Fluid Management in Neurosurgical Patients with Coexisting Cardiac Diseases

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Abstract

Neurosurgical patients with underlying cardiac diseases are very sensitive to fluid management. They need careful preoperative cardiac evaluation regarding myocardial ischemia, significant arrhythmia, valvular heart disease, and heart failure. Presence of heart failure in preoperative period will increase perioperative cardiac morbidity. Some may have heart failure. Some may have euvolemia or even hypovolemia. Fluid assessment and fluid responsiveness in cardiac patients are very important. Fluid challenge can be given in cardiac patients. In responders, hemodynamic parameters will change to favorable results. Bedside echocardiogram and passive leg raising (PLR) test are excellent tools for assessing fluid responsiveness. In patient with hypovolemia and hypotension, resuscitation fluid should focus on rapid restoration of circulating volume. Intraoperative hypotension can be minimized with goal-directed fluid combined with protocol management. Postoperative IV fluid requirements should be low in most cases. Fluid balance chart and serum electrolytes should be checked. Cardiac complications (myocardial ischemia, atrial fibrillation, and heart failure) occur commonly, and some neurological conditions such as neurogenic stunned myocardium (NSM) can cause myocardial dysfunction. Appropriate fluid and blood pressure management may decrease or prevent major cardiac events.

Keywords

Cardiac complications · Cardiac patient · Fluid assessment · Fluid responsiveness · Goal-directed therapy (GDT) · Hypotension

Introduction

Fluid management of neurosurgical patients with coexisting cardiac disease is a great challenge to anesthesiologists in perioperative period. Patients with underlying cardiac diseases are very sensitive to fluid management. Too much fluid can cause congestive heart failure. Too little fluid leads to inadequate preload, low cardiac output, hypotension, and poor tissue perfusion. Intravascular volume of neurosurgical patients can be altered by several etiologies, such as receiving diuretics (mannitol and furosemide) to reduce intracranial pressure or to provide brain relaxation. Anesthetics cause systemic vasodilatation and relative hypovolemia. Surgery can also result in massive bleeding. Development of diabetes insipidus (DI) or syndrome of inappropriate

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antidiuretic hormone (SIADH) secretion during perioperative period may further lead to fluid and electrolyte imbalance [1]. This chapter provides details about administering fluid in cardiac patients during perioperative period for neurosurgical procedures.

Preoperative Assessment of Cardiac Patients

Apart from neurological assessment, cardiac patients need careful preoperative evaluation regarding cardiac function and pathologies such as myocardial ischemia, significant arrhythmia, valvular heart disease, and heart failure. Presence of heart failure in preoperative period will increase perioperative cardiac morbidity. Stabilization of cardiac function and treating pulmonary congestion should be done before elective surgery. Evaluations with respect to exercise tolerance and functional capacity are also very important. They determine perioperative risk, need for invasive monitoring, and special postoperative care. Some may need further cardiac test or intervention [2–7].

Symptoms, signs, and investigation of patients with heart diseases are shown in Table 1.

Table 1 Symptoms, signs, investigation, and interpretations [2–7]

	Interpretation
History	
Breathlessness, orthopnea, nocturnal dyspnea Palpitation, dizziness, syncope Severity and triggers of dyspnea and fatigue	Symptoms of heart failure Symptoms of heart failure To determine NYHA class
Presence of chest pain, history of CAD History of long-standing or poorly controlled hypertension	Symptoms of coronary ischemia Hypertensive heart disease, diastolic dysfunction
Exercise capacity, physical activity	Functional capacity
Weight: loss or gain	GI dysfunction (cachexia) or fluid overloaded
Medication adherence	Access for medication, follow-up
Salt-containing diet	Sodium retention
Physical examination	
Blood pressure (supine and upright)	Hypertension or hypotension (postural change)
Pulse: tachycardia, irregularity, narrow pulse pressure	Signs of heart failure, arrhythmias
Tachypnea, lung crepitation Jugular venous pressure, hepatojugular reflux	Signs of heart failure Identify congestion
Size and location of apical impulse S 3 gallop Cardiac murmur Hepatomegaly, ascites, peripheral edema Cooled lower extremities, oliguria	Ventricular enlargement Ventricular dysfunction Structural heart disease Fluid overloaded Inadequate cardiac output
Investigation	
Electrocardiogram (ECG)	Ischemic pattern, left ventricular hypertrophy (LVH)
Chest X-ray	Cardiomegaly, congestion, Kerley B line, pleural effusion
Blood test: cardiac troponin, BNP or NT-proBNP Other blood test: hemoglobin and WBC—sodium, potassium, urea, creatinine (with eGFR), liver function tests (bilirubin, AST, ALT, GGTP), glucose, HbA1c, lipid profiles	Myocardial ischemia, heart failure For initial assessment for other organ functions
Echocardiogram Other cardiac testing such as cardiac magnetic resonance, stress imaging, coronary angiography (according to cardiologist and guidelines)	Structural heart, ventricular function, volume status, hemodynamic parameters Myocardial structure and function

CAD coronary artery disease, *NYHA* New York Heart Association, *BNP* B-type natriuretic peptide, *NT-proBNP* N-terminal pro-B-type natriuretic peptide, *WBC* white blood cell, *eGFR* estimated glomerular filtration rate, *AST* aspartate aminotransferase, *ALT* alanine aminotransferase, *GGTP* gamma-glutamyl transpeptidase, *HbA1c* hemoglobin A1c

Fluid Assessment and Fluid Responsiveness

Fluid assessment and fluid responsiveness in cardiac patients are very important. Only half of hypotensive patients respond to fluid resuscitation [8]. Some cardiac patients may have heart failure and overloaded intravascular volume. However, some may have euvolemia or even hypovolemia. Hypovolemia is common during perioperative period due to several factors such as overuse of diuretics (thiazides or furosemide or mannitol or combination of diuretics), and/or inadequate intake, and/or severe nausea or vomiting (in case of raised intracranial pressure) and/or ongoing blood loss in multiple trauma [1]. Despite advance in laboratory investigations and imaging technology, careful history taking and meticulous physical examination are essential for fluid status evaluation [9].

Common symptoms, signs, monitoring, and investigation for fluid status evaluation and fluid responsiveness are shown in Table 2 [8–13]. However, many symptoms and signs are too general and not specific to hypovolemia or hypervolemia. In those cases, some investigations may be needed. For example, hypotension can be caused by several factors, not only from hypovolemia. In hyperglycemic state, renal insufficiency or use of diuretics, SIADH or DI, urine output may be an inaccurate measure of volume status and resuscitation.

Fluid challenge can be given to cardiac patients with low systolic blood pressure (SBP <90 or 100 mmHg) or low mean arterial pressure (MAP <60 or 70 mmHg), with tachycardia (heart rate >90 or 100 beats/min), or with other signs of hypovolemia (Table 2). Crystalloid or colloid (in cases with no acute kidney injury or no sepsis) can be given in a bolus with volume of 4–10 mL/kg

Table 2 Assessment of fluid status and fluid responsiveness [8–14]

History	Monitoring (noninvasive or invasive)
Hypovolemia Fluid loss via urine, the gastrointestine, or the skin Blood loss Low or inadequate intake Hypervolumia Excessive intake in patients with heart failure or chronic kidney disease Excessive fluid resuscitation	Vital signs or national early warning scores (NEWS) Fluid balance chart, weight Blood urea nitrogen, creatinine, electrolytes Urine output Stroke volume variation or pulse pressure variation (if indicated) Cardiac output (if indicated) Central venous pressure (CVP) or pulmonary capillary wedge pressure (PCWP) (if indicated)

Physical examination	Investigation
Hypovolemia Altered mental status	Hypovolemia Elevated hematocrit
Dry mucous membranes	Low bicarbonate level
Poor skin turgor	Increased base deficit
Tachycardia (heart rate >90/min)	Increased lactate level
Hypotension (systolic blood pressure <100 mmHg or orthostatic changes)	Increased blood urea nitrogen/creatinine ratio
Capillary refill >2 s Peripheries cold to touch 45° passive leg raising suggests fluid responsiveness Weight loss	Increased urine osmolality or concentrated urine Decreased fractional excretion of sodium
Hypervolumia Weight gain High jugular pressure Hepatomegaly, ascites, peripheral edema Lung sound (rales or crepitation)	Hypervolemia Chest X-ray (lung congestion) Echocardiography (large left ventricular volume, large IVC diameter) Lung ultrasound (B line and pleural effusion)

National early warning scores (NEWS) = score developed by the Royal College of Physicians (UK) by using six physiologic parameters: respiratory rate, oxygen, temperature, systolic blood pressure, pulse rate, and consciousness

(100–500 mL or more) to most patients [8–12]. However, in cardiac patients with impaired cardiac function, a slower bolus rate is preferred. However, when infusion time was longer than 30 min, proportion of responder was decreased [8]. In responders, hemodynamic parameters (macrocirculation) will change to favorable results such as increasing in blood pressure, increasing in cardiac output, or decreasing in pulse rate. Microcirculation such as lactate, prolonged capillary refill time, and mottling score will also be improved [10, 11]. Frequent monitorings and re-evaluation are essential for fluid adjustment.

Those with negative response to passive leg raising (PLR) test are unlikely to be fluid-responsive. PLR can be used in combination with other goal-directed fluid therapies [8].

In sick patients on ventilator with an arterial line, hemodynamic parameters such as changes in pulse pressure (ΔPP) during positive pressure ventilation or pulse pressure variation (PPV) can be used for fluid responsiveness. In patients who breathe spontaneously, PPV can be used in conjunction with a maneuver that increases intrathoracic pressures, i.e., Valsalva maneuver or passive leg raising test [12].

Bedside echocardiogram is another excellent tool for assessing fluid responsiveness. Disadvantages of echocardiogram are noncontinuous monitoring and high inter-operator variability. Pulse contour analysis or other noninvasive cardiac output monitoring can also be used for fluid responsiveness [8, 13].

Echocardiogram/Transthoracic Ultrasound for Fluid Management

Myocardial dysfunction in cardiac patients may be diastolic, systolic, or both systolic and diastolic dysfunctions. Some cardiac patients have abnormalities in valve function (stenosis or regurgitant or both) or arrhythmias. Preoperative echocardiogram gives some valuable details about cardiac volume, cardiac function, and valve abnormalities, but it is not always needed in every case of cardiac patients. However, echocardiogram is essential for cardiac patients with unstable hemodynamics or doubtful volume status [13].

Heart failure may result from disorders of pericardium, myocardium, endocardium, heart valves, great vessels, or other metabolic abnormalities. Most commonly, HF patients have left ventricular (LV) myocardial disease. Some patients have normal LV size and preserved ejection fraction (EF), whereas others have cardiac chamber dilatation and/or reduced EF [5–7].

Optimal filling volume for cardiac patient has a very narrow range. Outside this narrow range, stroke volume and cardiac output may be decreased because of hypovolemia or hypervolemia. When stroke volume has been optimized with adequate fluid and/or the use of inotropes, cardiac output will undoubtedly be improved providing good tissue perfusion. Optimum intravascular volume will also prevent reflex tachycardia from hypovolemia. Tachycardia increases myocardial oxygen consumption and puts patients with coronary artery disease at risk [5–7].

Positive pressure ventilation increases the size of inferior vena cava (IVC), while negative pressure reduces its size. A large, noncompliant IVC implies patient is not a volume responder. IVC diameter is easily and reproducibly measured 1–2 cm from right atrial junction using transthoracic ultrasound [13]. Lung ultrasound can be used for fluid status assessment. B line and pleural effusion suggest fluid overloaded [14].

Types of Fluid Management

Different indications need different types of fluid. In patient with hypovolemia and hypotension, resuscitation fluid should focus on rapid restoration of circulating volume, so high sodium (Na 130–154 mmol/L) containing fluid such as 0.9% NaCl or balanced salt solutions should be used [9, 10, 15].

From neurosurgical view point, crystalloids should be used as first-line resuscitation fluid. Synthetic colloids should not be used as resuscitation fluid regarding its affecting platelet function. Glucose-containing hypotonic solutions, albumin, and other hypotonic solutions should not be used as resuscitation fluid [16].

Replacement fluid must mimic fluid that has been lost such as gastrointestinal loss from vomiting or urine loss from DI. Maintenance fluid must deliver basic electrolytes (approximately 1 mmol/kg/day of potassium, sodium chloride, and water 25–30 mL/kg) and glucose for metabolic needs (50–100 g/day of glucose to limit starvation ketosis) [9, 15].

Patient electrolytes also guide fluid management. For example, in patient with acute or symptomatic hyponatremia, a bolus of 100–150 mL of hypertonic saline (3% NaCl) over 10–20 min could be given. For a moderate symptom, use continuous infusion of 3% NaCl 0.5–2.0 mL/kg/h. However, in asymptomatic or mild hyponatremia, a slower rate of 3% NaCl or 0.9% NaCl can be used [17]. In patient with hypervolemic hyponatremia or cardiac patient with impending heart failure, hypertonic saline can be combined with loop diuretic. Frequent glucose and electrolyte monitoring is essential because electrolyte imbalance and hypoglycemia or hyperglycemia have deleterious effects on neurological function.

Dosage and duration are equally important for fluid management. For resuscitation, a bolus of 500 mL <15 min is recommended [9]. For replacement fluid, when cause of fluid loss is stopped, replacement is no longer needed. Final step in fluid therapy is to withhold fluid when it is no longer required, thus reducing the risk of fluid overload and deleterious effects [10]. In cardiac patient, invasive or sophisticated monitoring may be needed if, somehow, fluid responsiveness is in doubt.

Perioperative Fluid Management

To avoid a bolus fluid loading in cardiac patient, fluid deficit should be corrected or prevented before surgery.

Current guidelines allow intake of clear oral fluid up to 2 h before elective surgery [18]. For enhanced recovery after surgery (ERAS) guideline, carbohydrate-rich drink is suggested to be given 2–3 h before operation to decrease insulin resistance and avoid dehydration from prolonged fasting [19]. However, fluid may need to be restricted in patients with symptoms and signs of heart failure (HF). Symptoms and signs of HF are dyspnea, fatigue, limited exercise tolerance, and fluid retention. Cardiologist consultation is necessary in cardiac patients with acute heart failure. Elective surgery might need to be postponed because acute heart failure increases perioperative cardiac risk.

Intraoperative Fluid Management

Hourly fluid maintenance can be calculated by summation of fluid deficit + maintenance fluid + blood loss + surgical loss or third-space loss. Third-space loss remains unclear, and interstitial fluid loss is returned to circulation by lymphatic system. Interstitial fluid shift is related to endothelium glycocalyx. In sepsis or systemic inflammatory response, there is fluid leakage via endothelium glycocalyx. In conclusion, third-space loss is no longer included in calculation for fluid management [15].

Restrictive vs. liberal fluid therapy for major abdominal surgery (RELIEF) trial showed a high incidence of postoperative acute kidney injury (AKI) in restrictive group. Restrictive group received a <5 mL/kg bolus at anesthesia induction, followed by intraoperative crystalloid infusion at a rate of 5 mL/kg/h. Liberal group received a 10-mL/kg bolus at induction, followed by an intraoperative rate of 8 mL/kg/h [20]. Zero fluid balance is too restrictive, and a somewhat liberal approach may be needed. Therefore, evidence-based practices in non-thoracic surgeries are moving away from a restrictive fluid strategy toward maintaining euvolemic state or 1–2 L of positive fluid balance in general low-risk population [20, 21].

Individualized goal-directed fluid (GDT) therapy is proven to be beneficial in several outcomes. It can reduce mortality in high-risk patients or high-risk surgeries/procedures. GDT reduced rates of arrhythmia; however, myocardial infarction (MI), HF, and cardiac arrest were not significantly different between groups. High-risk patients included cardiac patients and patients with severe comorbidities [22]. Most neurosurgical procedures are classified as moderate- or high-risk procedures with a great change of fluid during operation. Arterial line or invasive blood measurement is normally used in craniotomy and major spine surgery, so pulse pressure variation can be used for fluid responsiveness. For very sick cardiac patient with low ejection fraction, continuous cardiac output monitoring may be useful, not only for fluid titration but also for inotrope and vasopressor management [23]. Combining goal-directed fluid monitoring with protocol management (Fig. 1) may improve patients' outcome.

Intraoperative Hypotension

After anesthesia induction, many cardiac patients may experience severe hypotension because of preexisting hypovolemia, anesthetic-induced vasodilatation, and positive pressure ventilation. These patients are on ascending limb of Frank-Starling curve and will benefit from individualized, goal-directed intravascular fluid administration. Factors associated with post-induction hypotension are pre-induction systolic blood pressure, elderly, and emergency surgery [24]. A slow fluid bolus (10–20 min) at time of induction and a slow titration of anesthetic agents or using induction agents such as thiopentone or etomidate have

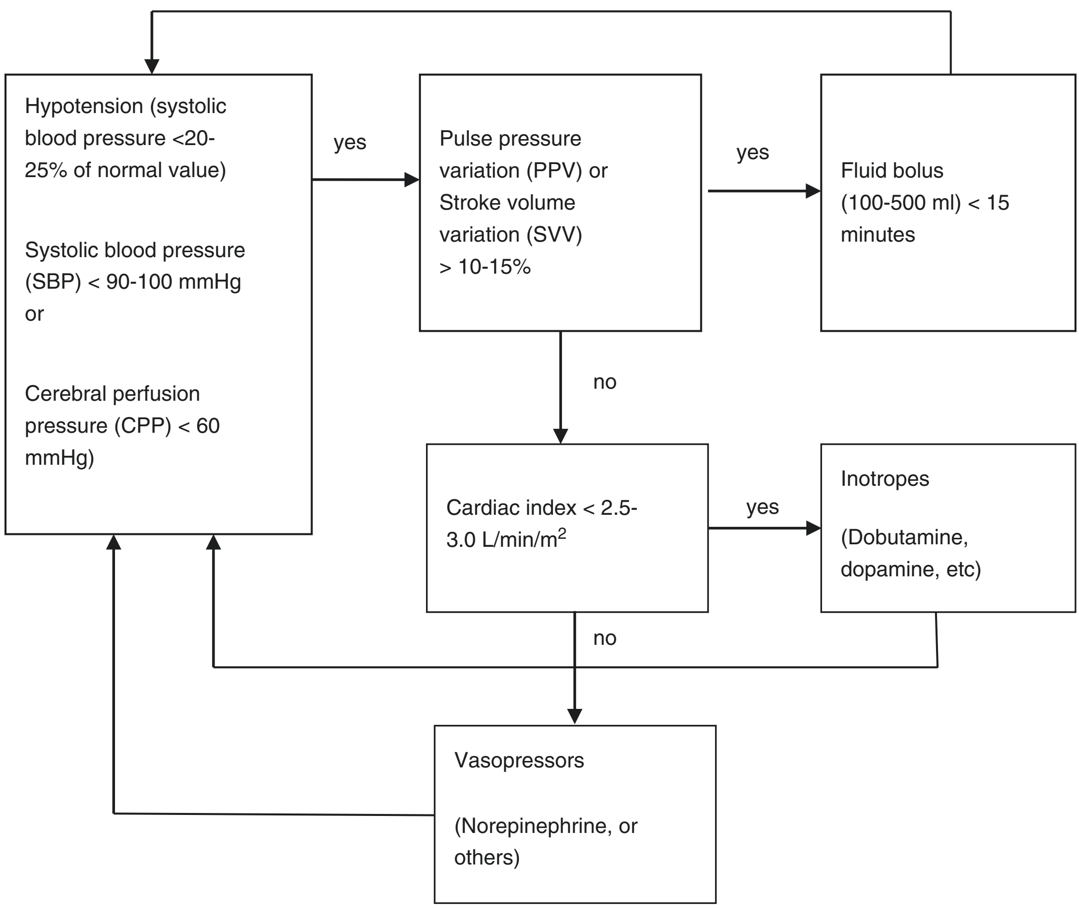

Fig. 1 Protocol for goal-directed fluid therapy in hypotensive patient

less myocardial depressant effect compared with propofol. A single dose of etomidate can decrease cortisol production, but adrenal insufficiency is unlikely [25].

Perioperative hypotension frequently occurs in critically ill or cardiac patients. Vasopressor and/or inotropic drugs should be prepared and ready for use. It has been shown in the study that intraoperative hypotensive episodes of as little as 1–5 min can be associated with an unfavorable outcome [26]. In contrast, routine use of vasopressor to prevent and treat hypotension might mark symptoms and signs of hypovolemia in some cardiac patients. In repeated hypotension despite adjustment in anesthetic agent, hypovolemia may be the cause, and fluid challenge might provide some benefits (Fig. 1).

To treat hypotension, balanced salt solution is preferred. However, acetate or lactate ringer solution is slightly hypotonic (273 mmol/L) which leads to cerebral edema. A large amount of 0.9% NaCl (308 mmol/L) leads to hyperchloremic metabolic acidosis. So mixed crystalloid can be used to avoid such side effects. However, hydroxyethyl starch (HES) solution can potentially cause acute kidney injury in septic patients. In hypovolemic or hemorrhagic patient, a small amount of colloid prior to transfusion can be given. Colloid lasts longer in intravascular space and reduces interstitial edema [21].

Blood Transfusion

There was no evidence that restrictive transfusion strategy (hemoglobin (Hb) 7 g/dL to 8 g/dL) affects 30-day mortality or morbidity (cardiac events, stroke, pneumonia, thromboembolism, infection) compared with liberal transfusion strategy (Hb 9 g/dL to 10 g/dL). There were insufficient data to draw a conclusion in clinical subgroups such as cardiac patients with acute coronary syndrome, myocardial infarction, or neurological patients with traumatic brain injury and stroke or patients with bleeding risk [27]. Recently published blood transfusion guidelines [28, 29] supported restrictive transfusion strategy. A restrictive red blood cell (RBC) transfusion threshold of 8 g/dL is recommended for patients undergoing orthopedic surgery and cardiac surgery and those with preexisting cardiovascular disease [28]. Implementation of patient blood management improved appropriate RBC utilization [29, 30].

For cardiac patient, acceptable level of hemoglobin is higher compared with other population, so triggering point for transfusion is 8 g/dL [28] or even higher. In neurosurgery, there were some evidences in patients with traumatic brain injury (TBI) and subarachnoid hemorrhage to study transfusion trigger of red blood cells. An Hb level <9 g/dL in patient with subarachnoid hemorrhage was associated with an increased incidence of brain hypoxia and cell energy dysfunction [31]. TBI patients with Hb concentration >9 g/dL were associated with an improved 6-month functional outcome [30, 31]. Clinical experiences played an important role in transfusion practice in neurosurgery.

Preoperative hemoglobin concentration is usually higher than normal due to fasting. Allowable blood loss from calculation is usually too high. In neurosurgical procedures, estimated blood loss is sometimes difficult due to irrigating fluid. Repeated hemoglobin measurement is essential during operation with ongoing blood loss. Interpretation of hemoglobin level should be adjunct with fluid status. A false high Hb level usually shows in acute hemorrhagic setting.

Hyperosmolar Therapy and Hemodynamic Changes

Hyperosmotic therapy (mannitol and hypertonic saline) is commonly used to reduce intracranial pressure and provide brain relaxation. Both agents increase osmolality and induce water shift through intact blood-brain barrier. They increase initially central venous pressure, cardiac output, cardiac index, lower systemic vascular resistance, and variable results with blood pressure [32, 33]. Administration of hypertonic saline heightens level of serum sodium and promotes a temporary reduction of potassium. In contrast, mannitol causes a transient acute dilutional hyponatremia with a concomitant increase of potassium [34, 35]. Urine output is significantly higher in patients treated with mannitol.

In cardiac patients, hyperosmolar therapy can induce heart failure due to initial increase in intravascular volume and can cause significant hypotension after mannitol-induced diuresis. Fluctuation in intravascular volume can cause hemodynamic instability, so lower dose and slower rate of infusion of hyperosmolar therapy in these cardiac patients might provide more stable hemodynamics. Electrolyte abnormalities undoubtedly affect cardiac patients than general population. Hypokalemia and hypomagnesemia can precipitate cardiac arrhythmias.

Postoperative Fluid Management

Excessive fluid administration has a negative impact on recovery after major surgery. Patients recovering from major surgery typically have a 3–4-kg weight gain secondary to fluid and salt overload, manifesting as tissue edema [15]. However, restrictive vs. liberal fluid therapy in major abdominal surgery (RELIEF) trial showed a high incidence of postoperative acute kidney injury (AKI) in restrictive group or zero balance group. Study suggested maintaining euvolemic state or 1–2 L of positive fluid balance in general low-risk population [20]. Excessive fluid can slowly be self-eliminated in postoperative period or with the use of diuretics.

Postoperative intravenous fluid requirements should be low in most cases unless there are ongoing fluid and electrolyte losses such as DI in pituitary surgery. Patients should be encouraged to resume oral fluid as soon as possible after surgery. Intravenous cannula can be locked, but no administered fluid, only for parenteral antibiotics or other drugs.

Patient's fluid balance should be charted and serum electrolytes checked, especially after osmotic therapy. Regular postoperative assessment of patient's fluid status and requirements should include asking for thirst and looking for signs of hypovolemia or hypervolemia (Table 2). Urine output can be an unreliable monitor of fluid status in postoperative neurosurgical patients.

Goal-directed fluid therapy and fluid responsiveness can continuously be used in postoperative period. However, this period's fluid shift or fluid loss is less than intraoperative period. Cardiac patient who is hemodynamically stable can be managed with standard care.

Common Cardiovascular Complications

Cardiac complications occur commonly in perioperative period, especially patients with coexisting cardiac diseases. Perioperative myocardial infarction or injury occurs up to 3% in patients undergoing elective major noncardiac surgery [34, 35]. Troponin surveillance provides early myocardial injury detection, and its level relates to cardiac prognosis. Stable hemodynamics can be achieved with appropriate fluid and electrolyte management. Mean arterial pressure <55 mmHg increases incidence of myocardial injury after noncardiac surgery (MINS) [26].

Apart from cardiothoracic surgery, incidence of postoperative atrial fibrillation (AF) is 3–10% [34]. Precipitating factors are not always known, but catecholamine stress, myocardial ischemia, pain, hypovolemia or atrial stretch, hypoxia, and electrolyte disturbances have all been implicated. Loss of atrial contraction reduces stroke volume, and tachycardia may cause myocardial ischemia. Some patients with postoperative AF episodes are asymptomatic, but cardiac patients with limited or reduced cardiac function can frequently become hemodynamically unstable. This acute AF may need pharmacological or electrical cardioversion to restore sinus rhythm. Some patients who fail from rhythm control AF should benefit from rate-controlled AF. Postoperative AF should be treated the same way as AF from any other cause, and stroke risk should be assessed with a validated scoring system such as CHADS2-VASC and anticoagulation started if indicated [36]. However, after major neurosurgery, bleeding risk should be assessed before starting anticoagulant. Atrial fibrillation is a leading cause of ischemic stroke from embolic event, but hemorrhagic stroke occurs from the use of anticoagulant. Management of arrhythmias is a shared responsibility of critical care intensivists and cardiologists. Arrhythmias may arise from intrinsic cardiac pathology or from secondary precipitating factors.

Majority of patients with postoperative heart failure also have preoperative heart failure. Heart failure is rare in noncardiac patient as a solely postoperative complication. Some of patients with acute coronary syndrome will develop systolic heart failure as a consequence of myocardial ischemia. Other causes are inappropriate fluid type and volume, perioperative lung injury as neurogenic pulmonary edema, preexisting renal impairment, and sepsis. Patients with preoperative diastolic dysfunction (long-standing hypertension with left ventricular hypertrophy) can experience postoperative pulmonary edema, myocardial infarction, and other major cardiac events. It has been suggested that using some forms of cardiac output monitoring to optimize stroke volume as well as avoiding tachycardia may lower cardiac morbidity [37].

Hypervolemia in triple-H therapy (hypervolemic, hypertensive, and hemodilution) was used to treat patients with delayed cerebral ischemia in postoperative period. However, it caused pulmonary edema without improving neurological outcomes. So hypervolemia is no longer recommended. In cardiac patients who are very sensitive to fluid overload, this therapy should not be used [16].

Some neurological conditions can cause myocardial dysfunction such as neurogenic stunned myocardium or neurogenic stress cardiomyopathy. It is characterized by ischemic electrocardiography (ECG) changes, increased cardiac biomarker, and reversible left ventricular dysfunction (cardiomyopathy) without evidence of epicardial coronary artery disease. It is related to subarachnoid hemorrhage or other neurologic etiologies. Neurogenic stunned myocardium sometimes can lead to cardiogenic shock and neurogenic pulmonary edema. Cardiogenic shock is treated with inotrope to optimize cardiac output and maintain cerebral perfusion pressure. Drugs that prolong QTc interval such as antidepressants should be avoided. Although this type of cardiomyopathy is reversible, patients with NSM exhibit poor outcome and high mortality [38].

Conclusion

Perioperative fluid management in cardiac patients is very crucial. Fluid should be optimized starting from preoperative period to intraoperative and postoperative period. Hemodynamically unstable cardiac patient may benefit from goal-directed individualized fluid therapy to prevent perioperative major cardiac events.

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