carbon dioxide (CO_2) pneumoperitoneum—is steep and creates a uniquely hostile environment for the HFrEF patient. The insufflation of the abdomen to pressures of 12-15 mmHg creates a state of controlled, iatrogenic abdominal compartment syndrome. The hemodynamic consequences are immediate and profound. The increased intra-abdominal pressure mechanically compresses the abdominal aorta and splanchnic arterioles, causing an acute and dramatic increase in systemic vascular resistance (SVR), which directly elevates left ventricular afterload.[8] For a healthy heart, this is a manageable stressor. For the failing ventricle with an LVEF of 24%, this is akin to clamping the aorta. Simultaneously, the respiratory effects are significant. The cephalad displacement of the diaphragm by the insufflated abdomen dramatically reduces functional residual capacity and pulmonary compliance.[9] This leads to atelectasis, ventilation-perfusion mismatching, and potential hypoxemia. Furthermore, the transperitoneal absorption of CO_2 imposes a significant acid-base challenge. If not met with a compensatory increase in minute ventilation, this will lead to hypercarbia and respiratory acidosis, which have direct negative inotropic effects, further depressing myocardial function and lowering the threshold for arrhythmias. The confluence of these factors—a massive afterload increase, unpredictable preload changes, vagal stimulation, hypercarbia, acidosis, and decreased pulmonary reserve—creates a perfect storm of physiological insults, all directed at a cardiovascular system with no capacity to compensate.

This clinical scenario presents a profound challenge to the anesthesiologist. Standard anesthetic techniques, particularly those reliant on potent vasodilating and cardiodepressant agents like propofol or high concentrations of volatile anesthetics, are often profoundly destabilizing and thus contraindicated.[10] The critical challenge, therefore, is to abandon a "one-size-fits-all" approach and instead design and execute a comprehensive, bespoke perioperative plan that actively shields the failing heart from these predictable

insults. While the principle of using hemodynamically stable anesthetic agents is well-established, detailed, granular reports illustrating the step-by-step anticipation and management of predictable intraoperative hemodynamic crises in HFrEF patients undergoing laparoscopy are less common in the literature. This report aims to fill this gap by presenting the case of a 63-year-old male with an LVEF of 24% who successfully underwent laparoscopic cholecystectomy. The novelty of this report is not merely in its description of using a specific drug, but in its detailed, real-world validation of a holistic, physiology-based management strategy. The primary aim of this manuscript is to deconstruct this comprehensive strategy, exploring in depth the intellectual and clinical rationale behind the choice of anesthetic modality over viable alternatives, the philosophy of proactive goal-directed hemodynamic therapy, and the critical importance of viewing the patient's journey as an uninterrupted continuum of perioperative care. We seek to illustrate how a management plan built upon a deep, mechanistic understanding of the patient's specific pathophysiology can facilitate a successful outcome in an extreme-risk clinical scenario, thereby offering valuable insights for clinicians facing this increasingly common and formidable challenge.

2. Case Presentation

A 63-year-old, 72 kg male with a body mass index (BMI) of 26.5 kg/m² was scheduled for an elective laparoscopic cholecystectomy for symptomatic cholelithiasis. His medical history was significant for long-standing hypertensive heart disease that had progressed to severe HFrEF, with a recent LVEF documented at 24% and corresponding New York Heart Association (NYHA) Class III symptoms of fatigue and dyspnea with minimal exertion. His significant comorbidities included type 2 diabetes mellitus controlled with oral hypoglycemics, chronic kidney disease stage 3, and chronic anemia of inflammation. A summary of his baseline characteristics is provided in Figure 1. Given the extreme-risk nature of the procedure, a formal multidisciplinary meeting was convened, involving the general surgery, cardiology, and anesthesiology teams. The discussion centered on a comprehensive risk-benefit analysis. Alternative, lower-risk management strategies, most notably percutaneous cholecystostomy, were extensively considered. However, due to the patient's recurrent biliary colic and the less definitive nature of a percutaneous drain, the consensus decision was to proceed with laparoscopic cholecystectomy following a rigorous period of medical optimization. This optimization phase focused on two key goals: ensuring the patient was on maximally tolerated guideline-directed medical therapy (GDMT) for his heart failure and confirming a state of clinical euvolemia. His GDMT consisted of a beta-blocker (Bisoprolol), an ACE inhibitor (Ramipril), and a loop diuretic (Furosemide). His volume status was confirmed as optimal through a combination of clinical examination revealing no peripheral edema or elevated jugular venous pressure, and a preoperative bedside lung ultrasound that demonstrated no evidence of interstitial edema (A-lines present, no B-lines). His final assigned American Society of Anesthesiologists (ASA) physical status was IV.

Upon arrival in the operating room, standard ASA monitors (5-lead ECG with continuous ST-segment analysis, pulse oximetry, non-invasive blood pressure cuff, temperature) were applied. A 20-gauge left radial arterial line was placed under local anesthesia for continuous, beat-to-beat hemodynamic monitoring. After a period of pre-oxygenation with 100% oxygen, a general anesthetic was induced. The complete timeline of the anesthetic induction, maintenance, intraoperative events, and key management decisions is detailed in Figure 2. The induction sequence was executed with the primary goal of ensuring hemodynamic stability. Following this, the patient's trachea was intubated, and mechanical ventilation was initiated with a lung-protective strategy. Anesthesia was maintained with a low concentration of sevoflurane to minimize cardiodepressive effects. The intraoperative course was marked by a

predictable, profound, but well-managed hemodynamic crisis immediately following peritoneal insufflation and placement in the Trendelenburg position. This was characterized by a precipitous drop in heart rate and blood pressure, as illustrated graphically in Figure 2. The crisis was managed with pre-planned pharmacological interventions. The remainder of the 75-minute procedure was hemodynamically stable. Fluid management was highly conservative, with a total of 400 mL of a balanced crystalloid solution administered against a minimal estimated blood loss (<50 mL) and a urine output of 50 mL. Neuromuscular blockade was reversed with sugammadex to ensure a smooth emergence with minimal risk of residual paralysis. Figure 2 illustrates the patient's heart rate (HR) and mean arterial pressure (MAP) over time. Key events are annotated, showing the baseline stability, the sharp decline upon CO_2 insufflation, and the successful restoration of hemodynamics following intervention with atropine and ketamine.

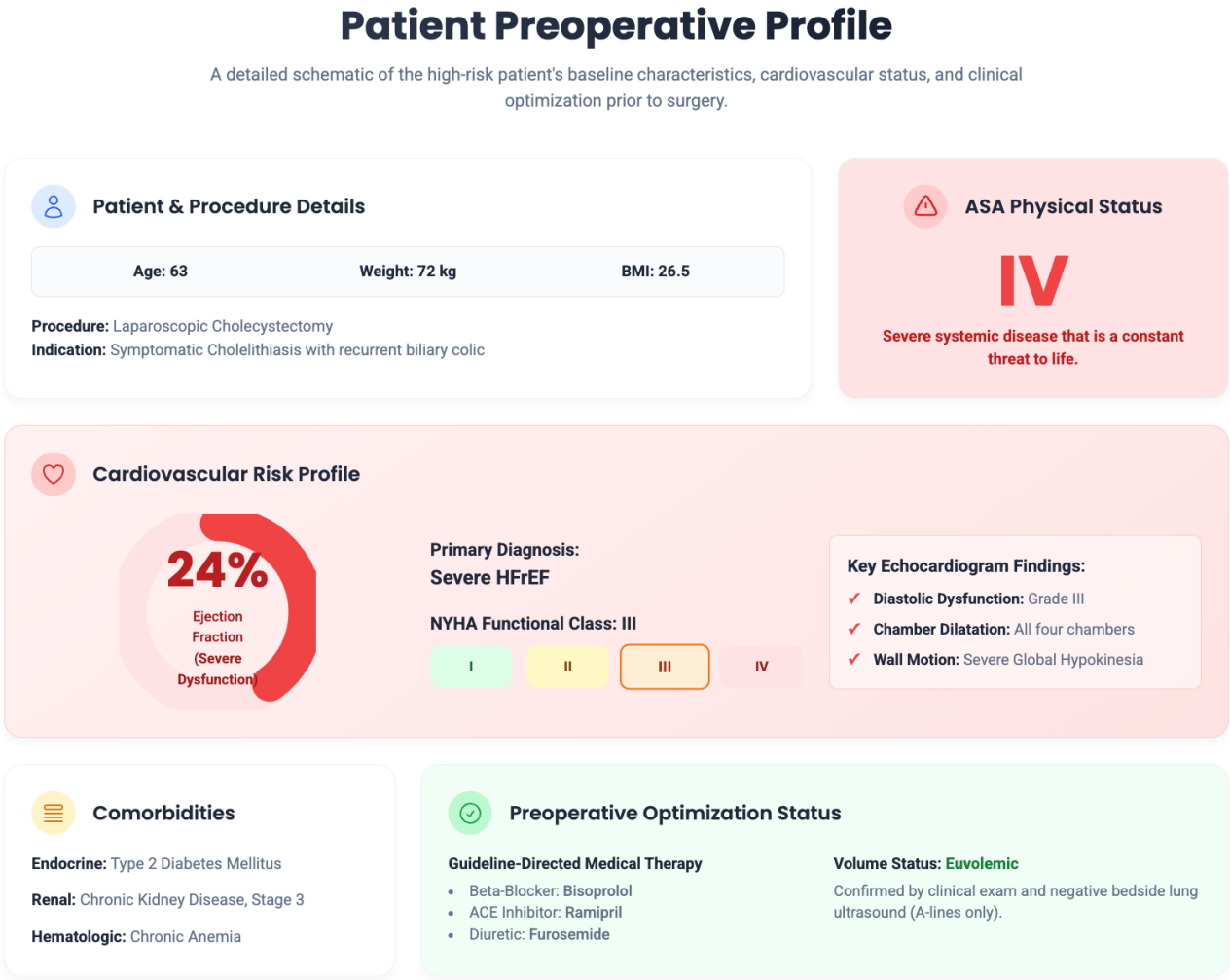

Figure 1. Summary of patient demographics and preoperative optimization.

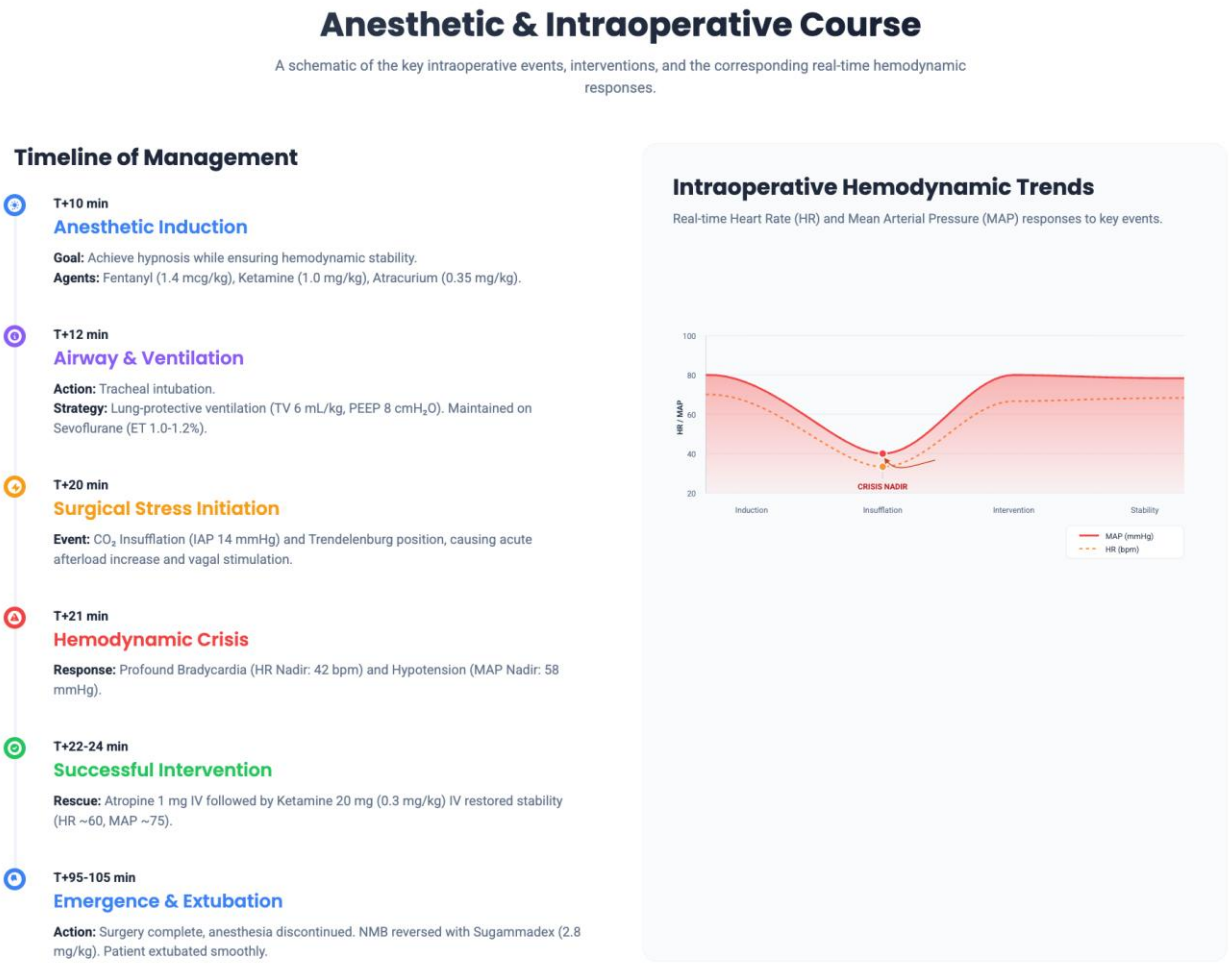

Figure 2. Timeline of anesthetic and intraoperative management.

Figure 2 provides a detailed schematic and graphical chronology of the anesthetic and intraoperative management, serving as a visual narrative of a high-stakes physiological challenge. This period represents the crucible of the entire perioperative plan, where extensive preoperative preparation is tested against the predictable and profound stresses of the surgical procedure. The timeline deconstructs the case into discrete, critical phases, each associated with a specific goal, a set of pharmacological or mechanical interventions, and a resulting hemodynamic response. This granular view allows for a deep, scientific analysis of the clinical decision-making process in real-time. The initial phase, Anesthetic Induction (T+10 min), was arguably the most perilous moment of the procedure. The primary goal was to achieve a state of hypnosis and amnesia without precipitating the cardiovascular collapse that would be expected with conventional anesthetic agents in a patient with an ejection fraction of 24%. The chosen agents—Fentanyl, Ketamine, and Atracurium—were selected for their synergistic effects and unique physiological profiles. Fentanyl was administered to blunt the intense sympathetic stress response to laryngoscopy and intubation, a reflex that could have dangerously increased myocardial oxygen demand. Atracurium provided reliable neuromuscular blockade. The cornerstone of the induction, however, was a carefully titrated dose of ketamine (1.0 mg/kg), chosen for its intrinsic sympathomimetic properties, which actively supported the patient's tenuous hemodynamics. Following a smooth induction, the

Airway & Ventilation (T+12 min) phase was established. The successful, atraumatic intubation was followed by the immediate implementation of a lung-protective ventilation strategy. This approach, utilizing a low tidal volume (6 mL/kg) and a moderate level of positive end-expiratory pressure (PEEP of 8 cmH_2O), was not incidental. It was a deliberate strategy to minimize ventilator-induced lung injury and, crucially, to mitigate the detrimental effects of high intrathoracic pressures on venous return—a critical consideration in a patient exquisitely dependent on adequate preload. Anesthesia was maintained with a low end-tidal concentration of sevoflurane to minimize its dose-dependent cardiodepressive and vasodilatory effects. The inflection point of the entire procedure occurred at the Surgical Stress Initiation (T+20 min). The insufflation of the peritoneal cavity with carbon dioxide to an intra-abdominal pressure of 14 mmHg, combined with the placement of the patient in a steep Trendelenburg position, represented a multi-faceted physiological assault. This maneuver acutely and dramatically increased systemic vascular resistance (afterload), mechanically compressed the great vessels, and triggered a potent vagal nerve-mediated cardioinhibitory reflex. As anticipated, this culminated in a Hemodynamic Crisis (T+21 min). The graphical representation of the intraoperative hemodynamic trends vividly captures this moment: a precipitous and simultaneous decline in both heart rate and mean arterial pressure to life-threatening levels (HR nadir of 42 bpm, MAP nadir of 58 mmHg). The subsequent phase, Successful Intervention (T+22-24 min), demonstrates the efficacy of a pre-planned, physiology-based rescue strategy. The management was not a panicked reaction but a choreographed sequence of targeted interventions. First, the profound, vagally-mediated bradycardia was immediately corrected with atropine, an anticholinergic agent that blocks the cardioinhibitory effects of the vagus nerve. This was followed by a small supplemental bolus of ketamine (0.3 mg/kg) to address the persistent hypotension. This choice was deliberate; unlike a pure vasopressor which would have further increased the already dangerously high afterload, ketamine provided balanced inotropic and chronotropic support, restoring blood pressure without placing undue strain on the failing ventricle. As the graph clearly shows, these interventions resulted in the successful restoration of hemodynamic stability to a safe and physiologically optimal range. The final phase, Emergence & Extubation (T+95-105 min), completed the intraoperative journey. The use of sugammadex for neuromuscular blockade reversal ensured a rapid, reliable, and complete return of muscle function, facilitating a smooth and calm emergence. The patient was successfully extubated in the operating room, a testament to the stability achieved and the careful titration of all anesthetic agents, which avoided profound postoperative sedation or respiratory depression. This controlled and stable conclusion to the intraoperative period was critical for ensuring a seamless and safe transition to the intensive care unit, setting the stage for the patient's ultimately successful recovery.

A structured, closed-loop communication handover was provided to the receiving Intensive Care Unit (ICU) team. The handover emphasized the patient's extreme cardiac risk, the details of the intraoperative course, and the predefined postoperative management goals. The postoperative plan, summarized in Figure 3, was centered on maintaining the physiological stability achieved in the operating room. The patient's ICU course was uneventful. He remained hemodynamically stable without any requirement for vasoactive or inotropic support. His pain was well-controlled with the multimodal regimen, and he was weaned from supplemental oxygen within 12 hours. Having met all predefined ICU discharge criteria (including hemodynamic stability, adequate pain control, and no signs of cardiac or pulmonary decompensation), he was discharged to the surgical ward after 24 hours of observation. His recovery continued smoothly, and he was subsequently discharged home on the third postoperative day.

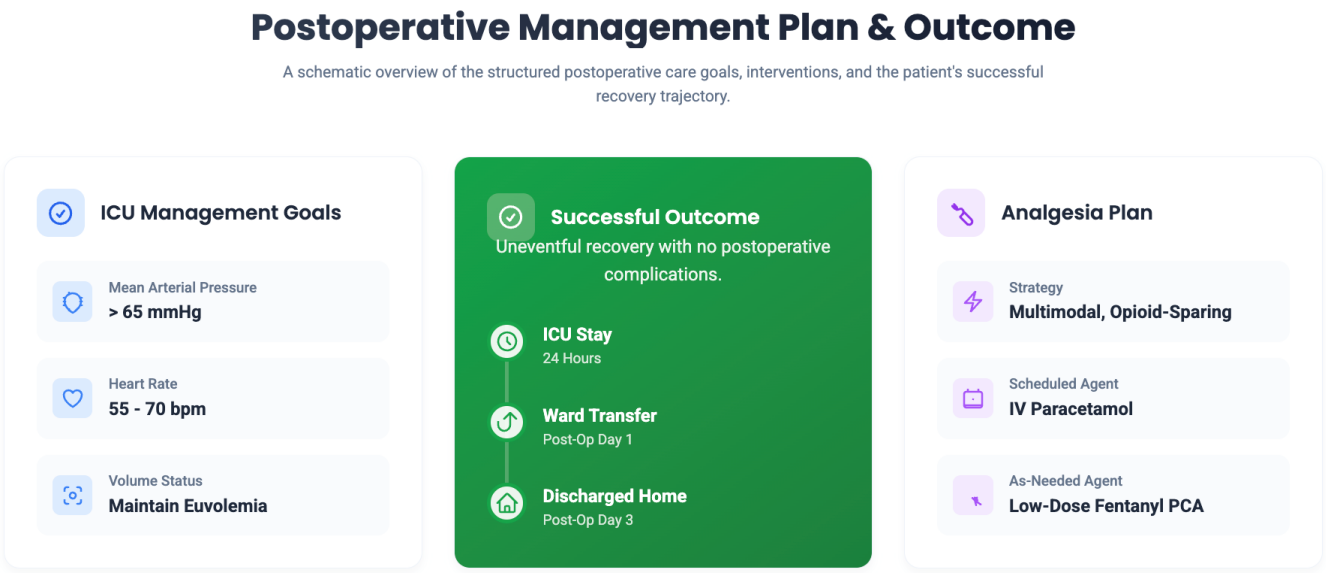

Figure 3. Postoperative management plan and outcome.

Figure 3 provides a comprehensive schematic overview of the final, critical phase of the patient's perioperative journey: the postoperative management and ultimate outcome. This phase is as crucial as the intricate intraoperative management, as it is during this period that the patient is most vulnerable to complications such as fluid shifts, cardiac decompensation, and respiratory failure. The figure deconstructs the postoperative plan into its core components—the specific, goal-directed therapies implemented in the Intensive Care Unit (ICU), the multimodal analgesic strategy, and the resulting successful recovery trajectory. It serves as a testament to the principle that a successful surgical outcome is not merely the absence of an intraoperative catastrophe, but the culmination of a meticulously planned and executed continuum of care. The first panel, ICU Management Goals, details the predefined physiological targets that guided the patient's care immediately following transfer from the operating room. These were not arbitrary numbers, but carefully selected parameters designed to maintain the delicate hemodynamic stability achieved during surgery and to provide the optimal physiological environment for recovery. The goal to maintain a Mean Arterial Pressure (MAP) > 65 mmHg was chosen to ensure adequate perfusion to vital end-organs, particularly the brain and the patient's already compromised kidneys, without imposing excessive afterload on the struggling left ventricle. The target Heart Rate of 55-70 bpm was equally critical; this range of "slight bradycardia" was intended to maximize diastolic filling time, which is essential for both coronary artery perfusion and for optimizing stroke volume in a heart with severe diastolic dysfunction. Finally, the directive to Maintain Euvolemia was perhaps the most crucial goal, requiring a vigilant and continuous assessment to prevent both fluid overload, which could precipitate pulmonary edema, and hypovolemia, which would have led to a collapse in preload and cardiac output. The fact that these goals were achieved without any need for vasoactive or inotropic support is a key indicator of the success of the intraoperative management. The second panel, the Analgesia Plan, highlights a cornerstone of modern, enhanced recovery pathways. The strategy was explicitly Multimodal and Opioid-Sparing. This approach is particularly vital in a patient with severe cardiac compromise. Uncontrolled postoperative pain would have triggered a deleterious stress response—

tachycardia and hypertension—dramatically increasing myocardial oxygen demand and potentially leading to ischemia. However, relying solely on high doses of opioids for pain control would have introduced the significant risks of respiratory depression, sedation, and gastrointestinal dysfunction. The chosen regimen elegantly balanced these risks by using scheduled, non-opioid IV Paracetamol as the foundational analgesic, supplemented by a patient-controlled analgesia (PCA) pump delivering very small, as-needed doses of Fentanyl. This provided the patient with excellent pain control while minimizing systemic opioid exposure and its associated adverse effects. The central and most prominent panel, Successful Outcome, serves as the summary of this entire process. It confirms that the patient's recovery was uneventful and free of any postoperative complications. The visual timeline within this panel—the Recovery Trajectory—provides a clear and concise summary of the patient's efficient and successful journey through the final stages of their hospital stay. The initial 24-hour ICU stay allowed for intensive monitoring during the most vulnerable period. This was followed by a safe transfer to the surgical ward on Postoperative Day 1, indicating that the patient was stable and no longer required intensive intervention. Finally, the patient was discharged home on Postoperative Day 3, a remarkably swift recovery for a patient with such an extreme level of underlying cardiac disease. This successful and rapid trajectory is the ultimate validation of the comprehensive, physiology-based, and multidisciplinary approach that was applied throughout the entire perioperative period.

3. Discussion

This case report provides a detailed validation of a comprehensive, physiology-based perioperative strategy for managing an extreme-risk HFrEF patient undergoing laparoscopic surgery. The successful outcome was not the result of a single drug or action, but rather the culmination of a deliberate, intellectually rigorous process that spanned the entire perioperative continuum.[11] This discussion will deconstruct this process, focusing on four core pillars: the foundational role of the perioperative care continuum, the critical justification for the chosen anesthetic modality over viable alternatives, the philosophy of proactive, goal-directed hemodynamic management, and a deep, mechanistic analysis of the intraoperative pharmacological and ventilatory strategy. The intraoperative management, while being the most dramatic phase of care, cannot be viewed in isolation.[12] Its success was predicated on a foundation of meticulous preparation and a clear vision for postoperative recovery. The decision to proceed with surgery was itself a carefully considered outcome of a multidisciplinary team discussion, which is the standard of care for such high-risk cases. This collaborative approach ensures that all therapeutic avenues are explored and that the perioperative plan is aligned across specialties. The cornerstone of the preoperative phase was optimization. Ensuring the patient was on maximal, guideline-directed medical therapy was not a passive check-box exercise; it ensured that his neurohormonal milieu was maximally suppressed and his failing ventricle was pharmacologically supported to the greatest extent possible. Even more critically, the confirmation of clinical euvolemia was paramount. A fluid-overloaded patient would have entered the operating room with an elevated left ventricular end-diastolic pressure, perilously close to the threshold for pulmonary edema, which the acute afterload increase from pneumoperitoneum would have almost certainly precipitated.[13] Conversely, an under-filled patient would have been unable to tolerate the anesthetic-induced vasodilation or the potential drop in venous return, leading to profound hypotension. The use of bedside lung ultrasound to confirm the absence of interstitial edema is a key example of employing modern, non-invasive tools to refine clinical assessment and increase confidence in the patient's physiological readiness for surgery.[14] This continuum of care extended seamlessly into the postoperative period. A vague plan to "transfer to ICU" is insufficient.

In this case, a structured handover with clearly defined hemodynamic and analgesic goals was essential. This ensured that the physiological stability so carefully curated in the operating room was maintained. The choice of a multimodal, opioid-sparing analgesic regimen was deliberate. Pain-induced tachycardia and hypertension would have dangerously increased myocardial oxygen consumption.[15] By providing a baseline of non-opioid analgesia with paracetamol and allowing the patient to self-titrate small doses of fentanyl, excellent pain control was achieved while minimizing the risks of respiratory depression, sedation, and opioid-related side effects that could have stressed the patient's limited reserve. This holistic approach, viewing the patient's care not as discrete phases but as a continuous, integrated process, was fundamental to the successful outcome. The choice of a general anesthetic-only technique in a patient so exquisitely sensitive to afterload warrants a critical and detailed examination, particularly when contrasted with a combined general-epidural anesthetic. A carefully titrated thoracic epidural offers profound and, in many ways, ideal physiological benefits for the failing heart. By inducing a controllable, segmental sympatholysis, it directly addresses the core pathophysiological problem: an inability to tolerate increased afterload. A thoracic epidural would have reduced the patient's baseline systemic vascular resistance and, in theory, could have powerfully blunted the massive SVR increase from pneumoperitoneum, effectively preempting the primary hemodynamic insult. This afterload reduction is a cornerstone of managing acute-on-chronic heart failure and could have improved the critical relationship between the ventricle and the arterial system, known as ventricular-arterial coupling.[16] However, the decision to omit neuraxial anesthesia in this specific case was the result of a careful, multifactorial risk-benefit analysis. While afterload reduction is desirable, the profound vasodilation from an epidural can lead to significant and difficult-to-manage hypotension in a patient who is also exquisitely preload-dependent due to severe diastolic dysfunction. An abrupt drop in venous return from venous pooling could have been as catastrophic as the afterload increase. The primary concern was the management of this "fixed-output" state, where both preload and afterload must be maintained within a very narrow therapeutic window.[17] This can be exceptionally challenging with the relatively fixed and long-lasting sympathectomy of an epidural. The potential for a "high block" and the ensuing profound bradycardia and hypotension would have left few rescue options. Secondly, the GA-only approach, utilizing rapidly titratable intravenous and inhaled agents, offered far greater minute-to-minute pharmacologic control and reversibility. It allowed the clinical team to anticipate and plan for specific, transient events. The plan was not just to induce anesthesia, but to be prepared to directly counter the anticipated vagal bradycardia with an anticholinergic and to actively support blood pressure with a sympathomimetic agent (ketamine) during the most intense period of surgical stress. This approach was deemed to provide a more adaptable and reversible strategy to navigate the rapid and profound hemodynamic swings expected during the brief but intense period of pneumoperitoneum. Once the abdomen was desufflated, the primary insult would be removed, and a long-acting epidural would no longer have been necessary and could have complicated postoperative fluid management.

Modern high-risk anesthesia has evolved beyond the simplistic, reactive normalization of vital signs. The philosophy underpinning this patient's management was one of proactive, goal-directed therapy.[18] The decision to proceed with standard ASA monitoring plus an arterial line, without more advanced cardiac output monitoring, was a deliberate choice based on a risk-benefit analysis for this specific, relatively short (<90 minute) procedure. The known risks associated with placing a central venous catheter (infection, pneumothorax) or a pulmonary artery catheter (arrhythmias, vessel rupture) were felt to outweigh the potential benefits, provided a clear set of physiologically-informed goals was established

beforehand. Advanced monitoring with a PAC or even a minimally invasive cardiac output monitor would have provided more data, but the fundamental management strategy would have remained the same: supporting perfusion while avoiding insults. The primary goal was not merely to "treat hypotension," but to maintain the mean arterial pressure (MAP) within 20% of the patient's optimized preoperative baseline. This baseline, established when the patient was in his best possible clinical state, served as the validated target for adequate end-organ perfusion. Equally important was the avoidance of excessive tachycardia that would drastically increase myocardial oxygen demand and shorten diastolic time, compromising coronary perfusion. The management of the intraoperative crisis exemplifies this goal-directed approach. The profound bradycardia was not just treated to achieve a "normal" heart rate. It was corrected to a state of "slight bradycardia" in the target range of 55-65 bpm. This specific target was chosen because a slower heart rate is known to prolong diastolic filling time, a critical factor for both coronary artery perfusion and for optimizing stroke volume in the stiff, non-compliant ventricle characteristic of this patient's Grade III diastolic dysfunction. The subsequent hypotension was treated not with a pure alpha-agonist vasopressor like phenylephrine, which would have dangerously increased afterload and worsened ventricular-arterial uncoupling, but with ketamine, which provided balanced inotropic and chronotropic support.[19] This demonstrates a management strategy where every intervention was aimed at achieving a specific, predefined physiological state, rather than simply normalizing a number on the monitor.

The intraoperative plan was a carefully choreographed sequence of pharmacological and physiological interventions designed to support the failing heart at every step. The choice of ketamine as the primary induction agent was central to this strategy.[20] A detailed pharmacological analysis reveals why it was superior to other options for this patient. While propofol would have caused catastrophic vasodilation and myocardial depression, the more common alternative for cardiac instability, etomidate, also presents issues. Etomidate maintains hemodynamic stability by preserving SVR, but it offers no inotropic support and is associated with significant adrenal suppression, which can be detrimental even after a single dose. Ketamine, in contrast, provides a unique combination of effects. Its indirect sympathomimetic action, via norepinephrine reuptake inhibition, actively supports heart rate, contractility, and blood pressure. This effect was critical for improving the patient's ventricular-arterial coupling—the efficiency of energy transfer from the ventricle to the arterial system—at a time when the system was about to be stressed by a massive afterload increase. This choice was not without a careful consideration of ketamine's potential downsides. Its sympathomimetic effects invariably increase myocardial oxygen consumption (MVO_2). This risk was weighed against the profound benefit of maintaining coronary perfusion pressure (diastolic blood pressure minus LVEDP). In this case, allowing permissive hypotension to avoid an increase in MVO_2 would have been far more dangerous. The risk was mitigated by continuous multi-lead ST-segment analysis, which showed no signs of ischemia throughout the case. Another concern is ketamine's potential to increase pulmonary vascular resistance (PVR). In a patient with severe left-sided heart failure, some degree of secondary pulmonary hypertension is likely. However, the risk was deemed low in the absence of known severe right ventricular dysfunction, and the benefit of maintaining systemic perfusion and right ventricular coronary perfusion pressure was prioritized. The ventilatory management strategy was an equally important, though less dramatic, component of the plan. A lung-protective strategy with a low tidal volume (6 mL/kg) was used to minimize ventilator-induced lung injury and reduce the detrimental effects of high intrathoracic pressures on venous return. The use of a relatively high level of PEEP (8 cmH_2O) was crucial. It served to prevent atelectasis, especially when the patient was in the Trendelenburg position, which

reduces functional residual capacity, thereby improving oxygenation and minimizing shunt. During insufflation, the respiratory rate was proactively increased to manage the absorbed CO_2 load. This prevented the development of a significant respiratory acidosis, which would have had a direct negative inotropic effect, further depressing myocardial function, and could have potentiated arrhythmias.

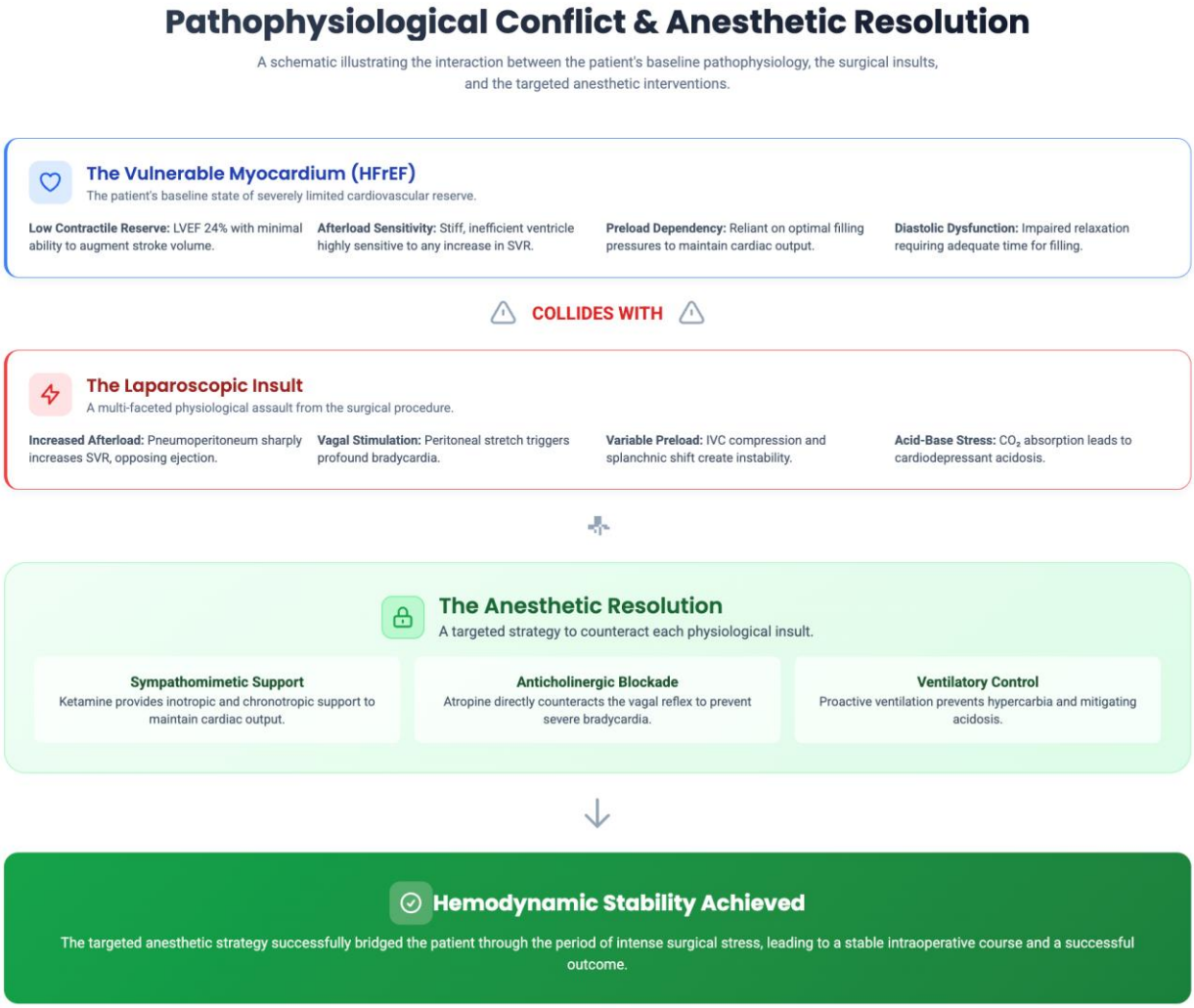

Figure 4. Pathophysiological conflict & anesthetic resolution.

Figure 4 presents a conceptual schematic that distills the core narrative of this entire case report into a single, comprehensive visual. It moves beyond a simple timeline of events to illustrate the fundamental and dynamic conflict between the patient's severely compromised baseline physiology and the multi-faceted insults imposed by the surgical procedure. More importantly, it demonstrates how a targeted, physiology-based anesthetic strategy acted as a decisive mediating force, neutralizing these threats and ultimately leading to a successful outcome. The figure is structured as a logical progression, detailing the initial state, the impending collision of opposing forces, the specific interventions of the Anesthetic Shield, and the final, stable resolution. The schematic begins with the Vulnerable Myocardium, which defines the patient's precarious baseline state. This is a heart defined by profound limitations. With a Low

Contractile Reserve (LVEF 24%), the ventricle has a critically impaired ability to increase its stroke volume in response to stress. It is characterized by extreme Afterload Sensitivity, meaning any increase in the resistance against which it must pump will cause a precipitous fall in cardiac output. Concurrently, it exhibits a strong Preload Dependency, requiring optimal filling volumes to even maintain its limited function on a flattened Frank-Starling curve. Compounding these systolic issues is severe Diastolic Dysfunction, an impaired ability to relax and fill, which makes the heart critically dependent on adequate time (a slower heart rate) for both ventricular filling and coronary perfusion. This section establishes the high-stakes battlefield: a cardiovascular system with virtually no reserve capacity, balanced on a knife's edge. Descending from this baseline is The Laparoscopic Insult, which represents the array of physiological assaults launched by the surgical procedure. These are not random complications but predictable, inherent consequences of the technique. The most significant is the Increased Afterload, a direct result of the CO_2 pneumoperitoneum sharply increases systemic vascular resistance (SVR) and creates an obstacle that the failing ventricle cannot overcome. This is compounded by potent Vagal Stimulation from peritoneal stretching, which threatens to cause profound, life-threatening bradycardia. Furthermore, the procedure introduces Variable Preload through a combination of vena caval compression and splanchnic blood volume shifts, creating instability. Finally, the systemic absorption of CO_2 creates an Acid-Base Stress, leading to a hypercarbic and acidotic state that can directly depress myocardial contractility. The schematic visually represents these distinct but simultaneous insults converging on the vulnerable myocardium, setting the stage for the inevitable hemodynamic crisis. At the fulcrum of this conflict is The Anesthetic Resolution. This section details the specific, targeted interventions that were designed not merely to provide anesthesia, but to act as a physiological shield against the laparoscopic insults. It was a multi-pronged defense. Sympathomimetic Support, delivered via ketamine, provided essential inotropic and chronotropic stimulation, actively augmenting the heart's contractility to help it overcome the high afterload and maintain cardiac output. Anticholinergic Blockade, via atropine, served to directly counteract the powerful vagal reflex, preventing a catastrophic drop in heart rate. Finally, proactive Ventilatory Control was employed to manage the absorbed CO_2 load, preventing the development of a severe respiratory acidosis and thereby mitigating its direct cardiodepressant effects. The final panel, Hemodynamic Stability Achieved, represents the successful culmination of this strategy. By accurately predicting the nature of the physiological conflict and deploying a set of specific, targeted countermeasures, the anesthetic plan successfully neutralized the threats. The schematic demonstrates that the patient was safely bridged through the period of most intense surgical stress. This led to a stable intraoperative course and, ultimately, a successful postoperative outcome, validating the entire physiology-based approach.

4. Conclusion

The anesthetic management of patients with severe HFrEF for laparoscopic surgery is a high-stakes endeavor that demands a comprehensive, multidisciplinary perioperative strategy. This case report illustrates the successful application of such a strategy, demonstrating that by anticipating predictable physiological insults and implementing a proactive, goal-directed management plan, it is possible to safely guide these critically ill patients through the profound challenges of minimally invasive surgery. The successful outcome hinged not on a single agent or a magic bullet, but on an integrated process. This process began with meticulous preoperative optimization, was executed through a deliberate choice of anesthetic modality based on the patient's specific pathophysiology, was guided by proactive intraoperative hemodynamic management, and was completed by a seamless transition to a

structured, goal-oriented postoperative care plan. This case serves as a powerful example of this process in action, underscoring the principle that individualized, physiology-based, and holistically planned.

5. References

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