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Review article

Ultrasound in Cardiopulmonary bypass in patients undergoing Mitral and Aortic Valve Surgery: A Card to be Played?

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Abstract

Mitral and aortic valve surgeries, necessitating open-heart procedures with cardiopulmonary bypass, mandate a comprehensive comprehension of the pathophysiological mechanisms underlying mitral valve insufficiency (MVI) and aortic valve insufficiency (AVI). This knowledge is indispensable for devising efficacious perfusion strategies during cardiopulmonary bypass. This narrative review seeks to transcend generalized perfusion paradigms by emphasizing the crucial role of ultrasound scans in delineating and optimizing cardiopulmonary bypass procedures for MI and AoI. The article underscores the significance of ultrasound in real-time monitoring of key perfusion parameters, specifically focusing on compromised vena cava, arterial resistance, and pulmonary congestion. The conjoined utilization of heart-lung-cava ultrasound furnishes insights into critical elements of perfusion management. For instance, the collapsibility index of the inferior vena cava prognosticates compromised venous drainage, while Doppler parameters gauge alterations in arterial compliance. Resistivity (RI) and pulsatility (PI) indices furnish valuable insights into distal and proximal vascular resistance. Recommendations predicated on ultrasound parameters encompass employing vacuum in confirmed cases of venous compromise and employing low-volume hemodilution and low volemia cardioplegic agents in instances of pulmonary congestion. The article further advocates for the utilization of mannitol and bicarbonate, along with perfusion techniques maintaining zero hemocentration balance.

Introduction

For many perfusionists, reaching the learning curve can be a challenging endeavor, due to the automated processes built upon well-established principles and standards. Consequently, this presents a surprising barrier between the realms of diagnostic imaging and their technical routines in perfusion. The standardization of a uniform perfusion process for all, irrespective of associated comorbidities, presents a significant obstacle that many perfusionists must conquer on their journey towards becoming highly competent professionals.

It is believed that standardized knowledge of predefined perfusion protocols reflects the indisputable realm of "generalized perfusion and patient casuistry." In order to transcend the paradigm of generalized perfusion, this review describes the importance of ultrasonographic examinations as a support in the planning of cardiopulmonary bypass (CPB) in the context of heart failure (HF) due to mitral valve insufficiency (MI) and aortic insufficiency (AoI), and discusses in detail their significance in evidence-based strategies, including pre and post-ECC procedures.

Thus, HF represents clinical syndrome, and it is acommon final clinical pathway resulting from pressure overload. Currently, it is challenging to provide a precise definition of HF due to evolving etiologies, variations in heart failure symptoms across different age groups, and shifting understandings of its pathophysiology over time. Drawing on the research by Heidenreich et al. (2021)1, it compels

us to assert that CHF is a clinical syndrome in which cardiac disease diminishes cardiac output, elevates venous pressures, and is accompanied by molecular abnormalities that lead to the progressive deterioration of the heart, ultimately resulting in myocardial cell failure and premature death.

For the purposes of this narrative review, the term HF is confined symptoms due to cases of MI and AoI that warrant surgical intervention. The signs and symptoms leading to hospitalization in these cases can be attributed to volume overload and pulmonary congestion (PC).

It is important to emphasize that both types of valve insufficiency are fundamentally distinct from each other. These distinct lesions result in varying loading conditions, diverse pathophysiological mechanisms, and require different surgical corrections. However, both types can lead to HF, arrhythmias, frequent hospitalizations, and ultimately, mortality2.

Cardiac overload typically necessitates myocardial remodeling and increased focus on the electrical potential and depolarization vector. As a result, the electrocardiogram reflect these changes, particularly in advanced stages. Furthermore, atrial fibrillation is often prevalent in these patients and serves as a common indicator of potential PC3.

Despite the prevalence of diagnostic findings associated with PC, chest X-rays serve as the primary diagnostic imaging procedure. They can reveal indicators such as peribronchial cuffing, cardiomegaly, pulmonary venous congestion, or pleural effusion. When comparing X-rays to other diagnostic imaging techniques, they have been found to be reasonably specific but not particularly sensitive to detecting PC4.

Pathophysiology of MI and AoI

Left ventricular (LV) wall stress is notably greater in AoI (the classical left ventricular pressure overload condition) compared to MI5. Consequently, these distinct loading conditions tend to result in different forms of ventricular remodeling6. In the case of MI, the LV may exhibit slight enlargement and thin-walled characteristics, where the mass/volume ratio is less than 1. Conversely, in AoI, there is a combination of modest concentric LV hypertrophy and eccentric hypertrophy, and normal mass/volume ratio can be present7.

Looking at overload from a different perspective, the average systolic blood pressure in AoI is higher than in MI, and correspondingly, the average LV wall thickness in AoI tends to exceed that in MI8. In both scenarios, the severe acute phase is generally poorly tolerated, often necessitating immediate intervention9. However, chronically most cases well tolerable (MI and compensated AoI) due to various electromechanical adaptations triggered by the hypertrophic process, including the addition of sarcomeres in series, which increases the length of cardiomyocytes, neurohormonal responses resulting in fluid and sodium retention, and subsequently, an increase in total LV volume10-12. This enables the ventricle to pump a larger total volume, compensating for the reduced output generated by the valvar regurgitation 13,14.

Simultaneously, both valvar regurgitations exhibit a progressive pattern of volume overload in the LA15,16. This, in turn, results in LA hemodynamic resistance being reflected in the pulmonary vascular bed, causing increased pressure, pulmonary edema, and subsequent overloading of the right chambers17,18. It is worth noting that in both the compensated and chronic phases, the LV ejection fraction is higher than normal. Even in the decompensated phase, the increase in preload keeps the ejection fraction (EF) within the normal range19-21 or even hiperdinamic, emphasizing the paradigm that "a normal EF does not necessarily indicate normal systolic function in these cases."

Pulmonary Congestion

Traditionally, PC stands out as the primary and conspicuous sign of a critical phase of LA overload in cases of MI or AoI, significantly contributing to the heightened risk of early hospitalization for acutely decompensated patients22,23. The exacerbation and persistence of subclinical pulmonary fluid overload remain unclear in certain cases of valvar regurgitation, as not all patients with MI or AoI exhibit universal pulmonary fluid congestion. Moreover, the correlation between post-capillary (venous) pulmonary pressure and CP is surprisingly weak. In fact, some patients can endure substantial increases in LA pressure without developing PC24.

In heart failure (HF), elevated left atrial (LA) pressure surpassing a critical threshold leads to increased lung water, resulting in heightened hydrostatic pressure within the pulmonary capillaries. While left ventricular filling pressures can be accurately estimated through cardiac catheterization or non-invasively assessed using echocardiography, the consequences of increased LA pressure may vary among patients. This divergence can stem from disparities in the thickness of the alveolar-capillary membrane or discrepancies in lymphatic drainage efficiency. When the rise in LA pressure exceeds the lymphatic drainage capacity, patients may present with dyspnea, diminished exercise tolerance, and, in severe cases, alveolar edema25.

However, it is crucial to emphasize that LA pressure overloads can trigger pulmonary adaptations to increased post-capillary pressure. These adaptations include the thickening of the basal membrane, enhanced alveolar fluid clearance, and increased lymphatic drainage25,26. This,

in turn, activates the hypothalamic-pituitary-adrenal axis, stimulating a reduction in urinary secretion and promoting sodium retention in acutely decompensated patients27-29.

Another contributing factor to fluid retention is the stimulation of natriuretic peptide (BNP) production30. Elevated natriuresis can result from the interruption of signaling at afferent sensing sites located in the cardiopulmonary system31. These sites include disrupted baroreceptors situated in the carotid sinus and aortic arch, as well as malfunctioning mechanosensitive nerve endings in cardiac chambers and the cardiopulmonary system, which can lead to the failure to detect elevations in circulating volume, thus contributing to congestion due to pressure overload and its associated cardiovascular outcomes32-34.

As a result, patients with PC exhibit alterations in the ventilation-perfusion axis due to increased lung compliance. This, in turn, leads to a compensated greater expansion of the respiratory muscles to enhance inspiratory flow and maintain an adequate tidal volume35. The elevation in negative intrathoracic pressure during inspiration exacerbates the development of pulmonary edema, increasing both ventricular preload and afterload36,37. Furthermore, the oxygen consumption for respiration increases, potentially compromising oxygen delivery to the myocardium. These alterations can be managed with the use of diuretics, vasodilators, and supplemental oxygen38.

Assessment of Pulmonary Congestion in MI and AoI

Several formulas have been developed to indirectly estimate plasma volume (PV) using hemoglobin and/or hematocrit, which can be valuable for monitoring both acute and chronic congestion39. One simple formula for assessing estimated changes in plasma volume (PV) is the Strauss formula, which is calculated as follows: Change in PV 100 x (hemoglobin (initial))/(hemoglobin (final)) x (1-hematocrit (initial))/(1-hematocrit (final))-100

Lung ultrasound

The implementation of Lung ultrasound (LU) has enabled the effective measurement of intrapulmonary fluid and its correlation with pulmonary arterial pressure40 with a facility to be available and simple to apply into surgical on bedside. LU can provide valuable information for prognostic stratification of patients admitted to the emergency department with dyspnea and/or chest pain syndrome41. Additionally, it can promptly detect changes in congestion and allow therapeutic intervention befora and after a surgical procedure.

Recent study results suggest that incorporating bedside imaging tests, such as ultrasound (US), can significantly contribute to the identification of residual and subclinical pulmonary congestion in patients hospitalized for congestive heart failure (HF) and, consequently, facilitate improved decision-making regarding treatment42-45.

Echocardiography

Currently, echocardiography (ECHO) serves as the gold standard test for indirectly diagnose and monitor intracardiac pressure overload generated by MI and AoI. It is also valuable for assessing the etiology of valve insufficiency and for planning the surgical approach46. In the context of cardiopulmonary bypass (CPB), ECHO provides essential parameters, including the severity of the intraventricular (IV) condition, left atrium (LA) diameter under pressure overload, pulmonary hypertension (PH), diameter of the vena cavae, diameter of the ascending aorta, and enables the estimation of systemic arterial resistance.

Perfusion Strategies Guided by Ultrasound Parameters During Mitral or Aortic Valve Surgery

The introduction of CPB machinery has revolutionized open-heart surgery, ensuring the preservation of cellular integrity. The development of modern CPB techniques is the collaborative effort of a multidisciplinary team, involving various healthcare and engineering professionals with extensive knowledge of anatomophysiology and cardiovascular hemodynamics47. In the present day, innovations continually emerge, reshaping methods and introducing novel technologies.

Therefore, all these advancements and innovations in perfusion aim to mitigate the detrimental effects of CPB. Consequently, the integrated use of cardiac-lung ultrasound plays a pivotal role in decision-making related to the optimal choice of oxygenator, calculation of arterial flow, selection of arterial and venous cannula sizes, and predicting conduct during perfusion. This integrated approach complements the existing routine parameters.

In summary, the CPB circuit comprises pumps, cannulas, tubes, a reservoir, an oxygenator, a heat exchanger, and an arterial line filter. Present-day machines are equipped with systems for monitoring pressure, temperature, oxygen saturation, hemoglobin, blood gases, and electrolytes. They also feature safety components like bubble detectors, oxygen sensors, and alarms for identifying low levels in the reservoir. Consequently, there are numerous scenarios where ultrasound plays a crucial role in CPB decision-making, particularly in mitral valve or aortic valve surgery.

Lung ultrasound in the Selection of Venous Drainage Type and Venous Cannula

During extracorporeal circulation, central venous pressure (CVP) is expected to be close to zero. However, in routine perfusion of patients undergoing mitral or aortic

valve surgery, it is often observed that there is an increase in CVP, accompanied by whipping of the venous line tubes, indicating impaired venous drainage into the reservoir. It has already been documented that this increase may be linked to inadequate cannula size, obstruction of the cannula line or tip, and an insufficient height difference between the patient and the reservoir to allow gravity drainage of the siphon48.

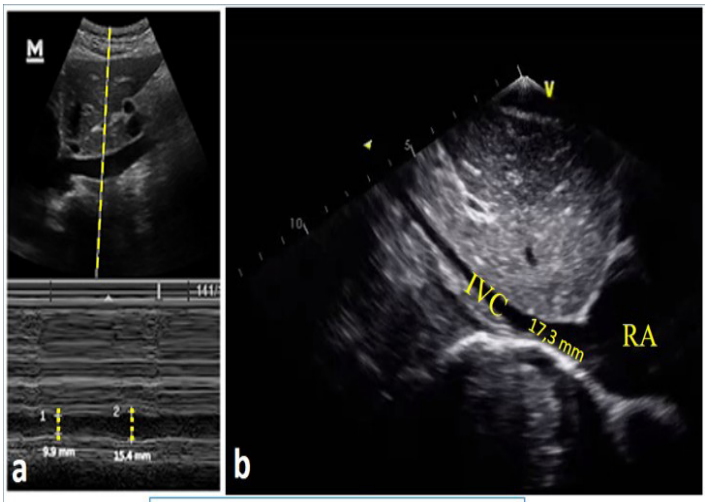
The choice of cannula is contingent on several factors, including the surgeon's preference (single cannula inserted into the right atrium), the target flow rate, vessel size, and the nature of the procedure. However, it is essential to note that many patients with MI and AoI exhibit elevated central venous pressure and increased intravascular volume, resulting in mechanical and hemodynamic alterations in the inferior vena cava (IVC)49, which can potentially impede venous drainage during CPB. Consequently, LU has gained recognition as a method for evaluating intravascular volume status.

Geometric measurements of the vena cava using LU represent a dependable non-invasive technique. Central venous pressure (CVP) demonstrates a positive correlation with IVC size and a negative correlation with the IVC collapsibility index50,51. Consequently, the IVC collapsibility index may serve as a superior indicator to IVC diameter, as it encompasses dynamic changes in diameter throughout the respiratory cycle52.

However, it's important to note that the diameter of the IVC measured by ultrasound (US) may be underestimated due to the "tangent cylinder effect," which occurs when the US beam traverses the vena cava longitudinally in the subcostal window, as illustrated in Figure 1. To mitigate this error, calculating the inferior vena cava index (IVCI) can be a valuable approach. This method helps reduce errors associated with minor variations in the sampling angle due to acoustic incidence53,54. Therefore, we recommend utilizing this index as a parameter for determining the size of the venous cannula and the type of drainage (siphoning or vacuum) based on the degree of involvement of the venous drainage of the IVC.

Ultrasound Combination: Supplementary Parameters for Flow Calculation and Oxygenator Selection

The preparation of the CPB machine commences with the selection of the oxygenator. This choice is guided by a flow calculation using the formula f = BS x CI, which represents the cardiac output during CPB. In this calculation, both the body surface area (BS) and the cardiac index (CI) are dependent on the patient's weight. However, as mentioned earlier regarding the physiological aspects of IV, these patients may experience issues like fluid retention and interstitial extravasation, along with alterations in



$$IVCI = x\frac{VCIe - VCIi}{VCIe}x100$$

Classification	Description
I	IVC is narrowed or completely collapsed
II	Maximum IVC diameter is <21mm with respiratory variation >50%
III	Maximum IVC diameter is ≥21mm or respiratory variation is <50%
IV	Maximum IVC diameter is ≥21mm with respiratory variation <50%
V	Maximum diameter of the IVC is ≥21mm with insignificant respiratory variation and dilation of the hepatic veins

Figure 1: *Geometric Assessment of the Inferior Vena Cava (a) This segment illustrates the use of ultrasound (US) imaging with yellow lines to longitudinally examine the inferior vena cava within the subcostal window. It also demonstrates the IVC's adaptability during changes in venous return, as observed using M-mode imaging. (b) This portion offers a 2D representation of the inferior vena cava as it enters the right atrium. Furthermore, the figure presents an index used to quantify the degree of venous drainage impairment.Also the RA pressure can be estimated from 3mmHg , 5, 10, 15 or 20 mmHg using the same graduation.*

systemic vascular resistances. As a result, these factors are not typically considered when calculating the flow.

It is common for patients with acute decompensated MI and AoI undergoing surgery to experience an increase in the volume of blood collected in the cardiotomy reservoir that exceeds the normal levels. This phenomenon is not solely a result of using hyperosmolar solutions in the priming process or the volume of cardioplegia administered. Instead, it is believed that part of this increase in volume is due to inadequate compensation for blood flow when faced with elevated systemic vascular resistance, particularly when a centrifugal pump is employed. The function of a centrifugal pump relies on both preload and afterload. This dependence becomes apparent when the initial blood gas results reveal a type B lactic metabolic acidosis.

In this regard, echocardiographic examinations and vascular ultrasound play a valuable role in strategic perfusion planning. They enable the pre-assessment

of the hemodynamic profile using Doppler techniques, documenting velocities of blood flow, and facilitating the identification of variables that indicate potential impairment of hemodynamic flow, as well as artery compliance55-59.

With this tool it is possible to map the blood flow profile, including peak systolic velocity, end-diastolic flow, pulsatility index (PI) and resistivity index (RI), providing information on the quantity and quality of blood flow in a given time interval60 figure 2. In clinical routines, RI is often used to estimate perfusion at tissue level, deducing that the higher the resistance of the vessels, the higher the resistance index and the lower the local perfusion, while the lower the PI, the lower the perfusion of distal tissues61,62. In addition to these indices, the peak systolic velocity can be obtained from the hemodynamic oscillation offered by the vessels, which is a dependent technical variable, for which the positioning of the angle is decisive to better characterize the degree of vascular involvement63.

In addition to the previously mentioned indices, the cardio-ankle vascular index (CAVI) can be regarded as one of the alternative tools for assessing the flow resistance that may arise during CPB in the valve surgeries mentioned earlier. CAVI is a relatively new method known for its robustness in estimating arterial stiffness. It serves as a direct indicator of arterial distensibility and acts as an independent predictor of prognostic outcomes in patients with cardiovascular disease. This index allows for the measurement of the arterial tree from the aortic origin to the ankle64,65, as depicted in Figure 2.

The calculation of the CAVI index is based on the principles of arterial malleability, supported by the 2D vascular ultrasound imaging technique (intraluminal volemia) and the Bramwell-Hill formula. Consequently, it derives the heart-ankle pulse wave velocity (haPWV) by dividing L (the length from the aorta to the ankle) by T (the time for the pulse wave to travel from the aortic valve to the ankle) Figure 2. This reinforces the critical importance of a reliable assessment of vascular resistance for estimating adequate flow and choosing the optimal oxygenator, emphasizing that "the place of blood is always within the patient and not in the cardiotomy reservoir."

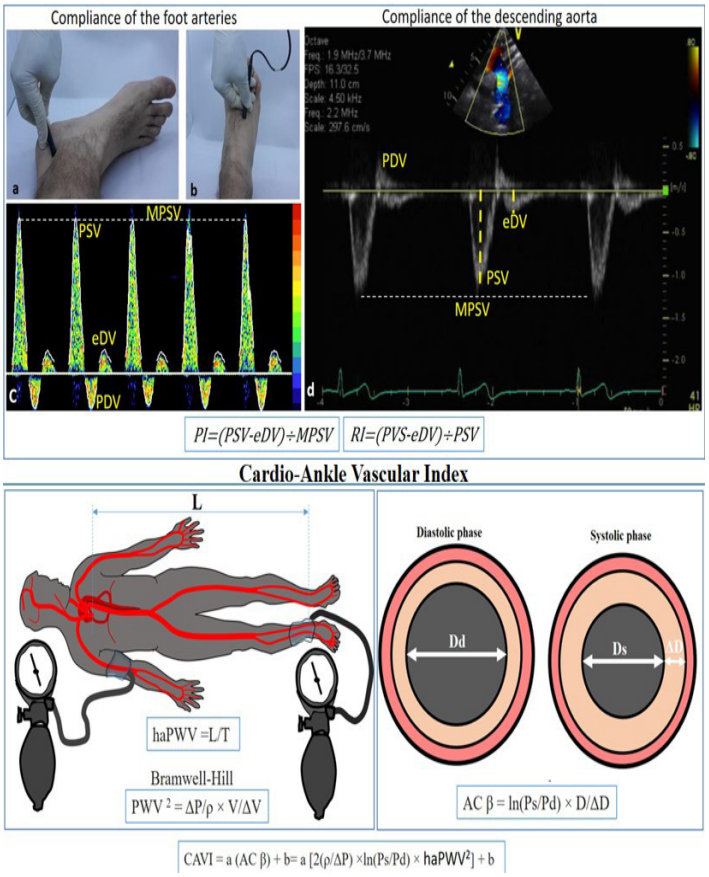


Figure 2: *Evaluation of systemic vascular resistance involves the measurement of distal arterial malleability in the flow of two specific arteries: (a) the posterior tibial artery and (b) the dorsal artery of the foot. This assessment includes the examination of their respective spectral flow graphs (c) using spectral Doppler ultrasound. Additionally, similar measurements can be taken near the aorta (d). These parameters allow for the mapping of the hemodynamic profile, documenting the quality of blood flow and the mechanical malleability of the arteries. Furthermore, the Cardio-Ankle Vascular Index (CAVI) can offer early insights into the state of systemic arterial resistance and may serve as a guide for the potential use of vasodilator drugs during cardiopulmonary bypass procedures.*

Lung Ultrasound in the Selection of Hemoconcentrator Type

The association between ideal weight and pulmonary congestion plays a pivotal role in assessing the volume status of patients with MI and AoI, given the chronic nature of these conditions. Many of these patients undergo antidiuretic therapy to alleviate congestion and ensure hemodynamic stability before surgery. However, this treatment does not reverse the thickening of the alveolar-capillary membrane, potentially leading to increased volume in the venous reservoir during cardiopulmonary bypass (CPB), as demonstrated in the attached videos. Therefore, depending on the severity of CHF, lung ultrasonography (LU) is crucial at the bedside before initiating CPB. Its significance lies not only in stratifying ideal weight but also in serving as a reference parameter for potential filtration.

Pulmonary congestion as observed on LU is characterized by the presence of multiple B-lines, often referred to as comets, which move in synchrony with lung sliding (if present)66. These B-lines are typically well-defined and appear like long lasers, extending to the lower part of the screen without fading, indicating the extravasation of fluid at the interstitial level67. Indeed, patients displaying B-lines on a LU often have increased pressure in their pulmonary arteries. Consequently, echocardiography can serve as a valuable method for measuring pulmonary artery pressure

and assessing its various branches. In addition to its diagnostic utility, echocardiography can assist perfusionists in determining the appropriate volume to use for such patients, considering their hypovolemic state (Figure 3).

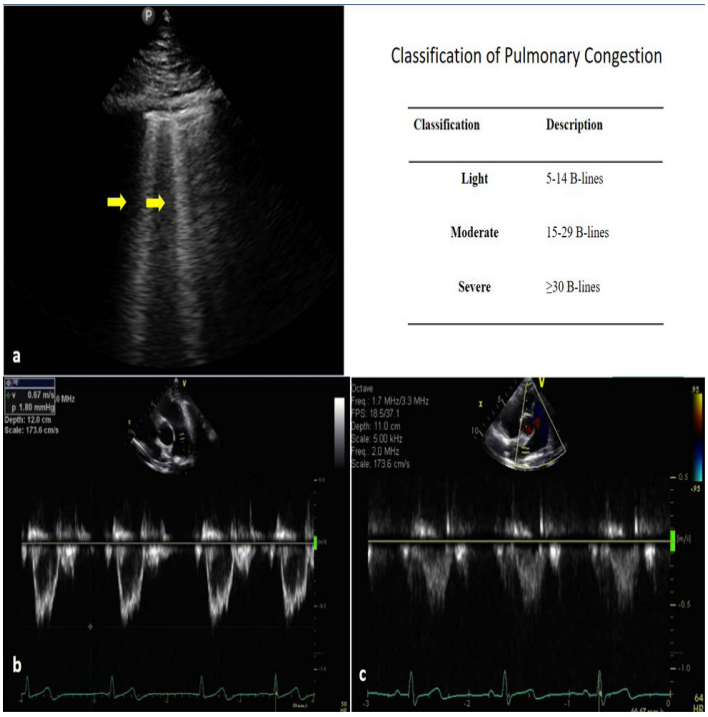


Figure3: *Pulmonary congestion plays a pivotal role in assessing the volume status of patients, serving as an important indicator for perfusionists to determine the appropriate volume of fluids to administer. (a) Real-time visualization of fluid accumulation in the interstitial spaces, denoted by B-lines (indicated with yellow arrows), allows for the classification of pulmonary congestion. B-lines are characteristic vertical lines seen on LU that signify increased lung density or fluid presence. (b) Evaluation of blood flow at the level of the pulmonary artery. (c) Assessment of blood flow in the right branch of the pulmonary artery.*

The total count of B-lines observed at each examination site yields a score that signifies the extent of extravascular fluid in the lung. In this scoring system, zero denotes the complete absence of pulmonary congestion in the area under examination, while the presence of B-lines is categorized into three grades: "mild" (5-14 comets), "moderate" (15-29 comets), and "severe" (≥30 comets)68,69, as depicted in Figure 3. These parameters for pulmonary congestion assessed by LU can play a crucial role in the selection of the appropriate type of hemofiltration and determining the volume of fluid that can be subjected to hemofiltration without inducing hypovolemia in the patient.

Ultrasound in the Composition of Priming and in the Choice of the Type of Cardioplegia

Building upon the pathophysiological mechanisms of Mitral Insufficiency and Aortic Insufficiency previously elucidated, it is universally acknowledged that distinct signs within cardiac-lung-vena cavae ultrasound combinations offer crucial parameters. These include systemic vascular resistance (IR, IP, and CAVI), pulmonary congestion (manifested by B-lines), and decreased renal output, characterized by fluid and sodium retention, heightened central venous pressure, and intravascular volume variations, which can be assessed through measurements of vena cava diameter and IVCI.

This valuable information compound the cornerstone for the strategic development of both the priming solution and the selection of cardioplegia. In this context, a suggestion arises concerning the utilization of mannitol as a key component within the priming solution to counteract interstitial edema, particularly the presence of pulmonary B-lines. This suggestion is founded on the premise that mannitol, functioning as an osmotic diuretic, holds the potential to mitigate renal damage when administered preemptively, prior to an ischemic event such as CPB or aortic clamping70.

The proposed mechanisms of action for mannitol encompass a 'washing' effect, clearing necrotic tubular debris, eliminating oxygen free radicals, and enhancing medullary blood flow through the reduction of endothelial edema71,72. On the other hand, we also recommend the inclusion of bicarbonate in the priming solution to reduce vascular resistance and prevent type B metabolic acidosis73. However, it is essential to note that patients with Mitral Insufficiency and compensated Aortic Insufficiency often exhibit normal blood gases due to increased hematocrit and hemoglobin levels74.

In contrast, patients with MI and decompensated AoI may present with anemia, resulting in reduced tissue perfusion and exacerbating type A lactic metabolic acidosis75. Furthermore, perfusion itself can induce metabolic acidosis due to low viscosity (as seen with Heparin use) and non-pulsatile flow, which stimulate the release of catecholamines (vessel constrictors)76-78. Considering these factors, the strategic use of bicarbonate can be beneficial in certain cases to address vascular resistance and metabolic acidosis.

Failure to correct metabolic acidosis before and after cardiopulmonary bypass can hinder the heart's recovery from asystole. This is due to a decrease in cardiac contractile function, centralization of blood volume (vasoconstriction and arteriolar dilation), reduced renal and hepatic blood flow, and a lowered pacing threshold.

Hence, it is crucial to address metabolic acidosis at the onset of extracorporeal circulation by manipulating the components of the prime. It's important to note that many centers do not have machines equipped with online gasometry evaluation tools. In other cases, which is the reality in many centers, gasmetry results take a considerable

amount of time to emerge from the laboratories. This often leaves perfusionists to work without the benefit of real-time data. As mentioned earlier, metabolic acidosis should also be corrected before weaning from the CPB machine. In many instances, there's a need to shock the heart to restore its autonomy or correct certain arrhythmias.

As for cardioplegia, given the hypervolemia (intravascular volume) presented by this patient, it is necessary to use cardioplegic agents that require minimal volume, in order to prevent further increases in the patient's blood volume and hemodilution.

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