

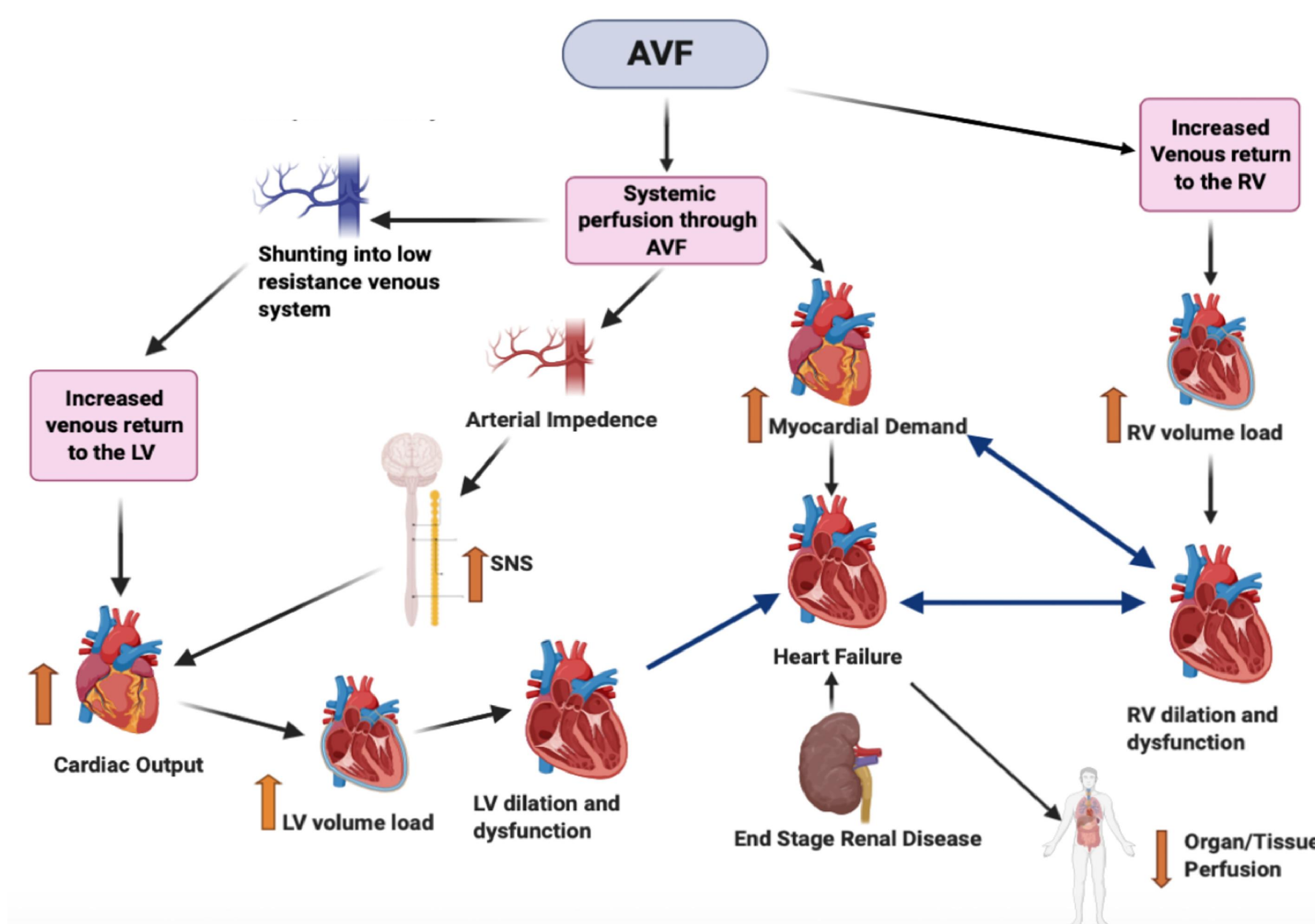
Perioperative Considerations for Emergent Arteriovenous Fistula Ligation in a Post-Cardiac Arrest Patient with High Output Heart Failure

Okola Tull MD, Leah Middleton MS, Edward Foley MD

Anesthesiology and Perioperative Medicine, Medical College of Georgia, Augusta University, Augusta, Georgia

INTRODUCTION

In patients undergoing hemodialysis, surgically created arteriovenous fistulas (AVFs) are the preferred option for vascular access. However, an often overlooked complication of AVFs is the development of high-output heart failure (HOHF). When a substantial amount of arterial blood is diverted from the left side to the right side of the circulatory system through the fistula, the resulting increase in preload can lead to elevated cardiac output (CO). Over time, this increased workload on the right heart will cause right ventricular dilation and eventually pulmonary hypertension.¹ Patients with HOHF typically exhibit symptoms such as tachycardia, elevated pulse pressure, a hyperdynamic precordium, and jugular venous distension. Anesthetic management must avoid myocardial depression and significant afterload reduction to prevent further cardiac strain.^{1,2}



CASE PRESENTATION

A 53-year-old dialysis-dependent male patient with a history of hypertension, renal cell carcinoma, ESRD on HD, bilateral native nephrectomy, and status post kidney transplant presented for left upper extremity AVF ligation under MAC and local anesthesia.

After receiving a 50 mg propofol bolus, followed by an infusion at 100 mcg/kg/min and 2 g cefazolin, the patient developed bradycardia with complete heart block.

CASE PRESENTATION

He then experienced a short burst of SVT before returning to bradycardia, leading to the case being aborted. The patient became unresponsive and required intubation. BP was unobtainable, and no pulsatile flow was detected. CPR was initiated, and ROSC occurred after three rounds. A right brachial arterial line and a RIJ central line were placed. The patient received 5 mg epinephrine, 10 units vasopressin, and 250mL albumin during the code. The patient was then transferred to CVICU for further stabilization.

Post-resuscitation, Swan-Ganz catheter placement revealed elevated R/L-sided filling pressures, increased cardiac output of 11.36 L/min with decreased SVR, and severe pulmonary HTN (PA 75/39 mm Hg) secondary to high output. While in the CVICU, the patient also developed new-onset atrial fibrillation with RVR and was started on an amiodarone infusion. After four days of hemodynamic improvement, he returned to the OR for a second attempt at ligation.

The case was done under GETA with hydromorphone and propofol infusions. For monitors and lines, the patient had a right internal jugular central line, right brachial line, and a PA catheter. Patient also remained on norepinephrine and amiodarone. The procedure was successful, with immediate improvement in mean arterial pressure, SVR and PA pressure, and he returned to the CVICU without issues. However, CT Head and EEG indicated severe hypoxic-ischemic encephalopathy. The patient's status was changed to comfort care, and he was terminally extubated.

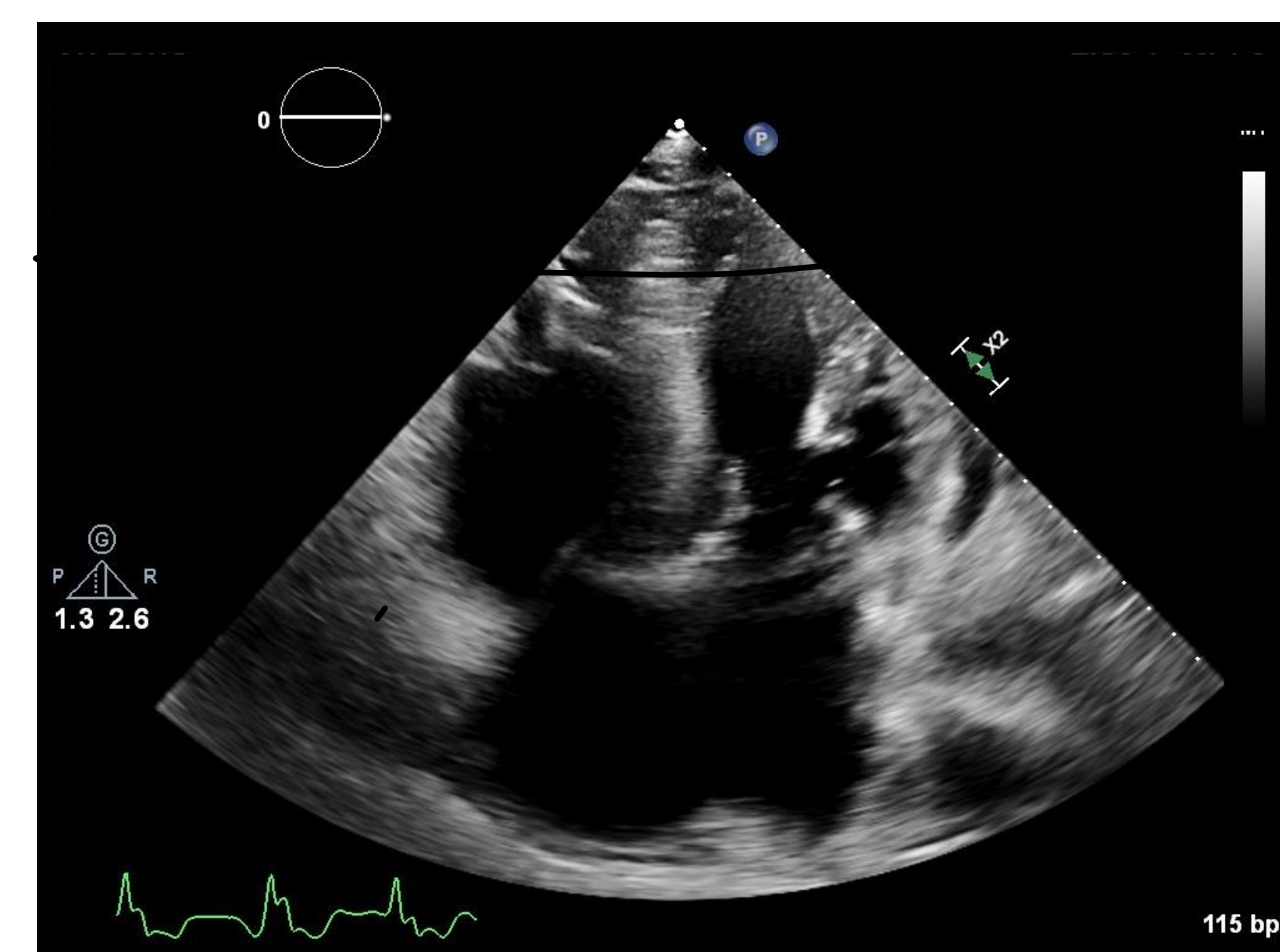


Figure 1: TTE- Apical 4 chamber view showing severe RV dilation

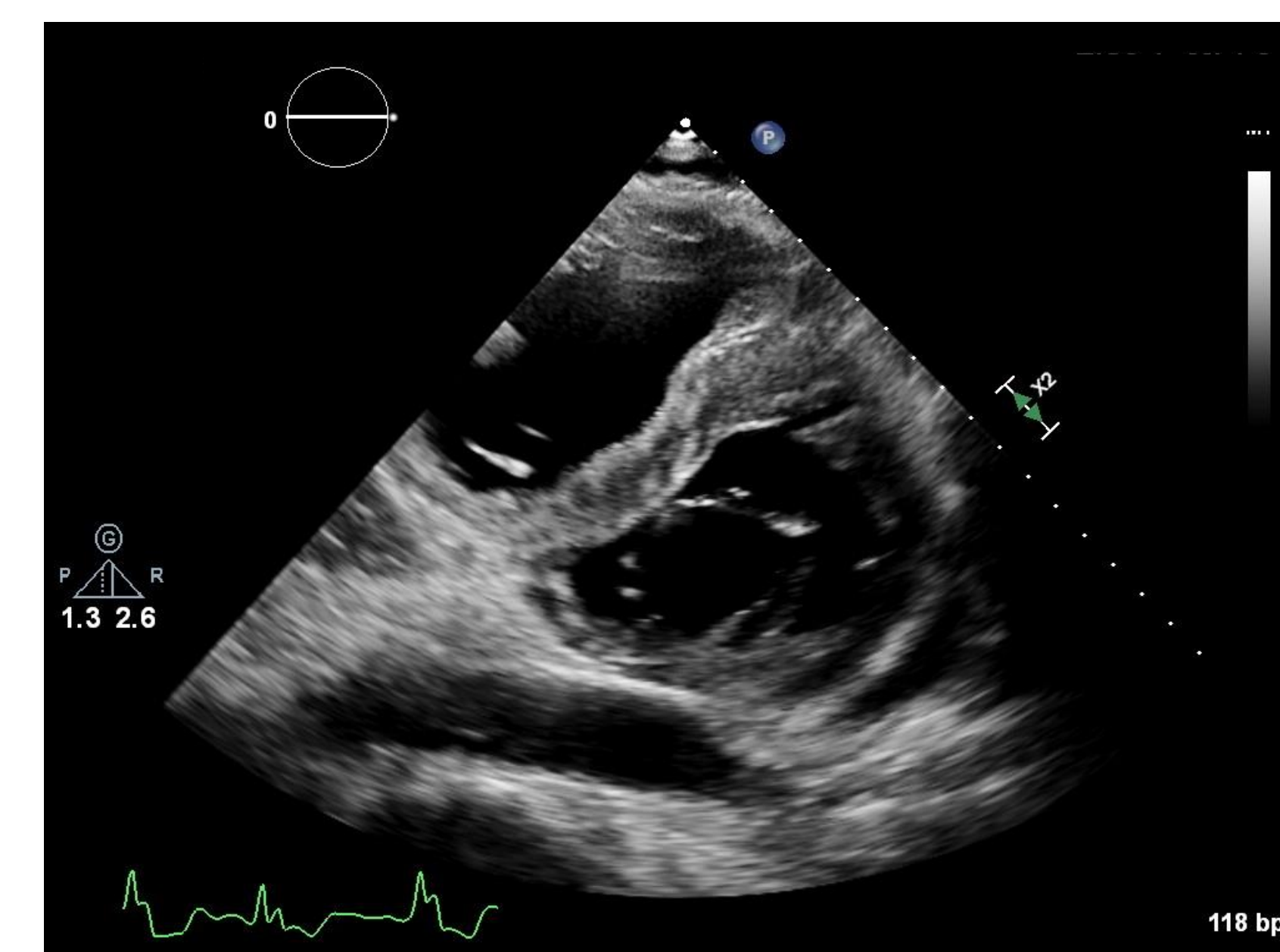


Figure 2: TTE- Parasternal short axis view showing severe RV overload secondary to HOHF

DISCUSSION

Arteriovenous fistulas can ultimately lead to high output heart failure (HOHF) due to the increased shunting of blood to the right-sided circulation. Increased venous return leads to decreased systemic vascular resistance (SVR). As a result, the sympathetic nervous system causes a compensatory increase in heart rate to improve cardiac output.¹

Anesthetic management for a dialysis-dependent patient with high-output heart failure undergoing arteriovenous fistula ligation requires careful hemodynamic and volume control. These patients are sensitive to rapid fluid shifts and hemodynamic instability, so anesthetic agents should be selected to minimize myocardial depression and avoid excessive vasodilation. Inhalational agents and propofol, which can cause myocardial depression and vasodilation, should be carefully dosed.²

Following ligation of an arteriovenous fistula in a patient with high-output heart failure, central venous pressure (CVP) typically decreases due to reduced venous return and preload. Systemic vascular resistance (SVR) increases as the blood flow is redirected back to normal systemic circulation, alleviating the high-output state. Pulmonary artery (PA) pressure also decreases, as the reduced preload lessens the cardiac workload and pressure within the pulmonary vasculature. However, the sudden increase in afterload from AVF ligation can strain the left ventricle, particularly in patients with ventricular dysfunction. Intraoperatively, a thermodilution catheter can aid in diagnosing and managing left ventricular decompensation and assessing the impact of the procedure on cardiac output.³

REFERENCES

1. Stern AB, Klemmer PJ. High-output heart failure secondary to arteriovenous fistula. *Hemodialysis International*. 2011;15(1):104-107.
2. Shroff PK, Martin TW, Schmitz ML. Successful anesthetic management of a child with an extensive facial hemangioma and high output cardiac failure for placement of a central venous catheter. *Pediatric Anesthesia*. 2005;16(1):77-81.
3. Ninagawa J, Yamada Y. General anesthesia in a patient with Parkes Weber syndrome with high-output cardiac failure due to multiple arteriovenous fistulas complicated by severe aortic regurgitation. *Journal of Anesthesia*. 2010;24(2):256-259.