

Rehabilitation of Anoxic Brain Injury

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Objectives

- Discuss common causes of anoxic brain injury
- Discuss pathophysiology of anoxic brain injury focusing on cardiac arrest
- Discuss rehabilitation of patients with anoxic brain injury
- Highlight patient cases



Etiology of Anoxic Brain Injury

- Cardiac failure
- Respiratory failure
- Carbon monoxide poisoning
- Cyanide poisoning

Cardiac failure

- Cardiac pathologies – ventricular arrhythmia; myocardial infarction
- Massive blood loss
- Traumatic or septic shock

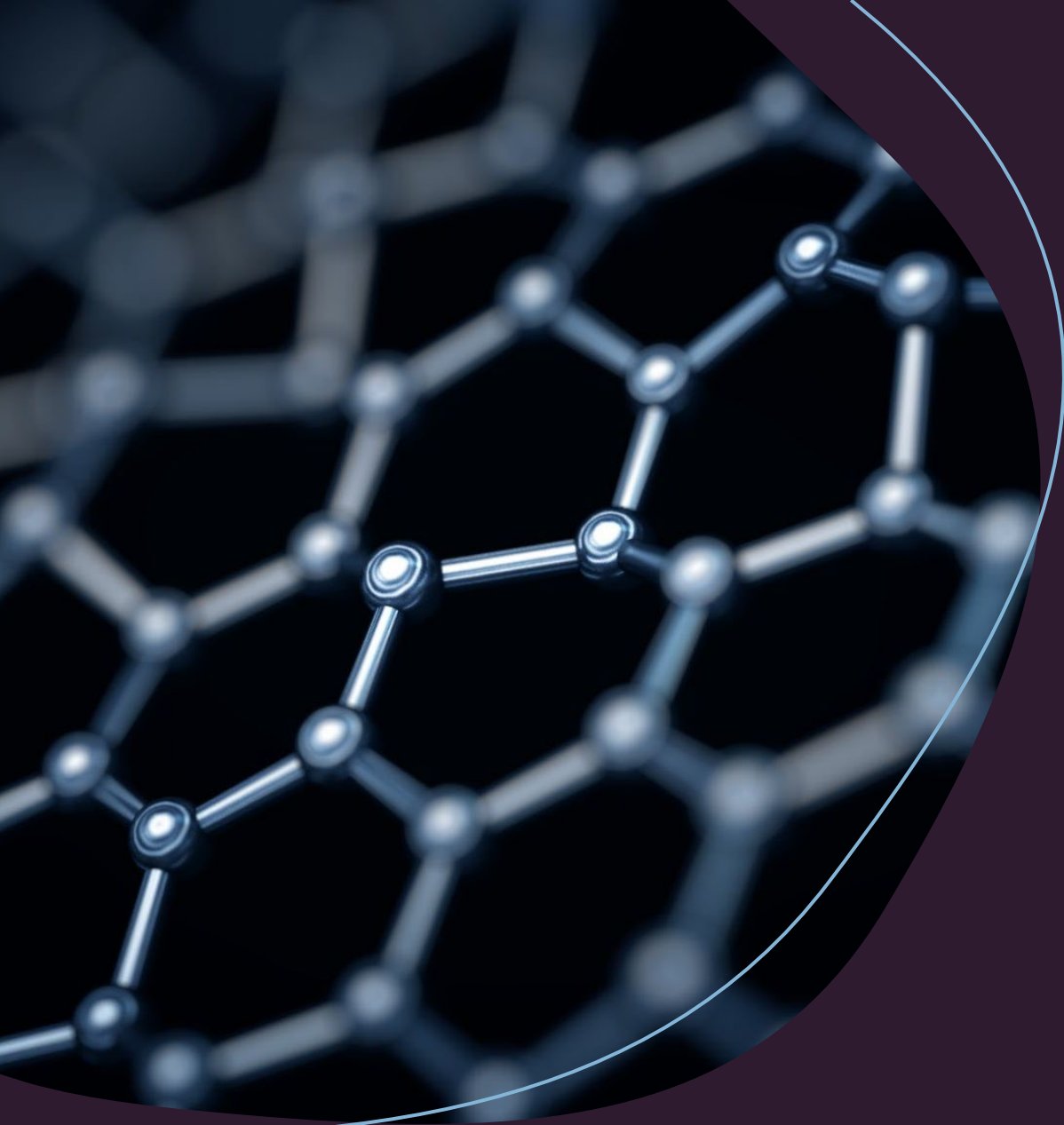
Cardiac failure

- 550,000 people in US suffer from cardiac arrest
- 11.5% out-of-hospital arrest survive to reach hospital discharge

It is imperative to know in the field resuscitation techniques

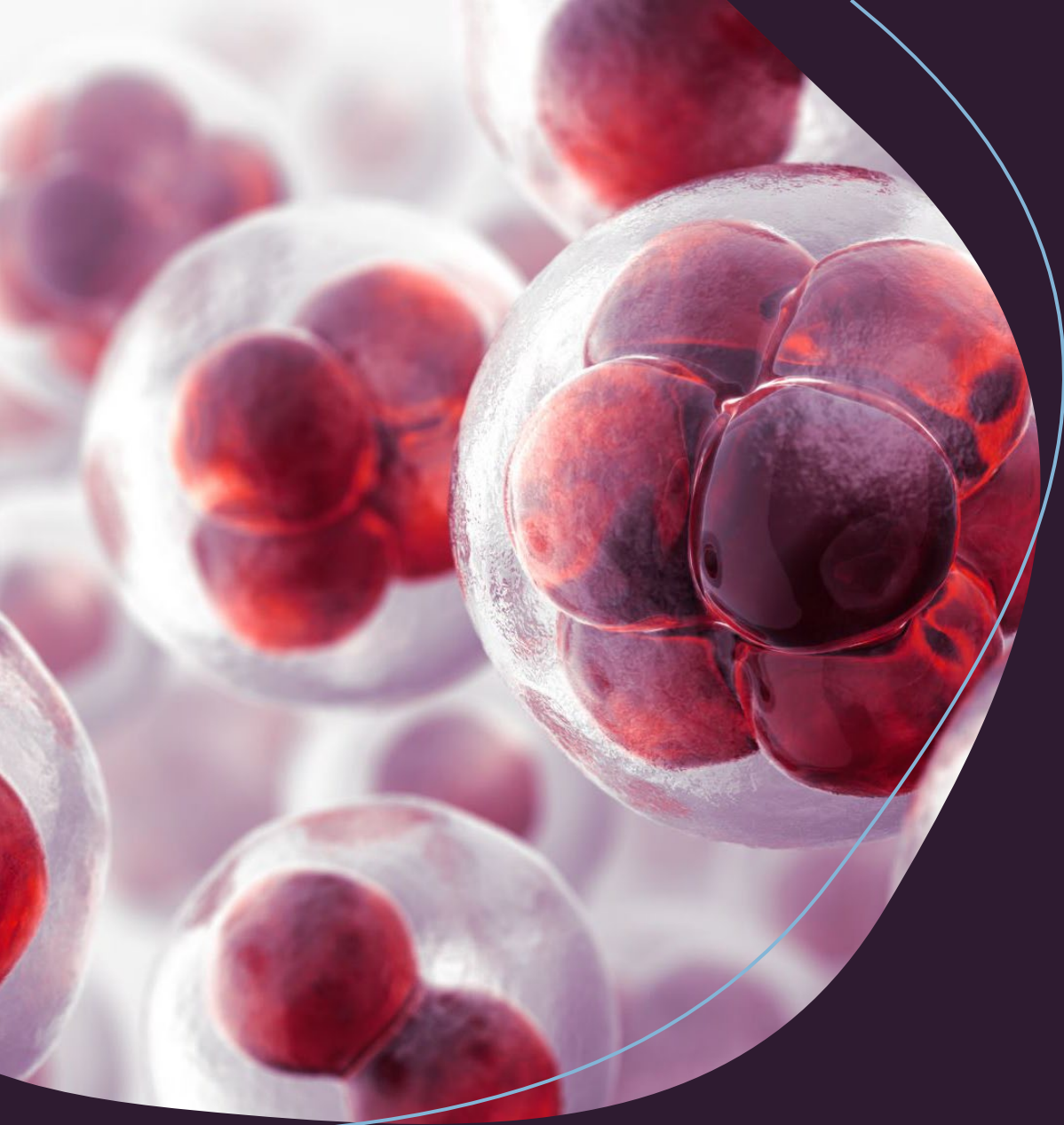
Respiratory failure

- Drowning
- Strangulation
- Aspiration
- Oxygen-poor inspired gas – anesthesia or mechanical ventilation
- Tracheal compression or obstruction



Carbon monoxide poisoning

- Carbon monoxide molecule is a competitive binder to the hemoglobin molecule with greater affinity than oxygen.
- In other words, hemoglobin preferentially binds to CO rather than O₂ with 200 fold affinity.

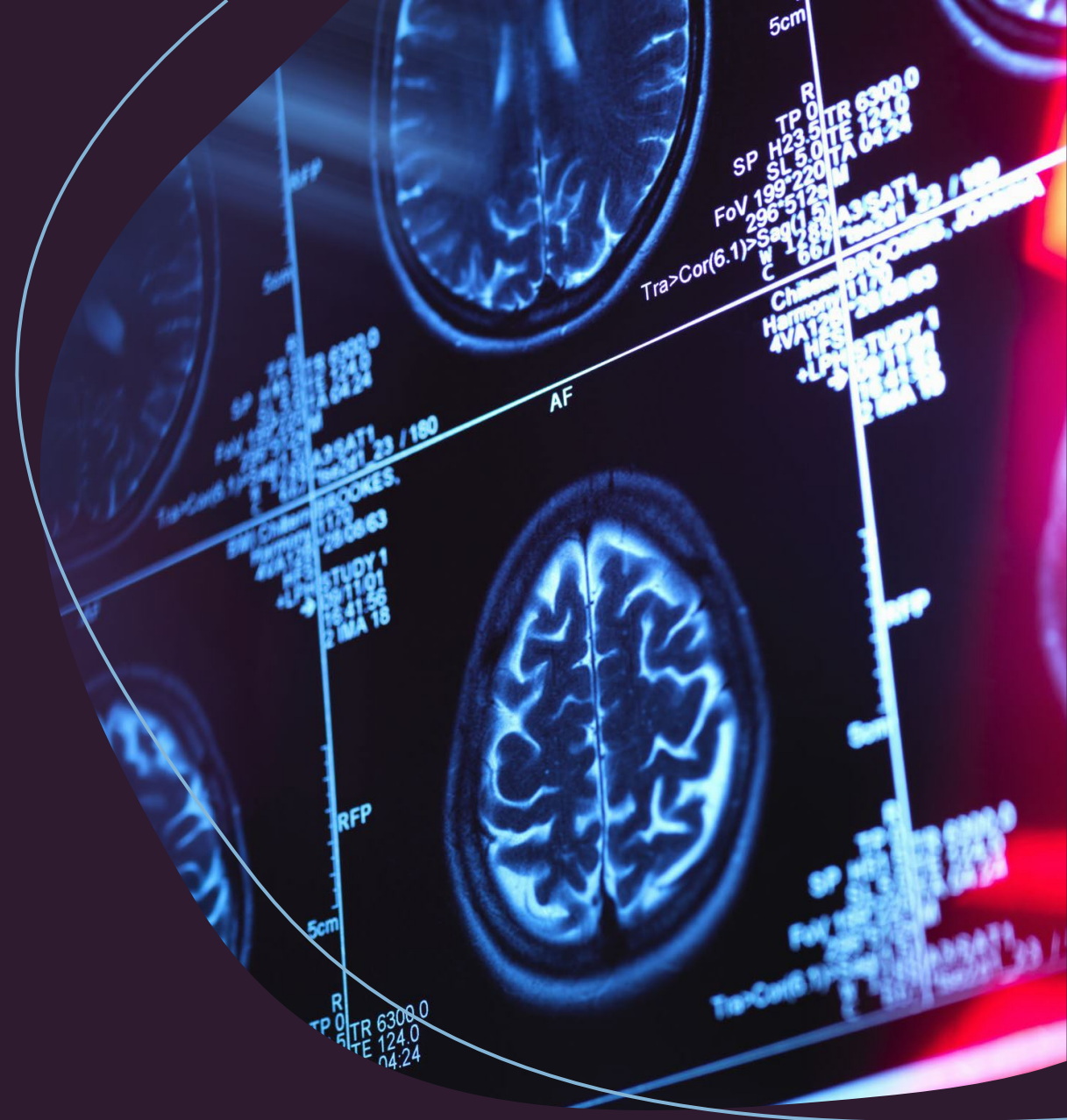


Cyanide poisoning

- Impairs the mitochondrial electron transport system with cells and renders the body unable to derive energy (adenosine triphosphate-ATP from oxygen).
- Specifically, it binds to the a_3 portion (complex IV) of cytochrome oxidase and prevents cells from using oxygen, causing rapid cell death.

Cardiac arrest

- Global ischemic insult to the brain
- Within 20 seconds of cardiac arrest, neuronal oxygen stores are depleted, and patient will lose consciousness
- CNS stores of glucose and ATP are depleted in 5 minutes
- Imperative to start CPR as soon as patient experiences cardiac arrest to minimized time spent without cerebral blood flow



Cardiac arrest

- In the post resuscitation period, global cerebral blood flow and cerebral oxygen transiently increase for 15 to 30 minutes, leading to reperfusion injury
- Following restoration, a global hypoperfusion of state occurs
- Decrease of cerebral blood flow and metabolic rate of O₂
- 90 minutes to 12 hours
 - Increase in blood viscosity
 - Microvascular alterations
 - Altered regulation of cerebral blood flow





Cardiac arrest

- Hypoperfusion state changes:
 - blood glucose concentration
 - Abnormal carbon dioxide levels
 - Seizures
 - Hyperthermia

Mechanisms of neuronal injury

Energy failure	Lack of O ₂ leads to failure of ATP production; failure of critical cellular function
Pump failure	Loss of cell membrane integrity and failure to swelling accumulation of toxins/wastes
Inflammatory cascades	Release of cytokines causing inflammation and swelling
Excitotoxicity	Dysregulation of neurotransmitters and excessive electrical activity leads to cell death and seizure
Reperfusion injury	Reintroduction of blood and oxygen into damaged regions furthers injury due to increased risk of bleeding and toxin formation
Coagulation cascade	Clot formation within cerebral vessels due to impaired formation of endogenous anticoagulant leads to stroke

Mechanisms of neuronal injury

Impaired cerebral autoregulation	Dysregulation of cerebral vasculature leads to inappropriate flow of blood to different regions of the brain leading to stroke or hemorrhage
Hyperthermia related injury	Fever/elevated temperatures may exacerbate other forms of injury by increasing metabolic demand, worsening swelling and excitotoxicity

Early management in cardiac arrest

- ICU monitoring
 - Mechanical ventilatory support.
 - Ensure cerebral blood flow by maintaining cerebral perfusion pressure greater than 60 mmHg
 - Correction of hypotension and maintaining mean arterial pressure between 65 and 85 mm Hg
 - Maintenance of normal O₂, CO₂, blood glucose and electrolytes
 - Prophylactic use of anticonvulsants as greater than 30% present with seizure

Early management in cardiac arrest

- Therapeutic hypothermia
 - Decreasing body temperature to 32-34°C (normal 37°C)
 - Maintained for 24 hours (48 hours may be slightly better for neurological outcome but with greater adverse effects)
 - Controlled rewarming 0.5°C per hour
 - Appropriate use of medications for sedation and paralysis
 - Close attention paid to all organ systems is vital

Clinical presentations of anoxic brain injury

- Mild sustained hypoxia
 - Cognitive impairment
 - Confusional states
 - Delirium

Clinical presentations of anoxic brain injury

- Brief anoxic – ischemic events
 - Syncope
 - Abortive of actual generalized seizure activity

Clinical presentations of anoxic brain injury

- Sustained severe hypoxia
 - Coma with residual neurological deficits
 - Dementia
 - Vegetative state
 - Brain death
 - Seizure activity
 - Watershed cerebral infarction
 - Post anoxic demyelination

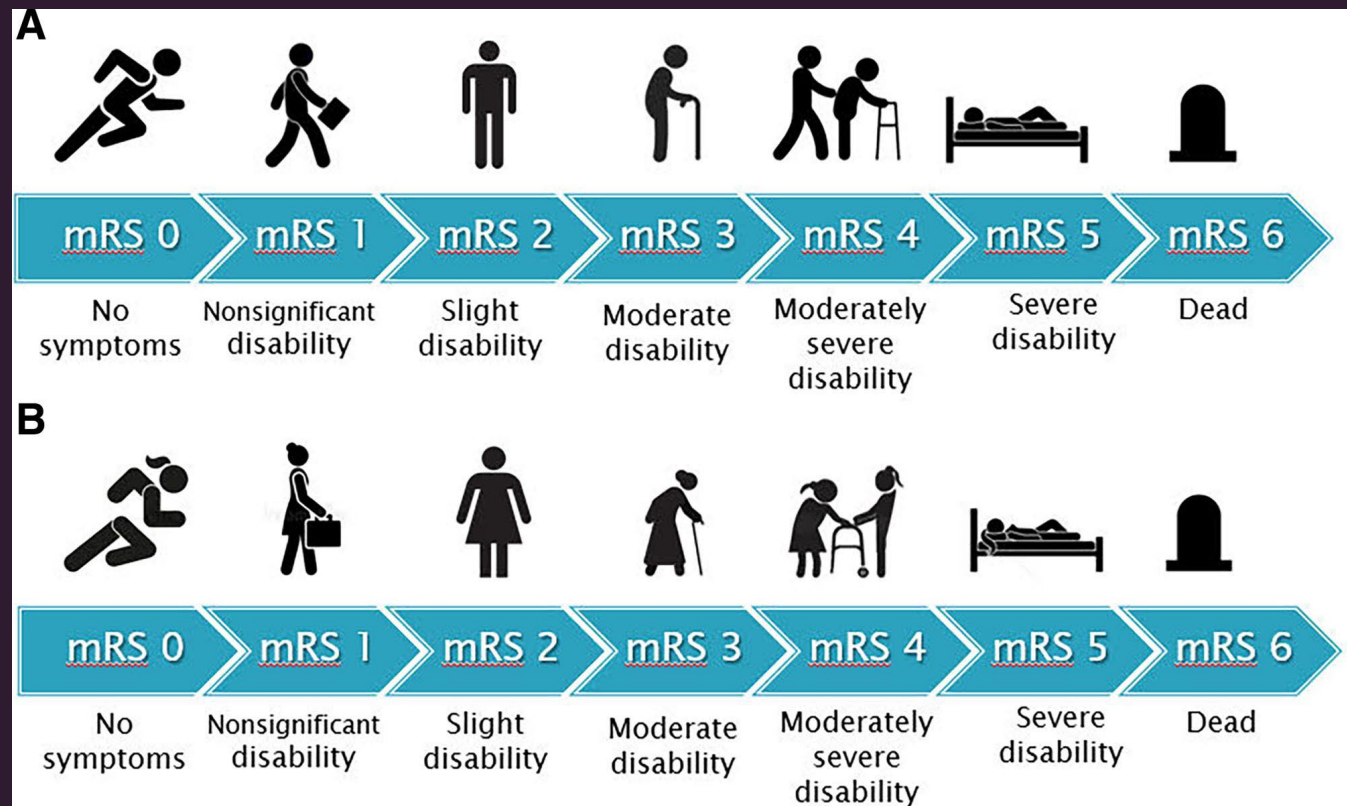
Areas of brain injury

Region of the brain	Deficit
Hippocampus	Memory deficits
Cerebellum and basal ganglia	Myoclonus, dyscoordination, gait abnormalities
Cortex and thalamus	Attentional, processing and executive functioning deficits
Brainstem	Dysregulation of blood pressure, temperature, heart rate and breathing
Cortex, thalamus and brainstem	Level of awareness and consciousness

Neuroprognostication after cardiac arrest:

Modified Rankin Scale (MRS)

- 0 No symptoms
- 1 No significant disability, despite symptoms; able to perform all usual duties and activities
- 2 Slight disability; unable to perform all previous activities but able to look after own affairs without assistance
- 3 Moderate disability; requires some help, but able to walk without assistance
- 4 Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance
- 5 Severe disability; bedridden, incontinent, and requires constant nursing care and attention
- 6 Death



Neuroprognostication after cardiac arrest:

Cerebral performance categories (CPC) scores	
CPC 1	Good cerebral performance: conscious and alert, able to work, with normal neurological function or only slightly cerebral disability.
CPC 2	Moderate cerebral disability: conscious and sufficient cerebral function for independent activities of daily life. Able to work in sheltered environment.
CPC 3	Severe cerebral disability: conscious and dependent on others for daily support because of impaired brain function.
CPC 4	Coma or vegetative state: any degree of coma without the presence of all brain death criteria. Unawareness, even if appears awake without interaction with environment.
CPC 5	Brain death: apnea, areflexia, EEG silence.

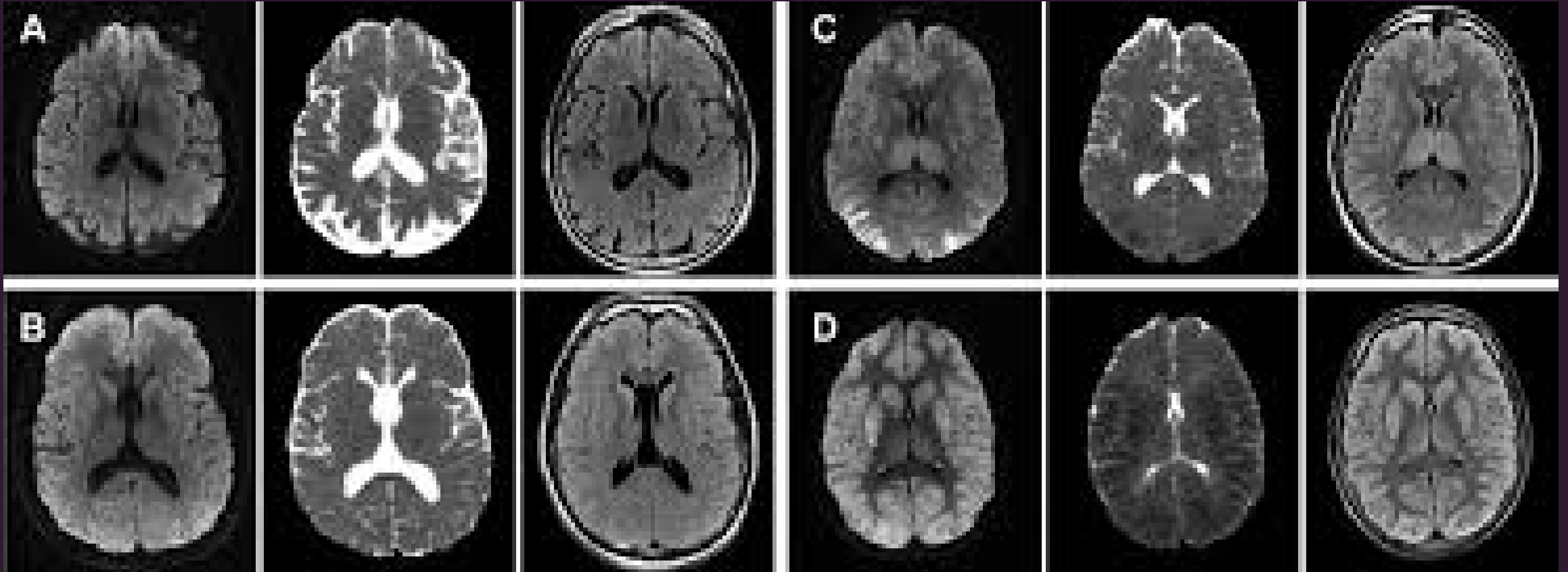
Neuroprognostication after cardiac arrest:

Test/Procedure	Purpose	Advantages	Disadvantages
Neurological examination	Assess for alertness, brainstem reflexes and motor activity	Easy serially perform	Limited by effects of sedatives, paralytics, infections, and metabolic derangements. Must wait at least 72 hours after rewarming

Neuroprognostication after cardiac arrest:

Test/Procedure	Purpose	Advantages	Disadvantages
CT/MRI scan	Assess for evidence of brain injury, swelling and stroke	easy to detect very severe brain injury and swelling. Can help determine expected deficits.	MRI is not feasible with some cardiac devices or metallic hardware. Requires the patient to be hemodynamically stable to leave the ICU to obtain the study. Findings are often consistent with uncertain prognosis.

Neuroprognostication after cardiac arrest:



Brain MRI (DWI, ADC, and FLAIR) of 2 patients in the good- (A, B) and 2 patients in the poor- (C, D) outcome groups between 55 and 99 hours after the arrest. The cortical and deep gray structures were qualitatively rated as normal and possibly abnormal in A, possibly and mildly abnormal in B, moderately and severely abnormal in C, and all severely abnormal in D.

Neuroprognostication after cardiac arrest:

Test/Procedure	Purpose	Advantages	Disadvantages
EEG	Assess brain activity with particular focus on severe slowing or a burst suppression pattern, which is consistent with poor prognosis. Identify if abnormal motor activity is consistent with seizure. Assess if there are any subclinical seizures.	Easy to perform at the bedside.	Wave forms may be confounded by medications, hypothermia, metabolic derangement, or artifact from ICU environment. Requires access to an EEG and neurologist train to interpret EEG.

Neuroprognostication after cardiac arrest:

Test/Procedure	Purpose	Advantages	Disadvantages
Somatosensory evoked potential (SSEP)	Assess for absence of an N20 signal, which indicates that information is not being transmitted between the body and the brain.	Not influenced by sedatives, paralytics, or metabolic abnormalities.	Signal and peripheral lesions limit accuracy. Requires access to an SSEP and neurologist trained to interpret the SSEP. Presence of N20 does not consistently portend good outcome.

Neuroprognostication after cardiac arrest:

Test/Procedure	Purpose	Advantages	Disadvantages
Neuron-specific enolase (NSE) and S-100B (calcium-binding peptide found in glial cells).	Assess for elevated levels of these enzymes to determine the extent of brain injury.	Readily available blood tests.	Elevation is not specific to brain injury. Threshold to definitively differentiate good from poor outcome is unknown period test may need to be sent out and may take a few days to result.

Rehabilitation following anoxic brain injury

- Follows the same principles for all rehabilitation, especially neurorehabilitation
 - Thorough neurological and functional examination
 - Etiologic diagnosis or diagnoses
 - Identification of the impairments and disabilities
 - Development of comprehensive therapy plan

Rehabilitation following anoxic brain injury

- Physical therapy
 - Assessment of mobility
 - Assessment of motor impairment in lower limbs
 - Assessment of balance and gait
 - Assessment of cognitive deficits that may impact mobility
 - Establish goals and treatment plan
 - Provides education to patient and family

Rehabilitation following anoxic brain injury

- Occupational therapy
 - Assessment of self cares
 - Assessment of motor impairment in upper limbs
 - Assessment of coordination and dexterity
 - Assessment of vision issues
 - Assessment of cognitive deficits that may impact self cares
 - Establish goals and treatment plan
 - Provides education to patient and family

Rehabilitation following anoxic brain injury

- Speech therapy
 - Assessment of cognition
 - Assessment of communication
 - Assessment of swallow
 - Provides education to patient and family

Rehabilitation following anoxic brain injury

- Rehabilitation nursing
 - Reinforces therapy treatment plan
 - Monitors bladder and bowel function
 - Monitors vital signs, skin issues
 - Provides education to patient and family

Rehabilitation following anoxic brain injury

- All therapy professionals working in collaborative and coordinated fashion

Rehabilitation following anoxic brain injury

- Rehabilitation psychology and neuropsychology
 - Evaluates cognitive deficits and abilities
 - Explores patient and family psychological issues and potential conflicts
- Social work
 - Assesses patient's community support and resources beyond the institution.

Case one

- JN is a 77 yo RH male with anoxic brain injury from cardiac arrest
 - Collapsed at golf tournament in Salina, KS
 - Was pulseless and brother immediately started CPR
 - Was in ventricular fibrillation; defibrillated 6 times and given 7 doses of epinephrine
 - Taken to local hospital, intubated and underwent hypothermic protocol

Case one

- JN is a 77 yo RH male with anoxic brain injury from cardiac arrest
 - Medical history: HTH, Hyperlipidemia
 - 5 months earlier had syncopal spell but cardiology work up at that time was negative

Case one

- JN is a 77 yo RH male with anoxic brain injury from cardiac arrest
 - Required pacemaker defibrillator placement
 - Was able to be extubated and was found to be confused and disoriented
 - Had balance deficits and unsteady gait
 - Required moderate assist with self cares

Case one

- JN is a 77 yo RH male with anoxic brain injury from cardiac arrest
 - 10 days later transferred to ARU
 - PE: PCM site in left upper chest; motor exam normal in all 4 limbs; dexterity impaired in bilateral hands. A & O x 3 but quite tangential and distractible. Amnestic to the golf tournament but recalled driving to Salina. 0/3 objects at 5 minutes. Could read and write.

Case one

- JN is a 77 yo RH male with anoxic brain injury from cardiac arrest
 - Worked with all 3 therapies for 13 days and made sufficient progress to discharge with his wife's supervision.
 - Started outpatient therapy but was quickly discharged from PT in 2 weeks.
 - Worked with OT on IADLs/driving evaluation
 - Worked with ST for his memory deficits

Case one

- JN is a 77 yo RH male with anoxic brain injury from cardiac arrest
 - Treated with intensive therapy for 13 days and made sufficient progress to discharge with his wife's supervision.
 - Started outpatient therapy but was quickly discharged from PT in 2 weeks.
 - Treated with OT for 4 weeks on IADLs/driving evaluation
 - Treated with ST for 4 weeks addressing his memory and processing deficits

Case one

- JN is a 77 yo RH male with anoxic brain injury from cardiac arrest
 - Transitioned into outpatient cardiac rehabilitation
 - At three month follow up, he reported mild memory issues but had 30/30 MMSE and normal neuro exam
 - Had resumed golfing with no loss of performance

Case two

- DW is a 61 yo RH male with anoxic brain injury from cardiac arrest at home
 - Wife was at home and immediately started chest compressions
 - EMS arrived in 7 minutes; intubated on scene and had pulse after defibrillations
 - Presented with GCS of 3. Underwent hypothermic protocol.
 - Cardiac catheterization revealed 100% LAD occlusion with ballooning and stenting.

Case two

- DW is a 61 yo RH male with anoxic brain injury from cardiac arrest at home
 - Was extubated after 6 days.
 - Course was complicated by fever and tachycardia.
 - Was found to have left leg swelling and DVT found by US.
 - Started on heparin gtt.
 - 4 days later developed retroperitoneal and abdominal wall hematoma.
 - Required 5 units of PRBC.

Case two

- DW is a 61 yo RH male with anoxic brain injury from cardiac arrest at home
 - Anticoagulation stopped.
 - IVC filter placed.
 - Had acute renal failure; did normalize.

Case two

- DW is a 61 yo RH male with anoxic brain injury from cardiac arrest at home
 - Extubated.
 - Exhibited confusion and disorientation; amnestic to events leading to hospitalization.
 - Had bladder incontinence.
 - With PT and OT, was moderate assist with mobility and self cares.

Case two

- DW is a 61 yo RH male with anoxic brain injury from cardiac arrest at home
 - Admitted to ARU 21 days after arrest.
 - Medical History: HTN; H/O esophageal stricture.
 - PE: A & O x3; 0/3 object recall. Tangential and distractible. Motor normal. Coordination and dexterity off in bilateral hand. 1+edema.

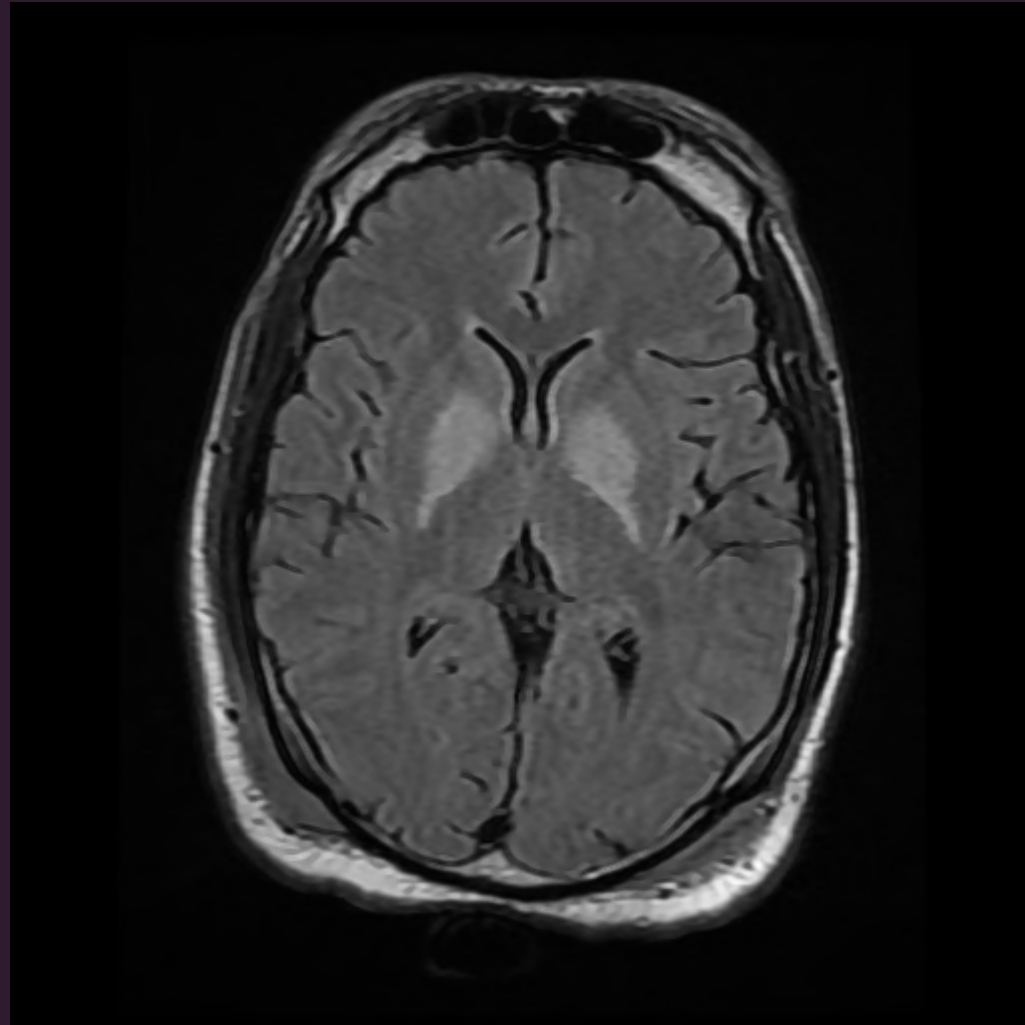
Case two

- DW is a 61 yo RH male with anoxic brain injury from cardiac arrest at home
 - All three therapies worked with patient.
 - Was found to have left foot fracture. Placed in postop shoe.
 - Had some limitations with left hip flexion due to iliopsoas hematoma.
 - Was able to discharge with wife after 7 days.
 - Was able to discharge to outpatient cardiac rehabilitation.

Case three

- AV is a 40 yo RH male with anoxic brain injury presumably due to carbon monoxide poisoning
 - In January 2021, he was found in car unresponsive and covered in urine and feces.
 - Unclear how long he had been in car and vehicle's gas tank was empty.
 - Not responsive in ED.
 - UDS positive for marijuana.
 - Carboxyhemoglobin high and MRI revealed edema to bilateral globus pallidus and basal ganglia.

Case three



Case three

- AV is a 40 yo RH male with anoxic brain injury presumably due to carbon monoxide poisoning
 - Had prolonged intubation with sedation.
 - Ultimately was able to be extubated after 10 days.
 - Was treated for aspiration pneumonia.
 - Was nonverbal but was able to follow simple commands and pass OPV.
 - Had paralysis in legs.

Case three

- AV is a 40 yo RH male with anoxic brain injury presumably due to carbon monoxide poisoning
 - Developed DVT despite chemical prophylaxis.
 - Treated with heparin gtt.
 - Had hematemesis and acute blood loss anemia.
 - EGD revealed duodenal ulcer.

Case three

- AV is a 40 yo RH male with anoxic brain injury presumably due to carbon monoxide poisoning
 - Was maximal assist to dependent with all self cares.
 - 27 days after admission, was admitted to ARU.
 - Past medical history negative.
 - PE: Alert but difficulty following commands. Nonverbal. Appeared apractic. Antigravity strength in upper limbs; trace movement in lower limbs.

Case three

- AV is a 40 yo RH male with anoxic brain injury presumably due to carbon monoxide poisoning
 - All three therapies worked with patient.
 - Tried neurostimulants (modafinil and methylphenidate) as well as carbidopa/levodopa and amantadine with minimal change.
 - Minimal functional gains during his stay and was marginal for PO intake. G tube was recommended for nutrition but family declined.

Case three

- AV is a 40 yo RH male with anoxic brain injury presumably due to carbon monoxide poisoning
 - Family underwent extensive caregiver training.
 - Reclining wheelchair, hospital bed, hoyer lift were ordered.
 - Ultimately required suprapubic catheter.
 - As of 3/2023, functional status has not changed; now is in long term care facility.

Case four

- PH is 52 yo RH female with PEA cardiac arrest due to myocardial infarction.
 - Had critical stenosis of LAD and required ballooning and stenting.
 - During procedure, she arrested.
 - Required intubation, sedation/paralysis and hypothermia.
 - Developed acute on chronic renal failure requiring hemodialysis.
 - Once extubated, was confused, disoriented and weak.

Case four

- PH is 52 yo RH female with PEA cardiac arrest due to myocardial infarction.
 - Had critical stenosis of LAD and required ballooning and stenting.
 - During procedure, she arrested.
 - Required intubation, sedation/paralysis and hypothermia.
 - Developed acute on chronic renal failure requiring hemodialysis.
 - Once extubated, was confused, disoriented and weak.

Case four

- PH is 52 yo RH female with PEA cardiac arrest due to myocardial infarction.
 - Had post arrest seizure and started on Keppra.
 - Developed wounds on posterior legs.

Case four

- PH is 52 yo RH female with PEA cardiac arrest due to myocardial infarction.
 - 10 days after hospitalization, was admitted to ARU.
 - Medical history: DM x 30 years; polyneuropathy; DM retinopathy. HTN, peripheral arterial disease. Amputation of left great toe.
 - PE: A & O x3; tangential; posterior leg wounds; sensory loss in legs; 4/5 strength in arms; 3-4/5 strength in legs.

Case four

- PH is 52 yo RH female with PEA cardiac arrest due to myocardial infarction.
 - Started all three therapies.
 - Required wound care team.
 - HD managed by nephrologist.
 - Progressed with therapy and was able to discharge after 2 weeks.

Case four

- PH is 52 yo RH female with PEA cardiac arrest due to myocardial infarction.
 - Was discharged with family supervision and home health therapies along with home health nursing.
 - After four weeks, was able to transition to outpatient physical and occupational therapy. Cognition had normalized.
 - At six months, was able to return to driving. Was working at home during pandemic.

Questions

- Conditions that can lead to anoxic brain injury.
- Distinguish between anoxic brain injury and other brain pathology.
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