Chapter 28
Anesthesia for Intracranial Vascular Surgery

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Abstract Subarachnoid hemorrhage (SAH) secondary to ruptured cerebral aneurysm carries a poor outcome and high mortality. There are various types of cerebral aneurysm and several risk factors for aneurysm development or rupture.

Preoperative grading scales, such as the Hunt and Hess scale, are useful to estimate prognosis. In addition to physical symptoms (sudden severe headache, nausea, or vomiting), SAH leads to systemic physiological responses in patients. Complications of SAH are not only intracranial but also extracranial (myocardial ischemia, arrhythmia, or neurological pulmonary edema). Recently, CT angiography has become essential to investigate ruptured aneurysms.

Preoperative management of cerebral vasospasm, arrhythmia, pulmonary edema, hypovolemia, and hyponatremia is required. Premedication differs according to the patient's grade. There are many monitoring systems (invasive and noninvasive) for the central nervous system. The induction of anesthesia and the hemodynamic response should be controlled adequately without compromising cerebral perfusion pressure. Total intravenous anesthesia is preferred particularly when motor evoked potential measurement is performed. Artificial ventilation is controlled to prevent hypercapnia which may increase intracranial pressure. Triple-H therapy may be a reliable method to prevent postoperative cerebral vasospasm. Adequate postoperative pain management is thought to inhibit hypertension or tachycardia, which may become worse in the patient.

Keywords Subarachnoid hemorrhage (SAH) from a cerebral aneurysm • Cerebral vasospasm • Anesthetic management

28.1 Introduction

Cerebral aneurysms are the most common cause of subarachnoid hemorrhage (SAH) excluding trauma. An SAH destroys cerebrovascular autoregulation and induces not only intracranial but also serious systemic complications, such as

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increasing intracranial pressure, cerebral vasospasm, or cardiopulmonary disturbance, with high mortality. Anesthesiologists should be familiar with the pathophysiology and treatment of SAH resulting from a cerebral aneurysm. At the beginning of this chapter, basic knowledge, clinical symptoms, and complications of cerebral aneurysm and SAH are reviewed, followed by a discussion of anesthetic management from the preoperative to postoperative period.

28.2 Pathophysiology of Cerebral Aneurysms

28.2.1 Epidemiology

SAH comprises only 1–7 % of all strokes; however, it has a relatively younger age of onset and poorer outcome than cerebral infarction [1]. The incidence of SAH secondary to ruptured cerebral aneurysm is 11–16 cases per 100,000 population per year [2, 3] and its mortality is 32–67 % [4], in agreement with previous reports. The incidence of unruptured aneurysm is 5 % [5], and if the size is < 10 mm and there is no history of subarachnoid hemorrhage, the incidence of rupture is 0.05 % per year [6].

28.2.2 Types and Location of Cerebral Aneurysms

28.2.2.1 Types

Saccular (berrylike) aneurysms are common and most are classified as small, being less than 12 mm in diameter. Giant aneurysms are more than 25 mm [7] and occasionally measure up to 10 cm. Fusiform aneurysms are often associated with severe atherosclerosis. Other types of aneurysms include dissecting, traumatic, and mycotic aneurysms.

28.2.2.2 Location

The most common locations are the anterior communicating artery (ACoA), internal carotid-posterior communicating artery (ICPCoA), and middle cerebral artery (MCA) bifurcation. The emergence of cross flow into the ACoA contributes to the formation of aneurysms [8]; therefore, hemodynamic stress may relate to the formation of cerebral aneurysms at a vessel's branching point, such as the ICPCoA or MCA bifurcation.

28.2.3 Risk Factors

28.2.3.1 Risk Factors for Developing Aneurysms

Risk factors for developing aneurysms include a family history of the disease (unruptured aneurysms or SAH), hypertension, smoking, atherosclerosis, previous infection, fibromuscular dysplasia, polycystic kidneys, coarctation or hypoplasia of the aorta, and connective tissue disorders (Ehlers-Danlos syndrome, Marfan syndrome, pseudoxanthoma elasticum).

28.2.3.2 Risk Factors for Ruptured Aneurysms

Risk factors for ruptured aneurysms depend on the size (more likely if > 6 mm [9]), morphology, location, and any previous history of SAH [10].

28.2.4 When an Aneurysm Ruptures

The pathophysiologic changes [10] include a sudden increase in intracranial pressure (ICP), decrease in cerebral blood flow (CBF) and cerebral perfusion pressure (CPP), loss of cerebrovascular autoregulation, spread of blood through the subarachnoid space causing inflammation and meningism, and cerebral vasoconstriction.

28.2.4.1 Treatment of Increased ICP

Treatments for increased ICP include maintaining a head-up position with sufficient fluid infusion, mild hyperventilation, intravenous infusion of osmotic diuretic agents, bolus or continuous administration of barbiturates, mild hypothermia, and external ventricular drainage of cerebrospinal fluid (CSF).

28.3 Preoperative Assessment

28.3.1 Preoperative Grading Scales

28.3.1.1 Neurological Symptom Grading Scales

The Hunt and Hess scale has been used widely as a preoperative neurological symptom grading scale (Table 28.1 [11]). As the World Federation of Neurological Surgeons (WFNS) grading scale is based on the Glasgow coma scale (GCS), the

Table 28.1 Hunt and Hess grading scale [11]

Grade	
I	Asymptomatic or minimal headache and slight nuchal rigidity
II	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
III	Drowsiness, confusion, or mild focal deficit
IV	Stupor, moderate to severe hemiparesis, possibly early decerebrate rigidity, and vegetative disturbances
V	Deep coma, decerebrate rigidity, moribund appearance

Table 28.2 World Federation of Neurological Surgeons (WFNS) grading scale [12]

WFNS Grade	GCS score	Motor deficit
I	15	Absent
II	14–13	Absent
III	14–13	Present
IV	12–7	Present or absent
V	6–3	Present or absent

scale is designed more simply and clinically estimates the prognosis by combining the GCS with major focal neurological deficits (aphasia, hemiparesis, or hemiplegia) (Table 28.2 [12]).

28.3.1.2 Grading of Blood by Computed Tomography (CT)

Although it has been pointed out that the Fisher grading system (Table 28.3 [13]) is not adapted to current technically sophisticated CT imaging, there are good correlations between this scale and symptomatic cerebral vasospasm [14].

28.3.2 Clinical Symptoms of SAH

28.3.2.1 Physical Symptoms

Cerebral aneurysms are most frequently manifested as SAH together with a sudden severe (thunderclap) headache, neck pain, nausea, vomiting, focal neurological signs, depressed consciousness, and prolonged coma.

28.3.2.2 Physiological Symptoms

Additionally, SAH leads to a range of systemic physiological responses and occasionally results in a hazardous situation or even death.

Table 28.3 Fisher grading system [13]

Group	Blood on CT
1	No subarachnoid blood detected
2	Diffuse deposition or thin layer with all vertical layers of blood (interhemispheric fissure, insular cistern, ambient cistern) < 1 mm thick
3	Localized clots and/or vertical layers of blood ≥ 1 mm thick
4	Intracerebral or intraventricular clot with diffuse or no subarachnoid hemorrhage

An abnormal ECG after SAH can be recognized in 70–100 % of the patients and presents variable waves as follows: peaked P waves, short PR interval, prolonged QT interval, large U waves, peaked T waves, and rarely electrical transformations of subendocardial ischemia or infarction. An overstressing of the sympathetic nervous system from increased ICP brings about these changes and is also fraught with excessive increases in blood pressure (Cushing reflex). The changes are often accompanied by mild elevation of cardiac enzymes, but do not usually correlate with significant myocardial dysfunction.

Leukocytosis, in which the white blood cell count increases to more than 20,000 per mm^3 after SAH, presents a poor clinical grading scale value, and the mortality reaches 50 % [15].

Other physiological changes after SAH include electrolyte imbalances, particularly hyponatremia, and acid-base abnormalities.

Caution from Drake [16]

An incident occurred which shows that, once alerted, both physicians and the public can recognize a warning bleed. In London, Ontario, a first year resident in neurology was sued by a widow for missing a "warning leak" some 2 weeks before her husband's fatal hemorrhage. A large judgment was rendered against him by the court and the story was featured in detail in the newspapers. During the following week, eight patients were referred to an emergency department by their physician for lumbar puncture, and in five of them the fluid was bloody. Also of interest was that they thought they had had a hemorrhage.

28.3.3 Complications of SAH

28.3.3.1 Intercranial Complications Secondary to SAH

Rebleeding is a fatal complication of SAH, which may form intracerebral or intraventricular hematomas and increase mortality and morbidity. The incidence of rebleeding peaks within 24 h after SAH; during the first 12 h, 12 % of the patients suffer from rebleeding and most of them receive poor grading at the initial symptom

evaluation [17]. A giant cerebral aneurysm occurring in a patient under 40 years old shows a higher tendency of rebleeding than in an older patient [18].

Vasospasm is generally manifested clinically 3–5 days after SAH and may induce cerebral ischemia or infarction, which are foremost causes of morbidity and death. Early vasospasm observed within 48 h after rupture of a cerebral aneurysm is recognized in 10 % of the patients and suggests a worse prognosis, but there is no relationship between delayed cerebral vasoconstriction and prognosis [19].

Hydrocephalus occurs in 10 % of the patients after SAH. Once it occurs, any aneurysm treatment cannot affect the progression of hydrocephalus. Acute hydrocephalus is characterized by the onset of lethargy and coma within 24 h of SAH, whereas chronic hydrocephalus develops weeks after SAH in surviving patients. Symptoms of chronic hydrocephalus include impaired consciousness, dementia, gait disturbance, and incontinence.

Seizures are deleterious because of increasing CBF and cerebral metabolic rate for oxygen ($CMRO_2$). Patients who present with lobar intracerebral hemorrhage on CT scan have a high rate of seizures.

28.3.3.2 Extracranial Complications

Extracranial complications include myocardial ischemia and infarction, arrhythmia, hemodynamic derangements, neurological pulmonary edema, and gastric hemorrhage (Cushing ulcer).

28.3.4 Investigations of SAH

28.3.4.1 Digital Subtraction Angiography (DSA)

Digital subtraction angiography (DSA) is still useful for investigation of cerebral aneurysms. When the presence of an aneurysm is suspected, repeat DSA after a suitable interval is required.

28.3.4.2 CT Angiography

The role of contrast-enhanced CT angiography is essential to investigate ruptured aneurysms, because of its ability to obtain important information about intracerebral or intraventricular hemorrhage, cerebral infarction, brain edema, and hydrocephalus that may be difficult to obtain by the use of DSA. In addition, CT investigation is noninvasive and may be repeated sequentially. The pulsation of cerebral aneurysms can be seen by recently developed four-dimensional CT [20].

28.3.4.3 Magnetic Resonance Imaging (MRI)

The detection of SAH by magnetic resonance imaging (MRI) is 94 % within 4 days after SAH and 100 % after 4 days using T2-weighted imaging [21].

28.3.4.4 Other Investigations

Transcranial Doppler (TCD) and cerebral arteriography can detect cerebral vasospasm before clinical symptoms occur [22].

28.3.5 How Should We Treat Aneurysms?

28.3.5.1 Treatment of Aneurysms

The treatment of a ruptured aneurysm should be considered first and then the prevention of rebleeding and removal of the hematoma. An unruptured aneurysm requires consideration regarding the cure and prevention of bleeding. A giant aneurysm must be prevented from rupturing, and the reduction in the diameter of the aneurysm needs to be planned.

28.3.5.2 Which Surgical Treatment Is Better?

Although there are no universal guidelines to choose whether neurosurgery (clipping) or neuroendovascular therapy (coiling) should be used, the general criteria are as follows:

- Neuroendovascular coiling is recommended to treat an aneurysm of the internal carotid-ophthalmic artery.
- Neurosurgical clipping is suggested to treat an aneurysm of the MCA.
- There are no indications for endovascular therapy for giant cerebral aneurysms, fusiform aneurysms, and small (<3 mm) thromboembolic aneurysms.

28.4 Anesthetic Management

28.4.1 Preoperative Management

28.4.1.1 Cerebral Vasospasm

Vasospasm of the cerebral artery may induce cerebral ischemia or infarction; therefore, early treatment of vasospasm is very important. “Triple-H therapy (HHH therapy)” which stands for hypertensive, hypervolemic, and hemodilution,

consists of intravenous administration of fluids or inotropic drugs, or both [22], and improves cerebral perfusion with increased blood pressure and intravascular volume accompanied by lower blood viscosity. Thus, the combination of hypervolemia and isoproterenol may be a reasonable therapy for untreated cerebral aneurysms [23]. A central venous pressure (CVP) of 10 mmHg or a pulmonary artery wedge pressure (PAWP) of 12–16 mmHg, or both, is considered as an index of optimal volume expansion. A hematocrit value of about 30 % is an advantage for the cerebral microvasculature.

28.4.1.2 Changes in the ECG Wave Tracing

Changes in the ECG wave tracing after SAH are well known and are correlated with increased catecholamine levels in the blood. Clinically, 33 % of SAH patients show some cardiac complications, mostly arrhythmia and lung edema [24].

Usually, the antiarrhythmic agents which do not prolong the QT interval, such as propranolol, lidocaine, and phenytoin, are prescribed. Contraindicated agents include procainamide, quinidine, and disopyramide.

28.4.1.3 Neurogenic Pulmonary Edema

Neurogenic pulmonary edema is caused by systemic and lung vasoconstriction-induced massive adrenergic discharge [25]. To determine whether the lung edema is neurogenic, it has to be distinguished from iatrogenic lung edema caused by excessive infusion of crystalloid or transfusion of blood, edema resulting from heart or renal failure, and aspiration pneumonia. Treatments include airway management, oxygenation, positive end expiratory pressure (PEEP), administration of adrenocortical hormone, and diuretic drugs.

28.4.1.4 Hypovolemia

Hypovolemia emerges necessarily after SAH because of excessive decreases in red blood cell mass or total blood volume [26]; thus, intravascular expansion therapy for symptomatic cerebral vasospasm becomes necessary (hypervolemia is part of "triple-H therapy"). Although cerebral vasospasm occurs from a combination of cerebral vasoconstriction and a decrease in total blood volume, cerebral ischemia can occasionally emerge with normal blood volume, even if there is vasoconstriction [27].

28.4.1.5 Hyponatremia

Hyponatremia occurs commonly after SAH. It is regarded as being induced by the cerebral salt-wasting syndrome (CSWS) rather than by the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) [28]. CSWS brings about excessive natriuresis by means of atrial natriuretic peptide (ANP) from the hypothalamus, and therefore we must treat to replace the sodium deficit with salt, using either normal or hypertonic (3 %) saline. Recently, brain natriuretic peptide (BNP) [29] or dendroaspis natriuretic peptide (DNP) [30] has been suggested as giving rise to hyponatremia.

28.4.1.6 Cerebral Ischemia

Cerebral ischemia worsens the morbidity and leads to poor overall mortality. Hyperglycemia associated with cerebral ischemia accelerates intracellular acidosis; hence, blood sugar level should be controlled to within 80–120 mg/dL to prevent brain injury. Similarly, hyperthermia worsens cerebral damage; the treatment by moderate hypothermia (34–35 °C) is effective to protect the brain. Otherwise, the calcium antagonist nimodipine (but not nicardipine) administered p.o. decreases the risk of morbidity, ischemic neurological disturbances, cerebral infarction, and rebleeding and also reduces death from vasospasm. Intravenous injection of a calcium antagonist cannot be recommended because blood pressure depression is an adverse event [31].

28.4.1.7 Acute Hydrocephalus

Acute hydrocephalus may require external ventricular drainage to normalize ICP. Hydrocephalus drained within 24 h preoperatively shows rebleeding in 4.4 % of the patients; the value is similar to later drainage [32].

28.4.1.8 Early Seizures

Early seizures occur in 3 % of the patients, frequently before hospitalization; prophylactic therapy is therefore controversial.

28.4.2 Premedications

Preoperative medications such as anticonvulsants or steroids, if prescribed, are continued. Drugs for gastric conditions, either an H_2 antagonist or metoclopramide,

are given before the induction of anesthesia. Giving small doses of opioids or benzodiazepine, or both, may be effective in good-grade patients. Conversely, almost all poor-grade patients do not receive hypnotic premedication, but receive blood pressure control. If an endotracheal tube is in place, medication for sedation and muscle relaxation may be given. Because of the hypovolemia after SAH, hydration with isotonic crystalloid solution is required before induction.

28.4.3 Monitoring

28.4.3.1 Noninvasive Conventional Monitoring

The V_5 lead of a 12-lead ECG represents heart rate, rhythm, and cardiac ischemia.

Intermittent noninvasive blood pressure can indicate the risk of rebleeding.

Urine output may be an estimate of the intravascular volume and renal function intraoperatively. When excessive urine output occurs, hypovolemia and electrolyte imbalances may exist, and diabetes insipidus (DI), SIADH, or CSWS needs to be considered.

End-tidal CO_2 concentration (P_{ETCO2}) conveniently suggests the status of the cerebrovascular autoregulation of the brain, relaxed or tight. In addition, a sudden decrease in the P_{ETCO2} may enable early detection of an air embolism.

Percutaneous oxygen saturation (S_PO_2) measurement via the pulse oximeter is helpful to prevent hypoxemia.

Deep body temperature measured at the tympanic or nasopharyngeal region reflects brain temperature and is useful to help protect the brain from hyperthermia.

28.4.3.2 Invasive Monitoring

Invasive arterial blood pressure measurement, performed before induction, is essential to strictly control systemic blood pressure and to prevent rupture of aneurysms and cerebral ischemia caused by violent fluctuations in blood pressure. Moreover, several blood samples via the arterial route may be obtained for analyzing blood gas, measuring blood sugar level, or other clinical factors.

CVP (central venous pressure) for the monitoring of preload is of great importance for several indications, such as preoperative hypovolemia, severe heart disease, and excessive bleeding intraoperatively.

Transesophageal echocardiography (TEE) is performed to evaluate left cardiac system function and air embolism.

28.4.3.3 Monitoring of the Central Nerve System (CNS)

Monitoring of motor evoked potentials (MEP) outperforms other CNS monitoring, such as somatosensory evoked potentials (SSEP) or microvascular Doppler ultrasonography (MDU), in detecting subcortical ischemia in the brain, which brings about motor dysfunction [33].

Brain tissue O_2 ($PtiO_2$) may be valuable in detecting ischemia expression in the brain [34].

TCD (transcranial Doppler) can show the condition of the CBF and appearance of air embolism.

ICP monitoring is helpful to preserve the CPP.

Jugular bulb oxygen saturation ($SjvO_2$) can show the O_2 supply and demand in the brain.

28.4.4 Goals of Anesthetic Management

Neuroanesthesiologists should make plans for the anesthetic management before surgery (Table 28.4 [22]). The basic goals are as follows.

28.4.4.1 Blood Pressure

Avoid sudden increases or rapid decreases in arterial blood pressure, which would induce aneurysm rupture due to a steep change in transmural pressure of the aneurysm.

Table 28.4 Anesthetic management of patients with intracranial aneurysms [22]

Preoperative	Neurologic evaluation is performed to look for evidence of increased intracranial pressure and vasospasm
	Electrocardiogram changes frequently are present
	HHH therapy is indicated if vasospasm is present
	Calcium channel blockers
Induction	Avoid increases in systemic blood pressure
	Maintain cerebral perfusion pressure to avoid ischemia
Maintenance	Opioid plus propofol and/or volatile anesthetic is the recommended regimen
	Mannitol (0.25–1.0 g/kg IV) also can be given
	Maintain normal to increased systemic blood pressure to avoid ischemia during temporary clipping
Postoperative	Maintain normal to increased systemic blood pressure
	Early awakening is recommended to facilitate neurologic assessment
	HHH therapy is given as needed

HHH hypervolemia, hypertension, hemodilution

28.4.4.2 CPP and CBF

Maintaining the CPP and CBF to prevent hypoxemia is important, even during temporary vessel occlusion. It is equally important to remember that CBF depends on systemic blood pressure because cerebrovascular autoregulation is lost after SAH.

28.4.4.3 ICP

Controlling ICP appropriately and retaining "brain relaxation (not tight or bulging brain)" are necessary to prevent cerebral edema or vascular engorgement.

28.4.5 Induction of Anesthesia

The hemodynamic response evoked by direct laryngoscopy, tracheal intubation, and particularly the placement of head pins should be controlled adequately without compromising the CPP. After optimal head position is obtained, an opioid (fentanyl, 2–5 mcg/kg; morphine, 20–50 mcg/kg; remifentanil, 0.5 mcg/kg/min) and a hypnotic agent (propofol, 1–2 mg/kg; thiopental, 3–5 mg/kg; midazolam, 0.1–0.2 mg/kg) are administrated intravenously, followed by a muscle relaxant (rocuronium, $\geq$0.6 mg/kg; vecuronium, 0.1 mg/kg) to facilitate insertion of an endotracheal tube. Lidocaine (1.5 mg/kg) is given before laryngoscopy and intubation, if needed. All procedures are carried out with a 100 % oxygen mask and in some cases adding a low-dose inhalation anesthetic (0.5 MAC).

28.4.6 Maintenance of Anesthesia

28.4.6.1 Management of Anesthetic Agents

A volatile anesthetic (isoflurane, sevoflurane, or desflurane) can be chosen under conditions of normal ICP, but usually total intravenous anesthesia (TIVA) with propofol, narcotic, and muscle relaxant is suitable for cerebral aneurysm surgery, particularly in a patient with a high ICP. Although lurching or other movement while the patient's head is fixed is very dangerous, no addition of muscle relaxant after intubation is required when MEP measurement is performed.

28.4.6.2 Artificial Ventilation

Pulmonary ventilation is controlled to maintain the $PaCO_2$ between 30 and 35 mmHg, averting hypercapnia which may increase the ICP. Generally, the respiratory rate is raised to add minute volume, but simultaneously high airway pressure, which also may increase ICP, should be inhibited.

28.4.6.3 Blood Pressure

The systemic blood pressure, before coiling or clipping, is maintained within the patient's normal range or a mean arterial pressure (MAP) between 70 and 80 mmHg, thus optimizing the CPP ($\geq$50 mmHg). During temporary proximal occlusion, MAP is maintained in the high-normal range to facilitate perfusion through the collateral circulation and to prevent distal cerebral ischemia or infarction caused by regional hypotension. At this time, a vasopressor (phenylephrine, 0.1–0.2 mg IV; dopamine, 3–5 mcg/kg/min; norepinephrine, 0.1–0.2 mcg/kg/min) can be used to raise the MAP gently. The MAP after embolization with coiling need not change from the normal range. On the other hand, after placement of a cerebrovascular clip, as the aneurysm is isolated from the systemic circulation, a MAP as high as 110 mmHg is acceptable, which may be of value in preventing postoperative vasospasm.

28.4.6.4 Fluid Management

Some type of lactated or acetated Ringer's solution is preferred to normal saline. However, it may aggravate cerebral edema if an excessive amount of hypoosmotic crystalloid solution is infused; therefore, maintaining euvolemia is an important matter. Colloids (5 % albumin or 6 % hetastarch) are acceptable, although hetastarch (>500 mL) may interfere with hemostasis.

28.4.6.5 Cerebral Protection (See Sect. 28.4.1.6)

When collateral cerebral circulation cannot be maintained by systemic hypertension alone, some means of cerebral protection must be employed. Intermittent intravenous administration of thiopental (3–5 mg/kg) or propofol (1–2 mg/kg) is considered a promising treatment to prevent cerebral ischemia. Mild hypothermia (33 °C) or a calcium antagonist (e.g., nimodipine) is also effective. A solution of 20 % mannitol offers cerebral protection in addition to its diuretic action.

28.4.6.6 If Brain Swelling Occurs

If cerebral edema occurs, it is important to first discontinue the administration of volatile anesthetics. The infusion of crystalloid solution should cease and be replaced with hypertonic saline (1.8–7.5 %). Mannitol (0.5–1 g/kg), an osmotic diuretic, reduces cerebral volume beginning 5–10 min after infusion. Rapid administration or large doses of mannitol cause several side effects, including hypotension, hyponatremia, and hyperosmotic serum. Furosemide (0.5–1 mg/kg), a diuretic agent, is also effective in decreasing the ICP. Barbiturates, which suppress cerebral metabolism and reduce cerebral blood flow, can also be used to decrease the ICP. Thiopental, a 5 mg/kg bolus infusion followed by 2–5 mg/kg continuous infusion, may be a conventional prescription. Other treatments to decrease the ICP include maintaining a head-up position (15–30°) and augmenting hyperventilation ($PaCO_2$ of 25–30 mmHg).

28.4.7 Emergence from Anesthesia

On awakening from anesthesia, a gentle extubation is required to prevent coughing which may increase the possibility of hemorrhage. A bolus intravenous infusion of lidocaine (1.5 mg/kg) may inhibit the response.

28.4.8 Postoperative Anesthetic Management

28.4.8.1 Cerebral Vasospasm

Cerebrovascular vasoconstriction, following neurosurgical procedures for SAH treatment, is the most important issue in postoperative management. Treatment reduced serious vasospasm and improved long-term prognosis after the mechanism of cerebral vasospasm was unraveled and several valid therapies were established [35]. Triple-H therapy, composed of hypertension, hypervolemia, and hemodilution, may be easy to understand and be an unfailing method to treat postoperative cerebral vasospasm, in the same way as preoperatively. However, if triple-H therapy continues until unnatural hypervolemia occurs, the patient's cardiac function would closely approach the conditions of heart failure and, in particular, the observed tachycardiac arrhythmia.

28.4.8.2 Postoperative Polyuria

Postoperative polyuria occurs from several causes and induces electrolyte imbalances and therefore should be treated adequately in concordance with each

etiology. DI caused by a decrease in ADH secretion includes polyuria, hypernatremia, low specific gravity of urine, and dehydration. Treatments for DI are infusion of 0.45 % saline solution and intramuscular injection of vasopressin (5–10 U) [36]. Treatments for SIADH are water restriction and infusion of isotonic solution. Treatment for CSWS has been mentioned previously (see Sect. 28.4.1.5).

28.4.8.3 Postneurosurgical Pain Management

Adequate sedation and analgesia are important postoperatively, because of the stress resulting in hypertension or tachycardia which may become worse in the patient. After brain surgery, it is necessary to assess neurological findings, including consciousness level; therefore, oversedation must be avoided. Other than the use of appropriate opioids, such as the patient-controlled analgesia system, scalp nerve block or administration of acetaminophen is effective.

28.4.8.4 Other Postoperative Management

Meningitis may result from an indwelling intracranial drain. Some neurological changes, such as delayed return of consciousness or neurological deterioration, may be observed. An immediate CT scan or MRI should be performed to investigate this condition.

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