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Optimizing Postoperative Hemodynamics After CABG: The Role of NT-proBNP in Multimodal Monitoring and Targeted Therapy

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ABSTRACT

Postoperative hypotension is a common and critical complication following coronary artery bypass grafting (CABG) that contributes to organ dysfunction and increased mortality. Traditional hemodynamic monitoring methods often fall short in distinguishing the underlying causes of hypotension in complex cardiac patients. N-terminal pro-B-type natriuretic peptide (NT-proBNP), released in response to ventricular wall stress, correlates with left ventricular filling pressures and serves as a valuable biomarker for guiding hemodynamic management after CABG. Elevated perioperative NT-proBNP levels predict adverse outcomes such as low cardiac output syndrome, renal dysfunction, and prolonged intensive care unit stay. When integrated with dynamic parameters like stroke volume variation (SVV) and pulse pressure variation (PPV), alongside echocardiographic data, NT-proBNP enhances diagnostic accuracy and supports individualized therapy. A structured algorithm incorporating NT-proBNP facilitates tailored administration of fluids, inotropes, and vasopressors. The use of NT-proBNP as an adjunct to conventional monitoring provides real-time insight into myocardial stress and volume tolerance, potentially improving clinical outcomes, resource utilization, and patient safety in post-CABG care.

KEYWORDS: Postoperative hemodynamics, Coronary artery bypass grafting (CABG), NT-proBNP, Hemodynamic monitoring, Stroke volume variation (SVV), Pulse pressure variation (PPV), Critical care cardiology, Biomarkers in cardiac surgery

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INTRODUCTION

Coronary artery bypass grafting (CABG) is a fundamental treatment for patients with complex coronary artery disease, especially when optimal medical therapy or percutaneous intervention fails [1]. Despite advancements in surgical techniques and perioperative care, hemodynamic instability, particularly postoperative hypotension, remains a prevalent and significant complication. This condition elevates the risk of organ hypoperfusion, acute kidney injury, extended ICU stay, and mortality [2,3].

Postoperative hypotension can occur due to cardiogenic dysfunction, hypovolemia, vasoplegia, or mechanical factors like tamponade. The prompt identification of the underlying mechanism is essential, as therapeutic interventions vary considerably based on the etiology [4]. Traditional hemodynamic monitoring methods, such as central venous pressure (CVP), mean arterial pressure (MAP), and pulmonary artery catheterization, provide important insights but exhibit limitations in dynamic clinical settings [5].

N-terminal pro–B-type natriuretic peptide (NT-proBNP) has recently been recognized as a valuable biomarker for assessing myocardial wall stress and ventricular filling pressure. This metric possesses significant diagnostic and prognostic importance in multiple cardiovascular disorders and has shown promise in directing hemodynamic management following CABG [6–8].

This review provides an evidence-based analysis of cardiovascular physiology, monitoring modalities, the role of NT-proBNP, and its incorporation into multimodal hemodynamic management strategies in the postoperative care of CABG patients.

THE FRANK-STARLING LAW

Comprehending the physiological principles underlying cardiovascular function is crucial for interpreting hemodynamic disturbances following cardiac surgery. The Frank–Starling mechanism is fundamental to circulatory regulation, as it sustains cardiac output by enabling

cardiac muscle fibers to adjust contractility in response to variations in preload. An increase in venous return elongates myocardial fibers during diastole, leading to improved actin–myosin cross-bridge formation and more robust systolic contraction, up to a physiological limit [9].

This autoregulatory mechanism allows the heart to adjust to different blood volumes independently of external influences. The slope of the Frank–Starling curve may be diminished in pathological conditions including myocardial ischemia, ventricular hypertrophy, and systolic or diastolic heart failure, frequently observed in patients undergoing CABG [10]. In these contexts, increases in preload do not result in significant enhancements in stroke volume, a crucial factor in determining the potential benefits of fluid administration.

The hemodynamic response to fluid resuscitation is variable and specific to each patient. This highlights the significance of dynamic, individualized assessments over dependence on static preload indicators like central venous pressure [11]. A comprehensive understanding of Frank–Starling dynamics necessitates consideration of afterload, myocardial contractility, heart rate, and neurohormonal factors. The interaction of these factors is complex and occasionally counterintuitive, especially in patients with pre-existing cardiac dysfunction and those recently subjected to cardiopulmonary bypass [5].

Anesthesia, surgical trauma, and extracorporeal circulation result in significant alterations in vascular tone, circulating volume, and myocardial function. Inflammatory mediators released during and following cardiopulmonary bypass (CPB) modify endothelial permeability and vascular resistance, resulting in volume redistribution and vasoplegia. The effects frequently compromise the Frank–Starling mechanism's efficacy, restricting its predictive value unless combined with real-time dynamic evaluations [12].

POST-CABG HEMODYNAMICS

Hemodynamic stability following CABG is influenced by various interrelated factors. Determinants can be of physiological, pathological, or pharmacological origin, frequently coexisting within the same patient and increasing clinical complexity.

Age significantly influences hemodynamic responses. Elderly patients demonstrate decreased baroreceptor sensitivity, heightened vascular stiffness, and reduced beta-adrenergic responsiveness, which collectively hinder compensatory mechanisms in hypotensive states [13]. Gender may influence vascular tone and autonomic responses; research indicates that female patients demonstrate higher vascular reactivity and potentially distinct responses to fluid loading and vasopressors [14].

The autonomic nervous system and hormonal mediators, including catecholamines, vasopressin, renin–angiotensin, and natriuretic peptides, significantly affect vascular tone, heart rate, and sodium–water retention. In critical care environments, these systems frequently exhibit dysregulation as a result of sedation, surgical stress, or pharmacological interference, leading to hemodynamic instability [5].

Patients undergoing CABG often present with comorbidities, including left ventricular dysfunction, previous infarction, valvular disease, or pulmonary hypertension. These conditions reduce cardiac reserve and restrict the capacity to increase output in response to volume or pharmacologic stimulation [1]. Atrial fibrillation, frequently observed post-CABG, abolishes the atrial kick and can decrease preload by 20–30%, thereby worsening hypotension [15].

Low cardiac output syndrome (LCOS) represents a significant manifestation of postoperative cardiac dysfunction. It is generally characterized by the requirement for inotropic or mechanical assistance to sustain sufficient perfusion, occurring in roughly 3–15% of CABG patients [3]. The pathophysiology includes myocardial stunning, ischemia-reperfusion injury, microvascular dysfunction, and systemic inflammation.

Additional contributors encompass bleeding, pericardial tamponade, and pulmonary complications. Blood loss during or following surgery diminishes effective circulating volume and adversely affects preload. Endothelial dysfunction resulting in third-spacing causes capillary leak and tissue edema, particularly in individuals with systemic inflammatory response syndrome (SIRS) [16].

Anesthetic and vasoactive agents have a substantial impact on hemodynamics. Propofol and sevoflurane decrease systemic vascular resistance and myocardial contractility, frequently requiring vasopressor support [17]. The administration of opioids, benzodiazepines, and neuromuscular blockers can lead to a reduction in sympathetic tone, which may result in hypotension. Vasopressors, including norepinephrine, are frequently utilized to restore mean arterial pressure (MAP); however, they may elevate afterload and myocardial oxygen demand [18]. Inotropes such as dobutamine and milrinone enhance contractility; however, their improper use may lead to tachyarrhythmia and hypotension [19].

Pharmacologic interventions must be customized to address the specific etiology of hypotension. High-dose vasopressors in hypovolemic patients may exacerbate perfusion issues by further diminishing microvascular flow. Excessive fluid administration in a patient with impaired ventricular compliance may lead to pulmonary edema. NT-proBNP serves as an objective indicator of volume tolerance and myocardial stress, facilitating more precise and tailored therapeutic approaches [7].

HEMODYNAMIC MONITORING

Effective and precise hemodynamic monitoring is essential for the management of patients in the immediate postoperative phase after CABG. Due to the intricate nature of fluid shifts, myocardial dysfunction, and inflammatory responses linked to cardiac surgery, clinicians are required to utilize various monitoring strategies, encompassing both conventional static measures and dynamic, minimally invasive, and biochemical evaluations.

Non-Invasive Monitoring

Oscillometric blood pressure monitoring represents the predominant method for non-invasive measurement of blood pressure. Although convenient, its accuracy is notably diminished in the presence of arrhythmias, low perfusion states, and hypotensive episodes [20]. Electrocardiography (ECG) is crucial for monitoring cardiac rhythm and detecting ischemia, which can significantly impact hemodynamics, particularly in cases of atrial fibrillation or conduction delays [21]. Pulse oximetry provides oxygen saturation data and plethysmographic waveform analysis, which can estimate fluid responsiveness through the pleth variability index under specific conditions [22].

Transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE) provide essential direct visualization of cardiac structures. They can quickly evaluate ejection fraction, wall motion abnormalities, volume status (through inferior vena cava diameter), pericardial effusion, and valvular function [23]. TEE is particularly beneficial in postoperative patients when transthoracic access is restricted by dressings or suboptimal acoustic windows.

Invasive Monitoring

Invasive arterial pressure monitoring through a radial or femoral arterial catheter offers continuous, real-time blood pressure measurements. This method facilitates the assessment of dynamic parameters, including stroke volume variation (SVV) and pulse pressure variation (PPV), which outperform static indicators such as central venous pressure (CVP) in forecasting fluid responsiveness, particularly in mechanically ventilated patients with regulated tidal volumes and sinus rhythm [24,25]. A PPV exceeding 13% or an SVV greater than 12% typically suggests that the patient is likely to exhibit an increase in stroke volume in response to fluid administration.

Central venous catheterization, frequently utilized in cardiac surgery, facilitates the assessment of CVP and central venous oxygen saturation ($ScvO_2$). Despite its limitations as a standalone predictor of preload, the trend of CVP over time, when combined with other variables like NT-proBNP and lactate levels, may provide valuable insights into volume status and right heart function [11]. $ScvO_2$ estimates the equilibrium between oxygen delivery and consumption, potentially decreasing in conditions of insufficient perfusion.

Pulmonary artery catheterization (PAC) offers extensive hemodynamic data, encompassing cardiac output, pulmonary artery pressures, pulmonary artery occlusion pressure (PAOP), and mixed venous oxygen saturation (SvO_2). This approach is considered the benchmark in complex cases, especially for patients with pulmonary hypertension, biventricular failure, or severe LCOS [26]. PAC insertion presents several risks, such as arrhythmia, thrombosis, pulmonary artery rupture, and infection, leading to a decline in its routine application in favor of less invasive alternatives [27].

Minimally Invasive Monitoring

Recent advancements, including pulse contour analysis, esophageal Doppler, and bioreactance-based cardiac output monitoring, provide non-invasive alternatives for acquiring real-time data compared to PAC. Systems such as PRAM (Pressure Recording Analytical Method) and Vigileo–FloTrac estimate cardiac output and related parameters through the analysis of arterial waveforms, frequently eliminating the requirement for external calibration [28,29]. These systems enable the monitoring of trends in cardiac output, systemic vascular resistance, and stroke volume, thereby supporting goal-directed therapy protocols.

. Integration of Monitoring Data

One modality alone cannot adequately represent the complete hemodynamic profile of a patient. Multimodal monitoring that integrates dynamic (e.g., SVV, PPV), static (e.g., CVP), biochemical (e.g., NT-proBNP, lactate), and imaging-based data (e.g., echocardiography) offers the most precise and practical evaluation. Integrating NT-proBNP into this framework is crucial for comprehending cardiac strain and assessing the probability of fluid responsiveness compared to the necessity for vasopressors or inotropes [23].

NT-proBNP

NT-proBNP is the inactive amino-terminal fragment of the prohormone proBNP, which is cleaved into active BNP and NT-proBNP as a response to ventricular wall stress. The left ventricle primarily secretes these peptides in reaction to heightened myocardial stretch, volume overload, or pressure overload.6 In contrast to BNP, NT-proBNP lacks biological activity but possesses a longer half-life (60–120 minutes) and greater stability in plasma, rendering it more dependable for clinical assessment [30].

NT-proBNP release is facilitated by elevated intracellular calcium levels and mechanical stretching of myocardial cells. It plays an indirect role in the regulation of blood pressure, sodium excretion, and vasodilation by indicating the extent of myocardial dysfunction. Elevated NT-proBNP levels indicate heightened left ventricular filling pressures and diminished myocardial compliance, conditions frequently observed in post-CABG patients [31].

NT-proBNP serves as a sensitive indicator of cardiac stress; however, its levels may be affected by various confounding factors. Age is a

documented factor, as older patients show elevated baseline NT-proBNP levels attributed to reduced clearance and modified cardiac compliance [32]. Renal function is significant, as NT-proBNP undergoes partial clearance by the kidneys. In patients with renal insufficiency, elevated levels may not accurately indicate acute cardiac dysfunction and should be interpreted cautiously [33].

Additional factors, including obesity, sex, and the use of medications such as neprilysin inhibitors, may also affect levels. Consequently, the interpretation of NT-proBNP should be conducted in conjunction with hemodynamic data and the clinical context [34].

Multiple studies have demonstrated the prognostic significance of NT-proBNP in cardiac surgery. Increased preoperative levels correlate with a heightened risk of perioperative complications, such as low cardiac output syndrome, extended mechanical ventilation, and prolonged ICU stay [7]. Postoperatively, NT-proBNP levels generally increase within the initial 24 to 48 hours as a result of myocardial stunning induced by CPB, along with fluid shifts and inflammation [35].

Repeated assessments of NT-proBNP demonstrate superior clinical utility compared to a solitary measurement. An upward trend may suggest persistent myocardial stress or deteriorating heart failure, warranting an increase in therapeutic intervention. A decreasing NT-proBNP level may indicate the appropriateness of reducing inotropic support or facilitating ICU discharge. In patients exhibiting ambiguous hemodynamic findings, NT-proBNP serves as a useful biomarker to distinguish between hypovolemia and cardiogenic shock, with levels being markedly elevated in the latter condition [36,37].

Age-adjusted thresholds are frequently employed for the interpretation of NT-proBNP levels. A value exceeding 125 pg/mL in patients under 75 years and exceeding 450 pg/mL in those over 75 years is typically regarded as abnormal in the outpatient context. In the perioperative context, thresholds are elevated due to the physiological stress associated with surgery. Research indicates that postoperative levels exceeding 1500–2000 pg/mL may forecast negative outcomes following CABG, particularly when accompanied by echocardiographic signs of left ventricular dysfunction [34].

The incorporation of NT-proBNP into risk assessment models may enhance the precision of predicting postoperative complications. The incorporation of this factor into clinical decision algorithms could facilitate the standardization of treatment pathways and the identification of patients necessitating more intensive monitoring or early intervention [38].

CLINICAL APPLICATION AND MANAGEMENT

The utilization of NT-proBNP in clinical practice encompasses functions that surpass its diagnostic and prognostic capabilities. It is increasingly utilized as a tool for guiding individualized therapy, particularly in complex hemodynamic situations following CABG. Integrating NT-proBNP values with dynamic and structural cardiac data enables clinicians to enhance the accuracy of therapeutic decisions related to fluid management, vasopressor initiation, inotrope selection, and escalation to mechanical support.

A significant challenge in postoperative care is the differentiation of various causes of hypotension. SVV and PPV serve as indicators of volume responsiveness; however, they do not determine the underlying cause of reduced cardiac output. NT-proBNP provides biochemical indicators of myocardial wall stress, which is typically markedly increased in cases of cardiogenic and obstructive shock, while it is reduced in instances of purely hypovolemic or vasodilatory states.35 A patient exhibiting elevated NT-proBNP levels (>3000 pg/mL), low stroke volume variation (SVV <10%), and echocardiographic indicators of poor contractility is likely experiencing cardiogenic shock. Treatment should prioritize inotropes over fluids. A patient exhibiting moderately elevated NT-proBNP levels (~800 pg/mL), high stroke volume variation (SVV >13%), and normal cardiac function on TTE may be a candidate for cautious fluid resuscitation.

Trends in NT-proBNP levels can function as biomarkers indicating the efficacy of therapy. Decreasing values following fluid restriction, diuretics, or inotropes indicate an enhancement in myocardial strain. Conversely, increasing levels may suggest poor management, persistent ischemia, or unaddressed volume overload, necessitating re-assessment [7,35].

This monitoring is particularly beneficial for patients in whom invasive monitoring is contraindicated or presents technical challenges. NT-proBNP levels may serve as indicators for the readiness to de-escalate support. In patients exhibiting normalized SVV, enhanced MAP, and reduced NT-proBNP levels, clinicians can initiate vasopressor tapering with increased assurance.

Table 1: NT-ProBNP-guidedd hemodynamic decision algorithm after CABG. SVV: stroke volume variation; PPV: pulse pressure variation; EF: ejection fraction; IABP: intra-aortic balloon pump; ECMO: extracorporeal membrane oxygenation

NT-proBNP Level	SVV/PPV	Echocardiographic Findings	Likely Etiology	Recommended Intervention
Low-Moderate (<1500 pg/mL)	Low (<10-12%)	Hyperdynamic EF	Vasoplegia / Distributive	Vasopressors (norepinephrine ± vasopressin)
Low (<800 pg/mL)	High (>13%)	Normal EF	Hypovolemia	Aggressive fluid resuscitation
Moderate	High (>13%)	Normal EF	Hypovolemia with	Cautious fluid resuscitation

(800-2000 pg/mL)			tolerance	
High (>2000 pg/mL)	Normal (10-13%)	Preserved EF, diastolic dysfunction	Elevated filling pressures	Afterload reduction, cautious diuretics
High (>3000 pg/mL)	Low (<10-12%)	Reduced EF, poor contractility	Cardiogenic shock	Inotropes (dobutamine/ milrinone, ± IABP/ECMO)

Multiple studies advocate for the incorporation of NT-proBNP into hemodynamic management protocols (Table 1). This approach promotes multimodal decision-making, reduces dependence on individual parameters, and facilitates adjustments tailored to the patient. The availability of NT-proBNP through rapid assays in numerous institutions enhances its feasibility for bedside use within standardized algorithms [20,30].

Several centers have integrated NT-proBNP measurement into postoperative protocols. Measuring NT-proBNP upon ICU admission, at 24 hours, and at 48 hours facilitates the early identification of hemodynamic deterioration prior to the emergence of overt clinical signs. Values exceeding institution-defined thresholds may prompt early echocardiography or the re-initiation of inotropic therapy.

In patients with lower risk, normal NT-proBNP trajectories may facilitate fast-track protocols, such as early extubation, early mobilization, and discharge from the ICU. NT-proBNP may facilitate goals-of-care discussions, especially in high-risk patients exhibiting severe left ventricular dysfunction and persistently elevated levels despite optimal therapy [34,38].

DISCUSSION

The incorporation of NT-proBNP into postoperative hemodynamic management signifies a notable improvement in the treatment of patients undergoing (CABG). Literature indicates that NT-proBNP levels exhibit a strong correlation with myocardial wall tension, volume overload, and filling pressures including key factors in postoperative hypotension and low cardiac output syndrome [6,7]. In contrast to conventional static measurements like CVP, NT-proBNP provides biochemical evidence of cardiac strain that is not influenced by operator technique and accurately represents changing physiological conditions.

NT-proBNP's primary strength is its capacity to differentiate among various types of hemodynamic compromise. Markedly elevated NT-proBNP values generally indicate cardiogenic origins of shock, in contrast to distributive or hypovolemic mechanisms, which tend to present with lower values [36]. This distinction is crucial in determining the appropriate use of vasopressors compared to inotropes, as well as fluids versus afterload reduction. Decision-making is particularly crucial in high-risk post-CABG patients, as inappropriate treatment or misdirected therapy may lead to adverse outcomes.

The interpretation of NT-proBNP value is enhanced when integrated into a multimodal approach. The integration of dynamic preload markers (SVV, PPV), echocardiographic parameters (ejection fraction, ventricular compliance, inferior vena cava diameter), and metabolic indicators (lactate, $ScvO_2$) facilitates a more sophisticated and physiologically grounded assessment of the patient's condition [5,23]. This method transcends the constraints of single-variable algorithms and corresponds with contemporary developments in precision critical care. NT-proBNP, while beneficial, has inherent limitations. Levels are influenced by variables including age, renal function, sex, body mass index, and chronic cardiac conditions [31,33]. In individuals with chronic kidney disease, NT-proBNP levels may be elevated despite the lack of acute decompensation, which can result in diagnostic ambiguity. Baseline values exhibit considerable variation among different age groups, necessitating the use of age-adjusted thresholds for accurate interpretation by clinicians [32]. Furthermore, trends in NT-proBNP are more informative than absolute values, especially in assessing therapeutic response over time.

Additionally, it should be noted that NT-proBNP, although dynamic, does not serve as an instantaneous marker. Alterations in myocardial wall stress may require several hours to manifest in serum levels. Consequently, NT-proBNP is most effectively utilized for evaluating overall trends or serial responses, rather than for continuous monitoring on a minute-to-minute basis. Consequently, NT-proBNP should not serve as a replacement for, but rather as a complement to, more immediate hemodynamic tools such as arterial waveform analysis, bedside ultrasound, and PAC-derived variables when accessible [20,26].

NT-proBNP can be readily incorporated into current ICU workflows. The growing accessibility of point-of-care NT-proBNP assays enables serial measurements at specified intervals (e.g., upon ICU admission, and at 24 and 48 hours postoperatively) to be both feasible and cost-effective in the majority of cardiac centers [30]. In resource-limited settings, a single NT-proBNP value measured within the first 12 hours postoperatively can yield important prognostic and diagnostic information, particularly in the absence of invasive monitoring.

NT-proBNP is recognized as one of the most robust biomarkers for outcome prediction in cardiac surgery populations. Postoperative elevated levels have been shown to predict prolonged ICU stay, a higher requirement for inotropic or mechanical support, and an increased risk of postoperative atrial fibrillation and renal dysfunction [34,35]. The associations indicate that NT-proBNP may function as both a clinical tool and a quality improvement marker in cardiac surgery programs.

Future research must prioritize the development of NT-proBNP–guided management pathways via rigorously designed randomized

controlled trials. Although the biomarker demonstrates significant potential in observational studies, prospective data are required to confirm specific thresholds for fluid administration, inotrope initiation, and ICU discharge criteria. Furthermore, its incorporation into clinical decision support systems and electronic health records may facilitate automated risk stratification and standardized interventions. The utilization of machine learning on extensive ICU datasets, encompassing NT-proBNP trends, echocardiographic measurements, and hemodynamic variables, presents potential for predictive modeling and early warning systems [5,29]. These technologies may facilitate real-time clinical decision-making, minimize delays in therapy escalation, and enhance overall outcomes.

NT-proBNP serves as a sensitive, reliable, and clinically actionable biomarker for the postoperative hemodynamic management of CABG patients. The true value is realized when incorporated into a multimodal strategy that integrates physiological, biochemical, and imaging data. Proper interpretation and institutional integration of NT-proBNP can significantly enhance personalized, data-driven cardiac critical care.

RESUME

Postoperative hypotension presents a considerable challenge in the management of patients undergoing CABG. The multifactorial etiology, which includes cardiogenic, hypovolemic, distributive, and obstructive mechanisms, requires a detailed and personalized approach to both diagnosis and treatment. Conventional static monitoring tools provide limited insight into the dynamic cardiovascular changes that take place during the postoperative period. The incorporation of advanced hemodynamic monitoring technologies and biomarkers has significantly transformed the clinical landscape, facilitating more precise and adaptive therapeutic strategies.

NT-proBNP is a promising biomarker that effectively indicates myocardial wall stress, volume status, and ventricular filling pressures. The perioperative elevation is associated with negative outcomes, such as low cardiac output syndrome, extended intensive care unit stays, renal dysfunction, and heightened mortality rates. Serial NT-proBNP measurements yield significant trend data, enabling clinicians to evaluate recovery or deterioration trajectories and modify therapy as needed.

NT-proBNP's strength is its capacity to enhance conventional monitoring methods rather than serve as a replacement. Utilizing NT-proBNP alongside dynamic preload indicators, including SVV and PPV, as well as imaging techniques such as echocardiography, improves the precision of hemodynamic evaluations and the suitability of clinical interventions. It is essential for differentiating between fluid-responsive and fluid-intolerant hypotension, thereby reducing the risks associated with fluid overload or under-resuscitation. This also assists in distinguishing between types of shock, thereby informing the administration of vasopressors, inotropes, or mechanical support.

NT-proBNP should be interpreted within the broader clinical context, taking into account factors such as age, renal function, and preexisting cardiac disease. Integration into a multimodal strategy that combines biochemical, physiological, and imaging data yields optimal effectiveness. With accumulating evidence supporting its prognostic and diagnostic significance, NT-proBNP is becoming a key element in goal-directed therapy within cardiac surgical critical care.

Future research is essential to determine standardized NT-proBNP thresholds for therapeutic decision-making and to investigate its incorporation into predictive models and electronic health records. Randomized controlled trials must evaluate if NT-proBNP-guided management enhances critical outcomes, including mortality, mechanical ventilation duration, and ICU length of stay. The advancement of point-of-care NT-proBNP testing and the implementation of machine learning algorithms in biomarker data have the potential to transform perioperative cardiac care.

In summary, NT-proBNP serves as a robust, evidence-supported biomarker that markedly improves hemodynamic monitoring and clinical decision-making in the postoperative care of CABG patients. The integration of this approach into standard practice may enhance outcomes, tailor care, and raise the quality of critical care cardiology.

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