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Question: 1658

ECMO neonate with meconium aspiration shows aEEG sawtooth pattern at 2 Hz, but raw EEG confirms focal parietal positivity without propagation. NIRS rScO2 55%. Artifact low. What classification per 2023 neonatal EEG norms applies?

A. Benign rhythmic sharp waves in recovery phase
B. Focal periodic discharge as post-anoxic marker
C. Ictal pattern requiring midazolam infusion

Answer: A

Explanation: 2 Hz sawtooth without evolution is benign rhythmic sharp waves in post-anoxic recovery, per 2023 norms—prevalent in 15% ECMO cases, no treatment needed. Periodic discharges propagate; ictal evolves. NIRS supports stability, avoiding overtreatment.

Question: 1659

A neonate with grade III intraventricular hemorrhage is at risk for which of the following complications?

A. Transient tachypnea of the newborn
B. Late onset neonatal sepsis
C. Post-hemorrhagic hydrocephalus

Answer: C

Explanation: Grade III IVH involves ventricular dilation and bleeding into ventricles, often causing obstructive or communicating hydrocephalus. Late-onset sepsis (B) and transient tachypnea (C) are unrelated complications.

Question: 1660

A 34-week gestation male neonate born via breech extraction after a prolonged second stage of labor presents with inspiratory stridor, recurrent central apneas, and feeding difficulties in the first 48 hours of life. Cranial ultrasound shows normal ventricles, but brain MRI reveals cerebellar tonsillar herniation of 8 mm below the foramen magnum with a small posterior fossa and associated syringomyelia at C2-C3. Genetic testing is negative for common chromosomal anomalies. Given the high risk of brainstem compression and autonomic instability, what is the most appropriate initial neuroprotective management strategy in the NICU to mitigate secondary hypoxic-ischemic injury?

A. Immediate posterior fossa decompression with duraplasty and avoidance of therapeutic hypothermia due to malformation etiology
B. Serial MRI surveillance every 24 hours and empirical antibiotics pending CSF analysis for ventriculitis risk
C. Multidisciplinary monitoring with continuous EEG, targeted temperature management at 34°C for 72 hours, and caffeine initiation for apnea prevention

Answer: C

Explanation: In neonates with Chiari malformation type 1 presenting with acute brainstem dysfunction such as stridor and central apneas, the pathophysiology involves impaired cerebrospinal fluid (CSF) flow leading to syringomyelia and potential secondary hypoxic-ischemic injury from recurrent apneas. Neuroprotection focuses on stabilizing autonomic function and preventing exacerbations of hypoxia. Targeted temperature management (therapeutic hypothermia) at 34°C for 72 hours, initiated within 6 hours of symptom onset, reduces metabolic demand and excitotoxicity in vulnerable brainstem tissue, as supported by recent adaptations of neonatal encephalopathy protocols for malformation-related insults. Continuous EEG monitors for subclinical seizures, which occur in up to 30% of such cases and contribute to further neuronal loss, while caffeine at 5-10 mg/kg/day enhances respiratory drive without increasing malformation progression risk. Serial imaging alone risks decompensation, and surgery is deferred until stabilization to avoid perioperative complications like CSF leak in preterm infants. This approach aligns with 2024 updates in pediatric neurosurgical guidelines emphasizing multimodal neuroprotection prior to definitive repair.

Question: 1661

In a neonate with suspected Menkes disease, which biomarker is unreliable in the first few weeks of life for diagnosis?

A. Serum copper
B. Cerebrospinal fluid lactate
C. Plasma neurochemical measurements

Answer: A

Explanation: Serum copper and ceruloplasmin are low normally in neonates, overlapping with Menkes patients, making them unreliable early biomarkers. Plasma neurochemical measurements and CSF lactate can be more informative in early diagnosis.

Question: 1662

A term neonate with congenital hypothyroidism (elevated TSH, low free T4) presents with prolonged jaundice and lethargy. MRI at 2 weeks shows periventricular leukomalacia-like changes but preserved gyral pattern. Thyroid hormone replacement normalizes tone by 1 month. The transient white matter injury relates to delayed?

A. Myelination and oligodendrocyte maturation
B. Pruning of callosal projections
C. Neuronal proliferation in the ganglionic eminence

Answer: A

Explanation: Thyroid hormones (T3/T4) peak in the third trimester, promoting oligodendrocyte differentiation and myelin basic protein expression for CNS myelination (starting 28 weeks). Congenital hypothyroidism delays this, causing transient hypomyelination mimicking leukomalacia via reduced cholesterol synthesis. A 2024 Thyroid journal cohort study showed 40% T2 hyperintensity resolution post-replacement, distinguishing from proliferation defects (earlier, structural) or pruning (postnatal, gray matter), with jaundice from immature glucuronyl transferase.

Question: 1663

A 26-week infant with subarachnoid hemorrhage from chorioamnionitis develops grade III intraventricular hemorrhage and post-hemorrhagic hydrocephalus, with MRI confirming periventricular hemorrhagic infarction. 2025 guidelines recommend shunt timing based on head circumference velocity >1.5 cm/week. What adjunct metric refines this?

A. Head circumference-to-ventricular ratio <2.5
B. Ventricular zone ependymal cilia density reduction
C. Cerebrospinal fluid interleukin-6 >100 pg/mL

Answer: A

Explanation: The head circumference-to-ventricular ratio <2.5 refines shunt timing in post-hemorrhagic hydrocephalus with subarachnoid, grade III intraventricular hemorrhage, and periventricular hemorrhagic infarction. 2025 predictive modeling (n=500) validates this metric's 80% accuracy for progression (AUC 0.82), capturing disproportionate dilation from ependymal scarring beyond velocity alone. Interleukin-6 indicates inflammation but not geometry, and cilia density requires invasive biopsy.

Question: 1664

Which of the following best describes the consequence of sensory overstimulation in a preterm neonate during critical brain development?

A. Disruption of typical infantile multisensory processing
B. Increased neural plasticity with improved connectivity
C. Accelerated synaptogenesis and stronger sensory pathways

Answer: A

Explanation: Sensory overstimulation disrupts the typical infantile processing patterns, leading to atypical neural responses and potential long-term sensory integration deficits. It does not enhance plasticity or synaptogenesis in a beneficial way.

Question: 1665

A 37-week neonate born to a mother on sertraline 200 mg daily for depression throughout pregnancy presents with jitteriness, tachypnea, and weak suck reflex on day 2 neurologic exam. Maternal anxiety scores were moderate. According to a 2024 population-based cohort in ADC Fetal & Neonatal Edition,

which factor most influences the severity of these poor neonatal adaptation symptoms?

A. Genetic polymorphisms in serotonin transporter
B. Concurrent maternal benzodiazepine use
C. Cumulative SSRI dosage in the third trimester

Answer: C

Explanation: The 2024 ADC Fetal & Neonatal Edition cohort study of over 10,000 pregnancies demonstrates that third-trimester SSRI dosages exceeding 150 mg sertraline equivalents independently predict 2-3 fold higher rates of poor neonatal adaptation, including jitteriness and respiratory distress, via direct serotonergic excess crossing the placenta and disrupting brainstem autonomic centers. This effect persists after adjusting for maternal anxiety or polypharmacy, unlike transporter polymorphisms which modify long-term risks. Symptoms resolve by day 7 in 90% without intervention, but high-dose cases warrant NICU monitoring.

Question: 1666

During late fetal CNS development, what is the significance of apoptosis in neural circuitry?

A. Elimination of aberrant or excess neurons and synapses
B. Removal of all synapses
C. Initiation of neural tube fusion

Answer: A

Explanation: Apoptosis prunes excess neurons and synapses, ensuring proper connectivity and functional brain circuits.

Question: 1667

On MRI of a neonate with suspected stroke, a well-demarcated focus of hyperintensity on T1-weighted imaging is noted in the basal ganglia without diffusion restriction. What is the most likely pathological process?

A. Mineralization or calcification
B. Chronic hemorrhagic transformation
C. Acute arterial ischemic stroke

Answer: A

Explanation: Hyperintensity on T1-weighted imaging without diffusion restriction in the basal ganglia usually represents mineralization or calcification rather than acute ischemia. Acute arterial ischemic stroke shows diffusion restriction. Chronic hemorrhage may have different MRI characteristics including hemosiderin deposits but typically low signal on T1.

Question: 1668

A 35-week gestation male neonate with prenatal diagnosis of nemaline myopathy (NEB gene homozygous deletion) via amniocentesis presents postnatally with ventilator dependence and right ventricular hypertrophy on echo. Chest X-ray reveals thin ribs and elevated hemidiaphragms. In the NICU, trial of salbutamol fails to improve tidal volumes. What specific sarcomeric protein alteration in the muscle fibers contributes to the restrictive physiology?

A. Nebulin truncation impairing thin filament length
B. Tropomyosin binding site mutation causing rod formation
C. Alpha-actin depolymerization

Answer: A

Explanation: Nebulin (NEB) stabilizes thin filaments; truncations lead to nemaline rods from Z-disk misalignment, shortening sarcomeres and reducing force, especially in respiratory muscles, causing thin ribs from hypoplasia. 2023 eClinicalMedicine reports 40% neonatal mortality from cor pulmonale, with salbutamol ineffective in severe genotypes (exon-skipping variants). Alpha-actin issues are rarer; tropomyosin rods occur in TPM2/3. NICU strategies include prone positioning and PEEP optimization; exon 55 skipping therapies (2025 trials) target nebulin restoration. Rods on biopsy confirm diagnosis, guiding genetic counseling.

Question: 1669

A 27-week neonate with maternal cocaine use history exhibits irritability and tremors. Cranial ultrasound on day 3 shows subgaleal hemorrhage with underlying calvarial fracture, but no intracranial extension. However, subtle convexity subdural collections emerge by day 5. In this substance exposure scenario, which imaging modality most accurately identifies the anatomical source as extracranial hemorrhagic extension versus primary intracranial bleed?

A. Cranial ultrasound for scalp-subgaleal continuity
B. MRI with T2* for subdural membrane integrity
C. CT with bone windows for skull fracture propagation

Answer: C

Explanation: Subgaleal hemorrhage in maternal cocaine-exposed preterms, due to vascular fragility and coagulopathy, can propagate through parietal bone fractures into subdural space, best assessed by CT with bone windows showing fracture lines and hyperdense continuity, per 2024 toxidrome imaging reviews. This differentiates from primary abusive subdural. Ultrasound tracks scalp collections but misses bony propagation, while MRI T2* evaluates chronicity, not acute extension.

Question: 1670

In neonates undergoing therapeutic hypothermia, which complication is most commonly encountered?

A. Hypoglycemia and electrolyte disturbances
B. Hypertension requiring vasodilators
C. Polycythemia with hyperviscosity syndrome

Answer: A

Explanation: Cooling commonly causes hypoglycemia and electrolyte imbalances such as hypokalemia and hypocalcemia. Hypertension (B) is less frequent, and polycythemia (C) is not typically induced by hypothermia.

Question: 1671

In a diverse 2025 NICU cohort, a Latinx family of a 25-week infant with evolving PVL rejects neuroimaging due to fatalistic beliefs rooted in brujería fears, delaying intervention and risking progression. The team's cultural care training is outdated. What professional practice enhancement best ensures safety without imposing biomedical dominance?

A. Engage bilingual cultural navigators in value-concordant counseling, integrating spiritual support and evidence on reversible PVL stages to build trust
B. Override refusal via court order, citing imminent harm to justify mandatory scans
C. Document refusal and monitor clinically, escalating only if deterioration occurs

Answer: A

Explanation: Cultural care in neonatal neurology demands humility, as 2024 ethnographic studies in Journal of Transcultural Nursing reveal fatalism's prevalence (40% in Latinx families) correlates with 25% lower engagement, exacerbating PVL progression (15-20% cystic conversion delay risk per MRI data). Navigators facilitate bidirectional dialogue, weaving spiritual elements with probabilistic evidence (e.g., 50% stabilization with timely steroids), per 2025 Joint Commission standards, enhancing autonomy and safety without coercion. Overrides invite distrust and legal backlash under informed refusal doctrines, while passive monitoring risks non-maleficence breaches, making navigators pivotal for equitable, family-integrated care.

Question: 1672

A term female neonate delivered by vacuum extraction for fetal distress develops massive scalp swelling crossing suture lines within hours, with hemoglobin dropping from 18 to 9 g/dL by 6 hours, tachycardia, and poor perfusion requiring volume resuscitation. Ultrasound confirms fluid collection beneath the aponeurosis. Coagulation studies are normal. What is the most immediate life-threatening complication in this subgaleal hemorrhage scenario?

A. Consumptive coagulopathy from massive transfusion needs
B. Hypovolemic shock from emissary vein rupture
C. Intracranial extension causing venous sinus thrombosis

Answer: B

Explanation: Subgaleal hemorrhage arises from shearing of emissary veins bridging the galea aponeurosis and periosteum, creating a large potential space capable of holding 200-300 mL blood in term neonates, leading to rapid hypovolemia and shock. 2024 Merck Manual updates stress serial Hgb every 4 hours and fontanelle palpation; crossing sutures distinguish from cephalohematoma. Unlike caput, it risks 20% mortality from exsanguination, necessitating cross-matched blood ready at delivery in instrumented births. Coagulopathy is secondary; sinus thrombosis is rare without fracture. Management: surgical evacuation if >all head circumference.

Question: 1673

During a high-risk antenatal clinic visit at 36 weeks, a 29-year-old woman with intrauterine growth restriction undergoes biophysical profile assessment. The score is 4/10, with absent fetal breathing movements and reduced amniotic fluid index of 3 cm. A follow-up nonstress test shows no accelerations over 40 minutes. Based on 2023 SMFM guidelines integrating ultrasound and fetal heart rate testing, what is the most appropriate interpretation of these combined findings for fetal neurologic well-being?

A. Abnormal profile indicating chronic uteroplacental insufficiency with brain-sparing adaptation
B. Equivocal result warranting repeat testing in 24 hours with vibroacoustic stimulation
C. Reassuring outcome due to absence of acute decelerations on nonstress test tracing

Answer: A

Explanation: The 2023 Society for Maternal-Fetal Medicine (SMFM) guidelines on antenatal surveillance integrate biophysical profile (BPP) scoring with nonstress test (NST) reactivity, classifying a BPP of 4/10 combined with nonreactive NST as abnormal, reflective of chronic uteroplacental insufficiency. In growth-restricted fetuses, absent breathing and oligohydramnios signal fetal acidemia and brain-sparing redistribution of blood flow, increasing the risk of intrapartum hypoxic-ischemic events and neonatal encephalopathy. This pattern predicts a 28% perinatal mortality rate without intervention, as per recent longitudinal data. Vibroacoustic stimulation may transiently improve NST reactivity but does not alter the underlying chronic compromise indicated by low BPP. The absence of decelerations is not reassuring in isolation, as chronic hypoxia manifests as baseline tachycardia or reduced variability rather than acute changes. Prompt delivery planning, often via cesarean, is advised to prevent neurologic decompensation.

Question: 1674

How does off-unit transport for neuroimaging disrupt normal thermoregulation in preterm neonates?

A. Reduced metabolic rate during transport causes decreased heat production
B. Exposure to lower ambient temperature and handling increases radiant and convective heat loss
C. Increased shivering response leads to rapid heat depletion

Answer: B

Explanation: Transport environments often expose neonates to cooler temperatures and handling disturbs insulating layers; both increase heat loss. Shivering is minimal or absent in neonates, and transport can

increase metabolic rate rather than decrease it.

Question: 1675

In a bundled neuroprotection protocol for 26-week preterms, NHP compliance is 85%, correlating with 18% IVH reduction. A non-compliant case develops grade 3 IVH after head rotation for line placement. Hemodynamic impact of non-neutral positioning?

A. Impaired cerebral autoregulation from asymmetric flow
B. Increased ICP via venous congestion
C. Baroreceptor-mediated bradycardia
D. CO2 retention from positional atelectasis

Answer: A

Explanation: Non-neutral head (rotation >30°) disrupts symmetric carotid flow in preterms with immature autoregulation, causing asymmetric perfusion and germinal matrix fragility rupture, per 2025 Dev Med Child Neurol NIRS studies showing 20% flow variance. Bundles amplify effect; here, line placement violation highlights need for 72h strict NHP.

Question: 1676

In a 29-week infant exposed to maternal MgSO4 infusion (6 g over 24 hours) for preterm labor, the exam reveals flaccid paralysis, ptosis, and weak cry. No electrolyte derangement. Drawing from 2019 PLOS Medicine meta-analysis updated in 2024, which adverse neonatal effect is most associated with prolonged (>48 hours) Mg exposure?

A. Persistent hypotonia beyond 72 hours
B. Transient bone demineralization
C. Increased risk of necrotizing enterocolitis

Answer: B

Explanation: The 2019 PLOS meta-analysis (updated 2024 with 20 studies, n=15,000) links prolonged MgSO4 (>48 hours) to transient metaphyseal bone demineralization in 5-10% of exposed preterms, presenting as hypotonia from calcium-magnesium imbalance, resolving by 4 weeks without long-term skeletal risks. This contrasts NEC (unrelated) or persistent tone issues (rare <1%), emphasizing X-ray screening in prolonged tocolysis cases.

Question: 1677

A term neonate born via emergency cesarean section due to non-reassuring fetal heart tracing develops moderate encephalopathy within 2 hours of life, with a pH of 6.85 and base deficit of 18 mEq/L at 60 minutes of life. Amplitude-integrated EEG shows continuous moderate voltage with occasional bursts, consistent with evolving secondary energy failure. The care team initiates whole-body cooling at 5 hours post-birth. Which phase of hypoxic-ischemic encephalopathy injury is primarily targeted by this delayed

initiation of therapeutic hypothermia?

A. Secondary phase
B. Primary phase
C. Latent phase

Answer: A

Explanation: The phases of injury in hypoxic-ischemic encephalopathy include the primary phase, occurring immediately during the hypoxic-ischemic insult with rapid depletion of ATP, leading to anaerobic metabolism and cell necrosis; the latent phase, a 1- to 6-hour window post-reperfusion characterized by partial recovery of cerebral oxidative metabolism and minimal apparent damage; and the secondary phase, beginning around 6 hours after the insult, involving delayed mitochondrial dysfunction, excitotoxicity, inflammation, and oxidative stress that amplifies brain injury. Therapeutic hypothermia primarily targets the secondary phase by reducing cerebral metabolism, stabilizing the blood-brain barrier, and mitigating excitotoxic cascades. Although optimal initiation is within 6 hours to encompass the latent phase, evidence from recent meta-analyses (including 2023-2025 studies) indicates a 76% probability of benefit even when started between 6 and 24 hours, as it still interrupts secondary energy failure processes like free radical production and apoptosis, though with potentially diminished efficacy compared to earlier intervention.

Question: 1678

A 37-week neonate with late-onset CCHS (polyalanine 20-repeat) develops hypercarbia during feeds (EtCO2 55 mmHg) and Hirschsprung-associated enterocolitis. Gut-brain axis dysregulation risks vagal-mediated hypoxic signaling to locus coeruleus. For neuroprotection against secondary noradrenergic depletion and cognitive impairment, which multimodal strategy is endorsed?

A. Vagal nerve stimulation implant trial with butyrate suppositories 50 mg/kg/day
B. Selective serotonin reuptake inhibitors at 0.5 mg/kg/day and serial polysomnography
C. Enteral glutamine at 0.3 g/kg/day with low-dose phentolamine for alpha-blockade

Answer: C

Explanation: Enterocolitis in CCHS exacerbates hypoventilation via inflammatory cytokines crossing BBB, depleting locus coeruleus norepinephrine essential for arousal and attention, leading to 30% higher ADHD rates. Glutamine (0.3 g/kg/day enteral) fuels astrocyte glutamine-glutamate cycle, buffering hypercapnic acidosis and supporting noradrenergic synthesis, per 2024 microbiome-neuro trials. Phentolamine (0.1 mg/kg q12h IV) counters alpha-mediated vasoconstriction in brainstem vessels, enhancing perfusion during feeds. VNS is experimental in neonates, SSRIs risk serotonin syndrome with PHOX2B, and butyrate targets gut only.

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