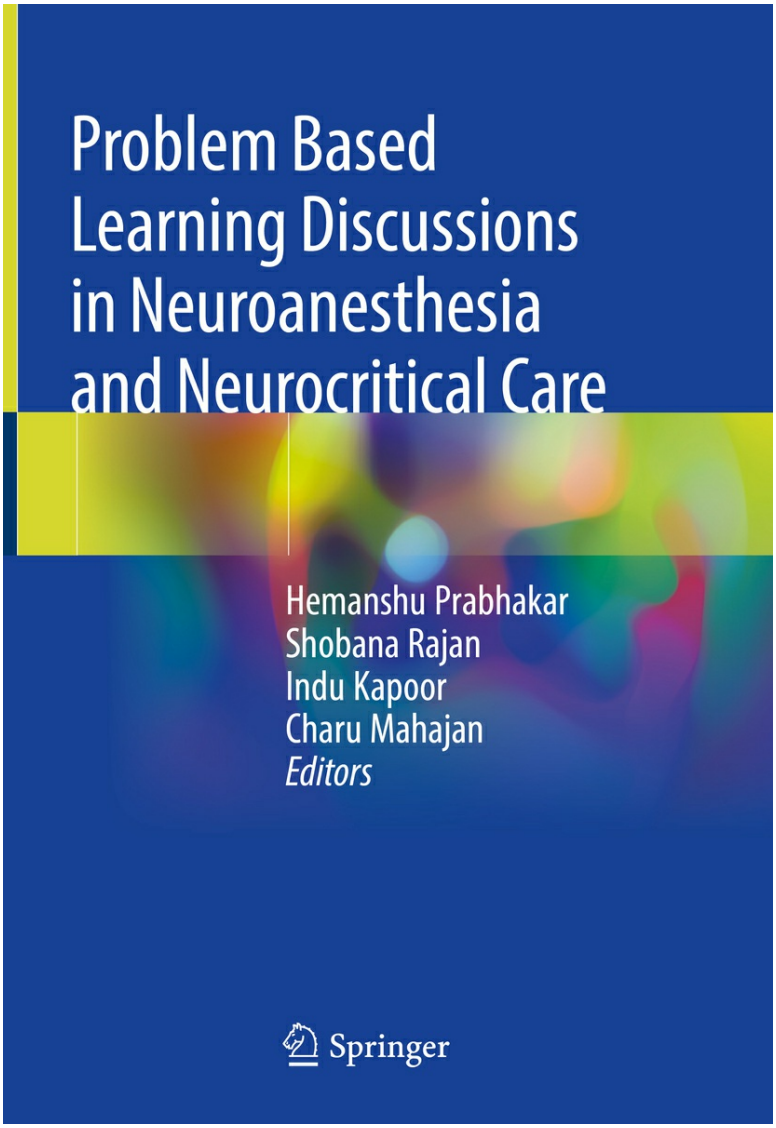
Problem Based
Learning Discussions
in Neuroanesthesia
and Neurocritical Care
Hemanshu Prabhakar
Shobana Rajan
Indu Kapoor
Charu Mahajan
Editors
Springer

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Problem Based Learning Discussions in Neuroanesthesia and Neurocritical Care

Editors

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To Anavi and Amyra, my precious angels and gifts from heaven

—Hemanshu Prabhakar

To my mother, Saraswathy Nagarajan, who has been a great source of enthusiasm, encouragement, and support for my academic endeavors, someone I could always turn to and the best part is that she leads by example.

—Shobana Rajan

To Namyah and Nyra, who mean all the world to me.

—Charu Mahajan

To Ansh, my bundle of joy, who made my life more beautiful.

—Indu Kapoor

Foreword

The Authors of this book, dedicated to clinical practice of*Neuroanesthesia and Neurocritical Care* , have adopted an innovative approach based on “case scenario” discussion. I am extremely pleased and honored to contribute to these forewords to present the result of a long-lasting effort in international networking aimed to fulfill educational need of our subspecialty. In time, I have met and had the opportunity to cooperate with several of the contributors of this book, and this makes it easy for me to witness their quality as scientists and clinicians.

The first notion of neuroanesthesia—the practice of anesthesia for brain or spinal surgery—dates back to the late 1940s. Since then, this subspecialty has experienced significant improvements in terms of quality of delivered care, complexity of treated patients and performed procedures, and the number of treated cases. Specific education path are now available, and this is a substantial contribution to reach high level of quality in patients care along with personal experience and case load. This book provides a substantial contribution within these three important aspects to obtain better short- and long-term outcomes in patients undergoing neurosurgical procedures and to better define the competences necessary for an accredited subspecialty practice.

According to a contemporary approach, clinical workup should take into account principles of “evidence-based medicine” that are derived by clinical experience and research results. In everyday neuroanesthesia, it is important to achieve a dedicated clinical training—that begins in general anesthesiology—and a full understanding of the unique features of the neurosurgical or neurologically injured patients. Education in neuroanesthesia and neurocritical care should also include simulation training and research activity so that residents and fellows could learn specific tasks and build confidence with essential features in order to provide excellent ability. In clinical practice, neuroanesthesiology requires also nonprofessional competencies such as effective communication and interdisciplinary interactions among all the members of the clinical team. Specific knowledge and competences required make neuroanesthesia and neurocritical care a unique medical field.

This book entitled*Problem Based Learning Discussions in Neuroanesthesia and Neurocritical Care* edited by Hemanshu Prabhakar and colleagues includes four major chapters: neurosurgical procedures; neurological patients; neuroradiology procedures; and “other” procedures and specific circumstances such as pregnant or HIV patients with brain tumor, or geriatric patients with intraparenchymal bleed. The chapter on neurosurgical procedures is dedicated to clinical management of intracranial A-V malformation, brachial plexus injury, brain abscess, cerebellopontine angle tumor, subarachnoid hemorrhage, cervical spine injury, craniosynostosis, craniopharyngioma, acromegaly, craniovertebral junction anomaly, hydrocephalus, lumbar PIVD, Moyamoya disease, meningomyelocele, pituitary tumor-Cushing’s disease, posterior fossa tumor, supratentorial tumor, traumatic brain injury—EDH, traumatic brain injury—SDH, motor strip gliomas—awake craniotomy and trigeminal neuralgia. The chapter on neurological patients is dedicated to clinical management of scoliosis, Guillain–Barre syndrome, myasthenia gravis, Parkinson’s disease, stroke, and polyneuropathy. The chapter on neuroradiology procedures is dedicated to clinical management of embolization-aneurysm, carotid stenting, and vein of Galen malformation.

I strongly recommend for those that approach the clinical practice of*Neuroanesthesia and Neurocritical Care* to go through this book in a systematic way and to use it to get ready for “best clinical management.” I also suggest this book to colleagues that are routinely involved in our discipline and to read the dedicated chapters when they will face one of the clinical scenario presented in this book; I am positive that it will be of great help in setting up a rationale workup for the daily practice.

Federico Bilotta

Preface

There have been some great textbooks in neuroanesthesiology. However, as editors of this book, we felt the need to bring out a book that would interest the millennial generation of anesthesiologists and trainees. They are not just interested in clinical facts but would like to know how things work out and why it works out. It is important to communicate the foundations of neuroanesthesiology to the next generation effectively. Hence, the idea of writing a book in a case-based, problem-oriented format was born.

Our highly qualified experienced team of editors then set to work to determine common yet challenging neuroanesthesia scenarios one would encounter during their practice. Due to advancements in the field of neurosurgery, there have been several new surgical procedures. Monitoring the brain has also taken a giant leap with the technology boom. “Time is brain” and we hope that reading this book will give all the insight and skills necessary to deal with these complex situations posed to the clinician practicing neuroanesthesia.

Each chapter begins with a case, and this is followed by pre-operative evaluation, intra-operative management, and post-operative complications in a question format with answers and references for these answers. Multiple choice questions at the end of each chapter serve as a test to assess the reader’s comprehension of the chapter.

As editors, we sought out neuroanesthesia experts throughout the world. Our authors have a high degree of expertise in their field, have been extremely willing and encouraging with this educational endeavor, and have shared their knowledge and experience with us.

Our team consisting of Hemanshu Prabhakar, Indu Kapoor, Charu Mahajan, and myself are happy to present you with this unique book. We think this would be of great benefit and we hope that you will enjoy reading every bit.

Happy reading,

Shobana Rajan
Cleveland, OH, Pittsburgh, PA, USA

Acknowledgements

We wish to acknowledge the support of the administration of the All India Institute of Medical Sciences (AIIMS), New Delhi, in allowing us to conduct this academic task.

Words are not enough to express our gratitude for the constant support and encouragement from the faculty and staff of the department of Neuroanesthesiology and Critical Care.

Special thanks are due to the production team of Springer—Dr. Naren Aggarwal, Dr. Eti Dinesh, Gaurav Singh, and Saanthi Shankhararaman.

Hemanshu Prabhakar

Charu Mahajan

Indu Kapoor

I would like to acknowledge the Society of Neuroscience in Anesthesiology and Critical Care (SNACC), where I have found inspiring mentorship and guidance. I would like to acknowledge the insight and support I have received from Dr. Rafi Avitsian who has always had time for me as a mentor from the early stages of my career in the United States.

It has been a great pleasure working with this wonderful team, Hemanshu, Indu, and Charu, and I thank them sincerely for their teamwork.

Shobana Rajan

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Part I
Neurosurgery

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1. Management of Patient with Intracranial A-V Malformation

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Keywords Cerebral arteriovenous malformation – Microsurgical resection – Perioperative complications – Multimodality monitoring

Stem Case Terminology
A 35-year-old male presented to your hospital with headache and vomiting for 5 months.

Question 1:
You are the neuroanesthesiologist on call and asked to assess this patient for future anesthetic and critical care management. How do you proceed?

Answer:
History taking and physical examination

Demographics: This patient is a 35-years-old gentle man hailing from Tamil Nadu. He is educated till twelfth grade and works as mason.

Chief complaints: Patient is a right-handed individual. He presented with episodic transient headache and vomiting since 5 months.

History of presenting illness: Patient was apparently normal 5 months back. That is when he was traveling in a bus and he had a sudden onset of headache. It was throbbing type and peaked over 10 min. Head ache was holocranial in location. It was so severe that the patient had to rush immediately to a regional hospital. After about 30 min, the patient had an episode of vomiting which relieved his headache.

Patient had four to five such episodes over the past 5 months with a frequency of one episode a month. Last two episodes were associated with giddiness.

There is no history of hypertension, seizures or weakness, visual loss, weakness in limbs, deviation of angle of mouth, loss of consciousness, and altered sensorium. There is no history of fever, alcoholism, vomiting, and diarrhea leading to dehydration. There is no history of fever and past history of tuberculosis. There are no signs of gait disturbances, difficulty in swallowing, or regurgitation of food particles and hoarseness of voice.

Treatment history: In the regional hospital, he was evaluated for migraine without any diagnostic success. Presently he is not on any medication.

Summarizing the history: This 35–year-old man is presenting with sudden onset of headache associated with features of raised intracranial pressure (ICP). His differential diagnosis would be causes of sudden onset head ache: (a) aneurysmal subarachnoid hemorrhage (b) cerebral arteriovenous malformation (cAVM) with bleed (c) intracranial tumor with bleed (d) cortical vein thrombosis (e) acute pyogenic meningitis.

General physical examination: Patient is middle aged, moderately built, and nourished. He does not have pallor, icterus, pedal edema, cyanosis, clubbing, and generalized lymphadenopathy.

Pulse is 82/min regular, blood pressure 126/80 mmHg in the right arm in supine position, and respiratory rate is 16/min regular.

1.1 Systemic Examination

1.1.1 Central Nervous System

Higher mental function: The patient is oriented with time, place, and person. Mini-mental state examination may be done for detailed assessment.

Cranial nerve examination: Clinical implications of cranial nerve examination to neuroanesthesiologist are given in Table 1.1.

Table 1.1 Clinical implications of cranial nerve examination to neuroanesthesiologist

Cranial nerve	Examination	Clinical implication to neuroanesthesiologist
1	smell	Not relevant

2	Visual acuity, afferent for pupillary reaction	Blurred vision in acute hydrocephalic attacks associated with headache and vomiting, pupillary asymmetry in cerebral herniation
3,4,6	Efferent for pupillary reaction	Lateral rectus palsy-one of the signs of raised ICP
	Extraocular movement	Tumor in proximity to cavernous sinus may hamper Extraocular movements
5	Sensory and motor testing	Intraoperative monitoring of cranial nerve using electromyography (EMG) of masseter
7	Motor testing	Intraoperative facial nerve monitoring using EMG of frontalis, orbicularis occuli, and orbicularis oris
8	Sensory testing	Brain stem auditory evoked potentials to monitor hearing as well as brain stem integrity
9,10,12	Motor testing	Intraoperative cranial nerve monitoring, caution during extubation if preoperative gag and cough are impaired
11	Motor testing	Lower cranial nerve monitoring

1.1.2 Motor System Examination

Bulk and tone of muscles—normal, power is 5/5 in all four limbs. Superficial and deep tendon reflexes are normal. Plantar reflex is down-going

Sensory system examination: Touch, pain, and temperature are normal in all dermatomes.

Proprioception is normal, vibration sense is normal, Romberg's sign is negative

No cerebellar signs. Gait and stance are normal

Clinical implication: It is essential to study from surgical notes about the preexisting sensory and motor deficits. Somatosensory and motor-evoked potentials may be used intraoperatively to monitor sensory and motor tracts, respectively.

Cardiovascular system: Normal heart sound is heard and no added sounds on auscultation.

Respiratory system: Normal vascular breath sounds are heard.

Gastrointestinal system: Normal on palpation and auscultation.

Question 2:

What are the diagnostic tools that help to arrive at diagnosis in this patient

Answer:

Imaging of the brain is an useful modality to arrive at diagnosis (Fig. 1.1)

1. Non-contrast computed tomography (CT): to rule out space occupying lesion. CT brain of patient showed hyperdense lesion in right parieto-occipital region with enlarged draining veins. There is no mass effect or midline shift.
2. CT angiogram: Right parieto-occipital arteriovenous malformation (AVM) with specks of calcifications. Nidus is 2 × 2.2 cm. Feeders are from right middle cerebral artery (MCA) and right posterior cerebral artery (PCA).
3. Magnetic resonance angiogram: T2-weighted image shows flow voids in right parieto-occipital region. Feeders are from MCA branches. Nidus drains into transverse sinus.
4. Digital subtraction angiogram (DSA): shows nidus measuring 1.6 × 2.2 × 2.6 cm^3 (Fig. 1.2). Feeders are from posterior temporal branch of right MCA and right PCA. Nidus drains into transverse sinus.

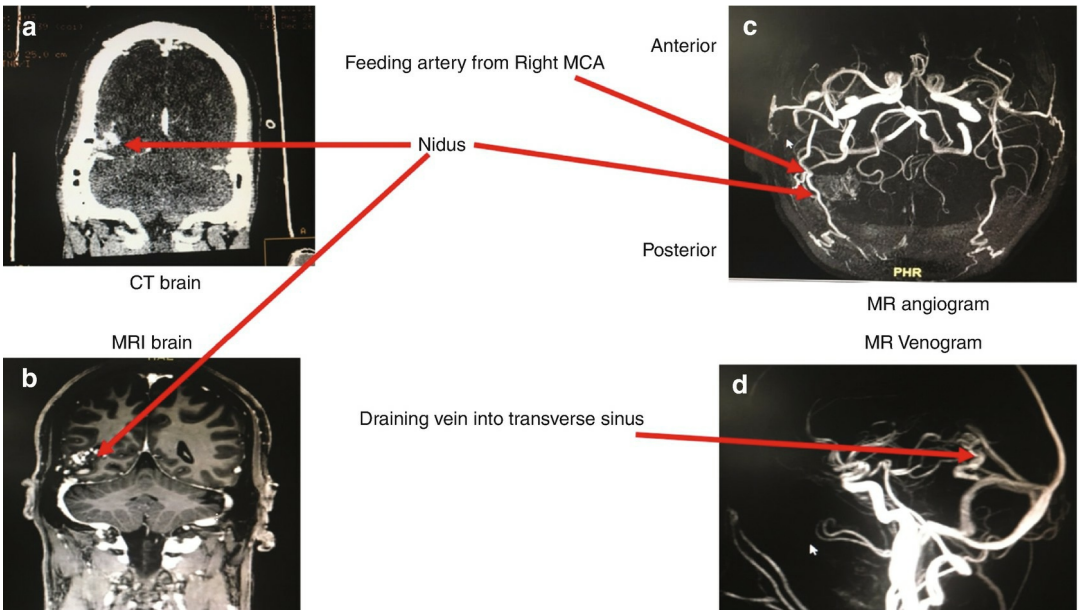

Fig. 1.1 Imaging modalities. (**a**) CT brain, (**b**) MRI brain, (**c**) MR angiogram, (**d**) MR venogram. *CT* computed tomography, *MRI* magnetic resonance imaging, *MR* magnetic resonance

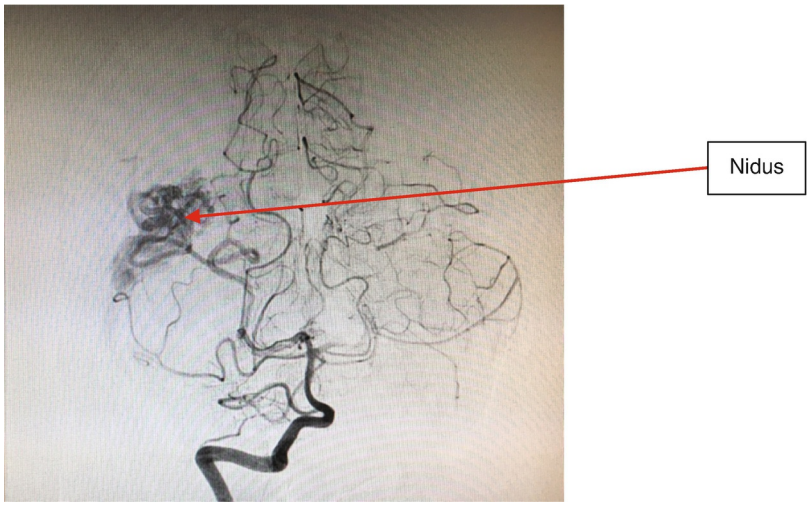

Fig. 1.2 Digital subtraction angiogram of cAVM

Question 3:
What is cAVM? Discuss its epidemiology.

Answer:
cAVMs are anomalies of intracranial vessels where an abnormal connection exists between the arterial and venous systems, and they lack normal capillary angio-structure [2].

Epidemiology [2]: Incidence: 1.3 per 100,000 person-years. Prevalence: 10–18 per 100,000 adults.

Question 4:
What is the pathophysiology of cAVM?

Answer:
In cAVM, arteries are directly connected to veins without intervening capillary bed [3]. A tangle of abnormal dilated channels that are neither arterial nor venous shunt blood from arterial end to venous end. This tangle is called the nidus. Thus cAVM has a single or multiple feeding arteries and a single or multiple draining veins. Figure 1.1c shows the feeding artery, and Fig. 1.1d shows draining veins of cAVM in the pateint. Blood flow in both the feeding artery and draining vein is higher than normal, and there exists higher than normal pressure on the venous side. Long-standing high flow rates may cause shear stress, intranidal flow-related aneurysms, arterial steal in the surrounding region and venous outflow obstruction. Mutation in RASA 1 gene is associated with capillary malformation-arteriovenous malformation syndromes [4]. However, cAVM may be an acquired condition as well.

Question 5:
What is the natural history and symptomatology of cAVM?

Answer:
Natural history is poorly understood. Spontaneous obliteration may occur with small AVMs (<2.5 cm) that present with intracerebral hemorrhage (ICH). Favorable anatomic features for spontaneous obliteration are single draining vein, small AVM, and presenting with ICH. cAVMs may present with ICH, seizures, headache, and long-term disability [5]. Majority of cAVMs are superficial and supratentorial.

ICH: Annual incidence of hemorrhage of unruptured cAVM is 2–4%. About 38–71% of patients with cAVM present with ICH [6]. Age of presentation of ICH with cAVM is 20–40 years. Risk factors for hemorrhage include (1) deep venous drainage (periventricular, galenic, or cerebellar) (2) flow-related nidal aneurysm (3) deep seated AVM, and (4) infratentorial AVM. Patients presenting with ICH may undergo emergency decompressive craniectomy in the presence of raised ICP features. History of ICH guides the need for therapeutic intervention of cAVM.

Seizures [7]: 18–40% of patients with cAVM present with seizures. Most commonly associated seizures are generalized seizures (30%).

Headache: Headaches [8] occur in about 5–14% of patients. Headache can be unilateral and bilateral and can have migrainous features with or without aura.

Focal neurological deficits (FND): 1–40% of patients with cAVM manifest with FNDs [9]. Pathophysiology of FNDs is multifactorial. They are (a) vascular steal phenomenon (b) venous hypertension. Vascular steal is centered around perinidal tissue. Venous dilatation may lead to mass effect and compression of brain tissue leading to FNDs. FNDs are independently associated with increasing age, female gender, deep brain location, and venous drainage pattern [10].

Question 6:
What are the diagnostic imaging modalities?

Answer:
Conventional DSA is the gold standard in the evaluation of cAVM angioarchitecture. Other initial modalities of imaging include—CT brain, CT angiography, MRI brain, MR angiography [11].

Question 7:
What are the treatment modalities available for cAVM?

Answer:
Important decision-making process in choosing the treatment modality for cAVM is to compare the risks of all treatment modalities against the natural history risks of cAVMs. Management of cAVM involves either single modality alone or multimodal treatment involving medical management [12], microsurgical resection [12], stereotactic radiotherapy [13], and endovascular embolization [14]. The factors that direct treatment modality are operator skill, cAVM size and location, surgical or endovascular accessibility, venous drainage, and presence of nidal flow aneurysm. ARUBA (a randomized trial of unruptured brain arteriovenous malformations) was the randomized controlled trial to assess surgical intervention versus medical management for unruptured cAVM. Fallacy in study design, implementation, short length of follow-up, and insufficient information regarding the treatment arm and the recruitment process invalidated the authors' conclusions [15].

Question 8:
Which is the mostly used grading system of cAVM in the context of micro-surgical resection?

Answer:
In 1986, Spetzler and Martin established a grading system (Table 1.2) for cAVMs based on the size of the nidus, location with respect to eloquent cortex, and venous drainage system [16].

Table 1.2 Spetzler Martin (SM) Grading Scale for cAVMs (this table is reproduced from Google images)

Characteristic	Number of points assigned
Size of AVM	
Small (<3 cm)	1 point
Medium (3–6 cm)	2 points
Large (>6 cm)	3 points
Location	
Non-eloquent site	0 points
Eloquent site[a]	1 point
Pattern of venous drainage	
Superficial only	0 points
Deep component	1 point

[a]Sensorimotor, language, visual cortex, hypothalamus, thalamus, internal capsule, brain stem, cerebellar peduncles, or cerebellar nuclei

Question 9:
What is the Spetzler Martin (SM) grading of cAVM of the above patient and what is the most suitable modality of treatment for him?

Answer:
SM grading of cAVM of the patient is

Size of cAVM is 1.6 × 2.2 × 2.6 cm^3 SM score = 1, Location—non eloquent—SM score = 0, Pattern of venous drainage-superficial component only—SM score 0

SM grade of the cAVM is 1 since it is a low-grade AVM in a surgically resectable, non-eloquent right parieto-occipital area with a single feeding artery, and a superficial draining vein.

Question 10:
Discuss anesthetic consideration for a patient with cAVM planned for a surgical resection.

Answer:
Neuroanesthesiologist encounters patients with cAVM for preoperative investigation (CT/MRI or DSA), preoperative endovascular embolization, surgical resection, or stereotactic radiosurgery.

Anesthetic drugs and cerebral physiology: Intravenous anesthetics reduce cerebral metabolic rate (CMR). There is also increase in vascular resistance resulting in decrease in cerebral blood flow (CBF). But most of the inhalational anesthetics except nitrous oxide cause vasodilation, increased CBF, and decrease in the cerebral metabolic rate of oxygen ($CMRO_2$). With normal anesthetic doses of inhalational agents, flow metabolism coupling is maintained [17]. Barbiturates, etomidate, and propofol may be used for brain protection during AVM embolization and/or surgical resection, when the brain is at the risk of focal ischemia [18].

Although not proved for each anesthetic, those that produce vasodilation may cause the steal phenomenon, and the anesthetics that constrict the vessels have the opposite effect, resulting in inverse steal, and may protect the brain or enhance the damage [19]. Effects of opioids on CBF, $CMRO_2$, and ICP are variable, slow administration in titrated doses, with care to maintain mean blood pressure is recommended [20]. Muscle relaxants, with the exception of succinylcholine have least effects on CBF and $CMRO_2$ as long as normocapnia is maintained, whereas succinylcholine causes an increase in ICP because of fasciculations [21].

Anesthesia for preoperative investigations: If the patient is a child or an uncooperative adult, monitored anesthesia care (MAC) or/and general anesthesia is required for preoperative imaging (CT/MRI/DSA). In our institution, MAC with intravenous midazolam 0.02–0.05 mg/kg is followed by intravenous infusion of propofol at the rate of 75 μg to 100 μg/kg/min. Injection midazolam followed by intravenous dexmedetomidine infusion at the rate of 0.02 μg to 1 μg/kg/h is also administered to patients for whom injection propofol is deemed unsuitable. Normoxia/normocarbia and ward blood pressures are maintained during MAC. General anesthesia with airway intervention is administered to cases in which MAC is failed or to cases requiring preoperative nidus embolization to occlude surgically inaccessible arterial feeder or to decrease the size of the nidus preoperatively. Imaging is done either electively or on an emergency basis. During preoperative assessment, neurological status of the patient including clinical assessment of ICP, CBF, cerebral oxygenation, airway, fasting status, and cardiovascular stability should be evaluated. Choice of anesthetic and technique of anesthesia is dictated by the clinical status of the patient at presentation.

Question 11:
What are the surgical considerations of cAVM?

Answer:
Standard surgical resection of cAVM involves microsurgical technique. The arterial feeders are tackled first, followed by excision of the nidus and finally resection of the draining veins. Veins are preserved till the very end of surgery [21]. Completeness of resection can be confirmed by intraoperative ultrasonography, intraoperative MRI, or DSA. If there is residual nidus, resection should be considered to avoid subsequent hemorrhage [21]. Stereotactic radiosurgery may also be considered for residual cAVM [22].

Question 12:
Discuss anesthetic management of surgical resection of cAVM

Answer:
Use of microsurgical technique combined with preoperative embolization and advanced neuroanesthesia techniques has enabled the total resection of cAVM, previously considered inoperable. General anesthesia is usually administered to patients undergoing surgical resection of cAVM. However, awake craniotomy may be required to resect cAVM in eloquent areas of brain.

Preoperative evaluation and premedication: Resection of cAVM is rarely performed as an emergency procedure [23]. Hence thorough preoperative evaluation is feasible. Preexisting comorbidities should be optimally treated and controlled prior to surgery. Existence of neurological dysfunction secondary to ICH due to ruptured cAVM, oligemic stroke, or mass effect due to cAVM itself should guide the choice of anesthetics and perioperative monitoring techniques to optimize postoperative neurological outcome [24]. An important consideration throughout the operation is the potential for rapid and massive blood loss [25]. Since most of the resection surgeries are elective, we counsel and explain the patient in detail of the perioperative management, and hence, we reserve anxiolytic medication like midazolam to apprehensive adults and to pediatric patients. Patients who have undergone preoperative embolization may have new onset neurological deficits [26], renal dysfunction [27] due to contrast used for angiography, and dehydration secondary to contrast agents. Accurate patient history and examination, optimization of fluid balance, renal parameters, and preparedness with blood and blood products prior to anesthetic induction are absolutely necessary.

Anesthetic technique: Choice of anesthetic agents is mainly guided by the coexistent morbidity. ICP is not a major concern as most of the cAVMs are operated on an elective basis [28]. Anesthetic agents whether intravenous or inhalational are titrated to maintain normoxia, normocapnia, and preoperative hemodynamics. Smooth and rapid emergence from anesthesia is also crucial for postoperative neurological assessment and avoidance of hemodynamic upheavals. The major differences between embolization and surgery are the presence of noxious surgical stimulation and possibility of sudden and profuse blood loss. Thiopentone, etomidate, and propofol are used as induction agents, and in children, we use sevoflurane for induction. Hemodynamic response to laryngoscopy, application of may field clamp to head, skin incision, periosteal elevation, and craniotomy should be anticipated and appropriate drugs like analgesics, local anesthetics (scalp block or pin site infiltration), thiopentone, propofol, and antihypertensives may be used. Careful patient positioning is essential to avoid nerve injury (padding pressure points), damage to eyes (eye padding and doughnut-shaped headrest), obstructed venous drainage (avoid excessive neck flexion and rotation), high airway pressure (avoid excessive neck flexion and rotation), and brain swelling (provide head end elevation). For awake craniotomy, we use MAC with injection dexmedetomidine. A bolus dose of 1 μg/kg/h is followed by an infusion of 0.2–1 μg/kg/h. Once the patient is asleep scalp block with 0.25% bupivacaine and 1% lignocaine with adrenaline calculated as per patient's body weight. After the craniotomy is completed, dexmedetomidine infusion is either stopped or lowered to keep the patient awake and cooperative for neurological assessment during cAVM resection. Post resection, dexmedetomidine infusion is resumed to keep the patient sedated during craniotomy wound closure.

Cerebral injury: Cerebral injury is caused by either surgical or anesthetic causes. Neurosurgeon-induced injury includes brain retraction, direct vascular injury (ischemia, thrombosis, and venous occlusion), and mechanical disruption of neuronal tissue or white matter tracts [24]. Anesthesia-induced injury may result in physiological trespass. Management goals should include ensuring brain relaxation and optimal systemic and cerebral hemodynamics, avoiding hypotonicity, maintenance of euglycemia, and a smooth emergence from anesthesia [24].

Cerebral protection: Pharmacological: Intraoperative brain swelling is the most common indication for pharmacological cerebral protection [29]. We use injection propofol or thiopentone titrated to burst suppression guided by scalp electroencephalogram (EEG). Avoid inhalational agents above mean alveolar concentration of 1. Use of appropriate vasopressors/inotropes and or intravenous fluids to maintain optimal cerebral perfusion pressure (CPP) is recommended

[30]. Intraoperatively we assess volume responsiveness guided by pulse pressure variation (PPV). After optimizing the PPV values and hemoglobin values, we use intravenous noradrenaline infusion at the rate of 0.05–0.1 μg/kg/min when indicated. If the systemic blood pressure is suboptimal at the end of surgery, we assess cardiac function by transthoracic echocardiography (TTE) at the earliest opportunity and add appropriate inotropes to improve cardiac function. We continue to follow improvements in cardiac function and fluid status periodically by assessing inferior vena cava (IVC) diameter and TTE.

Non-pharmacological interventions: Head end elevation, avoidance of extreme neck flexion to avoid kinking of neck veins which facilitates venous drainage. Hyperventilation for a shorter duration taking care to avoid fall in $PaCO_2$ below 30 mmHg. Hyperventilation may be guided by cerebral oxygenation monitors like near infrared spectroscope (NIRS) or jugular venous oximetry [31].

Question 13:
What are the perioperative complications of surgical resection of cAVM and how are they managed?

Answer:
Intraoperative bleeding: Neuroanesthesiologist should be prepared for rapid and torrential bleeding with blood and blood products.

Intraoperative brain swelling due to ICH may be tackled by techniques of pharmacological and non-pharmacological cerebral protection.

Intraoperative and postoperative brain edema and hemorrhage: Two propositions for the development of brain edema and hemorrhage during and after surgery are normal pressure perfusion breakthrough (NPPB) and occlusive hyperemia. The NPPB hypothesis proposes that postoperative hemorrhage and edema are caused by a preexistent deranged autoregulation in the ischemic brain around the AVM. Chronic oligemia in brain surrounding cAVM may produce maximal chronic vasodilation, which results in failed or insufficient vasoconstriction in response to the reinstitution of normal perfusion pressure after the AVM has been resected. According to this hypothesis, the key to avoid postoperative hemorrhage and edema is staged reduction of blood supply to the malformation. However, a number of observations suggest that the details of this theory are not applicable to most cases of malignant postoperative hemorrhage and edema. Some studies showed preserved autoregulation in the region surrounding a cAVM both before and immediately after its resection, and such cases went on to develop edema and hemorrhage. This observation argues against the theory of impaired autoregulation leading to NPPB. It has also led to the proposal of an alternative hypothesis termed “occlusive hyperemia.” This proposal postulates that malignant postoperative hemorrhage and edema are caused by either arterial stasis and obstruction or venous outflow obstruction, secondary to resection of cAVM. Consequences of NPPB like cerebral edema and hemorrhage are diagnosed by brain imaging, whereas occlusive hyperemia is diagnosed by DSA. NPPB requires lowering of MAP and antiedema measures. In our institute, MAP is maintained at 70–80 mmHg with labetalol infusion at the rate of 1–2 mg/min. We use clinical examination and NIRS to monitor cerebral oxygenation during labetalol infusion. When occlusive hyperemia is established, patient is hydrated to euvolemia, and MAP is maintained at 90–100 mmHg. Fluid infusion is guided by periodic TTE and/or IVC diameter assessments. Vasopressors and or inotropes are used to maintain desired MAP as and when needed.

Question 14:
How do you use bedside multimodality monitoring in the management of a case of cAVM?

Answer:
Perioperative bediside multimodality monitoring includes.

Preoperative monitoring: CBF velocities in feeding arteries are higher than in normal arteries remote from cAVM, and their pulsatality indices are lower than normal arteries [32]. Autoregulation may or may not be intact in the feeding arteries [33]. Jugular venous oximetry of the dominant jugular bulb demonstrates higher than normal (usually >70) jugular venous oxygen saturation as a result of shunting of arterial and venous blood without gas exchange occurring at nidus [34]. NIRS in the frontal region may display $rSO_2 > 70$ when the NIRS sensor picks up signals from shunted blood which is rich in oxyhemoglobin [35]. NIRS may display $rSO_2 < 40$ if it picks up signals from surrounding oligemic brain.

Intraoperative monitoring:
We monitor cerebral oxygenation using jugular oximetry or NIRS. EEG, somatosensory-evoked potentials and motor-evoked potentials are monitored for the detection of ischemia. We titrate hemodynamics when global suppression of cerebral oximetry and neurophysiological parameters occur. In the event of regional variation of cerebral oxygenation, EEG, and evoked potentials, neurosurgeon is warned about the changes, and appropriate measures of cerebral protection are executed. Intraoperative ultrasonography [36] (Fig. 1.3) or neuronavigation is gaining popularity because of their ability to localize the cAVM and also help to determine the extent of final resection. Intraoperative indocyanine green injection and examination under microscope also helps to determine the extent of resection of cAVM. Intraoperative DSA is the gold standard technique both for localization and to detect the presence of residual nidus.

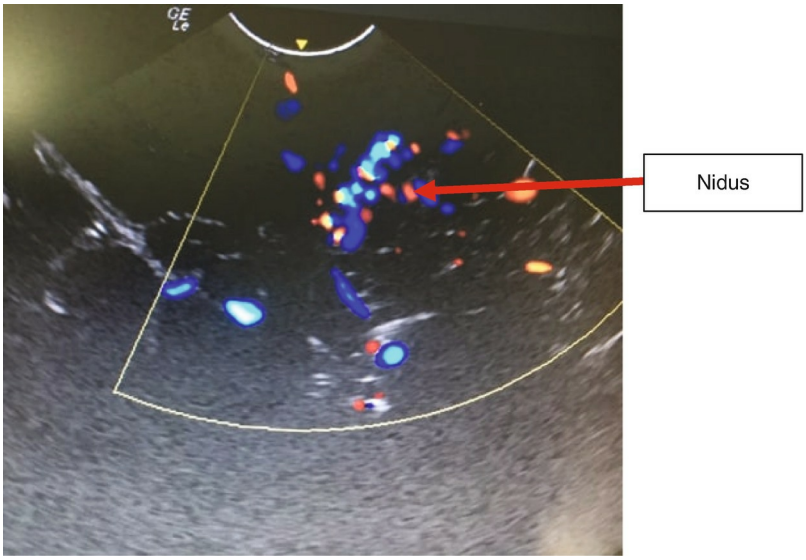

Fig. 1.3 Intraoperative ultrasonography showing nidus

Postoperative monitoring: Substantial fall in cerebral oximetry and jugular oximetry from baseline indicates significant shunt fraction reduction of cAVM [35]. Shunt fraction reduction is a reliable predictor of postoperative NPPB [37]. Increased CBF velocities demonstrated by TCD also indicates hyperemia.

Multiple Choice Questions:

1. Which of the following is not a component of cAVM?

   (a) Feeding artery
   (b) Draining vein
   (c) Nidus connecting artery to vein
   (d) Capillary-plexus connecting artery to vein

   Answer: d
   Explanation: cAVMs are anomalies of intracranial vessels where an abnormal connection exists between the arterial and venous systems, and they lack normal capillary angio-structure. cAVM has a single or multiple feeding arteries and a single or multiple draining veins. In cAVM, arteries are directly connected to veins without intervening capillary bed.

2. Which of the following is the definitive curative modality of treatment for cAVM?

   (a) Microsurgical resection
   (b) Stereotactic radiosurgery
   (c) Embolization
   (d) Medical management

   Answer: a
   Explanation: Microsurgical resection involves excision of all components of cAVM, namely feeding artery, draining vein, and the nidus. Whereas other modalities of treatment decrease the shunt fraction, but new feeder arteries may recruit with time when residual nidus exists.

3. NPPB is a postoperative complication of

   (a) Cerebral aneurysm
   (b) cAVM
   (c) Moyamoya disease
   (d) Pituitary apoplexy

   Answer: b
   Explanation: Chronic oligemia in brain surrounding cAVM may produce maximal chronic vasodilation, which results in failed or insufficient vasoconstriction in response to the reinstitution of normal perfusion pressure after the AVM has been resected. Such a phenomenon is known as NPPB. It is not seen with conditions a, c, and d.

4. Which of the following regarding multimodality monitoring in a patient with cAVM is incorrect?

   (a) Increased CBF velocities as measured by transcranial Doppler in feeding artery
   (b) Jugular venous oximetry ($SjvO_2$) of <40
   (c) Oligemia surrounding nidus on DSA
   (d) Raised ICP in a longstanding cAVM

   Answer: b
   Explanation: Jugular venous oximetry of the dominant jugular bulb demonstrates higher than normal (usually >70) jugular venous oxygen saturation as a result of shunting of arterial and venous blood without gas exchange occurring at nidus

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2. Management of Patient with Brachial Plexus Injury

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Stem Case Terminology
The incidence of perioperative brachial plexus injury (BPI) is not precisely known. Retrospective studies suggested that BPIs represent about 20-28% of the total perioperative peripheral nerve injuries (PNIs). There are various risk factors for BPI under anesthesia and surgery including preexisting disease, preexisting abnormal anatomical features, poor positioning, long surgical duration, hypotension, and hypothermia. BPI can be classified into preganglionic, postganglionic, or mixed pre and postganglionic. The most common perioperative BPI are ulnar nerve injury, upper/middle trunk injury and lower trunk injury. Pathologically, a BPI is classified into neuropraxia, axonotmesis, and neurotemsis. Such pathologic classification has a prognostic value and can determine treatment strategy. Treatment of BPI is essentially conservative. If surgical intervention is necessary, it is usually a lengthy procedure associated with specific anesthetic considerations including pressure care, temperature control, adequate fluid therapy to maintain normal volume status, prolonged ventilation strategy, control of blood sugar and acid-base balance, electromyography (EMG) testing, and thromboembolic prophylaxis.

Few hours after extubation, the patient complained of weakness in her right hand. Physical examination showed weak grip 1/5 of the right hand. The right hand attained a claw-like position with hyperextension at the metacarpophalangeal joints and flexion at the interphalangeal joints. Additionally, there was a narrow zone of lost sensation along the ulnar border of the forearm and hand. Nerve conduction studies confirmed BPI on the right side involving C8 and T1 distribution. The studies also indicated that the type of injury is probably at the level of the trunks involving the lower trunk (Fig. 2.1). Neurosurgery was consulted, and conservative treatment was recommended. The patient was discharged with weakness, paresthesia, and mild to moderate pain in the right hand. Extensive physiotherapy was performed. The patient's hand weakness improved slowly over 9 months and full motor power returned within 12 months with residual paresthesia.

2.1 Preoperative

Question 1:
What is the incidence of PNI?

Answer:
The incidence is not precisely known due to the absence of a reliable denominator and possible underreporting of PNI. Retrospective studies quoted 0.03–1.4% perioperative PNI [1]. It should be noted that some of these studies included patients who received neuraxial or peripheral plexus or single nerve blockade. Ulnar nerve, brachial plexus, and lumbosacral root injuries represent 28%, 20%, and 16%, respectively, of the total PNI events. Perioperative injuries to the sciatic, median, radial, and femoral nerves are less common. PNI in many cases have undetermined etiology or mechanism, e.g., 90% of ulnar nerve injuries [2].

Question 2:
What are the preoperative risk factors for PNI in this patient? Mention the risk factors for brachial plexus nerve injury during anesthesia and surgery.

Answer:
The preoperative risk factors for the brachial nerve injury in this case include preexisting diabetes and diabetic peripheral neuropathy, and the presence of cervical rib on the same side of injury. Other perioperative factors include park-bench position including abduction, posterior displacement of the shoulder, and increased surgical duration.

The perioperative risk factors are summarized in Table 2.1 [3].

Table 2.1 Perioperative risk factors for brachial plexus injury

Preoperative factors	Intraoperative factors
Preexisting diseases	Positioning
Diabetes	Shoulder/arm abduction >90°
Hypothyroidism	Steep Trendelenburg
Acromegaly	Posterior shoulder displacement
Neuropathy	External arm rotation
Polyarteritis nodosa	Rotation of head >20°
Herpes zoster	Anesthesia and surgery
Preexisting abnormal anatomy	Long duration
Anomalous nerve root origins	Sternotomy
Cervical rib	Hypotension

Shoulder deformity Hypothermia

Question 3:
Mention the neurologic effects of the presence of cervical rib.

Answer:
A cervical rib represents a persistent ossification and elongation of the transverse process of C7. The long transverse process can develop into full-size extra rib or fuse with the T1 rib.

Cervical ribs can cause a neurogenic type of thoracic outlet syndrome due to compression of the lower trunk of the brachial plexus [4]. Compression of the lower trunk of the brachial plexus causes weakness (weak grip) and sometimes atrophy of the thenar and hypothenar muscles. Additionally, sharp, burning, or aching pain can be present in the ulnar aspect of the arm and hand and reduced sensation to light touch in the fourth and fifth fingers. Patients may have pain in the side of the neck, the infraclavicular area, the axilla, and the upper back. Discoloration and coldness of the ipsilateral hand are not uncommon. If the upper trunk of the brachial plexus is involved, pain and paresthesias are experienced on the neck, shoulder, and face. Paresthesias radiate into the lateral arm and simulate fifth or sixth cervical nerve root compression.

2.2 Intraoperative

Question 4:
Discuss the common BPIs related to intraoperative patient positioning.

Answer:
The brachial plexus and its terminal branches are vulnerable to injury because of intraoperative malpositioning. The most common intraoperative injuries include ulnar nerve, upper/middle trunk, and lower trunk injuries [5].

Ulnar nerve injury: The incidence of intraoperative positioning-related ulnar nerve injury is between 1:215 and 1:385. If the ulnar nerve is subjected to injury, pain, paraesthesia, and/or weakness in its distribution usually occur in the immediate or early postoperative period. Ulnar nerve function recovers within 6 weeks in 50% of cases; however, the remaining 50% usually recovers within 24 months. Patients typically complain of numbness and/or pain in the ulnar-side of the hand (small finger and ulnar aspect of the ring finger). Additionally, motor functional impairment can be present in the form of clumsiness and loss of dexterity due to weakness of the intrinsic muscles of the hand.

Upper/middle trunk injury (C5/6): Such injuries have an overall incidence of approximately 1:2000. They present with motor deficit in the C5/C6 myotomes, often without sensory deficits. There is weakness of shoulder abduction and elbow flexion. The upper trunk is most at risk of stretch during prolonged and excessive shoulder depression and can be exacerbated by contralateral neck flexion. Full recovery is expected in about 80% of patients.

Lower trunk injury (C8/T1): It can complicate up to 1:20 cases of median sternotomy in cardiac surgery due to traction of the first rib against the lower trunk. Also, the lower trunk can be vulnerable during prolonged and excessive shoulder abduction (>90°), contralateral head rotation >20°, and when the arms are positioned below the height of the torso. Injury leads to numbness and may be pain in the distribution of the ulnar nerve. Motor impairment follows median and ulnar nerve distribution in the form of weak finger and thumb flexion as well as weakness in the small muscles of the hand.

Question 5:
Propose recommendations related to perioperative positioning to prevent BPIs?

Answer:
Several recommendations have been proposed for protecting the brachial plexus from injury during intraoperative positioning including [6, 7]:

- In the supine position, avoid extension and external rotation of the arm by limiting abduction to <90° and placing the arm supinated in the neutral position.
- In the prone position, avoid extreme arm abduction by tucking in and padding the arms by the patient's side, rather than abducting the arm above the head by more than 90°. Alternatively, the arms can be flexed at the elbow and the forearms placed on arm boards at a level lower than the torso and the upper arm placed almost vertical, and adducted without posterior displacement of the shoulder.
- In steep Trendelenburg position, tuck the arms in at the patient's side with draw sheets. Use "Butterfly Steep Trendelenburg Bean Bag Positioner" to support the patient and prevent the body from sliding cephalad. Using wrist suspensions or shoulder braces to prevent cephalad sliding increases the risk of BPI.
- In the lateral decubitus position, always use an axillary-chest roll and avoid suspension of the arm from an L-shaped bar or a sling.
- In any position, always keep the head in neutral position. Excessive rotation and lateral flexion of the neck can dangerously stretch the contralateral brachial plexus.

Question 6:
What are the anesthetic considerations and management if this patient had to undergo major brachial plexus exploration and repair?

Answer:
Major brachial plexus exploration and repair is a lengthy procedure. Hence, general anesthesia is usually preferred. Table 2.2 shows the anesthetic consideration and actions that can be done to provide safe anesthesia for those patients with prolonged surgery that may involve microvascular techniques [8, 9].

Table 2.2 Anesthetic considerations for prolonged brachial plexus repair

Anesthetic consideration	Action
Pressure care	Use of thick foam (3–4″) on top of the OR table mattress
	All dependent areas such as the heels, elbows, and occiput should be additionally protected by fluid filled bags or pillows
	All sites, where nerves lie superficially over bone, should be inspected and protected
Temperature control	Falls in core temperature must be vigorously treated
	Avoiding a skin/core temperature gradient during grafting
	Use forced air warming systems
	Use fluid warmers for IV fluids
	Use warmed irrigation solution
Fluid therapy and avoid hypotension	Maintain euvolemia
	Urinary catheterization is necessary for bladder decompression and urine output monitoring
	Arterial line may be necessary for blood pressure control and blood sampling
Prolonged ventilation	Maintain normocapnia
	Use lung protective ventilation strategies
Metabolic changes	Control blood sugar
	Maintain normal acid–base balance
Neuromuscular junction monitoring	Maintain 2/4 twitches for possible EMG monitoring
DVT prophylaxis	Use of intermittent pneumatic compression devices
	Use of unfractionated heparin or low-molecular weight heparin per institutional protocols
Pain control	Intraoperative long-acting opioids
	Postoperative PCA in case of large incisions
Immune status	N_2O may be avoided to prevent granulocyte and B- and T-immune cells depression
Personnel staffing and vigilance	To prevent failure of care, work in a “shift” pattern
	Plan to provide adequate number of anesthetists

2.3 Postoperative

Question 7:
Discuss the clinical anatomy of the brachial plexus.

Answer:
The brachial plexus consists of a complex system of nerves [10]. It passes through the posterior triangle of the neck into the axilla. The brachial plexus is bounded anteriorly by the anterior scalene muscle, posteriorly by the mid scalene muscle, and inferiorly by the outer border of the first rib. It is a complex intercommunicating network of nerves formed in the neck by the anterior rami of the spinal nerve roots C5, C6, C7, C8, and T1. Figure 2.1 summarizes the structure of the brachial plexus including roots, trunks, divisions, cords, and terminal branches. Various branches of the subclavian artery supply the brachial plexus along its length, i.e., vertebral artery; spinal arteries supply the roots; ascending and deep cervical arteries and superior intercostal artery supply the trunks and divisions; and the axillary artery supplies the cords.

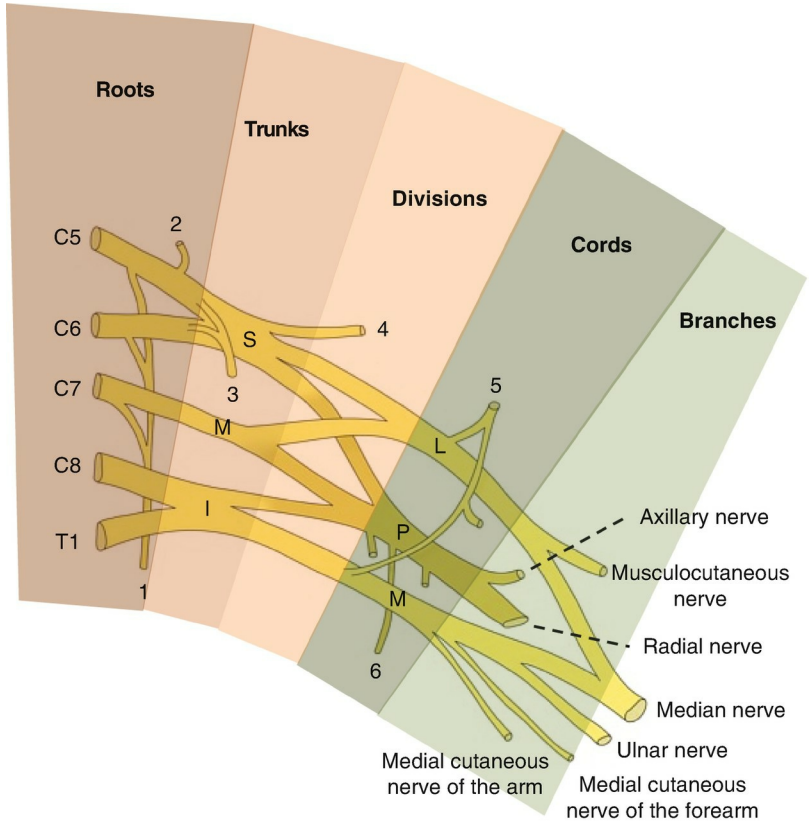

Fig. 2.1 Anatomy of the brachial plexus. The roots combine to form superior (S), middle (M), and inferior (I) trunks. Each trunk divides into anterior and posterior divisions. Posterior divisions unite to form the posterior cord (P). Anterior divisions of the superior and middle trunks unite to form the lateral cord (L). Anterior division of the inferior trunk continues to be the medial cord (M). The important nerves that originate proximal to the terminal branches include long thoracic nerve (*1*), dorsal scapular nerve (*2*), nerve to subclavius (*3*), subscapular nerve (*4*), pectoral nerve (*5*), and thoracodorsal nerve (*6*)

It supplies sensory innervation to the upper limb and most of the axilla except a small area in the medial upper arm and axilla (supplied by the intercostobrachial nerve).

It delivers motor innervation to the muscles of the upper limb and shoulder girdle except the trapezius muscle (supplied by the spinal accessory nerve).

It also supplies autonomic innervation to the upper limb by receiving sympathetic fibers from the stellate ganglion. Such autonomic supply has vasomotor, pilomotor, and secretomotor functions.

Question 8:
Classify BPIs.

Answer:
BPI is classified into three lesions: preganglionic, postganglionic, and mixed pre- and postganglionic [11]. A preganglionic lesion signifies avulsion of nerve roots, whereas a postganglionic lesion involves the nerve structure distal to the sensory ganglion. Postganglionic lesions can be in the form of nerve rupture or nerve injury in continuity.

Preganglionic injuries: Because it results from avulsion proximal to dorsal root ganglion, it does not regenerate. There is little potential for motor function recovery. Signs suggesting preganglionic injury include Horner's syndrome (ptosis, meiosis, anhidrosis of the cheek, and enophthalmos) due to disruption of sympathetic chain, winged scapula medially due to loss of motor fibers to serratus anterior through the long thoracic nerve, and rhomboids through the dorsal scapular nerve, flail arm, absence of a Tinel sign or tenderness to percussion in the neck, normal histamine test, i.e., intact triple response (redness, wheel, and flare), and elevated hemidiaphragm (phrenic nerve palsy), and evaluation by EMG may show loss of innervation to cervical paraspinals.

Postganglionic injuries: Because it is distal to the dorsal root ganglion, it involves the peripheral nervous system. Hence regeneration and better prognosis are possible. It presents with both motor and sensory deficits according to the postganglionic fibers disrupted (Table 2.4), abnormal histamine test (only redness and wheal, but no flare), and evaluation with EMG shows maintained innervation to cervical paraspinals.

Question 9:
Discuss BPI syndromes.

Answer:
BPI syndromes can be described according to the anatomical part that sustained injury (Table 2.3). Upper BPI (Erb–Duchenne palsy) is caused by difficult child birth, Burner syndrome, motor biking accidents, reduction of shoulder dislocations, direct trauma, e.g., injury by fractured clavicle, gunshot wounds, or stabbing. Lower BPI (Klumpke's palsy) is caused by difficult child birth, falling person grabbing on a tree, motor biking accidents, and direct trauma, e.g., injury by fractured clavicle, gunshot wounds, or stabbing.

Table 2.3 Brachial plexus injury syndromes

Syndrome	Mechanisms	Nerves injured	Features
Upper plexus injury[a] (C5/6 ± C7)	Excessive lateral neck flexion	Musculocutaneous	Loss of shoulder abduction, external rotation
	Excessive shoulder depression	Axillary	Loss of elbow flexion
		Suprascapular	Loss of wrist supination
		Nerve to subclavius	"Waiter's tip" position
		Radial	
Lower plexus injury[b] (C8/T1)	Excessive traction of abducted shoulder	Median	Loss of MCPJ flexion
		Ulnar	Loss of IPJ extension
			Loss of fingure abduction, adduction, and opposition
			Loss of wrist flexion
			"Claw hand" deformity
			Sensory loss of medial aspect forearm and hand
Total palsy (C5–T1)	Sever and complex traction injuries	Entire brachial plexus	Flaccid arm
			Paraesthesia of upper limb
Posterior cord injury	Direct injury	Subscapular nerves	Loss of arm extension
		Thoracodorsal	Loss of elbow extension
		Axillary	Loss of wrist extension
		Radial	

MCPI indicates metacarpal phalangeal joint, and IPJ indicates interphalangeal joint
[a]Upper plexus injury (Erb–Duchenne palsy syndrome) includes superior trunk (C5/6) ± middle trunk (C7)
[b]Lower plexus injury (Klumpke's palsy) includes the inferior trunk (C8/T1)

Also, injuries of the plexus can be described by the loss of motor function and sensations following dermatomal distributions (Table 2.4) [12].

Table 2.4 Motor and sensory loss in brachial plexus injury

Injury	Gross motor loss	Sensory loss
C5/C6	Shoulder abduction	Thumb
	Shoulder lateral rotation	Index finger
	Elbow flexion	
	(± wrist extension)	
C5/6/7	As above	As above
	Elbow extension	Middle finger
	Wrist extension	
	Finger and thumb extension	
C8/T1	Loss of wrist flexion	Medial forearm
	Finger and thumb flexion	Little finger
		Middle finger
C5–T1	Flail upper limb	Multiple dermatomes

Question 10:
What is Seddon's classification of nerve injury?

Answer:
Seddon in 1942 classified nerve injuries into three classes according to the extent of damage to axons and surrounding connective tissue layers. The classification has a prognostic significance and can help in planning treatment strategies [13]. In 1951, Sunderland expanded Seddon's classification to five degrees of PNI.

Neuropraxia (Sunderland type 1): There is no anatomical disruption. There is a localized transient electrophysiologic conduction block along the injured nerve without distal Wallerian degeneration. Clinical presentation is variable, but often it is associated with motor paralysis with residual sensory or autonomic function. Nerve conduction studies show a conduction block at the level of the injury. Good prognosis is expected with full recovery in 2–3 weeks. Conservative treatment is indicated.

Axonotmesis (Sunderland types 2, 3, and 4): There is anatomical disruption in the form of axonal damage within the nerve with axonal degeneration and myelin sheath, but endoneurial tubes and surrounding connective tissue elements remain intact. Distal Wallerian degeneration may occur in severe axonotmesis. Clinical presentation is in the form of complete muscle paralysis with progressive atrophy and complete sensory deficits. Nerve conduction studies show absent distal nerve conduction, absent motor unit action potential, and muscle fibrillation. Prognosis is fair with possible full recovery without surgery. Axons will grow by 1 mm/day; hence, full recovery may take weeks to months with possible residual deficits.

Neurotmesis (Sunderland type 5): There is complete anatomical disruption of the endoneurium, perineurium, and epineurium with complete nerve division. Wallerian degeneration occurs distal to the injury, usually caused by direct trauma to the nerve, i.e., open injuries and surgical transection. Prognosis is poor, and surgery is indicated. Full recovery is unlikely even with surgery.

Question 11: