Anesthetic Management of a Patient With Severe Heart Failure Undergoing Craniotomy

A case-based discussion of balancing cardiac output, cerebral perfusion and intracranial pressure

Patient Profile

- 68-year-old man • 78 kg •
- Elective right frontal craniotomy and tumor resection
- Ischemic cardiomyopathy
- Chronic heart failure with reduced ejection fraction (HFrEF)
- Previous myocardial infarction (4 years earlier)
- Hypertension
- Type 2 diabetes mellitus
- Functional status: NYHA class II; (Mild exertional dyspnea)
- No orthopnea, paroxysmal nocturnal dyspnea
- No recent heart-failure admission

Preoperative Assessment

Parameter	Finding
LVEF	25–30%
Left ventricle	Dilated
Right ventricular function	Mildly impaired
Mitral regurgitation	Moderate
Estimated pulmonary artery pressure	42 mmHg
ECG	Sinus rhythm; old anterior MI
BP , HR	110/68 mmHg , 76 bpm

Laboratory	Value
Hemoglobin	13.2 g/dL
Creatinine	1.3 mg/dL
Potassium	4.3 mEq/L
Sodium	138 mEq/L

Neurosurgical Diagnosis & Medication Review

- MRI: right frontal intra-axial mass with surrounding vasogenic edema
- Mild midline shift (5 mm); no signs of acute herniation
- Planned procedure: elective tumor resection
- Home medications:
  - ✓ Bisoprolol
  - ✓ Sacubitril/valsartan
  - ✓ Spironolactone
  - ✓ Furosemide
  - ✓ Empagliflozin
- Continue chronic beta-blocker unless contraindicated
- Individualize management of other HF therapies
- Withhold empagliflozin for at least 3 days before elective surgery to reduce perioperative ketoacidosis risk

Major Anesthetic Challenges

Cardiac priorities	Neurosurgical priorities
Preserve cardiac output	Maintain cerebral perfusion pressure
Avoid myocardial ischemia and arrhythmia	Avoid hypoxia and major CO_2 shifts
Prevent pulmonary edema	Control intracranial pressure
Avoid abrupt changes in preload and afterload	Provide adequate brain relaxation
Prevent hemodynamic instability	Avoid excessive hypertension

The central conflict: avoid hypotension without provoking excessive afterload or pulmonary congestion.

Monitoring & Hemodynamic Goals

Monitoring	Purpose / target
5-lead ECG, SpO_2, capnography, temperature, neuromuscular monitoring	Standard monitoring
Arterial line before induction	Beat-to-beat BP; blood sampling
Central venous access (if indicated)	Vasoactive infusion
Cerebral oximetry (if available)	Trend regional cerebral oxygenation
TEE	Evaluate unexplained instability or suspected cardiac dysfunction

Variable	Illustrative goal
MAP	Individualize; often 70–80 mmHg initially
Heart rate	Avoid tachycardia; maintain adequate rate
$PaCO_2$	Usually 35–40 mmHg
Oxygenation	Avoid hypoxemia ,SpO2 > 94%
Fluid balance	Euvolemia; avoid both overload and hypovolemia

Induction of Anesthesia

The objective is to prevent myocardial depression, tachycardia and HOTN

- Preoxygenate
- Establish invasive arterial pressure monitoring before induction
- Etomidate 0.2–0.3 mg/kg may be considered for relative hemodynamic stability
- Fentanyl 2–3 mcg/kg to blunt sympathetic response (dose individualized)
- Neuromuscular blockade
- Gentle laryngoscopy and intubation; prevent tachycardia and hypertensive surges
- Have norepinephrine and other rescue vasoactive agents immediately available

Maintenance of Anesthesia & Ventilation

Component	Approach
Technique	Balanced anesthesia with low-dose volatile agent or TIVA; opioid infusion as appropriate
Neuromuscular blockade	Titrate to surgical requirements; monitor quantitatively
Tidal volume	6–8 mL/kg predicted body weight
PEEP	Individualize; avoid excessive intrathoracic pressure
$PaCO_2$	Aim for normocapnia (35–40 mmHg)
Brain relaxation	Treat causes of raised ICP; brief controlled hyperventilation only when indicated

Avoid prolonged hypocapnia: cerebral vasoconstriction may compromise cerebral blood flow.

Intraoperative Hypotension: Case Scenario

Finding 20 minutes after induction	Value
Blood pressure	78/45 mmHg
Heart rate	88 bpm
SpO_2	98%
$ETCO_2$	34 mmHg

- First assess: anesthetic depth, rhythm, preload, vasodilation and ventricular function
- Use TEE if the cause is unclear or cardiac dysfunction is suspected
- If vasodilatory hypotension predominates: consider a carefully titrated norepinephrine infusion
- If low cardiac output is confirmed: consider inotropic support (e.g., dobutamine) with close monitoring
- Give fluid only when assessment suggests fluid responsiveness or true volume depletion

Do not reflexively give a fluid bolus to every hypotensive patient with HFrEF.

Brain Relaxation & Fluid Management

- Maintain euvolemia
- Avoid both excessive crystalloid administration and hypovolemia
- Use isotonic fluids; avoid hypotonic solutions
- Mannitol can initially expand intravascular volume, then cause osmotic diuresis and hypovolemia
- In severe HFrEF, consider whether hypertonic saline is a suitable alternative for the specific clinical situation
- Monitor sodium, osmolality, renal function, urine output and hemodynamics
- Coordinate osmotherapy and fluid strategy with the neurosurgical team

Neither mannitol nor hypertonic saline is risk-free in heart failure; choose according to physiology and monitoring.

Emergence & Postoperative Management

Emergence goals	Neuro-ICU priorities
Smooth emergence; avoid coughing and bucking	Continuous ECG and blood-pressure monitoring
Prevent hypertension and tachycardia	Frequent neurological assessment
Avoid hypoxia and hypercapnia	Fluid balance and pulmonary edema surveillance
Maintain adequate cerebral perfusion	Renal function and electrolyte monitoring
Extubate only if appropriate	Biomarkers when clinically indicated

Extubation depends on neurological status, respiratory function and hemodynamic stability—not EF alone.

Audience Discussion

1. Would you postpone elective surgery in a patient with an EF of 25%?

2. Would you choose propofol or etomidate for induction—and why?

3. In post-induction hypotension, when would you choose norepinephrine versus an inotrope?

4. Would you use mannitol or hypertonic saline for brain relaxation?

5. What criteria would guide immediate extubation versus postoperative ventilation?

Q1: Would you postpone elective surgery in a patient with an EF of 25%?

Not necessarily.

An EF of 25% alone is not an absolute contraindication to elective noncardiac surgery. However, I would postpone elective surgery if the patient has:

- ✓ Acute decompensated heart failure.
- ✓ Pulmonary edema.
- ✓ Unstable arrhythmias.
- ✓ Active myocardial ischemia.
- ✓ Hemodynamic instability.

If the patient has compensated HFrEF:

- ✓ Optimize heart failure treatment.
- ✓ Assess functional capacity and volume status.

Key point: Clinical stability is more important than EF alone.

Q2. Etomidate vs Propofol for induction

Comparison in a patient with severe LV systolic dysfunction

Feature	Etomidate	Propofol
Hemodynamic profile	Usually causes less reduction in SVR and myocardial contractility; BP is generally better preserved.	Dose-dependent reduction in SVR and myocardial contractility; hypotension is more likely, especially with rapid bolus or hypovolemia.
Practical use in severe HFrEF	Often favored when cardiovascular reserve is limited and induction-related hypotension is a major concern.	Not contraindicated; use a reduced, slowly titrated dose and be ready to treat hypotension.
Adverse effects	Myoclonus, nausea/vomiting, transient inhibition of adrenal steroid synthesis.	Hypotension, myocardial depression may be important in severe HFrEF.
Adrenal effects	Transient 11β-hydroxylase inhibition after a single dose; clinical significance is debated.	No comparable inhibition of adrenal steroid synthesis.
Typical adult induction approach	Often 0.2–0.3 mg/kg IV, titrated to response.	Individualize and titrate slowly; avoid routine full-dose bolus in poor cardiac reserve.

Etomidate is a reasonable first choice here—not mandatory, and not proven to improve outcomes.

Q3. Phenylephrine vs Norepinephrine

Vasopressor choice for hypotension in a patient with severe HFrEF

Feature	Phenylephrine	Norepinephrine
Receptor profile	Predominantly pure α_1-adrenergic agonist.	Predominantly α_1 with some β_1 activity.
Effect on SVR / BP	Marked increase in SVR and arterial pressure.	Increases SVR and arterial pressure, usually with less purely alpha-mediated afterload effect.
Effect on cardiac output	May reduce stroke volume and cardiac output by increasing LV afterload; reflex bradycardia can further reduce output.	Often better preserves cardiac output because of modest β_1 activity; it can still increase LV afterload and must be titrated.
Heart rate	Can cause reflex bradycardia.	Usually less reflex bradycardia; may cause or aggravate tachycardia in some patients.
Potential role in HFrEF	Less attractive when severe LV systolic dysfunction or low output is a concern. May be useful in selected settings, such as significant tachyarrhythmia or dynamic LVOTO.	Generally preferred for vasodilatory hypotension in severe HFrEF when a vasopressor is required, particularly when preserving cardiac output is important.
Main cautions	Excessive afterload increase, bradycardia, and deterioration in cardiac output.	Excessive vasoconstriction/afterload, tachyarrhythmia, and peripheral or splanchnic ischemia at high doses.

Take-home: Norepinephrine is usually favored over phenylephrine for vasodilatory hypotension in severe HFrEF; identify the cause and reassess cardiac output.

Q3. Norepinephrine or Dobutamine?

- First identify the mechanism: anesthetic-related vasodilation, low preload, bleeding, arrhythmia, or low cardiac output.
- Norepinephrine: appropriate when vasodilation/low SVR is dominant and arterial pressure needs support.
- Dobutamine: consider when there is evidence of low cardiac output from impaired contractility, particularly with adequate preload and perfusion pressure.
- If vasodilation and low output coexist, norepinephrine and a carefully titrated inotrope may be needed together.
- Reassess using arterial waveform, TEE when available, perfusion markers, and treatment response; do not treat the MAP number alone.

Do not give an inotrope for every low BP—or a vasopressor without considering LV afterload.

Q4. Mannitol vs Hypertonic Saline

Neither agent is universally safer in HFrEF

Feature	Mannitol	Hypertonic saline (HTS)
Main action	Osmotic diuretic; draws water from brain tissue, followed by renal excretion.	Raises serum sodium/osmolality and draws water from brain tissue into the intravascular compartment.
Intravascular volume	May initially expand plasma volume, then produce osmotic diuresis and volume loss.	Expands intravascular volume and may increase preload.
Potential HF concern	Subsequent diuresis can cause hypovolemia, hypotension, and reduced cardiac output.	Increased preload may worsen pulmonary congestion or edema in susceptible patients.
Renal / laboratory issues	Use caution in renal impairment or accumulation; monitor renal function, osmolality and electrolytes.	Monitor sodium, chloride, osmolality, acid–base status, and renal function; avoid excessive hypernatremia/hyperchloremia.
When it may be considered	When renal function and hemodynamics permit and volume depletion is not a concern.	When avoiding a diuretic phase is important, provided the patient is not congested or markedly preload-sensitive.

Choose according to congestion vs preload dependence, renal function, sodium/chloride, and osmolality.

Q5. Extubate or continue postoperative ventilation?

EXTUBATE WHEN	CONTINUE VENTILATION / ICU WHEN
➢ Hemodynamics stable without escalating vasoactive support. ➢ Adequate oxygenation, ventilation, and respiratory mechanics. ➢ Awake enough to protect the airway and permit neurologic assessment. ➢ Hemostasis satisfactory; no significant pulmonary edema or ongoing instability.	➢ Persistent low output, significant hypotension, or escalating vasoactive support. ➢ Pulmonary edema, poor gas exchange, or inadequate respiratory reserve. ➢ Delayed awakening, impaired airway protection, or concerning neurologic status. ➢ Ongoing bleeding, major surgical concerns, or need for controlled postoperative care.

An EF of 25% alone is not an indication for routine postoperative intubation.

Selected References & Key Caveat

Thompson A, et al. 2024 AHA/ACC Guideline for Perioperative Cardiovascular Management for Noncardiac Surgery. Circulation. 2024.

Heidenreich PA, et al. 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure. Circulation. 2022.

Miller’s Anesthesia. Sections on neuroanesthesia and perioperative cardiovascular management. Latest available edition.

Cottrell and Patel’s Neuroanesthesia. Relevant chapters on intracranial pressure, cerebral perfusion and neuroanesthetic management.

Educational case only. Drug choices, targets and timing must be individualized to the patient, procedure and institutional protocols.