Chapter 7

Neurological Monitoring and Management

Recovery after cardiac operation depends on total recovery of neurological function. Intact neurological function facilitates maintenance of adequate cardiorespiratory function and removal from the ventilator and other support systems.

It is difficult to evaluate neurological function until the effect of anesthesia is completely abolished. Complete neurological evaluation is possible only after the infant is fully awake, since most are maintained on IV analgesics and sedation post-anesthesia recovery.

Neurological management best addressed during preoperative, intra and postoperative periods:

1) Perform standard and orderly neurological evaluation postoperatively:

a) Glasgow coma scale.

b) Pupils: Reaction and size of pupils.

c) Motor function: Strength, movement of extremities, abnormal motor function, and activity (seizures).

2) Address predisposing factors of postoperative neurological dysfunction:

A) Hypoxemia, B) Hypothermia, C) Electrolyte imbalance, D) Metabolic acidosis, and E) Hypoglycemia.

3) Try to address intra-operatively several factors that result in neurological injury i.e., persistent right to left intra-cardiac shunts.

Acute postoperative neurological injury results in the adverse outcome after cardiac operation. The long term neurological sequelae involve impairment of cognitive function and focal neurological deficits.

Treatment of acute and severe postoperative neurological event is only supportive, and is often associated with poor prognosis. Therefore, identifying several risk factors for neurological injury and adopting several strategies to prevent injury are paramount.

7.1 Risk Factors for Neurological Injury

7.1.1 Preoperative Factors

Age and Periventricular leukomalacia (PVL):

Seen in infants undergoing CPB with or without deep hypothermic circulatory arrest (DHCA). It has high incidence in neonates compared to infants (54% vs 5%). Neonatal brain is more vulnerable to hypoxia-reperfusion injury due to immaturity of oligodendrogliocytes.

Preoperative neurological status:

↑ Prevalence of existing neuro-abnormalities in newborns (> 50%) and infants (38%) undergoing open heart surgery (OHS).

Preoperative clinical findings:

Hypotonia, jitteriness, motor asymmetries, microcephaly, and seizures (7%).

Above manifestations require thorough preoperative neurological assessment and neuroimaging for detection of ventriculomegaly, intraventricular hemorrhage, cerebral atrophy, and aqueductal stenosis.

Oxygen saturation:

↑ Incidence of neuro-abnormalities in infants with SaO_2 < 85% as compared to SaO_2 > 85%. Therefore, corrective surgery at a younger age is prudent.

Cardiac diagnosis:

Risk of perioperative cerebral injury and long-term neurologic sequelae correlate with the diagnosis.

Highest risk (class IV): Single ventricle with arch obstruction (HLHS).

Moderately severe risk (Class III): Single ventricle without arch obstruction.

Moderate risk (Class II): Two ventricles with arch obstruction.

Low risk (Class 1): Two ventricles without arch obstruction.

Genetic factors:

Following genetic abnormalities are associated with poor neurological outcomes.

1. Apolipoprotein E genotype (Ee2 allele) and postoperative neurodevelopmental dysfunction.

2. Velocardiofacial syndrome (partial deletion of chromosome 21).

7.1.2 Intraoperative Factors

(I) Low Flow CPB VS. Deep Hypothermic Circulatory Arrest (DHCA)

No difference in neurocognitive outcome between both strategies.

Both result in inferior long term neurodevelopmental outcome compared to normal population (see Figure 26).

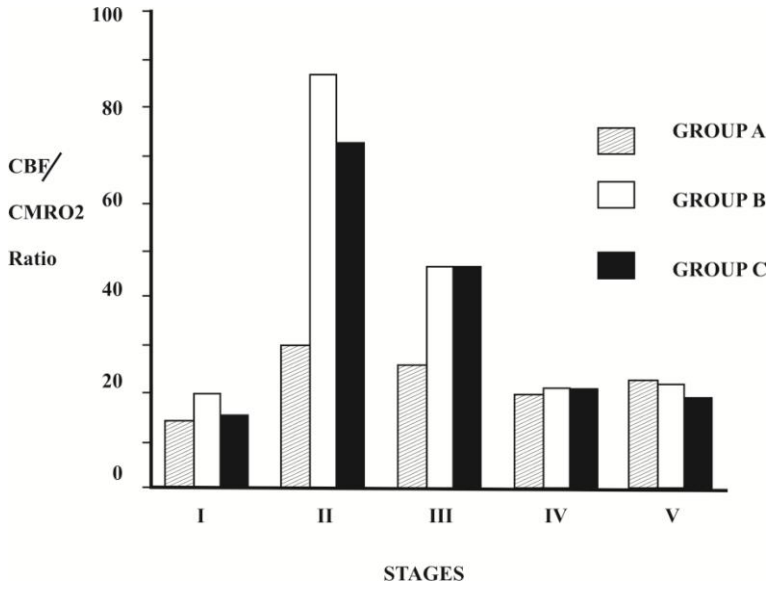

Figure 26 *Cerebral blood flow/cerebral metabolic rate of oxygen coupling (CBF/CMRO2) during cardiac surgery. Group A-moderate hypothermia; Group B-deep hypothermic cardiopulmonary bypass (CPB) with maintenance of continuous flow, Group C-deep hypothermic circulatory arrest. Stage I = pre CPB; Stages II and III = CPB cold; Stage IV = CPB rewarming; Stage V = post CPB. Note that despite reductions in CBF, the ratio of CBF/CMRO$_2$ returns to basleline levels during rewarming and Post CPB in groups B and C. In group B and C the increased ratios reflect significant reduction of cerebral metabolism due to deep hypothermia. *Reproduced from: Angelo Polito, Zaccaria Ricci et al, Cerebral blood flow during cardiopulmonary bypass in pediatric cardiac surgery: Cardiovascular Ultrasound 2006).*

Notes: (Cerebral metabolism: Though cerebral blood flow (CBF) decreases in a linear manner, CMRO$_2$ decreases exponentially with reduction of temperature, thus increasing ratio of CBF/CMRO$_2$ during deep hypothermic cardiopulmonary bypass (DHCPB) favoring luxury perfusion of the brain).

Cerebral blood flow and cerebral vascular resistance:

Profound hypothermia and low-flow CPB (DHCPB) or deep hypothermic circulatory arrest (DHCA) exhibit increased cerebral vascular resistance and decreased velocity of flow in the middle cerebral artery (V MCA) in the early postoperative period (occurs with increased frequency with DHCA). This is not seen with moderate hypothermic CPB. Normal coupling of CBF/CMRO$_2$ is present both before and after normothermic or moderate hypothermic CPB. During CPB rewarming, CBF returns to baseline values, except in DHCA where CBF remains decreased.

Deep Hypothermic circulatory arrest and CBF:

Low cerebral perfusion immediately following DHCA is characterized by a prolonged period of absent diastolic cerebral blood flow velocity (CBFV) in MCA. This finding was explained by an increased intracranial pressure after total circulatory arrest procedure, while patients subjected to

continuous low-flow perfusion technique showed CBFV close to baseline values at skin closure. Delay in rewarming on reperfusion after DHCA improved recovery of a diastolic doppler signal compared with patients who underwent immediate rewarming. In the group undergoing cold reperfusion, post bypass CBF velocity was not different from baseline.

Cerebral perfusion pressure, Cerebral blood flow, Pump flow rate, and Mean arterial pressure:

Loss of cerebral auto regulation occurs between 23 ℃ and 25 ℃. In the absence of cerebral vascular disease, the flow rate threshold for incurring functional cerebral injury during hypothermic (25 degrees C) nonpulsatile CPB is less than 1.0 L/min/m^2. Cerebral perfusion pressure (CPP) is a crucial parameter, rather then pump flow rate, in impacting brain perfusion. During moderate hypothermia and DHCPB (low-flow) there was immediate loss of detectable CBFV in MCA when cerebral perfusion pressure (CPP) decreased below 9 mm Hg. The cerebral perfusion can be detected in the MCA in some neonates at bypass flow as low as 10 mL/kg per minute. However a minimum bypass flow rate of 30 mL/kg per minute is needed to detect cerebral perfusion in all neonates. All patients with a mean arterial blood pressure (MAP) of 19 mm Hg or greater, regardless of pump flow rate, have detectable cerebral perfusion by trans-cranial doppler (TCD), but correlation between MAP and CPB pump flow rates was minimal. However, MAP alone is a poor indicator of CPP.

(II) pH Strategy: Alpha Stat / pH Stat

During CPB, CBF increases with increasing arterial carbon dioxide tension; this response is diminished by deep hypothermia and age less than 1 year.

In the pH-stat management, $PaCO_2$ is maintained at 40 mm Hg regardless of temperature changes (temperature-corrected), while in α-stat management $PaCO_2$ is not adjusted (temperature-uncorrected).

During moderate hypothermia CBF decreases significantly only with alpha stat management as compared to no decrease in pH stat management. However, other studies demonstrated that during α-stat management CBFV is less pressure passive than during pH-stat management, and that there is a better matching of cerebral metabolism to CBFV with α-stat management.

DHCA and pH stat:

pH stat is currently recommended for operations involving DHCA.

Alpha stat is favored for operations involving moderate hypothermia.

Advantages of pH stat: It has better cardiac output in the postoperative period, shorter recovery time to first EEG, a lower incidence of EEG seizures, lower perioperative morbidity and mortality,

and shorter intensive care unit (ICU) stay. There is no difference in psychomotor development between alpha and pH stat groups (see Figure 27).

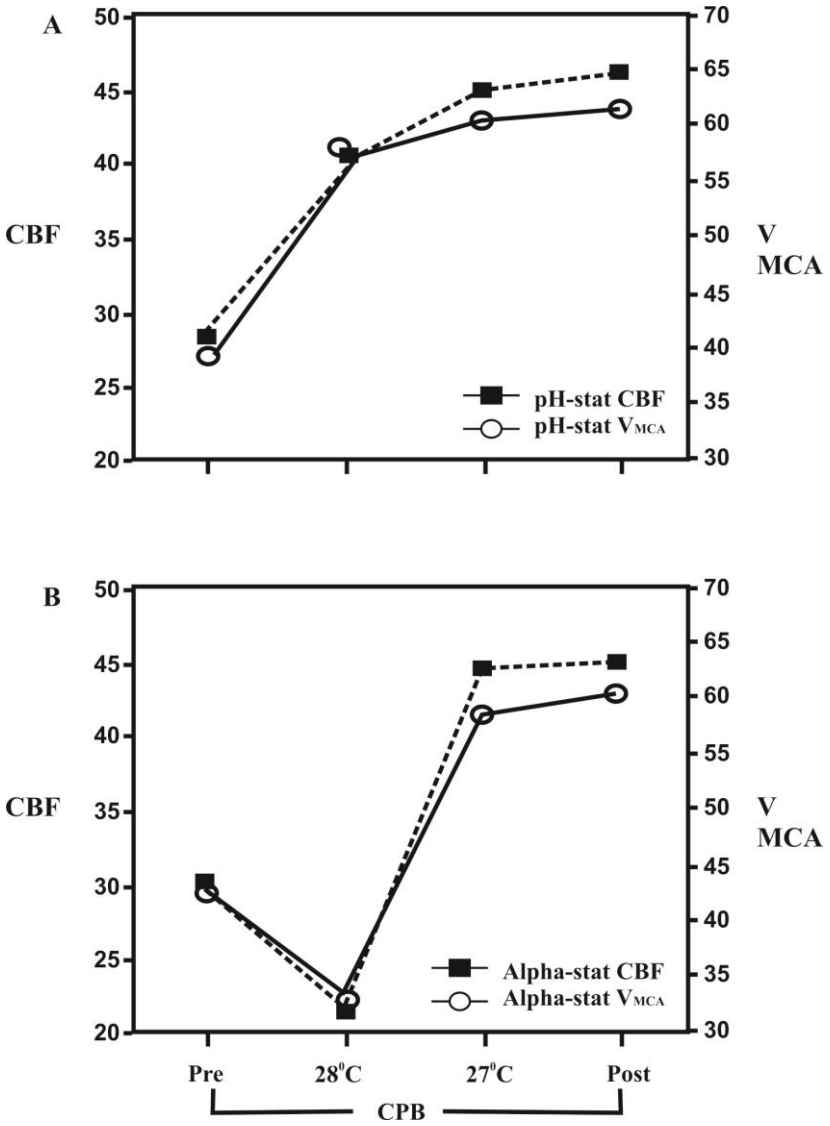

Figure 27 **A. pH-stat management and B. α-stat acid-base management on CPB and changes in cerebral blood flow (CBF) and cerebral blood flow velocity (CBFV) in infants. There is good correlation of CBF (by xenon-133 clearance technique) with V MCA (by TCD). CPB=Cardiopulmonary bypass, CBF= Cerebral blood flow (mL/100 g/minute). V MCA= Flow velocity in middle cerebral artery (cm/second) *Reproduced from: Angelo Polito, Zaccaria Ricci et al, Cerebral blood flow during cardiopulmonary bypass in pediatric cardiac surgery: Cardiovascular Ultrasound 2006).*

(III) Optimal Hematocrit

Hemodilution impairs oxygen delivery by shifting the oxygen dissociation curve to left. Extreme hemodilution (hematocrit < 10%) leads to inadequate O_2 delivery during early cooling. High hematocrit (30%, as blood prime) leads to improved cerebral recovery after DHCA.

(IV) Optimal Temperature and Post-Operative Hyperthermia

Hypothermia is the most powerful neuroprotective mechanisms during DHCA.

Cerebral metabolic demand for oxygen ($CMRO_2$) ↓ exponentially and results in progressive increase in ratio of CBF / $CMRO_2$.

(CBF = cerebral blood flow).

Gradual cooling prior to DHCA reduces neurological events compared to a rapid cooling. Hyperthermia in rewarming phase significantly increases neurological damage. Maintenance of normothermia in the postoperative period reduces the incidence of neurological injury.

(V) Pulsatile Flow (Pf) Versus Non-pulsatile Flow (NPf)

Critical opening pressure of the capillary bed is lower with pulsatile flow. At normothermia, CBF is higher over a wide range of mean arterial pressures with a pulsatile flow. In infants since the cerebrovascular resistance is low, Pf ↓ mean arterial pressure. There is no concrete evidence in favor of Pf regard to superior cerebral hemodynamics or neurological outcomes.

7.1.3 Postoperative Factors

(I) Longer ICU Stay

It is a marker for ↓ cardiac output, infection, end organ dysfunction/failure, etc, and is a risk factor for a long-term neurodevelopmental sequelae.

(II) Clinical or EEG Seizures

If these occur in the immediate postoperative period, they are associated with poor neurological outcome on a follow-up.

(III) Diastolic Hypotension / Hypoxemia

If any one of the above occurs in postoperative period, it is a risk factor for periventricular leukomalacia in neonates.

7.2 Neuroprotective Strategies

These involve inhibition of biochemical and metabolic cascade after ischemia to prevent neuronal death in pediatric cardiac surgery.

7.2.1 Optimal CPB Techniques

Recommendations:

1. Deep hypothermic circulatory arrest (DHCA) vs. Low flow cardiopulmonary bypass (LF-CPB): Immediate postoperative outcomes are better with LF-CPB, but there are no differences in long term outcomes.

2. DHCA: Minimize duration < 41 minutes. Provide intermittent cerebral perfusion for 1-2 minutes every 15 to 20 minutes.

3. pH management: pH stat for DHCA and alpha stat for moderate hypothermia.

4. Hematocrit: Optimal around 30.

5. Temparature: < 18 degree C for DHCA, No differences are observed between surface and core cooling. Avoid rapid cooling and cool > 20 minutes before DHCA. Avoid rapid re-warming and hyperthermia post-operatively.

6. Modified ultra filtration: Reduces cerebral edema and SIR (systemic inflammatory response).

7.2.2 Neuroprotective Drugs

Methyl prednisolone: Reduces inflammation with early recovery of cerebral function post-CPB.

Dose: IV 8 mg/kg 8 hours and 2 hours preoperatively in neonates (high risk group).

Following are not recommended or no data to support any benefit in humans.

Barbiturates: Reduces high energy phosphates (detrimental in DHCA), suppresses EEG activity.

Allupurinol, NAC (N-acetyl cysteine) reduces free radical injury. NAC improves myocardial function.

NMDA antagnosists (magnesium, selfotel): Reduce excitotoxicity.

Aprotinin: It helps recovery of cerebral high energy phosphates, but has no benefit in CPB of children.

Calcium channel blockers (nimodipine): It reduces reperfusion injury due to calcium influx.

7.2.3 Selective Cerebral Perfusion

(I) Retrograde Cerebral Perfusion (RCP)

Brain is perfused retrograde via superior vena cava. Used in adults undergoing aortic arch reconstruction, but has limited application in children as small amounts of blood returning into field retrograde will result in unsatisfactory exposure.

(II) Cerebroplegia

University of Wisconsin solution containing adenosine (NMDA blocker): It potentially reduces the excitotoxicity associated with ischemia-reperfusion injury. But no randomized controlled trials of this strategy have been done in children.

(III) Antegrade Cerebral Perfusion

A special and small aortic cannula placed through right innominate artery selectively perfuses the brain. Other brachiocephalic vessels and descending aorta are snared. This technique eliminates DHCA with improved neurological outcomes.

7.3 Neurological Monitoring

7.3.1 Functional Studies

Electro-encephalogram (EEG), somatosensory-visual, auditory evoked potentials, and electromyography (EMG).

7.3.2 Cerebral Blood Flow Velocity

It is estimated by doppler ultrasonography of middle cerebral artery (MCA) by a trans-cranial doppler. A 50% reduction in blood flow is considered significant.

7.3.3 Cerebral Oxygenation

Jugular venous bulb saturation and NIRS (near infra-red spectroscopy) is a method for monitoring the oxygenation in blood and in the brain tissue of a neonate. Absorption of light in the wavelength range 700-1000 nm through such tissue is measured, which is then used to calculate changes in the concentration of cerebral oxygenated and de-oxygenated haemoglobin (HbO_2 and Hb) and hence, cerebral blood volume (CBV). Studies have shown clear changes in HbO_2, Hb and CBV with hypoxia and bradycardia. These changes may be implied in the occurrence of hypoxic/ischemic brain injury.

7.3.4 Biochemical Markers

CPK-BB, lactate, neuron-specific enolase (NSE), S-100 protein, and myelin basic protein are markers for brain injury. Cerebral ischemia during CPB correlates with elevated lactate and CPK-BB. Duration of CPB correlates with appearance of S-100 protein. CNS dysfunction post-CPB correlates with presence of markers such as S-100 protein and NSE.

7.3.5 Glasgow Coma Scale (GCS)

The scale comprises three tests: Eye, verbal, and motor responses.

Three values separately as well as their sum are considered.

Lowest GCS (the sum) score is 3 (deep coma or death), highest is 15 (fully awake person) (see Table 7.1).

Table 7.1 *Glasgow Coma Scale.*

	1	2	3	4	5	6
Eyes	Does not open eyes	Opens eyes in response to painful stimuli	Opens eyes in response to voice	Opens eyes spontaneously	N/A	N/A
Verbal	Makes no sounds	Incomprehensible sounds	Utters inappropriate words	Confused, disoriented	Oriented, converses normally	N/A
Motor	Makes no movements	Extension to painful stimuli	Abnormal flexion to painful stimuli	Flexion / Withdrawal to painful stimuli	Localizes painful stimuli	Obeys Commands

Tests for assessing Glasgow Coma Scale:

Best eye response (E):

There are 4 grades.

1. No eye opening,

2. Eye opening in response to pain:

Responds to pressure on the patient's finger nail-bed. If this does not elicit a response, supraorbital / sternal pressure or rub may be used.

3. Eye opening to speech. But not to be confused with an awaking of a sleeping person; such receive a score of 4, not 3.

4. Eyes opening spontaneously.

Best verbal response (V):

There are 5 grades:

1. No verbal response.

2. Incomprehensible sounds (moaning but no words).

3. Inappropriate words. Random or exclamatory articulated speech, but no conversational exchange.

4. Confused. Responds to questions coherently, but there is disorientation and confusion.

5. Oriented. Patient responds coherently and appropriately to questions such as the name and age, where they are and why, the year, month, etc.

Best motor response (M):

There are 6 grades.

1. No motor response.

2. Extension to pain adduction of arm, internal rotation of shoulder, pronation of forearm, extension of wrist, (decerebrate response).

3. Abnormal flexion to pain adduction of arm, internal rotation of shoulder, pronation of forearm, flexion of wrist, (decorticate response).

4. Flexion/withdrawal to pain flexion of elbow, supination of forearm, flexion of wrist when supra-orbital pressure is applied; pulls part of body away if nail bed is pinched.

5. Localizes to pain purposeful movements towards painful stimuli; e.g., hand crosses mid-line and gets above clavicle when supra-orbital pressure is applied.

6. Obeys commands (the patient does simple things as asked).

Interpretation of Glasgow Coma Scale:

Individual elements as well as the sum of the score are important.

The score is expressed in the form "GCS 9 = E2 V4 M3 at a specific time."

Generally, comas are classified as:

Severe GCS $\leq$ 8, moderate GCS 9 – 12, and minor GCS $\geq$ 13.

If the patient is intubated and with severe facial/eye swelling, it is impossible to test the verbal and eye responses. In these circumstances, the score is given as 1 with a modifier attached, e.g., 'E1c' where 'c' = eyes closed, or 'V1t' where t = intubated.

A patient with eyes closed because of eye swelling (E1c), and intubated (V1t) with a motor response score of 3 for abnormal flexion has a composite score of 'GCS 5tc'.

Pediatric Glasgow Coma Scale (PGCS):

The verbal performance of even a healthy child is expected to be poor.

The GCS has limited applicability to children < 36 months of age.

PGCS is used for assessing younger children.

It comprises three tests: Eye response (E), verbal response (V), and motor reponse (M). The lowest PGCS (the sum) is 3 (deep coma or death).

The highest PGCS (the sum) is 15 (fully awake and aware infant).

Best eye response (E)

1. No eye opening

2. Eye opening to pain.

3. Eye opening to speech.

4. Eyes opening spontaneously.

Best verbal response (V):

1. No verbal response.

2. Infant moans to pain.

3. Infant cries to pain.

4. Infant is irritable and continually cries.

5. Infant coos or babbles (normal activity).

Best motor response (M):

1. No motor response.

2. Extension to pain (decerebrate response).

3. Abnormal flexion to pain in an infant (decorticate response).

4. Infant withdraws from pain.

5. Infant withdraws from touch.

6. Infant moves spontaneously or purposefully.

The lowest PGCS (the sum) is 3 (deep coma or death).

The highest PGCS (the sum) is 15 (fully awake and aware infant).

Any combined score of < 8 represents a significant risk of mortality.

7.4 Neurological Injury / Complications

These may present acutely in the postoperative period / or insidiously as transient / long-term permanent neurocognitive sequelae.

1) Acute neurological disorders: Seizures, cerebrovascular accidents, choreoathetosis, spinal cord injury, and delayed recovery from anesthesia.

2) Long-term neurodevelopmental sequelae:

i) Cognitive impairment, speech, and language problems.

ii) Visual-spatial and visual-motor skills impairment.

iii) Attention deficit / hyperactivity disorder, motor delays, and learning disabilities.

7.4.1 Seizures

The incidence is 6% for early seizures (occur in first postoperative week) and are transient. The incidence is 20% for ictal activity on (EEG) monitoring. These correlates with poor neurodevelopmental outcomes and abnormal MRI at ages 1 and 2½ years.

Seizures with stroke carries poor prognosis as compared to isolated “post-pump” seizures. The risk factor is DHCA if it’s longer duration, as in correction of D-TGA with VSD. Cerebrovascular accident (stroke): Occurs in patients with uncorrected congenital heart disease.

Clinical presentation of seizures:

The presentation varies depending on the age.

In infants, focal seizures or changes in mental status occur.

In older children: focal motor deficits, language, and visual deficits occur.

Contributing factors for seizures:

↑ CVP (predisposing to cerebral venous thrombosis), emboli, prosthetic material, residual defects, fenestration, procoagulant shift in the humoral clotting, and SIR (systemic inflammatory response) secondary to CPB.

7.4.2 Choreoathetosis (Movement Disorders)

Its incidence is of 0.5-19% of children undergoing OHS.

Transient and mild form: It is confined to distal extremities, and is seen in infants < 10 months.

Progressive and severe form: It involves proximal extremities, and occurs in children > 1 year, with high 40% incidence of mortality. Its associated with high incidence of persistent neurodevelopmental defects.

7.4.3 Spinal Cord Injury

Paraplegia:

Incidence: 0.41% and associated with surgery of descending thoracic aorta.

Risk factors: Poor collateral circulation (infants), pre-subclavian coarctation, stenosis of left subclavian artery, re-coarctation repair, prolonged duration of aortic cross clamping, hyperthermia, higher level of aortic clamping, hypotension of upper body, and elevated intraspinal pressure.

Preventive Techniques: Temporary ascending to descending aorta bypass, hypothermia, CSF drainage, localized cooling of spinal cord, and use of corticosteroids, naloxone, and papaverine.

7.5 Treatment of Neurological Complictions

7.5.1 Seizure Disorders

Treatment varies depending on the type of seizure disorder.

Definitions of seizure disorders:

Seizure: Paroxysmal cortical neuron discharge results in alteration of motor, sensory, or cognitive function.

Epilepsy: Occuerence of two or more seizures not precipitated by a nown cause.

Status epilepticus: Recurrent or prolonged seizure lasting 30 minutes without regaining consciousness.

Febrile seizure: Generalized seizure related to febrile illness without neural cause (low seizure threshold). Usually treatment is not indicated, but need neurologic evaluation if onset is 24 hours after febrile illness; seizure lasts for 15 minutes or more with focality, and two episodes of seizures occur within 24 hours.

Neonatal Seizures: Paroxysmal electrical or behavioral alterations including tonic, myoclonic, clonic, or subtle events due to CNS immaturity.

Etiologic agents:

i) Hypoxic brain injury, idiopathic (primary), metabolic, and genetic.

ii) Acquired cortical lesions (e.g., infection, air- embolus / stroke, hemorrhage, etc.).

Diagnosis: Determine whether the seizure is primary generalized or primary partial.

1. Primary generalized (generalized EEG changes at onset).

2. Primary partial (focal EEG changes at onset of seizure):

A. Partial-simple (consciousness preserved) with motor, sensory, and autonomic changes.

B. Partial-complex (unconsciousness).

C. Partial with secondary generalization: Grandmal tonic clonic seizures.

Treatment:

It is individualized; risk of further seizure is weighed against risk of treatment related complication.

Ensure adequate airway, oxygenation, and circulation.

Correct the metabolic abnormality (especially in neonatal seizures).

Hypoglycemia:

0.25-0.5 g/kg IV bolus of (10% dextrose solution) followed by 8 mg/kg/minute drip if required.

Hypocalcemia:

10 mg/kg of elemental calcium given as calcium gluconate by slow IV infusion.

Medications:

a) Phenobarbital: 15-20 mg/kg IV loading dose.

Repeat additional boluses of 5-10 mg/kg, IV as necessary up to 30-50 mg/kg.

If there is no satisfactory response, add phenytoin and / or benzodiazepine.

b) Phenytoin: 15-20 mg/kg IV loading. Do not give > 1 mg/kg/minute. Give drug under ECG monitoring.

c) Diazepam: 0.3 mg/kg IV bolus or 0.3-0.8 mg/kg / hour IV infusion.

d) Lorazepam: 0.05-0.1 mg/kg/IV over 2-5 minutes.

e) Pyridoxine: Supplement with 50-100 mg IV during seizure.

EEG monitoring should be done for refractory neonatal seizures.

Persistent Seizures (Status Epilepticus):

Immediate treatment:

1. Assure adequate airway (A), breathing (B), and circulation (C) (plus intravenous access). Stabilize vital signs.

2. Administer adequate $FI0_2$ and treat hypoglycemia.

3. Laboratory tests: CBC, serum electrolytes, Ca^{2+}, Mg^{2+}, glucose, urea, ceatinine, bilirubin, liver enzymes, and blood cultures (for suspected infection only).

4. Clinical assessment by history and physical exam.

5. Pharmacological therapy:

Lorazepam: 0.05-0.1 mg/kg IV up to 4-6 mg / or

Diazepam: 0.3 mg/kg IV (rectal dose 0.5 mg/kg) up to 6-10 mg.

May repeat lorazepam or diazepam every 5-10 minutes as necessary.

If seizures persist despite above medication:

Phenytoin 15-20 mg/kg at 1 mg/kg/minute slowly (maximum rate 50 mg/minute),

or

Phenobarbital: 20 mg/kg IV at 1 mg/kg/minute slowly.

If seizure still persists:

Give a loading dose of phenobarbital if phenytoin is used above or

Give additional loading of dose of phenobarbital 10 mg/kg IV.

Obtain neurological consultation.

7.5.2 Coma

1. Clinical Assessment:

a) History sequence, physical exam for BP, pulse, and respiratory pattern.

b) Temparature, abnormal posture, pupils, focal neurological signs and deficits, and monitor Glasgow coma scale.

2. Laboratory Evaluation:

Tests are based on above findings: CBC, electrolytes, serum glucose, ammonia, serum lactate, blood gas, and blood culture.

3. Radiological examination: CT scan of head and electroencephalogram (EEG).

4. Management: Immediately perform ABCs of resuscitation.

Airway and breathing: Assure adequacy of airway, adequate oxygenation, and gas exchange.

Circulation: Establish stable vital signs.

Dexstrostick: D 25W, 2-4 mL/kg IV bolus for hypoglycemia.

5. Optional therapy: (may not be indicated for postoperative hypoxic brain injury).

Naloxone: 0.1 mg/kg IV, maximum 2 mg/dose; may repeat if necessary.

Thiamine: 50 mg IV. It is given in deficiency states and before glucose administration.

Reduce intracranial pressure if it is suspected to be elevated.

6. Neurological consultation.

7.5.3 Increased Intracranial Pressure (ICP)

1. Clinical assessment:

a) History, visual and gaze changes, irritability, change in mental status, lethargy, and bulging fontanel.

b) Cushing's response (hypertension, bradycardia, abnormal respiratory pattern.), neck stiffness, cranial nerve dysfunction, i.e., paralysis of upward gaze or abduction, abnormal posturing, neurological deficit, and papilledema.

2. Management:

1) A. Elevate head of the bed 30 degrees and observe cardiac monitor.

B. Lab tests: CBC, electrolytes, glucose, urea, and blood culture (for suspected infection).

C. CT scan of head and neurological consult.

(Above are done in that order, presumed that ABC of resuscitation are taken care of).

2) Fluid restriction: It is done in hemodynamically stable patients.

Mannitol 0.25-1 gm/kg IV or / and furosemide 1 mg/kg to decrease intracranial pressure.

3) Maintain paralysis and sedation in intubated patient, but avoid ketamine in induction.

4) Keep $PaCO_2$ of 25-35 mm Hg.

5) Treat hyperthermia.

6) Do not lower BP in elevated ICP.