

ANESTHESIA MANAGEMENT IN SPINAL DURAL ARTERIOVENOUS MALFORMATION SURGERY

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Introduction

Among the AV malformations involving the spinal cord, the dural AVFs is the most common subtype. It occurs mostly in middle-aged men. Most of the cases are solitary, and multiple lesions are rare. Although the etiology has not been elucidated, the clinical symptoms are characteristic of the progressive spastic paraparesis, the micturition problem, and the numbness of the legs or buttocks. The MRI findings are characteristic of the perimedullary serpiginous flow voids, intramedullary T2 high-intensity signal, and swelling of the spinal cord. Patients will be in a severely disabled condition without proper treatment. There is a general agreement that endovascular embolization and the microsurgery are the treatments of choice. If the selective catheterization or embolization is difficult or failed, then surgical treatment is the choice. Although the microsurgery is safe, simple, and effective, the recovery after the treatment is not always complete.¹

ANATOMY

The anatomy of the spine can be divided into that pertaining to the vertebral bony column and the contents of the vertebral canal.

Vertebral Column

The vertebral column is composed of 33 vertebrae. In adult life this number is functionally reduced to 24 presacral vertebrae, the sacrum, and the coccyx. The presacral vertebrae consist of 7 cervical, 12 thoracic, and 5 lumbar bones. The 5 sacral and 4 coccygeal vertebrae fuse early in development. The vertebral column normally exhibits four curves in the anteroposterior (AP) plane. The two forward curves, or lordoses, are in the cervical and lumbar areas, and the two posterior curves, or kyphoses, are in the thoracic and sacral areas. The combination of these curves gives the normal bony spine the characteristic S shape when viewed from the side. Vertebral column showing 24 presacral vertebrae, sacrum, coccyx, and curvatures of the adult vertebral column. Note that the first coccygeal vertebra has fused with the sacrum. Most vertebral columns are 72 to 75 cm long; about one fourth of this length is contributed by the fibrocartilaginous intervertebral disks. The vertebral column supports the skull and transmits the weight of the body through the pelvis to the lower limbs (Stier et al).

The anterior longitudinal ligament and the posterior longitudinal ligament extend from the base of the skull and atlas to the sacrum. The anterior ligament is attached to the anterior surface of the vertebrae and intervertebral disks. The posterior ligament is attached to the posterior surface of the vertebrae and the intervertebral disks and lies within the vertebral canal. These two ligaments provide extension and flexion stability to the vertebral column. The supraspinal and interspinal ligaments join the spinous processes at each level, providing additional flexion stability. The ligamentum flavum unites the vertebral laminae at each level and forms part of the posterior border of the intervertebral foramen. The intervertebral disks are fibrocartilaginous joints composed of an interior nucleus pulposus surrounded and enclosed by a tough anulus fibrosus. Together, these two components provide a

strong attachment between adjacent vertebrae but allow some movement. In addition, the disks act as very efficient shock absorbers (Stier et al)

Spinal Cord Blood Supply

The spinal cord is supplied with blood from the aorta via the vertebral and segmental or radicular arteries; the three main arteries of the spinal cord are the single anterior spinal artery in the anterior or ventral median sulcus and two posterior spinal arteries located in the area of the dorsal nerve rootlets. These three arteries usually arise as branches of the vertebral arteries at the base of the brainstem and traverse the entire length of the cord. The blood flow is augmented by multiple segmental radicular and medullary arteries that enter at the intervertebral foramen. Blood supply of the spinal cord illustrating the single anterior spinal artery, paired posterior arteries, and feeding radicular branches from the aorta. IVC, inferior vena cava. Arteries of spinal cord. Regions most vulnerable to vascular deprivation when the contributing arteries are injured are T3-T5 and T12-L2 for anterior spinal artery and C8-T4 for dorsal circulation. Levels of entry of common radicular branches are shown (e.g., C5 and T5). Note that the spinal cord is enlarged in two regions for innervation of the limbs. Cervical enlargement extends from C4 to T1, and lumbosacral enlargement extends from L2 to S3. (Stier et al).

The anterior spinal artery supplies the anterior two thirds of the cord, and the posterior spinal arteries supply the posterior one third. Below the level of the cervical cord segments, additional blood supply is provided by segmental or radicular arteries that arise as branches of the aorta and enter the cord arterial system. The most consistent of these arteries is the artery of Adamkiewicz, which is the largest

segmental feeder in the thoracolumbar region of the cord. It usually enters as a single vessel between the ninth and the eleventh thoracic levels and arises on the left side of the aorta. This major arterial feeder vessel is thought to be the principal contributor to the arterial supply of the entire thoracic and lumbar cord distal to its entry. Loss of this artery after surgery or trauma to the aorta may produce paraplegia in the thoracic region. The arterial network of the three main blood vessels supplies blood to the interior of the cord through an extensive network of arterioles and capillaries. The density of the capillary bed reflects the metabolic demands of the different areas of the cord. Blood flow through these capillaries is very sensitive to compression of the cord, and ischemia may result (Stier et al).

Venous drainage of the spinal cord is through radial veins serving the parenchyma. The veins feed into the coronal venous plexus or longitudinal veins on the surface of the cord, which are, in turn, drained by medullary veins that penetrate the dura adjacent to the dural penetration of the nerve roots to join the epidural venous plexus. The epidural or internal vertebral venous system drains into the external vertebral venous system, which communicates with the caval veins. The veins in the epidural system are valveless and therefore subject to engorgement in certain normal and disease states, such as pregnancy and obesity, in which there is an increase in the intra-abdominal pressure or obstruction to venous flow through the inferior vena cava (Stier et al)

PHYSIOLOGY

Blood Flow

Spinal cord blood flow (SCBF) has been studied extensively in animal models. The values and data obtained from these studies are consistent with values obtained

for the brain; average SCBF is about 60 mL/100 g/min, including a threefold to fourfold gray matter–white matter differential in blood flow. Autoregulation in the cord mimics that in the brain, with flow well maintained with a mean arterial blood pressure (MAP) of 60 to 120 mm Hg. Likewise, the effects of arterial blood gas tensions are similar to those in the brain; hypoxemia and hypercapnia cause vasodilation, and hypocapnia causes vasoconstriction (Stier et al; Harrop et al 2011).

RADIOLOGIC CONSIDERATIONS

Imaging of the spine and spinal cord is an essential part of the diagnosis and treatment of spinal diseases. A variety of imaging modalities are available for the assessment of spinal pathology, the most common of which are plain radiography, computed tomography (CT), CT angiography, magnetic resonance imaging (MRI), MR angiography, bone scanning, single-photon emission computed tomography (SPECT), and positron emission tomography (PET). The choice of imaging modalities best suited for the patient depends on the history, physical findings, and differential diagnosis. A general review of the common imaging techniques used in spine disease can be found in the later section on traumatic spinal cord injury (SCI) (Stier et al; Varma et al; Prestigiacomo et al; Bogduk, 1999; Szolar et al; Akpinar et al 2009).

Case report

Here we have a successful report of anaesthetic management for male, 40 years old, and 100 Kgs bodyweight. He was diagnosed with Spinal Dural Arteriovenous Fistulae (SD AVFs). He underwent Laminectomy procedure to correct malformation. Patient was admitted in hospital at September 15, 2011 with Functional decline, multiple falls and severely weakening of lower legs, also bladder

and bowel incompetence history. Blood pressure (BP) was 140-150/65-77 mmHg, Heart Rate (HR) 62-72 bpm, Respiratory Rate (RR) 20 rpm, core temperature was 36⁰ C, and GCS was E₄V₅M₆, Upper Muscle Tone was 3/3 and Lower Muscle Tone was 0/0. Thyromental distance less than 3 cm, Mallampathy class II. MRI in September 16, 2011 is Edematous Spinal cord from C6-T5. MRI in September 19,2011 were Dilated vessel posterior to spinal cord at T5 or DAVF, Region and Spinal canal stenosis, with mild cervival spondylosis. The induction were with propofol and fentanyl titration, muscle relaxant facilitation was Atracurium 50 mg, then intubation use Simmac Video Laryngoscopy with Non Kinking Endotracheal Tube no.7.5 and maintenance with Oxygen:Air (2:2), and Desflurane. After 3 hours, the surgery procedure was ended. The patient was transferred to ICU. The next day patient was transferred to room care.

Discussion

The first descriptions of spine disorders were recorded nearly 4000 years ago in Egypt, when patients with such afflictions were left bedridden and death was considered unavoidable. One of the first extensive series on surgery of the spine was reported by Elsberg in 1925, in which the surgical treatment of spinal cord tumors was described. Since those early reports, spine surgery has made remarkable advancements, particularly since the 1980s. As surgical techniques have matured, complex operations are being performed on spine diseases once thought incurable. Moreover, increasingly older patients with multiple comorbidities are presenting for spine procedures. Consequently, the anesthetic approach to patients scheduled for spine surgery must consider the following issues: preoperative risk assessment, the specific spine pathology being treated, basic knowledge of spine

anatomy and imaging modalities, the surgical procedure planned, an awareness of the specific spine disorder being treated, potential airway difficulties, patient positioning, anesthetic choices, intraoperative medical management decision-making (blood replacement, blood salvage, hemodynamic goals, pulmonary function), and postoperative airway concerns and pain management.²

Preoperative Evaluation and Preparation

General

Preoperative considerations derive from the overall medical condition of the patient and the specific procedure that is planned. Patients presenting for surgery of the spine may manifest peripheral neuropathy, paraplegia, or spine instability, each with its attendant complications and anesthetic considerations. A comprehensive and coordinated anesthetic plan involving the surgeon and anesthesiologist that addresses the need for neurophysiologic or invasive monitoring (or both), the optimal approach to securing the airway, patient positioning, fluid requirements, special maneuvers such as an intraoperative “wake-up” test, and timing of extubation must be formulated in advance (Stier et al).

Airway Evaluation

The airway of the patient presenting for elective spinal surgery under general anesthesia requires meticulous evaluation, perhaps more so than for any other operation. Particular attention should be paid to the range of motion of the neck and to the presence of any neurologic symptoms or pain during such movement. The initial airway assessment is made by means of a general survey of the patient’s head and neck. Obvious problems such as morbid obesity, short neck, cervical collars,

and any breathing difficulties (e.g., stridor) should be noted. The presence of any craniofacial abnormalities may suggest a potentially difficult airway. The presence of a full beard may make mask ventilation more difficult. Mouth opening, a function of the temporomandibular joint, should be assessed. The extent of mouth opening is often related to the ease of laryngoscopy. Limited mouth opening can make visualization of any laryngeal structures challenging. The presence of loose teeth should be noted and documented on the record. Next, the oral pharynx is examined, with special notation made of the size of the tongue in relation to the mouth opening. During mouth opening, the ability to visualize the faucial pillars, soft palate, and base of the uvula with the tongue protruded maximally (Mallampati classification) has been shown to be an accurate predictor of difficulty with direct laryngoscopy and should be documented (Stier et al). This patient have prediction of difficult intubation preoperatively. The Mallampati was II, but the thyromental distance less than 3 cm and have a spondilosis cervicalis from MRI. So patient planned to be intubated with Simmac video laryngoscopy.

Moreover, the patient with moderate to severe limitation caused by mechanical or neurologic restrictions should be considered for an awake intubation under local anesthesia to minimize movement of the head and neck. If an awake intubation is considered optimal, a detailed discussion should take place with the patient, if possible, regarding the steps that will be required and in assuring the patient that care will be taken for comfort. Patients with only mild limitation of movement, in which a difficult intubation is not anticipated, may undergo anesthesia induction prior to laryngoscopy, depending on the comfort level of the anesthesiologist. However, it should always be brought to the patient's attention that the potential for further

neurologic injury exists, and the option of performing an awake intubation should be offered (Stier et al).

In the case of patient, 40 yrs old, with SD AVFs undergoing laminectomy correction procedure, BP 140-150/65-77 mmHg. After surgery, patients was awake extubation. Post surgery patient was moved to ICU. After 24 hours patient was discharged from ICU with adequate spontaneous breathing, Blood Pressure was 145 / 85 mmHg, HR 64 bpm, RR 20 rpm, and GCS was E₄M₆V₅. This surgery procedure, is fairly new categorized, utilizes micro-surgery with the addition of fluorescent dye which allows them to see the vein which is causing the problem. They could determine that it was a fistula (cluster of veins) where one tiny vein (smaller than a human hair) was shooting high pressure blood cells onto my spinal cord. The surgery took 3 hours and was not a simple one. It required them to cut patients back open, remove a portion of bone covering the spinal cord, and also go through the muscles in order to get to the tiny vein causing the problem. Using the fluorescent dye and a microscope, the surgeon was able to clamp off the vein, allowing the blood to circulate normally through the fistula.

This patient with cervical spondylosis. So we intubated with Simmac facilitations. Degenerative disease of the cervical spine affects more than 90% of individuals older than 65 years. The term *cervical spondylosis* refers to the nonspecific degenerative process of the spine that results in spinal stenosis as well as neural foraminal encroachment. Cervical spondylosis. Common sites of pathology that may result in compression of the spinal cord or nerve root. In those individuals who eventually experience symptoms of cervical degenerative disease, radiculopathy is the most common. *Cervical radiculopathy* is defined as a neurologic

condition characterized by dysfunction of a cervical spinal nerve, the nerve roots, or both. It is most commonly caused by lateral disk herniation, osteophyte overgrowth with narrowing of the lateral foramen (termed the *lateral recess syndrome*), or cervical spinal instability caused by subluxation of a cervical vertebra. Cervical radiculopathy caused by foraminal stenosis from a posterolateral herniation of the nucleus pulposus with compression of the exiting nerve root. MRI is the imaging modality of choice in the diagnosis of cervical radiculopathy; however, MRI is not indicated in the initial stages of management because the findings will not alter treatment. In general, medical management is attempted for 4 to 6 weeks, and if the patient remains symptomatic, an MRI study is appropriate. CT is of value primarily for defining the bony anatomy in the area of the spinal canal. Surgical treatment for cervical radiculopathy is indicated for severe clinical symptoms that medical therapy has failed to control combined with a compatible MRI study demonstrating nerve compression, for the persistence of pain despite medical management for at least 6 weeks, and for the presence of an evolving neurologic deficit (Stier et al). In this case we know the patient have cervical spondylosis by MRI, so we planned the intubation procedure with use Simmac Video Laryngoscope.

Once the airway is secured, delivery of anesthesia can begin. Induction of anesthesia for spinal surgery carries the same considerations as those for any other general anesthesia. Concerns related to patient comorbidities should be addressed as appropriate. As previously noted, a major issue is often whether to induce anesthesia before or after positioning. Muscle relaxants should be used with consideration of the potential for a hyperkalemic response in the instance of succinylcholine use in the patient sustaining a spinal cord injury. If muscle relaxants are used for induction, short-acting agents are recommended, allowing subsequent

evoked response monitoring. Following intubation and securing of the endotracheal tube, the patient can be prepared for positioning.

Concerns arise regarding protection of neurologic integrity after prone positioning when an area of the bony spine is unstable and susceptible to movement during positioning. If spinal instability is located in the cervical region, the patient may present for surgery with a cervical collar or in a halo immobilization or traction device. Other clinical conditions that may be present include spinal stenosis, severe root impingement by a disk fragment, and preexisting neurologic deficit. It is important that every effort be expended to keep the head and spine in a neutral position during the positioning of a patient with any of these conditions. Once the prone position has been attained, access to the head and endotracheal tube is restricted; thus, the use of a flexible armored endotracheal tube is advocated to avoid the risk of the tube kinking, which may result in difficulty with ventilation and oxygenation.

The anesthetic technique chosen for the majority of surgical procedures on the spine should be based primarily on the patient's underlying medical condition, the anticipated intraoperative conditions, and the preference of the anesthesiologist. If neurophysiologic monitoring is planned, an awareness of the effects of the various anesthetic agents on neurophysiologic testing is essential, and the anesthetic choices should be altered accordingly. Most importantly, a stable intraoperative anesthetic depth is essential so that any changes in evoked responses can be explained appropriately. An in-depth discussion of neurophysiologic monitoring techniques in the context of spine surgery is discussed in detail elsewhere in this book and is not covered in this discussion.

In addition to planning for neurophysiologic monitoring, the possibility of an intraoperative wake-up test should be determined before induction. If an intraoperative wake-up test is desired, either a total intravenous anesthesia technique or a balanced technique consisting of low doses of a volatile agent together with opioids is effective.^[204] With use of such regimens, there is an associated 25% incidence of patient intraoperative awareness and recall of the awakening event; however, the recall is not regarded as unpleasant in most instances

Intravenous anesthetics include barbiturates, propofol, etomidate and ketamine. Apart from anesthesia induction, propofol is being increasingly used for maintenance as a continuous intravenous infusion (often computer controlled). All the intravenous drugs mentioned are cerebral vasoconstrictors that act by depression of cerebral metabolic rate (CMR), except ketamine. Ketamine increases whole brain CBF without changing CMR in healthy volunteers. At subanesthetic doses, ketamine increases regional glucose metabolic rate and CBF. The other agents decrease CBF, CBV, and ICP while leaving autoregulation and vessel reactivity to PaCO₂ intact. CMR reduction reflects brain activity and is mediated through the electrical but not the basal metabolic activity of the neurons. Hence there is a ceiling effect for CMR reduction at electroencephalogram (EEG) burst suppression. In contrast to volatile anesthetics, propofol has been shown capable of suppressing the cerebrostimulatory effects of nitrous oxide. Etomidate directly inhibits adrenal cortisol secretion for 24 to 48 hours even after a single injection, and its use is often associated with myoclonic (not epileptic) movements.¹³

All volatile anesthetics are cerebral vasodilators, but isoflurane, sevoflurane, and desflurane also reduce CMR. A flat EEG is obtained with these three agents at around 2 minimum alveolar concentrations (2 MAC), a concentration at which maximum metabolic depression is achieved. The response of cerebral metabolism to rising concentrations of volatile anesthetics is not linear. The decrease in CMR is steep from 0 to 0.5 MAC and then more gradual up to 2 MAC. The effect of volatile anesthetics on CBF is the result of their vasodilatory properties and flow – metabolism coupling. At low concentrations (< 1 MAC), CBF is lower than in the awake person. But CBV is unchanged with isoflurane and decreased with propofol at comparable concentrations. Among the newer volatile anesthetics, sevoflurane is the least vasodilating and desflurane the most.¹³ ***In so that, for this case, we use Sevoflurane as the volatile maintenance anesthesia combine with propofol intravenous titration.*** The effects of xenon are more complex. This agent decreases CBF in gray matter, particularly in specific brain areas like the thalamus, the cerebellum, the cingulate gyrus, and the hippocampus, and increases CBF in white matter. It does not impair flow-metabolism coupling.¹³

For the normal brain and volatile concentrations below 1 MAC, PaCO₂ reactivity remains intact, permitting control of vasodilation by hypocapnia. However, the presence of a pathologic brain condition or use of a high – MAC volatile anesthetic may impair or even abolish PaCO₂ reactivity and autoregulation.¹³

Nitrous oxide is cerebrostimulatory, increasing CBF, CMR, and sometimes ICP. Its effect is not uniform throughout the brain but is limited to selected brain regions (basal ganglia, thalamus, insula), changing the regional distribution of CBF. If substituted for an equipotent concentration of a volatile anesthetic agent, nitrous oxide increases CBF. For the normal brain, the resulting cerebral vasodilation can be

controlled by hypocapnia or the addition of an intravenous anesthetic. However, volatile agents have no such attenuating effect; CMR and CBF are higher during 1 MAC anesthesia produced by a nitrous oxide – volatile anesthetic combination than that produced only by a volatile anesthetic. This effect is especially deleterious in the actual or potential presence of brain ischemia.¹³ In order that, for this case we must be without nitrous oxide application. But because of limited of qualification in anesthesia machine in operating theater was without room air facility, so that we give nitrous oxide in low concentration (Oxygen : Nitrous oxide equivalent was 75% : 25%). Particularly for repeat craniotomy, the potential of nitrous oxide, which is poorly soluble, to diffuse into and hence expand hollow spaces must be remembered as it could cause tension pneumocephalus in patients with intracranial air (repeat neurosurgery or head trauma).¹³

Opioids have been associated with short – term increases in ICP, particularly sufentanil or alfentanil. Reflex cerebral vasodilation after decreases in MAP and hence in CPP is the underlying mechanism for the transient increases in ICP, although a direct modest cerebral vasodilator effect has been demonstrated. This effect demonstrates the sensitivity of intracerebral drug effects to the intracranial and extracranial environment and the importance of maintaining normovolemia for ICP stability. Generally, opioids modestly reduce CMR and do not affect flow – metabolism coupling, autoregulation, or the carbon dioxide sensitivity of the cerebral vessels.¹³ In this case, we use fentanyl opioid for analgetic during surgery. Ramifentanil has been extensively studied. Its cerebral effects are comparable to those of other opioids, and its use in neuroanesthesia has been validated in clinical trials.¹³

Vasodilating antihypertensive agents such as nitroglycerine, nitroprusside, and nicardipine increase ICP and should be avoided.¹³ In this case, neurologist as the first medical care, was given nicardipine as an antihypertensive drugs for the patients is suspected hypertensive emergencies with target organs is cerebral haemorrhage. But in perioperative periode, we stopped nicardipine titrated slow down, to keep away from rebound effect. Cerebral vasodilation may result from a normal autoregulation response or direct arterial vasodilation. For axample, sodium nitroprusside does not change CBF. Conversely, verapamil decreases cerebrovascular resistance in humans by inducing direct cerebral vasodilation. Theophylline constricts cerebral vessels but increases CSF production and is a potent central nervous system (CNS) stimulant, raising the risk of convulsions. Most β – adrenergic blockers, especially esmolol, do not interfere with cerebral blood flow or metabolism.¹³

The anesthesiologist posseses a number of instruments to achieve ICP reduction and brain relaxation, and thus to improve the quality of surgical exposure and to reduce retractor pressure. The effectiveness of these instruments depends on intact intracerebral homeostatic mechanisms. Strategies to prevent intracranial hypertension and brain bulging are euolemia, sedation, analgesia, anxiolysis, no noxious stimulus aplied without sedation and local anesthesia, head – up position, no compression of the jugular veins, head straight, osmotic agents: mannitol, hypertonic saline, β – Blockers or clonidine or lidocaine, steroids, adequate hemodynamics: mean arterial blood pressure, central venous pressure, pulmonary capillary wedge pressure, hear rate, adequate ventilation: PaO₂ > 100 mmHg, PaCO₂ 35 mmHg, Intrathoracic pressure as low as possible, hyperventilation on demand before induction, use of intravenous anesthetic agents for induction and

maintenance in case of tensed brain.¹³ In this case, we kept the patient in euvolemia, by urinary monitoring, adequately sedation and analgesia with inhalation sevoflurane, intravenous propofol titration, and opioid fentanyl, with slight hyperventilation.

Treatment for managing intracranial hypertension and brain bulging are cerebrospinal fluid drainage if ventricular or lumbar catheter in situ, osmotic agents, hyperventilation, augmentation of anesthesia with intravenous anesthetic agents: propofol, thiopentone, etomidate, muscle relaxants, venous drainage: head up, no positive end – expiratory pressure, reduction of inspiratory time, mild controlled hypertension if autoregulation present.¹³ We do in this case with inhalation sevoflurane combined with intravenous propofol titration.

Hyperventilation result in hypocapnia and subsequent cerebral vasoconstriction. In the context of intact autoregulation, CBF is roughly linearly related to PaCO₂ between 20 and 70 mmHg. However, the carbon dioxide reactivity of cerebral vessels may be impaired or abolished in the presence of head injury or other intracerebral pathologic conditions, by high inspired concentrations of volatile anesthetics, or particularly if the vessels are already dilated, by nitrous oxide. The CBF-, CBV-, and ICP- reducing effects of hypocapnia are acute and apparent for less than 24 hours. A typical value to aim for is a PaCO₂ of 30 – 35 mmHg; arterial blood gas analysis rather than end – tidal CO₂ (ETCO₂) should be used as a controlling variable because of the possibility of large arterioalveolar CO₂ gradients in neurosurgical patients. The effectiveness of hyperventilation (PaCO₂ at 25 ± 2 mmHg) for controlling brain bulk in the patient under either isoflurane or propofol anesthesia has been demonstrated.¹³

The main complication associated with hyperventilation is reduction of CBF, which gives to cerebral ischemia. Thus, the anesthesiologist must balance the benefit of brain relaxation against the risk of cerebral hypoperfusion. Other side effects are linear reduction in coronary artery flow, reduced cardiac venous return, hypokalemia, and potentiation of the brain's response to opioids.¹³

Osmotic diuretics such as mannitol and hyperosmotic saline increase blood osmolality acutely, thus reducing brain water content (mainly in healthy brain tissue with an intact blood – brain barrier) and hence brain bulk and ICP. This response improves brain deformability and thereby facilitates surgical exposure. A further beneficial effect is improvement in blood rheology as a result of the reduction in edema of vascular endothelium and erythrocytes (increasing erythrocyte deformability) – the basis of mannitol's classic "antisludge" effect. A typical regimen is to give 0.5 to 1 g / kg mannitol (150 – 400 mL 20% Mannitol) intravenously, split between a more rapid pre-craniotomy dose and a slower infusion, until brain dissection is complete. The ICP effect is prompt, removes about 90 mL of brain water at peak effect, and lasts for 2 to 3 hours. Normally the aim is to keep osmolality at less than 320 mOsm/kg. Problems with the use of osmotic diuretics include hypernatremia, hypokalemia, and acute hypervolemia, which could be deleterious in patients with congestive heart failure. There is no additional benefit to using loop diuretics such as furosemide, which induced hypovolemia and does not reduce brain water content. On the contrary, serum saline should be infused to replace urinary losses in order to avoid hypovolemia and maintain blood pressure.¹³ In this case, we use mannitol 0,5 g/kbw because of cerebral oedema in that patient. Urinary output and fluid balance was the first priority.

Conclusion

Anesthesia management in Intracranial bleeding ec stroke haemorrhagic is very important for basic brain resuscitation perioperative with pharmacological and non pharmacological strategies, besides principle management of hypertensive emergencies.

Key Word: Intracerebral Haemorrhage, stroke, Brain Resuscitation, Hypertensive emergencies.

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