Encephalopathy, Postanoxic

Charles Weissman

Risk

- After successful prehospital cardiac resuscitation: 59–65% of pts remain comatose.
- 0–5% of successful resuscitations result in chronic vegetative state.

Perioperative Risks

- Worsening of neurologic status; blindness most common residuum.
- Postpone surgery in all but emergency situations.
- Do what is necessary to treat precipitating cause and to decrease sequelae (e.g., treat elevated ICP).

Worry About

- Repeat of events that initially caused encephalopathy (e.g., arrhythmias leading to cardiac arrest)
- Hypotension, hypercapnia, hypoxia, and sepsis that can exacerbate encephalopathy

Overview

- Brain injury resulting from prolonged period of insufficient cerebral oxygenation.
- Clinical picture ranges from mild confusion to brain death.
- Chances for acceptable neurologic recovery: 1% with continued coma after 24 h and lack of two of the following reflexes: Pupillary, corneal, and oculovestibular.
- Absence of brainstem function 72 h after event associated with irreversible coma.
- Therapeutic hypothermia (especially after cardiac arrest with initial VFIB or VTach) improves neurologic outcome.
- Good prognosis seen in 50% of pts awakening within 24 h of insult.
- Seizures occur in 25% of pts.
- Anoxic damage may have been sustained by other organs (e.g., MI, shock liver, acute renal failure, stress ulcers, ARDS).
- DI is poor prognostic sign.

Etiology

- Caused by inadequate O_2 delivery to CNS due to inadequate cardiac output, resp dysfunction, severe anemia, and/or increased ICP
- Most often secondary to primary cardiac (MI or arrhythmia) or pulm (asthma, pulm embolism) event
- May also be result of CO poisoning, suffocation, and cyanide poisoning

Usual Treatment

- Prevent recurrence of inciting event.
- Ventilatory and hemodynamic support as needed.
- Therapeutic hypothermia to 32–34° C for 12–24 h.
- Stress ulcer prophylaxis.
- Treatment of seizures (with anticonvulsants, e.g., phenytoin) and myoclonus.
- BP should be maintained at normotensive or mildly elevated levels in normotensives and higher in hypertensives.
- Treat fever promptly with antipyretic drugs.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	MI	Assess if cardiac disease was cause of arrest		ECG, other cardiac assessment Troponins, CK
RESP	ARDS	Assess if resp disease was cause of arrest Resp failure	Wheezing, stigmata of COPD	Pre-arrest PFTs ABGs
GI	Shock liver Stress ulceration	Hx of GI bleeding	Jaundice	AST, ALT, bilirubin, alkaline phosphatase Hct NG output
RENAL	Renal failure	Assess if lyte abnormality or acidosis caused initial event	Urine output	BUN/Cr
CNS	Altered mental status, diffuse and focal neurologic abnormalities	Changes in neurologic signs since hypoxic event, seizures	Neurologic and mental status exams, apnea test, brainstem reflexes	CT scan/CT angiography, MRI/MRA EEG SSEP, BAER
MS	Myoclonus, posturing Contractures	Abnormal movements, posturing Prolonged immobility	Decerebrate or decorticate postures, myoclonus Contractures	

Key References: Lippa CF, Moonis M: Generalized anoxia/ischemia of the nervous system. In Irwin RS, Rippe JM, editors: *Intensive care medicine*, ed 7, Philadelphia, PA, 2012, Wolters Kluwer/Lippincott Williams & Wilkins, pp 1768–1771; Topjian AA, Berg RA, Taccone FS: Haemodynamic and ventilator management in patients following cardiac arrest, *Curr Opin Crit Care* 21(3):195–201, 2015.

Perioperative Implications

Preoperative Preparation

- Assess and document neurologic function and mental status.
- Review cause of anoxic event.
- Assess damage to other organs.
- If pt hypothermic, beware of possible increased blood loss.

Monitoring

- If arrest was due to cardiac arrhythmias or MI/ischemia or if pt is hemodynamically unstable, may need specialized monitoring

Airway

- Assess potential for aspiration: Gag reflex and ability to cough and clear secretions.

Induction

- Avoid succinylcholine.

Maintenance

- Must consider that pts may have pain perception and will require analgesia.
- Do what is appropriate to decrease sequelae (e.g., treat increased ICP); therapeutic hypothermia.
- If being treated with therapeutic hyperthermia drug, clearance is reduced.

Extubation

- If unable to maintain patent airway or sustain adequate minute ventilation, pt should remain intubated.

Adjuvants

- Avoid long-acting anesthetics so that neurologic status can be assessed soon after surgery.
- Avoid drugs that decrease seizure threshold.

Anticipated Problems/Concerns

- Repeat of events (e.g., arrhythmias) that initially led to anoxic encephalopathy.
- Worsening of neurologic condition during periop period.
- Seizures and myoclonus.
- Postpone all but emergency surgery if fluctuating neurologic deficits or acute encephalopathic condition exists.

Endocardial Cushion Defect (Atrioventricular Canal)

Julie K. Freed | Paul S. Pagel

Risk

- 4% of all congenital heart disease and 0.3–0.4:1000 live births
- 40–50% of AV canal defects are associated with trisomy 21

Perioperative Risks

- Paradoxical air embolism
- Shunt reversal (from left to right to right to left) because of vasodilating volatile and IV anesthetics (reduced systemic vascular resistance)
- Endocarditis; prophylactic antibiotics for pts with a complete repair or a jet lesion
- Arrhythmias after AV canal repair
- Reactive pulmonary vasculature and PAH

Worry About

- Bradycardia
- PAH, RV failure, and shunt reversal
- Atrial arrhythmias resulting from atrial enlargement

Overview

- AV canal is associated with atrial and ventricular septal defects manifested by a variety of abnormal communications between the left and right heart structures.
- Categorized into atrial septal defects and partial or complete AV canal defect.
- Main hemodynamic problems include AV valve dysfunction, interatrial shunting, and interventricular shunting.
- L-to-R shunting results in RV or LV dysfunction or failure, frequent resp infections, and failure to thrive.
- Chronic L-to-R shunt causes increased pulmonary vascular resistance and shunt reversal (Eisenmenger syndrome), which may preclude surgical intervention.
- Diagnosis includes chest radiograph (enlarged heart), physical exam findings (murmur), prolonged electrocardiogram PR interval, and ECHO.

Etiology

- AV canal defects arise from abnormal endocardial cushion development between 4–5 wk gestational age.
- Failure of endocardial cushion fusion results in deficiencies in the interventricular septum that can form a common AV valve, common AV valve annulus, or interatrial communication.

Usual Treatment

- Medical management (before repair) directed to improve cardiac function and overall health (digitalis, diuresis, positive inotropic drugs, afterload reduction, adequate nutrition).
- Surgical management is definitive; includes repair of the septal defects and AV valves.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Feeding difficulties	Failure to thrive	Decreased weight/height for age	Compare with ideal weight/height
CV	CHF PAH	Fatigue, dyspnea, diaphoresis, coughing Dyspnea, tachypnea	Murmur, wheezing, rales, hepatosplenomegaly Increase in CHF Sx	CXR, TEE, cardiac cath TEE, cardiac cath
RESP	CHF, pneumonia	Dyspnea, tachypnea	Wheezing, rales	CXR
RENAL	Renal insufficiency			Cr, BUN
MS	Exercise intolerance			

Key References: Wenink AC, Zevallos JC: Developmental aspects of atrioventricular septal defects, *Int J Cardiol* 18(1):65–78, 1988; Bergin ML, Warnes CA, Tajik AJ, et al.: Partial atrioventricular canal defect: long-term follow-up after initial repair in patients ≥40 years old, *J Am Coll Cardiol* 25(5):1189–1194, 1995.

Perioperative Implications

Preoperative Preparation

- Midazolam (0.05–0.1 mg/kg) to reduce anxiety and facilitate cooperation.
- Anxiolytics not recommended for children <1 y of age.
- Use caution as anxiolytics may cause hypoventilation, hypercapnia, increased PVR, and shunt reversal.

Monitoring

- Standard ASA monitors; arterial and central venous cath
- CVP monitoring in the setting of PAH
- TEE if not contraindicated

Airway

- Anticipate difficulty when trisomy 21 is present

Induction

- Meticulous exclusion of air from IV tubing to avoid paradoxical air embolism.
- Inhalation induction time is minimally affected by L-to-R shunt but may be prolonged in R-to-L shunt.
- Choice of IV anesthetic for induction based on severity of heart failure.

Maintenance

- Decrease in afterload due to IV or volatile anesthetics may worsen R-to-L shunt.
- Adjuvant opioids to allow use of lower volatile anesthetic concentrations.

Extubation

- Extubation feasible in the operating for partial AV canal defects without heart failure or PAH.
- Airway obstruction or hypoventilation after extubation may increase PVR, requiring subsequent hyperventilation, increased FIO_2, or inhaled nitric oxide, or ECMO.

Adjuvants

- Positive inotropic drugs to enhance myocardial contractility
- Inhaled nitric oxide or prostaglandin I_2 to reduce PVR

Postoperative Period

- Closely monitor and reduce pulm artery pressures in pts with preop PAH.
- Reduced cardiac output may occur as a result of RV or LV dysfunction or LV outflow tract obstruction.
- May require temporary transvenous pacing or develop postop arrhythmias.

Anticipated Problems/Concerns

- Presence of arrhythmias, reduced cardiac output, moderate or severe mitral regurgitation, and elevated PVR is associated with greater risk of mortality

Endocarditis

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Risk

- Incidence: 3–10:100,000 population.
- Rheumatic heart disease is a key risk factor in lower-middle income countries.
- Valvular and cyanotic heart disease, DM, cancer, and IV drug use are risk factors in higher income countries.

Perioperative Risks

- Septic embolization to other organs (CNS, renal, and lung) is seen in 25–50% of pts.

Worry About

- Acute heart failure (valvular regurgitation or obstruction), stroke, and metastatic infection (i.e., epidural abscess/osteomyelitis)
- AV or bundle branch blocks due to infection extension

Overview

- IE is an infection of the heart, most commonly seen in pts with structural heart disease (i.e., CHD, mitral valve prolapse).
  - Acute infections commonly caused by *Staphylococcus aureus* and *S. epidermidis*.
  - Subacute infections commonly caused by *Streptococcus viridans* or HACEK group organisms (*Haemophilus* species, *Actinobacillus actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corrodens*, and *Kingella* species).
  - *S. aureus* bacteremia is highly associated with IE, and ECHO should be obtained with all *S. aureus*-positive blood cultures.
- Non-IE can be seen with systemic inflammatory disorders, such as SLE (Libman-Sacks endocarditis).
- Left-sided valves (mitral, aortic) are most commonly affected, except in IV drug users, in whom right-sided valves (tricuspid > pulmonary) predominate.
- IE diagnosed by the modified Duke criteria and presentation can be variable and nonspecific.

Etiology

- Autoimmune activity or bacterial colonization leads to endothelial injury and valvular damage.
- *S. aureus* is the most common bacterial isolate in high-income countries.
- Oral viridans group is the most common bacterial isolate in lower-middle income countries.
- Group D streptococci is classically seen in pts with colonic tumors.